

RESEARCH ARTICLE

Energy Trade-Offs Under Changing Environments

Hotter, faster, sicker? Warming shifts the cost of infection from individuals to populations

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Abstract

1. The thermal environment can profoundly modify the damage that pathogens cause to hosts. Yet while the interacting impacts of infection and temperature on individual life-history traits are well documented, how these effects scale to demographic consequences across thermal gradients remains poorly understood.
2. Using the *Daphnia magna*–*Pasteuria ramosa* system across a 10–30°C gradient, we tested whether virulence in individual-level life history metrics reliably reflects the consequences for predicted population growth rates.
3. Our results reveal a stark mismatch between scales: the cost of infection on individual life-history traits (fecundity, body size, and lifespan) was maximised at low temperatures, whereas the impact on population growth rate was maximised at high temperatures and at the thermal optima of healthy hosts.
4. These findings demonstrate that standard virulence metrics at the scale of individual life-history traits may underestimate demographic risk in warming environments. To reliably predict population persistence in the face of disease under a changing climate, it is important to account for the temperature-dependent scaling of pathogen virulence and host life-history to population growth rates.

KEYWORDS

climate change, disease, host–pathogen interactions, infection, life-history trade-offs, population growth rate, thermal performance, virulence

1 | INTRODUCTION

Predicting whether species persist or decline under environmental change requires understanding how various and interacting stressors shape the health of both individuals and populations (Altizer et al., 2013; Heard et al., 2013; Kirk et al., 2022; Murray et al., 2010). Infectious disease is a well-appreciated driver of declines at both scales (Burthe et al., 2008; De Castro & Bolker, 2005; Erens et al., 2023; Frick et al., 2010; Grimaudo et al., 2024; Heard et al., 2013; Martin et al., 2018; Smith et al., 2009; Valenzuela-Sánchez et al., 2021). By imposing physiological costs or causing

direct damage (i.e. virulence; Poulin & Combes, 1999), pathogens commonly reduce host survival, growth, and reproduction. However, while the damage infection causes to individuals can be severe, this does not necessarily predict the consequences at the level of populations (such as reductions in rates of population growth or persistence; Dziuba et al., 2025; Rózsa & Garay, 2023; Tompkins et al., 2011; Wells et al., 2017). Translating the impact of pathogen-induced damage into indicators of population performance therefore presents a scaling challenge: metrics assessing individual-level infection costs are not always reliable predictors of outcomes at the population level (Rózsa & Garay, 2023).

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This disconnect between the individual and population-level consequences of virulence arises because not all life-history traits contribute equally to population growth (Birch, 1948; Cole, 1954). Virulence is often quantified using cumulative life-history traits like total reproductive output or host lifespan (Poulin & Combes, 1999). However, population growth rates depend strongly on the timing of maturation and early reproduction, owing to the potential for early exponential population growth (Birch, 1948; Cole, 1954; Dean et al., 2023; Hoogendyk & Estabrook, 1984). Consequently, a pathogen whose damage primarily affects survival or fecundity later in life may seem highly virulent for an individual but have a reduced impact on population growth compared to a pathogen that makes seemingly more moderate reductions in early reproduction, survival or development (Rózsa & Garay, 2023). A focus on cumulative and individual-level metrics of virulence can therefore misrepresent the true threat that disease poses to populations, either overstating or understating the demographic consequences of infection.

Temperature can further exacerbate this disconnect between individual- and population-level metrics of virulence. In ectotherms (which represent 99% of all species; Atkinson & Sibly, 1997), warming (up to an ectotherm's thermal optimum) typically accelerates development and advances reproduction, compressing generation time and shifting reproductive effort earlier in life (Dell et al., 2011; Hector et al., 2021a; Huey Raymond & Berrigan, 2001; Kuparinen et al., 2011). At the same time, pathogens are thermally sensitive, such that the damage they can cause often varies across temperatures (Butterworth et al., 2025; Paull et al., 2015; Paull & Johnson, 2014; Vale et al., 2008). These combined effects mean that temperature can change not only the magnitude of virulence but may also fundamentally alter how virulence translates into population growth. Indeed, work by (Hector et al., 2021a) has shown how a rise in temperature can favour enhanced population growth at the expense of individual life-history traits, while simultaneously reducing the potential for a pathogen to damage the host. Yet despite the importance of predicting disease impacts under climate warming (Altizer et al., 2013; Gehman et al., 2018; Hector et al., 2021b), a formal test of whether individual-level metrics of virulence reliably predict population-level outcomes across broad thermal gradients remains lacking.

