# Ecological Modeling of Metabolic Rates Predicts Diverging Optima across Food Abundances

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abstract: Genetically based variation in metabolic rates of resting animals (RMR) suggests a potential role for evolutionary adaptations, but mechanistic models yielding evolutionary predictions are lacking. Here I utilize the increasingly recognized genetic correlation between RMR and activity metabolism and propose that optimality of the former is simply an outcome of selection on the latter. I develop a model for temporally stable environmental conditions that describes how the rate of acquisition of energy that can be converted into somatic growth and reproductive output can be expressed as a function of activity metabolism. One of the parameters in the model describes how food intake depends on activity and is hence a measure of food abundance. In contrast to the previously proposed hypothesis that individuals with a high RMR are at an advantage when environmental conditions are favorable, the model predicts that the optimal RMR is highest at an intermediate food abundance.

*Keywords:* standard metabolic rate, basal metabolic rate, resting metabolic rate, density-dependent selection, food availability.

## Introduction

Resting metabolic rate (RMR; basal metabolic rate in endotherms and standard metabolic rate in ectotherms) of nonfeeding, nongrowing individuals represents the energy loss experienced due to maintenance of vital organs and body functions. This energy loss varies greatly among individuals and species, being correlated with both characteristics of the organisms themselves (e.g., body size [Gillooly et al. 2001], life style [Killen et al. 2010], sex [Marhold and Nagel 1995], and reproductive stage [Vezina et al. 2006]) as well as environmental variables (e.g., temperature [Gillooly et al. 2001], parasitism [Scantlebury et al. 2007], and habitat structure [Millidine et al. 2006]). Additive genetic variation in this trait (Rønning et al. 2007; Nilsson et al. 2009; Wone et al. 2009) suggests that it can evolve and adapt given contrasting selective regimes, and this has been corroborated by artificial selection experiments (Harshman et al. 1999; Ksiazek et al. 2004; Brzek et al. 2007) as well as by genetically based differences among populations (e.g., Lardies and Bozinovic 2008). Furthermore, numerous studies demonstrate correlations between RMR and fitness-related traits (reviewed by Biro and Stamps 2010; Burton et al. 2011). Thus, the crucial remaining issue in understanding the evolution of variation in this trait is whether such correlations with fitness depend on environmental conditions and hence whether the optimal RMR might vary through time and space. There is a poor mechanistic understanding of how the optimum RMR varies across environments and hence which environmental factors might cause evolutionary responses in RMR and in which directions these might work. This also precludes a mechanistic understanding of empirical studies demonstrating context-dependent fitness effects of RMR, particularly in relation to food abundance (reviewed in Burton et al. 2011).

The aim of this article is therefore to develop a model that predicts how the optimal RMR changes qualitatively with changes in food abundance. I start by recognizing that an ideal organism would spend very little energy on RMR while being able to have a high food intake. The high food intake would require a high behavioral activity and a corresponding high-activity metabolic rate $R_A$, which is defined as the rate of energy allocation to activity. The high food intake would also result in a high specific dynamic action $R_{SDA}$, which is defined as the rate of energy allocation to digestion and synthesis of new tissue (i.e., growth overheads, e.g., Nisbet et al. 2000). These latter two terms would then be the dominating components of total metabolic rate $R_{TOT}$ ($R_{TOT} = \text{RMR} + R_A + R_{SDA}$). However, it seems likely that there must be a physiological constraint with respect to this, because organs required for higher behavioral activity and digestion rates must be maintained by metabolism even when individuals are resting. This is supported by observations of genetic correlations between RMR and maximum aerobic capacity (Sadowska et al. 2005; Wone et al. 2009), spontaneous movement activity (Gebczynski and Konarzewski 2009; Careau et al. 2011) and food intake (Ksiazek et al. 2004;

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Gebczynski and Konarzewski 2009). A correlation between RMR and peak metabolic rate also exists among species (Wiersma et al. 2007), and species that routinely perform energetically costly activities have higher RMRs than less active ones (Reinhold 1999). Furthermore, from an optimality perspective, there should be selection against increasing RMR, which represents an energy loss without providing a benefit per se, unless this increase allows for an increased $R_A$. Thus, here I propose that optimality of RMR across environments can be predicted qualitatively given correlations between RMR and $R_A$.

