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

ABSTRACT

Parasitism 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 parasitism within or on individual hosts (e.g., parasite load) compared to on population-level parasitism (e.g., prevalence, R_0). We adapted a simple temperature-dependent model and simulated several scenarios for individual- and population-level parasitism. Our model found that small differences in the underlying biology of host–parasite systems can substantially alter the expected relationship between the thermal optima of parasitism across levels of organization. In thirteen empirical host–parasite systems, we found a strong positive correlation between the thermal optima of individual- and population-level parasitism, suggesting that the effects of warming on parasitism may often be in the same direction across levels. 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. 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.

INTRODUCTION

Climate change is causing organisms to increasingly face temperatures warmer than their optima for physiological performance. These changes in temperature can alter species interactions (Thomas & Blanford 2003; Kordas *et al.* 2011), and there is now growing evidence that climate change has impacted interactions between individual hosts and their parasites as well as parasite dynamics and outbreaks in host populations (Koelle *et al.* 2005; Bruno *et al.* 2007; Rohr *et al.* 2011; Ben-Horin *et al.* 2013; Lafferty & Mordecai 2016; Harvell *et al.* 2019; Claar & Wood 2020). Predicting the effects of temperature and changing climate on infectious disease is an urgent priority (Altizer *et al.* 2013), yet research tends to focus on the level of either an individual host's biology or of host populations, but rarely both.

Understanding how individual-level *per capita* interactions in host–parasite systems scale to the population level is key for mitigating disease (Fenton 2008). Indeed, the many biological processes that occur in host–parasite systems occur across more than one level of biological organization, and bridging these levels is an important research area (Handel & Rohani 2015). These processes include those that affect individual host performance, individual parasite performance, host population dynamics, parasite population dynamics within a host, and parasite population dynamics among hosts within a population. Crucially, each of these processes and the traits that underlie them may be temperature dependent. However, whether we should expect the effects of temperature on individual-level parasitism (e.g., on parasite load or on within-host parasite population growth rate) to match the effects of temperature on population-level parasitism (e.g., on parasite prevalence or on the basic reproduction number R_0) remains an open research gap, as does whether small differences in the underlying host–parasite interaction or thermal biology should affect how thermal effects scale between levels. Understanding the

effects of temperature on both individuals and populations is critical for disease mitigation efforts as the world warms because if the effects are similar across levels of organization, we can leverage observations of the thermal dependence of parasitism at one level to inform predictions in more complex systems. However, if effects differ across levels, using observations on the thermal dependence at one level to extrapolate to the other could provide erroneous predictions for how warming will affect disease in a system.

Obtaining, summarizing, and comparing entire thermal responses can be difficult and sometimes unfeasible. Fortunately, most thermal response curves have a characteristic non-linear shape with a single thermal optimum at which performance is maximized (T_{opt}) (Huey & Stevenson 1979; Huey & Kingsolver 1989; Angilletta 2006). In this study, we therefore focus on and compare thermal optima, as this data is more widely available for many host–parasite systems than full thermal response curves. Though it does not represent the complete thermal response, T_{opt} is a useful metric for comparison both across levels of organization and among taxa because it determines the range of temperatures at which warming has a positive effect on parasitism (for $T < T_{opt}$) versus a negative effect (for $T > T_{opt}$). Other metrics, such as thermal breadth and the critical thermal minimum and maximum, would provide additional information about how climate change could affect host–parasite interactions at the margins of thermal tolerance.

The thermal optima of parasitism could either match or differ across biological levels of organization. First, we may expect the optimal temperature for parasitism at the individual level to reflect the optimal temperature of parasitism in populations because processes across the two levels can be linked (Ewald 1983; Frank 1996; Mideo *et al.* 2011; Handel & Rohani 2015). For example, if the rate-limiting or rate-determining process for population-level parasitism is

directly related to individual-level parasitism, the effects of temperature on individual-level parasitism may propagate directly to the population level. From this perspective, the thermal optima of parasitism at the two levels are unlikely to be independent in many systems. This could occur if the number of parasites within a host (i.e., individual-level parasitism) is the rate-determining process affecting the rate of parasite transmission (Ben-Ami *et al.* 2008; McCallum *et al.* 2017) or the rate at which hosts are killed by the parasite (Day 2001), both of which subsequently affect population-level parasitism metrics such as R_0 or prevalence (Anderson & May 1979).

On the other hand, temperature may not have the same effects across levels even in cases where population-level parasitism is dependent on the dynamics of individual-level parasitism. Indeed, when considering the effects of anti-parasite treatments instead of temperature, models and field experiments have shown that the effects of treatments on individual hosts do not necessarily lead to equivalent effects in host populations (Fenton 2013; Pedersen & Antonovics 2013). The process of parasite transmission among hosts can potentially decouple population dynamics of hosts and parasites from dynamics within host individuals, particularly if the traits that shape the transmission process are temperature dependent. For example, individual activity rate can vary with temperature (Casey 1976), thereby modifying the contact rate between susceptible and infected individuals and the overall transmission rate. If the thermal optima of traits that affect transmission are much warmer or much cooler than that of individual-level parasitism, we may expect the thermal optimum of population-level parasitism to be pulled away from the individual-level optimum towards that of these traits.