Integrating temperature-dependent effects of infection on multiple life-history traits with metrics of population growth begins by using life tables to derive the intrinsic growth rate of a population, denoted as r_m , and defined as the per capita potential growth rate of a population without density-dependent regulation (Birch, 1948; Krebs, 1972). Across a range of ectotherms (Frazier et al., 2006; Huey Raymond & Berrigan, 2001), the response of r_m across a temperature gradient (its thermal performance; Amarasekare & Savage, 2012; Huey & Kingsolver, 1989; Knies et al., 2009) has been shown to be left-skewed with a maximum value occurring at a temperature (T_{opt}) closer to the upper thermal limit in contrast to the underlying reproductive rates which are more symmetrical or survivorship which decreases with temperature. Comparing these thermal curves between healthy and infected hosts provides a useful framework

for assessing how the effects of infection on individuals scale to determine the sensitivity of populations to disturbance and stress under warming conditions (as reflected by changes to r_m ; as per; Amarasekare & Coutinho, 2013; Cortés, 2016; Deutsch et al., 2008; Pawar et al., 2024).

Here, we address this using the *Daphnia magna*-*Pasteuria ramosa* host-pathogen system (Ebert, 2008; Ebert et al., 2016). Across a 10–30°C thermal gradient, we quantify temperature-dependent effects of infection on lifespan, fecundity and body size, capturing multiple dimensions of individual-level virulence. We then integrate these traits to estimate intrinsic population growth rate (r_m) across temperatures. By directly comparing the thermal performance of individual traits and predicted population growth rates, we assess the impact of rising temperatures on multiple components of individual performance, contrast that with predicted population growth rates, and then ask if temperatures that maximize virulence in these traits are the same temperatures that maximize the population-level costs. In doing so, we provide empirical insight into how temperature and disease interact to reshape life-history trade-offs and population resilience.

2 | METHODS

2.1 | Study system

Daphnia magna Straus (Cladocera: Anomopoda) is a freshwater crustacean that can reproduce by facultative parthenogenesis. Typically, a female reaches maturity at approximately 10 days of age (at 20°C) and produces a clutch of 10–30 amictic eggs (diploid eggs that develop into female offspring without fertilization) after every adult moult (every 3–4 days at 20°C) until her death. In the laboratory, females may live for more than 3 months at 20°C (Ebert, 2005). In natural populations, *D. magna* is infected by a diverse assemblage of parasites (Ebert, 2005), including the gram-positive bacterium *Pasteuria ramosa* Metchnikoff (Green, 1974). Following ingestion, *P. ramosa* attaches to the host oesophagus and hindgut before proliferating within the haemolymph, where it produces transmission spores (Ebert et al., 2016). Infection substantially reduces female fecundity and lifespan while increasing host body size (Clerc et al., 2015; Hall et al., 2019). Transmission is exclusively horizontal and occurs only after spores are released from dead hosts (Aulsebrook et al., 2023; Hall & Mideo, 2018). Work on this system does not require ethical approval.

