Theoretical Population Biology **117** *(2017) 23–42*
doi:10.1016/j.tpb.2017.08.005
a Malthusian Relativity paper

$\iota^{**}=7/3\psi$
mrLife.org

The natural selection of metabolism and mass selects allometric transitions from prokaryotes to mammals

Lars Witting
Greenland Institute of Natural Resources, Box 570, DK-3900 Nuuk, Greenland

Abstract The exponents of inter-specific allometries for several life history (metabolism, lifespan, reproductive rate, survival) and ecological (population density, home range) traits may evolve from the spatial dimensionality (d) of the intra-specific interactive competition that selects net assimilated energy into mass, with 1/4 exponents being the two-dimensional (2D) case of the more general $1/2d$ (Witting 1995). While the exponents for mass-specific metabolism cluster around the predicted -1/4 and -1/6 in terrestrial and pelagic vertebrates, the allometries of mobile organisms are more diverse than the prediction. An exponent around zero has been reported for protists and protozoa (Makarieva et al. 2005, 2008), and the exponent appears to be strongly positive in prokaryotes with a value of about 5/6 (DeLong et al. 2010).

I show that the natural selection of metabolism and mass is sufficient to explain exponents for mass-specific metabolism that decline from 5/6 over zero to -1/6 in 3D, and from 3/4 over zero to -1/4 in 2D. These results suggest that mass-specific metabolism is selected as the pace of the resource handling that generates net energy for self-replication and the selection of mass, with the decline in the metabolic exponent following from a decline in the importance of mass-specific metabolism for the selection of mass. The body mass variation in prokaryotes is found to be selected from primary variation in mass-specific metabolism, while the variation in multicellular animals is selected from primary variation in the handling and/or densities of the underlying resources, with protists and protozoa being selected as an intermediate lifeform.

Keywords: Evolution, metabolism, body mass, life history, allometry, major transitions

1 Introduction

It is difficult to overestimate the evolutionary importance of body mass allometries as they reveal the joint evolution of the life history with mass across the tree of life. The most well-known allometry is Kleiber (1932) scaling in multicellular animals, with a negative 1/4 exponent for the dependence of mass-specific metabolism on mass. Yet, the real value, or rather values, of the exponent is still debated (e.g., McNab 2008; White et al. 2009; Isaac and Carbone 2010), and it is also uncertain whether the relationship between the two traits is a straight allometric line or a slightly bent curve (Hayssen and Lacy 1985; Dodds et al. 2001; Packard and Birchard 2008; Kolokotrones et al. 2010; Deeds 2011; Ehnes et al. 2011; MacKay 2011).

A value around $-1/4$ is though in general agreement with the average exponent for the basal (BMR) and field (FMR) metabolic rates across a wide range of vertebrates (e.g., Peters 1983; Savage et al. 2004; Glazier 2005; Duncan et al. 2007; Kabat et al. 2008; Capellini et al. 2010), and the exponents for BMR and FMR are statistically indistinguishable in most lineages of mammals (Capellini et al. 2010). It is therefore reasonable to assume that a value close to -1/4 is acting as an attractor for the natural selection of the allometric relationship in many taxa, and that the value may vary somewhat with variation in the underlying mechanism of natural selection.

It is important to recall that the metabolic allometry is only one of several essential allometries, with the empirical exponents in many studies approximating 1/4 for lifespan and reproductive periods, $-1/4$ for the rate of exponential population growth, $-3/4$ for the density of populations, and 1 for the area of the home range (Bonner 1965; Schoener 1968; Turner et al. 1969; Fenchel 1974; Damuth 1981, 1987; Peters 1983; Calder 1984). This existence of related exponents across several traits indicates that the evolution of allometries may be determined by a selection that involves not only a variety of life history traits, but also ecological traits like the density of the population and the home range of its individuals.

Allometries, however, are often studied for a single trait only, and the correlation between metabolism and mass is not only the most studied allometry empirically, it is also the most studied theoretically (reviewed by e.g. Glazier 2005; White and Kearney 2013). The widespread view has seen the allometry as a consequence of the physiology, where a physiological optimisation of metabolism in relation to resource trans-

portation networks is the cause of the metabolic exponent (e.g. West et al. 1997, 1999a,b; Banavar et al. 1999; Dodds et al. 2001; Dreyer and Puzio 2001; Rau 2002; Santillán 2003; Glazier 2010). This explanation, however, suffers from a circular argument of contingency (Witting 1997, 2008): as the overall physiology is optimised by natural selection it is *in principle* impossible to infer evolutionary causality from an observed physiological correlation. While it might be the metabolic exponent that evolves from the constraints of the evolved resource transportation network, it might just as well be the transportation network that is optimised to follow a metabolic allometry that reflects the primary selection of metabolism and mass (Witting 1998).

The physiological hypothesis is also insufficient from another natural selection point of view, as it does not show how the primary selection of metabolism and mass is causing the evolution of the allometric relation between the two traits, and nor does it select the allometric exponents of other traits. Even if the allometric relation is seem to be naturally selected by a physiological optimisation that operates relatively independently of the natural selection of mass, it is essential to show that the allometric hypothesis is consistent with the natural selection of mass. But the selection of mass that has been connected with the physiological cause for the metabolic allometry (Brown and Sibly 2006) is generally inconsistent with the inter-specific allometries of other traits. The assumed frequency-independent selection selects for an increase in average fitness [as given by the rate of exponential increase in population (r), or by the carrying capacity (K, denoted n^* in this paper)] with an increase in mass, but r and K are typically declining with mass across natural species (see Witting 2017 for more details).

A more parsimonious explanation, where allometric exponents are selected by the natural selection of mass, was proposed as the theory of Malthusian Relativity prior to the physiological hypothesis (Witting 1995, 1997). This mass selection hypothesis is parsimonious is the sense that it does not involve separate selection hypotheses for the selection of mass and the selection of allometries. The hypothesis assumes energy conservation and density-dependent population growth, where individuals meet more frequently in interactive competition as the abundance of the population increases. The interactive competition produces a density-frequency-dependent bias in resource assimilation in favour of the larger and competitively superior individuals, and this generates a population dynamic feed-back selection that selects the net energy that is obtained by foraging into non-negligible body masses. The assimilated net energy that is obtained by foraging is the essential component that drives the population dynamic feed-back selection of mass, with all of the above-mentioned allometric exponents following from the geometrical constraints (spatial dimensionality) of a foraging process that is optimised for a trade-off between the local resource exploitation of the individual and the density-dependent interactive competition between the individuals in the population.

With the predicted exponents following directly from a selection that is imposed by population growth and the conservation of energy, the mass selection hypothesis is deterministic in the sense that it avoids the circularity of contingent arguments (see review by Witting 2008). There is no chicken and egg dilemma, where we somewhat arbitrary need to choose one component of the phenotype (like the metabolic exponents) as an evolutionary function of another component (e.g., resource transportation networks). Apart from the life history constraints from population growth and energy conservation, and some initial conditions that does not affect the prediction, the mass selection model includes only the phenotypic properties that evolve directly from the natural selection of mass. And with the predicted exponents following from the ecological geometry of foraging, their values are dependent on the spatial dimensionality (d) of foraging, with $1/4$ being the two-dimensional case of the more general $1/2d$. The $1/4$ value is replaced by $1/6$ for species that forage and interact in three dimensions, with a $1/4 \rightarrow 1/6$ like transition being observed quite commonly between terrestrial and pelagic animals (Witting 1995, 1997).

The observed metabolic exponent is though more diverse than the predictions of the ecological mass selection model. The empirical exponent varies at least to some degree with mass (Kolokotrones et al. 2010), among major taxa and phylogenetic lineages (Peters 1983; Glazier 2005; Duncan et al. 2007; White et al. 2007a,b, 2009; Sieg et al. 2009; Capellini et al. 2010), and it is also dependent on the activity level of individuals (Darveau et al. 2002; Weibel et al. 2004; Glazier 2005, 2008, 2009; Niven and Scharlemann 2005; White et al. 2007).

More recent studies have found that the exponent tends to change across the tree of life. Instead of being negative, it is strongly positive in prokaryotes with an apparent value around 5/6 (DeLong et al. 2010), and it has been reported to be zero in protozoa (Makarieva et al. 2008; DeLong et al. 2010) and on the macro evolutionary scale across all non-sessile organisms (Makarieva et al. 2005, 2008; Kiørboe and Hirst 2014).

This variation is not explained by Witting (1995, 1997) where the selection of mass is dealt with independently of the primary selection of mass-specific metabolism. I extend the mass selection model with primary selection on mass-specific metabolism to examine if the joint selection of metabolism and mass will explain the wider set of allometries that is observed across the tree of life.

2 Basic selection relations

The proposed model is developed to explain the evolution of metabolism, mass, and exponents of inter-specific allometries as they are selected by the intra-specific interactive ecology between individuals in populations. The basic assumptions are the conservation of energy, the demography of age-structure, and the unfolding of interactive competition from the density-dependent growth of the population. Deviations in the interactive ecology from the assumed may select for alternative allometries, but this is not studied directly in the paper.

I will not attempt to explain absolute trait values, but only the selection response of the life history and the ecology to the primary selection of mass-specific metabolism and mass. This is done by two processes that I refer to as metabolic-rescaling and mass-rescaling selection. Metabolic-rescaling is associated with the primary selection of metabolism that generates net energy for the selection of mass, and mass-rescaling is the selection response of the life history and the ecology to the evolutionary changes in mass. Both selection responses are described by the first partial derivatives of the evolving traits with respect to the selected changes in metabolism and mass, with the integrals over mass being the inter-specific body mass allometries.

This level of explanation resembles the Newtonian tradition in physics, where we can explain the acceleration of an object, but not its absolute speed, from the action of a force. In order to use the proposed model to "predict" e.g. an absolute rate of reproduction, we will have to include an observed survival rate as an assumption. The explanation is then no longer deterministic, but contingent upon the observed life history.

To show that the allometric exponents follow from the primary selection of metabolism and mass I define all the exponents with unknown values. The allometries with unknown exponents are then included in a mathematical formulation of the selection process on metabolism and mass, with the resulting equations being solved to obtain the predicted values of the allometric exponents. This requires a detailed and consistent description of the essential life history energetics and ecological geometry of density regulation, i.e., of the density-dependent foraging and interactive behaviour in spatial dimensions.

These descriptions are relatively straight forward and they do not include new concepts. But they are nevertheless required to be formulated in a consistent way. For this I will not only refer to my earlier work, but formulate a complete model in the present paper and its appendices, with the essential (but not all) model symbols being explained in Table 1. I start in Section 2.1 with the new component that is included in the theory, i.e., mass-specific metabolism as a primary life history trait. The life history demography and density regulation are described in detail in Appendix A and B, with the relations that are necessary for the allometric deduction being included in Sections 2.2 and 2.3 of the main paper. A short section on the overall relationship between the primary selection of metabolism, net energy and mass is then following, before the deduction of the exponents of body mass allometries in Section 3.

2.1 Metabolism, net energy, and time

Being perhaps the most essential part of the physiology, metabolism is likely to be selected as a primary life history trait. This may reflect a metabolism that defines the pace of resource assimilation and energy use, as assumed in several publications (e.g., Calder 1984; Brown et al. 2004; Sibly et al. 2012; Humphries and McCann 2014; Padfield et al. 2016). As such a measure of joint biological activity, metabolism is highly dynamic, being dependent among others on chemistry, temperature, physiology, tissue maintenance and behaviour, with some of these components being controlled in part by ecological interactions and the age, sexual and informational state of the organism. Yet, for the selection model developed in this paper, all of this variation is integrated into a single measure of the average field metabolic rate per unit body mass.

Metabolism is transformed into pace by the biochemical, physiological and ecological work that is carried out by mass-specific metabolism. Let us therefore define metabolic pace

$$\tilde{\beta} = \beta / W \tag{1}$$

as the frequency (SI unit 1/s) of the mass-specific work ($W = 1\text{J/J}$) of one joule metabolised per unit body mass (with mass measured as combustion energy), with β (SI unit J/Js) being mass-specific metabolism, i.e., the field metabolic rate per unit mass.

S	SI	Basic relations	Description
w	J	$\frac{\partial \ln w}{\partial \ln \epsilon} = 1/\hat{\epsilon}$	Body mass of individual in joule (combustion energy).
$\ln w$	-	$\ln w = \ln[w/(1J)]$	Natural logarithm of mass.
β	J/Js	$\beta \propto \beta_\beta \beta_w$	Mass-specific metabolism; β_β:primary selected; β_w:mass-rescaling selected.
$\tilde{\beta}$	1/s	$\tilde{\beta} = \beta/W$	Metabolic pace in physical time.
W	J/J	W=1J/J	Mass specific work of one joule metabolised per unit mass.
x	-	$x = x_\circ w^{\hat{x}},\ \hat{x} = \hat{x}_\beta + \hat{x}_w$	Inter-specific allometry for trait x; $x_\circ$:intercept; $\hat{x}$:exponent.
x_β	-	$x_\beta = w^{\hat{x}_\beta}$	Metabolic-rescaling allometry (inter-specific).
x_w	-	$x_w = w^{\hat{x}_w}$	Mass-rescaling allometry (inter-specific).
t	s		Physical time.
τ	G	$\tau = t/t_g$	Biotic time, in generations (G).
t_x	s	$t_x = \tau_x t_g,\ x{:}l, g, m, j, r$	l:lifespan, g:generation, m:maturity, j:juvenile & r:reproductive period.
τ_x	G	$\tau_x = t_x/t_g,\ x{:}l, g, m, j, r$	l:lifespan, g:generation, m:maturity, j:juvenile & r:reproductive period.
ρ	J/m^d	$\rho = f\rho_u$	Realised resource per unit d dimensional habitat. ρ_u:unexploited resource.
f	-	$f = f_e f_\iota f_s$	Density regulation by exploitation (f_e), interference (f_ι) & self-inhibition (f_s).
α	J	$\alpha = \grave{\alpha}\rho^{**}$	Handling of net resource assimilation. $\grave{\alpha}$:intrinsic handling (Jm^d/J).
$\tilde{\alpha}$	1/s	$\tilde{\alpha}^{**} = \tilde{\beta}$	Pace of resource handling; selected to resemble metabolic pace.
ϵ	J/s	$\epsilon = \alpha\tilde{\alpha} = \alpha\tilde{\beta}$	Net assimilated energy (energetic state) per individual per unit t time.
ϵ_g	J/s	$\epsilon_g = \epsilon + \beta w$	Gross assimilated energy per individual per unit t time.
r_x	1/G	$r_x = \frac{d \ln x}{d\tau},\ x{:}\alpha, \beta_\beta, \epsilon$	Per-generation exponential increase in α, β_β & ϵ. $r_\epsilon = r_\alpha + r_{\beta_\beta}$.
p	-	$p = R_0/R$	Probability to survive to reproduce.
m	1/s	$m = \epsilon/\acute{\beta} w$	Reproductive rate in physical time.
R	-	$R = t_r m,\ R^* = 1/p^*$	Lifetime reproduction.
R_0	-	$R_0 = pR$	Expected lifetime reproduction.
λ	-	$\lambda = pR,\ \lambda^* = 1$	Population growth; per-generation multiplication factor.
r	1/G	$r = \ln\lambda = \frac{d \ln n}{d\tau},\ r^* = 0$	Population growth; per-generation exponential increase.
$\acute{\beta}$	-	$\acute{\beta} = 1 + \acute{w}_j \acute{\beta}_j / \acute{w}$	Invariant scaling of reproduction to account for offspring metabolism.
β_j	J/Js	$\acute{\beta_j} = \beta_j/\beta$	Average mass-specific metabolism of offspring during the juvenile period t_j.
w_j	J	$\acute{w_j} = w_j/w$	Average mass of offspring during t_j.
$\dot{w}$	J/s	$\acute{\dot{w}} = \dot{w}/w\beta$	Average ontogenetic growth during t_j.
d	-		Spatial habitat dimensions for interactive foraging behaviour. 1D, 2D & 3D.
n	1/m^d		Population density; individuals per unit d dimensional habitat.
I	1/s		Intra-specific interference; competitive encounters per individual per unit t time.
ι	-	$\iota^{**} = \frac{4d-1}{2d-1}\frac{1}{\psi},\ \bar{\iota}^{**} = \frac{1}{\psi}$	Log intra-specific interference, $\iota = \ln I$. $\bar{\iota}^*$:mass dependent maximum.
ψ	-		Fitness cost gradient per unit interference across body mass variants.
h	m^d		Home range of individual in d habitat dimensions.
v	m/s	$v = v_\circ \beta_\beta w^{\hat{v}},\ \hat{v} = \hat{t}$	Foraging speed of individual in physical time.
$\sigma^2_{\ln w}$	-		Additive heritable variance of a trait, here w on log scale.

Relation	Script	
Modification	Accent	$\tilde{x}$:pace of x; $\acute{x}$:relative, fractional, or conglomerate x; $\grave{x}$:intrinsic x; $\dot{x}$:dx/dt.
Allometry	Subscript/accent	β:metabolic-rescaling; w:mass-rescaling; $\circ$:intercept; $\hat{x}$:$w^{\hat{x}}$ exponent; $\check{x}$:$\beta^{\check{x}}$ exp.
Density regulation	Subscript	e:exploitation; ι:interference; s:local exploitation; u:unregulated; $\circ$:intercept.
Life periods/ages	Subscript	j:juvenile; m:maturity; r:reproduction; g:generation; l:lifespan.
Attractors	Superscript	$*$:population dynamic equilibrium; $**$:selection attractor.
Constraints	Bar	$\underline{x}$:lower limit on x; $\overline{x}$:upper limit on x.

Table 1: **Important symbols (S)** with SI units and basic relationships, including the interpretation of scripts and accents.