Beyond exploring correlations between the thermal optima of parasitism across levels, considering how the optima for parasitism are related to the optima for their uninfected potential

hosts can provide a more holistic view of how warming should affect a host–parasite system. One potential framework for doing this is the thermal mismatch hypothesis (Cohen *et al.* 2017). This 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 thermal responses 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 et al. (2020) analyzed >2000 host–parasite combinations at the population level and found evidence for population-level thermal mismatches: hosts from cool climates had increased disease prevalence at warm temperatures and vice versa. However, outside of amphibian–*Bd* systems, it is unclear if systems with thermal mismatches at the population level will also exhibit thermal mismatches at the individual level. How the optimal temperatures for individual- and population-level parasitism compare 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 first develop simple temperature-dependent models for individual-level parasitism and population-level parasitism to examine the temperature dependence of parasitism across levels. We then simulate several different scenarios—in which thermal responses of population-level processes are or are not related to the thermal responses of individual-level parasitism—to investigate how changes in the underlying processes and thermal biology of a

host–parasite system can alter the expected relationship between parasitism T_{opt} across levels. Next, after using our model to explore possible relationships between parasitism T_{opt} across levels, we sought to describe the observed relationship in nature. We identify thirteen systems that match our data requirements and re-analyze the data to investigate if the thermal optimum of population-level parasitism is positively related to the thermal optimum of individual-level parasitism across empirical host–parasite systems. Finally, we compare thermal optima of individual- and population-level parasitism to the thermal optimum of host performance in the thirteen systems to test if the thermal mismatch hypothesis holds across levels in these systems. Our findings provide a first step towards finding general rules for how warming temperatures will affect parasitism across biological levels of organization.

METHODS

Model

To formalize our intuitive arguments for why we may or may not expect the thermal optimum of parasitism in individual hosts to occur at the same temperature as the thermal optimum for parasitism in host populations, we adapted a simple temperature-dependent model for disease spread in a population that can be related to the thermal response for parasitism in an individual host. Here, we define host individual-level parasitism as parasite load, or the number of parasites within or on the host.

We first described the thermal response of individual-level parasitism using a Brière function. 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 $T < T_{min}$.

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

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

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

Here, χ is the contact rate between susceptible and infected hosts, σ is the probability of infection after contact, S is the density of susceptible hosts, μ is the background mortality rate, α is the parasite-induced mortality rate, and γ is the host recovery rate. We modeled all six parameters as temperature-dependent, and for all parameters except μ(T) we assumed thermal responses described by the Brière function (the same functional form used to describe the thermal response of individual-level parasitism; Eq. 1). We modeled the thermal response of background mortality rate using a concave-up quadratic function where mortality is minimized at the thermal optimum and increases at cooler and warmer temperatures, as mortality often shows relatively symmetrical responses across temperature (van der Have 2002; Angilletta 2009).

We simulated the model by randomly drawing different thermal response function parameters $\{c, T_{min}, T_{max}\}$ for each temperature-dependent trait (individual-level parasitism and parameters in Eq. 2). For each of 1000 simulations, T_{min} was drawn from a uniform distribution between 0-15, T_{max} was equal to T_{min} plus a value drawn from a uniform distribution between 10-20, and the rate constant c was drawn from a uniform distribution between 0.5-1.3. Similarly, for the concave-up quadratic function that describes background mortality rate, in each of the 1000 simulations we drew parameter a from a uniform distribution between 0.0008-0.0009, b from a uniform distribution between 0.02-0.03, and c from a uniform distribution between 0.1-1.

Thermal responses were scaled to realistic magnitudes for each temperature-dependent trait (e.g., all thermal responses for the contact rate parameter were scaled by a factor of 0.001), and we set background mortality rate to a minimum of 0.005 (see Supporting Information for more detail).

To explore if changes in the underlying biology of the host–parasite systems can alter the expected relationship between parasitism T_{opt} across levels, we modeled four main scenarios: (1) all temperature-dependent traits were independent; (2) parasite-induced mortality rate was proportional to parasite load (Anderson & May 1978); (3) per-contact infection probability was proportional to parasite load (McCallum *et al.* 2017); and (4) both parasite-induced mortality and per-contact infection probability were proportional to parasite load. We also explored seven additional model scenarios to determine the sensitivity of our results to our assumptions regarding both thermal response shape and the structure of the mechanistic model for population disease spread. These scenarios included changes to how thermal response function parameters were drawn, the breadth of the thermal response functions, the minimum parasite load across the temperature range, and the model used to describe population-level disease spread (see Supporting Information for more details). While the model scenarios explored in the main text and the Supporting Information represent only a small subset of the possible relationships between the thermal dependence of parasitism across levels, our aim was to investigate whether small changes to the underlying biology can result in qualitatively different patterns in the relationship of thermal optima in parasitism across levels, rather than to exhaustively explore potential outcomes.

