Opinion

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Local adaptation to temperature and the implications for vector-borne diseases

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Vector life-history traits and parasite development respond in strongly nonlinear ways to changes in temperature. These thermal sensitivities create the potential for climate change to have a marked impact on disease transmission. To date, most research considering impacts of climate change on vector-borne diseases assumes that all populations of a given parasite or vector species respond similarly to temperature, regardless of their source population. This may be an inappropriate assumption because spatial variation in selective pressures such as temperature can lead to local adaptation. We examine evidence for local adaptation in disease vectors and present conceptual models for understanding how local adaptation might modulate the effects of both short- and long-term changes in climate.

Why local adaptation?

The impact of climate change on the dynamics and distribution of vector-borne diseases is a topic of frequent and often contentious discussion [1–3]. What has been described as the 'conventional wisdom' [4] holds that climate change will result in dramatic increases in transmission of diseases such as malaria and dengue. Others have argued that shifts in disease prevalence will be negligible, or that disease burdens will even decline in some areas owing to overlooked ecological factors [3,5] or socioeconomic development [6,7]. Notably absent from these discussions, however, is a thorough consideration of adaptation and, in particular, local adaptation to temperature in parasites and vectors.

Local adaptation occurs when there is spatial variation in selection, either due to species interactions or environmental factors such as temperature (see Glossary). Understanding patterns of local adaptation could provide new insights into the impact of climate change on vector-borne diseases. If species are composed of multiple locally adapted populations, then assuming a single species-level response might produce inaccurate predictions for future disease risks. Relative to predictions based on species averages, local adaptation could lead to unexpected increases or decreases in transmission in response to warming (Figure 1). Local adaptation might also act to inhibit evolutionary responses through a reduction in the breadth of genotypic and phenotypic variation present in populations [8,9]. Furthermore, the extent of local adaptation in response to selection across contemporary temperature gradients could provide insights into the likely evolutionary response to future warming. The importance of local adaptation for predicting future change will depend on: (i) the nature of local adaptation, for example, whether populations are adapted in their operative ranges, optimal temperature, or both; (ii) the mean habitat temperature relative to optimal temperature (e.g., [10]); (iii) the

Glossary

Basic reproductive rate (R0): the expected number of infections resulting from the introduction of a single infected individual into a susceptible population. An increase in R_0 corresponds to an increase in disease prevalence.
Clinal patterns: genotypic or phenotypic gradations along a continuous sampling area, for example, with increasing latitude or altitude.
Ecotypes: distinct populations associated with different local environments. Genetic and phenotypic differences between ecotypes are generally inferred to be a result of local adaptation to the respective environments.
Entomological inoculation rate (EIR): the rate at which people are bitten by infectious vectors; typically the number of infectious bites per person per year.
Evolutionary adaptation: we use this term specifically to refer to genetically based changes in phenotypes that are a response to selection arising from a changing climate.
Extrinsic incubation period (EIP): the development time of parasites within the vector.
Local adaptation: adaptation in response to spatially heterogeneous selective pressures. Locally adapted organisms gain higher relative fitness in their native environment compared with foreign environments. Two frequently used approaches for studying local adaptation are common-garden experiments, where organisms are placed in a single environment, and one or a few aspects of the environment are manipulated, and reciprocal transplant, where organisms are moved between habitats. The former approach is often used for studying host–parasite systems, but it fails to capture adaptation to the environment as a whole.
Mesocosm: intermediate-scale experimental settings that incorporate realistic field conditions while still retaining reproducibility and the ability to manipulate variables.
Thermal response curve: phenotypic response over a range of temperatures; the outcome can be a process (e.g., enzyme activity), trait (e.g., size), or measure of fitness. The curve is characterized by its cold and warm extremes (CT_{min} and CT_{max}), the difference between these extremes (operative range), and the peak (T_{opt}). The position on the curve corresponding to the mean habitat temperature experienced by an organism is defined as T_{hab}. If the curve is based on the response of a single genotype over a range of environments then this is equivalent to a reaction norm.
Vector competence: the ability of a vector to acquire and transmit parasites; it is the product of the proportion of bites resulting in transmission from infected hosts to uninfected vectors and from infected vectors to uninfected hosts.

