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**Title**

Forecasting extinction risk of ectotherms under climate warming: an evolutionary perspective

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**Summary**

1. It has been postulated that climate warming may pose the greatest threat species in the tropics, where ectotherms have evolved more thermal specialist physiologies. Although species could rapidly respond to environmental change through adaptation little is known about the potential for thermal adaptation, especially in tropical species.
2. In light of the limited empirical evidence available and predictions from mutation-selection theory we might expect tropical ectotherms to have limited genetic variance to enable adaptation. However, as a consequence of thermodynamic constraints we might expect this disadvantage to be at least partially offset by a fitness advantage i.e. the 'hotter-is-better' hypothesis.
3. Using an established quantitative genetics model and metabolic scaling relationships we integrate the consequences of the opposing forces of thermal specialisation and thermodynamic constraints on adaptive potential by evaluating extinction risk under climate warming. We conclude that the potential advantage of a higher maximal development rate can in theory more than offset the potential disadvantage of lower genetic variance associated with a thermal specialist strategy.
4. Quantitative estimates of extinction risk are fundamentally very sensitive to estimates of generation time and genetic variance. However, our qualitative conclusion that the *relative* risk of extinction is likely to be lower for tropical species than for temperate species is robust to assumptions regarding the effects of effective population size, mutation rate and birth rate per capita.
5. With a view to improving ecological forecasts we use this modelling framework to review the sensitivity of our predictions to the model's underpinning theoretical assumptions and the empirical basis of macroecological patterns that suggest thermal specialisation and fitness increase towards the tropics. We conclude by suggesting priority areas for further empirical research.

**Keywords:** adaptation, ecological forecasting, extinction, hotter is better, metabolic scaling, temperature, thermal specialisation.

## Vulnerability of tropical species to climate warming

A diverse range of ecological responses to recent climate warming have been widely documented (Walther et al. 2002; Root et al. 2003; Parmesan & Yohe 2003), notably at mid to high latitudes where the rate of warming has been fastest and the extent of monitoring has been the greatest (Parmesan 2007). Although increases in temperature are predicted to be greatest at higher latitudes (IPCC 2007) it has been suggested that climate warming may pose a greater threat to species living in the biodiversity-rich tropics. This is because tropical (ectotherm) species are likely to have evolved a thermal specialist physiology that lowers tolerance to temperature change, an adaptation to the more stable climatic conditions typically experienced at lower latitudes (Tewksbury, Huey & Deutsch 2008). By operating at body temperatures close to their critical thermal maximum thermal specialists are vulnerable to even modest increases in regional temperature whereas temperate species may benefit from climate warming since many typically experience environmental temperatures below their thermal optimum (Deutsch et al. 2008; Huey et al. 2009). Although organisms are likely to avoid the lethal effects of hot temperatures through avoidance strategies such as thermoregulatory behaviour and physiological acclimation (Angilletta 2009; Huey & Tewksbury 2009; Kearney, Shine & Porter 2009) costs are likely incurred by changes to foraging behaviour and/or reduced opportunities for reproduction (Huey, Losos & Mortiz 2010). If tropical species cannot migrate to cooler areas quickly enough populations will have to effectively track climate change through adaptation to maintain positive population growth rate and avoid extirpation (Hewitt & Nichols 2005; Visser 2008; Hoffmann & Sgró 2011).

Evolutionary responses to climate warming are already evident (see Parmesan 2006 for a review) but it is debateable whether the rate of adaptation will be sufficient to avoid extinction (Skelly et al. 2007; Huey et al. 2010), especially in the species-rich tropics where local extirpations are already apparent (Sinervo et al. 2010). Two important macroecological patterns are predicted to influence the relative evolutionary potential of tropical species. First, the increase in thermal

specialisation towards the tropics (Tewksbury et al. 2008; Sunday, Bates & Dulvy 2011) is predicted to reduce genetic variance via stronger stabilising selection (Huey & Kingsolver 1993; Bürger & Lynch 1995; Angilleta 2009). The resultantly low trait heritabilities will reduce the capacity of the mean phenotype of a population to closely track environmental change reducing fitness and increasing the likelihood of extinction (Huey & Kingsolver 1993; Lynch & Lande 1993). Second, due to what is believed to be a thermodynamic constraint, adaptation to high temperatures is associated with a higher maximum population growth rate ($r_{max}$) (Huey & Kingsolver 1989; Frazier, Huey & Berrigan 2006; Kingsolver & Huey 2008; Knies, Kingsolver & Burch 2009; Angilletta, Huey & Frazier 2010). A high $r_{max}$ is predicted to lower the risk of extinction by minimising the costs of selection (Lynch & Lande 1993) and demographic stochasticity (Gomulkiewicz & Holt 1995).

In this paper, we demonstrate the relative importance of these opposing forces on extinction risk using an established modelling framework (Lynch & Lande 1993, reviewed by Huey & Kingsolver 1993, Angilletta 2009, Kingsolver 2009 and Hoffmann & Sgró 2011) and metabolic scaling relationships (Brown et al. 2004). Our primary prediction is that tropical species are at no greater risk of extinction than temperate species, a conclusion robust to assumptions regarding population size, mutation rate and birth rate. The extent of this potential evolutionary advantage is greatest for species with life-histories that already maximise $r_{max}$ i.e. small and fecund organisms. In light of these theoretical findings we review and critically discuss the empirical basis for these macroecological patterns and the theoretical assumptions underpinning these predictions with the view to improving ecological forecasts.

## A theoretical framework for thermal adaptation

Consider the performance curve of an ectotherm adapted to local thermal conditions. Fitness can be approximated as a concave quadratic function of temperature, or a simple transformation thereof (Lynch & Gabriel 1987; Angilletta 2006), where the maximum defines the thermal optimum, $T_{opt}$ and the breadth defines the thermal tolerance of the organism (Huey & Kingsolver 1989). Thermal tolerance is defined at the lower limit by the critical minimum temperature, $CT_{min}$ and at the higher

limit by the critical maximum temperature, $CT_{max}$. In reality, the $T_{opt}$ is often higher than the mean environmental temperature, a strategy that can help to maximise geometric mean fitness under variable conditions (Martin & Huey 2008). Since here we only consider a change in mean temperature the effects of selection on the breadth of the thermal reaction norm can be ignored under all but the most abrupt of environmental changes (Lande 2009). For simplicity we also assume the organism is a thermoconformer i.e. body temperature is equal to ambient environmental temperature. Without loss of generality, body temperature can be used instead of environmental temperature to accommodate complexities of thermoregulation (Gilchrist 1995; Angilletta 2009).

Under climate change the more closely the mean $T_{opt}$ of a population can continue to *evolutionarily* track changes in mean environmental temperature the more likely it is that the population can maintain positive growth rate and avoid extirpation. A suite of models have been developed to quantify such phenotypic changes for a range of life-histories in order to predict persistence time under directional selection (Lynch, Gabriel, & Wood 1991; Lynch & Lande 1993; Bürger & Lynch 1995; see box 1). Factors that are expected to promote the rate of adaptation include high levels of genetic variance, a high population growth rate and a short generation time. Huey and Kingsolver (1993) applied this general framework specifically to thermal adaptation to yield further insights into the role of performance breadth and the consequences of specialist-generalist trade-offs (reviewed by Angilletta 2009 and Kingsolver 2009). One clear consequence of being a thermal specialist is that a narrow performance breadth subjects the ectotherm to a stronger intensity of selection for a given rate of temperature change. By implication tropical species, as thermal specialists, are predicted to elicit a greater response to climate warming. But because strong stabilising selection is also predicted to erode away genetic variance, the resultantly lower heritabilities may ultimately limit any response to selection. The scale of the predicted heritability advantage for thermal generalists increases asymptotically with population size due to an increase in mutational input (box 1; figure 1A). For effective population sizes exceeding ca. 500, this heritability advantage is sufficient to offset a lower selection intensity (figure 1B). Although tropical species have the potential to avoid or delay time to extinction under slow rates of warming (figure 2), temperate

species are predicted to have a lower risk of extinction overall (figure 3). Here we demonstrate how other factors hereto unconsidered, namely an increase in maximal fitness, might influence the extinction risk of tropical thermal specialist species.

If fitness is subject to a specialist-generalist trade-off (Huey & Kingsolver 1989) we would predict that tropical species as thermal specialists would have a lower risk of extinction all else being equal (Huey & Kingsolver 1993), whether this advantage is mediated by greater potential fecundity or shorter generation time (Kingsolver & Huey 2008). Evidence that trade-offs observable at the enzyme-kinetic level (Hochachka & Somero 2002) scale up to that of the whole organism remains equivocal (Gilchrist 1995; Angilletta et al. 2009); nonetheless, there is strong evidence that tropical species do possess a fitness advantage, if only by virtue of being warm-adapted (Savage et al. 2004; Angilletta et al. 2010). This thermodynamic constraint on maximum population growth rate (Savage et al. 2004), otherwise known as the 'hotter is better' hypothesis (Frazier et al. 2006; Kingsolver & Huey 2008; Knies et al. 2009; Angiletta et al. 2010), is attributed to the fundamental role of enthalpy in driving metabolic processes (Gillooly et al. 2001). Thermodynamic constraints also affect other biological rates potentially important in promoting adaptation e.g. mutation rate (Gillooly et al 2005; Gillooly, McCoy & Allen 2007). However, because all these temporal rates appear to share the same dependence upon temperature and body size (Brown et al. 2004) they are not expected to have an additive influence on evolutionary potential. Moreover, because these temporal rates share the same temperature dependence as development rate (Gillooly et al. 2002) and lifespan (Munch & Salinas 2009), the temperature and body size dependence of evolutionary potential can be modelled simply as a function of generation time.

