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ORIGINAL ARTICLE

Survival in spatially variable thermal environments: Consequences of induced thermal defense

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Abstract

As Earth's climate warms, plants and animals are likely to encounter increased frequency and severity of extreme thermal events, and the ensuing destruction is likely to play an important role in structuring ecological communities. However, accurate prediction of the population-scale consequences of extreme thermal events requires detailed knowledge of the small-scale interaction between individual organisms and their thermal environment. In this study I propose a simple model that allows one to explore how individual-to-individual variation in body temperature and thermal physiology determines what fraction of a population will be killed by an extreme thermal event. The model takes into account the possibility that each individual plant or animal can respond to an event by adjusting its thermal tolerance in proportion to the stress it encounters. When thermal stress is relatively mild, the model shows that a graded physiological response of this sort leads to increased survivorship. However, the model predicts that in more severe events a proportional induced defense can actually reduce survivorship, a counterintuitive possibility that is not predicted by standard theory. The model can easily be tailored to different species and thermal environments to provide an estimate of when, where and how physiology can buffer the effects of climate warming.

Key words: induced defense, plasticity, small-scale variability, thermal physiology, thermal tolerance

INTRODUCTION

Over the next century, terrestrial plants and animals are likely to encounter an increased frequency and severity of extreme thermal events, and these events are likely to play an important role in restructuring ecological communities (IPCC 2013). However, at any given location, the effects of topography, inter-individual shading, and behavior ensure that individuals will differ in the temperature they experience during an extreme event (Gates 1980; Angilletta 2009; Denny 2016). Furthermore, individuals inevitably differ in their physiological tolerance, a result of variation in their genetics, development, thermal history, age, energy stores and reproductive status (Somero *et al.* 2017). Consequently, the fraction of individuals killed by a stressful thermal event depends on the pattern in which individual temperatures and tolerances co-occur. In particular, even

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when the mean maximum temperature of an event exceeds the mean tolerance (which at first glance might lead one to suppose that all individuals die), some individuals with above average tolerance will, by chance, experience below average temperatures and thereby survive (Denny *et al.* 2011). In short, latitudinal patterns and population-wide consequences of rising average temperatures are likely to be strongly affected by individual variation in body temperature and thermal physiology (Dong *et al.* 2017).

In this study I propose a simple model that allows one to explore the interaction between environmentally-imposed, stressful body temperatures and individuals' thermal tolerances. In particular, the model takes into account the possibility that an individual plant or animal can adjust its physiology in proportion to the thermal stress it encounters. For instance, at the cellular scale heat-shock proteins may be produced as a means to cope with the heat-induced denaturation of proteins (e.g. Feder & Hofmann 1999; Shudo *et al*. 2003; Somero *et al.* 2017), the composition of membranes can be adjusted to maintain the proper viscosity (Somero *et al.* 2017), and a variety of processes contribute to "heat hardening" (Bowler 2005). At the organismal scale, animals can sweat, pant, change color to absorb less solar irradiation, and direct additional blood flow to the periphery of their bodies to enhance convection heat transfer (Schmidt-Nielsen 1997). Trees can produce leaves of different shapes in response to the solar heat load (Vogel 1968).

The model I present here shows that mounting an induced thermal defense often leads to increased survivorship, a result that accords with standard theories of induced defense (e.g. Harvell 1990; Scheiner 1993). However, the model also shows how, in exceptionally stressful events, a proportional induced defense can reduce survivorship, a counterintuitive possibility that is not predicted by standard theory.

THEORY

Denny *et al.* (2011) developed a model describing how the distribution of individual thermal tolerances interacts with the distribution of maximum imposed body temperatures to predict the fraction of individuals surviving an extreme thermal event; that is, an event capable of killing a substantial fraction of organisms. The ideas proposed here build on the model of Denny *et al.* (2011), so I begin with a review of that model's key concepts and assumptions.

For simplicity, Denny *et al.* (2011) assumed that the distributions of thermal tolerance (T_{tol}, the highest body temperature an individual can survive) and body temperature (T_{max}, the maximum body temperature an individual experiences) are Gaussian (Fig. 1), each characterized by its mean (μ_{tol} and μ_{max}) and its standard deviation, an index of variability (σ_{tol} and σ_{max}). Each variable is measured in degrees Celcius.

Denny *et al.* (2011) assumed that T_{tol} is *constitutive*; that is, the maximum temperature an individual can tolerate is a fixed value, independent of the temperatures it experiences in a particular extreme thermal event. For present purposes, I note that constitutive tolerance is a fixed phenotypic trait set by both an individual's genotype and by what Lynch and Gabriel (1987) refer to as "developmental noise," random variation in developmental pathways as the organism grows.

Denny *et al.* (2011) evaluated the interaction between individual capacity (T_{tol}) and environmental stress (T_{max}) mathematically (see Appendix I), but given the above descriptions of T_{tol} and T_{max}, the fraction S of individuals surviving an extreme thermal event can equivalently be estimated through a simple simulation. One choses two values at random, one from the distributions of tolerance, *Tol*, and one from the distribution of imposed thermal stress, *Max*. The resulting pair of T_{tol} and T_{max} values

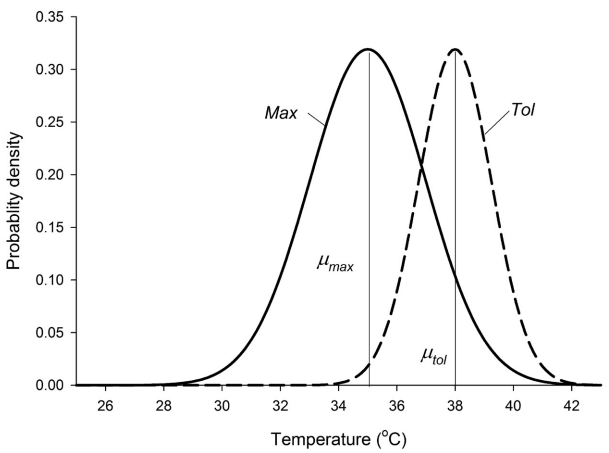

Figure 1 The distributions of maximum temperature and constitutive thermal tolerance in the model of Denny *et al.* (2011). Here, *Tol*, the distribution of constitutive tolerance has the mean and standard deviation appropriate for the mussel, *M. californianus* (μ_{max} = 38 °C, σ_{max} = 1.25 °C). *Max* is shown with a mean appropriate for the current climate (μ_{max} = 35 °C) and the standard deviation reported for beds in central California (2 °C).

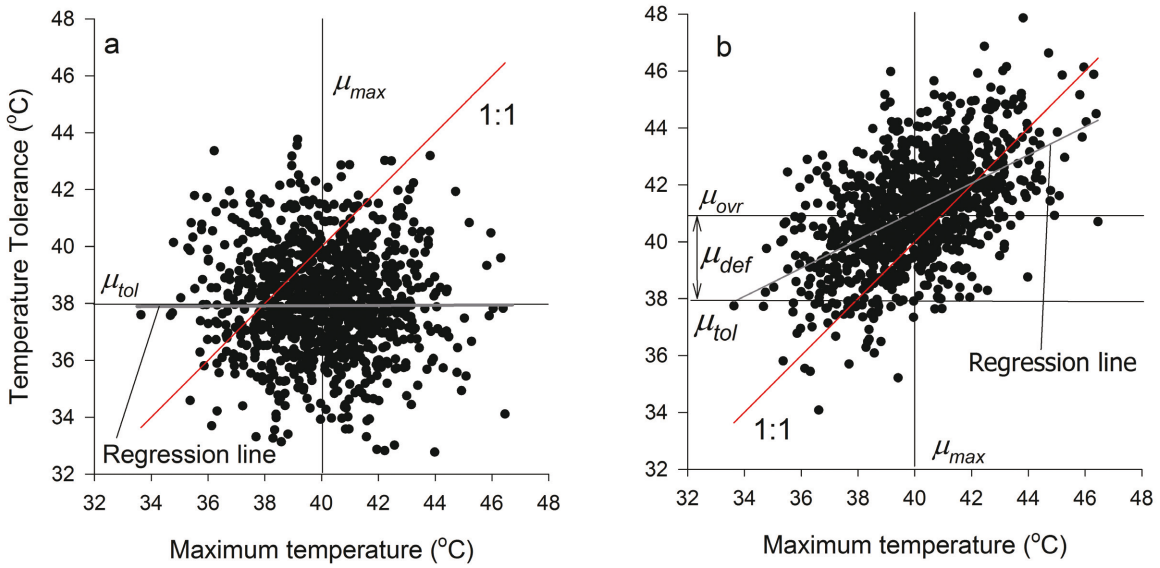

Figure 2 Graphical depictions of variation among individuals in maximum temperature and constitutive thermal tolerance. (a) Here, thermal tolerance is independent of the maximum temperature encountered. Note that mean maximum tolerance is less than mean maximum temperature; as a consequence, most points fall below the 1:1 line and these individuals die. (b) The addition of a proportional induced response increases the mean overall tolerance and gives the data cloud a positive slope. Both effects increase the number of points falling above the 1:1 line, indicating an increase in survivorship.

can be plotted as a point on a graph, and the process can then be repeated (e.g. Fig. 2a). Because each T_{tol} is chosen independently of the chosen T_{max}, the two variables are not correlated; that is, a regression line through the cloud of data has zero slope. (Note for future reference that the regression line passes through the point (μ_{max}, μ_{tol}). See Zar (1999) for an explanation of this characteristic of regression lines.) Inter-individual variability of T_{tol} can be visualized as the vertical spread of the data around μ_{tol}. Similarly, inter-individual variability in T_{max} is seen as the horizontal spread of data relative to μ_{max}. Drawing a line of equality between T_{max} and T_{tol} provides a means to visualize the fraction of individuals that survive: for data points on or above this 1:1 line, T_{tol} is at least as large as T_{max}, and those individuals survive; for points below the line, T_{tol} is less than T_{max}, and those individuals die.