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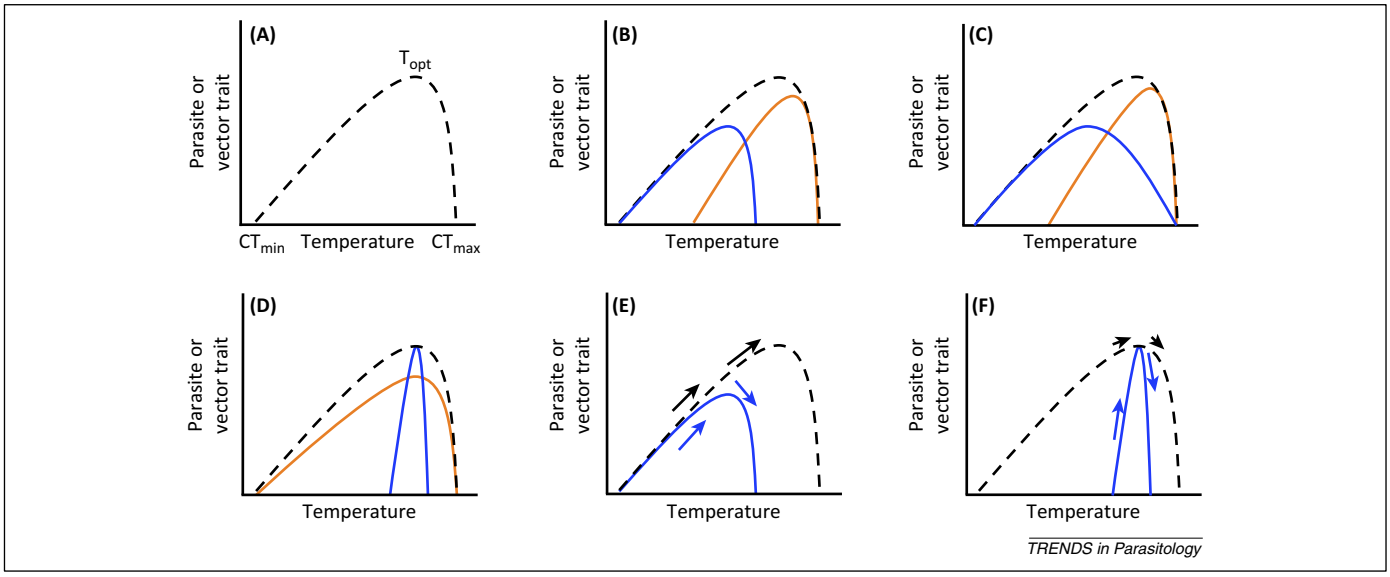

Figure 1. Conceptual models for thermal response curves. **(A)** One curve describing all populations of a species (i.e., no local adaptation). **(B)** Populations are locally adapted in their lower and upper thermal limits (CT_{min} and CT_{max}, respectively) and optimal temperature (T_{opt}). **(C)** Populations are locally adapted in their CT_{min} and T_{opt}, but all populations share a fixed CT_{max}. **(D)** Populations are locally adapted in their CT_{min} and CT_{max}, but share a fixed T_{opt}. The trait value in these examples could be either individual vector or parasite traits, or a composite epidemiological metric such as R_0. Depicted in **(E,F)** are the major insights from these conceptual models. If populations are locally adapted in their T_{opt} (E), the inflection points of the curves relative to temperature differ. If parasites and vectors inhabit a temperature below T_{opt} (i.e., $T_{hab} < T_{opt}$), increases in temperature can have the same effect on local populations as predicted by a species-level thermal response curve. However, if the T_{opt} of a locally adapted population is close to the ambient temperature (i.e., $T_{hab} \approx T_{opt}$), increases in temperature will lead to a decline in the trait value, even if a species-level curve predicts an increase. If populations are locally adapted in the width of the curve, but share a common T_{opt} (F), increases or decreases in trait values will be greater than expected given the broader, species-level curve as depicted by the length of the arrows showing an equivalent transition across the temperature axis. Moreover, because of the reduced operative range of the locally adapted curve, the T_{hab} of any other populations making up the species curve is less likely to fall within the operative range of the locally adapted population.

