A Framework for Elucidating the Temperature Dependence of Fitness

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abstract: Climate warming is predicted to cause large-scale extinctions, particularly of ectothermic species. A striking difference between tropical and temperate ectotherms is that tropical species experience a mean habitat temperature that is closer to the temperature at which fitness is maximized (T_{opt}) and an upper temperature limit for survival (T_{max}) that is closer to T_{opt} than do temperate species. Thus, even a small increase in environmental temperature could put tropical ectotherms at high risk of extinction, whereas temperate ectotherms have a wider temperature cushion. Although this pattern is widely observed, the mechanisms that produce it are not well understood. Here we develop a mathematical framework to partition the temperature response of fitness into its components (fecundity, mortality, and development) and test model predictions with data for insects. We find that fitness declines at high temperatures because the temperature responses of fecundity and mortality act in opposite ways: fecundity decreases with temperature when temperatures exceed the optimal range, whereas mortality continues to increase. The proximity of T_{opt} to T_{max} depends on how the temperature response of development mediates the interaction between fecundity and mortality. When development is highly temperature sensitive, mortality exceeds reproduction only after fecundity has started to decline with temperature, which causes fitness to decline rapidly to zero when temperatures exceed T_{opt}. The model correctly predicts empirically observed fitness-temperature relationships in insects from different latitudes. It also suggests explanations for the widely reported phenological shifts in many ectotherms and the latitudinal differences in fitness responses.

Keywords: development, fecundity, fitness, life-history traits, mortality, temperature variation.

Introduction

There is increasing evidence that climate warming can cause large-scale species extinctions (Walther et al. 2002; Root et al. 2003; Parmesan 2006; Sinervo et al. 2010). Ectotherms are particularly susceptible to extinction because they are directly affected by perturbations to the typical thermal environment. Since ectotherms are integral components of virtually every community, playing such key roles as decomposers, pollinators, predators, and parasites, their responses to climate warming are likely to have significant effects on community structure and functioning.

In ectotherms, fitness is strongly temperature dependent: increasing temperature causes a rise in fitness up to a maximum, followed by a rapid decline in fitness as temperature increases further. This dependence provides a quantifiable metric for assessing the effects of climate warming on population viability. Data show that tropical ectotherms experience mean environmental temperatures that are much closer to their physiological optima than temperate ectotherms, which puts them at greater risk of extinction due to climate warming (Deutsch et al. 2008; Tewksbury et al. 2008; Huey et al. 2009). Understanding the mechanisms that generate these differences is crucial in making informed predictions about how climate warming affects species extinctions.

Previous studies have quantified the temperature response of fitness by fitting phenomenological functions (e.g., Gaussian-Gompertz: Frazier et al. 2006; Gaussian-quadratic: Deutsch et al. 2008) to data on the intrinsic growth rate measured at several temperatures. These analyses show that the temperature response of fitness is unimodal and asymmetric (left skewed). Many performance traits (e.g., assimilation, development, metabolic rate) also exhibit left-skewed temperature responses, and these are typically explained in terms of the thermodynamics of enzyme reaction rates (Van der Have and de Jong 1996; Van der Have 2002; Kingsolver 2009). These models are based on the kinetics and denaturation of a single major enzyme (Johnson and Lewin 1946; Sharpe and DeMichele 1977; Schoolfield et al. 1981; Ratkowsky et al. 2005). They predict that the catalytic reaction, the rate of which increases exponentially with increasing temperature, com-

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bined with enzyme denaturation at low and high temperatures, which is unimodal (and typically Gaussian; Ratkowsky et al. 2005), give rise to a left-skewed temperature response for reaction rates. However, it is unclear whether this theory can explain the unimodal temperature response of a composite trait such as fitness, which arises from interactions between the temperature responses of life-history traits that constitute fitness components (e.g., fecundity, development, survivorship). There are currently no extensions of reaction rate theory that integrate the kinetics of several major enzymes that each drive separate fitness components. Moreover, empirically quantified temperature responses of fecundity and survivorship often deviate from the left-skewed response predicted by reaction rate theory (e.g., Dreyer and Baumgartner 1996; Morgan et al. 2001; Van der Have 2002; Angilletta 2009; Kingsolver 2009; Dannon et al. 2010; Hou and Weng 2010; Jandricic et al. 2010), suggesting that factors other than enzyme kinetics may be driving the temperature responses of fitness components themselves. For instance, reproductive traits are likely to be affected by temperature effects on hormonal regulation (Nijhout 1994), and the survivorship of free-living stages are likely to be affected by temperature effects on resource acquisition (Van der Have 2002). This mismatch between theory and data highlights the need for an alternative theoretical framework for elucidating the temperature dependence of fitness.

There is increasing recognition that the temperature dependence of fitness needs to be understood in terms of the temperature responses of fitness components. On the basis of a data analysis spanning seven ectothermic phyla, Huey and Berrigan (2001) showed that the temperature response of the intrinsic growth rate is more left skewed than that of the basic reproductive rate, which they attributed to temperature effects on development. Kingsolver et al. (2011) point out that predictions of extinction risk due to climate warming need to come from a consideration of how fecundity and survivorship contribute to overall fitness in variable environments. There is currently no theory that investigates how the temperature responses of fitness components interact to determine the temperature response of fitness.

Here we take a first step toward investigating this important question. We develop a mathematical framework based on age-structured population dynamics to investigate how the temperature responses of fitness components drive the temperature dependence of fitness. We find that the temperature responses of fitness components interact in complex and often nonintuitive ways. However, because the qualitative nature of these responses is conserved across taxa, there are only a limited number of ways in which their interactions could influence the overall temperature dependence of fitness. This allows us to generate testable predictions about the effect of climate warming on fitness, and hence on extinction risk, that apply across taxa, habitats, and latitude.

Background

Previous studies of temperature dependence (e.g., Savage et al. 2004; Frazier et al. 2006; Deutsch et al. 2008) have used the intrinsic growth rate or the Malthusian parameter (r_m) as the measure of fitness. This is the potential per capita growth rate of a population in the absence of density-dependent factors such as resource limitation and natural enemies (Krebs 1994), and it was previously used to elucidate latitudinal patterns in the temperature dependence of fitness (Deutsch et al. 2008). The ideas we develop here do not depend on the exact definition of fitness and could in principle be extended to the realized per capita growth rate that includes the effects of resources and natural enemies on birth, death, and development (Lotka 1907; Lotka and Sharpe 1911; Charlesworth 1980; Roff 1992). Using r_m as the fitness measure provides a useful bound, because populations cannot be viable unless $r_m > 0$. Moreover, this choice makes it easier to relate the theory to data because temperature dependence of life-history traits is typically measured in the absence of resource limitation or natural enemy pressure (see app. A, available online).

Numerous studies have measured the temperature response of r_m in ectotherms (e.g., references in Huey and Berrigan 2001; Savage et al. 2004; Frazier et al. 2006; Deutsch et al. 2008). They show that the temperature response of r_m is left skewed, with a maximum at a temperature (T_{opt}) that is closer to the upper temperature limit (T_{max}) above which $r_m < 0$ than to the lower temperature limit (T_{min}) below which $r_m < 0$. Because extinction occurs when $r_m < 0$, any function $r_m(T)$ that describes the temperature dependence of fitness must have the property that $r_m = 0$ at the lower and upper temperature limits (T_{min} and T_{max}, respectively) at which mortality balances reproduction. Following Deutsch et al. (2008), we can quantify the skewness of $r_m(T)$ using the ratio $r_{skew} = (T_{max} - T_{opt})/(T_{opt} - T_{min})$. This ratio is equal to 1 when r_m is symmetric around T_{opt}, less than 1 when T_{opt} is closer to T_{max} (left skewed), and greater than 1 when T_{opt} is closer to T_{min} (right skewed).

