INVITED REVIEW

Cold snaps, heatwaves, and arthropod growth

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Abstract. 1. Arthropod performance is a non-linear function of temperature, and thus global climate change may impact arthropods in a variety of non-obvious ways.

2. In this paper, the well-known thermal performance curve is reviewed briefly and attention is drawn to the importance of variance in temperature, particularly major weather events such as cold snaps and heatwaves.

3. A model is developed that considers the asymmetry between cold and heat stress and, particularly, the different timescales of recovery from these stressors: near-instantaneous for cold and lagged effects from heat.

4. Growth rate is evaluated as a function of weather-event intensity and length. Including the timescale asymmetry exacerbates both heat stress and, to a much lesser degree, cold stress.

Key words. Climate change, cold snap, heatwave, Jensen's inequality, poikilotherm, thermal performance.

Introduction

The world is, on average, clearly becoming warmer and more variable (Palmer, 2014). How such warming will impact individuals, populations and communities is not very clear, but for some groups of arthropods (e.g. bumblebees) extensive negative effects are already being documented (Kerr *et al.*, 2015). A wide range of recent studies, theoretical and empirical, on the effects of temperature on biological processes have sought to provide a unified understanding of the link between climate and biological function (e.g. Buckley *et al.*, 2010). In this paper, we focus on some of the non-obvious impacts of climate change and provide a new lens to examine these impacts.

Recent models of climate change predict more extreme weather events, including increased frequency of heatwaves, increased intensity and greater duration of such heatwaves (Meehl & Tebaldi, 2004), and more severe winter events (Palmer, 2014). In the following, we explain why these extreme events will be particularly important to arthropods and other poikilotherms and present new models to allow better understanding of the repercussions of extreme weather events.

Arthropods are predicted to be particularly vulnerable to exposure to detrimentally high and low temperatures. As a result of their size, most small organisms have low thermal inertia

and limited capacity to thermoregulate (Huey *et al.*, 1999). For much of what follows, we assume that body temperature closely matches ambient temperature. Although we focus on arthropods, our ideas should be applicable to a wide range of poikilotherms.

In this paper, we introduce a model that captures the asymmetric effects of hot and cold temperatures and then consider the implications for arthropod growth and fitness. We use a particularly simple model to illustrate how apparently elementary theory can provide subtle new insights. To begin, we briefly review the thermal performance curve (TPC) and the classic method for analysing the effects of fluctuating temperatures. We then describe a simple model that captures the effects of the asymmetries of hot and cold temperatures.

The thermal performance curve, fluctuating temperature, and Jensen's inequality

Temperature is a key component of performance for all living organisms, whether such performance is measured by development rate, foraging rate, immune function or respiration (Huey & Kingsolver, 1989). Thermal performance curves are concave, as shown in Fig. 1 (eqn 4). The standard approach for predicting performance from a curve such as that shown in Fig. 1 when the environment fluctuates is to use Jensen's inequality (e.g. Karban *et al.*, 1997; Mangel, 2006; Colinet *et al.*, 2015), which shows that average performance when temperature fluctuates differs from performance at the average temperature. For example, if
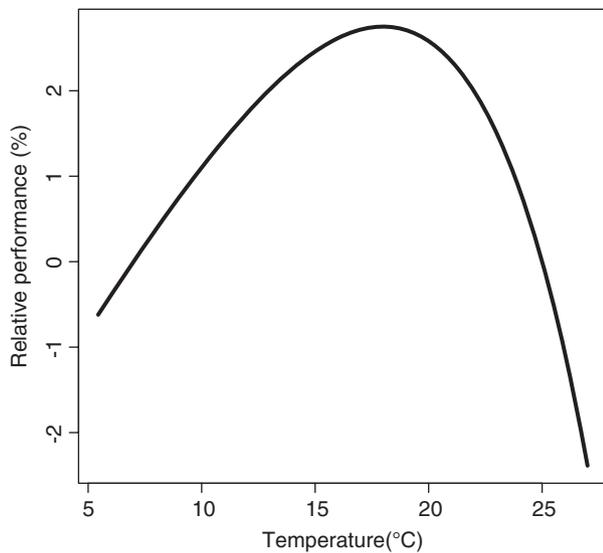
Fig. 1. A hypothetical performance-temperature profile, generated by the Ratkowsky curve (see below) for poikilotherms, assuming that performance is specific growth rate.

the organism experiences two temperatures, each for half of the time, we can draw a line segment between them and read off the average performance, thus estimating the loss in performance due to the fluctuations in temperature [see Estay *et al.* (2014) for a detailed analysis of the impact of temperature mean and variance on individual and higher-level processes].