existence of phenotypic plasticity [11,12]; (iv) interspecific interactions [13]; and (v) ultimately whether local adaptation explains a substantial amount of variation relative to the shorter-term ecological effects of changing temperatures (Box 1). In the following sections we expand on these arguments and outline a framework for integrating local adaptation into research on climate change and infectious diseases.

Local adaptation in existing populations

Temperature has a major influence on the growth and development of ectothermic species, including vectors and the parasites that they harbor [14,15]. Key traits of vectors including development rates [16,17], frequency of blood feeding [18], elements of the immune response [19], and susceptibility to parasite infection [20,21] are all influenced by temperature. Parasite replication and the duration of the extrinsic incubation period (EIP) are also strongly temperature-sensitive [22,23]. Accordingly, environmental temperature has a direct impact on vector-borne disease ecology and epidemiology. However, in addition to the ecological effects, temperature also represents a source of selection. This evolutionary dimension is relevant because, if local adaptation to temperature has occurred in parasite and vector populations, then the effects of temperature could vary across source populations; that is, there will be 'population-by-temperature' or 'genotype-by-environment' interactions, such that the effect of environment, including temperature, on phenotypes will depend on the genetic backgrounds of the vector and parasite. Moreover, if there is coevolution between vector and parasite populations, then local adaptation between the interacting species can be modulated by local conditions, resulting in a three-way 'genotype-by-genotype-by-environment' interaction [24,25].

There is evidence from a variety of species indicating local adaptation to temperature, particularly in ectothermic animals such as insects [26–28]. For example, in *Drosophila melanogaster* collected from Australian populations, and maintained in the laboratory for at least four generations, there was a negative relationship between heat-tolerance and sampling latitude, and a positive relationship between cold-tolerance and sampling latitude [28]. These results suggest that tolerance to extreme temperatures varies between *D. melanogaster* populations and is associated with the local temperature conditions experienced by the populations. Another study of Australian *D. melanogaster* found that simulating 'winter' and 'summer' temperature conditions during rearing affected both heat- and cold-tolerance across populations, but the differences due to rearing treatment were only slightly larger than the difference between the most divergent populations. In addition, there was no interaction between rearing temperature and source population, suggesting that clinal patterns in temperature-tolerance are maintained, regardless of rearing temperature [29].

In contrast to this research in non-disease systems, there has been relatively little research on local adaptation to temperature in vectors of human diseases. However, in *Anopheles* mosquitoes which vector malaria, there is extensive evidence that populations differ in the frequency of genetic markers such as single-nucleotide polymorphisms and chromosomal inversions, and these differences are often associated with habitat, or mosquito ecotypes [30–32].

Box 1. Quantifying the extent of local adaptation

The importance of local adaptation depends on the likely effect size relative to the ecological variation introduced by changes in environmental temperature alone. If local adaptation has resulted in thermal response curves that are substantially different between populations, then a large difference in trait values will occur at a common temperature (Figure I). 'Large', in this context, can be considered a difference similar in scale to the effects of short-term seasonal changes in temperature, or the predicted longer-term changes in temperature owing to anthropogenic warming. Alternatively, thermal response curves might be similar across populations and, thus, any variation introduced by local adaptation might be unimportant relative to predictable changes in life history driven by temperature. If this is the case there might be little need to include local adaptation explicitly in temperature-dependent epidemiological models. However, because composite epidemiological measures such as R_0 depend on several temperature-dependent vector and parasite traits [41], small effects of local adaptation could add up across traits and complicate predictions.