It is not, at least not in principle, absolutely certain that the work of eqn 1 is essential for the organism. If organism metabolism, contrary to our expectations, would evolve by neutral drift instead of by natural selection, the majority of the metabolism would be irrelevant for the ecological and physiological functioning of the organism. But it is fitness costly to burn energy in metabolism, and we do therefore expect that metabolism is optimised by natural selection to reflect the physiological and ecological work that is essential for the selected life history. While a perfect metabolic efficiency is impossible to obtain, I will quite generally assume that the energetics of the physiology is optimised by natural selection. This allows me to study life history evolution as it can be selected along an optimal fitness ridge that is defined by a physiology that is optimised by natural selection, assuming implicitly that optimal physiology is invariant of mass.

To aim for a natural selection explanation for the metabolic work, rate and pace, consider the net energy of the organism (ϵ, SI unit J/s) to be the energy that is available for self-replication per unit time. This energy is the net energy that is assimilated from resources, defined in physical time as a product

$$\epsilon = \alpha\tilde{\alpha} \tag{2}$$

between the ecological/physiological mechanical/biochemical handling of resource assimilation (α, resource handling in short, SI unit J), and the pace ($\tilde{\alpha}$, SI unit 1/s) of this process.

Let resource handling be defined by the work (W) that is carried out by one joule metabolised for handling per unit mass. The pace of handling $\tilde{\alpha} = \acute{\epsilon}_\alpha/W$ is thus proportional to the mass-specific energy ($\acute{\epsilon}_\alpha = \epsilon_\alpha/w$, SI unit J/Js; ϵ_α:total handling energy, SI unit J/s; w:body mass, SI unit J) that is used for handling per unit time. This energy is provided by metabolism $\acute{\epsilon}_\alpha = c\beta$ as the fraction ($0 \leq c \leq 1$) of mass-specific metabolism that is used for the handling of net resource assimilation, with $\tilde{\alpha} = c\beta/W = c\tilde{\beta}$. One, maybe unrealistic, but nevertheless potential example is invariance between handling speed and mass-specific metabolism ($c = c_0/\beta$, with $\tilde{\alpha} = c_0/W$ and c_0 being a constant). This is expected if a fixed amount of energy is used per unit mass on the handling of the resource, while the metabolic rate is evolving by other means.

To examine the natural selection of the relationship between metabolism and handling speed, consider net energy (ϵ) as the difference between gross energy (ϵ_g) and the total energetic cost of the metabolism of the individual ($w\beta$), i.e.,

$$\begin{aligned} \epsilon &= \epsilon_g - w\beta \\ &= \alpha_g\tilde{\alpha} - w\beta \\ &= \alpha_g c\beta/W - w\beta, \end{aligned} \tag{3}$$

where α_g (SI unit J) is the resource handling component that generates gross energy.

As natural selection favours an increase in ϵ (eqn 16), we find from the positive partial derivative $\partial\epsilon/\partial c = \alpha_g\beta/W$ that there is selection for a pace of handling that is as large as possible, with the evolutionary equilibrium (denoted by superscript $**$) being $c^{**} = 1$ with $\tilde{\alpha} = \tilde{\beta}$. This suggests that the primary function of metabolism (as defined by natural selection) is to enhance the net energy of the organism, with mass-specific metabolism being selected as a proxy for the pace of resource handling, with

$$\epsilon = \alpha_g\tilde{\beta} - w\beta = \alpha\tilde{\beta}, \tag{4}$$

where $\alpha = \alpha_g - wW$ and $\acute{\epsilon}_\alpha = \beta$.

The $w\beta$ difference between gross and net energy is the metabolic work that drives the gross assimilation of energy from the resource. Increasing the speed of handling requires more metabolic work, and eqn 4 defines that the metabolic work is selected to be paid back by a proportional increase in the net energy that is available for replication. As formulated here, the total metabolic cost ($w\beta$) includes not only the energy that is used directly in resource handling, but also the indirect energy of *i*) the catabolism and anabolism that goes into tissue maintenance, *ii*) other processes that are required to keep the organism running under the given ecological and physiological conditions, and *iii*) energy that is lost because metabolism is not 100% efficient in the transformation of energy.

With mass-specific metabolism being selected as the pace of the biochemical, physiological and ecological processes of resource assimilation, we may follow Pearl (1928) and others (Brody 1945; Hill 1950; Stahl 1962; Calder 1984) and extend the inverse of mass-specific metabolism to a general concept of biological time. This is straight forward for the physiological and behavioural processes that are defined more or less directly by metabolic processes. And with the energetic mass of the individual being formed by the anabolic metabolism that synthesises the organism from smaller molecules during ontogenetic growth (Brody 1945; Kleiber 1961; Kooijman 2000; West et al. 2001; Ricklefs 2003; Makarieva et al. 2004; Hou et al. 2008), the inverse dependence is extended to include the juvenile period where the individual is growing ($t_j = 1/\beta\acute{w}$; SI unit s; $\acute{w}$:the average growth of mass in joules relative to the mass and mass-specific metabolism of the

adult; see Appendix A.1). And with no delayed maturity, the age of maturity ($t_m = t_j$) is equivalent to the length of the juvenile period. The potential reproductive period (t_r), lifespan ($t_l = t_r + t_m$), and generation length (t_g) are also expected to have an inverse dependence on mass-specific metabolism, as the deterioration (senescence) of the organism depends, among others, on biochemical use.

Mass-specific metabolism is thus synchronising biological processes over time-scales from the metabolism of the physiology, over the ecological feeding behaviour and life history periods, to the population dynamic and natural selection that operate on the per-generation time-scale. Hence, we obtain a biological time in generations

$$\tau = t/t_g \propto t\beta, \tag{5}$$

which is running in proportion with mass-specific metabolism, with t being physical time (SI unit s). This relation does not only reflect an expected invariance that follows from a biotic time that is defined from metabolic pace; it is also a relation that evolves directly from the mass-rescaling selection of the life history (Section 3.1).

2.2 Life history

The age-structured life history with complete parental investment is described in Appendix A. An essential component is the total energetic investment in each offspring

$$\epsilon_j t_j = w + w_j t_j \beta \acute{\beta}_j = w(1 + \acute{w}_j \acute{\beta}_j / \acute{w}) = \acute{\beta} w, \tag{6}$$

which is the final adult mass w of the offspring at the age of independence (t_m), plus the energy $w_j t_j \beta \acute{\beta}_j$ that is metabolised by the offspring during the juvenile period (t_j), with ϵ_j being the energetic investment in the offspring per unit physical time, w_j the average juvenile mass during t_j with $\acute{w}_j = w_j/w$, $\acute{\beta}_j = \beta_j/\beta$ the average mass-specific metabolism of the offspring relative to that of an adult, and

$$\acute{\beta} = 1 + \acute{w}_j \acute{\beta}_j / \acute{w} \tag{7}$$

an invariant scaling that accounts for the energy that is metabolised by the offspring during t_j. The total investment in each offspring is thus proportional to mass and independent of mass-specific metabolism.

This investment implies an energetic quality-quantity trade-off (Smith and Fretwell 1974; Stearns 1992), where the reproductive rate

$$m = \epsilon / \acute{\beta} w \tag{8}$$

is inversely related to the energetic investment in each offspring. As there seems to be no simple way to quantify the absolute dependence of survival on net energy, I assume implicitly that ϵ relates to a physiology where the energetics of survival is already optimised by natural selection. Total lifetime reproduction

$$R = \frac{t_r}{t_j} \frac{\epsilon}{\epsilon_j} \tag{9}$$

is then, as a consequence of this (eqn 66), described by the product between the ratio of the reproductive period over the juvenile period (t_r/t_j) and the ratio of net energy over the energy that is allocated to an offspring (ϵ/ϵ_j).

The last life history constraint to be considered is the probability that a newborn will survive and reproduce

$$p = \frac{R_0}{R} = \frac{1}{t_r} \int_{t_r} l_t \, \mathrm{d}t, \tag{10}$$

where $l_t = \prod_0^{t-1} p_t$ is the probability that an individual will survive to age t, with p_t being the probability to survive from age t to age $t+1$. When l_t is invariant in biotic time, i.e., when l_{t/t_r} is invariant, it follows that the $\int_{t_r} l_t \, \mathrm{d}t$ integral reduces to $c_l t_r$ where c_l, and thus also p, are invariant parameters. This is expected if survival is determined entirely by intrinsic processes.

2.3 Optimal density regulation

The first density regulation constraint that is necessary for the allometric deduction is the adjustment of the life history to a population dynamic equilibrium (denoted by superscript $*$), where the per-generation replication

$$\lambda^* = pR = p\, t_r \epsilon / \acute{\beta} w = 1 \tag{11}$$

of the average reproducing unit in the population is one, where $\epsilon = \epsilon_u f^*$ with $0 < f^* < 1$ being a multiplicative density regulation at equilibrium, and ϵ_u the unregulated net energy.

A more essential constraint for the numerical values of the allometric exponents comes from the ecological geometry of the density regulated foraging that is optimised by natural selection. This was first shown by Witting (1995), and a more general version of his model is described in Appendix B.

The essential ecological geometry reflects a population where the individuals forage in home ranges that are spread out in one, two or three spatial dimensions. The arguments of the density regulation functions are thus reflecting, not only the density (n) of the population, but also the spatial dimensionality (d) of the ecology, and population traits like the average body mass

(w), home range (h), mass-specific metabolism (β), and foraging speed

$$v = v_\circ \beta_\beta t_w, \tag{12}$$

with the latter being defined by the primary selected mass-specific metabolism (β_β where $\beta = \beta_\circ \beta_\beta \beta_w$; β_w:mass-rescaling component; $\beta_\circ$:invariant initial condition), the mass-rescaling component for the length of biotic time-steps in physical time (t_w), and an invariant initial condition ($v_\circ$, see Appendix B for details).

The overall multiplicative regulation

$$f = f_e(nw\beta)\ f_\iota(nvh^{\frac{d-1}{d}})\ f_s(\beta h^{1/d}/v) \tag{13}$$

follows from regulation by exploitative competition (f_e), interactive (interference) competition (f_ι), and foraging self-inhibition (f_s) as caused by the local resource exploitation of the individual. The arguments of these regulation functions reflect the gross energy consumption of the population for f_e, the frequency of interactive encounters per individual in physical time ($e^\iota = I$) for f_ι, and the time interval between the individuals reuse of foraging tracks for f_s (see Appendix B for details).

Regulation by the cost of interference is increasing with an increase in the average home range (the f_ι function declines with an upward bend; Fig. 1, curve a), while regulation by the cost of foraging self-inhibition is declining (the f_s function increases with a downward bend; Fig. 1, curve b). There is thus an optimal home range, where the joint regulation by interference and self-inhibition is minimal (Fig. 1, optimum of c). The mathematical formulation

$$h^{**} \propto \left(\frac{v}{\beta}\right)^d \tag{14}$$

of the selection optimum follows from eqn 95, and it implies that the overall regulation at the population dynamic equilibrium is body mass invariant across the selected life histories [$f^* = f_e^* f_\iota^* f_s^* \propto w^0$]. This implies natural selection for a covariance

$$nvh^{\frac{d-1}{d}} \propto \beta h^{1/d}/v \propto nw\beta \propto w^0 \tag{15}$$

that will leave the regulation optimum, and the level of interference competition in the population, invariant of the life history (Appendix B.5).

2.4 Metabolism, net energy and mass

The next selection that we need to consider is primary selection on metabolism, net energy and mass. For this

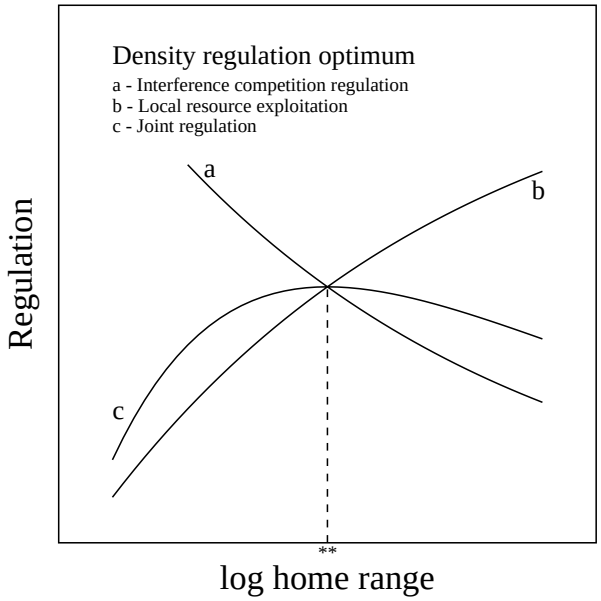

Figure 1: **The optimal home range** (**) as defined by the density regulation optimum for interference competition and local resource exploitation.

we can combine eqn 4, 11, and $r = \ln \lambda$ to find that the primary selection gradients

$$\partial r/\partial \ln \epsilon = \partial r/\partial \ln \alpha = \partial r/\partial \ln \beta = 1 \tag{16}$$

on the log of net energy (ϵ), and its subcomponents α and β, are unity. The secondary theorem of natural selection (Robertson 1968; Taylor 1996) is therefore predicting evolutionary rates of exponential increase

$$r_x = \mathrm{d} \ln x/\mathrm{d}\tau = \sigma^2_{\ln x} \partial r/\partial \ln x = \sigma^2_{\ln x} \tag{17}$$

on the per-generation time-scale (τ) of natural selection. These rates are proportional to the additive heritable variance ($\sigma^2_{\ln x}$) in net energy, resource handling, and mass-specific metabolism, with subscript $x \in \{\epsilon, \alpha, \beta_\beta\}$.

While this implies sustained selection for an increase in ϵ, the average net energy may decline due to environmental variation, or inter-specific interactions when a smaller species is excluded from essential resources by larger species. Owing to this competitive exclusion of species by inter-specific competition, we can expect a distribution of species with net energetic states that range from a possible minimum to a maximum, with the maximum increasing over evolutionary time due to eqn 17. I will not consider the evolution of the exponential increase in this paper [see Witting (1997, 2003, 2016a,b) instead], but only note that the increase al-

lows me to have a distribution of species that differ in α, β_β and ϵ.

Independently of the selection cause for the evolution of mass, the individuals of an evolutionary lineage cannot be large unless they have evolved the ability to consume plenty of resources. This implies that an evolutionary change in mass is selected, in one way or the other, as a consequence of the evolutionary change in net energy, i.e.,

$$\frac{\mathrm{d}\ln w}{\mathrm{d}\tau} = \frac{\partial \ln w}{\partial \ln \epsilon}\frac{\mathrm{d}\ln \epsilon}{\mathrm{d}\tau} \tag{18}$$

with

$$\partial \ln w/\partial \ln \epsilon = 1/\hat{\epsilon} \tag{19}$$

defining the selection dependence of mass on energy given a log-linear relation, where $\hat{\epsilon}$ is an invariant parameter. The specific mechanisms of mass selection are not considered here as they are described by Witting (1997, 2008, 2017). From eqns 18 and 19 we can conclude that natural selection creates an evolutionary function

$$w = \int \frac{\partial \ln w}{\partial \ln \epsilon}\,\mathrm{d}\ln \epsilon = (\epsilon/\epsilon_\circ)^{1/\hat{\epsilon}} \tag{20}$$

that defines mass as an evolutionary consequence of a selection that is imposed by the net energy of the organism, where $1/\hat{\epsilon}$ is an allometric exponent that is given by the selection relation of eqn 19, and the intercept parameter $\epsilon_\circ$ is an initial condition.

3 Body mass allometries

The allometries that can be observed across natural species are post-mass-rescaling allometries in the sense that they are the allometric correlations that have evolved by the complete selection process from the primary selection of metabolism and net energy, over metabolic-rescaling and body mass selection, to mass-rescaling selection from the evolutionary changes in mass.

The post-mass-rescaling allometry $x = x_\circ w^{\hat{x}}$ for the dependence of an unspecified trait x on mass is thus a product

$$x = x_\beta x_w = x_\circ w^{\hat{x}_\beta} w^{\hat{x}_w} = x_\circ w^{\hat{x}} \tag{21}$$

between the allometry of mass-rescaling

$$x_w = w^{\hat{x}_w} \tag{22}$$

that describes the complete allometric scaling with mass when there is no primary selection of mass-specific metabolism, and the allometry of metabolic-rescaling

$$x_\beta = w^{\hat{x}_\beta} \tag{23}$$

that is the additional allometric scaling that evolves from metabolic-rescaling and the dependence of body mass selection on the net energy that is generated by the primary selection of mass-specific metabolism ($x_\circ$ is the initial condition of the allometric intercept, and $\hat{x} = \hat{x}_\beta + \hat{x}_w$ the final allometric exponents, with hats denoting mass exponents).

Many of the allometries that have been established empirically across natural species have Kleiber scaling with exponents around $\pm 1/4$ or $\pm 3/4$. The life history covariance that evolves from my selection model identifies these exponents as the mass-rescaling exponents of eqn 22. Hence, in some instances we may regard the metabolic-rescaling allometries of eqn 23 as the evolution of the intercepts ($x_\circ w^{\hat{x}_\beta}$) of the more traditional mass-rescaling allometries; an evolution that is caused by the primary selection of metabolic pace.

3.1 Mass-rescaling

The inverse of eqn 20 is the $\epsilon = \epsilon_\circ w^{\hat{\epsilon}}$ allometry between mass and energy. And with net energy being a function of handling and metabolic pace (eqn 4), this implies that the allometric exponents of the three traits ϵ, α, and β are interrelated. To describe this, let the $\epsilon \propto \alpha\beta$ relation be rewritten as a product of sub-components of resource handling and mass-specific metabolism, where

$$\epsilon \propto \alpha\beta_\circ\beta_\beta\beta_w \tag{24}$$

with $\beta = \beta_\circ\beta_\beta\beta_w$ being split into a mass-rescaling component ($\beta_w = w^{\hat{\beta}_w}$) and a primary selection component ($\beta_\beta = w^{\hat{\beta}_\beta}$), where $w = \beta_\beta^{1/\hat{\beta}_\beta}$ is the selected dependence of mass on the primary selection of mass-specific metabolism.