2.2 | Experimental design

This experiment used stocks of *D. magna* of the genotype BE-OHZ-M10 which was originally hatched from a resting egg (ephippia) collected from Oud Heverlee, Belgium (coordinates: 50°50'14", 4°39'48"). The *P. ramosa* genotype C1 was used to infect which was originally isolated from a single female infected with pond sediment

from Moscow, Russia (Luijckx et al., 2011). Following established protocols, adult females were maintained under standard experimental conditions for three generations (Butterworth et al., 2024). Experimental offspring were then assigned unique individual identifiers and transferred to their respective temperature treatments at 1 ± 1 days of age. Individuals were reared across nine constant temperature treatments spanning 10, 12.5, 15, 17.5, 20, 22.5, 25, 27.5, and 30°C. Within each temperature treatment, individuals were randomly assigned to either an unexposed control treatment ($n=10$) or a pathogen-exposed treatment ($n=21$) for a total of 279 animals. After 16 animal losses or experimental errors there were 263 total animals assayed across 227 days of experimental observation.

Temperature treatments were maintained in temperature-controlled incubators, with temperatures monitored continuously and held within $\pm 0.5^\circ\text{C}$ of target values. Individuals were housed in 70 mL jars, filled with 50 mL of artificial media (ADaM; Ebert et al., 1998; Klüttgen et al., 1994). Animals were fed daily throughout the entire experiment with increasing numbers of algal cells (*Scenedesmus* sp.) – 1 million cells (at 1 day old), 2.5 million cells (at 2–5 days old), 3 million cells (at days 5–6 days old), and 5 million cells (from day 7 onwards). Animals were changed into 50 mL of fresh ADaM every 3 days throughout the entire experiment.

2.3 | Infection treatments

Pathogen exposure occurred when individuals were 4 ± 1 days old. All individuals assigned to the pathogen treatment were exposed to *P. ramosa* by administering a standardized dose of 25,000 spores in 20 μL of ADaM. The spore suspension was delivered directly into each individual's jar and were then immediately returned to their respective temperature cabinet. After 4 days of exposure to the spores, animals were moved to fresh jars filled with 50 mL of ADaM and then water was changed as usual after.

Unexposed control individuals received no spores and were otherwise handled identically. Any individuals that were dead or missing on the day of pathogen exposure were replaced with age-matched individuals drawn from large stock jars maintained at the same temperature. Replacement individuals experienced identical thermal conditions prior to exposure and were treated identically thereafter. No replacements occurred after pathogen exposure.

2.4 | Individual- and population-level metrics of performance

Individuals were monitored daily throughout their lifespan. Survival was recorded until death, and fecundity was assessed by counting the number of offspring produced at each clutch for each individual experimental animal. Body size was assessed by measuring body size at 14 ± 1 days of age. Intrinsic growth rate (r_m) was estimated for each temperature and exposure treatment by integrating survival and reproduction across the full life cycle using a standard

lifetable and then calculated as the solution to the Euler-Lotka equation (following Aulsebrook et al., 2022; Hector et al., 2021a; Shocket et al., 2018). Virulence was calculated for each temperature treatment as the mean difference between uninfected and infected hosts for each trait (fecundity, body size, lifespan and r_m).

2.5 | Statistical analyses

All analyses were conducted in R (version 4.5.2; R Development Core Team available at www.r-project.org). We first analysed the interaction between temperature and infection on our measures of individual and population performance using the mixed-effect model package glmmTMB (McGillucuddy et al., 2025). Two complimentary approaches were used: one where temperature was modelled as a factor and another where temperature was modelled as a second order orthogonal polynomial. In both cases, the use of the DHARMA package (github.com/florianhartig/dharma) to evaluate model fit identified the need for a heterogeneous diagonal residual structure with separate variances allowed for uninfected and infected animals. We then analysed the change in virulence (the reduction in trait values due to infection, see above) across temperature using a second order orthogonal polynomial.

