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COMMENTARY

CENTENARY ARTICLE

Optimisation and constraint: explaining metabolic patterns in biology

Craig R. White*,‡ and Dustin J. Marshall*

ABSTRACT

Constraint-based explanations have dominated theories of size-related patterns in nature for centuries. Explanations for metabolic scaling – the way in which metabolism changes with body mass – have been based on the geometry of circulatory networks through which resources are distributed, the need to dissipate heat produced as a by-product of metabolic processes, and surface-area-to-volume constraints on the flux of nutrients or waste. As an alternative to these constraint-based approaches, we recently developed a new theory that predicts that metabolic allometry arises as a consequence of the optimisation of growth and reproduction to maximise fitness within a finite life. Our theory is free of physical geometric constraints that limit the possibilities available to evolution, and we therefore argue that metabolic allometry can be explained without the need to invoke any of the assumed constraints traditionally imposed by metabolic theories. Our findings also suggest that metabolism, growth and reproduction have co-evolved to maximise fitness (i.e. lifetime reproduction) and that the observed patterns in these fundamental characteristics of life can similarly be explained by optimisation rather than constraint. In this Centenary Commentary, we present an overview of our approach and a critique of its limitations. We propose a suite of empirical tests that we hope will move the field forward, discuss the dangers of model overparameterisation and highlight the need to remain open to non-adaptive hypotheses for the origin of biological patterns.

KEY WORDS: Evolutionary physiology, Growth, Metabolic rate, Metabolic theory, Optimisation, Scaling

Introduction

Explanations for patterns of allometric scaling (see Glossary) in biology date back to at least the time of Galileo Galilei (1638), who observed that the mass of an animal's skeleton must increase out of proportion with the mass of its body, because the cross-sectional area – not the mass – of a bone must increase in proportion with the weight that it supports. This simple explanation, based on physical constraints imposed by area–volume relationships, exemplifies the dominant approach taken to the understanding of size-related patterns in nature: hypotheses rooted in the first principles of chemistry, geometry and physics. The invocation of physical constraints has dominated the field of metabolic scaling for nearly 200 years. The most prominent examples include theories based on the geometry of circulatory networks through which resources are distributed (West et al., 1997, 1999), the need to dissipate heat produced as a by-product of metabolic processes (Sarrus and Rameaux, 1839; Glazier, 2010; Speakman and Król, 2010), or surface-area-to-volume constraints on the flux of nutrients or waste (Pauly, 1979, 1997, 2010; Kooijman, 1986, 2010; Hirst et al., 2014).

In a recent paper, we offered an alternative to physical-constraint views on the origin of ontogenetic metabolic scaling (see Glossary) in animals (White et al., 2022). We proposed that the invocation of physical constraints is unnecessary for explaining why aerobic metabolic rate scales allometrically with body mass, and developed an optimality model that predicts that individual lifetime reproduction (our proxy of Darwinian fitness) is often maximised when metabolic rate scales allometrically. Our findings also suggested that metabolism, growth and reproduction have co-evolved to maximise fitness and that the observed patterns in them can similarly be explained by optimisation within the bounds of absolute constraints.

Although this idea is contentious, our purpose here is not to argue the relative merits of constraint and optimality approaches, or to critique alternatives to our approach. Constraint-based approaches are well established in the literature, and discussion about the relative merits of constraint and optimality approaches is already taking place elsewhere (Kearney, 2019, 2021; Marshall and White, 2019a,b; Pauly, 2019, 2021, 2022; White and Marshall, 2019; Kooijman, 2020; Kozłowski et al., 2020; Atkinson et al., 2022; Glazier, 2022; Froese and Pauly, 2023; Kearney and Jusup, 2023; White et al., 2023). Instead, we draw attention to the potential utility of optimality approaches by first presenting a brief overview and critique of our own approach and model, and then describing the empirical work that our optimality approach might inspire. In the spirit of a Centenary article, which celebrates the past and looks to the future, we hope that this serves as an introduction to the optimality modelling approach, demonstrates how the development of such a model can provide a guide to empirical research and encourages others to explore the evolution of physiological traits in a similar way.

A life-history optimisation model for metabolic scaling

Our model is built on a simple energy expenditure budget (see Box 1 for a technical description of the model). The model assumes that, when animals are inactive and post-absorptive, they allocate a fixed proportion of their metabolic activity to production. Before reproductive maturation, all of the energy expended on production is allocated to the energy cost(s) of somatic production (growth). Following reproductive maturation, the energy expended on production is divided among the energy costs of both growth and reproduction. Animals reach their maximum size and cease growing when no energy is allocated to growth. In such a framework, the ontogenetic trajectories of metabolism, growth and reproduction are viewed as emerging as an ultimate consequence of selection to maximise fitness, and as a proximate outcome of genetically regulated developmental programmes.