In practice, the three parameters that describe $r_m(T)$—T_{min}, T_{max}, and T_{opt}—can be estimated by measuring r_m at different temperatures and fitting a phenomenological function to the data (e.g., Huey and Berrigan 2001; Frazier et al. 2006). What we do not know is why a given species has particular values of these parameters and hence a particular shape/form of $r_m(T)$. To explain this, we must un-

derstand the mechanisms that drive the temperature response of fitness.

Mathematical Framework

Age-structured population dynamics provide a way to investigate the temperature dependence of r_m as a function of the temperature responses of underlying life-history traits. In a population that is growing at a rate r_m, the expected reproductive success of a newborn individual is given by the Euler-Lotka equation:

$$\int_{\alpha}^{\infty} \exp(-r_m x) l_x b_x dx = 1, \tag{1}$$

where α is the age at first reproduction (the time it takes for a zygote to develop into a reproductive adult), l_x is the age-specific survivorship (the proportion of individuals that survive from birth to age x), and b_x is the age-specific fecundity (the number of offspring produced by an individual of age x).

Equation (1) is the continuous form of the Euler-Lotka equation (Lotka 1907; Lotka and Sharpe 1911), which applies to multivoltine organisms that reproduce throughout the year. Tropical and Mediterranean species typically exhibit this type of life history, while temperate species are bivoltine or univoltine and reproduce only seasonally. However, data for all species come from experiments conducted under constant temperature and humidity and without any seasonal variation in temperature that limits reproduction to a few months of the year. Thus, our framework applies to data used to quantify $r_m(T)$ in species from all latitudes (e.g., references in Huey and Berrigan 2001; Savage et al. 2004; Frazier et al. 2006; Deutsch et al. 2008).

We do not consider the effect of body size on $r_m(T)$ because body size is fixed within a species and should have negligible effects on temperature dependence within species. Across species, body size may influence the magnitude of r_m but not the unimodality or asymmetry of $r_m(T)$ because $r_m(T)$ is determined by life-history traits that scale with body size (Savage et al. 2004).

We consider the common situation where fecundity declines with age (Charlesworth 1980; Roff 1992; Stearns 1992; Charnov 1993). Here, $b_x = 0$ for $x < \alpha$ and $b_x = b_{\alpha_{peak}} f(x - \alpha_{peak})$ for $x \geq \alpha$, where α_{peak} is the age at which fecundity is maximized and the function f describes the relationship between fecundity and age. This relationship is typically unimodal and by definition has a peak at $f(0)$ (Reznick 1985; Southwood 1988; Dreyer and Baumgartner 1996; Medeiros et al. 2000, 2003; Morgan et al. 2001; Dannon et al. 2010). Once the age at first reproduction is reached, survivorship can be approximated by an exponential decay; that is, $l_x = l_\alpha \exp[-d(x - \alpha)]$, where d is the instantaneous adult mortality rate (Charnov 1993; Savage et al. 2004). We assume that mortality does not increase with age, which is reasonable for species in which fecundity does not appreciably reduce longevity.

We can then express equation (1) as

$$b_{\alpha_{peak}} l_\alpha \exp(d\alpha) \int_{\alpha}^{\infty} f(x - \alpha_{peak}) \times \exp[-(d + r_m)x] dx = 1, \tag{2}$$

which, with a change of variables of $y = x - \alpha_{peak}$, becomes

$$b_{\alpha_{peak}} l_\alpha \exp[-d(\alpha_{peak} - \alpha)] \times \exp(-r_m \alpha_{peak}) \int_{\alpha - \alpha_{peak}}^{\infty} f(y) \times \exp[-(d + r_m)y] dy = 1. \tag{3}$$

Because $f(y)$ is unimodal, $f(0) = 1$ and $f(y) < 1$ everywhere else. In ectotherms, fecundity typically reaches a maximum relatively soon after sexual maturity and declines thereafter (e.g., Reznick 1985; Southwood 1988). For instance, in many insects, the age at which fecundity is maximized ($\alpha_{peak} - \alpha$) is less than 25% of the adult life span and can be as low as 5% (see app. B, table B1, available online). Consequently, f is sharply peaked such that adult mortality (d) determines how quickly the integrand declines and hence the age range contributing to the integral. In this case, the integral in equation (3) is dominated by $y = 0$ (i.e., $x = \alpha_{peak}$). We approximate this integral by evaluating f at its maximal point, $y = 0$, and performing the integration over the remaining exponential term (see app. C, available online). This gives us the following solution:

$$b_{\alpha_{peak}} l_\alpha \exp(-r_m \alpha) - (d + r_m) \approx 0. \tag{4}$$

The quantity l_α, the proportion of individuals that survive to the age at first reproduction, can be expressed in terms of the average mortality during the juvenile stage, $\bar{d}$, as $l_\alpha = \exp(-\int_0^\alpha d(x)dx) = \exp(-\bar{d}\alpha)$ (Charnov 1993). Then,

$$b_{\alpha_{peak}} \exp[-(\bar{d} + r_m)\alpha] - (d + r_m) \approx 0. \tag{5}$$

To investigate the temperature dependence of r_m, we must consider the temperature dependence of fecundity ($b_{\alpha_{peak}}$), mortality ($\bar{d}$ and d), and development (α). With

explicit temperature dependence of these traits, equation (5) becomes

$$b_{\alpha_{\text{peak}}}(T)\exp\{-[\bar{d}(T)+r_{\text{m}}(T)]\alpha(T)\}-[d(T)+r_{\text{m}}(T)]\approx 0, \tag{6}$$

which can be solved to give an analytical expression for the temperature response of fitness in terms of the temperature dependence of fitness components:

$$\begin{aligned} r_{\text{m}}(T) &= -d(T)+\frac{1}{\alpha(T)} \\ &\times W\left[b_{\alpha_{\text{peak}}}(T)\alpha(T)\exp\left[(d(T)-\bar{d}(T))\alpha(T)\right]\right], \end{aligned} \tag{7}$$

where W is the principal (positive) branch of the Lambert W function or the product logarithm (Corless et al. 1996).

Equation (7) provides a general expression for $r_{\text{m}}(T)$ that can accommodate any empirically determined or mechanistically derived temperature response function for fecundity, development, and mortality. In what follows, we discuss some commonly observed response functions and show that their incorporation into equation (7) yields an explicit analytical expression for $r_{\text{m}}(T)$ that consists entirely of measurable parameters.

Data from a large number of studies across all ectothermic taxa (e.g., references in Gillooly et al. 2001*b*) show that per capita mortality rates of juveniles and adults increase monotonically with increasing temperature. This relationship is well described by the Boltzmann-Arrhenius function (Gillooly et al. 2001*b*; Savage et al. 2004):

$$z(T) = z_{T_{\text{R}}}\exp\{A_z[(1/T_{\text{R}})-(1/T)]\}, \tag{8}$$

where z is the juvenile ($\bar{d}$) or adult (d) mortality rate at temperature T (in °K), $z_{T_{\text{R}}}$ is the mortality rate at a reference temperature (typically between 20° and 30°C, with 24°–35°C being the most common; Sharpe and DeMichele 1977; Schoolfield et al. 1981), and A_z is the Arrhenius constant ($A_z = E_{\text{A}}/k$, where E_{A} is the activation energy [cal] and k is the Boltzmann's constant [cal molecule^{-1} degree^{-1}]), which quantifies how fast mortality increases with increasing temperature. Some studies have found that mortality rates can increase at very low temperatures (Morgan et al. 2001), but because this typically occurs at temperatures too low for reproduction, such increases have no effect on r_{m}.

