Temperature effects on individual-level parasitism translate into predictable effects on parasitism in populations

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ABSTRACT

Parasitism – the interaction between a parasite and its host – is expected to change in a warmer future, but the direction and magnitude of this change is uncertain. One challenge is understanding whether warming effects will be similar on individual hosts (e.g., parasite burden) compared to on population-level parasitism (e.g., prevalence, R_0). Examining thirteen empirical systems, we found a strong positive relationship between the thermal optima of individual- and population-level parasitism. We also found that parasitism thermal optima were close to host performance thermal optima in mosquito – parasite systems but not in non-mosquito – parasite systems. A simple mechanistic model showed how population-level parasitism thermal optima can be similar to individual-level parasitism thermal optima even under conditions where parasite transmission has a considerably higher thermal optimum. These results provide a key step towards finding general rules for how warming temperatures should affect parasitism in individuals, populations, and ecosystems.

24 **INTRODUCTION**

25 Organisms increasingly face temperatures warmer than their optima for physiological
26 performance due to increasing and more variable temperatures caused by anthropogenic climate
27 change. Changes in temperature can alter species interactions (Thomas & Blanford 2003; Kordas
28 *et al.* 2011), and there is growing evidence that climate change has impacted host – parasite
29 interactions and infectious disease dynamics around the world (Koelle *et al.* 2005; Rohr *et al.*
30 2011; Lafferty & Mordecai 2016; Claar & Wood 2020). Indeed, a series of major disease
31 outbreaks in marine species have been linked to climate warming (e.g., seastar wasting disease,
32 Harvell *et al.* 2019; coral die-offs, Bruno *et al.* 2007; and black abalone die-offs, Ben-Horin *et*
33 *al.* 2013). These and other outbreaks have made predicting effects of temperature and changing
34 climate on infectious disease dynamics an urgent priority (Altizer *et al.* 2013). Importantly,
35 temperature affects biological systems across levels of organization, from individual physiology
36 and infection burden to host population and disease dynamics (Fig. 1). This means that
37 understanding the effects of temperature on both individuals and populations is critical for
38 disease mitigation efforts as the world warms. Yet this cross-scale investigation of thermal
39 disease ecology remains an open research gap.

40 Host–parasite systems involve multiple biological processes at more than one level of
41 biological organization: individual host performance, individual parasite performance, host
42 population dynamics, parasite population dynamics within a host, and parasite population
43 dynamics among hosts within a population. Each of these biological processes and the traits that
44 underlie them may be temperature dependent. One challenge for anticipating severe
45 consequences of parasitism is to understand how temperature effects emerge at the host
46 population level even when there can be a range of thermal responses among other biological

47 processes influencing the host and parasite (Fig. 1). For example, multiple host and parasite traits
48 and their respective temperature dependencies (typically described using *thermal performance*
49 *curves*, or TPCs) influence the effects of temperature on individual-level parasitism (Kirk *et al.*
50 2018). Moreover, individual-level parasitism outcomes, such as parasite burden (McCallum *et al.*
51 2017) and parasite-induced host mortality (Day 2001), can affect parasite transmission which
52 subsequently affects population-level parasitism, meaning that the thermal responses of
53 individual- and population-level parasitism are not independent.

54 In a simple case with temperature-independent transmission rates, we may expect the
55 thermal response of population-level parasitism to match the response of individual-level
56 parasitism. However, the temperature dependence of population-level parasitism is unlikely to be
57 so straightforward. Several potentially temperature-dependent traits can affect parasite
58 transmission, including demographic traits that influence host population dynamics and
59 behavioral traits of hosts, and these traits can therefore alter how individual-level parasitism
60 scales to the population level (Fig. 1). For example, activity rate of individuals can vary with
61 temperature (Casey 1976), thereby likely modifying the contact rate between susceptible and
62 infected individuals and the overall transmission rate. Additionally, the density of susceptible
63 hosts, often a key factor in parasite transmission, depends on demographic processes such as
64 birth and death rates, which can be strongly influenced by temperature (Savage *et al.* 2004;
65 Amarasekare & Savage 2012; Bernhardt *et al.* 2018). If the thermal responses of contact rates
66 and the density of susceptible hosts are distinct from the thermal response of individual-level
67 parasitism, we may also expect the thermal response of population-level disease to be distinct
68 from that at the individual level. Understanding the relationship between parasitism at these

levels is critical for understanding how disease dynamics, individual fitness, population dynamics, and evolution will change with climate-driven shifts in parasitism regimes.

One framework for understanding temperature-driven changes in disease centers on the thermal mismatch hypothesis (Cohen *et al.* 2017). Complementary to the approach we outline above and in Figure 1, which emphasizes parasitism at the host individual and host population levels, the thermal mismatch hypothesis relates the thermal response of parasitism to that of the host. The thermal mismatch hypothesis predicts that parasitism is maximized at temperatures away from the host's optimum—i.e., at cool temperatures for warm-adapted species and at warm temperatures for cold-adapted species—at both the host individual and host population levels (i.e., individual- and population-level parasitism TPCs should peak at temperatures offset from the host optimum in what is called a thermal mismatch; Cohen *et al.* 2017, 2019a, b). Thermal mismatches were first documented in amphibian – chytrid fungus (*Batracochytrium dendrobatidis*, *Bd*) systems at both the individual and population levels (Cohen *et al.* 2017). More recently, Cohen and colleagues (2020) analyzed >2000 host–parasite combinations at the population level and found that hosts from cool and warm climates had increased disease prevalence at warm and cool temperatures, respectively (i.e., there was evidence for population-level thermal mismatches). However, outside of amphibian – *Bd* systems, it is unclear if thermal mismatches at the population-level will generally correspond with thermal mismatches at the individual level. How the optimal temperature for both individual- and population-level parasitism compares to the optimal temperature for the host may mediate how climate change will affect host – parasite interactions: decreased host performance at warmer temperatures could either be compounded by increasing parasitism or mitigated by decreased parasitism.

