LETTER

Predicting natural mortality rates of plants and animals

Abstract

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Department of Zoology, University of Florida, 223 Bartram Hall, PO Box 118525, Gainesville, FL 32611, USA *Correspondence and present address: Department of Biology, Boston University, 5 Cummington St, Boston, MA, USA E-mail: mmccoy@zoo.ufl.edu Understanding the factors that control the mortality rates of species in their natural environments is important for understanding the structure and dynamics of populations, communities and ecosystems. Here, we test a model of natural mortality that yields explicit, quantitative predictions based on the constraints of body size and temperature on individual metabolism. Extensive field data from plants, invertebrates, fish, birds and mammals indicate that much of the heterogeneity in rates of natural mortality can be predicted, despite the many extrinsic sources of mortality in natural systems. These results suggest that common 'rule(s)' govern mortality rates in ecological communities for organisms as diverse as plants and animals.

Keywords

Allometry, free radical, life history, life span, metabolic theory, metabolism, mortality, Reactive oxygen species (ROS).

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INTRODUCTION

Birth and death rates largely govern the structure and dynamics of populations, communities and ecosystems. Consequently, understanding the factors that control birth and death rates among species has been a central focus of ecological and evolutionary theory, and the topic of many empirical studies. Across a broad array of species, rates of growth, reproduction and even maximum life span have been shown to scale predictably with body size and temperature (Blueweiss et al. 1978; Peters 1983; Calder 1996; Damuth 2001; Niklas & Enquist 2001; West et al. 2001; Gillooly et al. 2002; Brown et al. 2004; Savage et al. 2004a; Frazier et al. 2006; Marba et al. 2007), but to date, evidence for a general relationship between body size, temperature and rates of natural mortality is limited. This has led to a significant debate regarding the extent to which rates of natural mortality are idiosyncratic or whether these rates follow some general rules that apply across species and habitats (Ricklefs 1998; Mangel 2001; Ricklefs & Scheuerlein 2001; Speakman et al. 2002; Carey & Tuljapurkar 2003; Speakman 2005; Hulbert et al. 2007).

Explanations for rates of natural mortality have generally fallen into two classes: intrinsic or extrinsic. Intrinsic explanations are based on long-standing correlations reported between life span and individual metabolic rate, or the two primary determinants of metabolic rate, body size and temperature (Rubner 1908; Pearl 1928). Organisms with shorter life spans have been shown to have higher metabolic rates and incur higher rates of cell damage or decay (e.g. Sohal et al. 1989; Ku et al. 1993; Kapahi et al. 1999; Wright et al. 2004; Atanasov 2005; Hulbert et al. 2007) perhaps due to the accumulation of oxidative free radicals (a by product of metabolism) (Dufour & Larsson 2004; Huang & Manton 2004; Speakman 2005; Hulbert et al. 2007). Alternatively, extrinsic explanations of mortality are based on the observation that organisms in natural environments typically die as a result of disease, predation or accident, well before they reach their maximum possible life span (Ricklefs 1998). Under this explanation, organisms may show a regular pattern of intrinsic senescence or cellular decay (Mangel 2001; Ricklefs & Scheuerlein 2001; Charnov & Gillooly 2004), but this is expected to occur later in life, beyond the point where it could impair fitness (Hamilton 1966; Charlesworth 2000).

In this study, we take advantage of newly available theory and data to address the factors that control natural mortality rates across diverse species. Specifically, we test a model that yields predictions on rates of natural mortality, based on the body size and temperature dependence of individual metabolic rate. To do so, we use extensive data for species from different taxonomic groups living in their natural environments, including plants, fish, invertebrates, birds and mammals. Although, the size or temperature dependence of natural mortality within some of these groups has been previously shown (Pauly 1980; Gillooly *et al.* 2001; Marba *et al.* 2007), comparisons across such a broad diversity of organisms have not yet been undertaken. These comparisons will allow us to gain a better general understanding of the factors controlling rates of natural mortality in plants and animals.

MODEL AND PREDICTIONS

We test a previously published model of mortality from Gillooly *et al.* (2001; see also Brown *et al.* 2004; Savage *et al.* 2004a). The model builds on a long history of work that relates body size and temperature to biological rate processes (e.g. Peters 1983; Calder 1996) to propose that natural mortality rates, whether due to extrinsic and/or intrinsic factors, should show the same body size and temperature dependence as individual metabolic rate.