Consideration of Fig. 2a reveals that the fraction of individuals that survive can be affected by the means of both the temperature and tolerance distributions. Increasing mean maximum temperature (μ_{max}) would slide the cloud of points to the right, increasing the fraction that fall below the 1:1 line; decreasing μ_{max} would have the opposite effect. Increasing mean constitutive tolerance (μ_{tol}) would slide the cloud upward, increasing the fraction of individuals surviving; decreasing μ_{tol} would decrease the fraction surviving.

The effect of varying the standard deviations of the two distributions depends on the relative magnitudes of the two means. Consider, for instance, the scenario of Fig. 2a, a highly stressful environment in which mean tolerance is less than the mean maximum temperature. An increase in the variability of imposed temperature, σ_{max} (the result, for instance, of a more topographically complex landscape) would spread the points out along the T_{max} axis relative to μ_{max}, increasing the fraction of both benign, low temperatures and stressful, high temperatures. In the left half of the cloud, this spreading would move points to lower T_{max}, causing some points currently below the 1:1 line to move above it, thereby increasing the fraction of individuals that survive. In the right half of the cloud, increasing σ_{max} would spread points to higher T_{max}. However, all but a few of these points are already below the 1:1 line, and would remain so as they move to higher temperature; therefore, their survival status would not change. In short, because μ_{tol}

$< \mu_{max}$, the increase in the fraction of benign temperatures due to increased variability in T_{max} allows some individuals to survive who otherwise would not, while the increased fraction of high temperatures kills the same individuals who would already die. Thus, in a highly stressful environment ($\mu_{tol} < \mu_{max}$), increasing σ_{max} has the effect of increasing overall survivorship. By the same logic, increasing σ_{tol} (i.e. increasing the spread of points along the tolerance axis due to shifts in the genetics and/or physiology of individuals) would increase the fraction of both highly tolerant and highly susceptible individuals. The spread would preferentially cause some points currently below the line to move above it, increasing the fraction of survivors, while those point already below the line would move farther below and still die. In summary, when the environment is highly stressful ($\sigma_{tol} < \sigma_{max}$), increasing variability in either T_{tol} or T_{max} increases survivorship.

The results would be just the opposite in a benign environment where mean tolerance is greater than mean imposed temperature. In this case, increasing the fraction of high temperatures would kill individuals who would otherwise survive, while increasing the fraction of benign temperatures would only make life more comfortable for those individuals who would already survive. Similarly, when mean tolerance is greater than mean maximum temperature, increasing the spread of highly tolerant individuals would not affect their survival, but increasing the spread of susceptible individuals would cause some to die that would otherwise survive.

The pertinent results of the simulation described above can be summarized by applying it to a model organism, the California mussel *Mytilus californianus* Conrad, 1827, the dominant competitor for space in the mid-intertidal zone on the wave-exposed rocky shores of western North America. Its role in this dynamic ecosystem has been well studied (e.g. Dayton 1971; Paine & Levin 1980), and its thermal environment and thermal physiology are under active investigation (e.g. Gracey *et al.* 2008; Helmuth *et al.* 2010; Denny *et al.* 2011; Jimenez *et al.* 2015, 2016; Somero *et al.* 2017). Using the measured distribution of constitutive tolerances for *M. californianus* (μ_{tol} = 38 °C, σ_{tol} = 1.25 °C; Denny *et al.* 2011) and a representative spatial variation in imposed temperature (σ_{max} = 2 °C; Denny *et al.* 2011), one can predict the effects of interacting spatial variation and individual tolerance (Fig. 3). When the thermal event is relatively benign, that is, when mean tolerance temperature is greater than mean maximum temperature, increasing variation in maximum imposed temperature decreases the fraction of individuals surviving (Fig. 3a). By contrast, when the thermal event is highly stressful, that is, when μ_{tol} is less than μ_{max}, an increase in the variability of imposed temperature *increases* the fraction surviving. (For future reference, note that, regardless of the amount of thermal variation, when μ_{tol} =

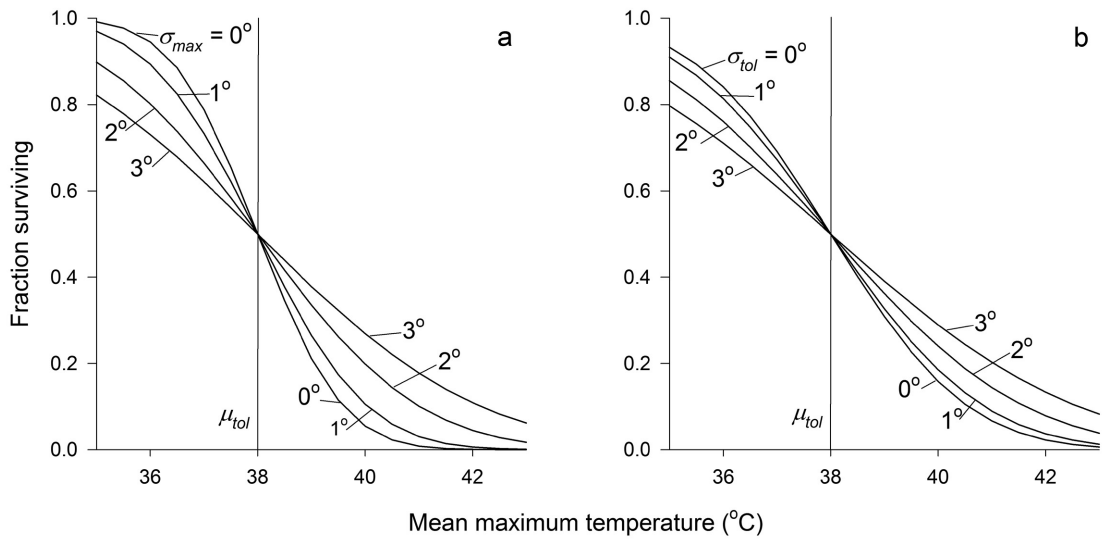

Figure 3 The fraction of mussels surviving as a function of mean maximum temperature and the variation in (a) maximum temperature and (b) constitutive thermal tolerance.

μ_{max}, 50% of individuals survive.)

Although Denny *et al.* (2011) did not explore the possibility, analogous results obtain for variation in thermal tolerance (Fig. 3b). For a relatively benign event in which mean maximum temperature is below mean tolerance, increasing the variability in tolerance among individuals decreases the chance of surviving. By contrast, when the event is exceptionally stressful (i.e. when $\mu_{tol} < \mu_{max}$), an increase in the variability of tolerance increases the likelihood of survival.

Generalizing from these results, Denny *et al.* (2011) conclude that: (i) simply comparing mean maximum temperature to mean tolerance cannot adequately describe the population-level effects of thermal stress; instead, spatial variation in thermal stress must be taken into account; and (ii) inter-individual variation in thermal stress can serve to buffer the consequences of highly stressful thermal events.

However, there are several ways in which the assumptions of this simple model clash with reality. In particular, the simulation's random choice of constitutive thermal tolerance and maximum imposed temperature ensures that an individual's ability to withstand thermal stress is independent of the stress to which it is exposed. In other words, the model assumes that each organism does not adjust its physiological defenses in response to a thermal threat, an assumption that seems blatantly unrealistic. Here I present a revised model that explores how the survival of organisms in a spatially variable thermal environment is affected by an inducible physiological response.

To begin, let's return to Fig. 2a in which individuals do not adjust their physiology in response to the temperature imposed (i.e. the cloud of points has zero slope). What if individuals were capable of responding to imposed thermal stress by mounting an induced defense? To put it another way, how would adaptive physiology affect the graph? To answer this question, I adjust the graph in accordance with four assumptions:

1. There is a cost to mounting an induced defense. For example, if the physiological response to high temperature is to increase the concentration of heat-shock proteins, metabolic expenditures must either be increased or diverted from other activities such as growth (e.g. Feder *et al.* 1992). Diverting energy available for growth potentially reduces reproductive output and evolutionary fitness.

2. Because defense is costly, response to thermal stress is graded. That is, I assume that an organism saves energy by increasing its thermal tolerance in proportion to the severity of imposed stress.

3. Individuals vary in their ability to mount a graded thermal defense.

4. This variability is independent of an individual's constitutive tolerance. The first 3 of these assumptions (defense has a cost, defense is employed in proportion to the stress applied and individuals vary) seem eminently reasonable; the 4th (independence between the ability to increase one's thermal tolerance and the magnitude of constitutive tolerance) is debatable, and I return to it in the Discussion.