Finally, having evaluated the potential for differences in the performance of uninfected and infected animals at different temperatures, we fit formal thermal performance curves for each trait as well as our virulence measures. Using the *rTPC* package (available at github.com/padpadpadpad/rTPC) we analysed mean trait values at each temperature (i.e. $n=8$ or 9) using a simplified Briere II model (Briere et al., 1999). This model was chosen as it can cover (via the scale and shape parameters) a range of thermal performance curves, including left and right skewed non-linear curves, as well as approximately linear curves, facilitating the use of a single model across all traits. From each curve (see Figure S1 for curve fits), we extracted the thermal optimum (T_{opt} , the temperature of peak performance, via the `get_topt()` function) and then used bootstrapping (via case resampling) to generate a mean and 95% confidence interval for each T_{opt} estimate. Although this approach ensures consistency across traits, it should be noted that a model incorporating an explicit T_{opt} parameter would be preferable. However, fitting a single model capable of capturing the diverse curve shapes observed in this study was not feasible.

3 | RESULTS

We found significant responses to temperature in all traits, but the exact responses depended on the infection status of an individual (significant interaction terms in all cases, Table 1). Response shapes (Figure 1 and Table 2) ranged from predominately linear across the temperature range (in the case of lifespan) to non-linear and saturating (size and intrinsic r), or even non-linear unimodal (fecundity), and appeared relatively concordant between control and infected

TABLE 1 Linear models for the analysis of trait variation and infection with temperature modelled as either (a) a second order orthogonal polynomial or (b) as a factor with eight levels. Models were run using a heterogenous residual structure with separate variances for uninfected and infected animals and evaluated as a Type III analysis of covariance (model a) or variance (model b).

Trait	Infection success			Temperature			Interaction			Random effect SD	
	χ^2	DF	p	χ^2	DF	p	χ^2	DF	p	Uninfected	Infected
(a) Temperature as polynomial											
Lifespan (days)	138.881	1	<0.001	287.108	2	<0.001	34.890	2	<0.001	30.797	9.954
Fecundity	926.206	1	<0.001	51.085	2	<0.001	17.295	2	<0.001	4.113	1.794
Size (mm)	0.066	1	0.797	170.489	2	<0.001	37.199	2	<0.001	0.311	0.441
Intrinsic r	241.368	1	<0.001	153.988	2	<0.001	36.523	2	<0.001	0.018	0.035
(b) Temperature as factor											
Lifespan (days)	107.081	1	<0.001	367.354	7	<0.001	72.126	7	<0.001	24.418	8.665
Fecundity	105.451	1	<0.001	63.379	7	<0.001	22.683	7	0.002	3.759	1.657
Size (mm)	0.768	1	0.381	187.520	7	<0.001	43.161	7	<0.001	0.301	0.414
Intrinsic r	1.123	1	0.289	157.440	7	<0.001	52.596	7	<0.001	0.018	0.034

animals. Instead, infection commonly led to a substantial reduction in trait values (Figure 1, and all positive virulence values) coupled with a reduced sensitivity to temperature as evidenced by generally smaller linear and quadratic terms (Table 2). The net result of the interaction between temperature and infection was that virulence was highly sensitive to temperature. For individual traits, however, virulence noticeably dropped as temperatures rose (see Figure 1a–c, Table 2), while for our measure of population growth (the intrinsic rate of increase), virulence instead increased with higher temperatures (Figure 1d, Table 2).

Exploring these patterns further using a simplified Brière II model (see Figure S1) revealed that the thermal optima for the individual characteristics of uninfected and infected animals did not necessarily differ (overlapping 95% CIs) and spanned a wide range of temperatures with lifespan maximized at 15°C or lower and body size maximized above 22°C (Figure 2). Yet in all cases the expression of virulence in these individual traits was maximized at lower temperatures, particularly for lifespan and fecundity with thermal optima of 15°C or lower (Figure 2). In contrast for the intrinsic rate of increase, we observed a clear separation in the thermal optima of infected and uninfected animals. Infection led to a 5°C drop in the thermal optima of the predicted population growth rates (i.e. uninfected versus infected T_{opt}), giving rise to virulence and the costs of infection in terms of the growth of a population being maximized above 25°C (Figure 2).