Gillooly et al. (2002) present a universal model for development time based on the principles of metabolic theory, which they validate with data drawn on a range of taxa from insects to birds. Empirical relationships for mean embryonic and mean post-embryonic development times have been determined as: $\ln\left(t/{m_0}^{1/4}\right) = -0.12T + 6.0$ and $\ln\left(t/M^{1/4}\right) = -0.11T + 7.2$ respectively, where $t$ is development time in days, $T$ is body temperature in $^{o}$C and $m_0$ and $M$ are body mass in grams measured at hatching and at final asymptotic size. Here we use the sum of these relationships to

estimate generation time as a function of temperature and body size. Since the above empirical relationship for post-embryonic development time is only based on data for zooplankton we calculate generation time using the most conservative theoretical estimate of the intercept value for taxa in general (Gillooly et al. 2002), accordingly generation time $t = {m_0}^{1/4} e^{-0.12T+6.0} + M^{1/4} e^{-0.11T+7.32}$. We further assume that $m_0/M = 0.01$, which is equal to the upper limit observed among ectothermic taxa (reptilia: Hendriks & Mulder 2008).

Using this estimate of generation time the critical rate of change $k_c$ can be calibrated in respect to absolute time (i.e. $^{o}$C yr$^{-1}$) in order to directly compare the increased risk of extinction associated with lower additive genetic variance (owing to thermal specialisation in the tropics) against the lowered risk of extinction associated with a shorter generation time, as predicted by the 'hotter-is-better' hypothesis. Figure 4 illustrates how extinction risk is predicted to vary for three hypothetical scenarios of thermal adaptation, in which the thermal optima for an extreme thermal specialist and an extreme thermal generalist differ by an arbitrary 0, 10 or 20 $^{o}$C. This analysis suggests that tropical species can offset any predicted disadvantage of thermal specialisation providing their body temperatures are 10+ $^{o}$C higher on average than those of equivalent temperate species (Figure 4E). This is before differential rates of temperature change and seasonal constraints between the tropics and temperate areas are taken into account.

To further determine the role of generation time on extinction risk in different regions we ran simulations to determine the largest body mass likely to survive respective predicted rates of temperature change. We refer to this extinction risk measure as the 'critical body size' ($M_c$). If we can assume (as in figure 4B) that 1) the body temperatures of tropical species are 10+ $^{o}$C higher than equivalently sized temperate species and that 2) those temperate species are likely to experience double the rate of temperature change relative to tropical species i.e. up to 0.06 $^{o}$C yr$^{-1}$ vs. 0.03 $^{o}$C yr$^{-1}$ (reflecting emissions scenario A2; IPCC 2007), then we can confidently expect extinction risk to decrease towards the tropics. This is evident as an increase in the critical body size with increased thermal specialisation (from right to left in fig 4G-I).

- INSERT FIGURE 4 HERE

In all scenarios, those organisms with the highest potential fecundity (per capita birth rate) have the lowest risk of extinction but show the highest sensitivity to a change in thermal specialisation. While this qualitative result is robust, quantitative estimates of critical body mass itself are extremely sensitive to assumptions relating to mutational variance and the possibility of seasonal constraints on the number of generations that can be completed within a given year (figure 5). Specifically, a one order of magnitude reduction in mutational input has the effect of reducing the critical mass by four orders of magnitude, while restricting development to one quarter of the year i.e. a single season, has the effect of reducing the critical mass by 2.4 orders of magnitude. This sensitivity of critical body mass to underlying assumptions can be generalised as: $M_c \propto a{k_c}^{1/b}$, where *a* is a constant that has a proportionate scaling effect on the critical rate of change and *b* is the scaling exponent of development time on body mass (i.e. ¼). The critical body mass, therefore, responds as a power to the fourth function to any proportional change in the critical rate of change. Since this is a power law the combined impact of *n* factors can be derived simply as: $\log(M_c) = \sum_{i=1}^{n}(1/b)\log(a_i)$. For example, the combination of development constrained to a single season per year (0.25) and a one order of magnitude reduction (0.1) in genetic variance leads to a 6.4 magnitude reduction in the critical body mass (figure 5).

- INSERT FIGURE 5 HERE

In summary, the combination of shorter generation times and slower predicted rates of temperature change in the tropics should enable tropical species to overcome any *relative* evolutionary disadvantage associated with lower genetic variance arising from increased thermal specialisation. This qualitative theoretical result appears to be robust to the parameterisation of variables, although it must be stressed that quantitative predictions, particularly of critical body mass, are extremely sensitive to actual generation time (i.e. number of generations per year vs. age at maturity), mutational input and estimates of how much of this genetic variance is actually adaptive. It should be noted also that the use of a common intercept and slope value effectively models the

response of the 'average' species. It is well known that the intercept value of metabolic scaling relationships can differ substantially between taxonomic groups (Brown 2004); however, the results of recent empirical analyses also question the supposed universality of the predicted allometric and exponential exponents. The greatest deviations from Kleiber's ¾ power scaling law appear to occur at the deepest evolutionary transitions between the prokaryotes and eukaryotes, and between unicellular and multicellular organisms (DeLong et al. 2010), but they are also evident at lower taxonomic levels (e.g. Capellini, Venditti & Barton 2010). If the mass scaling relationships of metabolic rate map onto development time as predicted i.e. $I \propto M^b \Rightarrow t \propto M^{1-b}$, we would predict the allometric exponent of mammal orders alone to range in value between 0.18 and 0.41 (cf. theoretical value of $b = 0.25$). A similar range of exponent values is predicted among classes of terrestrial invertebrates that also show substantial variation in their exponential exponent ($0.38 < E < 0.80$ eV), which determines sensitivity to temperature (Ehnes, Rall & Brose 2011). Much of this variation is expected to reflect differences in body architecture (constraints), and ecological lifestyles and habitats (adaptations). Where such taxonomic data are available the resolution of ecological forecasts should be parameterised accordingly.

Our discussion hereafter will focus on the macroecological patterns and theoretical assumptions underpinning our projections of latitudinal extinction risk in an effort to identify areas where future empirical research can best contribute to improving evo-ecological forecasts.

## Macroecological patterns

*Are tropical species thermal specialists?*

Analyses of global datasets demonstrate that in general the thermal tolerance range of ectotherms increases with latitude, especially in the northern hemisphere (Addo-Bediako, Chown & Gaston 2000; Ghalambor et al. 2006; Sunday et al. 2011). This pattern is most pronounced for terrestrial organisms, which show a 50% increase in thermal breadth due higher tolerance to cold temperatures. Marine organisms, however, appear to show little change in thermal breadth due to concomitant reductions in both the upper and lower tolerance limits (Sunday et al. 2011). The close association between these critical/lethal limits and climatic variation in these respective habitats

suggests that the increase in thermal specialisation in terrestrial ectotherms towards the tropics is an adaptive response to temperature per se, an assertion supported by further analyses of thermotolerance replicated at smaller spatial scales, e.g. in respect to altitude (Brattstrom 1968). However, the increase in the strength of stabilising selection associated with this increase in thermal specialisation towards the tropics appears to be modest.

In a separate study conducted on a smaller dataset of 38 insect species (data collated by Frazier et al. 2006) other measures of thermal tolerance were found to more strongly decrease towards the tropics. Based on this latter analysis, the "warming tolerance" (defined as the difference between the $CT_{max}$ and mean environmental temperature of the habitat, $T_{hab}$) of tropical species is only one third that of temperate species; the respective figure for the "thermal safety margin" (TSM; defined as the difference between the $T_{opt}$ and $T_{hab}$) is just one-fifth (Deutsch et al. 2008; Fig. 2). A reanalysis of the supplementary data for this article reveals that these various potential surrogate measures of thermal specialisation are poorly correlated to thermal tolerance ($CT_{max}$- $CT_{min}$) (linear and non-linear regression, whether hemispheres pooled or analysed separately: all $P$ values > 0.20). Although there is a trend for increasing thermal tolerance with latitude in this dataset it amounts to less than 21% over the whole range (3.0 to 52.4$^{o}$; hemispheres pooled). Consequently, the decrease in both "warming tolerance" and "TSM" towards the tropics is almost entirely attributable to variation the environmental temperature (mean annual temperature) rather than to evolved differences in thermal physiology. However, this conclusion is based on critical thermal limits that were determined by the statistical fitting of a function rather than by direct measurement.