These ideas were clearly articulated by Worner (1992), Ruel and Ayers (1999) and others, and were anticipated by Blackman (1905) more than a century ago. However, they are based on the assumption that the performance instantaneously adjusts when temperature changes. Indeed, most, if not all, models published to date are based on the assumption that recovery rates are instantaneous and identical, regardless of whether recovery is from a cold temperature or a warm temperature.

A single thermal performance curve but two mechanisms

We propose that the thermal performance curve is driven by two different mechanisms. The left-hand side of the curve is largely driven by enzyme kinetics. The conventional way to describe this is via an Arrhenius relationship (Hinshelwood, 1946; Brown *et al.*, 2004)

$$k(T) = Ae^{-E_a/RT} \tag{1}$$

where $k(T)$ is the rate of the process at temperature T, A is a constant, E_a is the activation energy and R is the universal gas constant. When $k(T)$ is plotted against T, we obtain an accelerating relationship, the left-hand side of the temperature-based performance curve (Fig. 1).

However, the increased performance predicted by eqn 2 does not continue indefinitely. As temperature continues to increase, there will be a point at which the slope abruptly changes direction, the so-called Arrhenius break temperature (ABT)

(Somero, 2002). In other words, at some point, performance begins to decline as a function of increasing temperature; this is the right-hand side of the curve in Fig. 1 but is not captured in eqn 2.

Why does performance decline even though reaction rates are expected to increase with temperature? At cold temperatures, eqn 2 predicts that life simply slows down. We hypothesise that physiological processes are disrupted at high temperatures in different ways from simply slowing down. These effects have been demonstrated at the levels of the organelle, organ and organism. For example, oxygen consumption by mitochondria is disrupted at high temperatures in a wide range of organisms (Pörtner, 2010). It should be noted that ABTs are often associated with an organism's common environment such that populations that typically live at warm temperatures have higher ABTs than those normally found in much cooler environments (Somero, 2002). Regardless of these variable set points, ABTs are commonly seen: there is an upper thermal limit. Indeed, researchers such as Deutsch *et al.* (2008) have attempted to capture this cold–hot asymmetry using a Gaussian curve and a parabolic function, respectively.

A variety of mechanisms underlie organ- and organism-level ABTs. For example, neuronal circuits tend to fail at high temperatures below those that cause cell death, due to loss of ion homeostasis (Robertson & Money, 2012). Clearly, failure in neural performance may lead to organism death even in the absence of neuronal death, due to inability to mitigate risk from the environment (e.g. predation, starvation). Similarly, antioxidant enzyme activity is greatly reduced at high temperatures, which can have serious long-term repercussions, especially given the fact that superoxide production increases at high temperatures (Mujahid *et al.*, 2007). A less explored mechanism is that insect immune function may be compromised at high temperatures (e.g. Karl *et al.*, 2011). Finally, costs of repair from heat damage can be very high, thus exacerbating an already difficult position for heat-stressed organisms (Kingsolver *et al.*, 2015; Kingsolver & Woods, 2016). Of course, at extremely low temperatures, qualitative effects are also seen (Chown & Nicolson, 2004); however, as arthropods typically live near their thermal optima (see Martin & Huey, 2008) and thermal performance profiles are asymmetric, deviation from mean temperature during the growing season more readily drives such individuals into their heat-damage zones than into their cold-damage zones.

When exposed to high temperatures, many, if not most, organisms will enter a heat coma because of the disruption of ion (K+) homeostasis. When an arthropod is exposed to very low temperatures, it will also enter into a low-activity state or even coma from which it may recover, although the mechanisms driving this phenomenon are not well understood (MacMillan & Sinclair, 2011). When the temperature returns to normal, individuals may recover and return to normal activities but this can take time. For example, there is evidence that there can be considerable lag between exposure to high temperatures and production of heat shock proteins (Bahrndorff *et al.*, 2009). Recovery times for ectotherms can vary from minutes for *Drosophila* recovery from heat-induced comas (Bozinovic *et al.*, 2011) to weeks for immunocompetence recovery in sticklebacks (Dittmar *et al.*, 2014). In addition, the effects of thermal stress

(hot and cold) can be seen well after exposure, e.g. days afterwards for sperm viability in bees (Stürrup *et al.*, 2013), months afterwards for swallowtail butterflies (Scriber *et al.*, 2012) and even across generations for flour beetles (Eggert *et al.*, 2015).

Data are beginning to accumulate which show that the rates of recovery from cold versus heat stress can differ widely (e.g. Diptera, Bozinovic *et al.*, 2011; Homoptera, Hazell *et al.*, 2008). Such differences in recovery rates have also been demonstrated in fungi; this delayed recovery has been called post-stress growth delay (Keyser *et al.*, 2014). In the very few controlled experiments published to date, including *Metarhizium* entomopathogenic fungi, there was an asymmetry between recovery from cold shock and heat shock with cold-based recovery occurring at a higher rate. While there are commonalities in response to heat versus cold stress [e.g. production of heat stress proteins (Udaka *et al.*, 2010); disruption of ion homeostasis (Findsen *et al.*, 2014)], they appear to operate independently (Hoffmann *et al.*, 2003; Udaka *et al.*, 2010).