Here, we re-analyze data from thirteen host – parasite systems to first test the hypothesis that the thermal optimum of population-level parasitism (e.g., prevalence, basic reproduction number R_0) is positively related to the thermal optimum of individual-level parasitism (e.g., parasite burden) across host – parasite systems. Next, we compare thermal optima of individual- and population-level parasitism to the thermal optimum of host performance in the thirteen systems to test the hypothesis that thermal mismatches at the individual level correspond with thermal mismatches at the population level. Finally, we use a mechanistic model to illustrate how even under conditions where parasite transmission has a thermal optimum much higher than individual-level parasitism, the thermal optimum of population-level parasitism can still be closer to the individual level than that of transmission. Our findings provide a first step towards finding general rules for how warming temperatures should affect parasitism across biological scale.

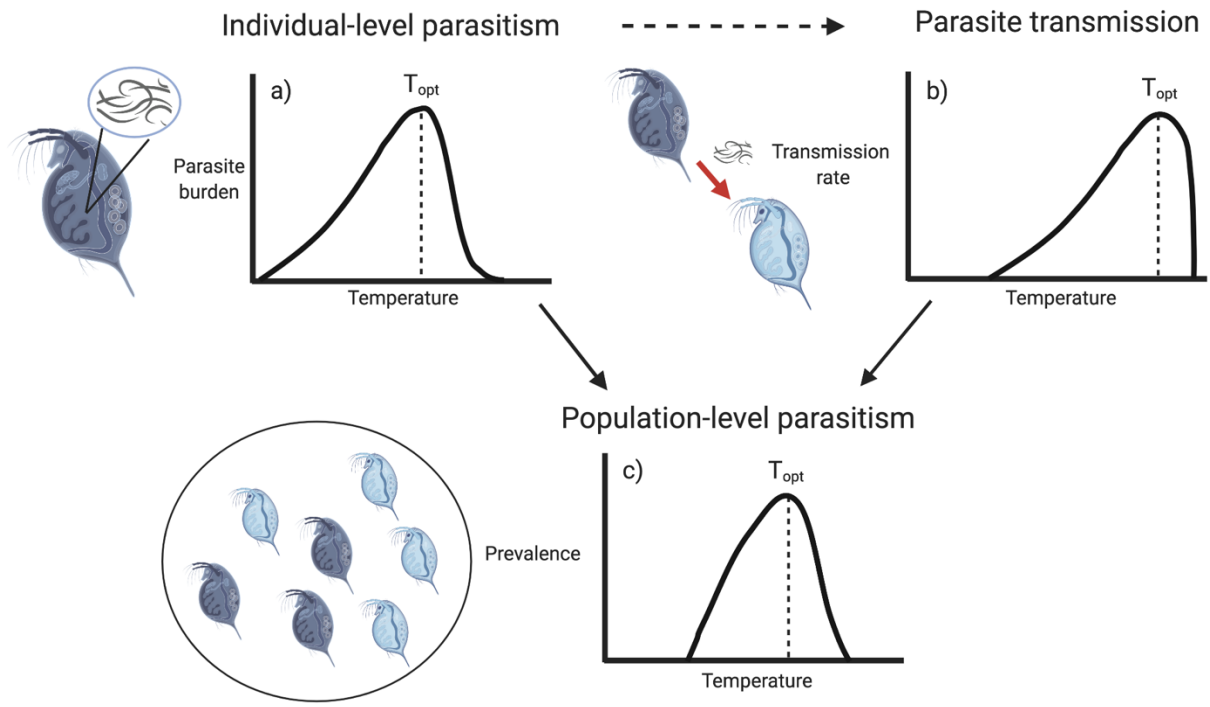

Figure 1. Illustration of a simplified host-parasite system that highlights temperature-dependent processes occur and interact across levels of biological organization. Temperature can influence parasite growth and host defenses against infection, which can ultimately lead to a thermal response for individual-level parasitism (e.g., parasite burden; a). Similarly, temperature can affect contact rates between hosts or hosts and parasites, the probability of infection after contact, and host density, all of which can lead to variation in parasite transmission rate across temperature (b). In some cases, the thermal response of individual-level parasitism (a) may also partially influence the thermal response of parasite transmission (b), as higher parasite burden can confer a higher probability of infection after contact or can alter contact rates between hosts. Together, the thermal responses of individual-level parasitism (a) and parasite transmission (b) will help determine the thermal response of population-level parasitism (e.g., parasite prevalence or the basic reproduction number R_0; c).

133 **METHODS**

134 ***Empirical analyses***

135 We tested the hypothesis that the thermal optimum of population-level parasitism is
136 positively related to the thermal optimum of individual-level parasitism across host – parasite
137 systems. Because it can be difficult to summarize an entire thermal performance curve (TPC)
138 with a single metric, we chose to focus on and compare the temperatures at which the curves are
139 maximized—the thermal optimum (T_{opt})—which is also more widely available for many host –
140 parasite systems than the full TPC. Though it does not represent the complete thermal response,
141 T_{opt} is a useful metric for comparison because it determines the range of temperatures at which
142 warming has a positive effect on parasitism (for $T < T_{opt}$) versus a negative effect (for $T > T_{opt}$).
143 Other metrics, such as thermal breadth and the critical thermal minimum and maximum, would
144 provide additional information about how climate change could affect host – parasite interactions
145 at the range margins. After testing for a relationship in the thermal optima of parasitism across
146 levels, we compared T_{opt} for individual- and population-level parasitism to T_{opt} of host
147 performance to determine if parasitism generally peaked at temperatures away from host thermal
148 optima as predicted by the thermal mismatch hypothesis (Cohen *et al.* 2017, 2019a, b), and if so,
149 whether these thermal mismatches at the individual level corresponded with thermal mismatches
150 at the population level.