Although these various measures of thermotolerance are correlated to performance breadth, they do not provide adequate measures of the strength of stabilising selection. Ideally, we require estimates of the intrinsic rate of increase $r$ (under non-limiting conditions) for multiple temperatures. The strength of stabilising selection can then be estimated by fitting a Gaussian distribution of the form: $r = r_{max} \exp\left[-0.5\left(\left|T - T_{opt}\right|/\omega\right)^2\right]$, where $T$ is temperature, $\omega$ is performance breadth and $r_{max}$ is $r$ at $T_{opt}$ (Angilletta 2009). By reanalysing the data collated by Frazier et al. (2006) we estimate $\omega$ typically varies between ≈3 (extreme thermal specialist) and ≈10 (extreme thermal

generalist), respectively. Assuming $V_E \approx 1$ these values fall within the range typically used by theoretical models (Bürger & Krall 2004) although they might be considered relatively weak compared to the median value of stabilising selection strength reported from empirical studies more generally (Johnson & Barton 2005). Nevertheless, were thermal specialisation to increase by this magnitude from temperate to tropical regions all else being equal it could have a substantial impact upon genetic variance for $T_{opt}$ (fig 1A), and consequently extinction risk (fig 1B).

While the fitting of a Gaussian distribution to thermal performance curves provides a practicable means of parameterising existing theoretical models of adaptation and extinction risk (Angilletta 2009), the accuracy of estimates strongly depends upon the number and range of temperatures at which performance was measured (see Angilletta 2006; Knies & Kingsolver 2010). The mean number of temperatures at which $r$ was measured in the empirical studies collated by Frazier et al. (2006) is 5.2 (range: 4 to 8) while the range of temperatures in some cases was limited to those where $r$ was greater than 0.64 of that achieved at the $T_{opt}$ (e.g. *Cotesia flavipes*; Mbapila & Overholt 2001). The fitting of curves to restricted data sets such as these is expected to yield poor estimates of $\omega$, a limitation that would also apply to the most comprehensive dataset recently compiled by Dell et al. (2011) on 1,072 thermal responses for 309 species (Huey & Kingsolver 2011). In light of the constraints posed by these studies, the wide variation in thermal responses (Huey & Kingsolver 2011) and the inherent difficulties of characterising fitness landscapes using the ordinary least squares approach in general (Shaw & Geyer 2010; Kingsolver & Diamond 2011), predictions that use mutation-selection balance theory to estimate how genetic variance will vary with thermal specialisation are likely to remain effectively qualitative. A more practical approach will be to estimate genetic variance for relevant traits directly.

*Is heritability for $T_{opt}$ lower among tropical species?*

We know of no study that has specifically tested whether tropical species possess less genetic variance for $T_{opt}$ or other measures of heat tolerance. However, a survey conducted on *Drosophila* species distributed along a latitudinal gradient has revealed that genetic variance for two other

273 ecological traits, cold tolerance and desiccation resistance, is substantially lower among tropical
274 species (Kellermann et al. 2009). These tropical species were also found to have narrow geographical
275 distributions. If a restricted range size reflects environmental specialisation then strong stabilising
276 selection could provide an explanation for why genetic variance is lower in these species.
277 Unfortunately, auxiliary data to test this hypothesis are not available. However, alternative
278 explanations for the erosion of genetic variance, including the roles of directional selection,
279 phylogenetic constraints, inbreeding and drift, could all be ruled out in this study. The authors'
280 favoured explanation is DNA decay - the process by which gene function is degraded under relaxed
281 selection due to the accumulation of neutral and conditionally deleterious mutations (Whitlock 1996;
282 Lahti et al. 2009; Hoffmann 2010). DNA decay can eventually lead to the complete and irreversible
283 loss of complex traits (Ostrowski, Ofria & Lenski 2007; Collin & Miglietta 2008). If this explanation
284 is correct, it would imply that tropical species are derived, and that past populations must have been
285 small enough for long enough to allow mutations to reach fixation (Hoffmann 2010).
286 Kellermann's (2009) study suggests that similar surveys of heritabilities for $T_{opt}$ are urgently
287 needed, a nontrivial task given that the estimation of $T_{opt}$ itself requires the rearing of organisms at
288 multiple temperatures. Nevertheless, the alternative, i.e. to estimate heritabilities for related or
289 surrogate traits such as heat knock-down resistance, the critical thermal maximum ($CT_{max}$) or the $LT_{50}$
290 (the temperature at which 50% of individuals die), is not without difficulties. This is exemplified by
291 the dependency of $CT_{max}$, and the heritability of $CT_{max}$, on the experimental protocol used to measure
292 it. Both traits are found to be markedly lower if temperatures are elevated ("ramped up") slowly. As a
293 consequence it was postulated that species would have less capacity to adapt to high temperatures
294 experienced under 'natural' field conditions (Chown et al. 2009; Mitchell & Hoffmann 2010).
295 However, this curious result could arise as an artefact of the experimental design, since the
296 physiological condition of flies tested under the ramping up regime is predicted to deteriorate during
297 the course of a trial simply due to desiccation (Rezende, Tejedo & Santos 2011). If we exclude studies
298 based on ramping temperatures then estimates of heritabilities for upper thermal limits, including
299 $CT_{max}$ ($h^2$=0.12- 0.22 in *Drosophila*: Gilchrist & Huey 1999; Mitchell & Hoffmann 2010) and $LT_{50}$
300 ($h^2$=0.32 in fish; Meffe et al. 1995), appear to fall within the general range for physiological traits

301 (Mousseau & Roff 1987). How these estimates vary with latitude has not been systematically
302 investigated though it is interesting to note that in one study conducted by Mitchell and Hoffmann
303 (2010) on *Drosophila melanogaster*, a simple comparison between two populations revealed that,
304 contrary to our predictions, narrow-sense heritability ($h^2$±1 SE: 0.22±0.07 vs. 0.14±0.05) and additive
305 genetic variance ($h^2$±1 SE: 24.32±7.84 vs. 14.40±6.51) for heat knockdown time were markedly
306 higher for the tropical population.
307
308 *Do tropical species have higher fitness? Is hotter really better?*
309 Higher temperatures reflect greater enthalpy or kinetic energy available for work – this basic
310 thermodynamic principle underpins the 'hotter is better' hypothesis proposed to explain why the
311 maximum rate of population increase ($r_{max}$) increases with body temperature in a range of organisms,
312 from bacteriophages to mammals (Savage et al. 2004; Angilletta et al. 2010). According to the
313 metabolic theory of ecology (MTE), if $r_{max}$ is fundamentally constrained by metabolic rate then we
314 would predict $r_{max}$ to scale with the average activation energy of rate-limiting biochemical metabolic
315 reactions ($E \approx 0.6$ to 0.7) (Savage et al. 2004). On a plot of log-transformed $r_{max}$ against the inverse
316 of mean body temperature ($1/kT$; where $k$ is the Boltzmann's constant and $T$ is absolute temperature)
317 this value is represented as the negative slope of the linear relationship (Gillooly et al. 2001). The
318 common slope observed among taxonomic groups is attributed to a universal temperature-dependence
319 i.e. a constraint. Nonetheless, there is considerable variation in slope evident at the intra-and
320 interspecific level which suggests there is substantial genetic variance for this trait (Clarke 2004;
321 Clarke 2006; Gillooly et al. 2006; Terblanche, Janion & Chown 2007). Given the potential for local
322 adaptation, it remains unclear why lower optimal temperatures should be associated with a lower
323 maximal performance.
324 To evaluate the extent to which local adaptation or 'temperature compensation' (Clarke 2003)
325 has occurred Frazier et al. (2006) collated a detailed dataset on the temperature-dependence of
326 maximal $r_{max}$ in insect species living at a range of latitudes to test whether the slope of temperature-
327 dependence significantly differs to theoretical predictions. If perfect temperature compensation were

possible we would predict no resulting relationship between log maximal $r_{max}$ and the inverse of thermal optima ($1/kT_{opt}$) i.e. $E = 0$. In other words, all species should achieve the same level of maximal performance irrespective of the temperature they are locally adapted to. However, if maximal performance is ultimately constrained by metabolic rate, and the thermodynamic constraints it is subject to, we would predict maximal fitness to increase with the temperature of the thermal optima. Specifically, in MTE terms, maximal fitness should decrease with the inverse of the thermal optima ($1/kT_{opt}$) with a slope approximately equal to -0.65. Activation energies lying between these two extreme values would provide evidence of partial compensation. The results of Frazier et al.'s meta-analysis provided strong evidence for a thermodynamic constraint as maximal $r_{max}$ was found to decrease on average by between 8 and 12% for every 1 $^{o}$C drop in the optimal body temperature of taxa. Nonetheless, the level of temperature-dependence ($-E$ =0.97) was much greater than predicted.