188 ***Empirical systems***

189 After using our model to investigate potential relationships for the thermal optima of
190 parasitism across levels, we sought to describe the observed relationship in empirical systems.
191 We searched for systems for which measures of host performance (e.g., lifetime reproduction),
192 individual-level parasitism (e.g., parasite load, parasite reproduction within the host, time spent
193 infected), and population-level parasitism (e.g., R_0, prevalence) were documented across
194 temperatures, allowing us to identify T_{opt} for each of the measures. While not an exhaustive list,
195 we identified thirteen systems that matched these requirements (Table 1): four mosquito–virus
196 systems (Mordecai *et al.* 2013; Shocket *et al.* 2018a; Tesla *et al.* 2018; Mordecai *et al.* 2019;
197 Shocket *et al.* 2020), four mosquito–malaria parasite systems (Villena *et al.* 2020), two
198 *Daphnia*–parasite systems (*D. magna–O. colligata*, Kirk *et al.* 2018, 2020; *D. dentifera–M.*
199 *bicuspidata*, Shocket *et al.* 2018b), two amphibian–*B. dendrobatidis* (*Bd*, the causative agent of
200 chytridiomycosis) systems (Cohen *et al.* 2017), and one crab–rhizocephalan barnacle parasite
201 system (*E. depressus–L. Panopaei*, Gehman *et al.* 2018). While the mosquito systems are mainly
202 studied with respect to human disease, here we leverage the rich data on their thermal
203 dependence to explore thermal scaling of the mosquito–parasite interaction, which in turn affects
204 transmission to humans.

205 Host performance, individual-level parasitism, and population-level parasitism were
206 measured using different metrics in different systems (Table 1; Supporting Information). We
207 report a single host T_{opt} value for eleven of the systems, but two host T_{opt} values (individual and
208 population) for the amphibian systems. This is because individual-level parasitism for cold-
209 adapted and warm-adapted amphibians was measured in the lab across one and two host species,
210 respectively (Cohen *et al.* 2017), but the thermal response of population-level parasitism was

211 reported as *Bd* prevalence in the field as a function of environmental temperature for 235
212 surveyed species, where cold- and warm-adapted amphibians were categorized as those at
213 locations where 50-year mean temperature was <15°C or >20°C, respectively (Cohen *et al.*
214 2017). We therefore used separate T_{opt} values for our individual-level host performance
215 (measured as thermal preference in the lab) and population-level host performance (measured as
216 the mean climatic temperature experienced in the field across surveyed species).

217 While we were generally constrained to use whichever metric of parasitism or host
218 performance was reported for a study, we had access to thermal response data for several
219 different metrics in the mosquito–parasite systems. We chose to use infected days as our measure
220 of individual-level parasitism in these eight systems because it is a composite metric using
221 thermal performance data on parasite development rate within the mosquito, vector competence
222 (the mosquito's ability to acquire and transmit the parasite), and mosquito survival. We were also
223 interested in how this choice affected our findings of how closely T_{opt} of individual-level
224 parasitism matched T_{opt} of population-level parasitism and T_{opt} of host performance. Generally,
225 using vector competence as a metric of individual-level parasitism gave similar results to our
226 main metric of infected days, but parasite development rate tended to exhibit higher T_{opt} values
227 than the other two metrics (Fig. S10). The Supporting Information contains further details on
228 both our model and the methods used to analyze the empirical systems.

229 Using these thirteen systems, we tested for a significant correlation by comparing
230 population-level parasitism T_{opt} to individual-level parasitism T_{opt} using the *cor.test* function in
231 the R package *stats* (R Core Team 2020; method = *pearson*). In some of these systems, metrics
232 used to calculate individual-level parasitism T_{opt} are also a subset of the metrics used to calculate
233 population-level parasitism T_{opt}. For example, the thermal response of mosquito mortality rate is

one of several components used to calculate individual-level parasitism in mosquito–parasite systems, and also one of many components used to calculate population-level parasitism in the same systems. As a result, observed thermal optima for population-level parasitism are not independent from observed thermal optima for individual-level parasitism. While this can affect the interpretation of the observed correlation across systems and whether or not it should be considered statistically significant, we argue that the partial dependence of population-level parasitism on what occurs at the individual-level is biologically realistic for many systems. Finally, using the same systems, we compared T_{opt} for individual- and population-level parasitism to T_{opt} of host performance to determine if parasitism generally peaked at temperatures away from host thermal optima as predicted by the thermal mismatch hypothesis (Cohen *et al.* 2017, 2019a, b), and if so, whether these thermal mismatches at the individual level corresponded with thermal mismatches at the population level.

248 TABLE 1. Thermal optima of parasitism and host performance for thirteen host – parasite
249 systems.
250

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

251 * T_{opt} for these values was determined as the temperature at which the measure was maximized
252 along a range of discrete experimental temperatures, rather than the temperature maximizing the
253 measure along a fitted continuous curve, as is the case for each other value in the table. For
254 discrete temperature range used, see Supporting Information.
255 † The first number listed is individual-level host T_{opt}. The number in parentheses is population-
256 level T_{opt}.
257

258

259 **RESULTS**

260 ***Differences in host–parasite biology and the expected relationship between parasitism T_{opt}***
261 ***across levels***