## The Model

My modeling approach focuses on the relationship between metabolism and the energy balance, assuming that evolution should act to maximize the rate with which surplus of energy can be converted into somatic growth and reproductive output. The model is restricted to populations in temporally stable environments and does not address the effects of environmental stochasticity or seasonality. For modeling purposes, the three metabolic components used throughout are defined as (1) resting metabolic rate, the metabolic rate of an inactive nonfeeding, nongrowing organism; (2) activity metabolic rate ($R_A$), the metabolic rate being due to behavioral activity; and (3) standard dynamic action ($R_{SDA}$), the metabolic rate being due to digestion as well as synthesis of new tissue (i.e., growth overheads). Although the growth overheads may to some extent be temporally unlinked to ingestion in a variable environment, this does not have any consequences for this model, which deals with temporally stable environments.

Using an approach equivalent to that used in the Holling disc equation (Holling 1959), I start by expressing the number of prey consumed, $N_C$, as a function of activity metabolic rate, $R_A$ (calories [cal] time$^{-1}$), and the duration of the search time period, $T_S$ (time):

$$N_C = \beta R_A T_S, \tag{1}$$

where $\beta$ is the experienced prey encounter rate per unit of activity (no. prey cal$^{-1}$) for a given prey density and searching efficiency. Thus, the value of $\beta$ increases with increasing food abundance. Due to handling time of individual prey caught, the total search time can be expressed as

$$T_S = T_{TOT} - T_H N_C, \tag{2}$$

where $T_{TOT}$ is the total time of the period of interest, and $T_H$ is the time spent on handling each unit of prey. By substitution and rearranging, equations (1) and (2) yield

$$N_C = \frac{\beta T_{TOT} R_A}{1 + \beta T_H R_A}. \tag{3}$$

To simplify, I set a common handling time $T_H = 1$. Dividing equation (3) by $T_{TOT}$ yields an intake rate $I$:

$$I = \frac{\beta R_A}{1 + \beta R_A}. \tag{4}$$

The energy assimilation rate, $A$, will depend on both intake rate and the proportion of the ingested energy that is assimilated, $p$. The proportion of the ingested energy that is assimilated varies relatively little with ration (e.g., Elliott 1976*b*). Thus, the rate of energy assimilation can be expressed as

$$A = pI = \frac{p\beta R_A}{1 + \beta R_A}. \tag{5}$$

The rate of energy surplus acquisition, $E$, that can be used for somatic growth and reproduction can be expressed as

$$E = A - R_{TOT}, \tag{6}$$

where $R_{TOT}$ is the total metabolism ($R_A$ + RMR + $R_{SDA}$). Since RMR is genetically correlated with $R_A$ (see "Introduction"), $R_{TOT}$ in equation (6) can be expressed as

$$R_{TOT} = R_A(1 + q) + R_{SDA}, \tag{7}$$

where $q$ is a positive constant determining the proportional relationship between RMR and $R_A$ (i.e., the ratio between these two). Furthermore, for a given type of food, $R_{SDA}$ increases linearly with ration (e.g., Beamish 1974; Caulton 1978). The causal relationship behind this observation is likely between $R_{SDA}$ and the energy assimilation rate $A$ (i.e., ingesting nondigestable food should not influence $R_{SDA}$). Thus, for a given type of food, and given that $R_{SDA} = 0$ for a nonfeeding, nongrowing individual:

$$R_{SDA} = \gamma A, \tag{8}$$

where $\gamma$ is the proportion of energy in assimilated food expended for $R_{SDA}$. By substitution with equations (5), (7), and (8), equation (6) can be expressed as

$$E = R_A\left[\frac{p\beta(1 - \gamma)}{1 + \beta R_A} - (1 + q)\right]. \tag{9}$$

Thus, by viewing the evolution of RMR as an outcome of selection on $R_A$, this solves the problem of predicting the optimal RMR, which by itself only represents an energetic cost and hence cannot, when viewed in isolation, have an optimal value $> 0$ from an energetic perspective.