School of Biological Sciences and Centre for Geometric Biology, Monash University, Clayton 3800, VIC 3800, Australia.
*These authors contributed equally to this work

‡Author for correspondence (craig.white@monash.edu)

C.R.W., 0000-0002-0200-2187

Glossary

Allometric scaling
A non-proportional relationship between a trait and body mass. Scaling relationships are typically described using power functions; when the scaling exponent of such a function differs from 1, the relationship is considered to be allometric. For example, metabolic rate typically scales allometrically with body mass, with an exponent of around 0.75 in metazoans. Scaling is referred to as hyperallometric or hypoallometric when the scaling exponent is greater than or less than 1, respectively.

Basal metabolic rate
The metabolic rate of an adult, non-reproductive, inactive, unstressed, postprandial endotherm that is thermoregulating in a thermoneutral environment during the inactive phase of its circadian cycle (see standard metabolic rate for a comparable state for ectotherms).

Carrying capacity
Denoted by the symbol *K*, carrying capacity is a demographic parameter that is defined as the maximum density of a population at which the growth rate of the population is zero because rates of birth and death are equivalent.

Density dependence
The relationship between population density and some metric of organismal performance or population growth.

Evolutionary metabolic scaling
The among-species relationship between metabolic rate and body mass.

Integral projection models
A class of demographic models that uses continuous-state variables, such as body size or metabolism, to model population dynamics including demographic parameters such as intrinsic rates of increase, population growth rate and carrying capacity. These models use information about how an individual's state (e.g. size, metabolism) affects the performance (survival, growth and reproduction) of that individual to make projections about populations. They are a useful bridge between empiricism and theory.

Life-history theory
An analytical framework for understanding how different components of an organism's life affect the overall performance of that organism. The theory considers how evolutionary optimisation has shaped the way in which resources are allocated to the various functions of life. Life-history theory often focuses on the fitness consequences of different allocations – for example, growth versus reproduction, producing many small versus few large offspring. While life-history theory has a long history, it is increasingly being applied to understand the evolution of metabolism.

Maintenance metabolic rate
The minimum level of metabolism (per unit time) necessary to sustain life. This level of metabolism can be operationally defined as the metabolic rate of a non-growing, non-reproducing, inactive, unstressed, postprandial animal measured under normothermic conditions during the inactive phase of its circadian cycle. For ectothermic animals, normothermic is defined as a temperature well within the tolerance limits of the species (see also basal metabolic rate). For endothermic animals, normothermic is defined as thermoregulating in a thermoneutral environment. See text for further discussion; see also basal metabolic rate and standard metabolic rate.

Metabolic theory
A broad term for a swathe of theories focused on how energy use at one scale might affect the properties of dynamics of another scale. Examples include the metabolic theory of ecology, Damuth's law and dynamic energy budget theory.

Ontogenetic metabolic scaling
The relationship describing how metabolic rate changes with mass specifically across the ontogenetic trajectory of individuals of the same species. Importantly, comparisons of individuals that vary in mass but not ontogeny should not be used to generate estimates of ontogenetic scaling (see also evolutionary metabolic scaling, static metabolic scaling).

Resting metabolic rate
The metabolic rate of an inactive animal (see also routine metabolic rate).

Routine metabolic rate
Metabolic rate, averaged over a specified time interval, of an animal exhibiting spontaneous 'routine' behaviours, or a specified behaviour.

Standard metabolic rate
The metabolic rate of an adult, inactive, unstressed, postprandial ectotherm measured under normothermic conditions during the inactive phase of its circadian cycle. For ectothermic animals, normothermic is defined as a temperature well within the tolerance limits of the species (see also basal metabolic rate).

Static metabolic scaling
The relationship describing how metabolic rate changes with mass assessed among different individuals at the same developmental stage within a population or species.

Total metabolic rate
The metabolic rate of an animal inclusive of the costs of maintenance, production (somatic growth and reproduction), digestion and activity (for free-living animals, this level of metabolism is referred to as daily energy expenditure or field metabolic rate). Note that, in our model, animals were assumed to be inactive and postprandial, such that empirical data for resting metabolic rate best approximate the definition of total metabolic rate applied in the model.