151 We sought out systems for which measures of host performance (e.g., lifetime
152 reproduction), individual-level parasitism (e.g., parasite burden, parasite reproduction within the
153 host, time spent infected), and population-level parasitism (e.g., R_0, prevalence) were
154 documented across temperatures, allowing us to identify T_{opt} for each of these measures. While
155 not an exhaustive list, we identified thirteen systems that matched these requirements (Table 1):

156 four mosquito – virus systems (Mordecai *et al.* 2013; Shocket *et al.* 2018a; Tesla *et al.* 2018;
157 Mordecai *et al.* 2019; Shocket *et al.* 2020), four mosquito – malaria parasite systems (Villena *et*
158 *al.* 2020), two *Daphnia* – parasite systems (*D. magna – O. colligata*, Kirk *et al.* 2018, 2020; *D.*
159 *dentifera – M. bicuspidata*, Shocket *et al.* 2018), two amphibian – *B. dendrobatidis* (*Bd*, the
160 causative agent of chytridiomycosis) systems (Cohen *et al.* 2017), and one crab – rhizocephalan
161 barnacle parasite system (*E. depressus – L. panopaei*; Gehman *et al.* 2018). While the mosquito
162 systems are mainly studied with respect to human disease, here we leverage the rich data on their
163 thermal dependence to explore thermal scaling of the mosquito – parasite interaction, which in
164 turn affects transmission to humans. Using these systems, we tested the null hypothesis that there
165 is no relationship between the thermal optima of parasitism across scales by regressing
166 population-level parasitism T_{opt} against individual-level parasitism T_{opt} using the *lm* function in
167 the R package *stats* (R Core Team 2020).

168 Ideally, the metrics used for host performance, individual-level parasitism, and
169 population-level parasitism would be the same across systems; however, that is not the case
170 because different studies report different metrics. For example, the four mosquito–virus systems
171 use R_0 as the metric for population-level parasitism, while the four mosquito–malaria systems
172 use a slightly different metric, transmission suitability, which ignores the temperature-
173 independent human density and human recovery rate parameters. Table 1 lists the metrics used
174 for each system, with further details listed in the Supporting Information. We report a single host
175 T_{opt} value for eleven of the systems, but two host T_{opt} values (individual and population) for the
176 amphibian systems. This is because individual-level parasitism for cold-adapted and warm-
177 adapted amphibians was measured in the lab across one and two host species, respectively
178 (Cohen *et al.* 2017), but the thermal response of population-level parasitism was reported as *Bd*

prevalence in the field as a function of environmental temperature for 235 surveyed species, where cold- and warm-adapted amphibians were categorized as those at locations where 50-year mean temperature was <15°C or >20°C, respectively (Cohen *et al.* 2017). We therefore used separate T_{opt} values for our individual-level host performance (measured as thermal preference in the lab) and population-level host performance (measured as the mean climatic temperature experienced in the field across surveyed species).

While we were generally constrained to use whichever metric of parasitism or host performance was reported for a study, we had access to TPCs of several different metrics in the mosquito – parasite systems. We chose to use infected days as our measure of individual-level parasitism in these eight systems because it is a composite metric of thermal performance data on parasite development rate within the mosquito, vector competence (the mosquito's ability to acquire and transmit the parasite), and mosquito survival. However, we were interested in how this choice affected our findings of how closely T_{opt} of individual-level parasitism matched T_{opt} of population-level parasitism and T_{opt} of host performance. Generally, using vector competence as a metric of individual-level parasitism gave similar results to our main metric of infected days, but parasite development rate tended to exhibit higher T_{opt} values than the other two metrics (Fig. S1). The Supporting Information contains further details on both our model and the methods used to analyze the empirical systems.

202 TABLE 1. Thermal optima of parasitism and host performance for thirteen host – parasite systems.

Host – parasite system	Measure of host performance	Measure of individual-level parasitism	Measure of population-level parasitism	Host performance T_{opt} (°C)	Individual-level parasitism T_{opt} (°C)	Population-level parasitism T_{opt} (°C)	Reference
Culex pipiens – West Nile virus	Adult reproduction weighted by lifespan	Infected days	R_0	15.9	23.5	24.5	Shocket et al. 2020
Aedes aegypti - dengue virus	Adult reproduction weighted by lifespan	Infected days	R_0	27.6	29.3	29.1	Mordecai et al. 2017
Aedes aegypti - Zika virus	Adult reproduction weighted by lifespan	Infected days	R_0	27.7	28.9	28.9	Tesla et al. 2018
Culex annulirostris – Ross River virus	Adult reproduction weighted by lifespan	Infected days	R_0	25.6	24.0	26.4	Shocket et al. 2018a
Anopholes gambiae - *Plasmodium vivax*	Adult reproduction weighted by lifespan	Infected days	Transmission suitability	24.4	23.6	25.0	Villena et al. 2020
Anopholes gambiae - *Plasmodium falciparum*	Adult reproduction weighted by lifespan	Infected days	Transmission suitability	24.4	23.4	25.0	Villena et al. 2020
Anopholes stephensi - *Plasmodium vivax*	Adult reproduction weighted by lifespan	Infected days	Transmission suitability	24.0	23.0	24.6	Villena et al. 2020
Anopholes stephensi - *Plasmodium falciparum*	Adult reproduction weighted by lifespan	Infected days	Transmission suitability	24.0	22.9	24.8	Villena et al. 2020
Cold-adapted amphibians - *Batrachochytrium dendrobatidis*	Temperature preference for individuals; mean climatic temperature for populations †	Parasite growth rate on host	Prevalence	17.9 (10.5) †	26.0	20.5	Cohen et al. 2017
Warm-adapted amphibians - *Batrachochytrium dendrobatidis*	Temperature preference for individuals; mean climatic temperature for populations †	Parasite growth rate on host	Prevalence	23.5 (23.9) †	12.0	15.9	Cohen et al. 2017
Eurypanopeus depressus - *Loxothylacus panopaei*	Uninfected host survival	Parasite lifetime reproduction	R_0	18.3	15.9	14.7	Gehman et al. 2018
Daphnia dentifera - *Metschnikowia bicuspidata*	Uninfected intrinsic growth rate	Spore load	R_0	26.0*	20.0*	26.0	Shocket et al. 2018b
Daphnia magna – *Ordospora colligata*	Lifetime reproduction	Spore load	R_0	16.2*	11.8*	18.8	Kirk et al. 2018, 2020