Regardless of the mechanism, the examples above demonstrate that the impacts of cold and heat stress probably operate at different timescales. Even if recovery rates are not asymmetric, recall that TPCs are not symmetrical and arthropods typically live near their thermal optima (see Martin & Huey, 2008). Thus, deviation from mean temperature during the growing season more readily drives such individuals into their heat-damage than cold-damage zones (Vasseur *et al.*, 2015). Schulte *et al.* (2011) and Colinet *et al.* (2015) noted such different timescales, without providing tools to deal with them. Our goal is to provide simple theory that generates new questions and suggestions for new lines of empirical inquiry.

Approaches to timescale effects

There are at least two ways of modelling the impact of differential timescales on arthropod performance: (i) mechanistic, and (ii) heuristic. While developing our paper, we learned of a forthcoming paper by Kingsolver and Woods (2016) that employs a suite of ordinary differential equations to track temperature-dependent changes in mRNA, stress proteins, and ingestion rate. This elegant mechanistic model predicts changes in growth rate as a function of exposure to high temperatures within and across days. By contrast, but in the same spirit, our heuristic approach, outlined in the following, quantifies timescale effects *per se* on performance. This allows us to consider the many possible ways that temperature stress might impact performance and also allows for a simple link to extreme temperature events of different intensities and length. The purpose of this latter model is to generate discussion of this phenomenon.

A simple model of growth

Although arthropods develop through a series of instars that are constrained at the upper end by exoskeleton (Dyar, 1890), for the purposes of this paper, inclusion of instar structure adds unnecessary complication. Thus, we use a particularly simple model for growth in mass, similar to the popular Briere

development rate curve (Briere *et al.*, 1999), which is amenable to inclusion of a timescale asymmetry, the primary focus of our discussion. We use a single variable $W(t)$ to represent mass at time t and assume that when temperature is $T(t)$.

$$\frac{dW}{dt} = \Omega[T(t)]\,W(t)^{2/3} \qquad (2)$$

where $\Omega[T(t)]$ is the temperature-dependent growth rate, with units mass$^{1/3}$/time.

We use the Ratkowsky curve (Ratkowsky *et al.*, 1982, 1983, 2005; McMeekin *et al.*, 1993) to characterize temperature-dependent growth

$$\Omega[T(t)] = d\left(T - T_L\right)\left[1 - e^{g(T-T_U)}\right] \qquad (3)$$

This function has four parameters: a lower temperature T_L below which the animal loses mass, an upper temperature T_L above which the animal loses mass, a characterization d of growth at low temperature, and a parameter g that determines, along with the lower and upper temperatures of zero growth, the growth giving maximum temperature.

For the curve shown in Fig. 1, we used $T_L = 7.0$, $T_U = 25.0$, $g = 0.12$, $d = 0.0044$, which we also used for the rest of the computations.

The solution of eqn 2, with thermal performance described by eqn 3, is

$$W(t + 1) = \left\{W(t)^{1/3} + 3\Omega[T(t)]\right\}^3 \qquad (4)$$

Beyond Jensen's inequality: the time course of relaxation of growth rate

When temperature is suddenly shifted from cold to optimal or warm to optimal, it may be that the animal requires some time before it can achieve the optimal growth rate. However, using Jensen's inequality assumes that this relaxation is instantaneous.

To capture this idea, we now let growth rate have its own dynamics. In particular, we assume that if $\Omega(t)$ denotes the growth rate at time t after the temperature has been shifted back from a non-optimal temperature T to the optimal one T^*, then $\Omega(0) = d\left(T - T_L\right)\left[1 - e^{g(T-T_U)}\right]$ and for subsequent times

$$\Omega(t + 1) = \Omega(T^*)(1 - e^{-r}) + \Omega(t)\,e^{-r} \qquad (5)$$

where r is the relaxation rate, which we assume is bigger for the cold to optimal shift than the warm to optimal shift. (For computations we used $r = 0.25$ when returning to optimal from a colder temperature and $r = 0.025$ when returning to optimal from a warmer temperature.) Equation 5 is a discretised version of the linear relaxation equation $\frac{d\Omega}{dt} = r\left[\Omega(T^*) - \Omega\right]$. If r is very large, then $\Omega(1) \approx \Omega(T^*)$ and we return to the previous application of Jensen's inequality, assuming instantaneous recovery.