Specifically, this theoretical framework proposes that mass-specific metabolic rate, or the rate at which organisms acquire and use energy per gram of tissue, *B*, scales with body size and temperature as:

$$B = b_0 m^{-1/4} e^{\left(-\frac{E}{k} \left[\frac{1}{T} - \frac{1}{T_{20} \circ c}\right]\right)},$$
(1)

where b_0 is a taxon-dependent normalization constant $(W/g^{-3/4})$. The negative 1/4 power scaling of metabolic rate with body mass (m in dry grams) is supported with empirical evidence (Savage et al. 2004b) and is consistent with several theoretical formulations including those of West et al. (1997, 1999). The Boltzmann–Arrhenius factor, $e^{-E/KT}$, describes the exponential increase in biochemical reaction rates with body temperature (Gillooly et al. 2001). Here, we standardize the metabolic rate to 20 °C leading to the standardized Boltzmann–Arrhenius factor, $e^{\left(-\frac{E}{k}\left[\frac{1}{T}-\frac{1}{T_{20}\circ C}\right]\right)}$, where E is the average activation energy of heterotrophic respiration in animals (c. 0.6-0.7 eV) (Gillooly et al. 2001, 2005) or photosynthesis in plants (c. 0.32 eV) (Allen et al. 2005), k the Boltzmann's constant (8.62×10^{-5} eV K⁻¹), T the absolute temperature in degrees Kelvin and $T_{20 \circ C}$ the standardization temperature (i.e. 293°).

When combined with the assumption that the amount of energy expended per lifetime is approximately constant following from the rate of living hypothesis, eqn 1 can also be used to make predictions on the rates of natural mortality. Specifically, under this assumption, natural mortality rate Z [individual/(individual × year)] is predicted to be proportional to the metabolic rate B, and therefore show the same body size and temperature dependence as eqn 1 (Gillooly *et al.* 2001; Brown *et al.* 2004):

$$Z = fb_0 m^{-1/4} e^{\left(-\frac{E}{k} \left[\frac{1}{T} - \frac{1}{T_{20} \circ c}\right]\right)},$$
(2)

where f is the taxon-specific mortality rate per unit of metabolic energy expended by a unit mass of tissue (g deaths individual⁻¹ Joules⁻¹). Note that eqn 2 predicts much weaker temperature dependence for plant mortality than for animal mortality based on the differences in the average activation energies of heterotrophic respiration vs. photosynthesis (Allen *et al.* 2005). Note also that this equation is not expected to apply in the early life-history stages of species that exhibit density-dependent mortality in these stages (Charnov & Gillooly 2004).

Equation 2 yields two quantitative predictions regarding mortality rates. First, the natural logarithm of masscorrected mortality rates, $\ln(Zm^{1/4})$, should be a linear function of inverse absolute temperature, $(1/k)[(1/T) - (1/T_{20^{\circ}C})]$, with a slope of *c*. -0.65 eV for animals and -0.32 eV for plants, reflecting the temperature dependence of metabolism. Thus, for a 30 °C decrease in temperature, a 16-fold decrease in mortality rates is predicted for animals and a fourfold decrease is predicted for plants (Allen *et al.* 2005).

Second, the natural logarithm of temperature-corrected mortality rate, $\ln(Ze^{\left(\frac{E}{k}\left[\frac{1}{t}-\frac{1}{T_{20}\circ c}\right]\right)})$, should be a linear function of the natural logarithm of body mass, $\ln(m)$, with a slope of c. -1/4, reflecting the size dependence of metabolic rate in both plants and animals. Thus, mortality rates are predicted to decrease by an order of magnitude for every 4 orders of magnitude increase in body mass in both groups.

MATERIALS AND METHODS

Data

For a broad array of plant and animal species, we compiled data on natural mortality rates, defined as the exponential rate of decrease of individuals in field censuses, or natural longevity. For each species, we also compiled data on average adult body masses and body temperatures. These data were compiled primarily from published data compilations and include species from five taxonomic groups [invertebrates = 128 species, fish = 168 species, plants = 278 species (including phytoplankton), mammals = 361 species, birds = 600 species; see Appendix S1 for a detailed list of data and sources], which span a broad range of body temperatures (-1 to 40 °C) and body masses (1.4×10^{-14} to 1.5×10^8 g). Each taxonomic group is represented by a diversity of species. Data for plants include phytoplankton, and a variety of multicellular plants (macroalgae, mosses, herbs, aquatic plants, woody shrubs and trees; Appendix S1). Data for both fish and invertebrates include freshwater and marine species, including species from both shallow water and deep-sea habitats (Appendix S1).