203 * T_{opt} for these values was determined as the temperature at which the measure was maximized along a range of discrete
204 experimental temperatures, rather than the temperature maximizing the measure along a fitted continuous curve, as is the case for
205 each other value in the table. For discrete temperature range used, see Supporting Information.
206 † Since amphibian measures of individual-level and population-level parasitism were undertaken in different conditions and
207 across a range of populations and species (laboratory experiments and field observations, respectively; Cohen *et al.* 2017), we
208 used separate T_{opt} values for our individual-level host performance (measured as thermal preference in the lab) and population-
209 level host performance (measured as the mean climatic temperature experienced in the field across surveyed species). See
210 methods for further details.

Model

After testing for relationships between parasitism T_{opt} across levels of organization in thirteen empirical systems, we sought to determine whether differences in the thermal responses of transmission rate and individual-level parasitism could lead to large differences in thermal optima between individual-level and population-level parasitism. Based on our findings in the empirical systems, we hypothesized that even if the rates and quantities underlying transmission (contact rate and host density, respectively) had thermal optima that were much higher than individual-level parasitism, the resulting population-level parasitism T_{opt} would still tend closer to individual-level parasitism T_{opt} than to transmission T_{opt}. We explored this using a heuristic model that relates a measure of population-level parasitism, the basic reproduction number R_0, to host and parasite parameters, some of which are measured at the level of the individual host. We modeled different scenarios for temperature dependence of these parameters. We first described a single theoretical thermal performance curve for individual-level parasitism using a Brière function that is maximized at an intermediate temperature (i.e., T_{opt} = 20.6°C). The Brière function (Eq. 1) is an asymmetrical unimodal function that is often used to describe thermal performance in biological systems (Briere *et al.* 1999), where c is a positive rate constant, T_{min} and T_{max} are the minimum and maximum temperatures, respectively, T is temperature, and $B(T)$ is set to 0 when $T > T_{max}$ or $T < T_{min}$. For this example, we define host individual-level parasitism as parasite burden, or the number of parasites within or on the host:

$$B(T) = cT(T - T_{min})(T_{max} - T)^{1/2} \qquad \text{Eq. 1}$$

We next developed a simple trait-based model of population-level parasitism by modeling the basic reproduction number of the parasite (R_0; Eq. 2).

$$R_0 = \frac{\chi(T)\cdot\sigma(B(T))\cdot S(T)}{\mu(T)+\alpha\big(B(T)\big)+\gamma} \qquad \text{Eq. 2}$$

Here, parameters that are functions of individual-level parasitism ($B(T)$) are parasite-induced host mortality (α) and the probability of infection after contact (σ). Background mortality rate of hosts (μ), a function of temperature, and host recovery rate (γ) are both individual-level rates but are assumed here to not be affected by individual-level parasitism. Contact rate between susceptible and infected hosts (χ) and host density (S), both of which are temperature-sensitive but neither of which are assumed to vary with individual-level parasitism, affect parasite transmission (the numerator in Eq. 2). We made this distinction explicit because we wanted to highlight that certain traits can vary with the number of parasites within a host, thereby connecting the individual and population levels, while other traits will affect population-level parasitism independent from individual-level burden.

We assume linear relationships between individual-level parasite burden and both the probability of infection after contact (σ) (though non-linear relationships are also possible; McCallum *et al.* 2017), and parasite-induced mortality (α) (Anderson & May 1978), respectively. Because σ and α are proportional to individual-level parasite burden, both parameters are maximized at the same temperature as burden (T_{opt} = 20.6°C). We also set background mortality rate (μ) to share the same thermal optimum, meaning that μ is minimized at 20.6°C and then increases at colder and warmer temperatures as described by a quadratic function, used rather than an asymmetrical Brière because mortality often shows relatively symmetrical responses across temperature (van der Have 2002; Angilletta Jr. 2009). We assumed that recovery rate was temperature-independent, though that need not be the case in all systems.

To test whether different thermal responses of contact rate between infected and susceptible hosts (χ) and susceptible host density (S) would shift the thermal optima of

population-level parasitism away from the thermal optima of individual-level parasitism, we allowed χ and S to each be described by one of three functions: a Brière function that has identical temperature-dependence as individual-level parasitism (i.e., T_{opt} = 20.6°C), a Brière function that is shifted along the x-axis to peak at higher temperatures (T_{opt} = 30°C), and a temperature-independent function in which the parameter takes the same value (the average value of the Brière functions) at each temperature. We calculated R_0 for each of the nine combinations, resulting in six unique R_0 responses as some combinations are redundant (e.g., the scenario with contact rate peaking at 20.6°C and density peaking at 30°C gives the same R_0 response as the scenario with contact rate peaking at 30°C and density peaking at 20.6°C).

RESULTS

Empirical analyses

The thirteen empirical systems we analyzed demonstrated a range of thermal optima for individual-level parasitism (11.8°C – 29.3°C) and population-level parasitism (14.7°C – 29.1°C). Population-level parasitism T_{opt} was significantly positively associated with individual-level parasitism T_{opt} (Fig. 2; $p = 0.0005$, adjusted $R^2 = 0.65$, $n = 13$). Of the thirteen systems, nine exhibited thermal optima at individual and population levels that were within 2.5°C of each other. The largest difference across scales appeared in the *D. magna – O. colligata* system in which individual-level parasitism peaked at a temperature that is 7°C cooler than the peak for population-level parasitism. Nine of the thirteen systems exhibited higher thermal optima for population-level parasitism than individual-level parasitism, though in most cases this difference was small (Fig. 2; points above the dashed 1:1 line).