Of the three parameters ϵ, α and β it is only metabolism that is split into a primary selection and a mass-rescaling component. Resource handling is defined as a pure primary selected parameter that is generating net energy for the selection of mass independently of the selected changes in the pace of resource handling (see appendix of Witting 2017). And net energy is found to be invariant of mass-rescaling on the per-generation time-scale (see Section 3.1.1) that is relevant for the natural selection of mass.

Relating to the relationship between the allometric exponents we have $\hat{\epsilon} = \hat{\alpha} + \hat{\beta}_\beta + \hat{\beta}_w$ from eqn 24. To identify the causal dependence in this expression, recall that the selection of mass is reflecting the exponent of net energy ($\hat{\epsilon}$), as it is defined by the partial derivative $\partial \ln w/\partial \ln \epsilon = 1/\hat{\epsilon}$ of eqn 19. This implies that we define the selection of mass from the changes in the

average net energy invariantly of the underlying causes (α, β_β & ρ) for the change in energy. We may thus expect that $\hat{\epsilon}$ is invariant of the other exponents $\hat{\alpha}$, $\hat{\beta}_\beta$ and $\hat{\beta}_w$, while the latter three are evolutionarily interrelated by the following trade-off

$$\hat{\alpha} = \hat{\epsilon} - \hat{\beta}_w - \hat{\beta}_\beta. \tag{25}$$

The expected invariance of the $\hat{\epsilon}$ exponent relative to the other three exponents in eqn 25 is confirmed later in this section, together with a similar invariance for the mass-rescaling exponent of metabolism ($\hat{\beta}_w$). Hence, in the end we will find a direct allometric trade-off between the exponent for resource handling ($\hat{\alpha}$) and the exponent for the primary selection of mass-specific metabolism ($\hat{\beta}_\beta$). This trade-off is not reflecting a metabolic-rescaling of resource handling. It is only reflecting the relative importance of resource handling and mass-specific metabolism for the generation of the variation in the net energy that is responsible for the natural selection of the variation in the body masses of the species that are compared in an allometric study.

Now, let us ignore variation in the primary selection of mass-specific metabolism, i.e., let

$$\hat{\alpha} = \hat{\epsilon} - \hat{\beta}_w \tag{26}$$

from eqn 25 with $\hat{\beta}_\beta = 0$ and $x_\beta = w^0$ for all traits x. This corresponds with the allometric model of Witting (1995), that determines the rescaling of the life history in response to the evolving mass. It will give us allometries for the limit case where all of the evolutionary variation in mass is induced by variation in resource handling and/or resource availability, with metabolism evolving exclusively by the allometric rescaling with mass.

In this case, for life history and ecological traits (x) we can expect an allometric mass-rescaling

$$\frac{\mathrm{d}\ln x_w}{\mathrm{d}\tau} = \frac{\partial \ln x_w}{\partial \ln w}\frac{\mathrm{d}\ln w}{\mathrm{d}\tau}, \tag{27}$$

where traits are selected in response to the selection change in mass (eqn 18). Given log-linear selection relations like

$$\partial \ln x_w / \partial \ln w = \hat{x}_w, \tag{28}$$

this implies the evolution of mass-rescaling allometries

$$x_w \propto \int \frac{\partial \ln x_w}{\partial \ln w}\,\mathrm{d}\ln w \propto w^{\hat{x}_w}, \tag{29}$$

where $\hat{x}_w$ is the mass-rescaling exponent.

3.1.1 *Metabolic trade-off selection*

Mass-rescaling selection is induced by a metabolism that trade-offs against the time that is needed for reproduction, when the parental energy that is allocated to the offspring has to be used either on the growing mass or on the metabolism of the offspring (eqn 54). Less energy is available for growth when more energy is metabolised, and this will cause the juvenile period to increase, and the reproductive rate to decline, with an increase in metabolic rate.

The evolutionary linking of the different life history traits by this metabolic trade-off selection is illustrated by the following causal relationships

$$\begin{array}{cccccc} & \underset{\text{selection}}{\mathrm{d}\epsilon/\mathrm{d}t>0} & & & & \\ \Rightarrow & \underset{\text{selection}}{\mathrm{d}w/\mathrm{d}t>0} & \Rightarrow & \underset{\text{constraint}}{\mathrm{d}t_j/\mathrm{d}t>0} & \Rightarrow & \underset{\text{problem}}{\mathrm{d}R/\mathrm{d}t<0} \\ \Rightarrow & \underset{\text{selection}}{\mathrm{d}\beta/\mathrm{d}t<0} & \Rightarrow & \underset{\text{solution}}{\mathrm{d}t_j/\mathrm{d}t<0} & \Rightarrow & \underset{\text{problem}}{\mathrm{d}\epsilon/\mathrm{d}t<0} \\ \Rightarrow & \underset{\text{selection}}{\mathrm{d}t_r/\mathrm{d}t>0} & \Rightarrow & \underset{\text{solution}}{\mathrm{d}R/\mathrm{d}t>0} & \Rightarrow & \underset{\text{result}}{R\propto\frac{t_r}{t_j}\frac{\epsilon}{\epsilon_j}\propto w^0} \end{array}. \tag{30}$$

Initially we have the selection induced increase in net energy ($\mathrm{d}\epsilon/\mathrm{d}t > 0$, eqn 17) that allows for a selection increase in mass ($\mathrm{d}w/\mathrm{d}t > 0$, eqn 18). Among variants with a similar larger than average body mass that is favoured by this selection, it is the variant that has life history traits that are correlated in such a way that the physiological (frequency-independent) replication rate is invariant of mass, that is selected over variants where the replication rate is declining with mass. Mass-rescaling by metabolic trade-off selection is the selection of these trait correlations as induced by the primary selection of mass.

From the physiological constraint of eqn 6 on net energy, metabolism and mass we find that the juvenile period, that defines the time that is needed to grow an offspring [$t_j = 1/(\epsilon_j/w - \acute{w}_j\beta\acute{\beta}_j)$], is increasing ($\mathrm{d}t_j/\mathrm{d}t > 0$) with mass as $\partial t_j/\partial w = \epsilon_j/w^2(\epsilon_j/w - \acute{w}_j\beta\acute{\beta}_j)^2 > 0$. Then, from the reproductive constraint $R = \frac{t_r}{t_j}\frac{\epsilon}{\epsilon_j}$ of eqn 9 we have that the increased juvenile period implies a lifetime reproduction and fitness decline ($\mathrm{d}R/\mathrm{d}t < 0$) that is avoided by selection if possible.

This selection conflict is solved in the third line of eqn 30, by trait correlations for an allometric rescaling where mass-specific metabolism is declining with mass ($\mathrm{d}\beta/\mathrm{d}t < 0$). This will shorten ($\mathrm{d}t_j/\mathrm{d}t < 0$) the juvenile period [$t_j = 1/(\epsilon_j/w - \acute{w}_j\beta\acute{\beta}_j)$] because a larger fraction of the parental energy is then allocated to the growth of the offspring at the cost of the energy that is burned

by the metabolism of the offspring. From the total energy invested in an offspring (eqn 6), the expression for lifetime reproduction is $R = \frac{t_r \epsilon}{w(1+\acute{w}_j t_j \beta \acute{\beta}_j)}$. Hence, provided that $t_r\epsilon$ is constant, the selection conflict on mass is cancelled when $\acute{w}_j t_j \beta \acute{\beta}_j$ is invariant of mass; implying selection for a mass-specific metabolism that is inversely proportional to the juvenile period $\beta_w \propto 1/t_{j,w}$.

But as $\epsilon = \alpha\tilde{\beta}$ we find that the net energy will decline ($\mathrm{d}\epsilon/\mathrm{d}t < 0$) with the decline in mass-specific metabolism, and given $R = \frac{t_r}{t_j}\frac{\epsilon}{\epsilon_j}$ this implies a decline in lifetime reproduction and fitness because the parent will no longer have the required energy available for reproduction. This selection conflict is solved in the last line of eqn 30, by trait correlations that extend the reproductive period ($\mathrm{d}t_r/\mathrm{d}t > 0$) until it is proportional with the juvenile period and inversely proportional with mass-specific metabolism. This leads to an increase in lifetime reproduction ($\mathrm{d}R/\mathrm{d}t > 0$) that results in a physiologically invariant fitness

$$R \propto \frac{t_r}{t_j}\frac{\epsilon}{\epsilon_j} \propto \frac{t_r}{t_j} \propto \frac{\epsilon}{\epsilon_j} \propto w^0 \tag{31}$$

that is selected by the selection for increased mass.

The evolutionary consequence of eqn 31 is a selection that dilates the per-generation time-scale of natural selection in order to maintain net energy and fitness invariant of a selection increase in mass. The result is an energetic state that is maintained constant in biotic time ($\epsilon t_r \propto \alpha\tilde{\beta}_\beta\tilde{\beta}_w t_\beta t_w \propto \alpha$, with $t_\beta t_w \propto 1/\tilde{\beta}_\beta\tilde{\beta}_w$) while it is declining in physical time ($\epsilon \propto \alpha\tilde{\beta}_\beta\tilde{\beta}_w$) with a decline in mass-specific metabolism with mass ($\beta_w \propto w^{\hat{\beta}_w}$, with $\hat{\beta}_w < 0$).

When the $R \propto w^0$ invariance is combined with the population dynamic constraint $pR = 1$, we obtain a mass invariant survival

$$p \propto w^0 \tag{32}$$

from a survival curve with age that is invariant in biotic time, as expected for time scaling of intrinsic survival (eqn 10). And when the $\epsilon/\epsilon_j \propto w^0$ invariance of eqn 31 is combined with the equation for the total energetic investment in each offspring (eqn 6), we obtain the following energetic constraint

$$t_w \propto w/\epsilon \tag{33}$$

that defines the mass-rescaling component of biotic time from the ratio of the selected mass over the selected net energy.

This mass-rescaling is including the optimal density regulation of Section 2.3, because the arguments of the three density regulation components $f_e(nw\beta)$, $f_\iota(nvh^{\frac{d-1}{d}})$ and $f_s(\beta h^{1/d}/v)$ are dependent on the traits that are rescaled by the selection increase in mass. The result is a trait covariance that is selected not only by the constraints of the metabolic trade-off (eqns 31 and 33), but also by the constraints of optimal density regulation (eqn 15). And where it is the metabolic trade-off selection that initiates the selection response to the evolutionary changes in mass it is, as we will see in the next sub-section, primarily the ecological geometry of optimal density regulation that explains the numerical values of the allometric response.

3.1.2 *Allometric deduction*

In order to deduce the allometric exponents from the selection conditions that we have described, let us use t as the scaling parameter for all biotic periods $t_x = \tau_x t_g$, and exchange v with t_w from eqn 12. Then, insert power relations $w^{\hat{x}_w}$ for the different traits $x \in \{p, t, \epsilon, \beta, n, h\}$ into eqns 5, 33, and 15 to obtain

$$\hat{t}_w = -\hat{\beta}_w, \tag{34}$$

$$\hat{t}_w = 1 - \hat{\epsilon}, \tag{35}$$

$$\hat{t}_w + \hat{n}_w + (d-1)\hat{h}_w/d = 0, \tag{36}$$

$$\hat{\beta}_w - \hat{t}_w + \hat{h}_w/d = 0, \tag{37}$$

$$\hat{n}_w + \hat{\beta}_w + 1 = 0, \tag{38}$$

and the following invariance

$$\hat{R}_w = \hat{p}_w = 0 \tag{39}$$

from eqns 31 and 32.

Now, from eqns 38 and 34 we have $\hat{n}_w = \hat{t}_w - 1$. Insert this expression into eqn 36 and obtain $2\hat{t}_w - 1 + (d-1)\hat{h}_w/d = 0$, and exchange $2\hat{t}_w$ with $\hat{h}_w/d$ from eqns 37 and 34 to obtain $\hat{h}_w = 1$. Then, from eqns 37 and 34 we have $\hat{t}_w = 1/2d$ and $\hat{\beta}_w = -1/2d$; and from eqns 26, 35, and 38 that $\hat{\epsilon} = (2d-1)/2d$, $\hat{\alpha} = 1$ and $\hat{n}_w = (1-2d)/2d$. And with the population dynamic growth rate in physical time being $r = \ln(pR)/t_g$ we have $\hat{r}_w = -1/2d$ given $pR \propto w^0$.

These mass-rescaling exponents are listed in Table 2a. They are the same as those of the original deduction in Witting (1995), except that the new deduction is more general as it explains also the exponent for survival (p) and the inverse link between the biotic periods and metabolism ($t_x \propto 1/\beta$).

One way to illustrate the evolutionary significance of the mass-rescaling allometries is to plot the average replication at the population dynamic equilibrium

a) Mass-rescaling as a function of d

$\hat{\epsilon}$	$\hat{\alpha}$	$\hat{\beta}_w$	$\hat{t}_w$	$\hat{p}_w$	$\hat{R}_w$	$\hat{r}_w$	$\hat{h}_w$	$\hat{n}_w$
$\frac{2d-1}{2d}$	1	$-\frac{1}{2d}$	$\frac{1}{2d}$	0	0	$-\frac{1}{2d}$	1	$\frac{1-2d}{2d}$

b) Metabolic-rescaling as a function of $\hat{\beta}_\beta$

$\hat{\alpha}$	$\hat{t}_\beta$	$\hat{p}_\beta$	$\hat{R}_\beta$	$\hat{r}_\beta$	$\hat{h}_\beta$	$\hat{n}_\beta$
$1-\hat{\beta}_\beta$	$-\hat{\beta}_\beta$	$\hat{\beta}_\beta$	$-\hat{\beta}_\beta$	$\hat{\beta}_\beta$	0	$-\hat{\beta}_\beta$

Table 2: **Allometric rescaling.** **a**) The predicted mass-rescaling exponents ($\hat{x}_w$) of body mass (w) allometries ($x \propto x_\beta w^{\hat{x}_w}$) as functions of the spatial dimensions (d) of the interactive behaviour. **b**) The predicted metabolic-rescaling exponents ($\hat{x}_\beta$) of the mass-rescaling intercepts ($x_\beta = w^{\hat{x}_\beta}$) as a function of the metabolic-rescaling exponent for mass-specific metabolism ($\hat{\beta}_\beta$). **Symbols**: ϵ:net energy; α:resource handling; β:mass-specific metabolism; t:biotic periods; p:survival; R:lifetime reproduction; r:population growth rate; h:home range; n:population density.

(pR) as a function of the potential rescaling exponents. This is done in Fig. 2, and it illustrates that it is only for the actual mass-rescaling of the allometric solution that natural selection can maintain the life history in the required balance where the average per-generation replication rate at the evolutionarily determined population dynamic equilibrium is one invariantly of mass.

3.2 Metabolic-rescaling

Let us now go beyond the mass-rescaling allometries and consider the influence of variation in the mass-rescaling intercepts as it evolves from primary selection of mass-specific metabolism. With mass-specific metabolism (β) being selected as the pace ($\tilde{\alpha}^{**} = \tilde{\beta}$) of resource handling ($\alpha$), it is providing part of, or the complete, net energy ($\epsilon = \alpha\tilde{\beta}$) that is driving the selection of mass, and it is thus influencing the allometric scaling independently of the mass-rescaling of the previous section.

The evolutionary increase in metabolism is causing a metabolic-rescaling

$$\frac{\mathrm{d}\ln x_\beta}{\mathrm{d}\tau} = \frac{\partial \ln x_\beta}{\partial \ln \beta_\beta}\frac{\mathrm{d}\ln \beta_\beta}{\mathrm{d}\tau}, \tag{40}$$

where rate related life history traits are scaled by the increase in metabolic pace, with subscript β denoting components that relate to this primary selection on metabolic pace. This rescaling will transform log-linear selection relations

$$\partial \ln x_\beta / \partial \ln \beta_\beta = \check{x}_\beta \tag{41}$$

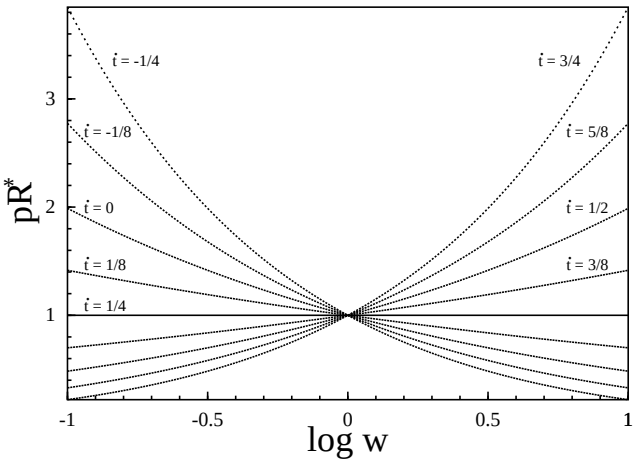

Figure 2: **The replication invariance.** A theoretical span (eqn 99) of the average per-generation replication rate (pR^*) in the population as a function of the selected mass (w) for a range of potentially possible mass-rescaling exponents $\hat{t}_w$ (numbers in plot). It is only the exponent of the allometric solution ($\hat{t}_w = 1/4$) that maintains the life history in the required balance where the average per-generation replication rate at the evolutionary optimum is one invariantly of mass. The model behind the figure is given in Appendix C, and the plot is for $z = 0.1$ for eqn 99, given 2D interactions where $\hat{\epsilon} = 3/4$.

with invariant exponents $\check{x}_\beta$ into metabolic allometries

$$x_\beta \propto \int \frac{\partial \ln x_\beta}{\partial \ln \beta_\beta}\,\mathrm{d}\ln \beta_\beta \propto \beta_\beta^{\check{x}_\beta} \tag{42}$$

where, among others, increased mass-specific metabolism is shortening biotic time periods like generation time and increasing the amount of resources that the individual assimilates per unit physical time. With the latter determining the selection pressure on mass, it follows that the $\partial \ln w/\partial \ln \epsilon$ relation of eqn 18 has a metabolic component

$$\frac{\partial \ln w}{\partial \ln \epsilon}\frac{\partial \ln \epsilon}{\partial \ln \beta_\beta}, \tag{43}$$

where the primary selection of mass-specific metabolism is generating net energy for the selection of mass. Given the log-linear selection relation of eqn 19, and $\partial \ln \epsilon/\partial \ln \beta_\beta = 1$ from $\epsilon = \alpha\tilde{\beta}$, we find that primary selection on metabolic pace selects mass as a partial function of mass-specific metabolism

$$w_\beta \propto \int \frac{\partial \ln w}{\partial \ln \epsilon}\frac{\partial \ln \epsilon}{\partial \ln \beta_\beta}\,\mathrm{d}\ln \beta_\beta \propto \beta_\beta^{1/\hat{\epsilon}}, \tag{44}$$

with the inverse

$$\beta_\beta \propto w_\beta^{\hat{\epsilon}} \tag{45}$$

of the selection function being the component of the post-mass-rescaling allometry for mass-specific metabolism that evolves from the primary selection of metabolic pace.