To predict growth, we now couple Eqns 2 and 5. We iterated deterministic runs of our model (in C++) in a factorial manner by varying the intensity of the temperature event period as well as its length. In each of the runs, individuals started at minimum mass and then grew according to daily temperatures, first

experiencing optimal temperatures (18 °C) for 15 consecutive days at which point a weather event ensued. At the completion of the weather event, temperature returned to optimal and remained there until day 60, at which point we terminated the run. We employed the same parameter values as earlier such that growth under optimal conditions generates individuals with maximum mass of 100 units. Finally, for comparison, we ran three different thermal response curves: (i) a quadratic (i.e. no asymmetry); (ii) a Ratkowsky curve with instantaneous response; and (iii) a Ratkowsky curve with relaxation response. We show only results using the Ratkowsky curve.

We show the results as heat maps in Fig. 2a (instantaneous Ratkowsky) and Fig. 2b (Ratkowsky with relaxation). As temperature moves away from optimal, performance declines. Note, however, that the effects are more exaggerated on the right-hand side of the graph where temperatures exceed optimum. When conditions are most difficult, i.e. hot, long heatwaves, there is a collapse towards minimum performance (shown in red); the sharp drop is denoted by the closely packed contour lines, especially when there is a timescale asymmetry (Fig. 2b). This tells us that the impact of heatwave intensity is contextual, i.e. it depends upon the length of the wave. The same occurs for cooler-than-optimal temperatures but with a gentler slope.

Some of these differences are simply due to the Jensen's inequality, but that is only part of the story. When we compared performance with the Ratkowsky versus the quadratic curve, we found a 5% loss in performance across the thermal landscape. Although individuals already perform poorly at high temperatures under the symmetric (quadratic) model, there is an additional impact from the asymmetric curve. Furthermore, the reduction in performance is much more pronounced when relaxation is not instantaneous (Fig. 2b) and this difference is not linear, i.e. this is not a simple effect of adding in the timescale. Comparing the two Ratkowsky models, we found a 90% congruence, i.e. 10% of the time, growth is worse when relaxation is required. Congruence was defined as the sum of the absolute difference for mass from the two models for each combination of event length and 0.5 °C above and below the optimal).

Discussion

Many populations of arthropods, and in particular tropical insects, live near their thermal optima and, as such, their thermal safety margin is small (recall the steep slope on the right-hand side of the thermal performance curve, Fig. 1). Thus, deviations from the mean during heatwaves and cold snaps associated with global warming suggest that the future for these organisms may be dire (Deutsch *et al.*, 2008; Vasseur *et al.*, 2015). To date, this discussion has largely dealt with impacts of temperature heterogeneity in space and/or time (Sears & Angilletta, 2015). Here, we add one more dimension to this discussion – the long-lasting effects of thermal stress, which may further challenge ectotherms in the future.

We introduced a simple model that shows how heat and cold stress may exacerbate the frequently discussed Jensen's inequality associated with thermal performance curves (e.g. Colinet *et al.*, 2015). The next step is to determine how well our model aids in predicting thermal performance across the life spans of ectotherms. Nearly all models to date assume an instantaneous response to temperature changes. Our model with relaxation of the thermal performance curve and subsequent sensitivity analysis points to important interactions between heatwave length and intensity that are not so obvious in the current models.

Several other points emerge from our approach

As noted in the introduction, current stress can lead to future impacts in performance. This would be the case, for example, when exposure to heatwaves leads to reduction in haemocytes and lysozyme activity in a tropical butterfly (Fischer *et al.*, 2014) and thus increases the chance of infection. Similarly, increased levels of superoxides in thermally stressed individuals will negatively impact their performance over time, often in a complex manner (Speakman *et al.*, 2015). In both of these cases, the impact of thermal stress will lag after the event has occurred and, as such, will require modifications to our current model. Although we did not employ lags in our earlier example, we can accommodate this in the response by replacing eqn 5 by the solution of:

$$\frac{\mathrm{d}\Omega(t)}{\mathrm{d}t} = r\left[\Omega(T^*) - \Omega(t-\tau)\right] \tag{6}$$

All else being equal, insects are predicted to suffer greater stress from two short heatwaves (or cold snaps) that are half the length of a single, long-lasting heatwave. This is because individuals in the former will be forced to recover twice and in the latter only once and recovery may not be complete at the time of the second heatwave or cold snap. Of course, 'all else' is rarely equal. Imagine an individual that is prevented from foraging for long periods of time due to excess heat or cold. An increased metabolic rate will exacerbate this asymmetry and cause the former individuals to suffer from energy reserve shortages more often than the latter. In other words, the full impact of extreme weather events will be state-dependent (Clark & Mangel, 2000). It is also important to note that impacts of long-term effects of exposure to high temperatures must be discounted by the probability that individuals will live long enough to experience such detriments, a concept that requires life-history models (Roff, 1992) or state-dependent life-history models (Clark & Mangel, 2000). The next iteration of our theory will include state variables such as energy (Colinet, 2011) and immune status.