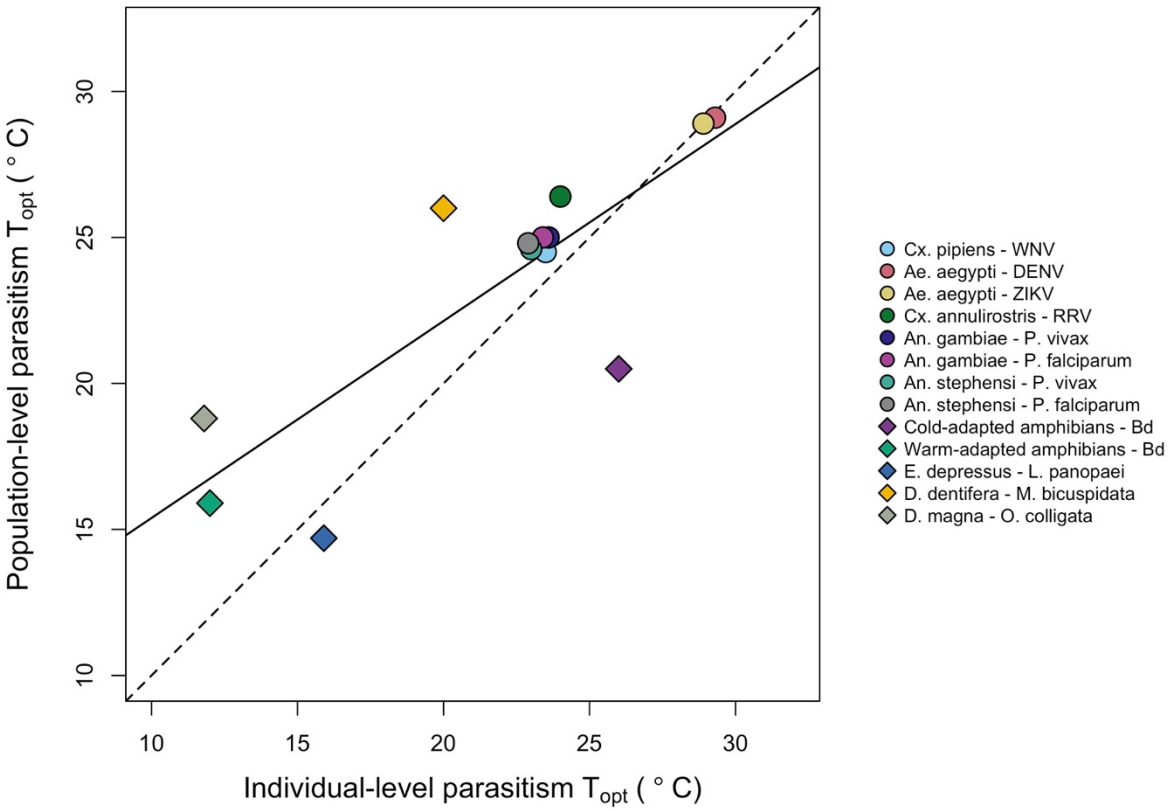

Figure 2. Empirical estimates of the optimal temperature (T_{opt}) for population-level parasitism versus T_{opt} of individual-level parasitism for thirteen host – parasite systems. We found a significant positive relationship between T_{opt} of population-level parasitism and T_{opt} of individual-level parasitism ($p = 0.0005$, adjusted $R^2 = 0.65$; solid line), and found that nine of the thirteen systems exhibited higher thermal optima for population-level parasitism than for individual-level parasitism (i.e., fall above the dashed 1:1 line). Circles represent mosquito – parasite systems and diamonds indicate non-mosquito – parasite systems.

Next, we compared how the thermal optima of parasitism at both levels compared to T_{opt} for host performance (Fig. 3). If a system was situated at the origin in Fig. 3, it would have individual-level parasitism, population-level parasitism, and host performance maximized at the same temperature (i.e., no thermal mismatches exist). Displacement from the origin represents parasitism peaking at temperatures away from where host performance peaks (i.e., thermal mismatches at one or both levels). We found that over half (7/13) of the systems were situated close to the origin (within Euclidian distance of 2.5°C, represented by the circle in Fig. 3).

However, all seven of these are mosquito – parasite systems, with the only mosquito system exhibiting strong thermal mismatches being *Cx. pipiens* – West Nile virus, in which both infected days and R_0 peak at temperatures nearly 10ºC higher than host performance (Fig. 3). The cold adapted amphibian – *B. dendrobatidis* system was also situated in the far upper-right quadrant (Fig. 3), showing strong thermal mismatches at both levels of parasitism, as shown in Cohen et al. 2017. Two systems showed cold-temperature mismatches at both levels: the crab *E. depressus – L. panopaei* and warm-adapted amphibian – *B. dendrobatidis* host – parasite systems. The zooplankton *D. dentifera – M. bicuspidata* system exhibited a thermal mismatch at cold temperatures at the individual level but no mismatch at the population level. Finally, the zooplankton *D. magna – O. colligata* system showed a distinct pattern of differing thermal mismatches across scales (Fig. 3): individual-level parasitism was maximized at a lower temperature than host performance while R_0 peaked at a higher temperature than host performance. None of the thirteen systems showed positive individual-level mismatches but negative population-level mismatches; therefore, the lower-right quadrant of Fig. 3 is empty. The six systems that experienced thermal mismatches generally supported our hypothesis that mismatches would occur at both scales and in the same direction, as more of these systems were situated in the upper-right or lower-left quadrants of Fig. 3 compared to the upper-left or lower-right quadrants.