262 As hypothesized, the model showed that T_{opt} for individual- and population-level
263 parasitism may be correlated or uncorrelated, depending on the relationships among the trait
264 thermal responses. When the thermal responses of all parameters in the population-level
265 parasitism model were independent from each other and from the thermal response of individual-
266 level parasitism, we did not observe any relationship between the thermal optima of parasitism
267 across levels (Fig. 1a). Similarly, there was no relationship between thermal optima in the
268 scenario in which the thermal response of parasite-induced mortality was proportional to the
269 thermal response of parasite load (Fig. 1b). This means that in this scenario, even though a
270 parameter that partially determined the thermal response of population-level parasitism was
271 directly dependent on the thermal response of individual-level parasitism, we still did not
272 observe a relationship between the thermal optima. Conversely, when the probability of infection
273 after contact was proportional to parasite load across the thermal range, we observed a strong,
274 positive relationship between the thermal optima for parasitism across levels (Fig. 1c). Though
275 the relationship was significant and positive, it does not fall upon the 1:1 line (95% confidence
276 interval; Fig. 1c). Additionally, while many simulated host–parasite systems are observed below
277 the 1:1 line (i.e., individual-level parasitism T_{opt} was greater than population-level parasitism
278 T_{opt}), there were no systems in which population-level parasitism T_{opt} greatly exceeded
279 individual-level parasitism T_{opt} under these model conditions. Finally, when both probability of
280 infection after contact and parasite-induced mortality were proportional to parasite load across
281 the thermal range, the correlation between individual-level parasitism T_{opt} and population-level

282 parasitism T_{opt} was strongly positive (Fig. 1d), closely resembling the scenario in which only

283 probability of infection after contact was proportional to parasite load (Fig. 1c).

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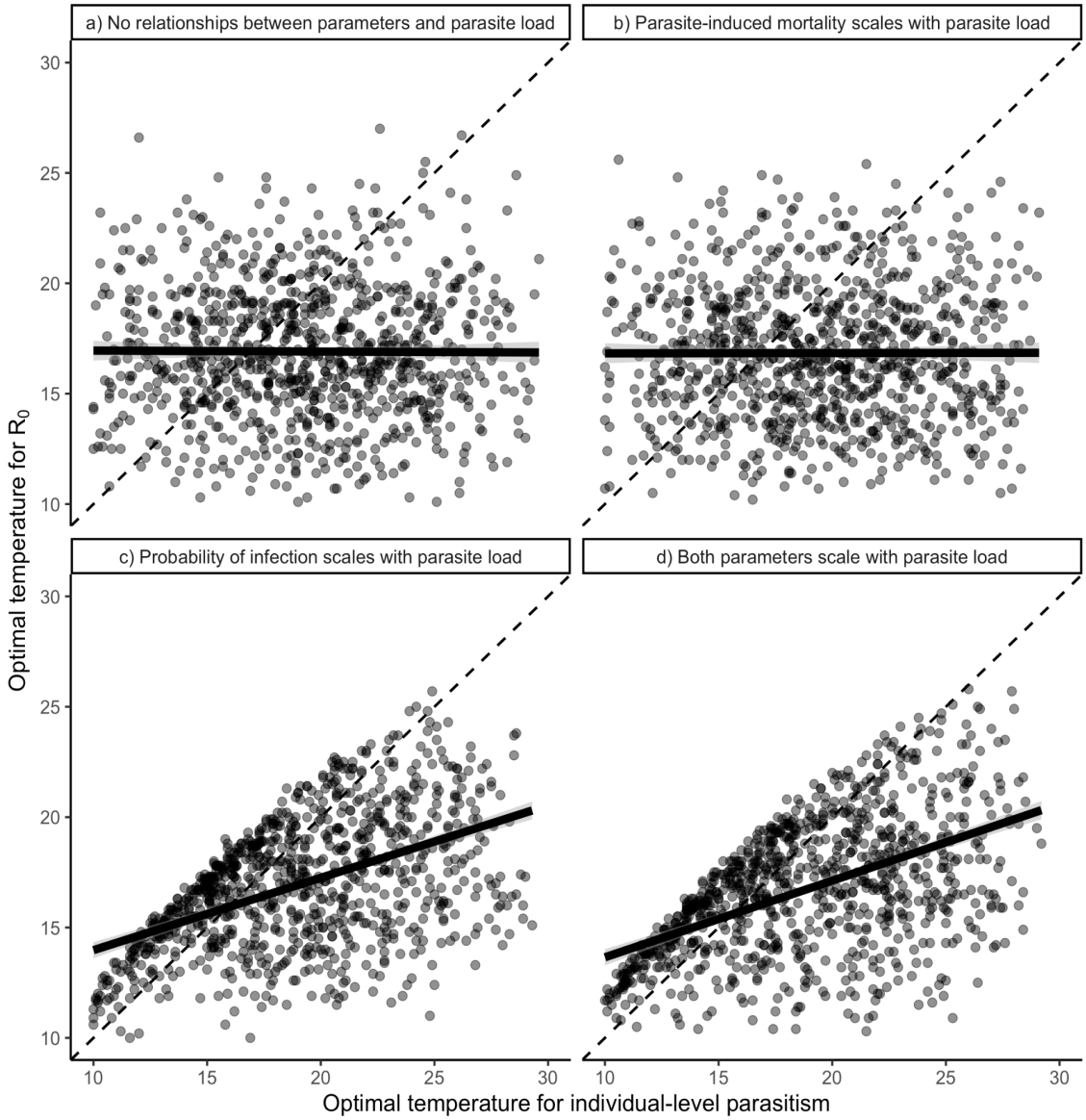