In addition, the invertebrate data include a tremendous diversity of species ranging from small zooplankton to large mollusks and crustaceans living in the benthos. A small number of lab studies also included the estimated minimum mortality rates (1/maximum life span) for several species of freshwater invertebrates (Appendix S1). Data for mammals and birds included field estimates of both average and minimum natural mortality rates for species (i.e. records from captive species were not included).

Minimum mortality rates (1/maximum life span) were converted to average mortality rates (1/average life span) based on the observation that minimum mortality rates are c. 2.5-fold lower than average mortality rates in endotherms (Charnov 1993; Ricklefs 2000; Ricklefs & Scheuerlein 2001). Average body temperature for birds was taken as 40 °C and for mammals as 38 °C (Schmidt-Nielsen 1997).

Statistical analysis

We compared the body size and temperature dependence of natural mortality rates among taxonomic groups, and the extent to which these differed from model predictions.

First, we used analysis of covariance (ANCOVA) to test for differences in the slopes and intercepts of the relationships among groups. In evaluating differences in intercepts given model predictions, ANCOVAS were performed with the slope fixed to the values predicted by eqn 2. Second, we used ordinary least-squares regression to determine how much of the variation in natural mortality rates within each group can be explained by the body size and temperature dependence predicted by the model (eqn 2).

RESULTS

Prediction 1: temperature dependence of natural mortality

Data support the predicted temperature dependence of natural mortality rates for those animals considered here (Fig. 1). For both fish and invertebrates, the natural logarithm of mass-corrected natural mortality rate is significantly negatively correlated with inverse absolute temperature (regression: $F_{1,430} = 368.69$, P < 0.0001, $r^2 = 0.46$). Surprisingly, there was no significant difference in the slopes (ANCOVA: $F_{1,428} = 1.08$, P = 0.30) or intercepts (ANCOVA: $F_{1,428} = 3.52$, P = 0.06) between fish and invertebrates. Moreover, the common slope estimated for the two groups gives an activation energy of -0.57 eV (95%) CI = -0.63 to -0.51 eV), which is close to the predicted value of -0.65 eV (Table 1; Fig. 1), and statistically indistinguishable from the range of average activation energies (i.e. -0.6 to -0.7 eV) for metabolic rate. The model (eqn 2) fitted to the data with the predicted slope of -0.65 explains 43 and 45% of the variation in body mass-corrected natural mortality rates for invertebrates and fish, respectively. Note that these relationships include organisms from both

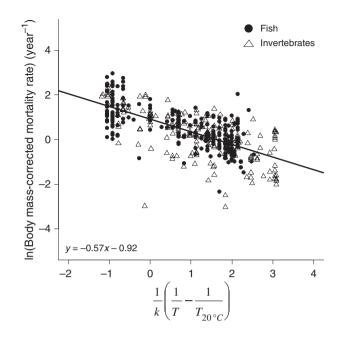


Figure 1 Temperature dependence of body mass-corrected natural mortality rates, $\ln(Zm^{1/4})$, for invertebrates and fishes. The solid line indicates the estimated slope and intercept from an ANCOVA, and the dashed line indicates the predicted slope of -0.65 eV for animals based on the average activation energy of metabolic rate.

shallow water and deep-sea environments. Although deepsea species are sometimes considered to be extremophiles with unusually long life spans (e.g. Cailliet *et al.* 2001), their life spans do not appear to be significantly different from other species after accounting for the effects of mass and temperature.

However, in multicellular plants, there was no statistically significant relationship between mass-corrected mortality rate and inverse absolute temperature ($F_{1,365} = 3.73$, P = 0.054, $r^2 = 0.01$; Table 1; see Marba *et al.* 2007). This is perhaps not surprising, as most of the data fall within a relatively narrow range of temperatures (6–22 °C; as reported in Marba *et al.* 2007), and the *a priori* expectation was only a twofold difference due to the relatively weak temperature dependence of photosynthesis (i.e. $E \sim 0.32$ eV; Allen *et al.* 2005). In contrasts, in phytoplankton, the slope was steeper than predicted. Clearly, other factors such as water availability are important to plant metabolism, and these have not been incorporated into the model.