Developmental rates of most ectotherms also increase monotonically with increasing temperature (Gillooly et al. 2001*b*; Savage et al. 2004), although in some species this occurs at a decelerating rate (e.g., Dreyer and Baumgartner 1996; Morgan et al. 2001) and in yet others, developmental rate declines at high temperatures (Van der Have 2002; Angilletta 2009). This pattern has been attributed to the reaction rate and high temperature inactivation of a single major enzyme that drives the developmental process (Johnson and Lewin 1946; Sharpe and DeMichele 1977; Schoolfield et al. 1981; Van der Have and de Jong 1996; Van der Have 2002; Ratkowsky et al. 2005); that is, the reaction rate increases monotonically with temperature while enzyme inactivation exhibits a unimodal (typically Gaussian; Ratkowsky et al. 2005) response to temperature. The net result is a left-skewed temperature response given by

$$\frac{1}{\alpha(T)} = \frac{[(TT_{\text{R}})/\alpha_{T_{\text{R}}}]\exp\{E_{1/\alpha}[(1/T_{\text{R}})-(1/T)]\}}{1+\exp\{E_{\text{H}}[(1/T_{\text{H/2}})-(1/T)]\}}, \tag{9}$$

where $1/\alpha(T)$ is the developmental rate at temperature T in degrees Kelvin, $1/\alpha_{T_{\text{R}}}$ is the developmental rate at the reference temperature T_{R}, $E_{1/\alpha}$ is the enthalpy of activation of the reaction (cal mol^{-1}) catalyzed by the enzyme divided by the universal gas constant (1.987 cal mol^{-1} degree^{-1}), E_{H} is the enthalpy change associated with the high-temperature inactivation of the enzyme (cal mol^{-1}) divided by the universal gas constant, and $T_{\text{H/2}}$ is the temperature at which the enzyme is 50% active. If the developmental rate does not decrease within the temperature range that allows for reproduction, the temperature response of development can be characterized by the Boltzmann-Arrhenius function (eq. [8], which is equivalent to the numerator of equation [9]) with the appropriate parameters ($\alpha_{T_{\text{R}}}$ and A_α). On the basis of our survey of the literature (P. Amarasekare, unpublished data), this is a reasonable approach for a large number of species in which a decline in the developmental rate at high temperatures is not observed within the biologically relevant temperature range (Trudgill et al. 2005; Bowler and Terblanche 2008; Dixon et al. 2009). It also has the advantage that it allows us to obtain an explicit analytical expression for $r_{\text{m}}(T)$, whereas the use of equation (9) requires that $r_{\text{m}}(T)$ be solved via numerical root finding.

The temperature dependence of maximum fecundity ($b_{\alpha_{\text{peak}}}(T)$) is rarely quantified empirically. We use the temperature dependence of average per capita fecundity ($\bar{b}(T)$) as a surrogate for $b_{\alpha_{\text{peak}}}(T)$. This is reasonable because fecundity is typically a sharply peaked function of age and $\bar{b}(T)$ is dominated by $b_{\alpha_{\text{peak}}}(T)$. Numerous studies of invertebrates, fish, and lizards show that per capita fecundity (averaged over the reproductive life span) exhibits a symmetric unimodal relationship with temperature (e.g., Dreyer and Baumgartner 1996; Carriere and Boivin 1997; Morgan et al. 2001; Dannon et al. 2010; Hou and Weng 2010; Jandricic et al. 2010; P. Amarasekare, unpublished data). This relationship is well-described by a Gaussian function:

$$\bar{b}(T) = \bar{b}_{T_R} \exp\{-[(T - T_{\text{opt}_{\bar{b}}})^2/2s^2]\}, \quad (10)$$

where $\bar{b}_{T_R}$ is the average per capita fecundity at the reference temperature, $T_{\text{opt}_{\bar{b}}}$ is the temperature at which average fecundity is maximal, and s gives the variability about the optimum. If the $\bar{b}(T)$ relationship of any species deviates from a Gaussian function (e.g., it is left skewed), one can use the appropriate function describing $\bar{b}(T)$ (e.g., a Gaussian-quadratic) instead.

Incorporating a Gaussian function for fecundity and exponential (Boltzmann-Arrhenius) functions for development and mortality into equation (7) and defining $TD = [(1/T_R) - (1/T)]$ gives

$$r_m(T) = -d_{T_R} \exp(A_d TD) + \frac{1}{\alpha_{T_R} \exp(A_\alpha TD)} \times W\Big(\bar{b}_{T_R} \alpha_{T_R} \exp\{A_\alpha TD - [(T - T_{\text{opt}_{\bar{b}}})^2/2s^2] + \alpha_{T_R} \exp(A_\alpha TD)[d_{T_R} \exp(A_d TD) - \bar{d}_{T_R} \exp(A_{\bar{d}} TD)]\}\Big). \quad (11)$$

The first term of equation (11) is the negative of the temperature response function for adult mortality, which is negative at all temperatures; the greater its magnitude, the greater the negative effect it has on fitness. The second term, which is positive for all temperatures, combines exponential, Gaussian, and Gompertz-like functions arising from the temperature response of development (exponential), fecundity (Gaussian), and the interaction between development and mortality (Gompertz-like). For $r_m(T) > 0$, the negative effect of increasing adult mortality with temperature must be counteracted by the positive effects of increasing fecundity and accelerated development as temperatures increase (fig. 1*c*–1*e*).

Results

Elucidating the temperature dependence of r_m in terms of underlying life-history traits allows us to explain two key empirical findings. The first is the unimodality of $r_m(T)$, in particular, the decline of r_m at high temperatures. The second is the asymmetry (left skew) of $r_m(T)$, with the lower critical threshold (T_{min}) being farther away from the optimum temperature (T_{opt}) than the upper critical threshold (T_{max}).

To understand why $r_m(T)$ is unimodal, we characterize the width and skew of $r_m(T)$. We obtain T_{min} and T_{max} by setting the right-hand side of equation (11) to 0 and solving for T; we obtain T_{opt} by setting $(\partial r_m(T))/\partial T = 0$ and solving for T. These solutions must be obtained numerically. We can understand the properties of $r_m(T)$ graphically by plotting the two terms of $r_m(T)$ as a function of temperature (fig. 1*c*–1*e*). The important point is that we can use the temperature responses of fecundity, development, and mortality, which are routinely measured in many ectothermic taxa, to predict the shape of $r_m(T)$ as characterized by T_{min}, T_{opt}, and T_{max}.

The empirically observed unimodality of $r_m(T)$ is the direct result of the qualitative differences between the temperature responses of fecundity, development, and mortality. Development and mortality rates increase with increasing temperature, while fecundity increases with temperature up to a maximum and declines thereafter. At low temperatures, therefore, all three life-history traits increase with increasing temperature. Provided that the increase in fecundity with increasing temperature is greater than the increase in mortality, r_m will increase as temperature increases. The greater the sensitivity of development to temperature, the greater the increase in fecundity relative to the increase in mortality and, hence, the greater the increase in r_m. As temperatures increase beyond the optimal range for reproduction, fecundity starts to decline while mortality continues to increase, causing r_m to decline at high temperatures. The wider the response of reproduction to temperature, the greater the temperature range over which r_m is positive (fig. 1).