Quantifying the importance of local adaptation relative to changes in temperature requires data from multiple populations over multiple temperatures. Such data are currently lacking for parasites and vectors that cause disease in humans, but are available for a limited number of plant pathogens and invertebrate species. Amongst these examples, some studies reveal no significant differences between populations in response to temperature [73–75], whereas others provide evidence for local adaptation [25–28,56,76–79]. In Table I we provide examples of studies reporting a significant interaction between rearing temperature and source population, or a significant effect of population on heat-tolerance. We have listed either the maximum and minimum differences between populations across all temperature conditions, or the difference between the most and least heat-tolerant populations.

Although the data in Table I are not from human parasite or vector species, they illustrate the wide range of effect sizes that can be observed due to local adaptation. In some cases, the differences in trait values between populations are <1%, whereas in other cases differences are >50%. To put this in context, based on the thermal response curves presented in [41], a 20–50% change in value of a key malaria transmission-related trait such as vector competence or EIP would equate to a 5–10°C change in environmental temperature. Thus, local adaptation could generate effect sizes equal to or greater than those expected from climate warming alone.

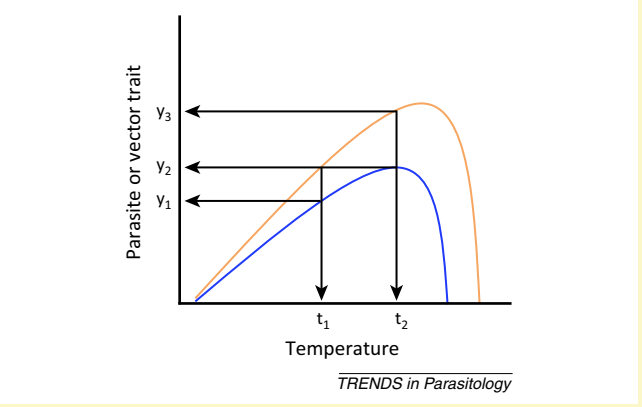

Figure I. Two illustrative population level thermal response curves describing changes in trait values in response to changes in temperature. If these curves are known, it is possible to estimate the effect of local adaptation relative to the effect of changes in temperature. For a given temperature t_2, the difference due to local adaptation is the difference between trait values y_2 and y_3, which is equivalent to change in temperature of t_2–t_1 along the upper orange curve. The magnitude of the effect depends on the specific habitat temperature, relative to the population thermal response curves. For example, at t_1 there is a smaller difference between trait values due to local adaptation (y_1 and y_2) than at temperature t_2 (y_2 and y_3).

Table I. Examples of population differences in response to temperature.

Species	Trait	Number of populations	Percent difference	Refs
Puccinia striiformis (wheat rust)	Proportion germinated spores	2	9–60	[76]
Drosophila buzzatii (fruit fly)	Time to knockdown	2	50	[79]
Drosophila melanogaster (fruit fly)	Time to knockdown	24	47	[28]
Drosophila melanogaster (fruit fly)	Fecundity	3	<1–52[a]	[26]
Papilio canadensis (swallowtail butterfly)	Development rate	2	<1–36[a]	[27]
Podosphaera plantaginis (fungal plant pathogen)	Fitness[c]	3	2–20[a,d]	[25]
Tigriopus californicus (marine copepod)	LT_{50}	8	9	[56]
Anaphes victus (parasitoid wasp)	Maximum development rate	3	6[b]	[78]
Anaphes victus (parasitoid wasp)	T_{opt}, development rate	3	4[b]	[78]
Stator limbatus (seed beetle)	Mass	2	3–5[b]	[77]
Stator limbatus (seed beetle)	Development time	2	<1[b]	[77]

Abbreviations: LT_{50}, 50% lethal temperature; T_{opt}, optimal temperature.