Figure 1. Thermal optima of individual parasite burden may be either uncorrelated (top panels) or positively correlated (bottom panels) with thermal optima of R_0 at the population level. The thermal optima of parasitism in 1000 simulated host–parasite systems at both the population level (R_0) and individual level (parasite load) for four different modeled scenarios: a) no relationship between any parameters in the R_0 model (Eq. 2) and parasite load (Eq. 1); b) parasite-induced mortality in the R_0 model is proportional to parasite load; c) probability of infection after contact in the R_0 model is proportional to parasite load; d) both parasite-induced mortality and probability of infection after contact in the R_0 model are proportional to parasite load. Dashed black lines represent the 1:1 lines. Solid black lines and shaded bands represent the best fit linear regressions and 95% confidence intervals, respectively.

We found qualitatively similar results for several of our additional model scenarios. Specifically, we found similar results when thermal response parameters were drawn from normal distributions (Fig. S2), when parameter thermal breadth was larger (Fig. S3), when peak contact rates occurred at warmer temperatures (Fig. S4), and when the population-level parasitism model (Eq. 1) was altered to allow for chronic infections (Fig. S7) or for environmentally-transmitted parasites (Fig. S8). However, we found that adding a baseline level of individual-level parasitism across the temperature range, such that individual-level parasitism (and probability of infection when it is modeled as proportional to individual-level parasitism) is low but not zero when $T < T_{min}$ and $T > T_{max}$, led to qualitatively different results when probability of infection was proportional to individual-level parasitism. In this case, a number of systems exhibited population-level parasitism T_{opt} much warmer than individual-level parasitism T_{opt} (Fig. S5c-d), a pattern we did not observe in Figs. 1c-d. Allowing for individual-level parasitism to have a baseline level and for peak contact rates to occur at warmer temperatures led to even more systems with population-level parasitism T_{opt} >> individual-level parasitism T_{opt} (Fig. S6c-d).

Relationship between parasitism T_{opt} across levels in empirical systems

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). We found that population-level parasitism T_{opt} was significantly correlated with individual-level parasitism (Fig. 2; Pearson correlation = 0.83, $p = 0.0005$, $n = 13$). 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 levels 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).

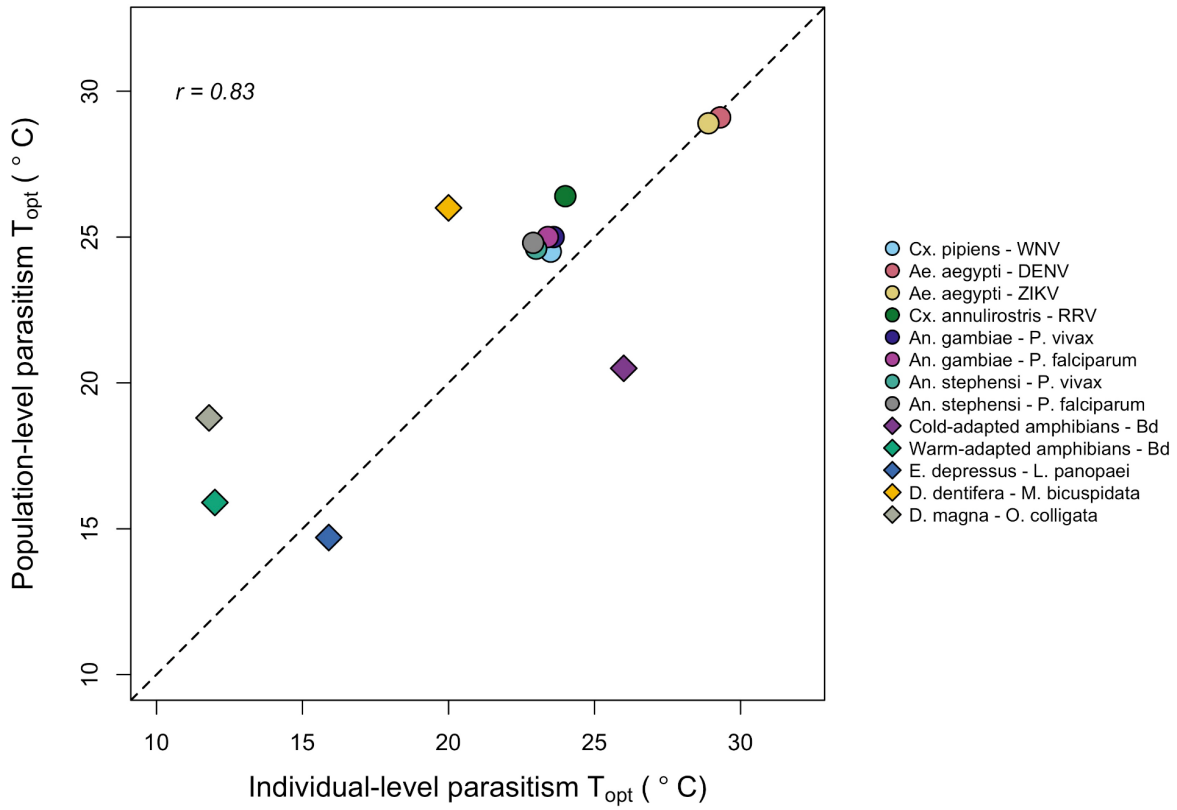

Figure 2. The thermal optima of population-level parasitism are positively correlated with the thermal optima of individual-level parasitism in empirical systems. Points show estimates of the optimal temperature (T_{opt}) for population-level parasitism versus T_{opt} of individual-level parasitism for thirteen host–parasite systems. We found a significant positive correlation between T_{opt} of population-level parasitism and T_{opt} of individual-level parasitism (Pearson correlation = 0.83, p = 0.0005, n = 13), 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.