4 | DISCUSSION

Predicting the consequences of climate warming and disease for the persistence of populations requires understanding how pathogens impact both hosts and populations across a wide range of thermal conditions (Gehman et al., 2018; Hector et al., 2021b, 2023; Langwig et al., 2015; Paull & Johnson, 2014; Smith et al., 2009). Our study reveals that while individual virulence—measured as the reduction

in lifespan, fecundity, and body size—was most pronounced at low temperatures, the cost of infection to population growth rate (r_m) peaked at high temperatures (Figure 1). This mismatch highlights that focusing on virulence in cumulative individual life-history traits can underestimate the threat pathogens pose to population growth rates under warming conditions.

Infection reduced r_m across all temperatures, as is expected when infection impacts host survival and fecundity—both traits that contribute directly to population growth rates (Anderson, 1978; Anderson & May, 1978; Hector et al., 2021a; Padfield et al., 2020). Beyond this, we show that infection also shifted the thermal optimum of r_m to lower temperatures, effectively narrowing the host's thermal niche. In other systems infection has also been shown to reduce host thermal optima (Gehman et al., 2018), however, this response is not universal, with increases in thermal optima reported in bacteria-phage systems (Padfield et al., 2020). Strikingly, the largest reductions in r_m occurred at the warmest temperatures and the thermal optimum of uninfected populations (Figure 2). Because r_m can reflect a population's capacity for persistence and recovery (Amarasekare & Coutinho, 2013; Cortés, 2016; Deutsch et al., 2008; Pawar et al., 2024), these results indicate that disease can most strongly constrain population growth precisely where performance would otherwise be maximized, revealing a masked population-level vulnerability to infection at thermal optima.

These findings are relevant to two general ideas in thermal ecology—that ‘warmer is better’, whereby species with higher optimal temperatures have higher maximal performances (Frazier et al., 2006) and that ‘warmer is sicker’ (Hall et al., 2006), whereby warming is expected to exacerbate the impacts of disease. Contrary to the latter, empirical work across ectotherm host–pathogen systems shows that warming can reduce disease severity and transmission (Hall et al., 2006). For example, in *Daphnia*, higher temperatures have been shown to reduce pathogen spore loads and infection success (Dziuba et al., 2023; Shocket et al., 2019); in crabs, warming may even drive some parasites to extinction (Gehman et al., 2018); and