We next explain the observed asymmetry of $r_m(T)$: the increase in fitness up to that optimum temperature is much slower than the decrease in fitness when environmental temperatures exceed the optimum (i.e., $T_{\text{max}} - T_{\text{opt}} < T_{\text{opt}} - T_{\text{min}}$; Deutsch et al. 2008). Numerical evaluation of equation (11) for different values of A_α, A_d, $A_{\bar{d}}$, and s shows that A_α and s are the key factors that determine the asymmetry of $r_m(T)$ (figs. 2, 3). The temperature sensitivities of juvenile and adult mortality ($A_{\bar{d}}$, A_d) have roughly equivalent effects on T_{min}, T_{opt}, and T_{max} and hence no strong qualitative effect on the unimodality or asymmetry of $r_m(T)$. They have the quantitative effect of reducing the magnitude of r_m at T_{opt}, which is stronger for $A_{\bar{d}}$ than it is for A_d (app. D, figs. D1–D4, available online).

The temperature sensitivity of development (A_α) is crucial in explaining the asymmetry of $r_m(T)$. When A_α is high, the positive contribution to fitness (via fecundity and development) increases with temperature at an accelerating rate. Since the negative contribution to fitness (via mortality) also increases at an accelerating rate, mortality can overwhelm reproduction only after the optimal temperature range for reproduction is exceeded and fecundity has begun to decline (fig. 2*c*). The net effect of declining fecundity and increasing mortality is a rapid decline in fitness when temperature exceeds the optimum (as opposed to the slow increase to the optimum when fecundity and mortality are both increasing with temperature). As a result, T_{max} is closer to T_{opt} than to T_{min} (fig. 2*d*). In contrast, when A_α is low, the positive contribution to fitness increases with temperature at a decelerating rate (fig.

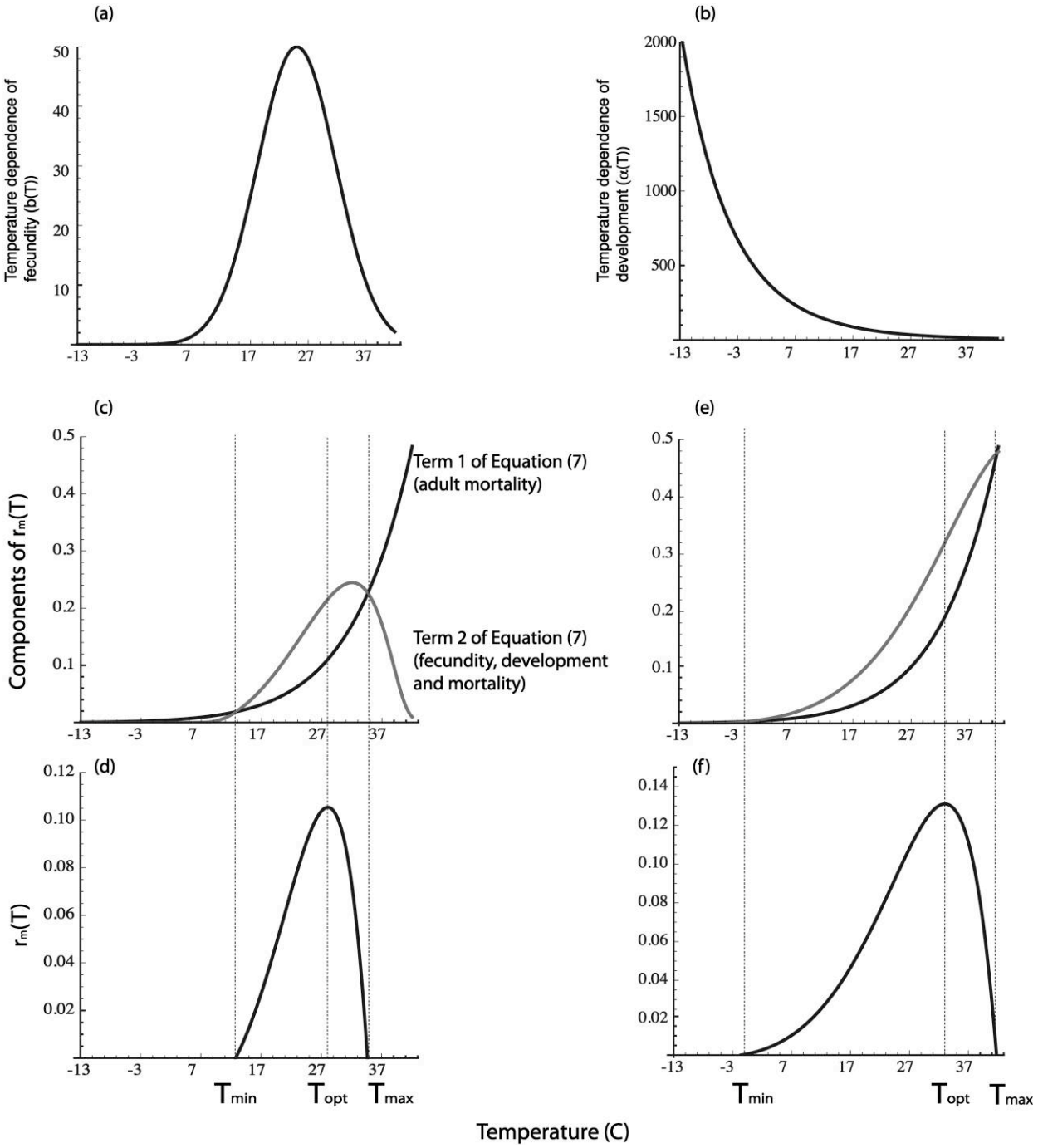


Figure 1: Temperature dependence of fecundity (*a*) and development (*b*; as specified by eq. [6]) and r_m (*c*, *d*). In *c*, the black curve is the negative contribution to fitness via adult mortality (negative of the first term of eq. [7]); the gray curve is the positive contribution to fitness via reproduction, development, and mortality (second term of eq. [7]). *d*, The function $r_m(T)$ resulting from *c*. The function $r_m(T)$ is 0 when the two curves in *c* cross, and these points correspond to T_{min} and T_{max}; $r_m(T)$ is maximal when the difference between the two curves is the largest, which occurs at T_{opt}. A comparison of *c* and *d* with *e* and *f* illustrates the effects of *s* on $r_m(T)$: as *s* increases ($s = 2.5$ in *c* and *d* and $s = 4.8$ in *e* and *f*), $r_m(T) > 0$ over a wider temperature range. Increasing $A_{\bar{d}}$ and A_d have no qualitative effect on $r_m(T)$ (figs. D1–D4, available online). Equation (7) requires that temperature be in Kelvin, but graphs are plotted in Celsius for ease of interpretation. Parameter values are as follows: $A_\alpha = -8{,}000$, $A_{\bar{d}} = 7{,}500$, $A_d = 10{,}000$, $T_R = 294$ K, $b_{T_R} = 50$, $\bar{d}_{T_R} = 0.03$, $d_{T_R} = 0.05$, $\alpha_{T_R} = 60$, $T_{opt_b} = 298$ K.