337 ***Relationship between parasitism T_{opt} and host performance T_{opt} in empirical systems***

338 Next, we compared the thermal optima of parasitism at both levels to T_{opt} for host
339 performance (Fig. 3). If a system was situated at the origin in Fig. 3, it would have individual-
340 level parasitism, population-level parasitism, and host performance maximized at the same
341 temperature (i.e., no thermal mismatches exist). Displacement from the origin represents
342 parasitism peaking at temperatures away from where host performance peaks (i.e., thermal
343 mismatches at one or both levels). We found that over half (7/13) of the systems were situated
344 close to the origin (arbitrarily defined here as within Euclidian distance of 2.5°C, represented by
345 the circle in Fig. 3). However, all seven of these are mosquito–parasite systems, with the only
346 mosquito system exhibiting strong thermal mismatches being *Cx. pipiens*–West Nile virus, in
347 which both infected days and R_0 peak at temperatures nearly 10ºC higher than host performance
348 (Fig. 3). The cold adapted amphibian–*B. dendrobatidis* system was also situated in the far upper-
349 right quadrant (Fig. 3), showing strong thermal mismatches at both levels of parasitism, as
350 shown in Cohen *et al.* (2017). Two systems showed cold-temperature mismatches at both levels:
351 the crab *E. depressus–L. panopaei* and warm-adapted amphibian–*B. dendrobatidis* host–parasite
352 systems. The zooplankton *D. dentifera–M. bicuspidata* system exhibited a thermal mismatch at
353 cold temperatures at the individual level but no mismatch at the population level. Finally, the
354 zooplankton *D. magna–O. colligata* system showed a distinct pattern of differing thermal
355 mismatches across levels (Fig. 3): individual-level parasitism was maximized at a lower
356 temperature than host performance while R_0 peaked at a higher temperature than host
357 performance. None of the thirteen systems showed positive individual-level mismatches but
358 negative population-level mismatches; therefore, the lower-right quadrant of Fig. 3 is empty. The
359 six systems that experienced thermal mismatches generally supported our hypothesis that if

mismatches occured they would be at both levels 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.