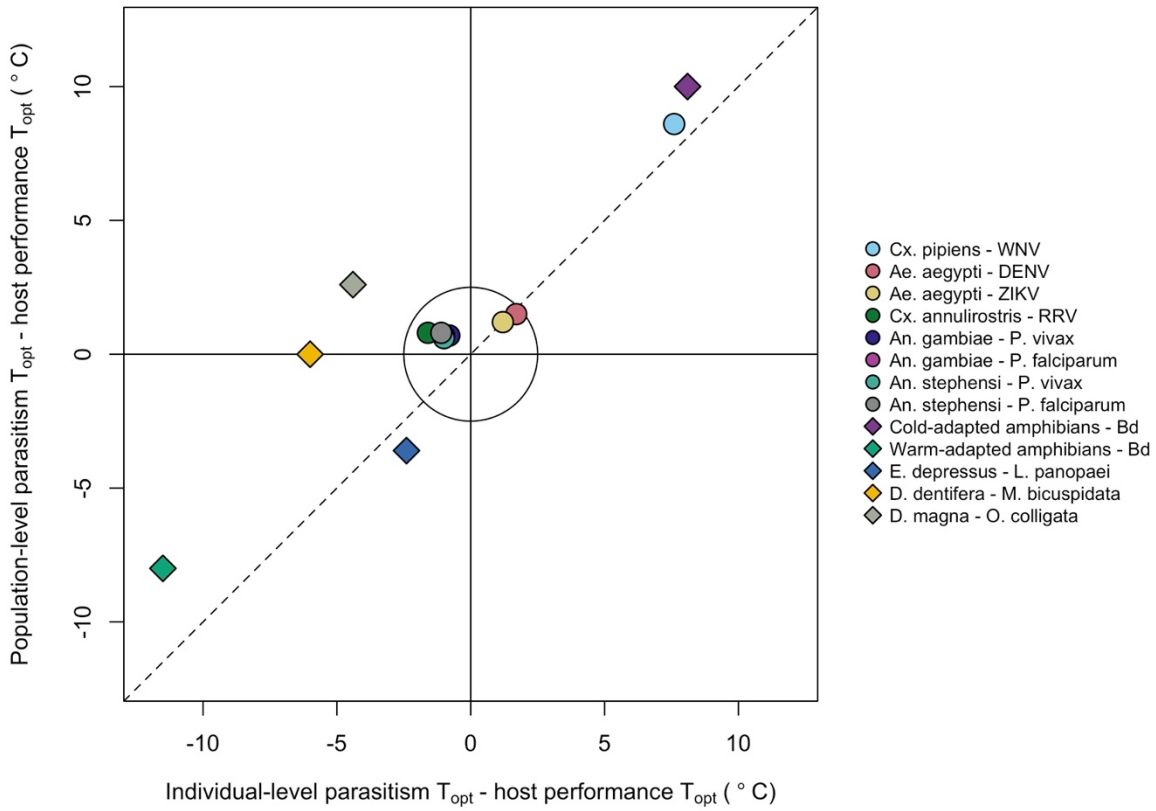

Figure 3. Thermal matches and mismatches tended to be correlated across scales. The difference between T_{opt} of population-level parasitism and host performance (y-axis) is plotted against the difference between T_{opt} of individual-level parasitism and host performance (x-axis) for thirteen host – parasite systems. Systems situated at the origin had population-level parasitism, individual-level parasitism, and host performance all maximized at the same temperature (i.e., no thermal mismatches exist), while displacement from the origin represents parasitism peaking at temperatures away from where host performance peaks (i.e., thermal mismatches at individual or population levels). All seven of the system situated close to the origin (within Euclidian distance of 2.5°C, represented by the black circle) were mosquito – parasite systems (circles; in contrast to systems with non-mosquito hosts: diamonds).

Model

We explored different scenarios for how thermal responses of traits related to transmission could shift the thermal optimum of population-level parasitism away from that of individual-level parasitism. As expected, we found that when contact rate and host density share the same thermal optimum as individual-level parasitism or are temperature-independent, population-level parasitism is also maximized at this temperature (20.6°C; Fig. 4). Alternatively, if either one of these traits has a thermal optimum that occurs at a higher temperature than individual-level parasitism, this difference can pull the population-level parasitism thermal optimum away from that of the individual level and towards the thermal optimum of the trait (30.0°C; Fig. 4). However, even in the most extreme scenario we explored in which both contact and host density peaked at 30.0°C (nearly 10°C warmer than individual-level parasitism T_{opt}), we found that R_0 T_{opt} occurred at a temperature only 3.3°C warmer than individual-level parasitism T_{opt} (Fig. 4). Intuitively, this is because population-level R_0 is constrained by individual-level parasite burden, which is equal to 0 at and beyond its critical thermal maximum temperature of 25°C, therefore leading to $R_0 = 0$ at and above 25°C in all nine scenarios (Fig. 4c) regardless of the thermal response of contact rate and host density.

In addition to small changes in the thermal optima, we found that the thermal response of contact rate and host density affected the magnitude of population-level parasitism. For example, when both contact rate and host density followed identical thermal responses to individual-level parasitism, it led to a maximum R_0 value that was nearly double the second highest maximum R_0 value (Fig. 4c). The three scenarios in which both contact rate and host density did not share a thermal optimum with individual-level parasitism and were either temperature-independent or peaked at 30.0°C resulted in the three lowest maximum R_0 values (Fig. 4c).

In sum, the model confirms the intuition that if rates or quantities that are related to parasite transmission rate, such as contact rate and host density, are either temperature-independent or have a thermal response that peaks at the same temperature as individual-level parasitism, we should expect T_{opt} of population-level parasitism to match that of individual-level parasitism. By contrast, if these key rates and quantities show thermal responses distinct from individual-level parasitism, it can lead to differences in the thermal responses of parasitism across biological scale, though we may still expect the thermal response of individual-level parasitism to constrain the population-level response from exhibiting a T_{opt} that is considerably different from the T_{opt} of individual-level parasitism.