Instead of dealing with mass as a joint parameter of several sub-components, like $w = w_\alpha w_\beta$, I will express the evolutionary dependence of mass on α and β_β, and the more usual inverse allometric correlations, as functions of total mass w. For this, let the dependence of w on β_β that is captured by the allometry of eqns 44 and 45, be expressed by the following allometry

$$\beta_\beta \propto w^{\hat{\beta}_\beta}. \tag{46}$$

Note that the relative dependence of total mass on mass-specific metabolism, as described e.g. by the w_β/w-ratio, is expressed differently by eqns 45 and 46. In eqn 45 we have an invariant exponent $\hat{\epsilon}$, with the dependence of mass on mass-specific metabolism being captured by the w_β component that is directly dependent on β_β. In eqn 46 it is instead the $\hat{\beta}_\beta$ exponent that will change as a function of the w_β/w ratio (eqn 25), to make any given dependence of total mass w on β_β consistent across the range of possible β_β values.

Now, if we insert eqn 46 into eqn 42, we find that the metabolic-rescaling of the life history can be expressed by an allometric relation of total mass

$$x_\beta \propto \beta_\beta^{\check{x}_\beta} \propto w^{\check{x}_\beta \hat{\beta}_\beta} \propto w^{\hat{x}_\beta}, \tag{47}$$

with $\hat{x}_\beta = \check{x}_\beta \hat{\beta}_\beta$ and $\check{\beta}_\beta = 1$.

3.2.1 *Allometric deduction*

To deduce the metabolic-rescaling exponents, from the inverse relationship between pace and biotic time periods, we have

$$\hat{t}_\beta = -\hat{\beta}_\beta. \tag{48}$$

And from $\hat{\epsilon} = (2d-1)/2d$, $\hat{\beta}_w = -1/2d$ and the $\epsilon \propto \alpha\beta_\beta\beta_w$ constraint of eqn 24 that links net energy, resource handling, and pace we have

$$\hat{\alpha} = 1 - \hat{\beta}_\beta. \tag{49}$$

From the invariant selection optimum of density regulation $h^{**} \propto (v/\beta)^d$ (eqn 14), and a foraging speed $v = v_\circ \beta_\beta w^{\hat{t}_w}$ that is defined by the mass-rescaling for lifespan ($w^{\hat{t}_w}$) and the primary selection of metabolism (β_β, eqn 12), we have $[v_\circ \beta_\beta w^{1/2d}/\beta_\beta w^{-1/2d}]^d = h_\beta w^1$ given $\hat{t}_w = 1/2d$, $\hat{\beta}_w = -1/2d$ and $\hat{h}_w = 1$. When solved for the mass-rescaling intercept for the home range, we find that it is an invariant intercept

$$\hat{h}_\beta = 0 \tag{50}$$

that will maintain the population at the selection optimum.

From the $nvh^{\frac{d-1}{d}} \propto w^0$ invariance of interference competition (eqn 15), with $v = v_\circ \beta_\beta w^{\hat{t}_w}$ we have $n_\beta w^{\frac{1-2d}{2d}} v_\circ w^{\hat{\beta}_\beta} w^{\frac{1}{2d}} w^{\frac{d-1}{d}} \propto w^0$ given $\hat{n}_w = (1-2d)/2d$, $\hat{t}_w = 1/2d$ and $\hat{h}_w = 1$. When solved for the mass-rescaling intercept of abundance, we find that it is a population density that scales inversely with the metabolic intercept

$$\hat{n}_\beta = -\hat{\beta}_\beta \tag{51}$$

that maintains a body mass invariant level of interference competition in the population. This rescaling will also maintain an invariant exploitation of the resource (eqn 13), with an invariant use of energy by the population.

If we turn to the population dynamic equilibrium ($p\, t_r \epsilon/\acute{\beta} w = 1$) it implies $p_\beta w^{\hat{p}_w} t_\beta w^{\hat{t}_w} \epsilon_\circ w^{\hat{\epsilon}} \propto w$, given $\acute{\beta} \propto w^0$. With $\hat{p}_w = 0$, $\hat{t}_w = 1/2d$, $\hat{\epsilon} = (2d-1)/2d$ and $t_\beta \propto 1/\beta_\beta$ we find that it is a survival mass-rescaling intercept that is proportional to the metabolic intercept

$$\hat{p}_\beta = \hat{\beta}_\beta \tag{52}$$

that maintains the balance of the population dynamic equilibrium. This increase in survival for the primary selection of mass-specific metabolism reflects a decline in the mortality rate in biotic time; a decline that may reflect a shortening of the physical time period where the individual is exposed to extrinsic mortality factors.

With $R^* = 1/p^*$ it follows that the mass-rescaling intercept of lifetime reproduction is inversely related to the intercept of the metabolic rate per unit body mass ($\hat{R}_\beta = -\hat{\beta}_\beta$), and from $r = \ln(pR)/t_g$ we have $\hat{r}_\beta = \hat{\beta}_\beta$. These selection changes in the mass-rescaling intercepts from metabolic-rescaling are listed in Table 2b.

3.3 Final allometries

Given the deduced exponents in Table 2 for the allometric rescaling with mass and metabolism we can calculate the $\hat{x}$ exponents of the final scaling of the life history with mass for a variety of situations. I list these exponents in Table 3 for interactive behaviour in one, two and three spatial dimensions.

Apart from the home range exponent that is always one, the exponents depend on the $\hat{\beta}_\beta$ exponent that describes the relative importance of metabolic pace for net resource assimilation and, thus, also for the selection of mass. This implies a post-mass-rescaling exponent for mass-specific metabolism ($\hat{\beta}$) that increases

a) One-dimensional interactions ($\hat{\epsilon} = 1/2$)

$\hat{\beta}_\beta$	$\hat{\alpha}$	$\hat{\beta}$	$\hat{t}$	$\hat{p}$	$\hat{R}$	$\hat{r}$	$\hat{h}$	$\hat{n}$
0	1	$-\frac{1}{2}$	$\frac{1}{2}$	0	0	$-\frac{1}{2}$	1	$-\frac{1}{2}$
$\frac{1}{2}$	$\frac{1}{2}$	0	0	$\frac{1}{2}$	$-\frac{1}{2}$	0	1	-1
1	0	$\frac{1}{2}$	$-\frac{1}{2}$	1	-1	$\frac{1}{2}$	1	$-\frac{3}{2}$

b) Two-dimensional interactions ($\hat{\epsilon} = 3/4$)

$\hat{\beta}_\beta$	$\hat{\alpha}$	$\hat{\beta}$	$\hat{t}$	$\hat{p}$	$\hat{R}$	$\hat{r}$	$\hat{h}$	$\hat{n}$
0	1	$-\frac{1}{4}$	$\frac{1}{4}$	0	0	$-\frac{1}{4}$	1	$-\frac{3}{4}$
$\frac{1}{4}$	$\frac{3}{4}$	0	0	$\frac{1}{4}$	$-\frac{1}{4}$	0	1	-1
$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{2}$	$-\frac{1}{2}$	$\frac{1}{4}$	1	$-\frac{5}{4}$
1	0	$\frac{3}{4}$	$-\frac{3}{4}$	1	-1	$\frac{3}{4}$	1	$-\frac{7}{4}$

c) Three-dimensional interactions ($\hat{\epsilon} = 5/6$)

$\hat{\beta}_\beta$	$\hat{\alpha}$	$\hat{\beta}$	$\hat{t}$	$\hat{p}$	$\hat{R}$	$\hat{r}$	$\hat{h}$	$\hat{n}$
0	1	$-\frac{1}{6}$	$\frac{1}{6}$	0	0	$-\frac{1}{6}$	1	$-\frac{5}{6}$
$\frac{1}{6}$	$\frac{5}{6}$	0	0	$\frac{1}{6}$	$-\frac{1}{6}$	0	1	-1
$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{3}$	$-\frac{1}{3}$	$\frac{1}{2}$	$-\frac{1}{2}$	$\frac{1}{3}$	1	$-\frac{4}{3}$
1	0	$\frac{5}{6}$	$-\frac{5}{6}$	1	-1	$\frac{5}{6}$	1	$-\frac{11}{6}$

Table 3: **Theoretical allometries.** Allometric exponents ($\hat{x}$) as they evolve from allometric rescaling given primary selection on metabolism and mass. The exponents depend on the dimensionality of the interactive behaviour (1D, 2D or 3D), and on the $\hat{\beta}_\beta$ exponent that describes the relative importance of mass-specific metabolism for the net energy of the organism.

with the relative importance of metabolism for the evolution of mass. This is illustrated in Table 3, where $\hat{\beta}$ is 3/4 for 2D, and 5/6 for 3D, interactions in the extreme case where all of the variation in ϵ, and thus also body mass, is caused by the primary selection of mass-specific metabolism ($\hat{\beta}_\beta = 1$). At the other extreme where all the body mass variation is caused by variation in resource handling and/or resource availability ($\hat{\beta}_\beta = 0$), the metabolic exponent takes the more well-known values of $-1/4$ (2D) and $-1/6$ (3D). For an intermediate case with a similar importance of handling and pace ($\hat{\beta}_\beta = 1/2$) we have 1/4 for 2D and 1/3 for 3D. The cases where mass-specific metabolism is independent of mass ($\hat{\beta} = 0$) is also shown. The latter depends on the spatial dimensionality of the interactive behaviour, with $\hat{\beta} = 0$ for $\hat{\beta}_\beta = 1/2d$.

The majority of the final allometric exponents are given as fractions, where $2d$ is the common denominator, with the most well-known set of allometric exponents for large bodied species, i.e., the set with $\hat{\beta} = -1/4$, evolving for two-dimensional interactions when all the variation in body mass is caused by variation in resource handling/availability. This case has net energy that scales to the 3/4 power of mass, biotic periods that scales to the 1/4 power, population densities that scale to the $-3/4$ power, and population growth that scales to the $-1/4$ power of mass. The corresponding exponents for one-dimensional interactions are $\pm 1/2$, while the $\pm 1/4$, and $\pm 3/4$, exponents are exchanged with $\pm 1/6$, and $\pm 5/6$, exponents for interactive behaviour in three spatial dimensions.

The evolutionary directions of the underlying selection components are listed in Table 4 for the two cases where all of the variation in net energy is generated either from primary selection on resource handling or from primary selection on metabolic pace. The primary selection of net energy generates the interactive selection of mass from increased population growth, with the selected mass and mass-rescaling selection adjusting the selected traits to maintain the selected net energy invariant on the per-generation time-scale of natural selection, and the interference competition invariant of mass.

4 Empirical allometries

How do the empirical exponents of inter-specific allometries compare with the predicted? As the exponents are predicted from the intra-specific feeding ecology of interacting individuals, we cannot expect a 100% match because the exponents may vary somewhat with variation in the underlying ecology. The model is currently not developed to reflect this type of variation; it is developed only for a base-case type of ecology, where it predicts allometric transitions from variation in the spatial dimensionality of the feeding behaviour and from variation in the importance of primary selection on metabolism for the natural selection of mass. In the evaluation of data, I will thus focus on the average and commonly observed empirical exponents across the tree of life to see whether the predicted exponents and their transitions are commonly observed and in general agreement with data.

4.1 Prokaryotes

Allometries in prokaryotes are maybe the most difficult to estimate, not only because of nonnegligible errors in most estimates of mass, but also because metabolism and mass are strongly dependent on an active or inactive physiological state. By correcting estimated exponents to their reduced major axis values

	Handling			Pace		
$\mathrm{d}x/\mathrm{d}\tau$	P	W	F	P	W	F
$\mathrm{d}\alpha/\mathrm{d}\tau$	+	0	+	0	0	0
$\mathrm{d}\beta/\mathrm{d}\tau$	0	−	−	+	−	+
$\mathrm{d}\epsilon/\mathrm{d}\tau$	+	−	+	+	−	+
$\mathrm{d}t_r/\mathrm{d}\tau$	0	+	+	−	+	−
$\mathrm{d}\epsilon t_r/\mathrm{d}\tau$	+	$0^\dagger$	+	0	$0^\dagger$	0
$\mathrm{d}p/\mathrm{d}\tau$	0	0	0	+	0	+
$\mathrm{d}R/\mathrm{d}\tau$	+	−	0	0	−	−
$\mathrm{d}r_\tau/\mathrm{d}\tau$	+	−	0	+	−	0
$\mathrm{d}n/\mathrm{d}\tau$	+	−	−	+	−	−
$\mathrm{d}\iota/\mathrm{d}\tau$	+	−	$0^\ddagger$	+	−	$0^\ddagger$
$\mathrm{d}w/\mathrm{d}\tau$	→	+	+	→	+	+

Table 4: **Selection directions.** The directional selection ($\mathrm{d}x/\mathrm{d}\tau = -, 0, +$) of the different traits (x) for the primary (P), mass-rescaling (W) and final (F) selection components of the proposed selection in high-energy organisms, given the two cases where all of the variation in mass is selected from net energy that is generated either by the primary selection of resource handling, or by the primary selection of metabolic pace. † denotes the invariance of mass-rescaling on net energy on the per-generation time-scale of natural selection, ‡ the invariance of interference competition for the selection attractor on mass, → the link between primary and mass-rescaling selection, and $r_\tau = \ln(pR)$ the exponential growth rate on a per-generation time-scale.

to account for uncertain estimates of mass, DeLong et al. (2010) estimated an allometric exponent for mass-specific metabolism of 0.96 ± 0.18 across active prokaryotes, and 0.72 ± 0.07 across inactive. Given the theoretical allometries in Table 3, these estimates indicate three-dimensional ecology with an average exponent of 0.84, and body mass variation that evolves from primary variation in mass-specific metabolism.

This fit should be considered more uncertain than indicated by the match of the theoretical and empirical exponents. Not only may the empirical estimates be more uncertain than indicated by their standard errors, but the feeding ecology of prokaryotes might also differ somewhat from the described model, and all prokaryotes may not have three-dimensional ecology.

4.2 Protists and protozoa

Based on the data from Makarieva et al. (2008), DeLong et al. (2010) estimated the average exponent across the complete range of inactive protozoa to be -0.03 (± 0.05), and that of active protists to be 0.06 (± 0.07).

$\hat{x}$	2D	Reptiles	Birds	Mammals
$\hat{\beta}$	-0.25	-0.24	-0.26	-0.26
$\hat{t}$	0.25	0.23	0.18	0.25
$\hat{p}$	0.00	-	0.01	-
$\hat{R}$	0.00	-	0.00	-0.03
$\hat{h}$	1.00	0.95	1.16	0.99
$\hat{n}$	-0.75	-0.77	-0.75	-0.78

Table 5: **Empirical exponents for different traits.** The theoretical 2D exponents compared with empirical exponents for reptiles, birds and mammals. From Witting (1997), except $\hat{t}$ for reptiles from Calder (1984).

These exponents are smaller than the exponent for prokaryotes and larger than the typical $-1/4$ and $-1/6$ exponents for multicellular animals, and they indicate a mass-specific metabolism that is invariant of mass.

A visual inspection of the data for protozoa, however, indicates that the inter-specific exponent for mass-specific metabolism may change from positive to negative with an increase in mass (see e.g., Fig. 4 in Witting 2017). This potential relationship is somewhat obscured by high metabolic rates in the four smallest protozoa in the dataset, yet being close to the upper size range of prokaryotes, these protozoa might be selected differently than the rest. So to estimate a potential range for the metabolic exponent in typical protozoa, I excluded the four smallest species from the data of Makarieva et al. (2008), and least-squares fitted a third degree polynomial to the remaining data for inactive protozoa ($n = 48$). This gave point estimates of the body mass exponent for mass-specific metabolism that declined from 0.61 across the smallest [$\log w(\mathrm{kg}) = -13.5$], over zero across intermediate [$\log w(\mathrm{kg}) = -11$], to a minimum of -0.20 among the largest protozoa [$\log w(\mathrm{kg}) = -8.0$].