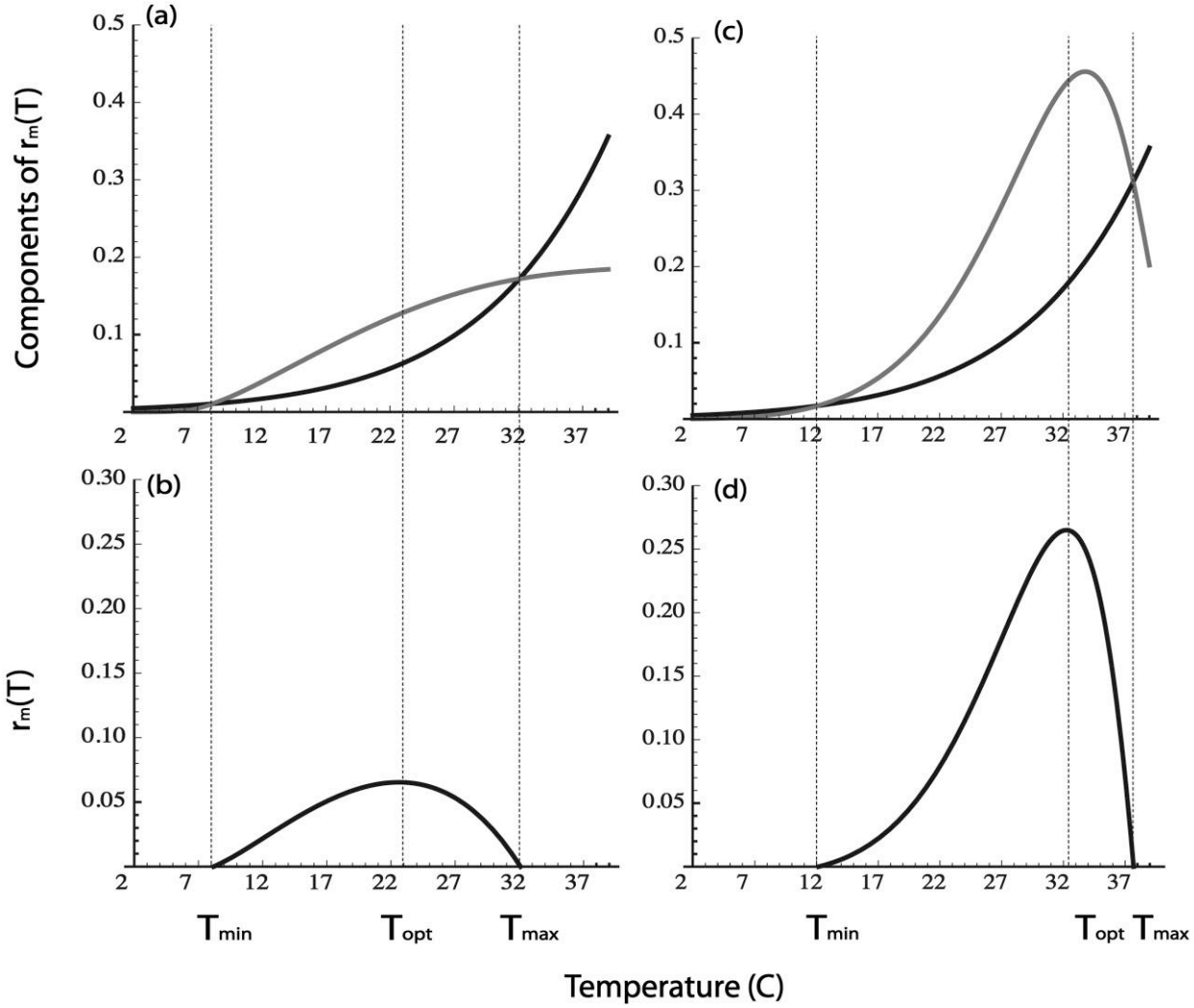


Figure 2: Effects of the temperature response of development on the asymmetry of $r_m(T)$. The temperature sensitivity of development (A_α) determines how close T_{max} is to T_{opt}. When A_α is low (*a*, *b*; $A_\alpha = -2{,}000$), the decline in r_m at high temperatures is relatively slow because organisms develop more slowly and their reproductive life span becomes shorter. Then mortality can exceed reproduction even before fecundity starts to decline with temperature, causing r_m to decline over a wider temperature range. As a result, T_{max} is farther away from T_{opt}. Conversely, when A_α is high (*c*, *d*; $A_\alpha = -15{,}000$), the decline in r_m at high temperatures is relatively fast because organisms develop faster and their reproductive life span is longer. Hence, mortality can overwhelm reproduction only after fecundity has started to decline with temperature. Since fecundity is declining while mortality is increasing, r_m declines to 0 within a narrow temperature range. As a result, T_{max} becomes closer to T_{opt}. Parameter values are as in figure 1 except $s = 3$, $A_{\bar{d}} = 9{,}000$, and $A_d = 10{,}000$.

2*a*). Since mortality increases with temperature at an accelerating rate, fitness is maximized at a relatively low temperature, one at which fecundity is still increasing (fig. 2*a*). Because fecundity continues to increase at temperatures past T_{opt}, the decline in fitness with increasing temperature is relatively slow. As a result, T_{max} is farther away from T_{opt} (fig. 2*b*).

Our formulation of $r_m(T)$ (eq. [11]) considers that developmental rate ($1/\alpha$) increases monotonically with temperature. In some species, the developmental rate can decelerate and even decline at high temperatures (Trudgill et al. 2005; Bowler and Terblanche 2008; Dixon et al. 2009). If the developmental rate starts to decline at temperatures that are too high for reproduction, it has no effect on $r_m(T)$; if it starts to decline within the temperature range that allows for reproduction, it will increase the left skewness of $r_m(T)$. This is because a decrease in the developmental rate reduces the reproductive life span of individuals, which in turn allows mortality to overwhelm reproduction at a lower temperature. As