We assumed no effect of duration or pattern of exposure for individuals growing in heatwaves and cold snaps, but that might not be the case (e.g. Kingsolver *et al.*, 2015; Kingsolver & Woods, 2016). As discussed earlier, this deficiency could be mitigated through the use of dynamic state variable models that include an exposure duration or pattern state as a kind of physiological memory (e.g. Mangel & Roitberg, 1989) or through the use of mechanistic models (e.g. Kingsolver & Woods, 2016) that track stress effects throughout a heatwave.

There is some evidence for very low genetic variance in heat resistance, supporting the notion of an upper limit that is
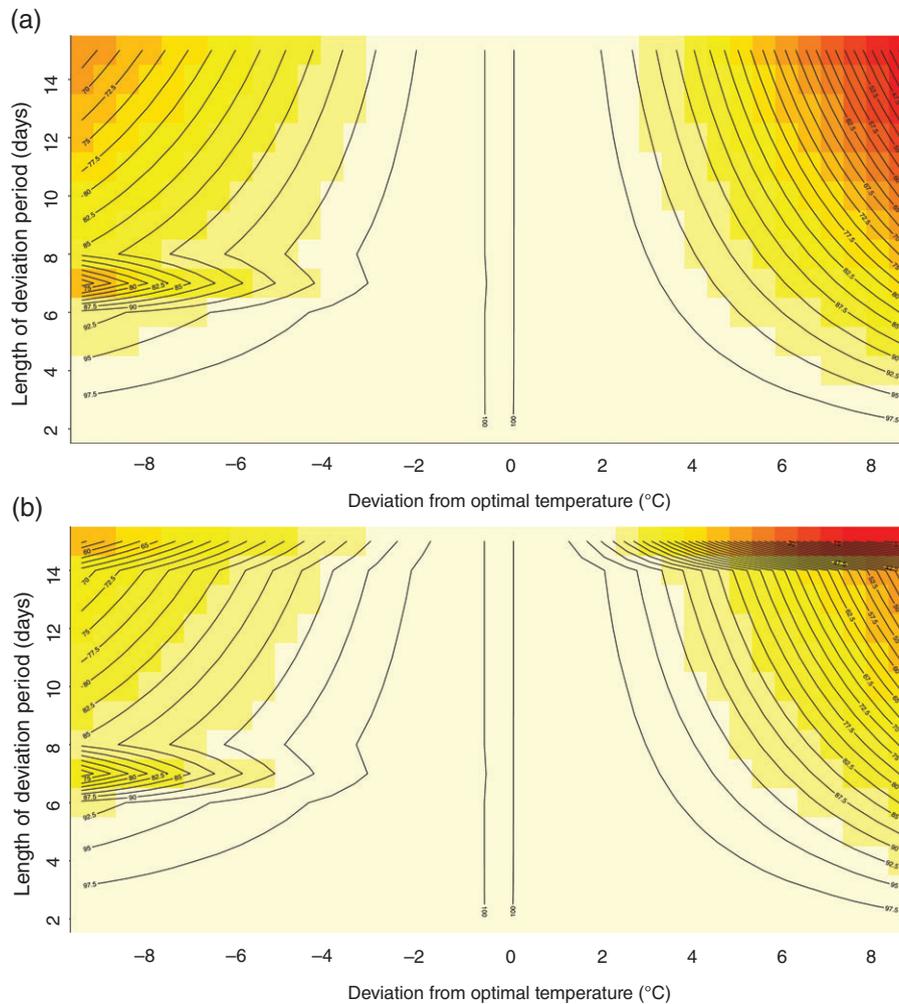
Fig. 2. (a, b) Impact of climate event intensity and duration on growth of hypothetical arthropods. The left-hand sides of the two plots indicate cold snaps and the right-hand sides indicate heatwaves. In (a), mass is shown for Ratkowsky growth performance when response to temperature is instantaneous and (b) shows the results when there is a lag in response upon return to optimal temperature. Final mass at the optimal temperature is set to 100 units and isoclines are set every 2.5 mass units.

qualitative in nature, i.e. simply increasing the concentration of enzymes will not solve the problem. For example, Schou *et al.* (2014) observed no effect of selection on *Drosophila* exposed to gradually increasing temperatures – this includes no change in genomic diversity between control and manipulated populations. The implication here is that it will be hard for populations to adapt to conditions when adaptation is based on a do-it-different versus do-it-better; other possible responses would be behavioural, i.e. migrate or change daily activity schedules. This will be particularly true if long-term effects of heat stress exacerbate the problem.