While compatible with an average invariance, the change in the exponent across the size range of protozoa indicates a shift in the selection of mass, starting with a mass that is selected predominantly from primary variation in mass-specific metabolism (as in prokaryotes). The importance of mass-specific metabolism for the selection of mass is then declining with an increase in mass towards the larger species, where mass variation is selected mainly from primary variation in the handling and/or density of the underlying energetic resources.

4.3 Multicellular animals

The often reported $-1/4$ exponent for mass-specific metabolism in multicellular animals indicates that the major component of the body mass variation in this group is selected from primary variation in the handling and/or density of the underlying energetic resources.

4.3.1 *Life history and ecological traits*

For any group of organisms, it is the terrestrial vertebrates that have been subjected to most allometric studies, and in Table 5 I show the predicted 2D exponents, together with some of the commonly observed exponents in mammals, reptiles and birds. It can be concluded that a reasonable resemblance exists between the theoretical and empirical exponents across traits ranging from metabolism, lifespan, survival and reproduction over population growth to ecological traits like the home range of individuals and the densities of populations.

4.3.2 *Invariant interference*

A key component of the model is the invariance in the level of interference competition as predicted by the density-dependent selection attractor for mass, given an invariant cost gradient (Witting 1997, 2017). While interaction levels have hardly been reported for any species, evidence for or against the existence of invariant interference competition may be examined by a comparison of empirical allometries in multicellular species. From eqn 13 we expect a level of interference that is proportional to $nvh^{\frac{d-1}{d}}$, with $v = v_\circ \beta_\beta w^{\hat{t}_w}$ (eqn 12). Concentrating on two-dimensional foraging with $\hat{\beta}_\beta = 0$, we find a level of interactive competition that scales as

$$\iota^* \propto w^{\hat{n}+\hat{t}+\hat{h}/2}. \tag{53}$$

Across 2D species in major taxonomic groups $\hat{h}$ is usually approximately 1, $\hat{t}$ approximately 1/4, and $\hat{n}$ around -3/4 (Table 6c; Peters 1983; Calder 1984; Damuth 1987; Nee et al. 1991; Witting 1995). From the exponents in Table 5 we find that $\iota^* \propto w^{-0.035}$ for mammals, $\iota^* \propto w^{0.01}$ for birds, and $\iota^* \propto w^{-0.065}$ for reptiles. This shows that the expected invariance is not contradicted by data.

Based on alternative estimates of the level of intraspecific interference, DeLong and Vasseur (2011), and DeLong (2014) concluded that the level of interference is invariant across species with a wide range of body sizes.

a) $\hat{\beta}$ exponent classification

Group	2D	3D
Mammals (all)[a,1]	-0.26 ± 0.01	
Pelagic mammals[b]		-0.16 ± 0.02
Primates[c]		-0.19 ± 0.03
Bats[a]	-0.26	
Birds[a,1]	-0.26 ± 0.01	
Reptiles[a,2]	-0.24 ± 0.02	
Snakes[a,3]	-0.26 ± 0.04	
Lizards[a,2]		-0.18 ± 0.02
Turtles[a,2]		-0.14 ± 0.03
Frogs[a,2]	-0.29	
Salamanders[a,1]		-0.18 ± 0.02
Fishes[a,3]		-0.20 ± 0.01
Molluscs[a]	-0.25	
Jellyfishes[a]		-0.15

b) 2D - 3D transitions

d	$\hat{\beta}$	$\hat{t}$	$\hat{n}$
2D	-0.26 ± 0.02	0.25 ± 0.04	-0.79 ± 0.09
3D	-0.17 ± 0.02	0.16 ± 0.02	-0.86 ± 0.08

Table 6: **Empirical 2D and 3D allometries. a)**: Taxa classified as 2D or 3D from estimates of the $\hat{\beta}$ exponent ($\pm$SD). **b)**: Exponents for 2D - 3D transitions: $\hat{\beta}$, as defined by the average exponents of Table a; $\hat{t}$, terrestrial versus pelagic mammals (Nowak, 1991); $\hat{n}$, 2D-3D classification and exponents from Pawar et al. (2012). a:Peters (1983) [1:estimate with largest sample size; 2:average estimate; 3:average estimate, but -1 outlier]; b:$\hat{\beta} = -\hat{t}$, Nowak (1991); c:Genoud (2002).

4.3.3 *2D versus 3D*

Another essential prediction is the transitions in the allometric exponents across species that differ in the spatial dimensionality of their interactive behaviour.

To examine the evidence for these transitions, I have in Table 6a listed allometric exponents for mass-specific metabolism ($\hat{\beta}$) across the species of major taxonomic groups, with the empirical $\hat{\beta}$ estimates being used to classify the taxa as having behavioural interactions in 2D or 3D (assuming that $\hat{\beta}_\beta = 0$). Most of these exponents are either the average exponent, or the exponent based on the largest sample size, from the tables in Peters (1983). I found no convincing 1D cases, but the exponents for mass-specific metabolism are in general agreement with the hypothesis that they evolve from the underlying spatial dimensionality of intra-specific

interactions.

The interactions of most terrestrial and benthic taxa are classified as 2D, and those of pelagic and tree-living taxa as 3D. This overall separation is likely to reflect that most terrestrial and benthic animals are constrained to behavioural interaction in the two horizontal dimensions, while pelagic and tree-living species have an extra vertical dimension in which to forage and interact.

With the available data the differentiation in the allometric exponents is maybe clearest in mammals, that are dominated by ground living 2D species and an overall exponent for field mass-specific metabolism of -0.25 ± 0.03 (Savage et al. 2004). Yet pelagic taxa like Cetacea, Pinnipedia and Sirenia have a 3D exponent of 0.16 ± 0.02 for lifespan (Witting 1995), and primates, where the majority of species are tree-living, are also classified as 3D with an exponent of -0.19 ± 0.03 for mass-specific metabolism (Genoud 2002).

Other plausible 2D-3D classifications of interactions from the exponent of mass-specific metabolism include reptiles, snakes, frogs and mollusks as 2D, and fishes, jellyfishes and salamanders as 3D (Table 6a). Although birds and bats move freely in 3D, it is most likely the packing of their breeding and/or feeding territories that are essential for the evolution of the allometric exponents. This packing is 2D in a majority of species with the reported exponents reflecting 2D interactions in birds and bats. A splitting of birds and bats into those that compete for territories and food in 2D and 3D would be desirable, but beyond the scope of this study that is based on empirical literature estimates of allometric exponents.

The dimensionality estimates of the intra-specific interactions from metabolic exponents in lizards, snakes, reptiles and turtles are also likely to benefit from a split into 2D (e.g., ground) and 3D (e.g., tree or pelagic) living species. The reported exponents reflect 3D for lizards and turtles, and 2D for reptiles and snakes.

The split between 2D and 3D interactions has been observed not only in the exponent for mass-specific metabolism, but also in exponents for lifespan and population density (Table 6b), and it seems to relate also to allometries of community ecology (Pawar et al. 2012).

4.3.4 *Mass-rescaling intercepts*

Evidence on the scaling of the intercepts of the traditional mass-rescaling allometries with Kleiber scaling is not as clear as for the mass-rescaling exponents. But with ectotherms having field metabolic rates that are 12 to 20 times smaller than in similar sized endotherms (Nagy 2005), from Table 2b we expect ectotherms to have longer lifespans and to be more abundant than similar sized endotherms, and this is generally the case (Peters 1983; Currie and Fritz 1993; de Magalhães et al. 2007). The predicted inverse relationship between lifetime reproduction and the mass-rescaling intercept for mass-specific metabolism is also in agreement with fecundity estimates that are about ten times higher in reptiles than in mammals (Peters 1983) reflecting, as predicted, a higher probability to survive to reproduce in mammals.

5 Discussion

In this paper I added primary selection on metabolism to the theory of Malthusian Relativity (Witting 1995, 1997, 2008), and showed that the joint selection of metabolism and mass can explain a wide range of allometric exponents that are observed across the tree of life.

5.1 Mass-specific metabolism

Given the importance of metabolism for animals (e.g., Lotka 1922; Pearl 1928; Maynard Smith and Szathmáry 1995; Brown et al. 2004) it is somewhat surprising that the metabolic rate has been the forgotten life history character, with not even a single model on its selection in seven review books on life history evolution (Charlesworth 1980, 1994; Roff 1992, 2002; Stearns 1992; Bulmer 1994; Stearns and Hoekstra 2000; but see Witting 2003; Barve et al. 2014; Artacho and Nespolo 2009; Boratyński et al. 2010; Versteegh et al. 2012).

The evolution of metabolism has instead been studied as a molecular process in relation to the origin of life (Horowitz 1945; Miller 1953; Haldane 1954; Oparin and Clark 1959; Ponnamperuma and Chela-Flores 1993; Chela-Flores et al. 1995; Baltscheffsky 1996; Cunchillos and Lecointre 2003; Ferry and House 2006; Melendez-Hevia et al. 2008; Fernando and Rowe 2007, 2008; Fry 2011; Marakushev and Belonogova 2013). And it has been examined empirically, where metabolism in natural species is linked to a range of extrinsic factors like temperature (e.g., McNab and Morrison 1963 Lovegrove 2003; Wikelski et al. 2003; Careau et al. 2007; Jetz et al. 2007; White et al. 2007), primary production (Mueller and Diamond 2001; Bozinovic et al. 2007, 2009), rainfall (Lovegrove 2003; Withers et al. 2006; White et al. 2007), and diet (McNab 2003; Anderson and Jetz 2005; Muñoz-Garcia and Williams 2005). Fitness components like survival and reproduction have been found to correlate with metabolism in a wide

range of species (see e.g. Table 4 in White and Kearney 2013), and these correlations indicate a stabilising selection (Artacho and Nespolo 2009; White and Kearney 2013). This suggests that natural species are optimised towards some central metabolic value that is given by the current evolutionary state of the species in a given environment. Yet, the study of empirical fitness correlations with metabolism does not reveal the underlying selection of the attracting metabolic rate.

Another widespread view is metabolic ecology (Brown et al. 2004; Sibly et al. 2012; Humphries and McCann 2014; Padfield et al. 2016), where metabolism is seen to determine the rate at which the organism assimilates, transforms and expends energy. Instead of formulating this as a life history trait that is selected by natural selection, metabolic ecology has treated metabolism as a passive physical parameter (Glazier 2015) that is determined by temperature and the geometry and physics of resource transportation networks (West et al. 1997, 1999a,b; Gillooly et al. 2001). While metabolism is indeed essential for the pace of physiological and ecological processes, and while physical factors may constrain the metabolic rate, metabolic ecology is insufficient in itself because there are no physical laws that will explain the elevated rates of metabolism in mobile organisms in general, and in birds and mammals in particular.

I partitioned resource assimilation into resource handling and the pace of handling. This handling and pace generate gross energy, and by defining net energy as the difference between gross energy and the total metabolism of the organism, I found the mass-specific work of handling to be selected as mass-specific metabolism. This implies that mass-specific metabolism is selected as the life history character that determines the pace of the net resource assimilation that generates net energy for self-replication. This provides an overall direction, where unconstrained selection is generating an exponential increase in mass-specific metabolism and the net energy of the organism. Associated with the increase there is a metabolic-rescaling of the rate dependent life history characters, and this rescaling is affecting the body mass allometries because the selection of mass is dependent on the energy that is generated by the selected increase in mass-specific metabolism.

5.2 Mass-rescaling selection

The well-known 1/4 exponents of Kleiber scaling, however, was found to be selected by a mass-rescaling response to the evolutionary changes in mass. While implicit in my original allometric model (Witting 1995), the first formal description of mass-rescaling selection is presented in the current paper. This selection emerges from the energetic trade-off between mass, metabolism and the time that it takes to grow a full-sized offspring, and it selects for a secondary decline in mass-specific metabolism as a response to a primary increase in mass.

With an increasing mass, reproduction will decline as it takes longer to grow larger offspring when the rate of mass-specific metabolism is the same. But the reproductive rate can be maintained if metabolism is reduced so that more of the parental energy is allocated into the mass of the offspring. This selection, however, goes not only contrary to the primary selection for increased pace, but the selection is also dependent upon the organism energy that is generated by metabolism and, thus, it cannot just reduce the metabolic pace as this would eliminate the net energy that generates the selection of mass. The evolutionary solution is an inverse scaling between mass-specific metabolism and biotic time periods, as this will maintain organism energy, reproduction and metabolic pace constant on the per-generation time-scale of natural selection, while the three traits are declining in physical time with an evolutionary increase in mass.

While it is the energetic trade-off between mass, metabolism and time that makes metabolism and biotic time periods respond to the evolutionary changes in mass, it is primarily the ecological geometry of optimal density regulation that was found to determine the actual values of the response as defined by allometric exponents. The mass induced changes in metabolic pace and biotic time is affecting the foraging process, and consequently the selection for a home range that satisfies the conditions of optimal density regulation. The dependence of interactive competition and local resource exploitation on the spatial dimensionality (d) of organism behaviour is then transferred by the selected density regulation optimum to the values of the allometric exponents, with the 1/4 exponent being the 2D case of the more general $1/2d$.

5.3 Diverse allometries

While mass-rescaling selection was found to be responsible for the evolution of the often observed Kleiber scaling, it was also found that a broader understanding of allometries is dependent on the inclusion of primary selection on metabolic pace.

The $-1/4$ exponent of Kleiber scaling was found to be restricted mainly to the taxa of multicellular animals that evolve masses from intra-specific interactions in

two spatial dimensions, when these taxa diversity by species that evolve into a multitude of ecological niches. Such a diversification will allow the variation in resource handling and resource availability to dominate variation in the primary selection of metabolic pace, with final allometries that evolve primarily from mass-rescaling with a $-1/4$ exponent. The corresponding 3D exponent is $-1/6$, and a $-1/4 \leftrightarrow -1/6$ like transition is observed quite commonly between terrestrial and pelagic taxa (Table 6a).

An average exponent around 0.84 for mass-specific metabolism across the masses of prokaryotes (DeLong et al. 2010) is instead consistent with 3D selection and body mass variation that is selected by variation in the primary selection of mass-specific metabolism.

An observed decline in the exponent for mass-specific metabolism from 0.61 over zero to -0.20 in protozoa (Section 4.2) is predicted by a gradual change in the natural selection of mass; suggesting that the mass variation of the smallest protozoa is selected from primary variation in mass-specific metabolism, while the mass variation of the largest is selected from primary variation in the handling and/or density of the underlying resource. These results indicate that unicellular protozoa may evolve as a continuum that spring from the selection mechanism in prokaryotes, and undergoes a gradual change with an increase in mass towards the selection mechanism in multicellular animals.

Evidence in favour of the proposed selection of metabolism and mass is not restricted to the predicted allometries. The apparent shift in the natural selection of mass across the tree of life is studied by Witting (2017), who shows that lifeforms from virus over prokaryotes and larger unicells to multicellular animals are selected by a unidirectional unfolding of the population dynamic feed-back selection of mass. This prediction of lifeforms is not restricted to mass related transitions in allometric exponents, it includes also mass related transitions in cellularity and reproduction levels, ranging from asexually reproducing cells, to sexually and eusocially reproducing multicellular organisms (Witting 2002a, 2017). The proposed allometric model is supported also by its ability to predict the curvature of the metabolic allometry (Witting 2018b), as well as the time-bend of body mass trajectories in the fossil record (Witting 2016a), from the primary selection of mass-specific metabolism.

Inter-specific exponents that are observed across natural species may nevertheless differ from the theoretical results. As the predicted exponents evolve from the spatial dependence of density regulation on traits like metabolism, foraging speed, home range, population density and mass, it is only natural to expect some variation in the exponents. While the base-case description of the spatial density dependence (Appendix B) appears to be representative for the average exponents across a broad range of mobile organisms, we expect some variation in the density-dependent ecology and this may result in deviating allometric exponents.

Reasons for exponents that deviate from the predictions in this paper may include inter-specific interactions that bias the resource distribution with mass in local communities (Brown and Maurer 1986; Nee et al. 1991) away from the invariance that applies at scales where the competitive effects from inter-specific interactions are minimal. Small islands, let it be real islands or habitat islands on a mainland, may limit the size of home ranges causing deviations in allometric exponents. Finally, the deduction in this paper assumes some invariance in the life history, and is therefore not strictly valid across gradients where, e.g., the age of maturity is correlated with mass when measured in biotic time. Empirical examples of inter-specific exponents that deviate from the expected can be found in the tables of Peters (1983), and they are discussed by McNab (1988, 2008), Lovegrove (2000), Glazier (2010, 2015), Kolokotrones et al. (2010) and others.

5.4 Parsimonious evolution

The proposed selection was elaborated from the foraging ecology in one of the earliest deductions of allometric exponents (Witting 1995), and it explains allometric exponents from the primary selection of metabolism and mass with not even a single parameter being estimated empirically. Yet, the more widespread view has seen the metabolic exponent as a mechanical or evolutionary consequence of physiological constraints. Let it be from a surface rule in four spatial dimensions (Blum 1977), resource uptake and use at cell or body surfaces (Davison 1955; Patterson 1992; Makarieva et al. 2003), tissue demands for resources (McMahon 1973; Darveau et al. 2002), resource demand with cellular and demographic constraints (Kozlowski and Weiner 1997; Kozlowski et al. 2003a,b), resource demand and exchange (Sibly and Calow 1986; Kooijman 2000; Banavar et al. 2002a,b), geometric constraints on resource transportation systems (West et al. 1997, 1999a,b; Banavar et al. 1999; Dodds et al. 2001; Dreyer and Puzio 2001; Rau 2002; Santillán 2003), thermodynamic constraints at the molecular level (Fujiwara 2003), quantum mechanical constraints on proton and electron flow in metabolic pathways (Demetrius 2003, 2006), ecological metabolism constrained by physical limits on

metabolic fluxes across surface-areas in relation to volume dependent resource demands (Glazier 2005, 2010), or from scaling in the four dimensions of space and time (Ginzburg and Damuth 2008).