A simulation implementing these assumptions (explained below) results in relationships such as that shown in Figure 2b. The regression line through the data cloud is shifted upward by the average magnitude of induced thermal defense, μ_{def}, and the line now has a positive slope because (per assumption 2) the higher the T_{max} to which an individual is exposed the larger the magnitude of its increased ability to withstand high temperatures (on average). The upward shift by itself would inevitably increase the fraction of points above the 1:1

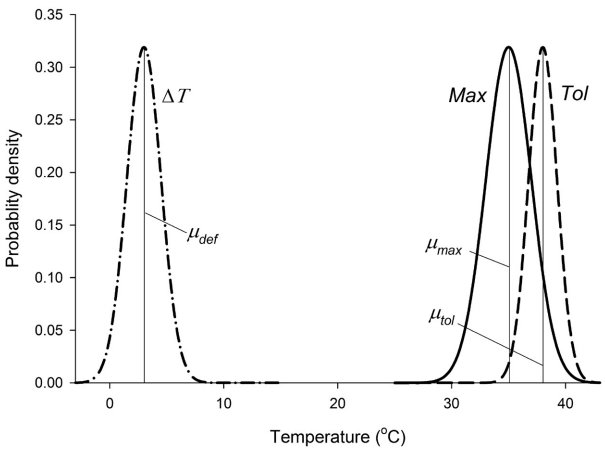

Figure 4 The distributions of maximum temperature, constitutive thermal tolerance and induced response used in the current model. Here, *Tol*, the distribution of constitutive tolerance, has the mean and standard deviation appropriate for the mussel, *M. californianus* (μ_{max} = 38 °C, σ_{max} = 1.25 °C). *Max* is shown with a mean appropriate for the current climate (μ_{max} = 35 °C) and the standard deviation found for beds in central California (2 °C). The distribution of induced responses has a mean of 3 °C and a standard deviation of 1.5 °C, the standard values used in the simulations for *M. californianus*.

line, but the correlation between T_{tol} and T_{max} introduces complexity.

The complex effects of the correlation between T_{tol} and T_{max} can be quantified by a simple extension of the model of Denny *et al.* (2011) (Fig. 4). This extended model includes a 3rd Gaussian distribution, ΔT, the distribution among individuals in a population of their ability to mount an induced thermal defense. The distribution has mean μ_{def} and standard deviation σ_{def}, and individual samples from the distribution provide values of ΔT_{def}, the magnitude of an individual's incremental defensive response (°C) at the time the maximum temperature is imposed (see Appendix II). Taken together, the distributions of ΔT_{def} and T_{tol} form a "snapshot" of how a population of organisms is physiologically poised at the time of the extreme event in question.

Because the distribution of defensive responses is a snapshot in time, it is a much simplified representation of reality. It obscures the fact that both the mean and variation of the defensive response in a particular extreme event will themselves be complicated functions of the interaction between each organism's physiology and its thermal history leading up to the event's climax. The magnitude of an individual's induced defense will depend (at least in part) on the temperature it has experienced in the past and the duration of the peak thermal stress. It is also debatable whether, in nature, ΔT would be Gaussian. I return to these complexities in the Discussion, but in the meantime I will try to convince you that this simple model is, nonetheless, useful.

The extended model (again a simulation) begins as before with the independent, random selection of a T_{max} and an individual's constitutive T_{tol}. A ΔT_{def} is then chosen from the distribution of defensive capability, and this induced response is added to the chosen T_{tol} to determine the individual's overall thermal limit, T_{ovr}:

$$T_{ovr} = T_{tol} + \Delta T_{def}. \quad (1)$$

However, ΔT_{def} is not chosen randomly; instead it is chosen such that ΔT_{def} has a specified correlation with the chosen values of T_{max} (see Appendix III). In other words, if the chosen maximum temperature is above the mean maximum temperature, a ΔT_{def} is chosen that is above the mean induced response and vice versa.

The simulation proceeds by choosing points at random from the distributions of T_{max} and T_{tol} and a point from the distribution of ΔT_{def} that is correlated with the chosen T_{max}. Each chosen value of ΔT_{def} is added to the corresponding choice of T_{tol}, resulting in a distribution of total overall tolerances (T_{ovr}), which has a mean of

$$\mu_{ovr} = \mu_{def} + \mu_{tol} \quad (2)$$

and a standard deviation of

$$s_{ovr} = \sqrt{\sigma_{def}^2 + \sigma_{tol}^2}. \quad (3)$$

(See Taylor [1997] for an explanation of why the standard deviations sum in quadrature.) In sum, given the means and standard deviations of imposed temperature, constitutive tolerance and induced defensive capability, and the physiological correlation between imposed temperature and defensive response, one can prescribe the relationships shown in Figure 2b. The consequences of these relationships can then be explored through simulation. The simulations were programmed in Matlab (ver. R2016a, The MathWorks, Natick, Mass.). A thousand paired samples were taken in each simulation, and the results were averaged over 1000 simulations.

I quantify the results of these simulations using three metrics. The first is simply the fraction of individuals surviving, essentially the same metric employed by Denny *et al.* (2011). However, the fraction surviving fails to take into account the cost of mounting a thermal defense. I assume that the cost of defense (in terms of either metabolic expenditure or fitness) increases in linear proportion to the maximum temperature that can be resisted. In that case, the expenditure incurred by a surviving individual (relative to the average constitutive expenditure among all sampled individuals, μ_{tol}) is a dimensionless index of its cost of survival,

$$cost = \frac{T_{ovr}}{\mu_{tol}}, \quad (4)$$

and this index can be averaged among all survivors:

$$\overline{cost} = \frac{1}{k}\sum_{i=1}^{k}\frac{T_{ovr,i}}{\mu_{tol}}, \quad (5)$$

where k is the number of surviving individuals. Note that $\overline{cost}$ is a dimensionless index rather than a measure of cost per se. In the Discussion, I explore the potential ways in which $\overline{cost}$ might be translated into traditional units.

While the relative cost of survival is a valuable metric, a comparison of that cost to the benefit accrued is equally important. The primary benefit of mounting a thermal defense is survival. Thus, R, a dimensionless

index of the cost–benefit ratio for induced thermal defense, is

$$R = \frac{\overline{cost}}{S} \quad . \tag{6}$$

Here, mean cost is defined by Equation (5) and S is the fraction of individuals surviving, estimated from the simulation.

I explore how the means and standard deviations of imposed temperature, constitutive tolerance and induced defense affect these metrics. Results depend on the values chosen for the model's various parameters; however, a representative picture can be gained by again applying the model to the mussel *M. californianus* using a set of reference values for the model's parameters. From Denny *et al.* (2011) we know that for mussels at Hopkins Marine Station in central California, μ_{tol} = 38 °C and σ_{tol} = 1.25 °C. (This σ_{tol} is representative of intertidal plants and animals in general. Denny *et al.* (2011) compiled a list of known values of σ_{tol}; the mean was 1.08 °C, and values ranged from 0 to 2.5 °C.) For individuals in 1 m^2 of bed, σ_{max} is approximately 2 °C (Denny *et al.* 2011), although there is likely to be larger variation among mussels spread over a larger area (Denny *et al.* 2009). Values for μ_{def} and σ_{def} have not been measured for *M. californianus*. For the sake of argument, I use what *a priori* seem like reasonable values (μ_{def} = 3 °C, σ_{def} = 1.5 °C), and explore the consequences of varying these values. To visualize the effect of these reference values, see Fig. 4.

The primary purpose of these simulations is to quantify the effects of inducible thermal defense. To that end, the correlation between ΔT_{def} and T_{max} was varied from r = 0 (resulting in an increase in defense independent of T_{max}) to r = 1 (which results in an induced defense in perfect proportion to T_{max}). To explore the effects of a warming climate, I used mean maximum temperatures ranging from 3 °C below to 5 °C above the current mean tolerance; that is, from 35 to 43 °C. Harley (2008) and Gracey *et al.* (2008) measured maximum mussel body temperatures of 36 and 38 °C (respectively) during stressful thermal events on the central California coast, so the low end of this temperature range is an approximation of the current thermal environment.

To describe the interacting effects of a warming environment and an induced defense, I calculated survival and the cost and cost–benefit ratio as a function of μ_{max} and r for the reference values of σ_{max}, σ_{tol} and σ_{def}. To explore the interacting effects of inducibility and the variation in temperature, tolerance and defense, I calculated the difference in each metric between r = 1 and r = 0 as functions of σ_{max}, σ_{tol} and σ_{def}. For example, while holding all other variables at their reference values, I calculated the difference in survival between r = 1 (an accurately proportional induced defense) and r = 0 (an uncorrelated defense) as a function of the variability in maximum temperature (σ_{max}) for a range of mean maximum temperatures (μ_{max}) (μ_{tol} – 3 °C to μ_{tol} + 5 °C). This procedure was then repeated, sequentially substituting σ_{tol} and σ_{def} for σ_{max}.

For the sake of simplicity, I apply the model to extreme thermal events involving high temperatures. The model can be applied equally well to extreme low temperatures; see Appendix IV.