If it can be assumed that maximal performance is subject to a specialist-generalist trade-off then this stronger than expected temperature-dependence could simply reflect the parallel trend for increased thermal specialisation towards the tropics. Using a structural equation modelling approach Frazier et al. (2006) attempted to control for thermal specialisation in their analysis indirectly via (a presumed adaptation to) seasonal variation. Although the addition of this surrogate measure failed to improve their model it is debatable whether the use of path analysis with its demanding statistical methods (Petraitis, Dunham and Niewiarowski 1996) was completely suitable for this dataset. Further comparative studies that aim to address the influence of taxonomic level (e.g. among vs. within species), life-history (e.g. nocturnal vs. diurnal) and environment (e.g. terrestrial vs. aquatic organisms) could help to further clarify the nature of the thermodynamic constraint by decoupling the relationship between thermal specialisation and maximal performance. However, only a systematic approach to the study of thermal reaction norm evolution is expected to establish the respective roles of phylogenetic constraints and adaptation to climate variability in shaping the temperature-dependence of fitness (Dell et al. 2011; Huey & Kingsolver 2011).

The results of experimental evolution experiments undertaken in different thermal environments have so far failed to support the basis of a thermodynamic constraint (Knies et al 2006;

355 reviewed in Angilletta et al. 2010). This might simply be due to a lack of statistical power or it may
356 reflect the lack of suitable control for thermal specialisation (Joel Kingsolver Pers. Comm.). In the
357 latter case, it is interesting to note that small-scale comparative studies of thermal reaction norms
358 suggest that thermal specialisation alone could underpin the 'hotter-is-better' response (Latimer,
359 Wilson & Chenoworth 2011). In the theoretical analysis presented here it is important to bear in mind
360 that we have considered temperature only as a proxy for predicting how (a constant) evolutionary
361 potential varies with latitude. In other words, we have not assumed a 'universal thermodynamic
362 constraint' (Clarke 2004; Clarke & Fraser 2004; Clarke 2006; Gillooly et al. 2006). To do so
363 otherwise would give rise to a positive feedback under climate warming between the evolution of a
364 higher thermal optimum and the maximal rate of adaptation (maximum development rate); thus in this
365 respect our analysis can be considered a conservative approach to forecasting extinction risk.
366

367 **Theoretical assumptions of quantitative genetic models**

368 *Do heritability estimates overestimate long-term evolutionary potential?*

369 Although mutational heritability provides a reliable measure of total genetic input, it is likely
370 to substantially overestimate the generation of potentially beneficial mutations required to maintain a
371 long-term adaptive response to environmental change (Lynch & Walsh 1998). This is because
372 standing genetic variance for fitness includes contributions from unconditionally deleterious
373 mutations. Lande (1995) estimates that up to 90% of new mutations could be deleterious and on this
374 basis recommended a revised-down figure of $V_m = 10^{-4}V_E$ when calculating the minimum effective
375 population size necessary for maintaining evolutionary potential (figure 5D-F). Recent molecular
376 analyses confirm that only around 10% of mutations are shown to be adaptive (Eyre-Walker &
377 Keightley 2007). Nonetheless, this figure is likely to exclude mutations that may influence fitness
378 through genotype-environment interactions and which may improve fitness in another environment.
379 For this and other reasons Franklin and Frankham (1998) disputed Lande's correction factor as being
380 too high. Accordingly, the range of mutational heritabilities we have evaluated here ($10^{-4} < V_m/$
381 $V_E < 10^{-3}$) is likely to represent the lower and upper limit. Note that even on the basis of this
382 restricted parameter range the predicted critical body mass, the largest ectotherm body size assured of

survival under predicted rates of climate change, can vary enormously e.g. by between 100 g and 1 metric tonne for an organism of intermediate thermal specialisation, a per capita birth rate of 10 and with an effective population size of 1000 (figure 5).

*How reliable are heritability estimates under environmental change?*

Estimates of heritability are by definition specific to the environment in which they were measured. Although estimates of heritability measured in the laboratory are not found to substantially differ on average to those measured in the field (Roff 2002), in general 'unfavourable' growth conditions tend to lower heritability estimates by inflating environmental variance while novel conditions tend to increase heritability estimates by revealing hidden additive genetic variance maintained by genotype-environment interactions (Charmantier & Garant 2005). It is unclear whether these factors are likely to systematically bias quantitative forecasts of extinction risk but one factor that could influence our qualitative conclusion regarding regional extinction risk is the tendency for additive genetic variance, and therefore heritability, to increase under stressfully high temperatures (Agrawal & Whitlock 2010).

Heat stress under climate warming will be felt most strongly by tropical species, which operate at body temperatures closer to their upper thermal limits (Deutech et al. 2008; Tewksbury et al. 2008). Whether this additional heat stress will enhance the adaptive potential of tropical species relative to that of temperate species will depend upon the underlying mechanism generating increases in genetic variance with temperature. For example, any increase due to higher rates of recombination and/or mutation (Hoffmann & Parsons 1997) is likely to consist largely of non-directed and generally deleterious genetic changes (Eyre-Walker & Keightley 2007). These are likely to impose a demographic cost on fitness that could potentially outweigh any advantage of producing rare beneficial alleles under directional selection. Under mild heat stress these deleterious effects may in part be offset by an elevated production of heat shock proteins responsible for molecular chaperoning (Casanueva, Burga & Lehner 2012), although under moderate to severe heat stress this reservoir of heat shock proteins may become depleted to reveal cryptic genetic variance (Rutherford 2003; Jarosz & Lindquist 2010). In the latter case, it has been argued, heat stress could promote the evolvability of

411 the species by exposing 'preadaptations' to selection (Eshel & Matessi 1998; Masel & Trotter 2010;
412 Rajakumar et al. 2012).

413 The relative roles that heat induced mutations and molecular chaperoning will have the risk of
414 extinction will depend upon the rate of temperature change for the organism. At slow rates of change
415 i.e. $k \leq k_c$, when the selection intensity is weak and heat stress is mild the demographic cost of
416 producing deleterious mutations is likely to be minimised and also partially offset by molecular
417 chaperoning. At fast rates of change i.e. those exceeding the critical rate $k_c$, when the selection
418 intensity is strong and heat stress is moderate to severe the likelihood of adaptive rescue under a finite
419 temperature change will be primarily dependent upon existing and cryptic genetic variance rather than
420 novel mutations. Accordingly, heat stress could in theory favour the adaptive potential of tropical
421 species irrespective of the rate of change, strengthening our qualitative conclusion that extinction risk
422 will be lower towards the tropics. Nonetheless, whether this increase in additive genetic variance
423 revealed at high temperatures will contribute to an adaptive response remains to be tested.

424

425 *Can a low heritability pose an absolute genetic constraint?*

426 Low heritability imposes a quantitative genetic constraint on trait responses to selection. In
427 cases where demographic stochasticity is high, such as in small populations, it can in effect pose an
428 absolute constraint to adaptation (Gomulkiewicz & Houle 2009). Though there is little utility in
429 forecasting the evolutionary trajectory of small populations, there are other circumstances in which a
430 low heritability could impose an absolute constraint irrespective of population size. It has been shown,
431 for example, that in some populations of *Drosophila* low heritability for ecological traits such as
432 desiccation resistance is associated with a lack of response to artificial selection (Hoffmann et al.
433 2003). If, as has been proposed (Hoffmann 2010), this lack of genetic variance is a result of DNA
434 decay it will be difficult, if not impossible (e.g. Wiens 2011), for new beneficial mutations to arise.
435 The finding that heritabilities for both desiccation and cold resistance are very low in tropical vs.
436 temperate populations that otherwise show an abundance of neutral genetic variance (Kellermann et
437 al. 2009) is therefore a cause for concern. Nonetheless, it is also the case that low heritability
438 estimates can often underestimate potential for adaptation. For instance, it appears that the grayling, a

freshwater fish, has undergone rapid physiological adaptation to differences in stream temperature between closely situated sites despite showing low levels of genetic variance for thermal tolerance and a lack of gene-flow among populations (Kavanagh et al. 2010; Skelly 2010). Ultimately, the only way to determine whether genetic limits really do exist for particular traits or trait combinations is to conduct resource-intensive selection experiments on the trait in question (Hoffmann et al. 2003).

The primary method to infer the potential role of genetic constraints is to evaluate phylogenetic inertia among closely related species (Huey & Bennett 1987; Somero 2011). A prerequisite for this comparative approach is the ability to disentangle the influence of a common evolutionary history from a common selective environment (Losos 2011), often a difficult task in the case of thermal adaptation (Angiletta 2009). Consider, for example, the apparent lack of variability in the critical thermal maximum among reptiles (Huey & Bennett 1987). A lack of genetic variation in this trait among terrestrial species has been interpreted as evidence of a genetic constraint (Huey & Kingsolver 1989); alternatively it might simply reflect adaptation to a weak latitudinal trend in maximum temperatures (Addo-Bediako et al. 2000).