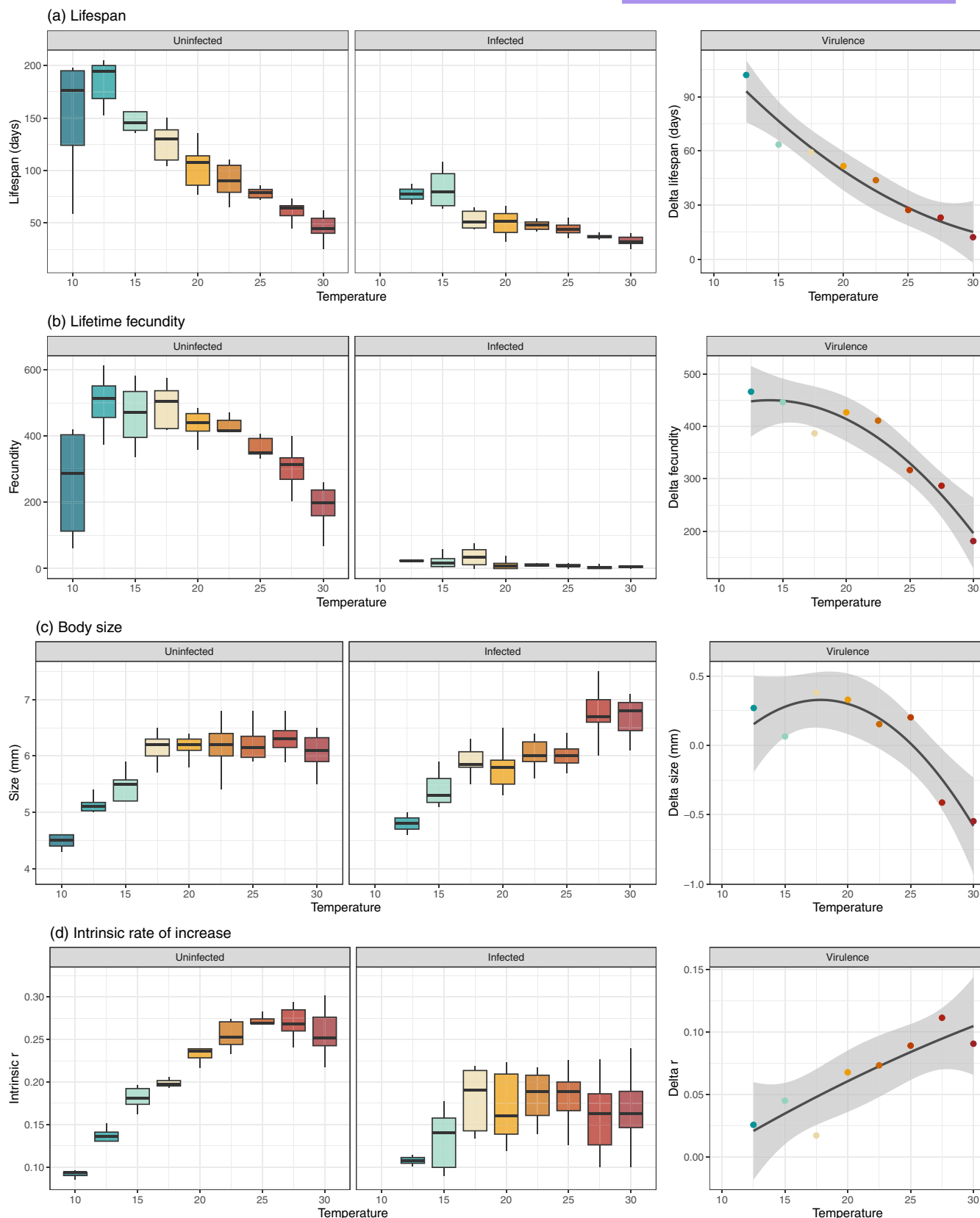


FIGURE 1 Trait values of uninfected hosts, infected hosts, and virulence (reduction in trait value for infected hosts relative to uninfected hosts) for (a) Lifespan, (b) Lifetime fecundity, (c) Body size at 14 days of age and (d) intrinsic rate of increase as calculated by the solution to the Euler-Lotka equation (following Aulsebrook et al., 2022; Hector et al., 2021a; Shocket et al., 2018).

TABLE 2 Linear and quadratic effects of temperature on the characteristics of (a) uninfected animals and (b) infected animals, as well as (c) the expression of virulence and the difference between uninfected and infected means at each temperature. In all cases, temperature was using orthogonal polynomials.

Trait	Overall fit			Linear term			Quadratic term		
	χ^2	DF	<i>p</i>	Estimate	SE	<i>p</i>	Estimate	SE	<i>p</i>
(a) Uninfected characteristics									
Lifespan (days)	157.693	2	<0.001	-386.715	30.796	<0.001	-3.400	30.796	0.912
Fecundity	30.650	2	<0.001	-13.753	4.113	<0.001	-18.145	4.113	<0.001
Size (mm)	281.833	2	<0.001	4.436	0.312	<0.001	-2.807	0.313	<0.001
Intrinsic <i>r</i>	971.327	2	<0.001	0.520	0.018	<0.001	-0.199	0.018	<0.001
(b) Infected characteristics									
Lifespan (days)	215.352	2	<0.001	-140.877	9.954	<0.001	38.605	9.954	<0.001
Fecundity	36.389	2	<0.001	-10.798	1.794	<0.001	0.717	1.794	0.689
Size (mm)	115.404	2	<0.001	4.735	0.441	<0.001	0.079	0.441	0.858
Intrinsic <i>r</i>	16.969	2	<0.001	0.059	0.038	0.126	-0.136	0.037	<0.001
(c) Virulence characteristics									
Lifespan (days)	135.484	2	<0.001	-72.095	6.273	<0.001	11.543	6.273	0.066
Fecundity	99.293	2	<0.001	-232.491	24.663	<0.001	-79.650	24.663	0.001
Size (mm)	43.818	2	<0.001	-0.680	0.127	<0.001	-0.498	0.127	<0.001
Intrinsic <i>r</i>	29.604	2	<0.001	0.077	0.014	<0.001	-0.004	0.014	0.774