A note on terminology. The model makes a clear, mechanistic distinction between constitutive defense (which varies among individuals but is fixed for each individual) and induced defense (which varies among individuals and for each individual may vary in response to thermal stress; Fig. 4). For a population, the magnitude of the induced defense can be independent of the maximum temperature individuals experience (r = 0) or correlated to some extent with T_{max} ($0 < r \leq 1$). It is here that the terminology becomes tricky. If the induced response is uncorrelated with T_{max} (i.e. if r = 0), the results are indistinguishable from an elevated constitutive defense, one with a mean tolerance $\mu_{ovr} = \mu_{tol} + \mu_{def}$. However, even though the tolerances in this scenario appear outwardly as a constitutive defense, because we know that μ_{ovr} is, mechanistically, a combination of constitutive and induced defense, I will refer to the situation of r = 0 as an *uncorrelated induced defense* to differentiate it from both a *correlated induced defense* and the constitutive defense.

RESULTS

Effects of correlation and mean maximum temperature

Results using the reference values for σ_{tol}, σ_{def}, σ_{max}, μ_{tol} and μ_{def} are shown in Fig. 5 as a function of μ_{max} and r. In the absence of correlation between ΔT_{def} and T_{max} (i.e. for an uncorrelated induced defense, r = 0), the results are similar to those reported by Denny *et al.* (2011): survival decreases as mean maximum temperature increases (Fig. 5a). However, due to the average (although uncorrelated) increase in tolerance due to induced defense, 50% survival is now reached when $\mu_{max} = \mu_{ovr}$ rather than when $\mu_{max} = \mu_{tol}$. For relatively benign events (when μ_{max}

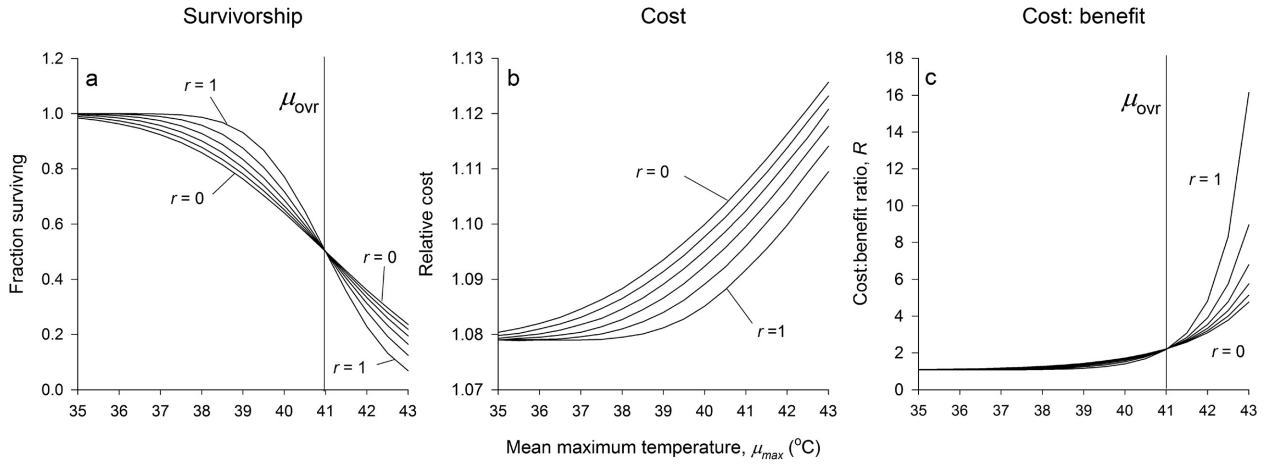

Figure 5 Predictions of survivorship, cost, and cost–benefit ratio. (a) The fraction of individuals in the population surviving a thermal event as a function of mean maximum temperature and the correlation (r) between maximum temperature and induced response. (b) The relative cost of surviving a thermal event as a function of mean maximum temperature and the correlation (r) between maximum temperature and induced response. (c) The cost–benefit ratio associated with surviving a thermal event as a function of mean maximum temperature and the correlation (r) between maximum temperature and induced response. In each panel, r varies sequentially between 0 and 1 in steps of 0.2.

is below μ_{ovr}), a correlated induced thermal defense increases survivorship. By contrast, for exceptionally stressful events (when μ_{max} is above μ_{ovr}) correlated induced thermal defense (surprisingly) *decreases* survivorship relative to an uncorrelated induced defense. In other words, when thermal events are highly stressful, a correlated induced defense is disadvantageous.

Why might a correlated induced defense reduce survivorship when the mean maximum temperature is high? The answer is best illustrated graphically (Fig. 6) using a simplified, heuristic example in which σ_{tol} is set to 0. In this case, the only source of variation in thermal tolerance is that due to variation in induced tolerance. This highlights the effects of a correlated response: when $r = 0$, induced defense is independent of the imposed thermal stress; when $r = 1$, induced defense is perfectly correlated with the imposed stress. Consider first a case in which $\mu_{max} < \mu_{ovr}$ (Fig. 6a,b). When $r = 0$ and T_{tol} is plotted as a function of T_{max} (Fig. 6a), the data form a cloud without any slope. Some points fall below the 1:1 line, so the fraction of individuals surviving is less than 1. However, when $r = 1$ (Fig. 6b), the resulting re-organization of the same T_{tol} and T_{max} data ensures that all individuals survive. This explains why in a relatively benign event ($\mu_{max} < \mu_{ovr}$), induced defense is clearly advantageous.

The converse holds for exceptionally stressful events ($\mu_{max} > \mu_{ovr}$, Fig. 6c,d). When $r = 0$ (Fig. 6c), the data again form a cloud without any slope, although in this case most points fall below the 1:1 line and the fraction of individuals surviving is substantially less than 1. However, because $\mu_{max} > \mu_{ovr}$, a correlated induced defense makes matters even worse. When $r = 1$ (Fig. 6d), the resulting re-organization of the T_{tol} and T_{max} data ensures that all individuals die. This effect explains why, when $\mu_{max} > \mu_{ovr}$, a graded induced defense is disadvantageous.

Adding variation in constitutive tolerance to this heuristic scenario and allowing for different values of r, σ_{max} and σ_{def} would make for quantitative differences in the fraction of individuals surviving, but the qualitative result would be the same: when mean maximum temperature exceeds mean overall tolerance, induced defense is disadvantageous.

Returning to Figure 5b and again using reference values for σ_{tol}, σ_{def}, σ_{max}, μ_{tol} and μ_{def}, we see that the relative cost of survival increases with increasing mean maximum temperature, but, at any μ_{max}, cost decreases with increasing correlation between ΔT_{def} and T_{max} because individuals pay only approximately as much as they need to for induced defense.