None of these alternative models explain how natural selection is supposed to generate the evolution of the required co-existence of mass and metabolism, and they do thus fail to explain the span of organisms that are a pre-condition for the existence of allometries. Several of the studies have though identified empirical 1/4 exponents for diverse physiological processes, and this may illustrate just how deep into the physiology the ecological constraints of optimal density regulation are selected. Resource transportation networks, e.g., are selected to supply the organism with energy. A potential solution is a fractal network of branching tubes (West et al. 1997) that is adjusted and optimised by natural selection to comply with a -1/4 exponent that is mass-rescaling selected by the ecological geometry of the foraging that generates the essential energy for the natural selection of mass.

Acknowledgements I am grateful to anonymous reviewers that helped me to improve the article.

Appendix

A Life history

A.1 Metabolism, mass and time

The conservation of energy is constraining the possible trait space of the life history (e.g., Charlesworth 1980; Roff 1992, 2002; Stearns 1992; Bulmer 1994; Stearns and Hoekstra 2000), with some of the essential constraints between net energy, metabolism, mass and time being described in this section.

Let us assume a somewhat simplified life history, where the age of maturity (t_m, age of first reproductive event in physical time) divides the potential lifespan (t_l) into a juvenile period (t_j from age 0 to t_m) and a potential reproductive period (t_r from t_m to t_l).

Consider then the energetics of body mass, where mass is given by the biochemical (combustion) energy of the organic matter of the mass. The mass of an adult is formed by the anabolic metabolism that is used to synthesise the organism from smaller molecules during ontogenetic growth. There is a wide range of models available to describe this growth (e.g., Brody 1945; von Bertalanffy 1957; Kleiber 1961; Kooijman 2000; West et al. 2001; Ricklefs 2003; Makarieva et al. 2004; Hou et al. 2008), and I use a rather simple allometrically oriented model with a minimum of implicit assumptions.

For simplicity reasons, I assume that the complete mass of the offspring is grown from energy that is supplied by the parents. And with the energy (ϵ_j) that is allocated to an offspring per unit physical time being used either on body growth or metabolism, the increase in mass ($\dot{w} = \mathrm{d}w/\mathrm{d}t$; SI unit J/s) at age t

$$\dot{w}_t = \epsilon_j - w_t \beta \acute{\beta}_t \tag{54}$$

is the allocated energy minus the total metabolism of the offspring, where w_t is the mass at age t, β the mass-specific metabolism of an adult, and $\acute{\beta}_t = \beta_t/\beta$ the mass-specific metabolism of an offspring with mass w_t at age t relative to that of an adult, with $\acute{\beta}_{t_m} = 1$ (I assume for simplicity that no energy is lost in the transfer of energy from the parent to the offspring).

With mass being synthesised by anabolic metabolism, the increase in mass during ontogenetic growth may also be written as a product

$$\dot{w}_t = c_{\dot{w}} w_t \beta \acute{\beta}_t \acute{\beta}_{\dot{w},t} \tag{55}$$

between the fraction ($\acute{\beta}_{\dot{w},t}$) of the total metabolism ($w_t \beta \acute{\beta}_t$) that is used for the synthesis of body mass and a growth constant ($c_{\dot{w}}$) that specifies the amount of matter that is created per joule that is used in the anabolic metabolism, with $\acute{\beta}_{\dot{w},t_m} = 0$, and $\acute{\beta}_{!\dot{w},t} = 1 - \acute{\beta}_{\dot{w},t}$ being the fraction of the offspring metabolism that is used for other purposes. For a given supply of energy (ϵ_j) and growth efficiency ($c_{\dot{w}}$), it follows from eqns 54 and 55 that the rate of ontogenetic growth at a given mass can increase only by a decline in mass-specific metabolism ($\beta \acute{\beta}_t$), with a differentiation [$\dot{w}/\mathrm{d}t > 0$] of eqn 55 showing that increased growth will require also a reallocation where more of the metabolism is used for the synthesis of mass, with $\mathrm{d}\acute{\beta}_{\dot{w},t}/\mathrm{d}t = -\mathrm{d}\acute{\beta}_{!\dot{w},t}/\mathrm{d}t > -(\acute{\beta}_{\dot{w},t}/\beta)\mathrm{d}\beta/\mathrm{d}t$ for $\mathrm{d}\beta/\mathrm{d}t < 0$.

Adult mass

$$w = \int_{t_j} \dot{w}_t \mathrm{d}t = w\beta \int_{t_j} c_{\dot{w}} \acute{w}_t \acute{\beta}_t \acute{\beta}_{\dot{w},t} \mathrm{d}t = w\beta t_j \acute{\dot{w}} \tag{56}$$

is given by the integral of growth over the juvenile period, with $\acute{w}_t = w_t/w$, and

$$\acute{\dot{w}} = \frac{1}{t_j} \int_{t_j} c_{\dot{w}} \acute{w}_t \acute{\beta}_t \acute{\beta}_{\dot{w},t} \mathrm{d}t = \dot{w}/w\beta \tag{57}$$

being a parameter that specifies the average growth in joules ($\dot{w}$) relative to the mass and mass-specific metabolism of the adult.

The juvenile period and the age of maturity are thus synchronised by ontogenetic growth (eqn 56) to be inversely related with mass-specific metabolism ($t_j = t_m = 1/\beta\acute{w}$). And with the deterioration (senescence) of organisms from the age of maturity to the potential lifespan being depend, among others, on biochemical use, the reproductive period ($t_r = s/\beta$, with s being a bauplan dependent senescence parameter) and potential lifespan [$t_l = t_r + t_m = (s + 1/\acute{w})/\beta$] are also expected to be inversely related with mass-specific metabolism. Metabolic pace may thus, as argued previously by Pearl (1928) and others like Brody (1945), Hill (1950), Stahl (1962), Calder (1984) and Witting (1997), synchronise rate dependent processes and periods over time-scales from the metabolism of the physiology, over the ecological feeding behaviour (where $\epsilon = \alpha\tilde{\beta}$), and life history periods (t_j, t_m, t_r, t_l), to the population dynamic and natural selection processes that operate on the per-generation time-scale.

Generation time $t_g = \int_{t_r} t\, m\, l_t\, \mathrm{d}t / \int_{t_r} m\, l_t\, \mathrm{d}t$ is maybe best seen as the average age of reproduction, that reduces to $t_g = \int_{t_r} t\, l_t\, \mathrm{d}t / \int_{t_r} l_t\, \mathrm{d}t$ with a constant reproductive rate m, given the probability $l_t = \prod_0^{t-1} p_t$ that an individual will survive to age t, with p_t being the probability to survive from age t to age $t+1$. Given the rate of living, generation time is also

$$t_g = \acute{t_g}/\tilde{\beta}, \tag{58}$$

with $\acute{t_g}$ being an invariant unitless scaling between generation length in physical time and metabolic pace. Biotic time

$$\tau = t/t_g \propto t\beta, \tag{59}$$

as measured in generations is running in proportion with mass-specific metabolism. The relationship between a period, or age, x of an organism in physical (t) and biotic (τ) time is thus

$$t_x = \tau_x t_g, \tag{60}$$

with $\tau_g = t_g/t_g = 1$.

A.2 Net energy and reproduction

The total energetic investment in each offspring is the final adult mass w of the offspring at the time of independence, plus the energy $w_j t_j \beta \acute{\beta}_j$ that is metabolised by the offspring during the juvenile period, i.e.,

$$\epsilon_j t_j = w + w_j t_j \beta \acute{\beta}_j = w(1 + \acute{w}_j \acute{\beta}_j / \acute{w}) = \acute{\beta} w \tag{61}$$

with $t_j = 1/\beta\acute{w}$,

$$\acute{\beta}_j = \frac{1}{t_j} \int_{t_j} \acute{\beta}_t \tag{62}$$

being the average mass-specific metabolism of the offspring relative to that of an adult,

$$w_j = \frac{1}{t_j} \int_{t_j} w_t\, \mathrm{d}t \tag{63}$$

the average mass of the offspring during t_j, $\acute{w}_j = w_j/w$ the relative mass, and

$$\acute{\beta} = 1 + \acute{w}_j \acute{\beta}_j / \acute{w} \tag{64}$$

an invariant parameter that represents the energy that is metabolised by the offspring. The total investment in each offspring is thus proportional to mass and independent of mass-specific metabolism.

The reproductive rate in physical time

$$m = \epsilon/\epsilon_j t_j = \epsilon/\acute{\beta} w, \tag{65}$$

is the net energy available for reproduction (ϵ) divided by the total energetic investment in each offspring. This generates a quality-quantity trade-off, where parents can produce many small, or a few large, offspring (Smith and Fretwell 1974; Stearns 1992). Total lifetime reproduction

$$R = t_r m = \frac{t_r}{t_j} \frac{\epsilon}{\epsilon_j} = \frac{\tau_r}{\tau_j} \frac{\epsilon}{\epsilon_j} \tag{66}$$

is thus constrained by the τ_r/τ_j and ϵ/ϵ_j ratios.

A.3 Survival

As the expected lifetime reproduction of a new-born is

$$R_0 = m \int_{t_r} l_t\, \mathrm{d}t, \tag{67}$$

we find that the probability that a new-born individual will survive the complete reproductive period is

$$p = \frac{R_0}{R} = \frac{1}{t_r} \int_{t_r} l_t\, \mathrm{d}t. \tag{68}$$

For this relation we note that when the l_t function is invariant in biotic time, i.e., when l_{t/t_r} is invariant, it follows that the $\int_{t_r} l_t\, \mathrm{d}t$ integral reduces to $c_l t_r$ where c_l, and thus also p, are invariant parameters. This is expected if survival is determined entirely by intrinsic processes.

B Density regulation

Some of the most essential constraints for the evolution of the allometric exponents are imposed by the ecological geometry of the density-dependent foraging and

interactive competition that is regulating the dynamics of the population. This was first described by Witting (1995), and his model is given here in a more general version.

The potential importance of density dependence for life history evolution has been realised since MacArthur (1962). First by the verbal formulation of r and K selection (Pianka 1970; Stearns 1976, 1977; Parry 1981). Then by its more formal mathematical theory with frequency-independent selection for an increase in r or K (Anderson 1971; Roughgarden 1971; Charlesworth 1971; Clarke 1972), as noted by Fisher (1930) when he formulated his fundamental theorem of natural selection (Witting 2000a, 2002b). It was found later that the incorporation of the frequency-dependent interactive behaviour is essential for a more general understanding of density-dependent selection (e.g. Abrams and Matsuda 1994; Mylius and Diekmann 1995; Metz et al. 1996; Witting 1997; Heino et al. 1998; Gyllenberg and Parvinen 2001; Dercole et al. 2002; Dieckmann and Metz 2006), with the density-frequency-dependent selection being essential for a realistic evolution of mass and allometries (see Witting 2017 appendix for details).

As natural populations on evolutionary time-scales are expected to be close to their population dynamic equilibria on average, it is only the selection and behaviour of density regulation in the surroundings of the population dynamic equilibrium that is essential for the allometric deduction. We are therefore not interested in functional relationships that necessarily are realist across all possible densities, but more so in descriptions that are mathematically easy to handle and useful as realistic approximations for the potential functional relationships near the equilibrium.

Density regulation is generally known to include the two components of interference (interactive) and exploitative intra-specific competition. I include a third component in the description, which is the foraging self-inhibition that is imposed by the local resource exploitation of the individual itself. The latter is not density regulation in itself, but it is needed for the natural selection of a realistic density regulation (Section B.5).

While there might be interactions between the three components that are difficult to describe by separate functions, I model the overall regulation

$$f = f_e f_\iota f_s \tag{69}$$

as a product between the three subcomponents (f_e:exploitation; f_ι:interference; f_s:local exploitation) with interactions between the subcomponents following by the constraints that the natural selection of the three subcomponents impose upon one another. This might be regarded as simplistic, but it is sufficient to capture the essential features that explain the natural selection of mass and associated allometries.

B.1 The overall regulation (f)

To formulate the overall regulation f, let the average net energy

$$\epsilon = \alpha\tilde{\beta} = \grave{\alpha}\tilde{\beta}\rho^{**} \tag{70}$$

be defined for an implicit resource (ρ^{**}) at the equilibrium population density (n^{**}) of the relevant selection attractor on mass, with $\grave{\alpha}$ being an intrinsic resource handling component that is independent of the density of the resource. This defines resource handling ($\alpha = \grave{\alpha}\rho^{**}$) by the net energy that is obtained per metabolic pace at the density-dependent equilibrium for the selection attractor on mass.

The density regulation of net resource assimilation may then be given by $\epsilon\rho/\rho^{**}$, with ρ being the density (n) regulated [$f(n)$] resource

$$\rho = \rho_u f(n) \tag{71}$$

that declines monotonically from ρ_u over ρ^{**} to zero as n increases from zero to infinity, with subscript u denoting the lower unregulated limit at $n \approx 0$, assuming no depensation. The population dynamic equilibrium abundance

$$n^* = n^*(r_u) = f^{-1}\left(1/\lambda_u\right) \tag{72}$$

is constrained by the $\lambda^* = pt_r\epsilon/\acute{\beta}w = 1$ relation of eqn 11 where $\epsilon = \epsilon_u f(n)$, and it increases monotonically with the maximal growth rate $r_u = \ln\lambda_u = \ln(p\,t_r\epsilon_u/\acute{\beta}w)$, with $\epsilon_u = \epsilon\rho_u/\rho^{**}$.

As an approximation of density regulation in the surroundings of the equilibrium I assume log-linearity

$$f(n) = f_\circ n^{-\gamma}, \tag{73}$$

where $f_\circ$ is a constant, and

$$n^* = (\acute{\epsilon_u} f_\circ/w)^{1/\gamma} = c\,\lambda_u^{1/\gamma} \tag{74}$$

is the equilibrium with $\acute{\epsilon_u} = pt_r\epsilon_u/\acute{\beta}$ and $c = f_\circ^{1/\gamma}$. This provides a simple conversion from a multiplicative regulation function on the scale of net life time reproduction ($R_0 = R_{0,u} f_\circ n^{-\gamma}$, $R_{0,u}$ unregulated R_0), to an additive function on the scale of exponential growth ($r \propto r_u - \gamma\ln n$, $r = \ln R_0$).

B.2 Exploitative competition (f_e)

Regulation by exploitative competition occurs through the reduction in resource density that is caused by the consumption of resources by the population. The correct argument in the regulation function is thus not the population density (n) per se, but a more complex expression that reflects the traits that are connected with the populations use of resources. As the energy that is used by the juvenile component (n_j) is supplied by the parental generation ($n_a = n - n_j$), the argument is the gross resource consumption (eqn 3) of the adult component

$$\begin{aligned} n_a\epsilon_g &= n_a w\beta + n_a\epsilon \\ &= n_a w\beta + n_j(w\acute{w}_j\beta\acute{\beta}_j + w/t_j) \\ &= n_a w\beta + n_j(w\beta - w\beta + w\acute{w}_j\beta\acute{\beta}_j + w\beta\acute{\dot{w}}) \\ &= nw\beta[1 + \acute{n}_j(\acute{w}_j\acute{\beta}_j - 1 + \acute{\dot{w}})], \end{aligned} \tag{75}$$

where the net energy of the adult component ($n_a\epsilon$) is used by the juvenile component (eqn 61) for metabolism ($n_j w\acute{w}_j\beta\acute{\beta}_j$) and mass ($n_j w/t_j$), with $\acute{n}_j = n_j/n$.

As it is reasonable to assume that $\acute{n}_j$, $\acute{w}_j\acute{\beta}_j$ and $\acute{\dot{w}}$ are invariant of mass, we obtain

$$\rho_e = \rho_u f_e(nw\beta) \tag{76}$$

as a general expression of the exploitation function, with f_e declining monotonically from one to zero as $nw\beta$ increases from zero to infinity, and ρ_e being the density of the exploited resource. The resource density ρ_e is different from the realised resource $\rho = \rho_u f = \rho_e f_\iota f_s$, as the latter refers to the resource component that can effectively be exploited after exploitation, the cost of interference, and local exploitation. In order to maintain the log-linearity of the overall regulation (f), I use

$$f_e \propto (nw\beta)^{-\gamma_e} \tag{77}$$

as an approximation in the surroundings of n^*, with γ_e being the density regulation parameter of exploitative competition.

B.3 Interactive competition (f_ι)

Density regulation by interactive competition reflects the costs of interference, with the cost to an individual including both the cost of losing access to resources that are controlled by competitively superior individuals, and the cost of using time and energy on competitive interactions with other individuals. As for exploitative competition, the correct argument in the regulation function is not the population density per se. It is instead the interference or, for a more detailed description, the underlying traits that are generating the interference between the individuals in the population.

Where it is the bias in the cost of interference across the individuals in the population that is essential for interactive selection, it is the average cost that is essential for density regulation. This regulation is broken up into two components. This first is the density dependence function

$$e^\iota = I = g_\iota\left(nvh^{\frac{d-1}{d}}\right) \tag{78}$$

that determines the level of interference ($I > 1$) as proportional to the frequency of the number of interactive encounters per individual per unit physical time, on ordinary (I) and log ($\iota = \ln I$) scale, from, among others, the density of the population. The second component is the regulation function

$$f_\iota(I) \tag{79}$$

that declines monotonically from one to zero as I increases from one to infinity.

The $nvh^{\frac{d-1}{d}}$ argument of eqn 78 reflects a population where the individuals forage in home ranges that are spread out in one, two or three spatial dimensions (d). The frequency of competitive encounters per individual is then expected to be proportional to the degree of overlap between the home ranges of the individuals, times the frequency by which the individuals reuse the foraging tracks within the home range. It is this frequency that defines how often the individuals will meet in overlapping areas.