The situation is more complicated if one considers the thermal optimum. A substantial mismatch between the optimal or preferred body temperature and the mean environmental temperature might appear mal-adaptive at first sight but it can serve to maximise geometric mean fitness in a variable environment (Gilchrist 1995; Martin & Huey 2008). This is true whether we consider the asymmetry of the thermal reaction norm to be a constraint imposed at the enzyme kinetic level (Knies & Kingsolver 2010) or an adaptation to thermodynamic constraints imposed upon maximal performance i.e. 'hotter is better' (Asbury & Angilletta 2010). These alternative scenarios serve to underscore our poor understanding of thermodynamic constraints and the resulting difficulty to quantitatively define a null model for thermal adaptation.

*Genetic covariance*

The rate at which a trait can respond to selection is also influenced by genetic correlations between multiple traits (Walsh & Blows 2009). A negative genetic covariance is indicative of antagonistic pleiotropy or trade-offs, which can severely constrain the rate of adaptation (Etterson &

467 Shaw 2001). Genetic variance-covariance matrices (G) can be used to quantify the strength and
468 direction of these correlations to more accurately predict the fate of individual populations (Blows &
469 Hoffmann 2005). But since positive genetic correlations, which can otherwise promote a response to
470 selection (Agrawal & Stinchcombe 2009), appear to be as common as negative correlations,
471 predictions of mean extinction risk at the macroecological level are not expected to be systematically
472 biased. Moreover, although G-matrixes can be used to predict short-term responses to environmental
473 change, they are themselves moulded by selection. Theoretical simulations suggest that the
474 evolutionary dynamics of G is highly dependent on its dimensionality and its alignment with the
475 selection gradient but that directional selection may contribute to its stability (Jones, Arnold and
476 Bürger 2004). On the other hand the breaking down of genetic constraints can occur extremely
477 quickly. For instance, in a recent study on the annual plant *Brassica rapa* five generations were
478 sufficient to overcome the constraints predicted by genetic correlations and enable the rapid adaptive
479 evolution of multiple traits in response to drought selection (Franks & Weis 2008).
480
481 *Mutations of large effect*
482 Mutational effects for individual traits are often leptokurtic, not normally distributed as often
483 assumed by theory (Eyre-Walker & Keightley 2007). Although such traits are still influenced by
484 many genes, responses to selection are dominated by a very few genes of large effect, which tends to
485 increase genetic stochasticity and lower mean time to extinction (Bürger & Lynch 1995). This is
486 because the disproportionate influence of a limited number of genes necessarily limits the number of
487 evolutionary trajectories towards a new adaptive peak evolution (Kopp & Hermisson 2007). The
488 potential cost to population fitness in producing less than optimal phenotypes is expected to increase
489 with the strength of selection (e.g. abrupt climate warming) and the rarity and size of the mutational
490 effect (Collins, de Meaux & Acquisti 2007; Collins & de Meaux 2009). While estimates of genetic
491 variance can be adjusted where distributions deviate from normality, such corrections tend to be
492 unnecessary for complex traits such as fitness where the influence of individual loci are ameliorated
493 by the averaging effects of multiple additive loci (Lynch & Lande 1993). This apparent robustness of
494 predicted changes in fitness and population persistence under directional selection to the uncertainties

associated with genetic architecture further supports the general use of quantitative genetic models in global change biology.

*Summary*

Quantitative genetics continues to provide researchers with a practical and general tool for modelling phenotypic evolution under directional selection (see Stockwell, Hendry & Kinnison 2003; Skelly et al. 2007; Visscher, Hill & Wray 2008; Hoffmann & Sgró 2011 for recent reviews and commentaries), especially thermal adaptation under climate change (Huey & Kingsolver 1993; Angilletta 2009; Kingsolver 2009; Chevin, Lande & Mace 2010). Here we used the modelling framework developed by Lynch and Lande (1993) and Bürger et al. (1995) to consider the potential role of adaptation and a temperature-dependent rate of evolution on the extinction risk of ectotherms with latitude. Contrary to previous ecological forecasts based solely upon macroecological patterns in ecophysiology i.e. thermal specialisation (Deutsch et al. 2008), we found that tropical species should be as, if not more, resilient to climate change than temperate species.

The principle evolutionary advantage tropical species are predicted to possess is a shorter development time at their respective thermal optima. We showed here that the combination of a shorter generation time and a lower predicted rate of regional temperature change can more than offset the disadvantage of low genetic variance associated with thermal specialisation. This qualitative theoretical prediction is robust to assumptions regarding population size, birth rate and mutation rate. Moreover, this prediction can be considered conservative for a number of reasons. Firstly, we assumed an extreme trend in thermal specialisation, and therefore genetic variance, with latitude although it remains to be established whether tropical species do have lower genetic variance for heat tolerance. Secondly, since we neither assumed a 'universal thermodynamic constraint' (and therefore a feedback between higher thermal optima and adaptive potential) nor a generalist-specialist trade-off in performance we have in effect restricted our analysis to the impacts of an existing trend in fitness with latitude. Nonetheless, if it the case that tropical species have low genetic variance and that this level poses an absolute rather than merely a quantitative genetic constraint to adaptation our analysis will inevitably prove to be overly optimistic.

*Improving ecological forecasts*

There appear to be three key areas where further empirical research could greatly improve the accuracy and therefore value of evolutionary forecasts not just to climate warming but to environmental change in general. Firstly, and arguably most importantly, it remains to be established whether tropical species possess lower heritabilities in general for ecological traits as a result of ecological specialisation and whether this limited adaptive potential represents an absolute vs. merely a quantitative genetic constraint. Few large-scale studies have been undertaken to investigate such patterns but since these questions are intrinsically linked to the wider question of what determines the evolution of range size further insights could be drawn from studies conducted in respect to other traits, organisms and other geographical regions. Secondly, although a number of mechanisms are known to increase genetic variance under heat stress to what extent this release of novel genotypes can contribute to the rate of adaptation remains to be tested. Interestingly, this potential to accelerate adaptation could apply to any new environments as a general consequence of low genetic quality manifested as poor phenotypic condition (Sharp & Agrawal 2012). Thirdly, if the 'hotter-is-better' response is caused by a thermodynamic constraint then we would predict potential rates of adaptation to accelerate under climate warming, and decelerate under climate cooling. Manipulative experiments are therefore necessary to test whether thermodynamic constraints shape the evolution of thermal reaction norms as predicted (Angilletta 2009). Although we have only explicitly considered the potential for the 'hotter-is-better' response to contribute to the thermal adaptation of tropical species under climate warming, the benefits of a faster generation time apply to any trait under selection.

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806

807 **Box 1 - Maintenance of genetic variance under stabilising selection**

808 The amount of genetic variance, $V_G$, that can be maintained within a finite population is dependent
809 upon a balance between mutation, selection and drift processes. At equilibrium, this can be modelled
810 using the stochastic house-of-cards approximation (SHC) (Bürger, Wagner & Stettinger 1989; Bürger
811 & Lynch 1995):

812

813 $$V_G(\mathrm{SHC}) = \frac{4n\mu\alpha^2 N_e}{1+\frac{\alpha^2 N_e}{V_S}}, \qquad \text{(eq. 1)}$$

814 where mutational input is determined as the product of the effective number of additively contributing
815 loci, $n$, mutation rate per generation, $\mu$, mutational effect, $\alpha^2$ and effective population size, $N_e$. The
816 loss of alleles by selection is governed by $V_S$, an inverse measure of the strength of stabilising
817 selection, which is equal to the sum of the squared width of the fitness function, $\omega^2$, and $V_E$, the
818 environmental component of phenotypic variance, $V_P$.

819 This particular formulation (eq. 1) applies to a sexual organism (see Lynch & Lande 1993 for
820 an approximation for asexual organisms). The model assumes there is no dominance or epistasis, thus
821 additive genetic variance is equal to the total genetic variance ($V_G = V_A$) and narrow sense heritability
822 is given by $h^2 = V_G/V_P$. Because quantitative traits are mainly determined by additive genes (Hill,
823 Goddard & Visscher 2008), the exclusion of non-additive genetic interactions from such models has
824 little to no negative effect on the efficacy of predictions (Crow 2010). Although the SHC
825 approximation provides the best estimate of mean genetic variance maintained at mutation-selection-
826 drift equilibrium (Bürger et al. 1989; Bürger & Lynch 1995) populations are rearly at equilibrium,
827 especially those with a low effective population size (Willi, Van Buskirk & Hoffmann 2006). This has
828 the effect of increasing variance around predictions but should not affect the mean risk of extinction.
829 To parameterise the breadth of the fitness curve we draw on reviews of common-garden studies,
830 which measured the thermal sensitivity of maximal population growth rate on a range of ectothermic
831 taxa from a range of latitudes (Frazier et al. 2006; Deutsch et al. 2008). These studies reveal a trend in
832 thermal specialisation towards the tropics. This increase in the strength of stabilising selection from

weak ($\omega \approx 10$) to moderate ($\omega \approx 3$) is within the range tested by theoretical models (Bürger & Krall 2004; Johnson & Barton 2005). The parameterisation of genetic variables is more problematic as estimates tend to vary substantially. Nevertheless, values for mutational heritability ($V_m/V_E$), a standardised composite measure of the input of mutational variance per generation, are remarkably consistent among traits and taxa, varying between $10^{-3}$ and $10^{-2}$ (Lynch & Walsh 1998 pp 335-340). Accordingly, we follow Bürger and Lynch (1995) in assuming that $V_m = 2n\mu\alpha^2 = 0.001$ and standardise parameter values to $V_E = 1$. On the conservative basis that genomic mutation rate ($2n\mu$) equals $0.02$, $\alpha^2$ is determined to be 0.05. To assess the sensitivity of our predictions of extinction risk to genetic input we also tested the effect of a lower mutational variance: $V_m = 10^{-4}V_E$ to account for the observation that up to 90% of mutations could be unconditionally deleterious (Lande 1995). The combined effects of thermal specialisation, effective population size and mutational variance on estimates of heritability are illustrated in Figure 1A.