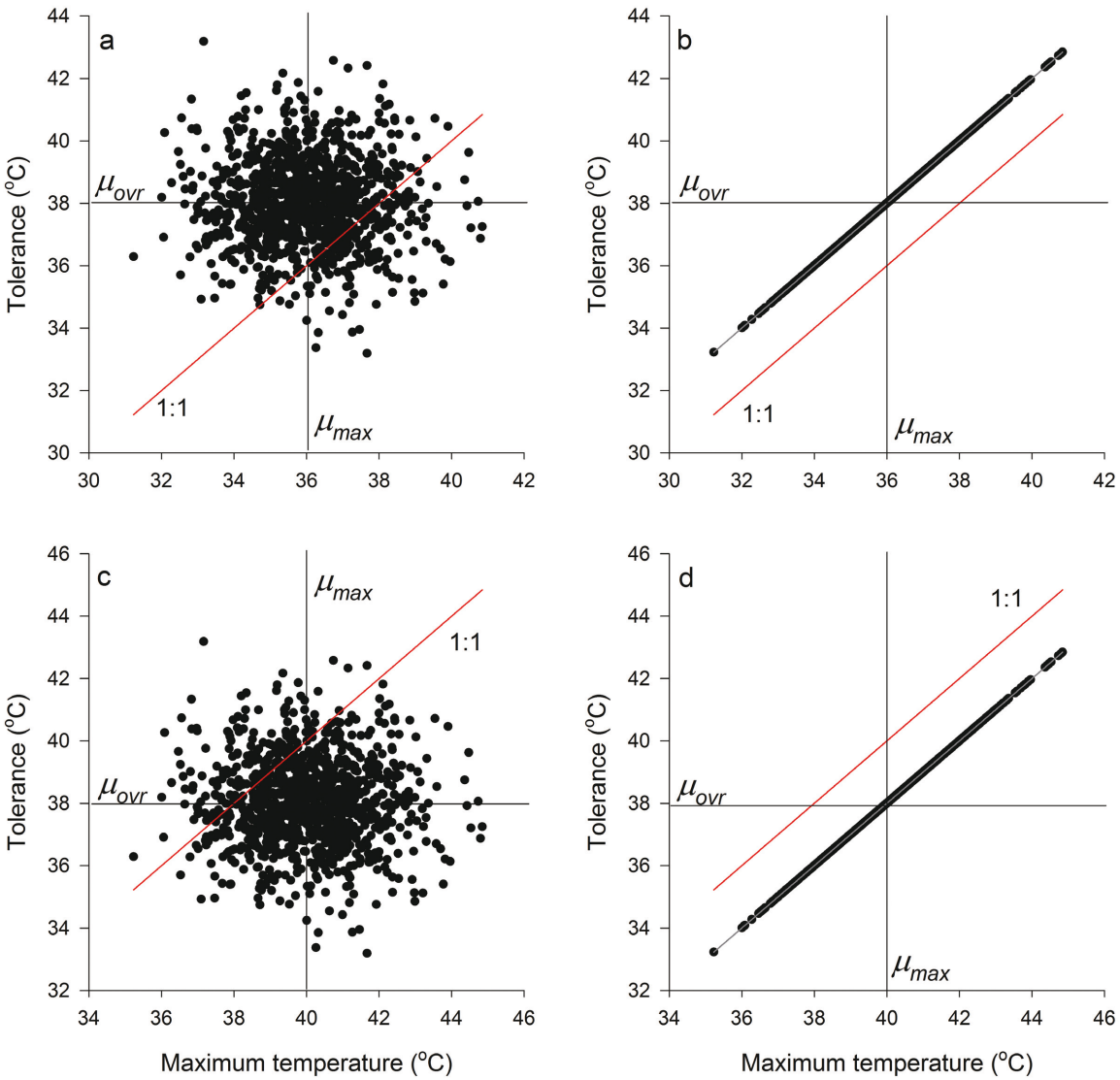

Figure 6 A heuristic example of how the correlation of induced defense with maximum temperature can affect survivorship. In panels (a) and (b), mean overall tolerance is greater than mean maximum temperature. (a) In the absence of correlation, some points fall below the 1:1 line, indicating that these individuals die. (b) When the same tolerance data are perfectly correlated with maximum temperature, all individuals survive. In panels (c) and (d), mean overall tolerance is less than mean maximum temperature. (c) In the absence of correlation, some points fall above the 1:1 line, indicating that these individuals survive. (b) When the same tolerance data are perfectly correlated with maximum temperature, all individuals die.

The ratio of cost to benefit, R, increases with increasing mean maximum temperature for all degrees of correlation (Fig. 5c). At low μ_{max}, R is highest for $r = 0$ and lowest for $r = 1$, although the difference in R between $r = 1$ and $r = 0$ is small. By contrast, at high μ_{max} ($>\mu_{ovr}$) R is highest for $r = 1$ and lowest for $r = 0$, and the disparity between correlated and uncorrelated induced defenses becomes large. In short, when a thermal event is relatively mild, correlated induced defenses are associated with a slightly advantageous cost–benefit ratio. However, when an event is exceptionally stressful, correlated induced defenses lead to a strongly disadvantageous R.

Note that a change in the arbitrarily chosen value of μ_{def} (3 °C) would shift the location of μ_{ovr} in Fig. 5, but would have no qualitative effect on the results.

In summary, a correlated induced defense is advantageous when the mean maximum temperature is less than the mean overall tolerance, but, surprisingly, in highly stressful thermal events a correlated induced defense decreases survival and increases the cost–benefit ratio of survivors, both of which are likely to be detrimental to the population.

Effects of variation in maximum temperature, constitutive tolerance and induced defense

Variation in maximum temperature

The effects of the magnitude of variation in maximum imposed body temperature (σ_{max}) are explained in Fig. 7. In Fig. 7a the difference in fraction of individuals surviving between perfectly correlated and uncorrelated induced response is shown as a function of the difference between mean maximum temperature and mean overall tolerance. When the environment is relatively benign, correlated induced defense is advantageous. When $\mu_{max} = \mu_{ovr}$, the difference in survival is zero independent of σ_{max}. By contrast, when mean maximum temperature is greater than μ_{ovr} (i.e. when $\mu_{max} - \mu_{ovr} > 0$), the effect of correlated induced defense is always negative, initially becoming increasingly negative with increasing σ_{max}, then reaching a nadir and subsequently increasing. In short, whether a correlated induced defense decreases or increases survivorship depends on whether mean maximum temperature is greater than or less than μ_{ovr} (respectively). The magnitude of the effect depends on the difference between μ_{max} and μ_{ovr}, but it is greatest when the inter-individual variation in maximum temperature (σ_{max}) is similar to the inter-individual variation in tolerance and induced defense, σ_{tol} and σ_{def} (1−2 °C in this representative case).

The difference in cost associated with correlated inducible defense is shown as a function of σ_{max} in Fig. 7b. When $\mu_{max} < \mu_{ovr}$ (i.e. when $\mu_{max} - \mu_{ovr} < 0$), an increase in σ_{max} results in a relatively small decrease in cost. By contrast, when $\mu_{max} \geq \mu_{ovr}$ (i.e. when $\mu_{max} - \mu_{ovr} > 0$), correlated induced defense initially results in an increased change in cost, but the change reaches a peak and then decreases, eventually becoming negative. The effects of correlated induced defense on cost are generally larger in magnitude when $\mu_{max} > \mu_{ovr}$ than when $\mu_{max} < \mu_{ovr}$. In short, as for survivorship, whether correlated induced defense increases or decreases cost depends on whether mean maximum temperature is greater or less than μ_{ovr} (respectively), and the negative effect of variation in maximum temperature (σ_{max}) increases with increasing variation.

The difference in cost–benefit ratio between correlated and uncorrelated defenses (Fig. 7c) is qualitatively inverse to that for survival; however, the disparity between benign and stressful environments ($\mu_{max} < \mu_{ovr}$ and $\mu_{max} > \mu_{ovr}$, respectively) is magnified: the increases in R associated with correlated induced defense at high mean

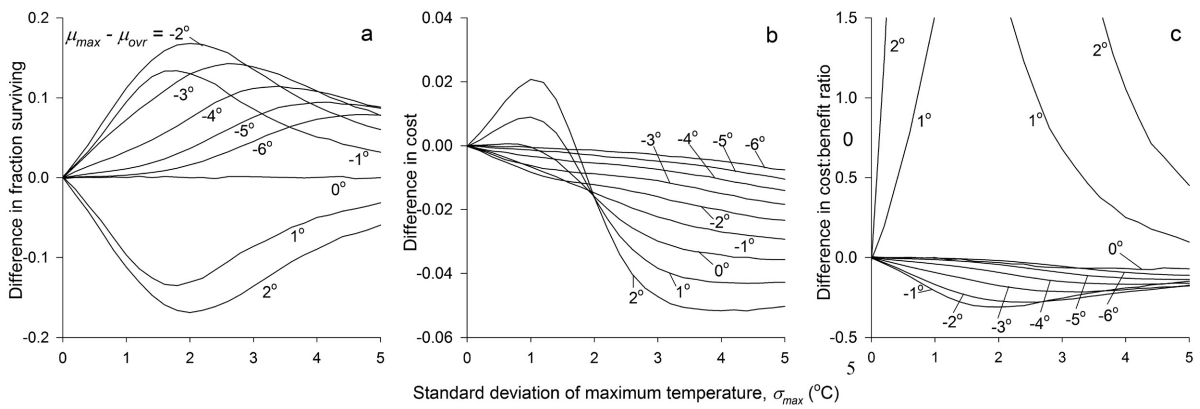

Figure 7 Predictions of the difference in survivorship, cost, and cost–benefit ratio as a function of the standard deviation of maximum temperature and the difference between mean maximum temperature and mean overall tolerance ($\mu_{max} - \mu_{ovr}$). (a) The difference in fraction of individuals in the population surviving thermal events between a correlated induced defense (r = 1) to an uncorrelated induced defense (r = 0). (b) The difference in cost between a correlated induced defense and an uncorrelated induced defense. (c) The difference in cost–benefit ratio between a correlated induced defense and an uncorrelated induced defense.

maximum temperature are vastly larger than the decreases at low μ_{max}. As for survivorship and cost, the effects of induced defense have their greatest magnitude for values of σ_{max} similar to σ_{tol} and σ_{def}.

In summary, the magnitude of the effects of correlated induced defense vary with the magnitude of variation in maximum temperature, with the sign of the effect typically depending on whether mean maximum temperature is greater or less than mean overall tolerance. In terms of survivorship and cost–benefit ratio, the effects of induced defense are greatest when σ_{max} is comparable to the reference σ_{tol} and σ_{def}.

Variation in tolerance

The effects of variation in constitutive tolerance (σ_{tol}) are shown in Fig. 8. The magnitude of the difference in survivorship associated with correlated induced defense generally decreases with an increase in σ_{tol}, and again the sign of the effect depends on whether μ_{max} is greater or less than μ_{ovr}; correlated induced defense is advantageous at low mean maximum temperature (i.e. $\mu_{max} - \mu_{ovr} < 0$) and detrimental at high mean maximum temperature ($\mu_{max} - \mu_{ovr} > 0$) (Fig. 8a).

Given the reference values for σ_{max}, μ_{tol}, μ_{def} and σ_{def}, correlated induced defense always decreases cost regardless of the magnitude of σ_{tol} (Fig. 8b). For relatively benign events ($\mu_{max} < \mu_{ovr}$), the magnitude of the reduction initially increases with an increase in σ_{tol}, and then decreases; for exceptionally stressful events ($\mu_{max} \geq \mu_{ovr}$), the magnitude decreases monotonically. The effect of correlated induced defense is much larger when $\mu_{max} \geq \mu_{ovr}$, but it is substantial only when σ_{tol} is small (<2 °C). Note that the difference in cost is not plotted for μ_{max} = 2 °C above μ_{ovr} and small σ_{tol}; in this case, no individuals among the 1000 sampled survived when r was equal to 1, so the change in cost could not be calculated.