Previous models of energy budgets have had great success in describing patterns of allocation of energy to the

different metabolic components and biomass production throughout life (i.e., the ontogenetic growth model [e.g., West et al. 2001; Hou et al. 2008] and dynamic energy budget models [e.g., Nisbet et al. 2000; Sousa et al. 2010]). However, these approaches do not consider activity metabolism to be a potentially evolving trait that influences energy acquisition rates. Rather, energy acquisition is either determined by body size and food density (e.g., Nisbet et al. 2000) or is allowed to match metabolic demands (e.g., Hou et al. 2008). Furthermore, for dynamic energy budget models activity metabolism is not modeled explicitly but is included in the somatic maintenance term (Nisbet et al. 2000). Thus, although these models have allowed great advances in understanding patterns of energy allocation, it is not obvious how they can be used to obtain predictions of how optimal levels of metabolism should depend on food abundance. The approach used here and those previous ones should therefore be considered complementary rather than competing.

### Analytical Approach

To evaluate the effect of the different model parameters on the optimal activity metabolic rate I differentiate equation (9) with respect to $R_A$ and set this equal to 0:

$$\frac{dE}{dR_A} = \frac{p\beta(1-\gamma)}{\beta R_A + 1} - \frac{p\beta^2 R_A(1-\gamma)}{(\beta R_A + 1)^2} - 1 - q = 0. \quad (10)$$

Solving this equation yields one positive (and hence biologically relevant) solution for values of $R_A$ maximizing energy surplus acquisition rates:

$$R_A = \frac{\sqrt{p(1+q-\gamma-\gamma q)}}{\sqrt{\beta}(1+q)} - \frac{1}{\beta}. \quad (11)$$

Equation (11) can then be used to evaluate how the optimal $R_A$ changes with a change in the model parameters.

### Numerical Example

To visualize the results from this model I give a numerical example, focusing on the effect of changes in the parameter values of $\beta$, that is, the experienced prey encounter rate per unit of activity, on the optimal metabolism while keeping the remaining parameters constant. Values of $\beta$ were allowed to vary between 0.1 and 100. For $q$, considerations of data from across taxa suggest that RMR typically constitutes around 20%–50% of total metabolism (Hulbert and Else 2000), corresponding to the magnitude of RMR relative to $R_A + R_{SDA}$ to range from 25% to 100%. The magnitude of RMR relative to $R_A$, and hence the parameter $q$, would then be somewhat larger. Unfortunately, studies providing estimates for $R_A$ in ecologically relevant settings remain rare. Some studies provides such estimates based on heart rate telemetry (e.g., Lucas et al. 1993), but this method has its limitations and uncertainties (Thorarensen et al. 1996). Thus, for simplicity I set the value of $q = 1$, while acknowledging that the empirical basis for this is weak. The values of the two remaining parameters, $p$ and $\gamma$, are also set as constants. Although their values are likely to be influenced by digestive systems of organisms as well as the quality of the food, this is beyond the scope of this article. I therefore set $p = 0.75$ and $\gamma = 0.15$, which provides representative values for at least one taxon (i.e., fish: Elliott 1976*a*; Jobling 1981). For each parameter combination, equation (11) was used to calculate the optimal $R_A$, and equations (4) and (9) to calculate the corresponding food intake rates and energy surplus, respectively.

## Results

### Analytical Approach

Equation (11) shows that $\beta$ must be larger than some threshold value for the optimal $R_A$ to be positive and hence biologically relevant (corresponding to positive values of energy surplus acquisition; see eq. [9]). This threshold value can be found by setting the right-hand side of equation (11) equal to zero, yielding the solution $\beta = (q + 1)/p(1 - \gamma)$. Thus, the minimum value of $\beta$ is larger if $q$ (the ratio between RMR and $R_A$) or $\gamma$ (the proportion of assimilated energy used for $R_{SDA}$) is larger or if $p$ (the proportion of ingested energy that is assimilated) is smaller.