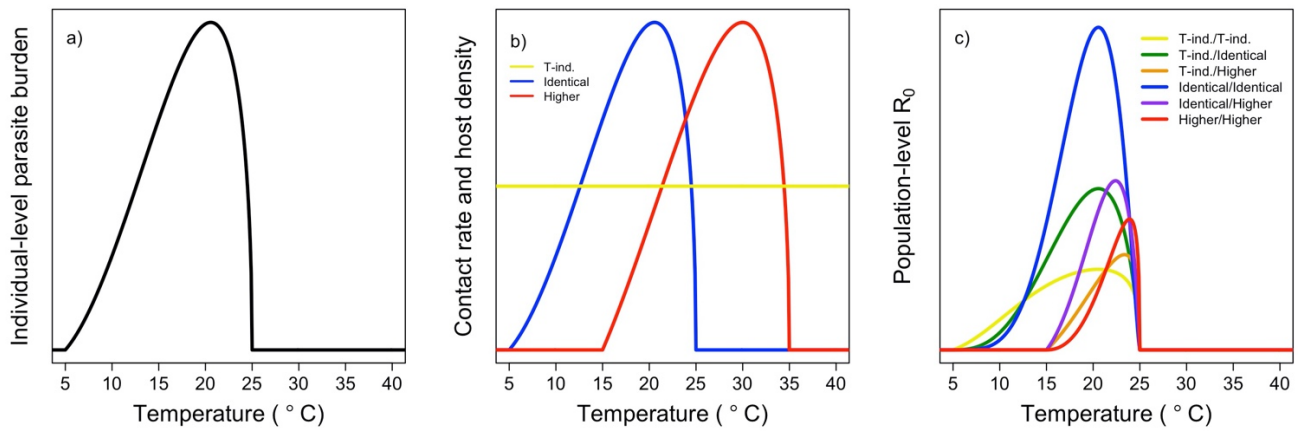

Figure 4. Individual parasite burden and transmission-related traits combine to determine the temperature-dependence of population-level R_0. Individual-level parasitism (a) interacts with traits and quantities related to transmission such as contact rate and host density (b) to determine population-level parasitism (c). We explored how one specific thermal response of individual-level parasite burden (a) can interact with different combinations of responses for contact rate and host density that were either temperature-independent (yellow line, b), temperature-dependent and peaking at the identical temperature as burden (blue line, b), or temperature-dependent and peaking at a higher temperature than burden (red line, b). These combinations generated six unique thermal responses for R_0 (c). The scenario with the warmest T_{opt} for R_0 (red line, c) exhibited a thermal optimum that was only 3.3°C warmer than individual-level parasitism T_{opt} (a).

DISCUSSION

Temperature impacts parasitism at the individual host level by influencing parasite growth and host defenses against infection, ultimately leading to differential levels of parasite burden across temperature. This thermal response of individual-level parasitism is intrinsically tied to the thermal response of parasitism at the population level (Fig. 1, 4). In our examination of thirteen empirical systems, we found a significant positive relationship between T_{opt} for parasitism at the two levels (Fig. 2). Additionally, while individual- and population-level parasitism both peaked at temperatures away from the host optimum in some systems, supporting the thermal mismatch hypothesis, this was not the case in seven of the eight mosquito – parasite systems (Fig. 3). Generally, our results suggest that information on the temperature-dependence, and specifically the thermal optima, of a host – parasite system at either the individual- or population-level should provide a useful—though not quantitatively exact—baseline for predicting temperature-dependence at the other level in a variety of host – parasite systems.

We found a significant positive relationship between T_{opt} of population-level parasitism and T_{opt} of individual-level parasitism and found that in the majority of systems, population-level parasitism tended to peak at warmer temperatures than individual-level parasitism (Fig. 2). The greatest differences in T_{opt} were observed in the zooplankton *D. magna – O. colligata* system, cold-adapted amphibians – *B. dendrobatidis* system, and the zooplankton *D. dentifera – M. bicuspidata* system (Fig. 2, Table 1). It is difficult to definitively parse out the causes of T_{opt} differences in the two amphibian systems since individual-level *Bd* parasitism was measured on either one or two frog species in the lab while population-level prevalence by the same parasite was synthesized across many field studies encompassing 235 host species (Cohen *et al.* 2017). However, the *D. magna – O. colligata* system can provide an illuminating example. Contact rate

in this host – microsporidian parasite system is maximized at 30.1°C (Kirk *et al.* 2019). This is nearly 20°C warmer than T_{opt} for individual-level parasitism (11.8°C) and is thus one of the key factors that pulls the thermal optimum of R_0 away from individual-level parasitism T_{opt} to a warmer temperature (18.8°C). Moreover, in the mosquito – parasite systems, which tended to have high population-level parasitism T_{opt} (Fig. 2), biting rate—a driver of host contact—usually has one of the highest optimal temperatures of all measured traits (Mordecai *et al.* 2013, 2017, 2019; Shocket *et al.* 2018a, 2020; Villena *et al.* 2020). These empirical systems are in line with our model results that show that transmission-related traits with high thermal optima can somewhat shift thermal optima of R_0 away from that of individual-level parasitism, though not to such an extent that population-level parasitism will peak at the same temperature as the transmission-related traits (Fig. 4). However, these few systems do not exhaustively represent all host – parasite systems. More empirical work measuring thermal performance of traits related to parasite transmission in other types of host – parasite systems, such as those with helminth or bacterial parasites, can help to broaden our perspective on scaling of parasitism from individuals to populations.