The pattern shown by the cost–benefit ratio R (Fig. 8c) is approximately the inverse of the pattern of survivorship. Correlated induced defense decreases R relative to uncorrelated induced defense when $\mu_{max} < \mu_{ovr}$, and increases R when $\mu_{max} > \mu_{ovr}$. The effect is much greater when the environment is exceptionally stressful, but in all cases it is substantial only when the variation in constitutive tolerance is small.

In summary, the magnitude of the effects of correlated induced defense vary with the magnitude of variation in constitutive thermal tolerance, σ_{tol}, with the sign of the effect typically depending on whether mean maximum temperature is greater or less than mean overall tolerance. In contrast to the effects of σ_{max}, which are largest at intermediate value, the effects of σ_{tol} are greatest when σ_{tol} is small; that is, less than the reference σ_{max} and σ_{def}.

The suggestion that a correlated induced defense has its greatest impact when variation in constitutive defense is small may be relevant to the theory of evolutionary bet hedging. In a nutshell, bet-hedging theory proposes that, when exposed to an unpredictability vari-

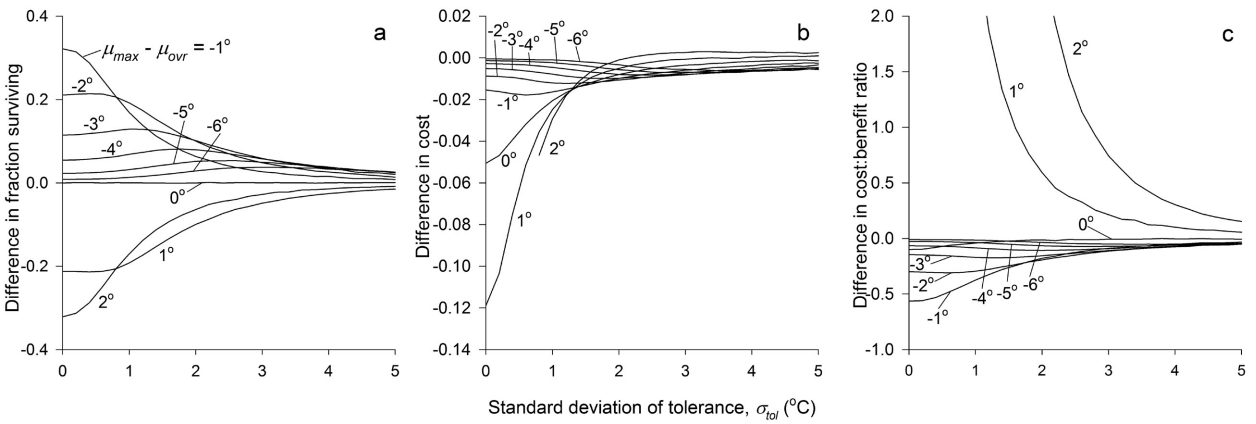

Figure 8 Predictions of the difference in survivorship, cost and cost–benefit ratio as a function of the standard deviation of constitutive tolerance and the difference between mean maximum temperature and mean overall tolerance ($\mu_{max} - \mu_{ovr}$). (a) The difference in fraction of individuals in the population surviving thermal events between a correlated induced defense (r = 1) and an uncorrelated induced defense (r = 0). (b) The difference in cost between a correlated induced defense and an uncorrelated induced defense. (c) The difference in cost–benefit ratio between a correlated induced defense and an uncorrelated induced defense.

able environment, a species gains a long-term selective advantage by producing a variety of phenotypes that, although they have a suboptimal fitness in any given environmental state, perform better when averaged across environmental states than do species with less phenotypic variability (e.g. Phillip & Seger 1989; Hopper 1999; Starfelt & Kokko 2012). Thus, in some unpredictable environments, there can be an evolutionary tendency to increase σ_{tol}. Because my model deals only with the consequences of a single extreme event, it cannot directly address bet-hedging strategies that play out over many random events. However, the results of my model suggest that in any single extreme thermal event, the increased constitutive breadth accruing from bet hedging may diminish the efficacy of a proportional induced response.

Variation in induced defense

The effects of the magnitude of variation in induced defense are shown in Fig. 9. For the difference in survivorship and cost–benefit ratio, the results are similar to those for variation in maximum imposed temperature (Fig. 7a,c). By contrast, the effects of variation in induced tolerance on cost are the inverse of those of the variation in maximum temperature. Cost is reduced by increased variation in σ_{def} when $\mu_{max} < \mu_{ovr}$. However, for $\mu_{max} > \mu_{ovr}$, cost is reduced at low σ_{def}, but then increases dramatically at high σ_{def}.

In summary, the magnitude of the effects of induced defense varies with the magnitude of σ_{def}, with the sign of the effect yet again depending on whether μ_{max} is greater or less than μ_{ovr}. In terms of survivorship and cost–benefit ratio, the effects of induced defense are again greatest when the variation in induced response is 1–2 °C, comparable to the reference variations in maximum temperature and constitutive tolerance, σ_{max} and σ_{tol}.

DISCUSSION

Three major conclusions emerge from these results: First, the relationship between mean overall tolerance and mean maximum temperature is critical. In relatively benign environments where mean tolerance is greater than mean maximum temperature, an induced defensive response is advantageous; however, in exceptionally stressful environments where mean tolerance is less than mean maximum temperature, an induced defensive response is disadvantageous. Second, substantial interaction among maximum temperature, constitutive tolerance and induced response is confined to cases in which the variation in the parameters is of roughly equal magnitude. Third, within this range, the effects of variation in constitutive tolerance and induced defense are complementary; when variation in constitutive tolerance is small, variation in induced defense has a large effect and vice versa.

Although the model proposed here explores the effects of a single extreme event, and, therefore, cannot

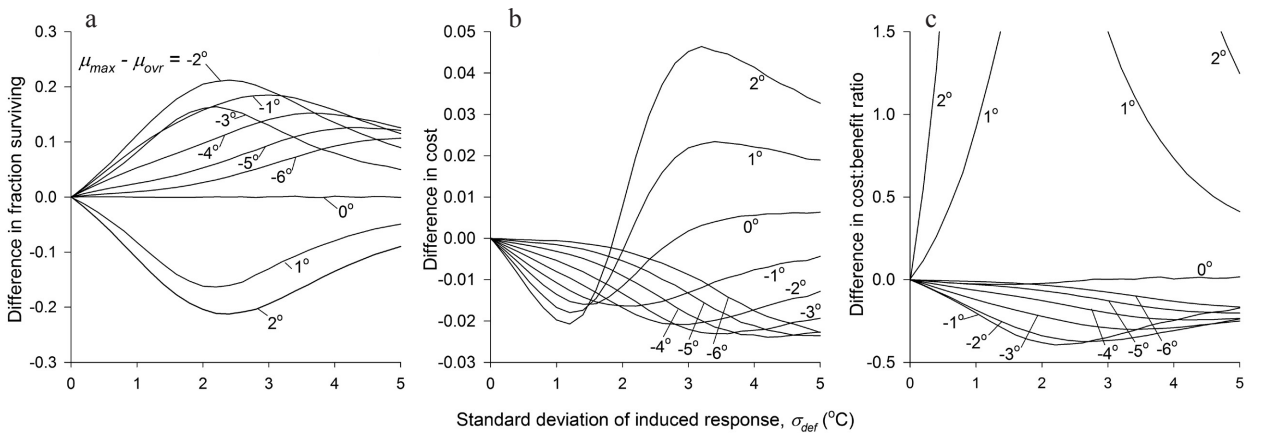

Figure 9 Predictions of the difference in survivorship, cost and cost–benefit ratio as a function the standard deviation of induced tolerance and the difference between mean maximum temperature and mean overall tolerance ($\mu_{max} - \mu_{ovr}$). (a). The difference in fraction of individuals in the population surviving thermal events between a correlated induced defense ($r = 1$) and an uncorrelated induced defense ($r = 0$). (b) The difference in cost between a correlated induced defense and an uncorrelated induced defense. (c) The difference in cost–benefit ratio between a correlated induced defense and an uncorrelated induced defense.

directly address issues of how organisms might evolve when subjected to a series of events, these conclusions have ties to some aspects of evolutionary theory. For example, theory developed by Lynch and Gabriel (1987) suggests that, when subjected to an unpredictable thermal environment, organisms will evolve a thermal tolerance that is greater than the maximum temperature they are likely to encounter in any given generation. This suggestion is supported by the model of Denny and Dowd (2012) in which hypothetical intertidal gastropods exposed to a random series of extreme thermal events evolved a mean thermal tolerance 7 °C above the mean maximum temperature imposed by extreme events; this calculated maximum tolerance is quite close to the empirically measured value. If these results can be generalized, they imply that, given time to evolve, species' mean tolerance will usually be greater than the mean maximum temperature, even in extreme events. This implies that the negative effects of induced thermal defense predicted by my model may seldom be realized in nature, although it remains to be seen whether the evolution required to appropriately adjust constitutive tolerance can keep pace with the current rate of climate change. In the few cases where mean extreme temperature might exceed species' mean tolerance, the counterintuitive results of the model presented here may help to explain why some organisms in stressful environments have evolutionarily opted for what presents as a costly escalated constitutive defense rather than a potentially more economical correlated induced thermal defense. For example, Dong *et al.* (2008) measured the production of heat-shock proteins in two limpet species found in the thermally stressful high intertidal zone. They found that the species that inhabited vertical walls (and was, therefore, less thermally stressed) induced a graded production of heat-shock proteins in response to thermal stress, whereas the other species, which was found on horizontal surfaces (and was, therefore, subjected to more frequent and more stressful high temperatures), had only an elevated constitutive defense.