Equation (11) can also be used to identify the value of $\beta$ where the optimal $R_A$ is largest. This value can be found by differentiating the right-hand side of the equation (i.e., the expression for the optimal $R_A$) with respect to $\beta$ and setting it equal to zero, yielding the solution $\beta = (4q + 4)/p(1 - \gamma)$. Remembering that both $p$ and $\gamma$ per definition are constrained to take on values between zero and one and that $q$ is positive, this shows that the optimal $R_A$ is largest at an intermediate value of $\beta$. More specifically, the value of $\beta$ yielding the maximum optimal $R_A$ is larger if $q$ (the ratio between RMR and $R_A$) or $\gamma$ (the proportion of assimilated energy used for $R_{SDA}$) is larger or if $p$ (the proportion of ingested energy that is assimilated) is smaller.

Although the focus of this article is how the optimal $R_A$ changes with food abundance, equation (11) can also be used to evaluate how the optimal $R_A$ changes with changing values of the remaining parameters (i.e., $p$, $q$, and $\gamma$). It can be seen directly that increasing the value of $p$ or decreasing the value of $\gamma$ increases the optimal $R_A$. Furthermore, a given increase in $q$ will result in a larger increase

in the denominator than in the numerator (remembering $0 < \gamma < 1$), and hence cause a decrease in the optimal $R_A$.

According to equation (11), the parameters $q$ and $\gamma$ are structurally constrained in terms of their relative values in order to allow for an optimal value of $R_A$. Specifically, for the numerator in the first term to have a solution, the condition $q \geq (\gamma - 1)/(1 - \gamma)$ must be met. However, since $\gamma$ is defined to take on values between 0 and 1 (causing the right-hand side in the above expression to be negative), and $q$ is defined as a positive constant, this does not cause a constraint in a biological sense.

## Numerical Example

As expected, higher values of $\beta$, providing a more rapid increase in assimilation with increasing activity metabolism (fig. 1), also yields a higher energy surplus for a given metabolism (fig. 2). Equation (9) produces a dome-shaped relationship between activity metabolism and the energy surplus, $E$, such that an optimal metabolism at an intermediate value can be identified (fig. 2).

Figure 2 also suggests differences in optimal activity metabolism across values of $\beta$. This is presented more systematically in figure 3*A*. Below the threshold value of $\beta$ ($\beta = (q+1)/p(1 - \gamma)$) total metabolism will exceed assimilation independent of the level of activity metabolism, such that no positive optimal value can be obtained. As $\beta$ is increasing beyond this threshold, there is an initial increase in the optimal activity metabolism before it flattens out and then decreases. At the optimum activity metabolism, the corresponding food intake rate and energy surplus both increase with increasing values of $\beta$ (fig. 3*B*, 3*C*).

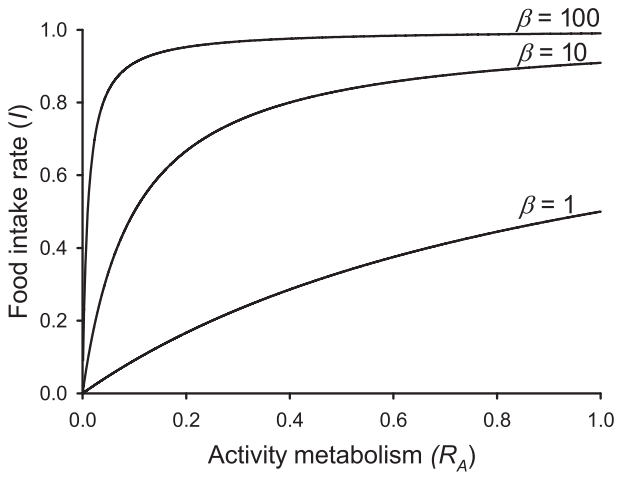


**Figure 1:** Modeled food intake rate as a function of activity metabolism according to equation (4) for different values of the parameter $\beta$.