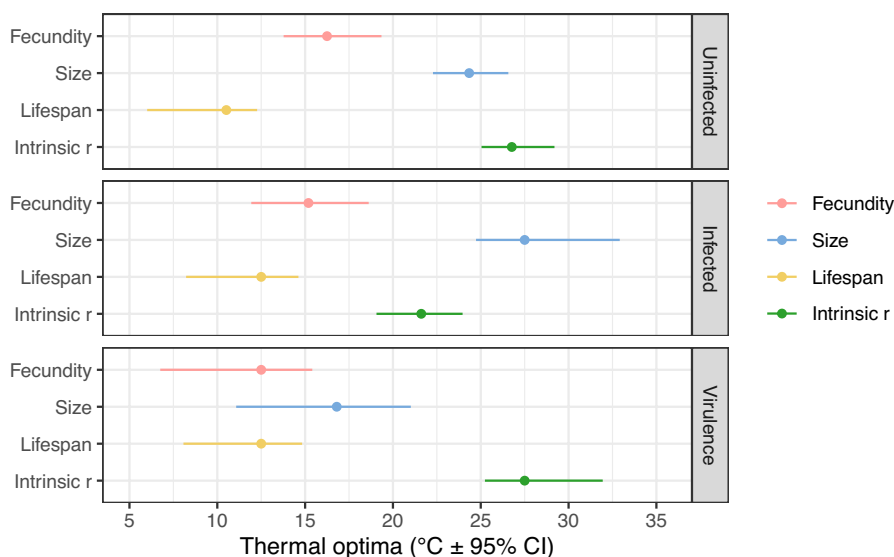


FIGURE 2 Thermal optima as calculated at the temperature with the highest trait value from the Brière II models for fecundity, body size, lifespan and intrinsic rate of increase for uninfected hosts, infected hosts, and virulence (calculated from curves based on the difference between trait values between uninfected and infected hosts).

in mosquitoes, West Nile virus transmission declines at high temperatures (Shocket et al., 2020). In the present study, by focusing on pathogen-mediated damage to hosts and its consequences for population growth, we show that warming can nevertheless amplify the impacts of disease. Although infected individuals experienced the lowest relative virulence at warmer temperatures, this coincided with the largest reductions in intrinsic population growth rate (r_m) occurring at the thermal optimum of uninfected hosts. This indicates that pathogen-mediated risk to population growth can be greatest under warm conditions, and specifically near host thermal optima—reflecting that warmer is both worse and sicker for infected hosts. This is particularly interesting to consider in light of the thermal mismatch hypothesis (Cohen et al., 2017; Nowakowski et al., 2016)

which posits that parasite performance in or on a host should be maximized away from the host's thermal optima. Our data suggest that pathogen induced *virulence* at the population-level is maximized precisely at host thermal optima (though importantly, virulence does not necessarily reflect pathogen performance; Acevedo et al., 2019). One possible follow up question would be whether population-level virulence is often maximized nearest the thermal optima of hosts in other genotype combinations or host–pathogen systems.

The observed mismatch in virulence between individual and population-level scales likely reflects the disproportionate importance of early-life traits to r_m . In ectotherms such as *Daphnia magna*, warming (up to the hosts thermal optima) typically accelerates development, advances the onset of reproduction, and shortens lifespan, collectively

compressing the life cycle and typically inflating r_m by reducing generation time (Dell et al., 2011; Hector et al., 2021a; Huey Raymond & Berrigan, 2001; Kuparinen et al., 2011). Therefore, under infection at high temperatures, even though the reduction in total fecundity appeared modest relative to other temperatures, it did not accurately reflect the outsized consequences for r_m . This was likely underpinned by a more consequential decline in the average clutch size of infected hosts at warmer temperatures (Figure S2). Because reproduction early in life contributes disproportionately to population growth, early losses in average fecundity may have driven the strongest reductions in population-level growth rates at high temperatures—effects that were not captured by cumulative fecundity. This demonstrates that the timing of life-history impacts of virulence, as well as its magnitude, is critical for understanding population responses across varied environments—reinforcing recent calls to more closely integrate host life-history in studies of disease impacts across diverse host–pathogen systems (Valenzuela-Sánchez et al., 2021).