[a]Exact values provided in the text or supplementary material.

[b]Data separated by sex, percent difference is based only on females.

[c]Based on the spread of infection through a population, estimated from measurement of fungal sporulation time and rates of spore production.

[d]Based only on sympatric plant–pathogen combinations.

For example, the 2La inversion increases with aridity along latitudinal clines in Cameroon and Nigeria [33,34], and is associated with improved heat- and desiccation-tolerance in the laboratory [35,36]. Similarly, in tsetse flies (*Glossina* spp.), which vector trypanosomes, populations have been found to differ in desiccation and thermal tolerances [37,38]. There is also evidence of extensive genetic structuring within tsetse fly species [39], and this may be

associated with local environmental conditions [40]. Although these studies show that vector populations differ phenotypically and genetically, possibly as a result of local adaptation to environmental conditions, the implications for disease transmission remain unclear. Moreover, almost nothing is known about local adaptation to temperature in the parasites transmitted by these insect vectors.

Locally adapted thermal response curves

The intensity of vector-borne disease transmission is frequently characterized using the basic reproductive number, R_0. This epidemiological metric depends on a combination of vector and parasite traits including vector density, biting rate, vector competence, adult mortality, and the EIP. Vector density can be further partitioned as the product of the number of eggs laid per female per day, larval development rate, and the probability of vector survival to adulthood. All these traits are affected by temperature [41]. The temperature-dependencies tend to be nonlinear and are generally well described by an asymmetrical, unimodal curve (Figure 1A), where the left and right boundaries of the curve correspond to critical temperature minima (CT_{min}) and maxima (CT_{max}), respectively, and the peak of the curve (T_{opt}) represents the temperature corresponding to optimal performance or maximum trait value [42]. In a recent study examining the temperature-dependence of malaria transmission, the use of such nonlinear relationships in a temperature-dependent model captured well the maximum values of transmission intensity (entomological inoculation rate, EIR) reported across the temperature range for transmission in Africa [41]. However, for a given mean temperature, there was extensive variation in EIR below the maximum. Although many factors could contribute to this variation, the relative importance of differences in thermal responses between mosquito populations – as well as between species – remains largely unexplored.

Local adaptation could potentially occur in any aspect of the thermal response curve, but CT_{max} appears to be less variable and to have a limited capacity for response to selection [43–46]. Furthermore, local adaptation might occur in response to conditions other than mean temperature, including precipitation and vegetation cover [47]. For example, in an analysis of data from almost 400 reptile species, mean temperature was the best climatic predictor only for CT_{min}. The best predictors for CT_{max} and T_{opt} were diurnal temperature variation and precipitation, respectively [48]. Precipitation is clearly a prerequisite for transmission of vector-borne diseases such as malaria, where the vector has an aquatic larval stage. However, in the previously mentioned analysis of EIR across Africa, rainfall explained little of the variation at sites once the minimum rainfall requirement to enable successful breeding had been met [41].

Notably, the shape of a thermal response curve is driven not only by mean temperatures, but also by the magnitude of fluctuation in temperature over time. Tropical species often have narrower thermal response curves compared with temperate species [49], and this reduced range is possibly a result of the relatively constant conditions experienced by tropical species. Similarly, Antarctic marine species experience extremely cold but stable thermal environments, and this is thought to result in increased sensitivity to relatively small variations in temperature – that is, a reduced operative breadth [50]. If narrow thermal response curves are a result of stable environments and not of the tropical environment *per se*, then we expect vector and parasite populations inhabiting stable microclimates to have reduced operative ranges compared to populations inhabiting highly variable thermal environments. For example, there is evidence that experimental exposure to fluctuating temperatures negatively influences the larval development and survival of the malaria vector *Anopheles funestus*, whereas there is no corresponding effect on *Anopheles arabiensis*, another important malaria vector species. This might be because *An. arabiensis* prefers smaller water bodies and therefore experiences more thermally unstable larval environments in the wild [51]. Consequently, latitude alone might not be sufficient to explain or predict local adaptation to temperature in vector and parasite populations with different ecological profiles [52]. Furthermore, small-scale habitat changes such as clearing land or changes in housing structure [53,54] can lead to dramatic variation in local environments within similar geographic contexts and over short periods of time.