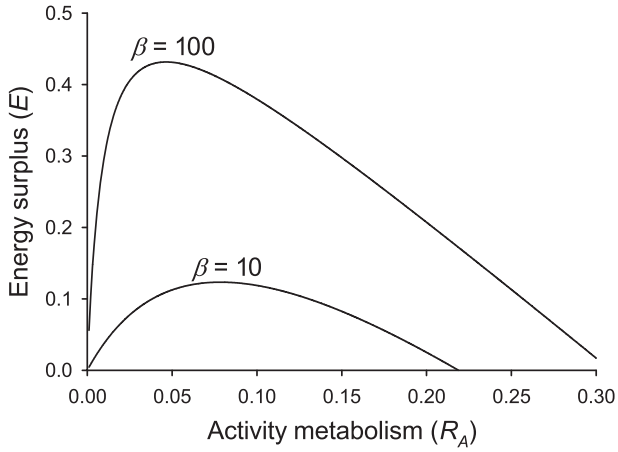


**Figure 2:** Modeled energy surplus as a function of activity metabolism according to equation (9) for two values of the parameter $\beta$.

## Discussion

In this study, I utilize the increasingly recognized relationship between RMR and activity metabolism to provide a modeling framework that can address how the optimal RMR varies as a response to food abundance. Rather than directly attempting to understand variation in RMR, which per se represents a fitness cost (and hence should always be minimized if viewed in isolation), population and species divergence in RMR can be considered to be a correlated response to diverging optima in metabolism allocated to activity. The main pattern predicted by the model was that the optimal activity metabolism ($R_A$), and hence RMR, is highest at an intermediate increase in energy assimilation with increasing activity (i.e., intermediate $\beta$). This suggests an important role for resource availability in shaping selection on RMR. The per capita food abundance, being determined by inherent characteristics of the environment as well as population density, will influence the $\beta$, which in turn influences the optimal RMR. A very low or very high food abundance causes the optimal RMR to be low compared to intermediate food abundance levels. This result contrasts with the previous hypothesis, supported by verbal arguments, that individuals with high RMR are at an advantage when environmental conditions are favorable (e.g., Biro and Stamps 2010; Burton et al. 2011). Intuitively, the results from this model can be understood by considering that as food abundance becomes sufficiently high, a lower activity (and hence RMR) is required to approach maximum assimilation.