- INSERT FIGURE 1 HERE

848 **Box 2 - Modelling adaptation and extinction risk under environmental change**

849 To estimate mean time to extinction under directional selection it is necessary to model the
850 dynamics of the distribution of phenotypes over time. The expected mean phenotype ($E[\bar{p}]$) and
851 expected variance in mean phenotype ($V[\bar{p}]$) can be obtained by recursion using the following
852 difference equations (Bürger & Lynch 1995; see original equations 6a and b).

853 $$E[\bar{p}_{t+1}] = E[\bar{p}_t] + s(kt - E[\bar{p}_t]), \qquad \text{(eq. 2)}$$

854 $$V[\bar{p}_{t+1}] = \frac{V_A}{N_e} + (1-s)^2 V[\bar{p}_t] + s^2 V_\theta, \qquad \text{(eq. 3)}$$

855 where *t* is time in generations, $k$ is the rate of environmental change per unit time, $V_\theta$is a measure of
856 environmental stochasticity, $N_e$ is effective population size and $s$ is a measure of the strength of
857 directional selection, equal to $V_A/(V_P + \omega^2)$. Where direct estimates of genetic variance are not
858 available, $V_A$ can be estimated as a function of mutation-selection-drift balance (see box 1).

859 Under directional selection the mean phenotype is predicted to eventually evolve at rate $k$, but
860 lag behind the mean environment $\theta$ by $k/s$. If the lag between the mean phenotype and the
861 environment becomes too large population growth rate becomes negative, at which point the
862 population rapidly goes extinct. The expected population growth rate at time *t* is given as:

863 $$r_t = r_{max,t} - \frac{(\bar{p}_t - \theta)^2}{2V_{\lambda,t}} \qquad \text{(eq. 4)}$$

864 where the maximal population growth rate can be given as, $r_{max,t} = \ln\left(B\omega/\sqrt{V_{\lambda,t}}\right)$, where $B$
865 offspring per capita when the mean phenotype is equal to its environment i.e. $E[\bar{p}] = \theta$, and where
866 $V_{\lambda,t}$, is equal to the sum: $\omega^2 + V_P + V[\bar{p}_t] + V_\theta$. In the simplest case of density dependence, for a
867 population size of $N$ breeding adults limited by a carrying capacity $K$, of the $N\exp^r$ offspring
868 produced per generation only a maximum of $K$ will survive; when $N\exp^r < K$ all offspring are
869 assumed to survive to the next generation. Extinction is assumed to occur once the population reaches
870 a minimum threshold (e.g. $N < 2$ for sexually reproducing organisms). To evaluate the relative
871 contribution of adaptation to reducing extinction risk, mean time to extinction of an adapting

population can be compared to that of a population where the mean phenotype is otherwise held constant i.e. $E[\bar{p}_{t+1}] = E[\bar{p}_0]$.

- INSERT FIGURE 2 HERE

An alternative measure of extinction risk that can be derived analytically is the theoretical maximum rate of environmental change at which a population could continue to just replace itself (i.e. $r_{max} = 0$) under directional selection. This measure of extinction risk is referred to as the critical rate of environmental change, $k_c$ (Lynch & Lande 1993). Beyond this rate population growth rate becomes negative leading to rapid extinction. Bürger and Lynch (1995) derive the critical rate of change as:

$$k_c = \frac{V_A}{V_P+\omega^2}\sqrt{2V_\lambda r_{max}}. \qquad \text{(eq. 5)}$$

where $V_\lambda = V_{\lambda,\infty}$ and $r_{max} = \ln\left(B\omega/\sqrt{V_\lambda}\right)$.

Obviously no trait can be expected to evolve indefinitely. But on the basis that long-term selection experiments on small populations have shown responses of ten or more phenotypic standard deviations a low critical rate of change could sustain an adaptive response to selection for hundreds, perhaps even thousands of generations before pleiotropic constraints are encountered (Lynch & Lande 1993; Bürger & Lynch 1995). Note that while these solutions can provide good estimates of *mean* time to extinction they fail to account for the skewed distributions of extinction times expected to occur at rates of environmental change close to the critical rate. Caution should be taken therefore when interpreting the risk of extinction.

- INSERT FIGURE 3 HERE

895 **Figure legends**

896 **Figure 1.** A) Theoretical estimates of heritability at mutation-selection-drift equilibrium and B) the
897 respective predicted critical rates of change calculated for thermal specialists (dashed lines; $\omega$ = 3)
898 and thermal generalists (solid lines; $\omega$ = 10). Black lines are predictions based on the lower estimate
899 of mean observed mutational variance ($V_m = 10^{-3}V_E$) and grey lines are predictions based on
900 expected proportion of mutations that are quasineutral in their effects i.e. Lande's discount factor
901 ($V_m = 10^{-4}V_E$). Other parameters: $B = 2$; $\alpha^2 = 0.05$.

902

903 **Figure 2.** Mean time to extinction calculated for A-B) a thermal specialist ($\omega = 3$) and C-D) a
904 thermal generalist ($\omega = 10$) as a function of A and C) birth rate per capita (bottom left to top right: $B$
905 = 1.2, 2, 10, 10000; $N_e$= 500) and B and D) effective population size (bottom left to top right: $N_e$=
906 100, 200, 500, 10000; $B = 2$). Solid lines represent the adaptive genetic response to temperature
907 change and dashed lines represent the null hypothesis of no adaptive change. Note the log-scale of
908 both axes. Other parameters: $V_m/V_E = 5 \times 10^{-4}/1$; $\alpha^2 = 0.05$; $V_\theta = 1$.

909

910 **Figure 3.** The predicted critical rate of change $k_c$ ($^{o}$C gen$^{-1}$) plotted against the breadth of the thermal
911 reaction norm and per capita birth rate for an effective population size of A) 100, B) 500, C) 1000 and
912 D) 10000. At low effective population sizes thermal specialists have a lower relative risk of extinction
913 but at high effective population sizes thermal generalists have a lower relative risk of extinction.
914 Other parameters: $V_m/V_E = 5 \times 10^{-4}/1$; $\alpha^2 = 0.05$; $V_\theta = 1$.

915

916 **Figure 4.** The effects of temperature-dependent fitness, as mediated by development rate, on
917 predicted extinction risk. Hypothetical scenarios depict the consequences of a thermodynamic
918 constraint ($-E = 0.65$) on the relative performance of an extreme thermal specialist (grey lines;
919 $\omega$=3) and an extreme thermal generalist (black lines; $\omega$=10). In A) species share the same thermal
920 optima (temperate $T_{opt}$= tropical $T_{opt}$=20 $^{o}$C), in B) the $T_{opt}$ differs by 10 $^{o}$C (temperate $T_{opt}$=15 $^{o}$C;
921 tropical $T_{opt}$=25 $^{o}$C) and in C) the $T_{opt}$ differs by a 20 $^{o}$C (temperate $T_{opt}$=10 $^{o}$C; tropical $T_{opt}$=30 $^{o}$C).

Panels D-F illustrate the respective consequences for the critical rate of change $k_c$ (contours indicate equivalent rates in $^{o}$C yr$^{-1}$) for the full range of thermal specialisations and birth rates (body mass = 1 gram). In panels G-I contours indicate an equivalent critical body mass (log10(grams)) based on calculations including the assumption that 'temperate' thermal generalist ($\omega$=10) species are exposed to double the rate of temperature change of 'tropical' thermal specialist ($\omega$=3) species (0.06 vs. 0.03 $^{o}$C yr$^{-1}$). Other parameters: $N_e$=1000; $V_m/V_E = 5 \times 10^{-4}/1$; $\alpha^2 = 0.05$; $V_\theta = 1$.

**Figure 5.** The predicted critical body mass (log10(grams)) of ectotherms in respect to effective population size, mutational variance and seasonal constraints (where development is restricted to a single season i.e. ¼ of the year). Calculations are based on the assumption that 'temperate' thermal generalist species ($T_{opt}$=15 $^{o}$C; $\omega$=10) are exposed to a higher rate of temperature change than tropical thermal specialist species ($T_{opt}$=25 $^{o}$C; $\omega$=3): 0.06 vs. 0.03 $^{o}$C yr$^{-1}$ (see scenarios in figure 4B). Contours depict equivalent values of the critical body mass (see text) calculated for various combinations of variables. Other parameters: $-E = 0.65$; $V_E = 1$; $\alpha^2 = 0.05$; $V_\theta = 1$.