Upper thermal limits are less spatially variable than lower limits (e.g. Addo-Bediako *et al.*, 2000). Hoffmann *et al.* (2013) cite data showing that many terrestrial insects live under conditions that are close to their thermal maxima but might be constrained from evolving new thermal maxima as the world becomes hotter

and more variable. The situation may be even more constraining as this notion is based on the simple instantaneous response model.

As noted by a number of authors (e.g. Hoffmann *et al.*, 2013), many temperature–stress experiments are univariate and yet we know little about how insects react to temperature in the context of other environmental stressors. For example, Phelan and Roitberg (2013) showed that mosquito larvae respond to temperature in highly contextual ways with regard to the age and size at emergence. Similarly, few populations live in isolation from other species and thus long-term effects of heatwaves and cold snaps could profoundly affect community dynamics (e.g. Bannerman & Roitberg, 2014).

Although our model allows for variation in temperature, the variation is deterministic in that during the first 15 days the temperature is optimal for growth, during the cold snap or heatwave of variable length (2–15 days; Fig. 2) the temperature is different from optimal but constant, and during the remaining

time up to day 60, the temperature is once again optimal. In order to simplify the presentation (Fig. 2 is complicated enough), we ignored short-term fluctuations with a period of constant temperature. Jensen's inequality tells us that such shorter-term fluctuations will only make the situation worse (see Vasseur *et al.*, 2015 for such a case with instantaneous response to temperature). Thus, in some sense we have presented a 'best case' scenario.

It is true that we have added another complication to the already complex problem of climate change. But our model is general and heuristic. The new feature – relaxation of the instantaneous response assumption in already accepted models – requires empirical work to measure the relaxation rates from cold snaps and heatwaves to assess its importance; few hard data are currently available for this potentially important parameter. Controlled state-dependent experiments wherein recovery and latencies are elucidated for both cold and heat stress, in the same system, are needed. Currently, there is a tendency to focus on one extreme weather event or the other, but not both. There is much to be done.

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References

Addo-Bediako, A., Chown, S.L. & Gaston, K.J. (2000) Thermal tolerance, climatic variability and latitude. *Proceedings of the Royal Society of London Series B*, **267**, 739–745.

Bahrndorff, S., Marien, J., Loeschcke, V. & Ellers, J. (2009) Dynamics of heat induced thermal stress resistance and Hsp70 expression in the springtail, *Orchesella cincta*. *Functional Ecology*, **23**, 233–239.

Bannerman, J.A. & Roitberg, B.D. (2014) Impact of extreme and fluctuating temperatures on aphid–parasitoid dynamics. *Oikos*, **123**, 89–98.

Blackman, F.F. (1905) Optima and limiting factors. *Annals of Botany*, **19**, 281–295.

Bozinovic, F., Bastias, D.A., Boher, F., Clavijo-Baquet, S., Estay, S. & Angilletta, M.J. (2011) The mean and variance of environmental temperature interact to determine physiological tolerance and fitness. *Physiological and Biochemical Zoology*, **84**, 543–552.

Briere, J.F., Pracros, P., le Roux, A.V. & Pierre, J.S. (1999) A novel rate model of temperature-dependent development for arthropods. *Environmental Entomology*, **28**, 22–29.

Brown, J.H., Gillooly, J.F., Allen, A.P., Savage, V.M. & West, G.B. (2004) Toward a metabolic theory of ecology. *Ecology*, **85**, 1771–1789.

Buckley, L.B., Urban, M.C., Angilletta, M.J., Crozier, L.G., Rissler, L.J. & Sears, M.W. (2010) Can mechanism inform species' distribution models? *Ecology Letters*, **13**, 1041–1054.

Chown, S.L. & Nicolson, S.W. (2004) *Insect Physiological Ecology: Mechanisms and Patterns*. Oxford University Press, Oxford, U.K.

Clark, C.W. & Mangel, M. (2000) *Dynamic State Variable Models in Ecology. Methods and Applications*. Oxford University Press, Oxford, U.K.

Colinet, H. (2011) Disruption of ATP homeostasis during chronic cold stress and recovery in the chill susceptible beetle (*Alphitobius diaperinus*). *Comparative Biochemistry and Physiology A*, **160**, 63–67.

Colinet, H., Sinclair, B.J., Vernon, P. & Renault, D. (2015) Insects in fluctuating thermal environments. *Annual Review of Entomology*, **60**, 123–140.

Deutsch, C.A., Tewksbury, J.J., Huey, R.B., Sheldon, K.S., Ghalambor, C.K., Haak, D.C. *et al.* (2008) Impacts of climate warming on terrestrial ectotherms across latitude. *Proceedings of the National Academy of Sciences of the United States of America*, **105**, 6668–6672.