Future tests of this model would require the quantifi-

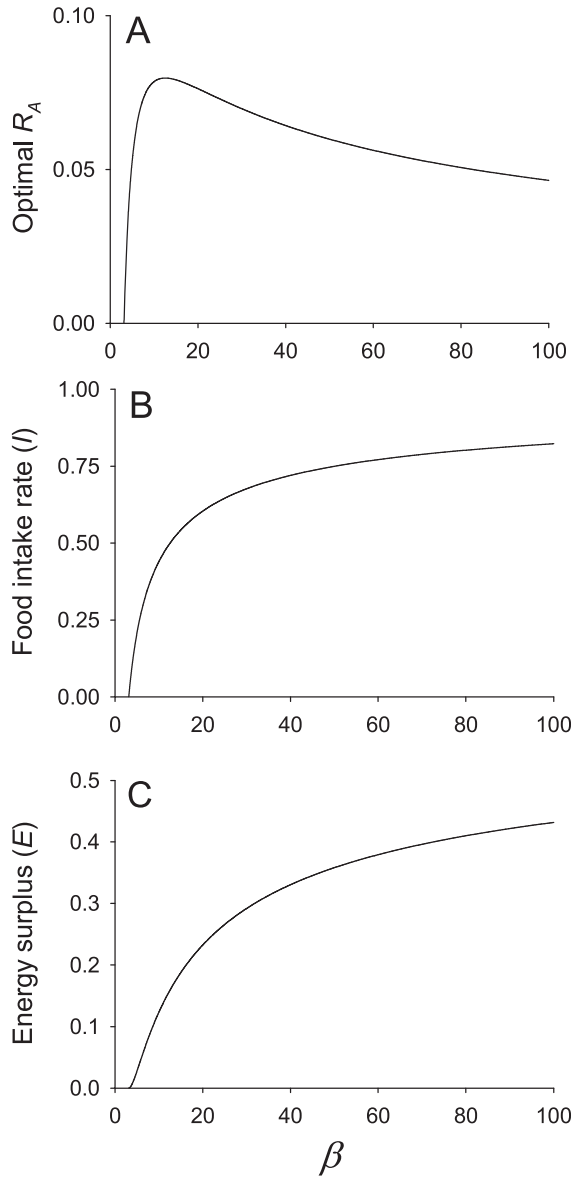


**Figure 3:** Relationship between the parameter $\beta$ and the optimal activity metabolism ($A$), the corresponding food intake rate ($B$), and energy surplus ($C$).

cation of effects of variation in RMR across a wide range of food abundances. Yet it may be useful to evaluate existing empirical data for two reasons. First, even though this model does not yield a simple prediction for the sign of a correlation between the optimal RMR and the value of $\beta$ (this will depend on the range of $\beta$ values considered) it does suggest that the optimum should vary with $\beta$. Second, the verbal model presented previously (e.g., Biro and Stamps 2010; Burton et al. 2011) hypothesizes a monotonic increase in the optimal RMR with increasing values of $\beta$, and this may be easier to reject based on qualitative observations. Specifically, fitness effects of metabolic rate can be estimated at a very low or high value of $\beta$, such that most individuals would be expected to have a RMR that is above or below the optimum. The previous verbal model then predicts that metabolic rate is negatively correlated with growth under low food conditions (i.e., small optimum when $\beta$ is small) and positively under high food conditions. In a recent review, Burton et al. (2011) identified studies quantifying the relationship between RMR and fitness-related traits. Different studies typically focus on different components of fitness (growth, survival, reproduction, senescence). For comparisons with this model, studies focusing on growth are most relevant. On first inspection of empirical data they appear to be consistent with the verbal model predictions; four of the five studies reporting relationships under what were classified as ad lib. under controlled conditions showed a positive relation between metabolic rate and growth. However, one of these (on zebra finch *Taeniopygia guttata*; Mathot et al. 2009) was in fact not conducted under ad lib. feeding conditions but rather by providing food resulting in a "neutral energy budget" (i.e., close to zero growth). Furthermore, the remaining three studies (Yamamoto et al. 1998; McCarthy 2000; Alvarez and Nicieza 2005) were conducted on groups of juvenile salmonid fish (*Salmo* sp.), which are territorial and show high levels of intraspecific aggressiveness and where social status is highly dependent on metabolic rate (Metcalfe et al. 1995; Yamamoto et al. 1998). In such species, it is difficult to provide ad lib. feeding conditions in groups of individuals, because high metabolic rates are likely to provide benefits in terms of defense of favorable feeding locations even when food is abundant. This may be particularly true in laboratory studies such as these where food is introduced at a specific predictable location within a tank or a stream channel. A subsequent study of Atlantic salmon (*Salmo salar*) juveniles in seminatural streams illustrates this problem. Reid et al. (2012) showed that somatic growth rate is positively related to metabolic rate under a predictable spatial variation in food abundance. However, when the spatial variation in food abundance was made temporally unpredictable, the advantage of a high metabolic rate disappeared, likely due to a decreased effect of social status on the quality of occupied territories (Reid et al. 2012). Data from wild populations regarding the interaction between metabolic rate and environmental conditions on growth rates are lacking. A recent study on chipmunks (*Tamias striatus*) demonstrated a positive relationship between metabolic rate and somatic growth during a year of low food abundance (Careau et al. 2013). This appears to be inconsistent with the verbal model, which would predict that individuals in such a year would have metabolic rates above the optimum, and with

individuals with higher metabolic rates being further from the optimum. Thus, empirical data on the relationship between metabolism and somatic growth do not provide strong support to the hypothesis that there is a continuous increase in the optimal metabolism with increasing environmental quality.