The thermal mismatch hypothesis (Cohen *et al.* 2017, 2019a, b, 2020) has been proposed as an approach for understanding the effects of climate change on host – parasite systems, predicting that T_{opt} for parasitism should occur at temperatures away from host T_{opt} (i.e., a thermal mismatch) due to larger organisms (hosts) generally having narrower thermal breadth than smaller organisms (parasites; Rohr *et al.* 2018). While thermal mismatches have been shown to occur widely at the population level across systems (Cohen *et al.* 2020), we tested the hypothesis that thermal mismatches at the individual level will correspond with similar thermal mismatches at the population level. Of the five non-mosquito systems we explored—including

the two amphibian – *Bd* systems investigated previously (Cohen et al. 2017)—parasitism tended to peak at temperatures away from the host's optimum in each, meaning these systems experience thermal mismatches at one or both levels. If our hypothesis was to be supported, we would expect most systems experiencing thermal mismatches to fall in the upper-right or lower-left quadrants of Fig. 3, rather than the upper-left or lower-right. We found that this was generally the case, with two systems experiencing thermal mismatches at warmer temperatures at both levels (Fig. 3; upper-right) and two systems experiencing thermal mismatches at cooler temperatures at both levels (Fig. 3; lower-left). The major exception was the *D. magna – O. colligata* system, in which host T_{opt} occurs at a temperature intermediate to the relatively cool T_{opt} for individual-level parasitism and relatively warm T_{opt} population-level parasitism (Fig. 3; upper-left). The other zooplankton host system we explored, *D. dentifera – M. bicuspidata,* exhibited a thermal mismatch at cooler temperatures at the individual level but no mismatch at the population level.

Evidence for thermal mismatches occurring at either level was weak in the mosquito – parasite systems. Seven of eight mosquito – parasite systems were situated close to the origin (Fig. 3), suggesting that parasitism and host performance are maximized at close to the same temperatures and that no thermal mismatches are occurring at either level. The exception, *Cx. pipiens* – West Nile virus, in which parasitism at both levels peaks at temperatures much higher than host performance because *Cx. pipiens* adult lifespan is much longer at low temperatures (Shocket *et al.* 2020), is a system with a predominantly temperate distribution in North America, compared to more the sub-tropical and tropical distributions of malaria, dengue, Zika, Ross River virus, and their respective mosquito vectors. Notably, when testing for thermal mismatches at the population level, Cohen *et al.* (2020) also found that thermal mismatch effects were strongest in

systems without vectors (or intermediate hosts). Why we observed this pattern in mosquito systems will require further investigation in different vector-borne systems across biological scale, including those systems with non-mosquito vectors such as ticks or sandflies.

We propose that situating host – parasite systems along both individual- and population-level axes as in Fig. 3 can provide a diagnostic framework for looking for thermal mismatches across biological scales. While our results do not conclusively address how general thermal mismatches at both levels may be, our finding that individual- and population-level T_{opt} are strongly related (Fig. 2) provides a general rule of thumb that, if a host – parasite system does experience thermal mismatches, they are likely to occur at both scales and in the same direction (though note the exception of *D. magna – O. colligata*; Fig. 3). This has major implications for our understanding of the effects of climate change on host-parasite systems, because if T_{opt} for both individual- and population-level parasitism is at a higher temperature than T_{opt} for host performance, warming may not only decrease host performance itself, but also lead hosts to experience increasing effects of parasitism (Fig. 3; upper-right quadrant). Alternatively, if T_{opt} for parasitism is at temperatures cooler than host performance optima, decreased host performance at warmer temperatures may be partially offset by decreased effects of parasitism (Fig. 3; lower-left quadrant). This is in line with recent projections that climate change may lead to hosts from cooler and warmer climates experiencing increased and decreased parasitism, respectively (Cohen *et al.* 2020).

To better understand how temperature affects parasitism and to make better predictions for how climate change will affect host – parasite systems, we highlight two important research directions. First, when possible, studies that use experiments to investigate effects of temperature at the individual level can also be used to parameterize simple models of population-level

parasitism (a 'bottom-up' approach). Second, field studies investigating population-level parasitism under different climate or weather conditions, often undertaken by measuring the proportion of individuals infected, should also aim to record measures of individual-level parasitism (a 'top-down' approach). The best direct measure would be to record the parasite burden or an analogous metric measuring the parasite on or within the host, but in cases where this is not possible, indirect measures such as host condition may still be informative.

Overall, we found a strong relationship between thermal optima of parasitism across scales in the thirteen systems we analyzed, and our results suggest that in many host – parasite systems, individual-level outcomes may beget similar outcomes across temperature at the population level. Many studies have investigated the effects of temperature on parasitism (reviewed, for example, in Marcogliese 2008; Lafferty 2009; Rohr *et al.* 2011; Altizer *et al.* 2013; Lafferty & Mordecai 2016; Claar & Wood 2020), though often at only one of either the individual or population levels. Our findings build upon these studies by emphasizing that how temperature affects individual-level parasitism can scale up to affect parasite transmission, prevalence, and the potential for epidemics in host populations. While this is an important first step toward predicting the effects of climate change on systems by allowing researchers to primarily focus on parasitism at one level, future empirical work that investigates the thermal dependence of a wider range of host – parasite systems across scales, including in systems with different types of hosts (e.g., plants, non-amphibian vertebrates) and other types of parasites (e.g., helminths, bacterial parasites), will be necessary for investigating the generality of this result. More generally, tying together more theory and empirical results to provide general predictions for how climate change will affect parasitism from individuals to ecosystems in a

diverse range of hosts and parasites remains an urgent priority as accelerating climate change makes likely catastrophic temperature increases of > 2°C increasingly likely by 2050.

ACKNOWLEDGEMENTS

We thank members of the Mordecai and O'Connor labs for valuable feedback on the manuscript. EAM and DK were funded by NIH (R35GM133439); EAM was funded by NSF (DEB-1518681 and DEB-2011147, also supported by the Fogarty International Center), a King Center for Global Development seed grant, and a Terman Award. Figure 1 was created using BioRender.com.

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