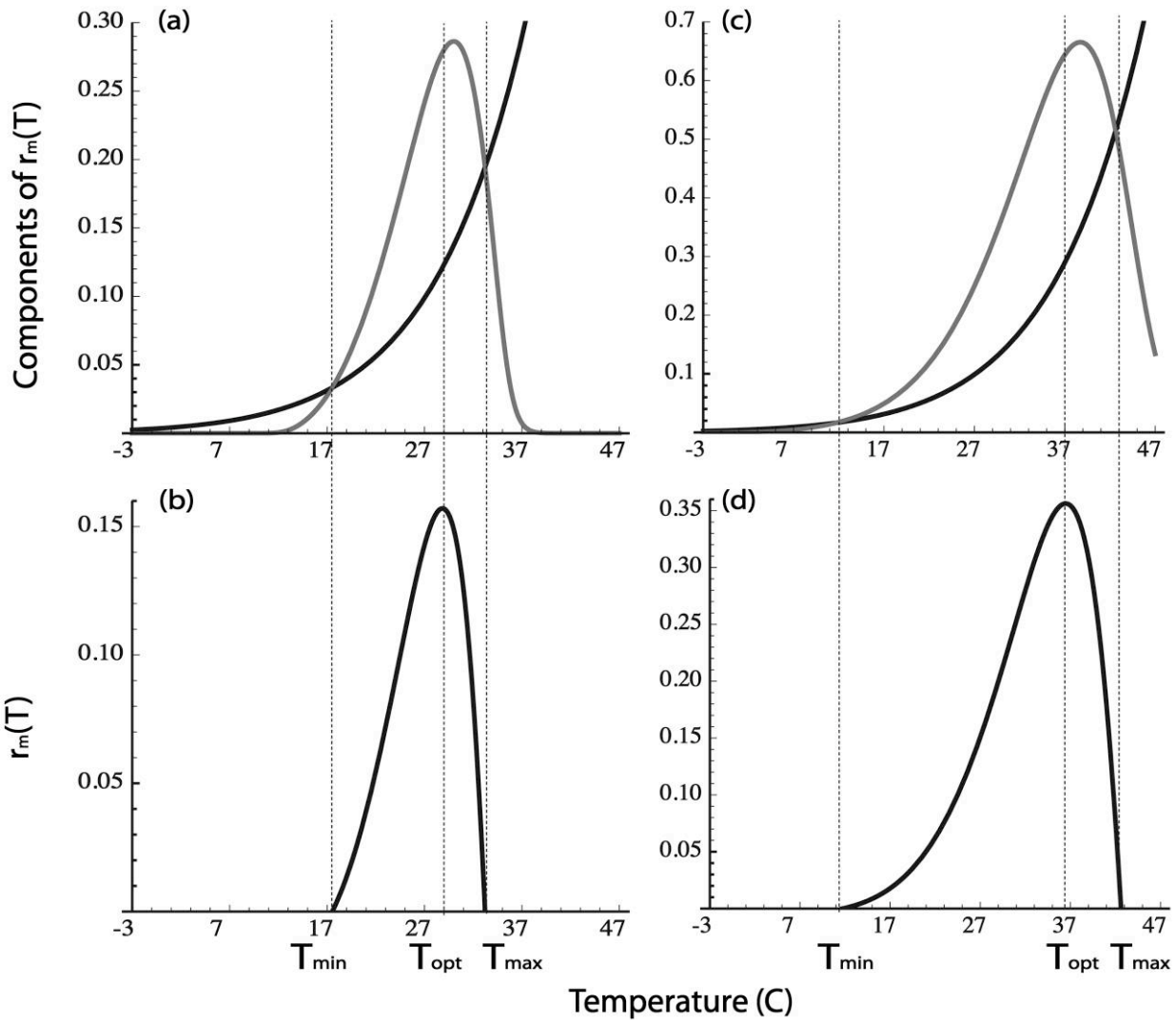


Figure 3: Effects of the temperature response of reproduction on the asymmetry of $r_m(T)$. The width of the temperature response (s) determines how close T_{min} is to T_{opt}. When s is low (a, b; $s = 2.0$), $T_{opt} - T_{min}$ is only slightly greater than $T_{max} - T_{opt}$. As s increases (c, d; $s = 4.0$), T_{min} decreases while T_{opt} and T_{max} increase. The decrease in T_{min} is greater than the increase in T_{opt} or T_{max}, and hence the left tail increases with s even faster than the right tail. These two effects result in a greater asymmetry of $r_m(T)$. Parameter values are as in figure 1 except $A_\alpha = -15{,}000$, $A_{\bar{d}} = 9{,}000$, and $A_d = 10{,}000$.

a result, T_{max} will be closer to T_{opt} than when the developmental rate increases monotonically with temperature.

While the decline in fitness at high temperatures occurs within a narrow temperature range, the increase in fitness up to the optimum temperature occurs over a much wider temperature range. This is because when environmental temperatures are low, the increase in fecundity with increasing temperature is not much greater than the increase in mortality. Hence, the increase in fitness with temperature ($\partial r_m(T)/\partial T$) is relatively slow. It is only when temperatures approach the optimum range for reproduction that the increase in fecundity substantially exceeds the increase in mortality, causing fitness to increase with temperature relatively quickly. As a result, T_{min} is farther away from T_{opt} than T_{max}. The width of the temperature response of reproduction (s) has a strong effect on the proximity of T_{min} to T_{opt} (fig. 3*a*–3*d*). When s is high, fitness increases with temperature at an accelerating rate; that is, $\partial r_m(T)/\partial T$ is very small at low temperatures and increases rapidly as temperatures approach the optimum for reproduction. This causes T_{min} to be farther away from T_{opt}, thus

making for a highly asymmetric temperature response (fig. 3*c*, 3*d*).

These findings suggest that species with low s and high A_α values are likely to exhibit a narrower and more symmetric temperature response of fitness than species with high s and low A_α values. Previous studies suggest that the asymmetry of the fitness response is a function of latitude (Deutsch et al. 2008; Tewksbury et al. 2008). If so, we expect temperate ectotherms to exhibit greater asymmetry in $r_m(T)$ than tropical ectotherms. We also expect the asymmetry of $r_m(T)$ in subtropical and Mediterranean ectotherms to be intermediate between those of tropical and temperate ectotherms.

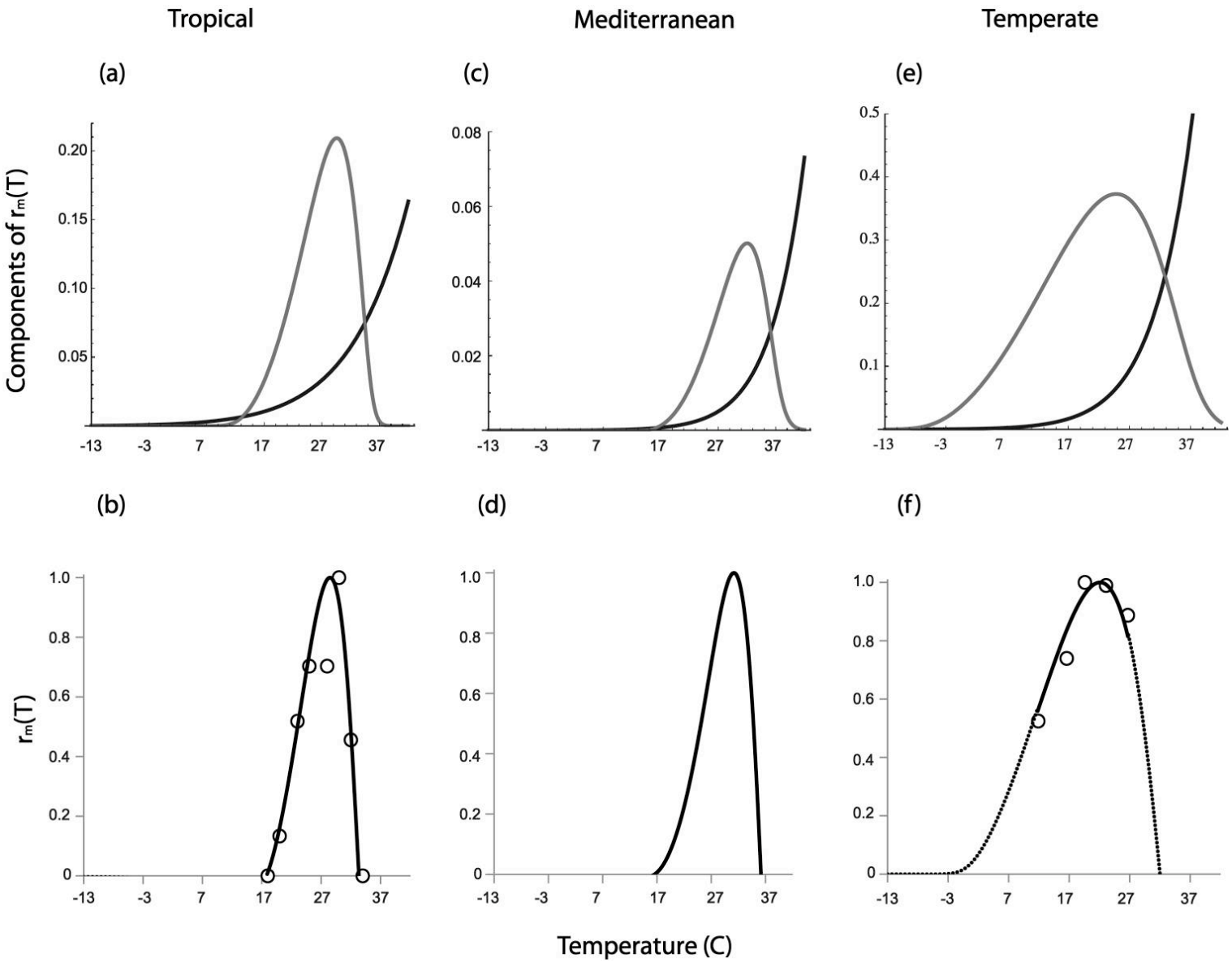
We tested these predictions with data from three Hemipteran species from tropical, Mediterranean, and temperate localities. Our goal was to validate our model with data from representative species inhabiting different latitudes rather than perform an exhaustive analysis of a large number of species; such an analysis is an important next step (see "Discussion"). The tropical species is a pod-sucking bug (*Clavigralla shadabi*; Hemiptera: Coreidae) from Benin (8°20′N; Dreyer and Baumgartner 1996), the Mediterranean species is the harlequin bug (*Murgantia histrionica*; Hemiptera: Pentatomidae) from coastal southern California (33°37′8″N; Amarasekare 2000*a*, 2000*b*, 2003, 2007), and the temperate species is the pea aphid (*Acyrthosiphon pisum*; Hemiptera: Aphididae) from York, England (53°57′30″N; Morgan et al. 2001). The tropical and temperate species are the same ones used by Deutsch et al. (2008) in their study of extinction risk due to climate warming. We used empirically measured temperature responses of reproduction, development, and mortality for each species (Dreyer and Baumgartner 1996; Morgan et al. 2001; P. Amarasekare, unpublished data) to parameterize equation (11) and to predict the expected $r_m(T)$ (see app. E, available online, for details). The predicted $r_m(T)$ for the tropical and temperate species provide a good match with the empirically measured $r_m(T)$ (fig. 4). The asymmetry of the empirically measured $r_m(T)$ is greater in the temperate species compared with the tropical species, but the asymmetry of the Mediterranean species is not greater than that of the tropical species (fig. 4). While these results contradict expectations based purely on latitude, they are easily explained within our framework. Data show the temperate species to have a wider temperature response of reproduction and a lower temperature sensitivity of development than the tropical species. As we have shown above, this combination of responses can cause $r_m(T)$ to become highly asymmetric. The similarity between the tropical and the Mediterranean species arises because the latter has as narrow a temperature response of reproduction as the former and a much higher temperature sensitivity of development (table 1). Of note, the tropical and the Mediterranean species, both pod-sucking bugs, are more closely related to each other than to the temperate species, the pea aphid, suggesting a possible phylogenetic influence on the temperature responses of fitness components.