### *Model Applicability*

In this first optimality model for metabolic rate, I focus on optimality under temporally stable environmental conditions. In the absence of such variation the only relevant parameter spaces to evaluate are those that provide a positive net energy intake, whereas under a variable environment populations may persist under periods of negative energy intakes. If populations experiencing a mean (i.e., over time) low food abundance are more likely to undergo periods of extreme food limitation that cause starvation mortality, one may expect that this will favor individuals with a low metabolism (see discussion above in relation to starvation experiments). It has been argued that large-scale patterns in metabolic rates across mammals provide support for such a role of environmental predictability (Lovegrove 2000). On the other hand, it may be of particular importance for organisms in such environments to take advantage of periods of higher food abundance, enabling them to build up energy reserves that allow them to sustain periods of food shortage (Finstad et al. 2004). Thus, verbal models for selection under temporally variable conditions are just as fraught with problematic simplifications as those proposed for stable conditions. In a recent examination of interspecific patterns, Kooijman (2013) suggested that species with a high somatic maintenance cost (i.e., high RMR) typically live in environments that are temporally variable in their food abundance. This enables them to benefit from periods of high food abundance by expressing a high population growth rate, and is consistent with predictions from the dynamic energy budget theory. However, this only applies to the situation where the period of high food abundance lasts for several generations. Furthermore, most of the high RMR species identified by Kooijman (e.g., copepods, cladocerans) are able to survive periods of food shortage by switching to some resting stage, which is not an option for many other organisms. It therefore remains unknown how the optimal RMR changes with unpredictability in food supply, and mechanistic models should be developed for dealing with such situations. However, this is not straightforward, particularly in terms of environmental variation occurring over a short timescale (i.e., within generation), due to the ability of organisms to adjust their metabolism in response to such variation (Wang et al. 2006).

In this model, I also make a number of simplifying assumptions. First, the model assumes that evolution acts to maximize the rate with which surplus of energy can be converted into somatic growth and reproductive output ($E$ in eq. [9]). This may be considered simplistic in the way that it lacks an incorporation of effects of metabolism on age and size at maturation. An evolutionary change in metabolism and corresponding effects on energy surplus acquisition will have correlated effects on such other phenotypic traits. The mechanistic basis for this is well explained by previous energy budget models (Nisbet et al. 2000; West et al. 2001; Hou et al. 2008; Sousa et al. 2010). It is these correlated responses that would be expected to cause fitness effects, not energy surplus acquisition per se. However, by increasing energy surplus acquisition rates (through evolution of the optimal metabolism), organisms would necessarily either mature at a lower age (causing lower juvenile mortality) or a larger size (if maturation age does not change, causing larger potential fecundity). Thus, while the model does not include these correlated responses explicitly, the fitness consequences of metabolism implicitly result from them.

A second assumption that requires mentioning is the fact that having a high metabolic rate may impose selection not considered here due to predation or oxidative stress. A high metabolism is generally believed to increase the production of reactive oxygen species and hence increase oxidative stress in cells, causing damages to DNA (including telomere abrasion), RNA and other macromolecules (reviewed by Jennings et al. [2000]; Speakman [2005]), which is likely contributing to the negative effects of having a high growth rate on longevity (Lee et al. 2013).

A third simplifying assumption in the model is that handling time was set to be a constant. This assumes that the handling time is not (or to a very limited extent) influenced by $R_A$. To my knowledge, no empirical data exist to evaluate this, but it may be likely that this is an unrealistic assumption for situations where the prey is large relative to the body size of the consumer, and hence a considerable amount of work is devoted to handling. Thus, the results should be considered with care under such situations.

In conclusion, this model provides a first theoretical framework for understanding variation in selection for RMR across environments. The most important component of the model is a genetic correlation between RMR and metabolism used for activity. Using this model, I predict that there is a dome-shaped relationship between food abundance and the optimal RMR. This contrasts with the commonly expressed hypothesis that the optimal RMR should increase continuously with increasing food abundance (e.g., Biro and Stamps 2010; Burton et al. 2011). Future empirical studies should test these alternative mod-

els, and they can most easily be distinguished within the range of high to very high (ad lib.) food abundances, where they make qualitatively different predictions. Further theoretical developments are required to predict how the optimal RMR is influenced by temporal environmental variation in food abundance.

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