Home range overlap may be defined as the average home range (h) divided by the per capita availability of space, which is proportional to $1/n$; the inverse of population density. Hence, overlap is proportional to nh.

The time between reuse of foraging tracks is the length of the tracks divided by the foraging speed (v). Given d-dimensional foraging, the length of the foraging tracks is expected to be proportional to the dth root of the home range, as empirically confirmed for mammals (Garland 1983; Calder 1984). Hence, the interval between track reuse scale as $h^{1/d}/v$. Multiplying the frequency of track reuse ($v/h^{1/d}$) with home range overlap (nh) we find the level of interference competition (eqn 78) to increase monotonically with $nvh^{\frac{d-1}{d}}$.

The interference of eqn 78 applies when the interactive behaviour of the individuals in the population is unaffected by the resource density ρ_e. While this may be expected for low energy organisms with a passive interactive behaviour, the level of interference in populations of high energy organisms might

be inversely related to the underlying resource density $[I = g_\iota \left(nvh^{\frac{d-1}{d}}/\rho_e\right)]$ given that the individuals fight more often over resources when these are limited.

Foraging speed has been found to be proportional to lifespan and other organism periods on the body mass axis across species (Garland 1983; Calder 1984), with mechanistic explanations provided by Calder (1984). We expect also that foraging speed reflects metabolic pace, suggesting that the allometric intercept of foraging speed is proportional to the intercept of mass-specific metabolism. Given these relations, we defined foraging speed as

$$v = v_\circ \beta_\beta t_w. \tag{80}$$

As an approximation in the surroundings of n^*, I use

$$\iota \propto \gamma_\iota \ln\left(nvh^{\frac{d-1}{d}}\right), \tag{81}$$

and

$$f_\iota \propto I^{-\mu} \propto e^{-\iota\mu} \propto \left(nvh^{\frac{d-1}{d}}\right)^{-\gamma_\iota\mu}, \tag{82}$$

where γ_ι is the density dependence of interference, and μ the average cost. This will maintain the log-linearity of the overall regulation f, and by inserting eqn 74 into eqn 81 we find the level of interference competition at the population dynamic equilibrium to be

$$\iota^* = (\gamma_\iota/\gamma)\ln(\acute{\epsilon}_0/w), \tag{83}$$

with $\acute{\epsilon}_0 = \acute{\epsilon_u} f_\circ \left(vh^{\frac{d-1}{d}}\right)^\gamma$. This level will decline monotonically with w, with $\overline{\iota}^* = (\gamma_\iota/\gamma)\ln(\acute{\epsilon}_0/\underline{w})$ defining the maximum from an average minimum mass $\underline{w}$.

B.4 Local resource exploitation (f_s)

The resource assimilation of an individual is also influenced by the local resource exploitation of the individual itself. The availability of food along the foraging tracks of the individual is expected to be proportional to the time interval between the individuals reuse of foraging tracks, with longer intervals allowing more time for resource re-growth and/or dispersal into the area. Above we expressed this interval as $h^{1/d}/v$.

But foraging self-inhibition by local resource exploitation is a relative term, relative to the frequency of re-harvesting when foraging tracks are infinitely long and never reused by the individual itself. In this case, re-harvesting is occurring because of the overall resource exploitation from all the individuals in the population, with a frequency that is expected proportional with biotic pace (β). When scaled accordingly we find self-inhibition expressed as

$$f_s = f_s(\beta h^{1/d}/v), \tag{84}$$

where f_s is a downward bend function that increases monotonically from zero to one as the home range increases from zero to infinity.

As an approximation in the surroundings of the evolutionary equilibrium (Section B.5) I use

$$f_s \propto c_s - (\beta h^{1/d}/v)^{-\gamma_s}, \tag{85}$$

where c_s is a scaling constant and γ_s the strength of self-inhibition. And for the density regulation approximation $f(n) = f_\circ n^{-\gamma}$, from eqns 77, 82, and 84 we have

$$\gamma = \gamma_e + \gamma_\iota\mu \tag{86}$$

and

$$f_\circ = (w\beta)^{-\gamma_e}\left(vh^{\frac{d-1}{d}}\right)^{-\gamma_\iota\mu} z f_s \tag{87}$$

where z is a scaling parameter.

B.5 Selection of density regulation

Natural selection on the exploitative component of density regulation occurs through selection on the net assimilation of energy. This represents the ability of the organism to exploit resources, and it is covered by Section 2.4.

Relating to regulation by interactive competition $[f_\iota(nvh^{\frac{d-1}{d}})]$ we note that, when this component is considered in isolation, we expect selection for home ranges that are so small that there are no interactions between individuals and no cost of interference. This does not coincide with natural conditions where interactions between individuals are common. Interactions, however, are expected because local resource exploitation $[f_s(\beta h^{1/d}/v)]$ is counteracting interference, as it increases with a decline in home range. In fact, if we were considering only local resource exploitation, we would expect selection for infinitely large home ranges with no self-inhibition.

When the joint regulation $f_\iota(nvh^{\frac{d-1}{d}})f_s(\beta h^{1/d}/v)$ of the two functions are considered together, we have that $f_\iota f_s$ is increasing initially around $h \approx 0$ where f_ι is flat around zero, and declining as $h \to \infty$ where f_s is flat around zero. The home range will thus evolve to an intermediate size where foraging is optimal and the joint regulation by self-inhibition and interference is minimal, i.e., the value of $f_\iota f_s$ is at a maximum.

This equilibrium home range is defined by the optimum

$$\frac{\partial f_\iota^{**}}{\partial h} f_s^{**} + \frac{\partial f_s^{**}}{\partial h} f_\iota^{**} = 0. \tag{88}$$

To solve, apply

$$f_\iota \propto \left(Yh^{(d-1)/d}\right)^{-\gamma_i} \tag{89}$$

with $Y = nv$, $\gamma_i = \gamma_\iota\mu$, and

$$f_s \propto c_s - (Sh^{1/d})^{-\gamma_s} \tag{90}$$

with $S = \beta/v$, as local approximations. Then,

$$\partial f_\iota/\partial h = Y^{-\gamma_i} h^{\frac{\gamma_i(1-d)}{d}} h^{-1} \frac{\gamma_i(1-d)}{d} \tag{91}$$

and

$$\partial f_s/\partial h = S^{-\gamma_s} h^{\frac{-\gamma_s}{d}} h^{-1} \frac{\gamma_s}{d}. \tag{92}$$

Hence, from eqn 88, we have

$$X\left[\frac{\gamma_i(1-d)}{d}\left(c_s - S^{-\gamma_s}h^{\frac{-\gamma_s}{d}}\right) + S^{-\gamma_s}h^{\frac{-\gamma_s}{d}}\frac{\gamma_s}{d}\right] = 0 \tag{93}$$

with

$$X = Y^{-\gamma_i} h^{\frac{\gamma_i(1-d)}{d}} h^{-1}. \tag{94}$$

Then, divide eqn 93 with X on both sides, rearrange, and substitute $\gamma_i = \gamma_\iota\mu$ and $S = \beta/v$ to find the optimal home range

$$h^{**} = \left(\frac{v}{\beta}\right)^d \left(\frac{\gamma_s + \gamma_\iota\mu(d-1)}{\gamma_\iota\mu(d-1)c_s}\right)^{d/\gamma_s}. \tag{95}$$

For the four traits (v, β, n, h) in the density regulation functions of interference and local resource exploitation, we find the home range (h) of the density regulation optimum to be density (n) independent, being dependent only on foraging speed (v) and mass-specific metabolism (β).

If we inset eqn 95 into eqn 84 we find that the local resource exploitation at the foraging optimum (f_s^{**}) is independent of β, h and v, being dependent only on the other parameters of eqn 95. These are not part of the phenotype and are therefore not modified by natural selection (at least not directly), and self-inhibition at the foraging optimum is therefore expected to be invariant of the life history. The invariant local resource exploitation of optimal regulation will have an invariant derivative ($\partial f_s^{**}/\partial h^{**}$), implying that eqn 88 reduces to

$$\partial f_\iota^{**}/\partial h^{**} = -c_o f_\iota^{**} \tag{96}$$

where $c_o = \partial f_s^{**}/\partial h^{**} f_s^{**}$ is invariant. Hence, for a given functional relation $f_\iota(\iota)$, we have invariant interference, and with interference in at least high-energy organisms being likely to reflect the exploitation level of the resource (ρ_e), we may expect exploitation $f_e(nw\beta)$ and regulation as a whole $f = f_e f_\iota f_s$ to be body mass invariant; generating natural selection for a trait covariance

$$nvh^{\frac{d-1}{d}} \propto \beta h^{1/d}/v \propto nw\beta \propto w^0 \tag{97}$$

that will leave the regulation optimum invariant of the life history.

Note that it is only the inter-specific variation in the regulation at the population dynamic equilibrium (the values of f^* across populations) that is predicted to be invariant of the life history when the regulation parameters γ_e, γ_ι and γ_s are invariant. The density regulation response, as expressed across a gradient of densities within a population, is not invariant of the life history. The regulation response by exploitation, e.g., is stronger for the same density in species with a larger body mass or a larger mass-specific metabolism (see eqn 76).

C Replication invariance

One way of illustrating the evolutionary importance of the mass-rescaling allometries is to focus on the average replication in a population as a function of the average mass [$pR^* = f(w)$] for a range of potential mass-rescaling options (variation in $\hat{t}_w = -\hat{\beta}_w$). Of these it is only those with an equilibrium per-generation replication rate of one ($pR^* = 1$) that may actually be selected by natural selection.

To construct this function let $\hat{t}_w^{**}$ be the mass-rescaling exponent of the allometric solution (Section 3.1), let $\hat{t}_w$ be a chosen rescaling exponent, and let the mass dependence of the juvenile period in biotic time be $\tau_j = \tau_{j,\circ} w^{\hat{\tau}_j}$, noting that this dependence in positive ($\hat{\tau}_j = z > 0$, eqn 30) in the absence of mass-rescaling ($\hat{t}_w = 0$), and invariant ($\hat{\tau}_j = 0$) at the allometric solution ($\hat{t}_w^{**}$, eqn 31). Hence, assuming local linearity, we find the exponent of the juvenile period $\hat{\tau}_j = z(1 - \hat{t}_w/\hat{t}_w^{**})$ as a function of the chosen mass-rescaling exponent $\hat{t}_w$. And with net energy ($\epsilon = \alpha\tilde{\beta}$) being a function of resource handling (α) and metabolic pace ($\tilde{\beta} \propto 1/t$), the resource handling exponent (eqn 25) is also a function of the chosen mass-rescaling with $\hat{\alpha} = \hat{\epsilon} + \hat{t}_w$. Hence, given physiological invariant survival (eqn 32), the per-generation replication rate

$$pR = \frac{pt_r\alpha\tilde{\beta}}{w(1 - \acute{w}_j\tau_j)} \propto \frac{p\alpha(\tau_l - \tau_j)}{w(1 - \acute{w}_j\tau_j)} \tag{98}$$

is a function of w given $\hat{t}_w$ and z, with

$$pR \propto \frac{p\alpha_\circ w^{\hat{\epsilon}-\hat{t}_w}\left[\tau_l - \tau_{j,\circ} w^{z(1-\hat{t}_w/\hat{t}_w^{**})}\right]}{w\left[1 + \acute{w}_j \tau_{j,\circ} w^{z(1-\hat{t}_w/\hat{t}_w^{**})}\right]}. \quad (99)$$

This rate is either declining or increasing with the average mass for all possible mass-rescaling options except for the allometric solution, where the per-generation replication is invariant of mass (Fig. 2).

References

Abrams P. A. Matsuda H. (1994). The evolution of traits that determine ability in competitive contests. Evol. Ecol. 8:667–686.

Anderson K. J. Jetz W. (2005). The broad-scale ecology of energy expenditure of endotherms. Ecol. Lett. 8:310–318.

Anderson W. W. (1971). Genetic equilibrium and population growth under density-regulated selection. Am. Nat. 105:489–498.

Artacho P. Nespolo R. F. (2009). Natural selection reduces energy metabolism in the garden snail, *Helix aspersa* (*Cornu Aspersum*). Evolution 63:1044–1050.

Baltscheffsky, H., ed (1996). Origin and Evolution of Biological Energy Conversion. VCH Publishers, New York.

Banavar J. R., Damuth J., Maritan A., Rinaldo A. (2002a). Modelling universality and scaling. Nature 420:626.

Banavar J. R., Damuth J., Maritan A., Rinaldo A. (2002b). Supply-demand balance and metabolic scaling. Proc. Nat. Acad. Sci. USA 99:10506–10509.

Banavar J. R., Maritan A., Rinaldo A. (1999). Size and form in efficient transportation networks. Nature 399:130–132.

Barve A., Hosseini S. R., Martin O. C., Wagner A. (2014). Historical contingency and the gradual evolution of metabolic properties in central carbon and genome-scale metabolisms. BMC Syst. Biol. 8:48.

Blum J. J. (1977). On the geometry of four-dimensions and the relationship between metabolism and body mass. J. theor. Biol. 64:599–601.

Bonner J. T. (1965). Size and cycle. Princeton University Press, Princeton.

Boratynski Z., Koskela E., Mappes T., Oksanen T. A. (2010). Sex-specific selection on energy metabolism selection coefficients for winter survival. J. Evol. Biol. 23:1969–1978.

Bozinovic F., Muñoz J. L. P., Croz-Neto A. P. (2007). Intraspecfic variability in the basal metabolic rate: testing the food habits hypothesis. Physiolo Biochem Zool 80:452–460.

Bozinovic F., Rojas J. M., Broitman B. R., Vasquez R. A. (2009). Basal metabolism is correlated with habitat productivity among populations of degus (*Octodon degus*). Comp. Bioch. Physiol. A. 152:560–564.

Brody S. (1945). Bioenergetics and growth. Hafner, New York.

Brown J. H., Gillooly A. P., Allen V. M., Savage G. B. (2004). Towards a metabolic theory of ecology. Ecology 85:1771–1789.

Brown J. H. Maurer B. A. (1986). Body size, ecological dominance and cope's rule. Nature 324:248–250.

Brown J. H. Sibly R. M. (2006). Life-history evolution under a production constraint. Proc. Nat. Acad. Sci. 103:17595–17599.

Bulmer M. (1994). Theoretical evolutionary ecology. Sinauer Associates Publishers, Massachusetts.

Calder W. A. I. (1984). Size, function, and life history. Harvard University Press, Cambridge.

Capellini I., Venditti C., Barton R. A. (2010). Phylogeny and metabolic scaling in mammals. Ecology 91:2783–2793.

Careau V., Morand-Ferron J., Thomas D. (2007). Basal metabolic rate of canidae from hot deserts to cold arctic climates. J. Mamm. 88:394–400.

Charlesworth B. (1971). Selection in density-regulated populations. Ecology 52:469–474.

Charlesworth B. (1980). Evolution in age-structured populations. Cambridge University Press, Cambridge.

Charlesworth B. (1994). Evolution in age-structured populations. 2nd edn. Cambridge University Press, Cambridge.

Chela-Flores, J., Chadha, M., Negrón-Mendoza, A., & Oshima, T., eds (1995). Chemical Evolution: Self-Organization of the Macromolecules of Life. A. Deepak Publishing, Hampton.

Clarke B. (1972). Density-dependent selection. Am. Nat. 106:1–13.

Cunchilos C. Lecointre G. (2003). Evolution of amino acid metabolism inferred through cladistic analysis. J. Biol. Chemi. 278:47960–47970.

Currie D. J. Fritz J. T. (1993). Global patterns of animal abundance and species energy use. Oikos 67:56–68.

Damuth J. (1981). Population density and body size in mammals. Nature 290:699–700.

Damuth J. (1987). Interspecific allometry of population density in mammals and other animals: the independence of body mass and population energy-use. Biol. J. Linnean Society. 31:193–246.

Darveau C.-A., Suarez K. S., Andrews R. D., Hochachka P. W. (2002). A general model for ontogenetic growth. Nature 417:166–170.

Davison J. (1955). Body weight, cell surface and metabolic rate in anuran Amphibia. Biol. Bull. 109:407–419.

De Magalhães J. P., Costa J., Church G. M. (2007). An analysis of the relationship between metabolism, developmental schedules, and longevity using phylogenetic independent contrasts. J. Geron. Biol. Sci. 62A:149–

160.

Deeds E. J. (2011). Curvature in metabolic scaling: A reply to MacKay. J. theor. Biol. 280:197–198.

DeLong J. P. (2014). The body-size dependence of mutual interference. Biol. Lett. 10:20140261.

DeLong J. P., Okie J. G., Moses M. E., Sibly R. M., Brown J. H. (2010). Shifts in metabolic scaling, production, and efficiency across major evolutionary transitions of life. Proc. Nat. Acad. Sci. 107:12941–12945.

DeLong J. P. Vasseur D. A. (2011). Mutual interefrence is common and mostly intermediate in magnitude. BMC Ecology 11:1.

Demetrius L. (2003). Quantum statistics and allometric scaling of organisms. Physica 322:477–490.

Demetrius L. (2006). The origin of allometric scaling laws in biology. J. theor. Biol. 243:455–467.

Dercole F., Ferriére R., Rinaldi S. (2002). Ecological bistability and evolutionary reversals under asymmetrical competition. Evolution 56:1081–1090.

Dieckmann U. Metz J. A. J. (2006). Surprising evolutionary predictions from enhanced ecological realism. Theor. Pop. Biol. 69:263–281.

Dodds P. S., Rothman D. H., Weitz J. S. (2001). Re-examination of the "3/4-law" of metabolism. J. theor. Biol. 209:9–27.