Dittmar, J., Janssen, H., Kuske, A., Kurtz, J. & Scharsack, J.P. (2014) Heat and immunity: an experimental heat wave alters immune functions in three-spined sticklebacks (*Gasterosteus aculeatus*). *Journal of Animal Ecology*, **83**, 744–757.

Dyar, H.G. (1890) The number of moults of lepidopterous larvae. *Psyche*, **5**, 420–422.

Eggert, H., Diddens-de Buhr, M.F. & Kurtz, J. (2015) A temperature shock can lead to trans-generational immune priming in the Red Flour Beetle, *Tribolium castaneum. Ecology and Evolution*, **5**, 1318–1326.

Estay, S.A., Mauricio Lima, M. & Bozinovic, F. (2014) The role of temperature variability on insect performance and population dynamics in a warming world. *Oikos*, **123**, 131–140.

Findsen, A., Pedersen, T.H., Petrsen, A.G., Nielsen, O.B. & Overgaard, J. (2014) Why do insects enter and recover from chill coma? Low temperature and high extracellular potassium compromise muscle function in *Locusta migratoria. The Journal of Experimental Biology*, **217**, 1297–1306.

Fischer, K., Michael Klockmann, M. & Reim, E. (2014) Strong negative effects of simulated heat waves in a tropical butterfly. *The Journal of Experimental Biology*, **217**, 2892–2898.

Hazell, S.P., Pederson, B.P., Worland, M.R., Blackburn, T.M. & Bale, J.S. (2008) A method for the rapid measurement of thermal tolerance traits in studies of small insects. *Physiological Entomology*, **33**, 389–394.

Hinshelwood, C.N. (1946) *The Chemical Kinetics of the Bacterial Cell*. Oxford University Press, Oxford, U.K.

Hoffmann, A.A., Sørensen, J.G. & Loeschcke, V. (2003) Adaptation of Drosophila to temperature extremes: bringing together quantitative and molecular approaches. *Journal of Thermal Biology*, **28**, 175–216.

Hoffmann, A.A., Chown, S.L. & Clusella-Trullas, S. (2013) Upper thermal limits in terrestrial ectotherms: how constrained are they? *Functional Ecology*, **27**, 934–949.

Huey, R.B. & Kingsolver, J. (1989) Evolution of thermal sensitivity on ectotherm performance. *Trends in Ecology and Evolution*, **4**, 131–135.

Huey, R.B., Berrigan, D., Gilchrist, G.W. & Herron, J.C. (1999) Testing the adaptive significance of acclimation: a strong inference approach. *American Zoologist*, **39**, 323–338.

Karban, R., Agrawal, A.A. & Mangel, M. (1997) The benefits of induced defenses against herbivores. *Ecology*, **78**, 1351–1355.

Karl, I., Stoks, R., De Block, M., Janowitz, S.A. & Fischer, K. (2011) Temperature extremes and butterfly fitness: conflicting evidence from life history and immune function. *Global Change Biology*, **17**, 676–687.

Kerr, J.T., Pindar, A., Galpern, P., Packer, L., Potts, S.G., Roberts, S.M. *et al.* (2015) Climate change on bumblebees converge across continents. *Science*, **349**, 177–180.

Keyser, C.A., Fernandes, E.K.K., Rangel, D.E. & Roberts, D.W. (2014) Heat-induced post-stress growth delay: a biological trait of many *Metarhizium* isolates reducing biocontrol efficacy? *Journal of Invertebrate Pathology*, **10**, 67–73.

Kingsolver, J. & Woods, A. (2016) Beyond thermal performance curves: modeling time-dependent effects of thermal stress on ectotherm growth rates. *The American Naturalist*, **187**, 283–294.

Kingsolver, J., Higgins, J. & Augustine, K. (2015) Fluctuating temperatures and ectotherm growth: distinguishingnon-linear and time-dependent effects. *The Journal of Experimental Biology*, **218**, 2218–2225.

MacMillan, H.A. & Sinclair, B.J. (2011) Mechanisms underlying insect chill-coma. *Journal of Insect Physiology*, **57**, 12–20.

Mangel, M. (2006) *The Theoretical Biologist's Toolbox. Quantitative Methods for Ecology and Evolutionary Biology*. Cambridge University Press, Cambridge, U.K.

Mangel, M. & Roitberg, B. (1989) Dynamic information and oviposition decisions in a fruit fly. *Ecological Entomology*, **14**, 181–189.

Martin, T. & Huey, R. (2008) Why "suboptimal' is optimal: jensen's inequality and ectotherm thermal preference. *American Naturalist*, **117**, E102–E118.

McMeekin, T.A., Olley, J.N., Ross, T. & Ratkowsky, D.A. (1993) *Predictive Microbiology. Theory and Application*. John Wiley and Sons, New York, New York.