We assumed that the ontogenetic scaling of metabolic rate with body size is well described by a power function, and sought to identify the value of the metabolic scaling exponent (b, where metabolic rate is proportional to mass^b) that maximised fitness (approximated by lifetime reproductive output). To estimate lifetime reproduction, we assumed that maximum longevity is inversely proportional to metabolic rate and that the probability of surviving to maximum longevity is equal to 0.001. We then simulated the growth of an individual animal over 1,000,000 time steps from birth to death; we calculated reproduction for each time step as the product of the rate of energy allocation to reproduction and the probability of surviving to that time step, and summed reproduction over the life of the animal to calculate lifetime reproduction. Our estimate of lifetime reproduction incorporates the effects of both size and mortality. By making some reasonable assumptions about model parameters (see Box 1) and optimising the value of the metabolic scaling exponent to maximise lifetime reproduction, we were able to show that fitness is often maximised when metabolic rate scales allometrically with size, with an exponent consistent with the mean value of around 0.75 observed in empirical data (White et al., 2022).

Although the parameters we use and assume can and should be debated, to us at least, there is a more critical issue with our optimality model (and indeed optimality models more generally):

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Box 1. The life-history optimisation model for the origin of metabolic scaling

Our model is built upon an energy-expenditure budget for an animal in which the total rate of energy expenditure (E_T, J h^{-1}) is equal to the sum of the rates of allocation of energy expenditure to self-maintenance (E_M, J h^{-1}), production (E_P, J h^{-1}), digestion (E_D, J h^{-1}) and activity (E_A, J h^{-1}). E_P is equal to the sum of the rates of energy allocation to growth (E_G, J h^{-1}) and reproduction (E_R, J h^{-1}). The overhead costs of production (tissue synthesis costs) continue when an animal is postprandial (i.e. no longer spending energy on digestion) (Rosenfeld et al., 2015), and so for an inactive postprandial animal when both E_A and E_D are zero:

$$E_T = E_M + E_P = E_M + E_G + E_R. \quad (1)$$

We assume that production is a fixed fraction f of total metabolism; that animals allocate production only to growth prior to reaching maturity at size M_{Mat} (g); that they grow until maximum mass M (g), which they reach when allocation to growth is zero (and so all of production is allocated to reproduction); and that both metabolism and reproduction are well described by power functions ($E_T = a_{E_T} m^{b_{E_T}}$, $E_R = a_{E_R} m^{b_{E_R}}$), where a and b are scaling coefficients and exponents, respectively, and m is body mass in g. By assuming that $b_{E_M} = b_{E_T}$, calculating the scaling coefficient of reproduction as $a_{E_R} = f a_{E_T} M^{b_{E_T}} / M^{b_{E_R}}$, and introducing a term that represents the overhead cost of tissue biosynthesis and serves to convert from units of energy to units of mass (C_m, J g^{-1}), post-maturation growth rate is estimated as:

$$\frac{dm}{dt} = \frac{f}{C_m} a_{E_T} \left[m^{b_{E_T}} - M^{b_{E_T}} \left(\frac{m}{M} \right)^{b_{E_R}} \right]. \quad (2)$$

The integral of Eqn 2 yields a growth trajectory, and b_{E_T} can be optimised to maximise lifetime reproduction by (1) assuming that lifespan (maximum age; h) is proportional to a constant (C_l) divided by a_{E_T}, (2) setting mortality such that the probability of surviving until maximum age equals 0.001 (White et al., 2022), and (3) estimating reproduction at any point in time from the time dependence of m and the scaling of E_R with m for values of m greater than M_{Mat}. We optimised the parameter b_{E_T} to maximise lifetime reproduction using a numerical model to estimate growth and reproduction through 1,000,000 time steps from t=0 to t=C_l/a_{E_T} (i.e. from birth to maximum longevity). Because mortality was set such that the probability of surviving until maximum age equals 0.001, the probability of surviving each of the 1,000,000 time steps equals $0.001^{1/1,000,000}$. The reproduction predicted to occur in a given time step was calculated as the product of the reproductive allocation occurring during that time step and the probability of surviving to that time step, and lifetime reproduction was calculated by summing reproduction over the life of the animal. This optimisation process, which requires the assumption that lifespan is inversely proportional to a_{E_T}, yields a local optimum for b_{E_T}. The inverse relationship between lifespan and a_{E_T} results in the prediction that fitness is independent of a_{E_T} because increases in E_P are offset by reductions in lifespan (White et al., 2022). At any given a_{ET}, however, the value of b_{E_T} that maximises fitness is often allometric, as dictated by the trade-off between growth and reproduction that accompanies changes in b_{E_T} (White et al., 2022).

the fitness metric that is assumed to be maximised. In our case, we chose to maximise lifetime reproductive output, and although we believe it to be the most pragmatic, we also think it is the most important limitation of our model. Below, we consider the issue of maximisation in more detail.