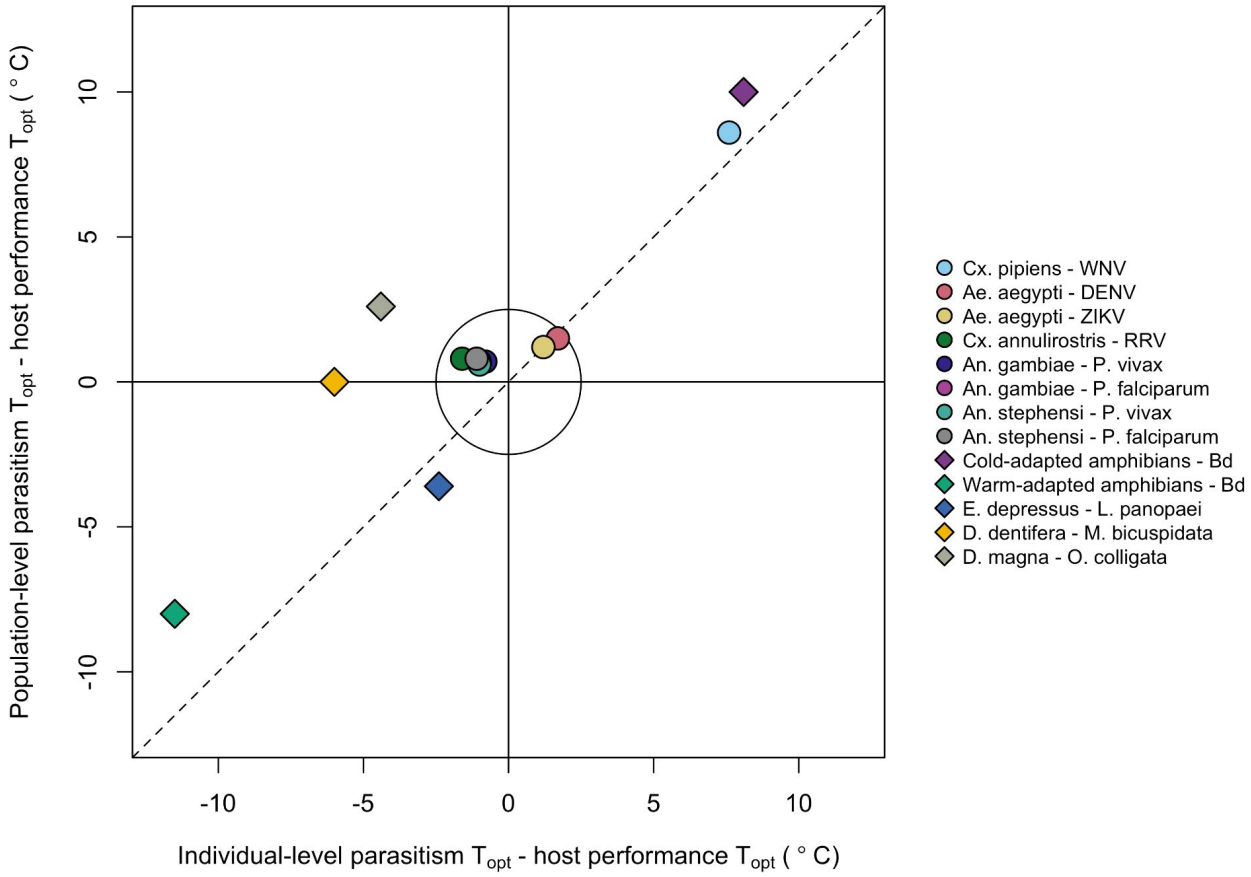

Figure 3. Thermal matches and mismatches tended to be correlated across levels of biological organization. The difference between T_{opt} of population-level parasitism and host performance (y-axis) is plotted against the difference between T_{opt} of individual-level parasitism and host performance (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 systems 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).

DISCUSSION

If we are to best predict how warming temperatures will affect host–parasite systems, we need to understand if effects on parasitism at one level of organization correspond with similar effects at another level. Moreover, understanding how these effects on parasitism compare to thermal effects on uninfected hosts can offer a more complete lens into how climate change will affect hosts. Our model found that small differences in the underlying biology of host–parasite systems can substantially alter the expected relationship between the thermal optima of parasitism in individual hosts and host populations, ranging from strong positive correlations to no correlation of T_{opt} across levels (Fig. 1). 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), suggesting thermal mismatches may be more common in certain types of host–parasite systems. Generally, our results show that information on the temperature-dependence, and specifically the thermal optimum, 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 correlation between T_{opt} of population-level parasitism and T_{opt} of individual-level parasitism, suggesting that the effects of warming on parasitism may often be in the same direction across levels. However, we also found that population-level parasitism tended to peak at slightly warmer temperatures than individual-level parasitism in the majority of systems (Fig. 2), meaning that these systems may experience small temperature ranges in which warming leads to increases in population-level parasitism but decreases in

individual-level parasitism. The greatest differences in T_{opt} were observed in the zooplankton *D. magna–O. colligata* system, the 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; Villena *et al.* 2020). These few systems represent only a small slice of the diversity of host–parasite systems that exist; however, they may represent a relatively greater portion of the diversity in thermal trait biology and temperature-dependent metabolic rates because these rates are often conserved across systems (Brown *et al.* 2004; Dell *et al.* 2011; Molnár *et al.* 2017)**.** More empirical work measuring thermal responses of traits related to parasite transmission in other types of host–parasite systems, such as those with helminth or bacterial parasites—both of which have been shown to be affected by climate (Ben-Haim *et al.* 2003; Ben-Horin *et al.* 2013; Mignatti *et al.* 2016)—can help broaden our perspective on the scaling of parasitism from individuals to populations in light of climate change.

423 The positive relationship observed for T_{opt} across levels in the thirteen empirical systems
424 qualitatively matched the positive relationship observed in the two modeled scenarios in which
425 probability of infection was proportional to parasite load (Fig. 1c-d). However, this should not be
426 taken as evidence that these empirical systems follow the same described dynamics or meet the
427 same assumptions of these modeled scenarios, as the underlying biology differs greatly between
428 many of these systems, and the models that have been used to describe their dynamics differ both
429 between the systems and from the simple model developed here (e.g., Briggs *et al.* 2010;
430 Mordecai *et al.* 2017; Gehman *et al.* 2018; Shocket *et al.* 2018b; Kirk *et al.* 2020). Taken
431 together, this means that the model results (Figs. 1, S2-S8) are useful for our understanding of
432 how different T_{opt} relationships are possible under different plausible scenarios, but should not be
433 used as statistical null hypotheses for our thirteen empirical systems. The contribution of this
434 approach—pairing mechanistic models and thermal responses across biological levels under
435 different assumptions—is that we can observe how different systems or thermal biology can lead
436 to different patterns in T_{opt} across levels. Indeed, while our additional model scenarios generally
437 supported our main finding that individual- and population-level parasitism can be correlated or
438 uncorrelated and showed that this is qualitatively robust across different model structures and
439 assumptions regarding thermal response shape, they also illustrated how changes to thermal
440 biology can alter these patterns. For example, if peak contact rates tend to occur at warmer
441 temperatures than other traits, we may observe more systems that have population-level
442 parasitism T_{opt} > individual-level parasitism T_{opt} (Fig. S4). Also, despite not observing systems
443 in the far upper-left space (where population-level parasitism T_{opt} >> individual-level parasitism
444 T_{opt}) for our model simulations when probability of infection scaled with individual-level