Importantly, we only focus on a single ectothermic host and pathogen genotype under constant temperatures and a pathogen that has markedly strong effects on host fecundity (Clerc et al., 2015; Hall et al., 2019). Nevertheless, substantial reductions in host fecundity are a common pattern of host–pathogen interactions (Anderson, 1978; Armstrong & Bass, 1989; Futerman et al., 2006; Hurd, 2001), and thermal sensitivity in the timing of reproduction is ubiquitous among ectotherms (Dell et al., 2011; Hector et al., 2021a; Huey Raymond & Berrigan, 2001; Kuparinen et al., 2011)—so we may expect a similar mismatch between individual- and population-level virulence in many other ectothermic species. Furthermore, our predictions of population growth rate are based only on individual-level measurements, and we did not empirically measure population-scale outcomes. It is very likely that density-dependent factors such as crowding or resource limitation could cause realized rates of population growth to differ from our predictions. It will thus be important that future work assesses population-level outcomes and also extends these questions to more diverse host–pathogen systems, fluctuating thermal environments (Ferguson & Sinclair, 2019; Krichel et al., 2023), and additional stressors (such as predation; Hall et al., 2006); or pollution; (Aulsebrook et al., 2022; 2023). Such work will be essential for understanding the generality of our findings and for predicting the population persistence for the vast majority of species under global change.

In conclusion, our results show that individual life-history traits may not always reliably predict risks to population growth rates across environmental gradients. As warming reshapes both host life histories and host–pathogen interactions, linking pathogen-mediated damage into life-history theory and demography is increasingly important (Valenzuela-Sánchez et al., 2021). Quantifying virulence in terms of more sophisticated demographic metrics derived from structured population models and models that can equate for density dependence, pathogen transmission, and demographic feedbacks between hosts and pathogens (Anderson, 1978; Anderson & May, 1978) will be particularly important for predicting how disease impacts scale across environments. Such approaches will improve our ability to anticipate if, when, and where warming will amplify disease-driven population

declines, particularly in ectotherm populations that may otherwise appear well adapted to high temperatures.

AUTHOR CONTRIBUTIONS

Nathan J. Butterworth: Conceptualization (lead), data curation, formal analysis, investigation, methodology, validation, writing—original draft preparation, writing—review & editing, project administration. Kerri J. Moore: Investigation, methodology, writing—review & editing. Matthew D. Hall: Conceptualization (supporting), formal analysis, validation, visualization, writing—review & editing, funding acquisition, resources.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

Data and code are publicly available from the figshare repository <https://doi.org/10.6084/m9.figshare.32118460> (Butterworth et al., 2026).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Figure S1. Briere type II curves of all traits (fecundity, body size at 14 days of age, lifespan and intrinsic population growth rates r_m) for uninfected and infected hosts. Virulence curves are calculated from the difference between trait values of uninfected and infected hosts.

Figure S2. Thermal response of average offspring number per clutch (total offspring number divided by total number of clutches per individual) for infected and uninfected hosts. Virulence was calculated as the difference in trait values between uninfected and infected hosts.

Figure S3. Intrinsic rate of increase (r) of unexposed and pathogen-exposed *Daphnia magna* across a thermal gradient. Each panel shows r values (points) calculated for individual hosts and their distribution (boxes). r was calculated for each individual female using the Euler-Lotka equation, incorporating individual reproductive schedules and survival. Colours represent temperature treatments (°C).

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