Discussion

Understanding how climate change influences species extinction is an issue of great ecological and environmental concern. Because fitness is strongly temperature dependent in ectotherms, it provides a metric for assessing extinction risk. Here we have developed a mathematical framework that partitions the temperature dependence of fitness into its components of fecundity, development, and mortality. Because the qualitative properties of the temperature responses of fitness components are robust to taxonomic and geographic variation (Gillooly et al. 2001*a*, 2001*b*; Savage et al. 2004), our partitioning applies to ectothermic taxa inhabiting all latitudes. Most importantly, it allows us to both explain the observed fitness-temperature relationships and predict their consequences for species extinctions.

The key prediction of our framework is that species exhibiting a narrower temperature response of fitness, with T_{opt} closer to T_{max}, should have a narrower temperature response of reproduction and a greater temperature sensitivity of development (i.e., lower s and higher A_α) compared with species that have a wider temperature response of fitness, with T_{opt} farther away from T_{max}. In testing this prediction with insects from different latitudes, we found that the tropical species, which exhibits a narrower temperature response of fitness, does indeed have lower s and higher A_α values than the temperate species. However, the Mediterranean species has similar s and higher A_α values than the tropical species and a comparably narrow fitness response. This finding highlights an important point: any ectothermic species, regardless of its geographic location, will be at greater risk of extinction because of climate warming if it has a narrow temperature response of reproduction and a high temperature sensitivity of development.

Previous explanations of the temperature response of fitness (e.g., Angilletta 2009; Kingsolver 2009) are based on reaction rate theory, which considers temperature effects on the kinetics and inactivation of a single major enzyme (Johnson and Lewin 1946; Sharpe and DeMichele 1977; Schoolfield et al. 1981; Van der Have and de Jong 1996; Van der Have 2002; Ratkowsky et al. 2005). However, fitness is a composite trait consisting of individual components (e.g., fecundity, development, survivorship). While enzyme kinetics may play a role in the temperature responses of fitness components, these responses are also


Tropical
Mediterranean
Temperate
(a)
(c)
(e)
(b)
(d)
(f)
Components of $r_m(T)$
$r_m(T)$
Temperature (C)
0.20
0.15
0.10
0.05
0.08
0.06
0.04
0.02
0.5
0.4
0.3
0.2
0.1
1.0
0.8
0.6
0.4
0.2
0
-13
-3
7
17
27
37

likely to be affected by other physiological processes that are temperature dependent. For instance, cumulative survivorship (l_x), especially embryonic viability, exhibits a temperature response that is inverted U-shaped rather than left skewed (Van der Have 2002; Angilletta 2009; Kingsolver 2009), and per capita mortality rates exhibit a monotonic temperature response (Gillooly et al. 2001*a*; Savage et al. 2004). Reproductive traits (e.g., mating, egg laying) show a more symmetric temperature response than the left-skewed one predicted by reaction rate theory (e.g., Dreyer and Baumgartner 1996; Carriere and Boivin 1997; Medeiros et al. 2000, 2003; Morgan et al. 2001; Dannon et al. 2010; P. Amarasekare, unpublished data; app. E). Because reproductive traits are also under strong hormonal regulation, their temperature responses are likely to be driven by temperature effects on hormonal activity (Nijhout 1994). Developmental rates exhibit temperature responses that are most consistent with the left-skewed response predicted by the reaction rate theory (Sharpe and DeMichele 1977; Schoolfield et al. 1981; Van der Have and de Jong 1996; Van der Have 2002), but in many species the developmental rate increases with temperature at a decelerating rate and does not appear to decline within the biologically relevant temperature range (Trudgill et al. 2005; Bowler and Terblanche 2008; Dixon et al. 2009). Although there is no doubt that the thermodynamics of enzyme reaction rates plays an important role in the organism-level temperature responses of life-history and performance traits, a mechanistic understanding of the temperature dependence of fitness requires that we also understand how temperature affects other physiological processes such as hormonal regulation. It may well be that the temperature responses of reproductive traits result from the interplay between temperature effects on rate processes such as enzyme kinetics and on regulatory processes based on negative feedback pathways such as hormonal action. Ultimately, it is through a mechanistic understanding of how temperature affects key life-history traits that we can hope to understand and predict how ectotherms respond to perturbations such as climate warming (Huey and Berrigan 2001; Kingsolver et al. 2011).

Our mathematical framework, and the studies that motivated it (e.g., Deutsch et al. 2008), are based on the temperature dependence of fitness quantified as the intrinsic growth rate. In real systems, fitness is likely to be influenced by density-dependent factors such as resource limitation and natural enemy pressure. It is therefore important to verify that predictions of extinction risk that are based on the temperature dependence of r_m also hold in population dynamical models that incorporate resource competition and interactions with natural enemies. If predictions based on such models match those based on r_m, we can have greater confidence in using the temperature dependence of r_m to predict extinction risk due to climate warming. Studies are currently under way (P. Amarasekare, unpublished manuscript) to investigate this issue.