445 parasitism (Fig. 1c-d), additional scenarios showed that this is possible if individual-level
446 parasitism does not decrease completely to zero when $T < T_{min}$ and $T > T_{max}$ (Figs. S5-S6).
447 The thermal mismatch hypothesis (Cohen *et al.* 2017, 2019a, b, 2020) has been proposed
448 as an approach for understanding the effects of climate change on host–parasite systems,
449 predicting that T_{opt} for parasitism should occur at temperatures away from host T_{opt} (i.e., a
450 thermal mismatch) due to larger organisms (hosts) generally having narrower thermal breadth
451 than smaller organisms (parasites; Rohr *et al.* 2018). While empirical evidence shows that
452 thermal mismatches do occur at the population level across many systems (Cohen *et al.* 2020),
453 the novel hypothesis we tested here is that thermal mismatches at the individual level will
454 correspond with similar thermal mismatches at the population level. Of the five non-mosquito
455 systems we explored—including the two amphibian–*Bd* systems investigated previously—
456 parasitism tended to peak at temperatures away from the host's optimum in each, meaning that
457 these systems experience thermal mismatches at one or both levels. If our hypothesis was to be
458 supported, we would expect most systems experiencing thermal mismatches to fall in the upper-
459 right or lower-left quadrants of Fig. 3, rather than the upper-left or lower-right. We found that
460 this was generally the case, with two systems experiencing thermal mismatches at warmer
461 temperatures at both levels (Fig. 3; upper-right) and two systems experiencing thermal
462 mismatches at cooler temperatures at both levels (Fig. 3; lower-left). The major exception was
463 the *D. magna–O. colligata* system, in which host T_{opt} occurs at a temperature intermediate to the
464 relatively cool T_{opt} for individual-level parasitism and relatively warm T_{opt} population-level
465 parasitism (Fig. 3; upper-left). The other zooplankton host system we explored, *D. dentifera–M.*
466 *bicuspidata,* exhibited a thermal mismatch at cooler temperatures at the individual level but no
467 mismatch at the population level.

468 Evidence for thermal mismatches occurring at either level was weak in the mosquito–
469 parasite systems. Seven of eight mosquito–parasite systems were situated close to the origin (Fig.
470 3), suggesting that parasitism and host performance are maximized at close to the same
471 temperatures and that no thermal mismatches are occurring at either level. Notably, when testing
472 for thermal mismatches at the population level, (Cohen *et al.* 2020) also found that thermal
473 mismatch effects were strongest in systems without vectors (or intermediate hosts). The
474 exception in our study, *Cx. pipiens*–West Nile virus, in which parasitism at both levels peaks at
475 temperatures much higher than host performance because *Cx. pipiens* adult lifespan is much
476 longer at low temperatures (Shocket *et al.* 2020), is a system with a predominantly temperate
477 distribution in North America, compared to the more sub-tropical and tropical distributions of
478 malaria, dengue, Zika, Ross River virus, and their respective mosquito vectors. Why we
479 observed this pattern in mosquito systems will require further investigation in different vector-
480 borne systems across biological levels, including those systems with non-mosquito vectors such
481 as ticks or sandflies.

482 We propose that situating host–parasite systems along both individual- and population-
483 level axes as in Fig. 3 can provide a diagnostic framework for looking for thermal mismatches
484 across biological levels. While our results do not conclusively address how general thermal
485 mismatches at both levels may be, our finding that individual- and population-level T_{opt} are
486 strongly related (Fig. 2) provides a general rule of thumb that, if a host–parasite system does
487 experience thermal mismatches, they are likely to occur at both levels and in the same direction
488 (though note the exception of *D. magna–O. colligata*; Fig. 3). This has major implications for
489 our understanding of the effects of climate change on host-parasite systems, because if T_{opt} for
490 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 climates to experience increased parasitism, and hosts from warmer climates to experience decreased parasitism (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 across biological levels, 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 load 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, after our model illustrated that either positive or flat relationships between T_{opt} across levels were plausible, we found a strong positive relationship in the thirteen systems we analyzed, suggesting that the effects of warming on individual-level outcomes may beget similar outcomes at the population level in various host–parasite systems. Many studies have investigated the effects of temperature on parasitism (reviewed, for example, in Marcogliese

514 2008; Lafferty 2009; Rohr *et al.* 2011; Altizer *et al.* 2013; Lafferty & Mordecai 2016; Claar &
515 Wood 2020), though often at only one of either the individual or population levels. Our findings
516 build upon these studies by emphasizing that how temperature affects individual-level parasitism
517 can scale up to affect parasite transmission, prevalence, and the potential for epidemics in host
518 populations, though not always in a 1:1 manner. While this is an important first step toward
519 predicting the effects of climate change on systems by allowing researchers to primarily focus on
520 parasitism at one level when necessary, future empirical work that investigates the thermal
521 dependence of a wider range of host–parasite systems across levels, including in systems with
522 different types of hosts (e.g., plants, non-amphibian vertebrates) and other types of parasites
523 (e.g., helminths, bacterial parasites), will be necessary for investigating the generality of this
524 result. More broadly, tying together more theory and empirical results to provide general
525 predictions for how climate change will affect parasitism from individuals to ecosystems in a
526 diverse range of hosts and parasites remains an urgent priority as accelerating climate change
527 makes potentially catastrophic temperature increases of > 2°C increasingly likely by 2050.
528

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