Dreyer O. Puzio R. (2001). Allometric scaling in animals and plants. J. Math. Biol. 43:144–156.

Duncan R. P., Forsythe D. M., Hone J. (2007). testing the metabolic theory of ecology: allometric scaling exponents in mammals. Ecology 88:324–333.

Ehnes R. B., Rall B., Brose U. (2011). Phylogenetic grouping, curvature and metabolic scaling in terrestrial invertebrates. Ecol. Lett. 14:993–1000.

Fenchel T. (1974). Intrinsic rate of natural increase: The relationship with body size. Oecologia 14:317–326.

Fernando C. Rowe J. (2007). Natural selection in chemical evolution. J. theor. Biol. 247:152–167.

Fernando C. Rowe J. (2008). The origin of autonomous agents by natural selection. Bio Systems 2:355–373.

Ferry J. G. House C. H. (2006). The stepwise evolution of early life driven by energy conservation. Mol. Biol. Evol. 23:1286–1292.

Fisher R. A. (1930). The genetical theory of natural selection. Clarendon, Oxford.

Fry I. (2011). The role of natural selection in the origin of life. Origins Life Evol. Bios. 41:3–16.

Fujiwara N. (2003). Origin of the scaling rule for fundamental living organisms based on thermodynamics. BioSys. 70:1–7.

Garland T. (1983). Scaling the ecological cost of transport to body mass in terrestrial mammals. Am. Nat. 121:571–587.

Genoud M. (2002). Comparative studies of basal rate of metabolism in Primates. Evol. Anthro. (Suppl) 1:108–111.

Gillooly J. F., Brown J. H., West G. B., Savage V. M., Charnov E. L. (2001). Effects of size and temperature on metabolic rate. Science 293:2248–2251.

Ginzburg L. Damuth J. (2008). The space-lifetime hypothesis: viewing organisms in four dimensions, literally. Am. Nat. 171:125–131.

Glazier D. S. (2005). Beyond the '3/4-power law': variation in the intra- and interspecific scaling of metabolic rate in animals. Biol. Rev. 80:611–662.

Glazier D. S. (2008). Effects of metabolic level on the body size scaling of metabolic rate in birds and mammals. Proc. R. Soc. B. 22:821–828.

Glazier D. S. (2009). Metabolic level and size scaling of rates of respiration and growth in unicellular organisms. Funct. Ecol. 23:963–968.

Glazier D. S. (2010). A unifying explanation for diverse metabolic scaling in animals and plants. Biol. Rev. 85:111–138.

Glazier D. S. (2015). Is metabolic rate a universal 'pacemaker' for biological processes? Biol. Rev. 90:377–407.

Gyllenberg M. Parvinen K. (2001). Necessary and sufficient conditions for evolutionary suicide. Bull. Math. Biol. 63:981–993.

Haldane J. B. S. (1954). The origins of life. New Biol. 16:12–27.

Hayssen V. Lacy R. C. (1985). Basal metabolic rates in mammals: taxonomic differences in the allometry of BMR and body mass. Comp. Bioch. Physiol. 81A:741–754.

Heino M., Metz J. A. J., Kaitala V. (1998). The enigma of frequency-dependent selection. Trends Ecol. Evol. 13:367–370.

Hill A. V. (1950). the dimensions of animals and their muscular dynamics. Science of Computer Programming 38:209–230.

Horowitz N. H. (1945). On the Evolution of Biochemical Syntheses. Proc. Nat. Acad. Sci. 31:153–157.

Hou C., Zuo W., Moses M. E., Woodruff W. H., Brown J. H., West G. B. (2008). Energy uptake and allocation during ontogeny. Science 322:736–739.

Humphries M. M. McCann K. S. (2014). Metabolic constraints and currencies in animal ecology. Metabolic ecology. J. Anim. Ecol. 83:7–19.

Isaac N. J. B. Carbone C. (2010). Why are metabolic scaling exponents so controversial? Quantifying variance and testing hypotheses. Ecol. Lett. 13:728–735.

Jetz W., Freckleton R. P., McKechnie A. E. (2007). Environment, migratory tendency, phylogeny and basal metabolic rate in birds. PLOS One 3:e3261.

Kabat A. P., Blackburn T. M., McKechnie A. E., Butler P. J. (2008). Phylogenetic analysis of the allometric scaling of therapeutic regimes for birds. J. Zool. 275:359–367.

Kiørboe T. Hirst A. G. (2014). Shifts in mass scaling of respiration, feeding, and growth rates across life-

form transitions in marine pelagic organisms. Am. Nat. 183:E118–E130.

Kleiber M. (1932). Body and size and metabolism. Hilgardia 6:315–353.

Kleiber M. (1961). The fire of life. Wiley, New York.

Kolokotrones T., Savage V., Deeds E. J., Fontana W. (2010). Curvature in metabolic scaling. Nature 464:753–756.

Kooijman S. A. L. M. (2000). Dynamic energy and mass budgets in biological systems. Cambridge University Press, Cambridge.

Kozlowski J., Konarzewski M., Gawelczyk A. T. (2003a). Cell size as a link between noncoding DNA and metabolic rate scaling. Proc. Nat. Acad. Sci. USA 100:14080–14085.

Kozlowski J., Konarzewski M., Gawelczyk A. T. (2003b). Intraspecific body size optimization produce interspecific allometries. In: Blackburn T. M. Gaston K. J. (eds). Macroecology: Concepts and consequences: Blackwell, Malden, Massachusetts, pp 299–320.

Kozłowski J. Weiner J. (1997). Interspecific allometries are by-products of body size optimization. Am. Nat. 149:352–380.

Lotka A. J. (1922). Contribution to the energetics of evolution. Proc. Nat. Acad. Sci. 8:147–151.

Lovegrove B. G. (2000). The zoogeography of mammalian basal metabolic rate. Am. Nat. 156:201–219.

Lovegrove B. G. (2003). The influence of climate on the basal metabolic rate of small mammals: a slow-fast metabolic continuum. J. Comp. Physiol. B. 173:87–112.

MacArthur R. H. (1962). Some generalized theorems of natural selection. Proc. Nat. Acad. Sci. USA 46:1893–1897.

MacKay N. J. (2011). Mass scale and curvature in metabolic scaling. J. theor. Biol. 280:194–196.

Makarieva A., Gorshkov V. G., Li B.-L. (2003). A note on metabolic rate dependence on body size in plants and animals. J. theor. Biol. 221:301–307.

Makarieva A. M., Gorshkov V. G., Bai-Lian L. (2004). Ontogenetic growth: models and theory. Ecol. Model. 176:15–26.

Makarieva A. M., Gorshkov V. G., Bai-Lian L. (2005). Energetics of the smallest: do bacteria breathe at the same rate as whales. Proc. R. Soc. B. 272:2219–2224.

Makarieva A. M., Gorshkov V. G., Li B., Chown S. L., Reich P. B., Gavrilov V. M. (2008). Mean mass-specific metabolic rates are strikingly similar across life's major domains: Evidence for life's metabolic optimum. Proc. Nat. Acad. Sci. 105:16994–16999.

Marakushev S. A. Belonogova O. V. (2013). The origin of ancestral bacterial metabolism. Paleo. J. 47:1001–1010.

Maynard Smith J. Szathmáry E. (1995). The major transitions in evolution. W.H. Freeman Spektrum, Oxford.

McMahon T. A. (1973). Size and shape in biology. Science 179:1201–1204.

McNab B. K. (1988). Complications unherent in scaling the basal rate of metabolism in mammals. Quart. Rev. Biol. 63:25–54.

McNab B. K. (2003). Standard energetics of phyllostomid bats: the inadequacies of phylogenetic-contrast analyses. Comp. Bioch. Physiol. A. 135:357–368.

McNab B. K. (2008). An analysis of the factors that influence the level and scaling of mammalian BMR. Comp. Bioch. Physiol. A 151:5–28.

McNab B. K. Morrison P. (1963). Body temperature and metabolism in subspecies of *Peromyscus* from arid and mesic environments. Ecol. Monogr. 33:63–82.

Melendez-Hevia E., Montero-Gomez N., Montero F. (2008). From prebiotic chemistry to cellular metabolism The chemical evolution of metabolism before Darwinian natural selection. J. theor. Biol. 252:505–519.

Metz J. A. J., Mylius S. D., Diekmann O. (1996). When does evolution optimize? On the relation between types of density dependence and evolutionary stable life history parameters. IIASA WP:96–04.

Miller S. L. (1953). A production of amino acids under possible primitive Earth conditions. Science 117:528–529.

Mueller P. Diamond J. (2001). Metabolic rate and environmental productivity: well-provisioned animals evolved to run and idle fast. Proc. Nat. Acad. Sci. 98:12551–12554.

Muñoz-Garcia A. Williams J. B. (2005). Basal metabolic rate in carnivores is associated with diet after controlling for phylogeny. Physiolo Biochem Zool 78:1039–1056.

Mylius S. D. Diekmann O. (1995). On evolutionarily stable life histories, optimization and the need to be specific about density dependence. Oikos 74:218–224.

Nagy K. A. (2005). Field metabolic rate and body zise. J. Exp. Biol. 208:1621–1625.

Nee S., Read A. F., Greenwood J. J. D., Harvey P. H. (1991). The relationship between abundance and body size in british birds. Nature 351:312–313.

Niven J. E. Scharlemann J. P. W. (2005). Do insect metabolic rates at rest and during flight scale with body mass? Biol. Lett. 1:346–349.

Nowak R. M. (1991). Walker's mammals of the world volume I–II. 5th edn. The Johns Hopkins University Press, Baltimore.

Oparin, A. I. & Clark, F., eds (1959). The origin of life on Earth. Pergamon Press, New York.

Packard G. C. Birchard G. F. (2008). Traditional allometric analysis fails to provide a valid perdictive model for mammalian metabolic rates. J. Exp. Biol. 211:3581–3587.

Padfield D., Yvon-Durocher G., Buckling A., Jennings S., Yvon-Durocher G. (2016). Rapid evolution of metabolic traits explains thermal adaptation in phytoplankton. Ecol. Lett. 19:133–142.

Parry G. D. (1981). The meanings of r- and k-selection. Oecologia 48:260–264.

Patterson M. R. (1992). A mass transfer explanation of metabolic scaling relations in some aquatic invertebrates and algae. Science 255:1421–1423.

Pawar S., Dell A. I., Savage V. M. (2012). Dimensionality of consumer search space drives trophic interaction strengths. Nature 486:485–489.

Pearl R. (1928). The rate of living. Alfred A. Knopf, New York.

Peters R. H. (1983). The ecological implication of body size. Cambridge University Press, Cambridge.

Pianka E. R. (1970). On r- and k-selection. Am. Nat. 104:592–596.

Ponnamperuma, C. & Chela-Flores, J., eds (1993). Chemical Evolution: Origin of Life. A. Deepak Publishing, Hampton.

Rau A. R. P. (2002). Biological scaling and physics. J. Biosci. 27:475–478.

Ricklefs R. E. (2003). Is rate of ontogenetic growth constrained by resource supply or tissu growth potentiel? A comment on West *et al.*'s model. Funct. Ecol. 17:384–393.

Robertson A. (1968). The spectrum of genetic variation. In: Lewontin R. C. (ed). Population Biology and Evolution: Syracuse University Press, New York, pp 5–16.

Roff D. A. (1992). The evolution of life histories. Theory and analysis. University of Chicago Press, New York.

Roff D. A. (2002). Life history evolution. Sinauer Associates, Inc., Massachusetts.

Roughgarden J. (1971). Density-dependent natural selection. Ecology 5:453–468.

Santillán M. (2003). Allometric scaling law in a simple oxygen exchanging network: possible implications on the biological allometric scaling laws. J. theor. Biol. 223:249–257.

Savage V. M., Gillooly J. F., Wooduff W. H., West G. B., Allen A. P., Enquist B. J., Brown J. H. (2004). The predominance of quarter-power scaling in biology. Funct. Ecol. 18:257–282.

Schoener T. W. (1968). Sizes of feeding territories among birds. Ecology 49:123–131.

Sibly R. M., Brown J. I., Kodric-Brown A. (2012). Metabolic ecology: A scaling approach. John Wiley & Sons, Chichester.

Sibly R. M. Calow P. (1986). Physiological ecology of animals. Blackwell, Oxford.

Sieg A. E., O'Connor M. P., McNair J. N., Grant B. W., Agosta S. J., Dunham A. E. (2009). Mammalian metabolic allometry: do intraspecific variation, phylogeny, and regression models matter? Am. Nat. 175:720–733.

Smith C. C. Fretwell S. D. (1974). The optimal balance between size and number of offspring. Am. Nat. 108:499–506.

Stahl W. R. (1962). Similarity and dimensional methods in biology. Science 137:205–212.

Stearns S. C. (197). The evolution of life-history traits: A critique of the theory and a review of the data. Ann. Rev. Ecol. Syst. 8:145–171.

Stearns S. C. (1976). Life-history tactics: A review of the ideas. Quart. Rev. Biol. 51:3–47.

Stearns S. C. (1992). The evolution of life histories. Oxford University Press, Oxford.

Stearns S. C. Hoekstra R. F. (2000). Evolution: an introduction. Oxford University Press, Oxford.

Taylor P. D. (1996). The selection differential in quantitative genetics and ess models. Evolution 50:2106–2110.

Turner F. B., Jennrich R. I., Weintraub J. D. (1969). Home range and body size of lizards. Ecology 50:1076–1081.

Versteegh M. A., Schwabl I., Jaquier S., Tieleman B. I. (2012). Do immunological, endocrine and metabolic traits fall on a single Pace-of-Life axis? Covariation and constraints among physiological systems. J. Evol. Biol. 25:1864–1876.

von Bertalanffy L. (1957). Quantitative laws in metabolism and growth. Quart. Rev. Biol. 32:217–231.

Weibel E. R., Bacigalupe L. D., Schmitt B., Hoppeler H. (2004). Allometric scaling of maximal metabolic rate in mammals: muscle aerobic capacity as determinant factor. Resp. Physiol. Neurobiol. 140:115–132.

West G. B., Brown J. H., Enquist B. J. (1997). A general model for the origin of allometric scaling laws in biology. Science 276:122–126.

West G. B., Brown J. H., Enquist B. J. (1999a). A general model for the structure and allometry of plant vascular systems. Nature 400:664–667.

West G. B., Brown J. H., Enquist B. J. (1999b). The fourth dimension of life: Fractal geometry and allometric scaling of organisms. Science 284:1677–1679.

West G. B., Brown J. H., Enquist B. J. (2001). A general model for ontogenetic growth. Nature 413:628–631.

White C. R., Blackburn T. M., Martin G. R., Butler P. J. (2007). Basal metabolic rate of birds is associated with habitat temperature and precipitation, not primary productivity. Proc. R. Soc. B. 274:287–293.

White C. R., Blackburn T. M., Seymour R. S. (2009). Phylogenetically informed analysis of the allometry of mammalian basal metabolic rate supports neither geometric nor quarter-power scaling. Evolution 63:2658–2667.

White C. R., Cassey P., Blackburn T. M. (2007). Allometric exponents do not support a universal metabolic allometry. Ecology 88:315–323.

White C. R. Kearney M. R. (2013). Determinants of interspecific variation in basal metabolic rate. J. Comp. Physiol. B. 183:1–26.

Wikelski M., Spinnery L., Schelsky W., Scheuerlein A., Gwinner E. (2003). Slow pace of life in tropical sedentary birds: a common-garden experiment on four stonechat populations from different latitudes. Proc. R.

Soc. B. 270:2383–2388.

Withers P. C., Cooper C. E., Larcombe A. N. (2006). Enviromental correlates of physiological variables in marsupials. Physiolo Biochem Zool 79:437–453.

Witting L. (1995). The body mass allometries as evolutionarily determined by the foraging of mobile organisms. J. theor. Biol. 177:129–137, https://doi.org/10.1006/jtbi.1995.0231.

Witting L. (1997). A general theory of evolution. By means of selection by density dependent competitive interactions. Peregrine Publisher, Århus, 330 pp, URL http://mrLife.org.

Witting L. (1998). Body mass allometries caused by physiological or ecological constraints? Trends Ecol. Evol. 13:25, https://doi.org/10.1016/S0169–5347(97)01269–X.

Witting L. (2000). Interference competition set limits to the fundamental theorem of natural selection. Acta Biotheor. 48:107–120, https://doi.org/10.1023/A:1002788313345.

Witting L. (2002a). From asexual to eusocial reproduction by multilevel selection by density dependent competitive interactions. Theor. Pop. Biol. 61:171–195, https://doi.org/10.1006/tpbi.2001.1561.

Witting L. (2002b). Two contrasting interpretations of Fisher's fundamental theorem of natural selection. Comm. Theor. Biol. 7:1–10.

Witting L. (2003). Major life-history transitions by deterministic directional natural selection. J. theor. Biol. 225:389–406, https://doi.org/10.1016/S0022–5193(03)00274–1.

Witting L. (2008). Inevitable evolution: back to *The Origin* and beyond the 20th Century paradigm of contingent evolution by historical natural selection. Biol. Rev. 83:259–294, https://doi.org/10.1111/j.1469–185X.2008.00043.x.

Witting L. (2016). The natural selection of metabolism bends body mass evolution in time. Preprint at bioRxiv http://dx.doi.org/10.1101/088997.

Witting L. (2017). The natural selection of metabolism and mass selects lifeforms from viruses to multicellular animals. Ecol. Evol. 7:9098–9118, http://dx.doi.org/10.1002/ece3.3432.

Witting L. (2018). The natural selection of metabolism explains curvature in allometric scaling. Oikos 127:991–1000, http://dx.doi.org/10.1111/oik.05041.