Meehl, G.A. & Tebaldi, C. (2004) More intense, more frequent, and longer lasting heat waves in the 21st century. *Science*, **305**, 994–997.

Mujahid, A., Akiba, Y. & Toyomizu, M. (2007) A cute heat stress induces oxidative stress and decreases adaptation in young white leghorn cockerels by downregulation of avain uncoupling protein. *Poultry Science*, **86**, 364–371.

Palmer, T. (2014) Record-breaking winters and climate change. *Science*, **344**, 803–804.

Phelan, C. & Roitberg, B. (2013) An age-size reaction norm yields insight into environmental ineractions affecting life-history traits: a factorial study of larval development in the malaria mosquito *Anopheles gambiae sensu stricto*. *Ecology and Evolution*, **3**, 1837–1847.

Pörtner, H.O. (2010) Oxygen- and capacity-limitation of thermal tolerance: a matrix for integrating climate-related stressor effects in marine ecosystems. *Journal of Experimental Biology*, **213**, 881–893.

Ratkowsky, D.J., Olley, J., McMeekin, T.A. & Ball, A. (1982) Relationship between temperature and growth rate of bacterial cultures. *Journal of Bacteriology*, **149**, 1–5.

Ratkowsky, D.A., Lowry, R.K., McMeeking, T.A., Stokes, A.N. & Chandler, R.E. (1983) Model for bacterial culture growth rate throughout the entire biokinetic temperature range. *Journal of Bacteriology*, **154**, 1222–1226.

Ratkowsky, D.A., Olley, J. & Ross, T. (2005) Unifying temperature effects on the growth rate of bacteria and the stability of globular proteins. *Journal of Theoretical Biology*, **233**, 351–362.

Robertson, M.R. & Money, T.G. (2012) Temperature and neuronal circuit function: compensation, tuning and tolerance. *Current Opinion in Neurobiology*, **22**, 724–734.

Roff, D.A. (1992) *The Evolution of Life Histories: Theory and Analysis*. Chapman and Hall, New York, New York.

Ruel, J.J. & Ayers, M.P. (1999) Jensen's inequality predicts effects of environmental variation. *Trends in Ecology and Evolution*, **14**, 361–366.

Schou, M.F., Kristensen, T.N., Kellermann, V., Schlötterer, C. & Loescheke, V. (2014) A *Drosophila* laboratory evolution experiment points to low evolutionary potential under increased temperatures likely to be experienced in the future. *Journal of Evolutionary Biology*, **27**, 1859–1868.

Schulte, P.M., Healy, T.M. & Fangue, N.A. (2011) Thermal performance curves, phenotypic plasticity, and the time scales of temperature exposure. *Integrative and Comparative Biology*, **51**, 691–702.

Scriber, J.M., Emily Maher, E., Matthew, L. & Aardema, M.L. (2012) Differential effects of short term winter thermal stress on diapausing tiger swallowtail butterflies (Papilio spp.) *Insect Science*, **19**, 277–285.

Sears, M.W. & Angilletta, M.J. (2015) Costs and benefits of thermoregulation revisited: both the heterogeneity and spatial structure of temperature drive energetic costs. *American Naturalist*, **185**, E94–E102.

Somero, G.N. (2002) Thermal physiology and vertical zonation of intertidal animals: optima, limits and costs of living. *Integrative and Comparative Biology*, **42**, 780–789.

Speakman, J.R., Blount, J.D., Bronikowski, A.M., Buffenstein, R., Isaksson, C., Kirkwood, T.B. *et al.* (2015) Oxidative stress and life histories: unresolved issues and current needs. *Ecology and Evolution*, **5**, S745–S757.

Stürrup, M., Baer-Imhoof, B., Nash, D.R., Boomsma, J.J. & Baer, B. (2013) When every sperm counts: factors affecting male fertility in the honeybee *Apis mellifera*. *Behavioral Ecology*, **24**, 1192–1198.

Udaka, H., Chiaki, U. & Goto, S. (2010) Survival rate and expression of Heat-shock protein 70 and Frost genes after temperature stress in Drosophila melanogaster lines that are selected for recovery time from temperature coma. *Journal of Insect Physiology*, **120**, 67–73.

Vasseur, D.A., DeLong, J.P., Gilbert, B., Hamish, S., Greig, H.S., Harley, C.D.G. *et al.* (2015) Increased temperature variation posesa greater risk to species than climate warming. *Proceedings of the Royal Society of London Series B*, **281**, 20132612.

Worner, S.P. (1992) Performance of phenological models under variable temperature regimes: consequences of the Kaufmann or rate summation effect. *Environmental Entomology*, **21**, 689–699.