Prediction 2: mass dependence of natural mortality

Data also support the predicted mass dependence of natural mortality rates for both plants and animals (Fig. 2a-e). The natural logarithm of the temperature-corrected mortality

Independent variable	Group(s)	Predicted slope (eV)	Fixed slope intercept	Fitted slope	Fitted intercept
Temperature, $\left(\frac{1}{k}\left[\frac{1}{T}-\frac{1}{T_{20^{\circ}C}}\right]\right)$	Fish and invertebrates	-0.65	1.00 (0.93 to 1.07)	-0.57 (-0.63 to -0.51)	0.92 (0.83 to 1.01)
	Multicellular plants	-0.32	-0.68 (-0.83 to -0.53)	0.13 (-0.02 to 0.28)	-0.82 (-0.97 to -0.67)
	Phytoplankton	-0.32	-3.11 (-3.39 to -2.83)	-0.96 (-1.26 to -0.66)	-3.25 (-3.51 to -2.98)
Body mass, ln(<i>m</i>)	Birds	-0.25	-2.18 (-2.21 to -2.14)	-0.20 (-0.23 to -0.17)	-2.36 (-2.48 to -2.25)
	Fish	-0.25	1.05 (0.96 to 1.13)	-0.27 (-0.35 to -0.19)	1.16 (0.80 to 1.53)
	Invertebrates	-0.25	0.94 (0.83 to 1.05)	-0.26 (-0.32 to -0.19)	0.91 (0.62 to 1.20)
	Mammals	-0.25	-1.19 (-1.24 to -1.14)	-0.24 (-0.31 to -0.17)	-1.30 (-1.64 to -0.97)
	Multicellular plants	-0.25	-0.68 (-0.83 to -0.53)	-0.30 (-0.36 to -0.25)	-0.48 (-0.74 to -0.22)
	Phytoplankton	-0.25	-3.14 (-3.41 to -2.87)	-0.22 (-0.30 to -0.13)	-2.18 (-3.83 to -0.54)

Table 1 Parameter estimates and confidence intervals estimated via ANCOVA for the data shown in Figs 1-3

Numbers in parentheses represent the upper and lower bounds of the 95% confidence intervals.

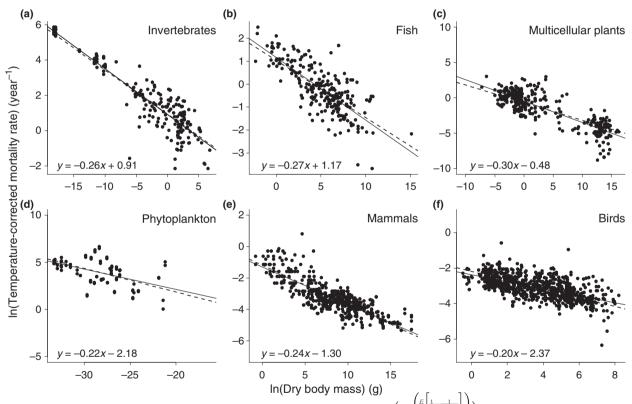


Figure 2 Body mass dependence for temperature-corrected natural mortality rates, $\ln \left(Ze^{\left(\sum_{k} \left\lfloor \frac{1}{t} - \frac{1}{T_{20}\circ c} \right\rfloor} \right)} \right)$, for: (a) invertebrates, (b) fish, (c) multicellular plants, (d) phytoplankton, (e) mammals and (f) birds. Solid lines indicate the estimated slope and intercept from an ANCOVA. Dashed lines indicate the predicted slope of -1/4 based on the mass dependence of individual metabolic rate.

rate for all plants and animals (pooled) is negatively correlated with the natural logarithm of body mass (regression: $F_{1,2101} = 2883$, P < 0.0001), with a fitted slope (-0.25, 95% CI = -0.26 to -0.24) that is identical to the predicted value of -0.25 (Fig. 3). Within taxonomic groups, the slopes range from -0.20 to -0.30, and the 95% CIs for fish, invertebrates, mammals, phytoplankton and multicel-

lular plants overlap the predicted value of -0.25 [Table 1; see previous studies for plants (Marba *et al.* 2007), fish (Savage *et al.* 2004a), mammals (Gaillard *et al.* 2003) and invertebrates (Gillooly *et al.* 2001)]. The model, assuming a slope of -1/4 explains considerable variation in natural mortality rates for both the pooled data (59%) and for fits to individual taxonomic groups (mammals = 66%,

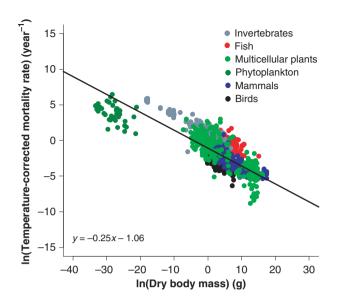


Figure 3 The body mass dependence of temperature-corrected natural mortality rates, $\ln(Ze^{(\frac{E(1)}{kT}-\frac{1}{2u\circ c})})$, for all species shown in Fig. 2a–e. The solid line indicates the estimated slope and intercept from an Ordinary least squares (OLS) regression. The slope is equal to the predicted value of -0.25.

fish = 58%, invertebrates = 84%, phytoplankton = 73%, multicellular plants = 72% and birds = 36%).