Depending on the exact nature of local adaptation, it could alter the pattern and magnitude of response to climate warming (Figure 1). For example, if changing climate causes the habitat to become warmer than the optimal temperature for the local population, but not for the species as a whole, the predicted response would be positive based on the species-level curve whereas, in actuality, a decline could occur. Similarly, if the local population inhabits a much narrower thermal range than the species as a whole, the response to changing temperatures could be larger than expected.

Evolutionary responses to climate change

We have thus far focused on adaptation in contemporary populations. However, the extent to which populations might adapt in response to future climate change is also of considerable interest. Evolutionary adaptation could allow threatened species to persist in the face of climate change, but it could also facilitate range expansions of invasive pest or vector species. For example, models of habitat suitability for *Aedes aegypti* mosquitoes, a vector of yellow fever and dengue, predict a range expansion in Australia with changing climate. This expansion is exacerbated when the capacity for adaptation in egg desiccation-tolerance is included in the simulations [55].

The capacity for adaptive responses depends in part on preexisting variation [8], and local adaptation can reduce the variation present in populations [9]. In a non-disease system, for example, when copepods were sampled from the pacific coast of North America and subjected to high temperatures for 10 generations, northern populations became more heat-tolerant but never to the extent found in copepods from southern populations [56]. Likewise, simulations of range dynamics including local adaptation suggested that cold-adapted genotypes could block the invasion of warmer-adapted genotypes. As a result, species with broader ranges might be more susceptible to extinction due to the persistence of cold-adapted genotypes [57].

The extent to which these results apply to vectors and parasites is unclear, but evolutionary responses to a warming climate could be inhibited by populations that are currently locally adapted to cooler temperatures.

Although the impact of local adaptation on evolutionary potential remains untested in vector and parasite populations, it is a compelling issue given the implications for disease transmission over both the short and long term. If vector and parasite populations at high altitudes or latitudes are adapted to cooler temperatures, they might not respond to warming temperatures in the same way as lower-altitude or -latitude populations. In the short term, climate change might drive populations along their locally adapted thermal response curves, and the differences between these curves will result in different epidemiological outcomes (Figure 1). In the longer term, further adaptation could cause thermal response curves to shift rightwards towards the upper limit of the species, but such shifts could be limited in populations strongly adapted to cooler temperatures because of the absence of genotypes able to cope with warmer temperatures.

The influence of contemporary local adaptation and the capacity for further change in response to warming will depend heavily on rates of environmental change. In principle, ongoing adaptation is more likely to keep pace with environmental change if the rate of change is relatively slow. It remains an open question whether this will be the case, with global surface temperature changes of 2°C or more being projected by the end of this century [58]. Very rapid shifts which might occur through instantaneous changes in habitat features [53,54], however, are more likely to lead to a transient mismatch between locally adapted populations and their environment. Furthermore, although climate change is predicted to increase mean temperature relatively slowly, it will probably also result in more extreme and unpredictable weather, which can constrain successful adaptive responses [59].