Beyond these implications, productive ties between my model and evolutionary theory may have to wait until we have better information about the mechanics of induced thermal response. For example, some issues of the temporal pattern of thermal stress have been addressed by Shudo *et al.* (2003) in the context of heat-shock proteins in bacteria. They explored the factors that affect the optimum combination of constitutive (what they term "feedforward") tolerance and proportional induced ("feedback") thermal defenses when organisms are exposed to an unpredictable thermal environment, and find that an optimal strategy always includes some degree of constitutive tolerance, but the efficacy of an induced response depends on the relationship between the fixed temporal lag involved in mounting the induced defense and the temporal pattern of variation in temperature. However, for organisms more complicated than bacteria, temporal lag may not be a fixed factor; instead, it is likely itself to depend on past thermal history. Furthermore, the model developed by Shudo *et al.* (2003) takes into account only the duration and intensity of stressful thermal events, but for some organisms it is the number of events that most influences survival (e.g. Marshall & Sinclair 2015). Only when we better understand these and similar factors regarding the history-dependent mechanics of induced thermal defense will it become clear how to integrate the model proposed here into evolutionary theory.

Distinguishing between induced and constitutive defenses

Indeed, the complexities of whole-organism physiology can make it difficult even to distinguish induced from constitutive defenses unambiguously. The model presented here assumes that constitutive and induced thermal defenses are separate entities. However, practical separation of constitutive and induced tolerances is problematic because it depends on the temporal scale over which stress is applied and tolerance is measured (e.g. Levins 1968; Bowler 2005). Thermal tolerance is measured empirically by increasing an organism's body temperature and noting the temperature at which it dies or ceases to function effectively (e.g. Angilletta 2009; Somero *et al.* 2017). For instance, consider an experiment in which an animal's temperature is increased over the course of an hour, and the temperature at which it dies is noted. Is this temperature a measure of constitutive thermal tolerance or induced thermal defense? It depends on the time course of induction. If, for instance, it takes the animal a week to increase its level of heat-shock proteins or to reduce the fluidity of its membranes, its tolerance after 1 h of thermal stress is indicative primarily of its constitutive defense. By contrast, if heat-shock proteins and membrane fluidity can be adjusted in 10 min, tolerance after 1 h of exposure to stress represents T_{ovr}, a combination of both inducible and constitutive defense. The production of heat-shock proteins can often ramp up over the course of a few minutes (Feder & Hofmann 1999; Somero *et al.* 2017), but the temporal characteristics of even this relatively well-studied response can be complicated. Tomanek and

Somero (2000) found that the rate at which the heat-shock response was mounted in intertidal snails was inversely proportional to the intensity of thermal stress: the higher the temperature, the slower the response. In addition, measuring the production of heat-shock proteins provides only indirect (and potentially misleading) information about thermal tolerance (Morris *et al.* 2013). Furthermore, the heat-shock response is only one part of an overall induced response (e.g. heat hardening; Bowler 2005), and the time course of the whole system is less well known. For instance, gene expression patterns in *M. californianus* vary over the course of a few hours in the field, at least in part in apparent response to changes in temperature (Gracey *et al.* 2008), but it remains unclear how these expression patterns translate into changes in thermal tolerance. The problem is compounded even further by the likelihood that thermal tolerance varies through ontogeny (e.g. Brett 1970; Kingsolver *et al.* 2011). In short, much work remains to be done before we will have a sufficiently thorough understanding of the time course of induced thermal responses to allow a definitive differentiation between constitutive and induced defenses.

Behavior and mobility

In describing my model, I have presented the inter-individual variability in maximum temperature as something imposed solely by the environment. This is appropriate for sessile organisms such as plants and the mussels used as an example here, and the model is, therefore, applicable to many of Earth's inhabitants. However, for mobile organisms, variability in maximum body temperature can be affected by behavior. The ability to run and hide when times are tough can affect the mean and standard deviation of maximum body temperature (T_{max}), and may change the shape of the T_{max} distribution as well (e.g. Huey *et al.* 2009; Kearney *et al.* 2009; Hayford *et al.* 2015; Dong *et al.* 2017). Empirical measurements of the effects of behavior could easily be incorporated into the model. For instance, they could potentially result in distributions of ΔT_{def} and T_{max} that are non-Gaussian, and the effect of non-Gaussian (e.g. skewed) distributions on survivorship could be a productive avenue for future exploration.

Calibrating cost

The indices of cost and cost–benefit ratio used here assume that the cost of mounting a thermal defense rises in proportion to the imposed stress. This seems likely to be true in general, but in contrast to the strictly linear proportionality assumed here, it is probable that the actual proportionality is nonlinear. However, as long as cost is a monotonic increasing function of stress, nonlinearity should not affect the qualitative patterns shown

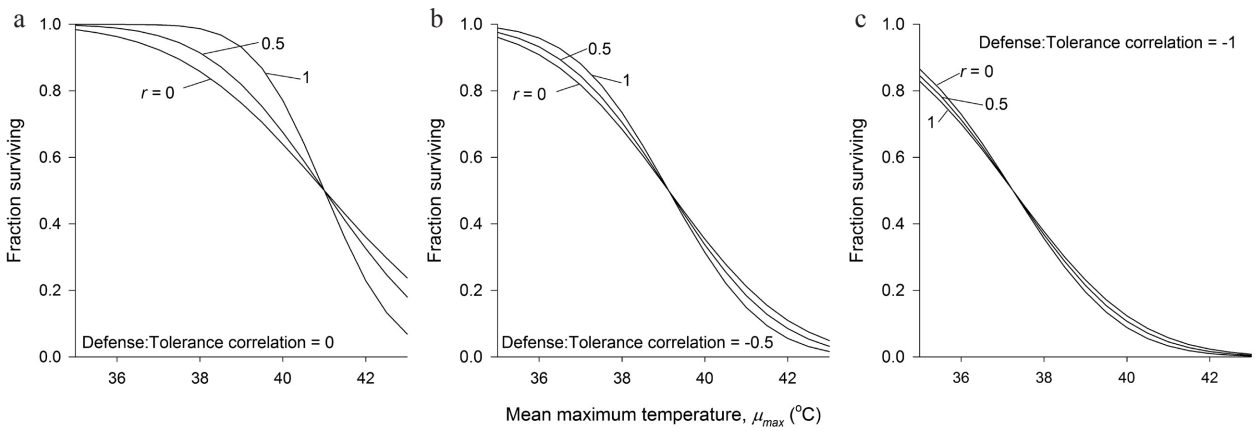

Figure 10 The effect of correlation between constitutive tolerance and induced tolerance as a function of mean maximum temperature and *r*. (a) When there is no correlation, the results are the same as those shown in Fig. 5a; an induced defense correlated with maximum temperature increases survivorship for low mean maximum temperatures but reduces survivorship for high mean maximum temperatures. (b) A moderate negative correlation (correlation coefficient = −0.5) between induced and constitutive tolerance has the same pattern as in (a), but reduces the fraction of individuals surviving. (c) Perfect negative correlation (correlation coefficient = −1) between induced and constitutive tolerance reverses the pattern seen in (a). Now an induced tolerance correlated with maximum temperature reduces survivorship at low mean maximum temperatures and enhances survivorship at high mean maximum temperatures, although overall a smaller fraction of individuals survive.

by the existing model; it will affect only the magnitude of the various effects. In any case, it will be interesting to calibrate the model through measurement of both the actual cost (in terms of growth or fitness) of maintaining a constitutive defense and that of mounting a correlated induced defense.

Independence of mean tolerance and mean inducible response

As shown in Fig. 5, mean overall tolerance, μ_{ovr}, represents a critical temperature separating the advantages of correlated and uncorrelated induced defense. Recall, however, that μ_{ovr} is the sum of mean constitutive tolerance (μ_{tol}) and mean inducible capacity (μ_{def}). If there is a negative correlation between μ_{tol} and μ_{def}, that is, if the most warm-adapted species (the species with the highest constitutive tolerance) has the lowest ability to plastically increase its tolerance, the sum of μ_{tol} and μ_{def} (i.e., μ_{ovr}) will vary less among species than one might expect. Any negative correlation between μ_{tol} and μ_{def} could, therefore, establish a relatively conserved threshold for the severity of extreme events that will favor uncorrelated induced defenses. Stillman (2002, 2003) and Stenseng *et al.* (2005) note that in intertidal crabs and snails there is, indeed, a negative correlation between μ_{tol} and μ_{def}, and the variation in overall tolerance among these species is substantially less than the variation among their mean constitutive tolerances. It remains to be seen whether this type of negative correlation is common among other species. If it is, it suggests that constitutive tolerance will be increasingly favored by evolution as the climate warms.