Differences in rates of mortality among taxonomic groups

Perhaps most surprisingly, analysing the data with the slope fixed to the predicted value of -1/4, we found considerable overlap in the temperature-corrected rates of mortality among taxonomic groups (Fig. 3). Still, there were differences in the intercepts among some groups (Table 1). Among invertebrates and fish, the intercepts were not significantly different (note that 'minimum' mortality rates estimated in the laboratory fall within the range of natural rates estimated in the field). But, bird and mammal mortality rates were, on average, c. 24- and 9-fold lower, respectively, than invertebrates and fish. Multicellular plants had mortality rates that were on average approximately fivefold lower than ectothermic animals, but higher than those for endotherms. Phytoplankton had the lowest mortality rates: c. 12-fold lower than multicellular plants. Differences in intercepts among groups shown in Fig. 3 are due to differences in b_0 values among these groups (eqn 2; Gillooly et al. 2001) and/or taxon-specific differences in lifetime energy expenditure (i.e. differences in f from eqn 2).

DISCUSSION

Overall, data support model predictions. In fishes and invertebrates, the observed temperature dependence of

mortality was statistically indistinguishable between groups and only marginally different from model predictions. This was surprising given the enormous differences in form, function and life history among these organisms. Moreover, for all taxonomic groups, the observed body size dependence was not significantly different from that predicted by eqn 2 (Figs 2 and 3). This indicates that, on average, at least within individual taxonomic groups, the amount of energy expended in a lifetime is an approximately invariant quantity consistent with the 'rate of living hypothesis'. In principle, this quantity can be calculated based on the y-intercepts of Figs 2 and 3, $\ln(f b_0)$ given an independent estimate of b_0 , similar to previously published calculations of energy expenditure for maximum life span in birds and mammals (Rubner 1908; Austad & Fischer 1991; Hulbert et al. 2007). But perhaps more importantly, Fig. 3 suggests that, much like the 'mouse to elephant curve' describing the general relationship of metabolic rate to body mass, there appears to be a general relationship of natural mortality that applies across species. For example, the body size dependence of mortality in plants was similar to that of animals. This is consistent with recent work showing that metabolic rates (Gillooly et al. (2001), as well as rates of growth (Damuth 2001; Niklas & Enquist 2001) and production (Ernest et al. 2003), are similar in plants and animals.

However, Figs 2 and 3 also point to substantial variation in natural mortality rates that cannot be explained by body size and temperature alone. As such, this model may be useful as a point of reference for understanding the ecological and evolutionary mechanisms that may be responsible for this variation. This variation might be viewed as indicative of life-history trade-offs by species from particular environments in an attempt to maximize fitness (Brown & Sibly 2006; Sibly & Brown 2007). Such trade-offs could also be reflected in the energy invested by species to reduce cell damage by reducing oxygen-related free radical production, neutralizing free radicals or repairing damage caused by free radicals. Indeed, the differences in mortality rates among groups observed here are consistent with previous research that suggests ectotherms are more susceptible to oxidative damage than endotherms (Venditti et al. 1999) and that rodents are more susceptible to oxidative damage than similarly sized birds (Barja et al. 1994; Barja 1998; Perez-Campo et al. 1998; Pamplona et al. 1999a,b; Ogburn et al. 2001).

Finally, these results may provide some insights regarding the relative importance of intrinsic vs. extrinsic controls on mortality. The general agreement of the data with model predictions are certainly consistent with the hypothesis that mortality is under intrinsic control and that these intrinsic mechanisms (e.g. free radical production) are constrained by the body size and temperature dependence of metabolism. However, natural mortality is clearly not exclusively under intrinsic control – extrinsic factors such as predation or disease do cause mortality. Yet, while many ecological factors leading to mortality (competition, predation) may be ultimately constrained by individual metabolic rate (Peters 1983; Brown *et al.* 2004), it is difficult to see how they could combine to generate the observed size and temperature dependence given the vast differences in species and habitats examined here. Perhaps in all communities, the cumulative effects of cell damage with age may increase an organism's vulnerability to extrinsic factors. In any case, intrinsic and extrinsic factors that control natural mortality rates are clearly linked through individual metabolism.

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SUPPLEMENTARY MATERIAL

The following supplementary material is available for this article:

Appendix S1 Temperature, body mass and mortality data with sources.

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/full/10.1111/j.1461-0248.2008.01190.x

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