Patterns of response to environmental change will be further influenced by plasticity, both in the immediate, short term response to changing temperature as well as in the capacity for an evolutionary response to new environmental conditions [11,60]. Plasticity in response to environmental conditions can result in non-genetic changes in thermal response curves, and these changes could be more rapid, but possibly smaller, than changes due to adaptation [59,61]. For example, laboratory experiments with tsetse flies, *Glossina pallidipes*, and the mosquitoes *Anopheles gambiae*, *An. arabiensis*, and *An. funestus*, show that acclimation can affect tolerance to temperature extremes and desiccation [35,36,62,63], indicating a plastic response in thermal tolerance. Caution is necessary when estimating such plasticity, however, because both the mean values and the variance in critical limits depend on the rate of change in temperature [64]. Plasticity can also have costs, which impose trade-offs with fitness under optimal environmental conditions, and such costs can limit the critical rate of environmental changes that allow for species persistence [12]. When considering local adaptation in vector and parasite populations, it is necessary to understand variation, not only due to plasticity but also in the capacity for plasticity and any costs thereof.

Characterizing local adaptation

Studies of local adaptation have a long history in the field of evolutionary biology and, conceptually, testing for local adaptation is straightforward; it is simply the comparison of populations to test whether individuals are, on average, fitter in their local environments [65]. Nevertheless, there

Box 2. Using laboratory colonies to study adaptation to temperature

The usefulness of established laboratory strains of vectors and parasites is unequivocal; strains that can be maintained in the laboratory over many years provide a valuable tool for disentangling the genetic and physiological mechanisms that contribute to adaptation. However, laboratory populations can differ substantially from their wild counterparts. Colonies often originate from a relatively small sample of wild populations, which creates an initial bottleneck, and additional bottlenecks can occur with subsequent passaging. Consequently, founder effects and genetic drift can reduce diversity in laboratory strains. One study of *An. gambiae* found a depletion of rare alleles (frequency <0.05) and an enrichment of common alleles (frequency >0.50) in laboratory populations compared to a field population [80]. Similarly, a study of *Schistosoma mansoni* also found that parasites isolated in the field had greater allelic diversity and more rare alleles compared to parasites maintained in the laboratory [81].

Another potential issue with laboratory strains is adaptation to the laboratory environment [82]. In *An. gambiae*, the transcriptome of individuals collected from wild populations but reared in the laboratory differed markedly from the transcriptomes of individuals sampled from a laboratory colony. Moreover, many of the genes that were differentially expressed belonged to functional classes that are likely to be important for laboratory versus field conditions, including immunity, host-sensing, and energy balance [83].

Because of the genetic and phenotypic differences between laboratory and wild populations, careful consideration is necessary when using laboratory strains. Broadly speaking, studies that are useful for understanding the responses of vectors and parasites to climate change can be divided into three categories: (i) laboratory experiments testing the immediate effects of variation in temperature on phenotype and/or identifying the genetics underlying thermal responses, (ii) selection experiments in the laboratory that attempt to measure adaptation in response to different temperature regimes imposed over multiple generations, and (iii) spatial and longitudinal studies that sample directly from wild populations to measure phenotypic and genotypic variation over space and time. Laboratory strains can be appropriate for (i) and (ii), but there are several caveats associated with experimental evolution approaches, which can make results difficult to interpret and extrapolate to wild populations (see recent discussion in [84]). Lastly, we note that infection responses may depend on vectors and parasites being co-evolved [85], which is a dimension that is generally absent from laboratory infections.

Research topics that benefit from the use of laboratory colonies:

- Identifying potential genetic targets of temperature-mediated selection.
- Characterizing the physiological traits that determine thermal tolerance.
- Identifying trade-offs that may limit evolution in response to changing climate.

Research topics that benefit from field sampling of wild populations:

- Quantifying the effect of local adaptation relative to other sources of variation.
- Estimating the standing heritable variation present in existing populations.
- Measuring concurrent genetic and environmental change.

are multiple ways to conduct these population comparisons, and methodologies vary in their robustness and interpretation (recently detailed in [66]). As a first step, we suggest sampling vector and parasite populations along latitudinal clines or altitudinal gradients to determine whether reaction norms for various life-history traits differ between populations. Such an approach has been used to characterize local adaptation in heat- and cold-tolerance in *Drosophila* populations (e.g., [28]) as well as cold-tolerance in tsetse flies, *G. pallidipes* [38]. The major advantage of this approach is the capacity for replication by sampling multiple populations along independent temperature gradients. This replication is essential for disentangling the effects of temperature as a selective force, separate from other environmental factors.