Independence of ΔT_{def} and T_{tol}

Similar considerations apply to the variation within rather than among individuals. In its current form, the model presented here assumes that the magnitude of induced defense exhibited by an individual is independent of its level of constitutive tolerance. Instead, it is conceivable that the extra effort an organism can contribute to induced defense depends on the effort it already exerts constitutively. If so, an individual with a higher than average T_{tol} might have a lower than average ΔT_{def}. Cavicchi *et al.* (1995) and Bettencourt (1999) have noted this kind of negative correlation in fruit flies. The effect of such a negative correlation between T_{tol} and ΔT_{def} would reduce the variance of T_{ovr}, and reduce (or even reverse) the correlation between ΔT_{def} and T_{max}.

We can explore these effects, again using *M. californianus* (Fig. 10). For low to moderate levels of negative correlation between ΔT_{def} and T_{tol} (Fig. 10a,b), the effect is to mute the consequences of induced defense: induced defense is still advantageous when mean maximum temperature is low and disadvantageous when μ_{max} is high, but the difference in survival between constitutive and induced defenses is reduced. In an extreme case, in which the negative correlation between ΔT_{def} and T_{tol} is perfect (Fig. 10c), induced defense has the opposite effect it would without this correlation: induced defense is disadvantageous when μ_{max} is low and advantageous when μ_{max} is high (Fig. 10c). In short, negative correlation between ΔT_{def} and T_{tol} could substantially modify the effects of individual variation in T_{max} and overall thermal tolerance. It remains to be seen whether there is a general negative correlation between ΔT_{def} and T_{tol} in plants and animals; in contrast to the findings of Cavicchi *et al.* (1995) and Bettencourt (1998) for fruit flies, Perieira *et al.* (2017) found that, for intertidal copepods, there was no correlation between the level of constitutive tolerance and the ability for a plastic increase in tolerance.

CONCLUSIONS

The model presented here provides a simple framework for exploring how small-scale variation in body temperature and individual variation in physiological defense interact to predict the consequences of extreme thermal events. When applied to a well-studied organism (the mussel *M. californianus*), the model highlights the fact that variation can be as important as the average when predicting the effects of increasing temperature, and reveals the counterintuitive likelihood that when thermal stress is extreme, a proportional induced defense can be disadvantageous. The model can be readily applied to other species given distributions for spatial variance in body temperature and individual variation in thermal physiology. Once these distributions are known, the model can be used to provide a better understanding of how individual-by-individual variation in maximum body temperature and thermal tolerance interact to potentially buffer (or amplify) the population-level effects of climate change.

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Appendix I

The distribution of tolerances among individuals, the probability density function, *Tol*, is:

$$Tol(T_{tol}) = \frac{1}{\sigma_{tol}\sqrt{2\pi}} exp\left[-\frac{(T_{tol}-\mu_{tol})^2}{2\sigma_{tol}^2}\right], \quad (7)$$

where T_{tol} is the highest body temperature that an individual can survive. Denny *et al.* (2011) assumed that T_{tol} is *constitutive*; that is, that the maximum temperature an individual can tolerate is a fixed value independent of the temperatures it experiences. Here, μ_{tol} is the mean constitutive tolerance (the average maximum temperature individuals can withstand), and σ_{tol} is the standard deviation of tolerances among individuals in a population. Each variable is measured in degrees Celcius. For the distribution of maximum temperatures imposed by the environment during a stressful thermal event, the probability density function, *Max*, is:

$$Max(T_{max}) = \frac{1}{\sigma_{max}\sqrt{2\pi}} exp\left[-\frac{(T_{max}-\mu_{max})^2}{2\sigma_{max}^2}\right], \quad (8)$$

where T_{max} is the maximum body temperature that an individual experiences, μ_{max} is the mean maximum temperature among individuals in the population and σ_{max} is the standard deviation of maximum temperatures among individuals. Again, each variable is measured in degrees Celcius.

Given these descriptions of individual capacity and of environmental stress, one can calculate the fraction S of individuals that will survive the thermal event:

$$S = \int_{-\infty}^{\infty} CMax(T_{max})Tol(T_{max})dT_{max}, \quad (9)$$

where *CMax* is the cumulative probability of *Max*. That is, *CMax* is the probability that a maximum temperature chosen at random from *Max* is less than or equal to T:

$$CMax(T) = \int_{-\infty}^{T} Max(T_{max})dT_{max}. \quad (10)$$

Appendix II

The probability density distribution of induced defense, ΔT, is

$$\Delta T(\Delta T_{def}) = \frac{1}{\sigma_{def}\sqrt{2\pi}} exp\left[-\frac{(\Delta T_{def}-\mu_{def})^2}{2\sigma_{def}^2}\right], \quad (11)$$

where ΔT_{def} is the magnitude of an individual's incremental defensive response (°C), μ_{def} is the mean induced response and σ_{def} is the standard deviation of induced response.

Appendix III

If $[x]$, $[y]$ and $[z]$ are independent sets of normally distributed random values, each with a mean of 0 and a standard deviation of 1, the set of maximum imposed temperatures used in the simulation is:

$$[T_{max}] = \sigma_{max}[x] + \mu_{max}, \quad (12)$$

the set of constitutive tolerances is

$$[T_{tol}] = \sigma_{tol}[y] + \mu_{tol} \quad (13)$$

and the set of defensive responses is

$$[\Delta T_{def}] = \sigma_{def}\left\{r[x] + \sqrt{(1-r^2)}[z]\right\} + \mu_{def}, \quad (14)$$

where r is a correlation coefficient that varies from 0 (no correlation) to 1 (perfect correlation).

Given these sets of data, the regression line of the (T_{max}, T_{ovr}) data passes through (μ_{max} μ_{ovr}), and b, the slope of the regression, is

$$b = \frac{r\sigma_{ovr}}{\sigma_{max}}. \quad (15)$$

This can be verified through reference to the standard derivations of the equations for statistical analysis of correlation and linear regression. Let

$$x = x_i - \bar{x}, \quad (16)$$

$$y = y_i - \bar{y}, \quad (17)$$

where x_i is an individual measurement of the independent variable (T_{max} in our case) and y_i is an individual measurement of the dependent variable (T_{ovr} in our case), and $\bar{x}$ and $\bar{y}$ are the means of x and y, respectively. Given this notation, the slope of the regression through a set of n data points is (Zar 1999):

$$b = \frac{\sum_{i=1}^{n} x_i y_i}{\sum_{i=1}^{n} x_i^2}. \quad (18)$$

The correlation coefficient for x and y is (Zar 1999):

$$r = \frac{\sum_{i=1}^{n} x_i y_i}{\sqrt{\sum_{i=1}^{n} x_i^2 \sum_{i=1}^{n} y_i^2}}. \quad (19)$$

From the definition of the variance (Zar, 1999), and given that $x_i = T_{max,i}$ and $y_i = T_{ovr,i}$:

$$\sigma_{max}^2 = \frac{1}{n}\sum_{i=1}^{n} x_i^2 \quad (20)$$

$$\sigma_{ovr}^2 = \frac{1}{n}\sum_{i=1}^{n} y_i^2. \quad (21)$$

Rearranging Equations (20) and (21), we find that:

$$n\sigma_{max}^2 = \sum_{i=1}^{n} x_i^2 \quad (22)$$

$$n\sigma_{ovr}^2 = \sum_{i=1}^{n} y_i^2. \quad (23)$$

Substituting Equations (22) and (23) into Equation (19):

$$r = \frac{\sum_{i=1}^{n} x_i y_i}{\sqrt{n^2 \sigma_{max}^2 \sigma_{ovr}^2}} = \frac{\sum_{i=1}^{n} x_i y_i}{n \sigma_{max} \sigma_{ovr}}. \quad (24)$$

Substituting this expression for r into Equation (15), and recalling Equations (22) leads to the conclusion that, according to my proposal,

$$b = \frac{(\sum_{i=1}^{n} x_i y_i)\sigma_{ovr}}{(n \sigma_{max} \sigma_{ovr})\sigma_{max}} = \frac{\sum_{i=1}^{n} x_i y_i}{n \sigma_{max}^2} = \frac{\sum_{i=1}^{n} x_i y_i}{\sum_{i=1}^{n} x_i^2}, \quad (25)$$

which, by comparison to Equation (18) demonstrates the validity of the proposal (Equation 15). QED.

Appendix IV

Organisms can be stressed by low as well as by high temperatures. The model can easily be modified to address the consequences of low-temperature stress. In this case, one deals with the distribution of minimum temperatures in a stressful event:

$$Min(T_{min}) = \frac{1}{\sigma_{min}\sqrt{2\pi}} exp\left[-\frac{(T_{min} - \mu_{min})^2}{2\sigma_{min}^2}\right], \quad (26)$$

where T_{min} is the minimum body temperature imposed on an individual, μ_{min} is the mean minimum temperature and σ_{min} is the standard deviation of minimum temperatures among individuals. The distribution of tolerances, *Tol*, is the same as before but now refers to an individual's ability to survive low temperatures. The fraction of individuals surviving a cold event is

$$S = 1 - \int_{-\infty}^{\infty} CMin(T) Tol(T) dT, \quad (27)$$

where *CMin*(*T*) is the cumulative probability of *Min*, the probability that a minimum temperature chosen at random from *Min* is less than *T*:

$$CMin(T) = \int_{-\infty}^{T} Min(T) dT. \quad (28)$$

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