The crucial insight to emerge from our analysis is the effect of development on the temperature dependence of fitness. While the effects of reproduction and mortality are relatively easy to predict (e.g., the decline in fitness at high temperatures is the result of mortality exceeding reproduction), the effects of development are indirect and could not have been elucidated without mathematical theory. As we have shown, temperature effects on development are the key to explaining both the unimodality and the asymmetry of the temperature response of fitness. Huey and Berrigan (2001) found that the temperature response of r_m was more left skewed than that of the basic reproductive rate (R_0), which they predicted should occur because r_m depends on the generation time, which itself decreases with increasing temperature. This study, which was based on empirical measures of r_m and R_0 in several ecothermic taxa, highlighted how temperature effects on development could lead to qualitatively different temperature responses for r_m and R_0. However, it did not explain why the temperature response of r_m should be left skewed at all or why there exist species-specific differences in the degree of skewness of $r_m(T)$ (e.g., tropical species exhibit $r_m(T)$ values that are significantly less left skewed than those of temperate species; Deutsch et al. 2008). Our mathematical analyses show that the temperature sensitivity of development is key to generating the left-skewed temperature response of r_m but that this effect can be counterintuitive. For instance, species with greater a temperature sensitivity of development (and hence a shorter generation time) exhibit a less left-skewed temperature response than species with a lower temperature sensitivity of development (and hence a longer generation time), but the optimal temperature at

Figure 4: Temperature responses of fitness for three Hemipteran species inhabiting tropical (*a*, *b*), Mediterranean (*c*, *d*), and temperate (*e*, *f*) latitudes. In *b*, *d*, and *f*, the solid curve is the predicted $r_m(T)$ from equation (7) parameterized with empirically quantified temperature responses of fecundity, development, and mortality (table 1; app. E, available online). The open circles are direct measurements of r_m that we have normalized to 1 for ease of comparison. Direct measures of r_m are not available for the Mediterranean species. For the tropical and temperate species, r_m predicted from the model provides a good fit to the empirical measurements of r_m (Kolmogorov-Smirnov test; tropical species: $D = 0.375$, $P = .6272$; temperate species: $D = 0.2$, $P = 1$; note that higher P values imply a better fit between observed and predicted $r_m(T)$).

Table 1: Key parameters of the temperature responses of fecundity, development, and mortality (eqq. [8]–[10]) for three Hemipteran species from tropical, Mediterranean, and temperate latitudes

	Species					
	Tropical (*Clavigralla shadabi*)		Mediterranean (*Murgantia histrionica*)		Temperate (*Acyrthosiphon pisum*)	
Life-history trait	Mean ± SE ($N = 8$)	P	Mean ± SE ($N = 9$)	P	Mean ± SE ($N = 5$)	P
Fecundity ($\bar{b}(T)$):						
$T_{\text{opt}_{\bar{b}}}$ (°K)	299.5 ± .41	<.00001	298 ± .29	<.00001	290.3 ± .7	<.0001
s	2.67 ± .29	<.00001	3.39 ± .25	<.00001	4.13 ± .58	.006
Development ($\alpha(T)$):						
A_α	−11,112 ± 731	<.00001	−15,299 ± 654	<.00001	−5,108 ± 687	.002
Juvenile mortality ($\bar{d}(T)$):						
$A_{\bar{d}}$	31,910 ± 1,201	<.00001	10,486 ± 2,001	.002	14,518 ± 1,901	.002
Adult mortality ($d(T)$):						
A_d	10,035 ± 1,117	<.00001	16,826 ± 655	<.00001	15,004 ± 2,679	.0049

Note: See appendix E for details on parameter estimation. N values are the sample sizes, that is, the numbers of constant temperatures at which temperature responses were measured.

which r_m is maximized (T_{opt}) is greater for species with a greater temperature sensitivity of development.

Our findings suggest that temperature effects on development are likely the cause of the widely reported phenological shifts in many ectotherms (Dunn and Winkler 1999; Walther et al. 2002; Root et al. 2003; Inouye 2008; Miller-Rushing and Primack 2008; Post et al. 2008). Climate warming will cause species with a high temperature sensitivity of development to reach the age at first reproduction earlier in the year. If such species depend on biotic resources (e.g., herbivores on plants, insect parasitoids attacking herbivorous hosts) that have a lower temperature sensitivity of development, resource abundance will lag behind consumer abundance, causing the consumer species' fitness to decline.

An important implication of our findings is that factors other than latitude may be important in predicting extinction risk due to climate warming. Even within a given latitude, species may differ in the temperature responses of reproduction and development due to their ecological roles. In fact, temperature responses of a species' life-history traits may depend on its trophic position (e.g., resource, intermediate consumer, predator) and the degree of trophic specialization. For instance, temperature responses of specialist consumers and predators may be narrower than those of generalists because the former are likely to be more constrained by the temperature responses of their resources and prey. Along the same lines, properties such as invasiveness may be a result of the relative flexibility in the temperature responses of life-history traits. Many insect pests with tropical or subtropical origins have proved to be highly successful in Mediterranean and temperate regions (Hill 1983, 1987). Similarly, natural enemies such as insect parasitoids used for pest control have also proved to be successful when introduced outside their native range (Clausen 1978). Conversely, species inhabiting different latitudes may show similar temperature responses because of their shared phylogenetic history (Bogert 1949); this could be the case with the pod-sucking bugs from the tropical and the Mediterranean regions (fig. 4). Given that data on the temperature responses of life-history traits are available for numerous ectothermic taxa in different habitats across all latitudes, an important future direction would be to evaluate the roles of geography, phylogeny, and ecology in driving the temperature dependence of fitness.

In conclusion, partitioning of the temperature response of fitness into its basic components highlights how differential effects of temperature on life-history traits such as fecundity, development, and survivorship can affect overall fitness. It provides a mechanistic basis for identifying which species are more likely to be impacted by perturbations to their thermal environments, and hence a more rigorous means of predicting extinction risk due to climate warming. By focusing on the interaction between the temperature responses of fitness components, this study provides a way to integrate thermal biology, which focuses on temperature responses of fitness components at the physiological level, with life-history theory, which links fitness components to demographic processes.

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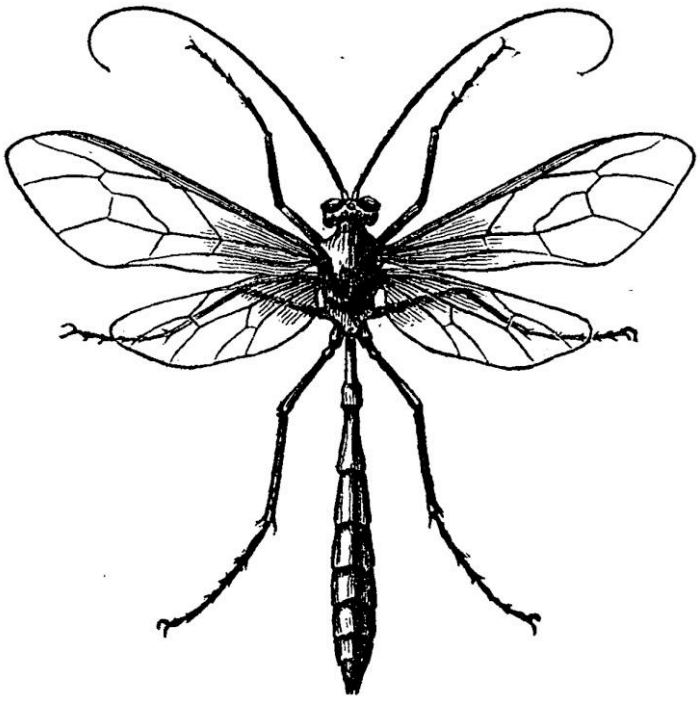

Ophion macrurum Linn. Ichneumon parasite on the larva of *Telea polyphemus*. "When an Ichneumon detects the presence of a worm, she flies around it for a few seconds, and then rests upon the leaf near her victim; moving her antennae very rapidly above the body of the worm but not touching it and bending her abdomen under the breast, she seizes her ovipositor with the front legs, and waits for a favorable moment, when she quickly deposits a little oval white egg upon the skin of the larva." From "The American Silk Worm" by L. Trouvelot (*American Naturalist*, 1867, 1:85–94).