Several other approaches for studying evolutionary adaptation in response to climate change have been outlined [9]. In addition to the spatial approach described above, potential approaches include longitudinal studies measuring genetic change over time and experimental evolution in the laboratory. Although similar approaches could be used for vector-borne diseases, it is worth noting that their power depends heavily on sampling from natural populations. Laboratory strains are valuable for developing an understanding of how temperature variation can affect disease transmission and for identifying environmentally relevant genes where variability is informative for predicting adaptive potential [67], but they are not always suitable for parameter estimates of wild populations. Some of the strengths and weaknesses of using laboratory strains are discussed in Box 2.

Another consideration is that vector and parasite populations could coevolve to each other, as well as adapt to their local environment. Three-way interactions between vector population, parasite population, and thermal environment create a challenge for conducting the classic common-garden experiments often used for host–parasite systems, and reciprocal transplant experiments are not feasible with motile organisms. Mesocosm or semi-field experiments might, therefore, offer a reasonable compromise between the need for experimental manipulation while still capturing the essential variation in biotic and abiotic factors that shape selection on vectors and parasites [68].

Concluding remarks

Local adaptation has previously been invoked as a potentially important factor for predicting the effects of climate change on vector-borne disease transmission [69,70], but the issue has not yet been explored in a detailed and systematic way. We have summarized some outstanding questions in Box 3. As a first step towards addressing this knowledge gap, we have outlined a conceptual framework highlighting how local adaptation might shape the responses of vector-borne diseases to climate warming. Based on this framework, we identify three complementary areas for future research. First, empirical studies are necessary to characterize local adaptation to temperature in vector and parasite populations, and to provide validated parameter estimates for the suite of vector and parasite traits that combine to determine transmission intensity. By sampling multiple populations and characterizing their thermal response curves, this will address the pressing question of whether such local adaptation occurs and how much variation is due to local adaptation compared to the variation in climate across the ranges of vectors and parasites. As we have outlined in Box 1, quantifying the difference in trait values between populations is necessary to determine if the extent of local adaptation is likely to have a measurable effect relative to the influence of variation in environmental temperature alone. Second, if substantial differences due to local adaptation are detected, transmission models using modified temperature-dependent relationships should incorporate varying thermal response curves to address whether such variation significantly changes the predicted outcome for disease transmission. Third, properly characterizing local adaptation will require an improved understanding of the microclimates actually experienced by vectors and parasites. For example, a 3 month study examining microclimate in an urban malaria-transmission setting in India found that daily mean temperatures and daily temperature variation differed markedly between a suite of possible mosquito resting habitats, and the temperatures of the resting habitats differed from those recorded at a local weather station. Furthermore, the differences in mean temperature and daily temperature variation were generally greater between the habitats at any one time than over time for a single habitat. Thus, at least during the course of the study, local spatial variation in temperature was greater than temporal variation [71]. Adaptation to fine-scale climate conditions could result in populations that share similar thermal performance profiles, despite inhabiting climates that are dissimilar on a larger scale. Likewise, populations inhabiting habitats that appear similar on a large scale might differ due to local adaptation to microclimate conditions. Because coarse-scale environmental estimates based on measures from remote meteorological stations do not necessarily represent the local conditions in which transmission take place, predictions from standard global climate models will have to be downscaled appropriately to define spatial and temporal variability in temperature at scales relevant to local transmission and local adaptation [71,72].

Box 3. Outstanding questions

- How much variation is there between populations in relevant traits?
- Does this change the output of models?
- Does temperature modulate local adaptation in vector and parasite pairs?

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