Figure 1

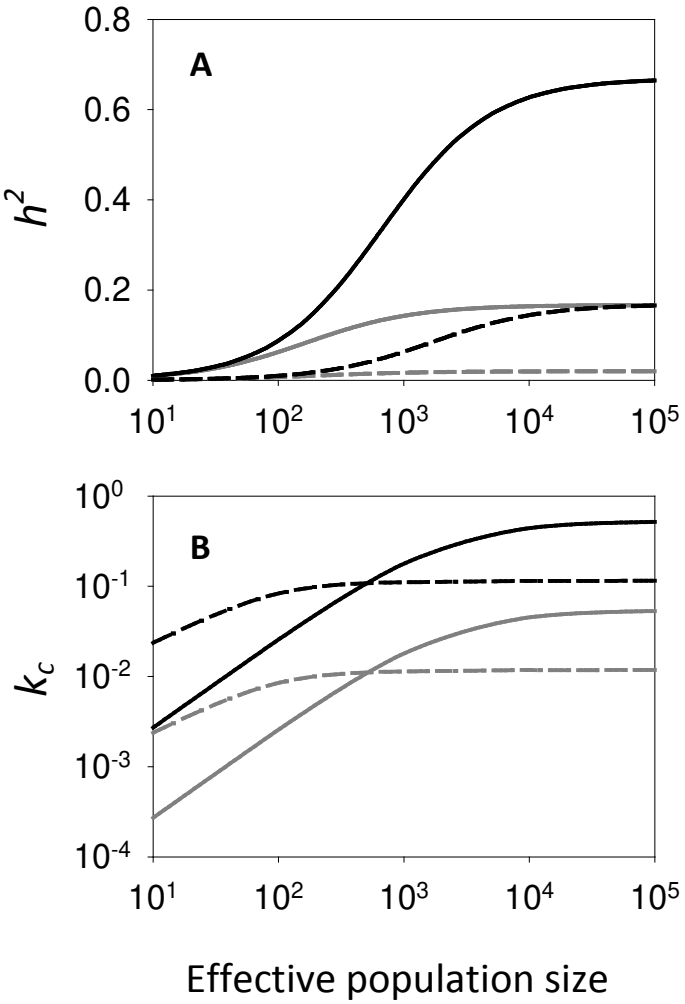

A
B
$h^2$
$k_c$
0.8
0.6
0.4
0.2
0.0
$10^0$
$10^{-1}$
$10^{-2}$
$10^{-3}$
$10^{-4}$
$10^1$
$10^2$
$10^3$
$10^4$
$10^5$
Effective population size

Figure 2

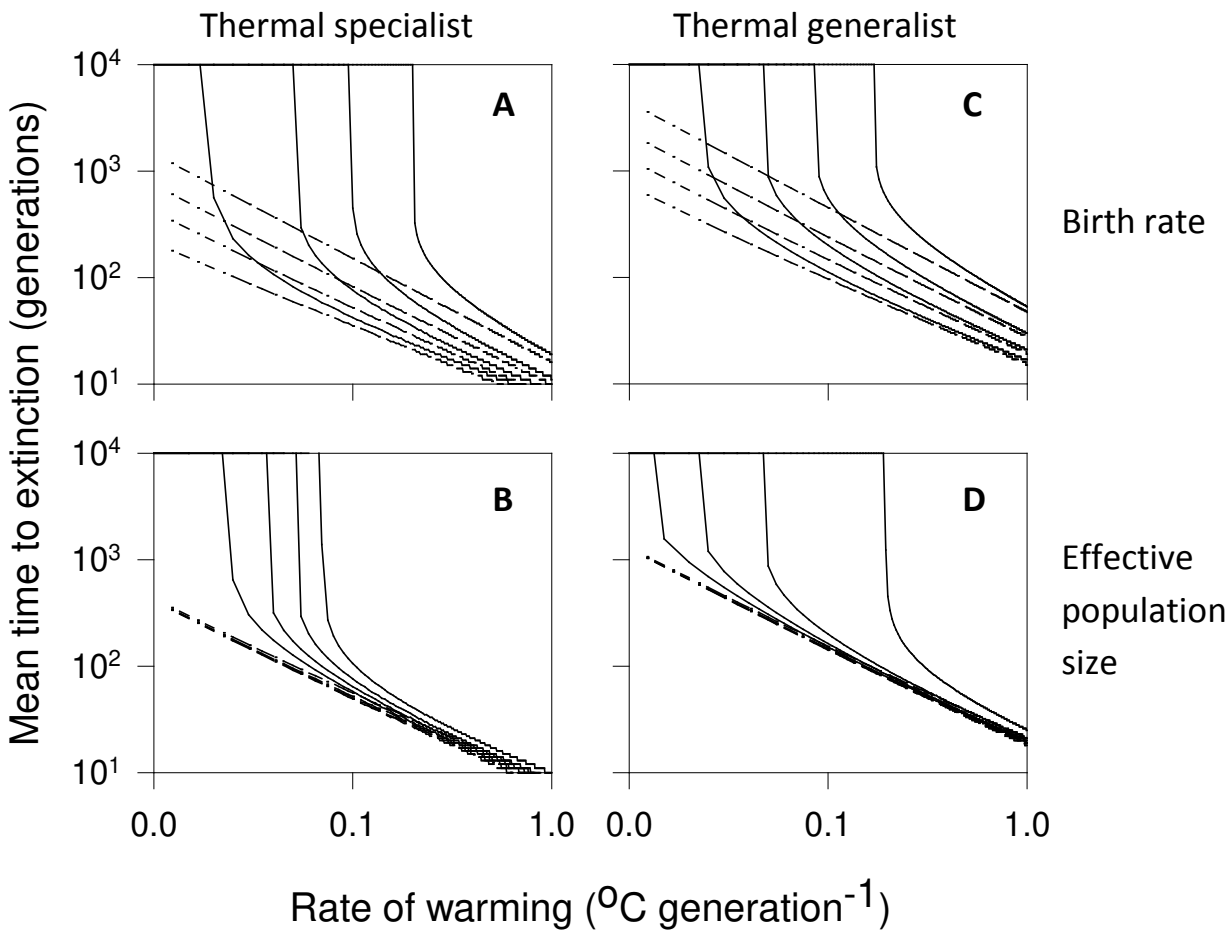

Thermal specialist
Thermal generalist
A
B
C
D
Birth rate
Effective population size
Mean time to extinction (generations)
$10^4$
$10^3$
$10^2$
$10^1$
0.0
0.1
1.0
Rate of warming ($^oC$ generation$^{-1}$)

Figure 3

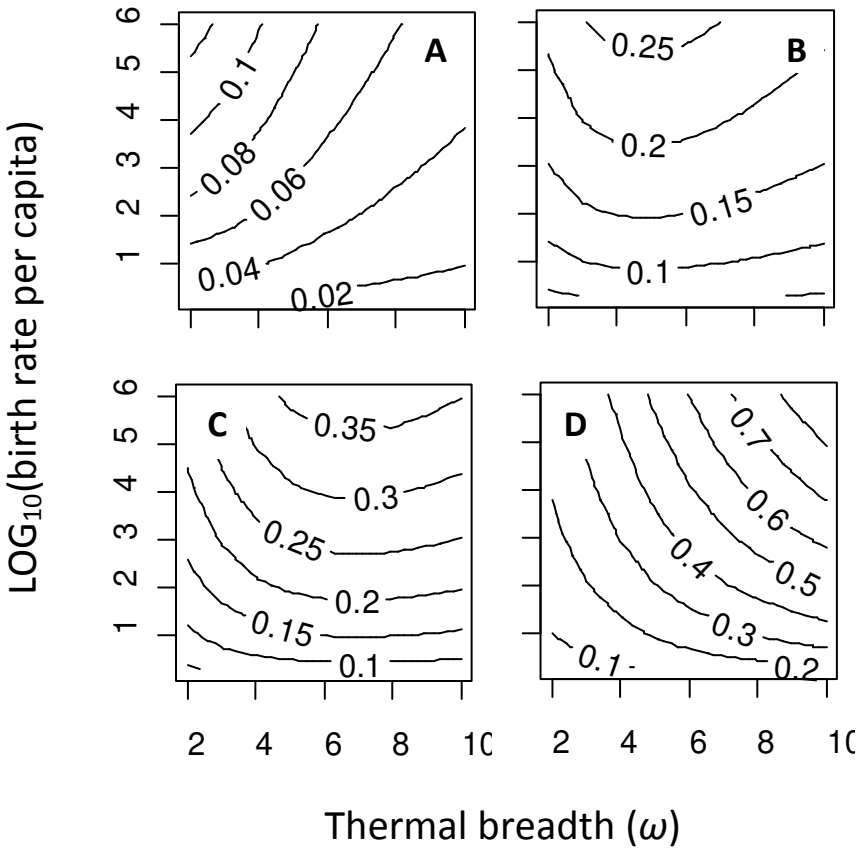

A
0.1
0.08
0.06
0.04
0.02
B
0.25
0.2
0.15
0.1
C
0.35
0.3
0.25
0.2
0.15
0.1
D
0.7
0.6
0.5
0.4
0.3
0.2
0.1
1 2 3 4 5 6
2 4 6 8 10
LOG10(birth rate per capita)
Thermal breadth (ω)

Figure 4

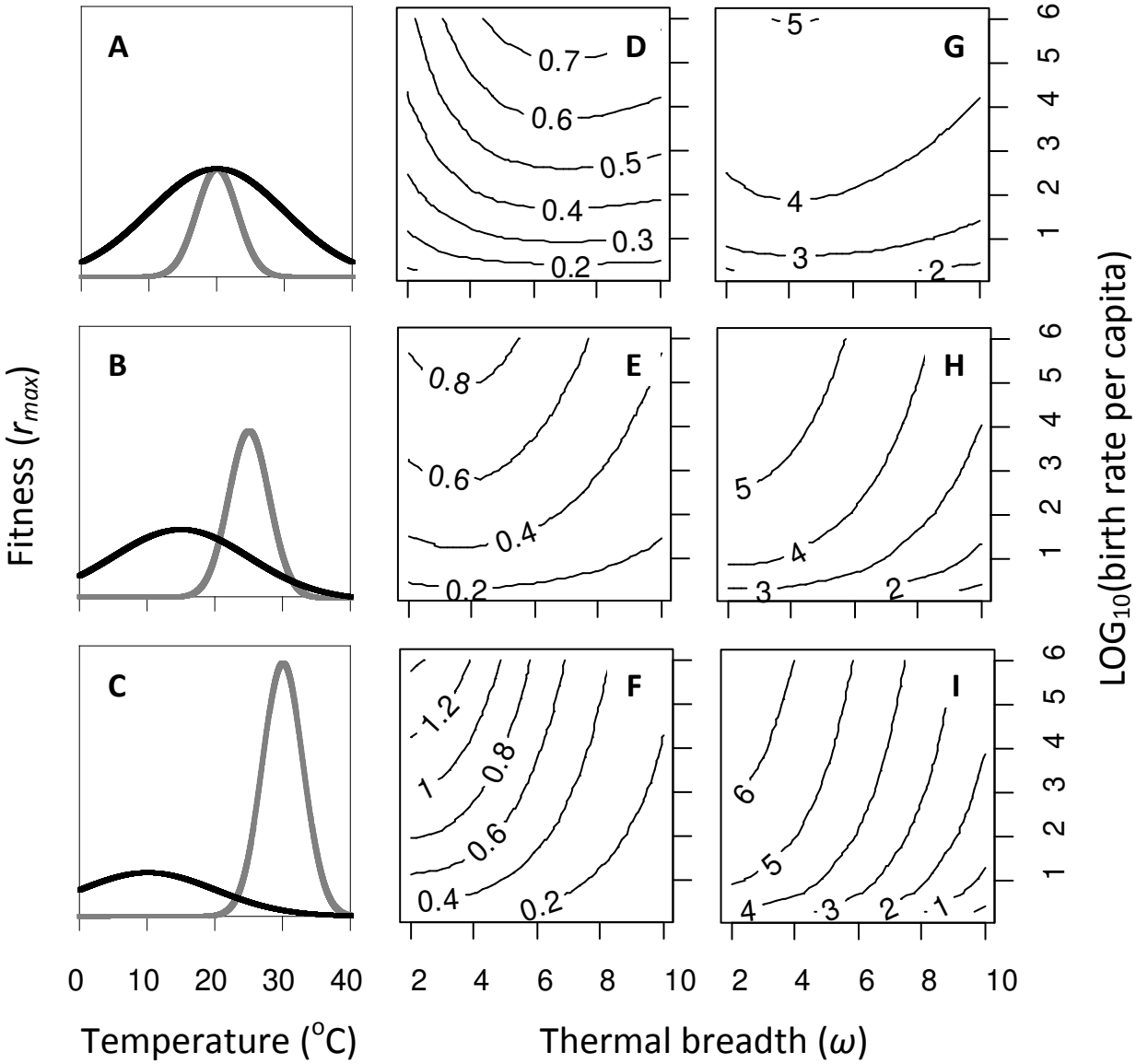
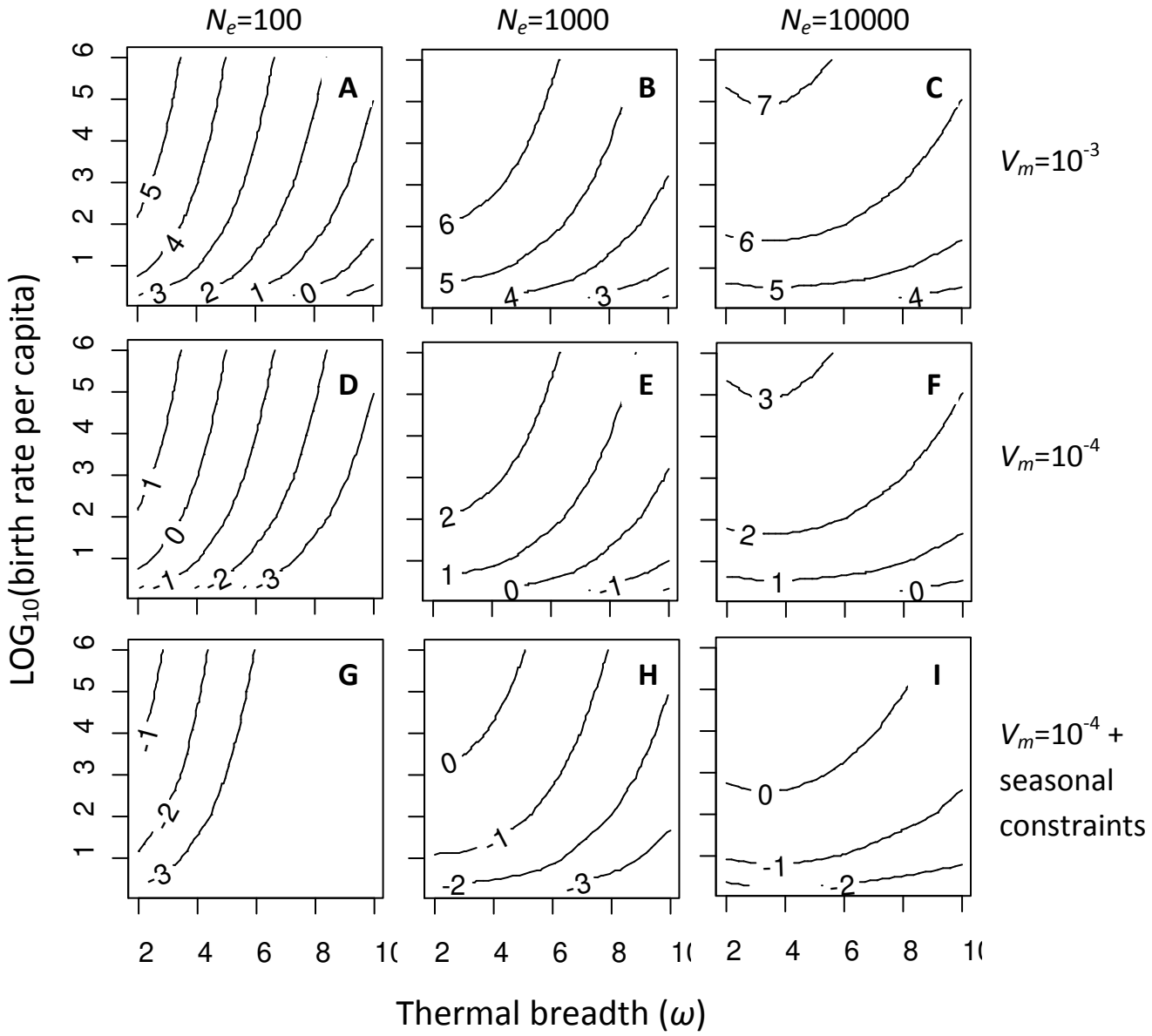

A
B
C
D
E
F
G
H
I
Fitness ($r_{max}$)
$LOG_{10}$(birth rate per capita)
Temperature (°C)
Thermal breadth ($\omega$)
0 10 20 30 40
2 4 6 8 10
1 2 3 4 5 6
0.7 0.6 0.5 0.4 0.3 0.2
0.8 0.6 0.4 0.2
1.2 1 0.8 0.6 0.4 0.2
5 4 3 2
6 5 4 3 2 1

Figure 5


$N_e$=100
$N_e$=1000
$N_e$=10000
A
B
C
D
E
F
G
H
I
$V_m$=$10^{-3}$
$V_m$=$10^{-4}$
$V_m$=$10^{-4}$ + seasonal constraints
$LOG_{10}$(birth rate per capita)
Thermal breadth ($\omega$)