What does evolution maximise?

Reproductive output versus population growth rate

Describing a combination of traits as 'optimal' is shorthand for saying that those are the trait combinations that yield the highest fitness. Although this approach might seem straightforward, estimating fitness is challenging, and most common measures are imperfect. Worse still, the quantity that evolution maximises in the long term is not always connected to the fitness of individuals and instead depends on a range of factors. Here, we will critique the metric that we used and compare it with alternative metrics of fitness before exploring other issues associated with evolutionary maximisation more generally.

As discussed above, we used lifetime reproductive output as our fitness metric. Specifically, because our model used energy as the currency and converted this to mass using the constant C_m (J g^{-1}), lifetime reproductive output is defined as the total energy expended on reproduction over the lifetime, which is assumed to be proportional to the total number of offspring multiplied by the mass of individual offspring. This seems like a reasonable measure – total fecundity will determine how many offspring are produced and individual offspring mass is a decent predictor of offspring survival (Marshall et al., 2018). Accordingly, in both theory and empiricism, the product of offspring quantity and quality is the currency of fitness (e.g. Stearns, 1992). Lifetime reproductive output is a reasonable measure of fitness for a population in equilibrium in which density dependence acts on the juvenile phase (Kozłowski, 1993; Dańko et al., 2018), but otherwise it is an imperfect proxy for the quantity that evolution maximises. Since Fisher (1958), abundant theory has demonstrated that evolution does not maximise reproductive output per se: instead (and assuming no density dependence), evolution maximises population growth rates. Although lifetime reproductive output certainly contributes to the rate of population growth, it does not define it.

To illustrate why reproductive output is an imperfect proxy for population growth rate, consider the following extreme and simplified example with two hypothetical species. Imagine that after maturity, Species A produces 5 offspring every year and lives for 4 years. Meanwhile, Species B, which matures at the same age as Species A, produces 10 offspring every day and lives for only 2 days. The two species have the same lifetime reproductive output (20 offspring), but obviously Species B will have a much higher rate of population growth than Species A, because shorter generation times mean higher population growth rates per unit time. Hence, although our model treats these two hypothetical species as equal in terms of fitness, they are clearly different in terms of population growth rate. Put another way, a limitation of our model is that a species that reproduces sooner than another species has a fitness advantage in terms of evolutionary maximisation in nature, but our model is ignorant of this. It is easy to imagine that metabolic rate would affect generation times (indeed metabolic theory more generally makes this precise assumption: Savage et al., 2004; see Glossary for a description of metabolic theory), and so the timing of reproduction is likely to covary with the key parameters of our model in ways that the model does not capture.

Why then did we not use population growth rate instead of lifetime reproductive output in our model? Mainly pragmatic reasons. Ideally, we would have used population growth rate as our fitness metric but the problem with this approach is that unless the model is artificially and arbitrarily constrained, it will 'select' for instantaneous reproduction – in essence all organisms should be unicells, which divide as soon as possible. But introducing such a constraint seemed undesirable to us because there are simply too few data on the relationship between our parameters of interest and the timing of reproduction. We need a much better understanding of how the relative timing of maturation covaries with our parameters of interest, and we need to incorporate the demographic consequences of such a coupling into our modelling approach. If we were to simply

maximise population growth rate, all reproduction would occur in a single suicidal bout where all available production was allocated to reproduction as early in life as possible. Again, we could have constrained the model to prevent this outcome because we know that reproduction is spread in time for many species, but we do not know how this spread in time covaries with metabolic rate. Lifetime reproductive output was therefore the only currency we could use without introducing arbitrary constraints into our model and, as we will discuss in the next section, the inclusion of additional parameters is rarely a good thing. Although lifetime reproduction is an imperfect currency for evolutionary maximisation, it requires fewer assumptions and probably captures a non-trivial fraction of the variation in population growth rate, particularly when comparing closely related species with similar generation times. But we view this as the most important weakness of our model, and strongly encourage empirical research exploring the covariance between maturation and reproductive timing and metabolic rate, so that future iterations of the model maximise population growth rate rather than lifetime reproduction.

Even without a model that explicitly deals with population growth rate, we believe that empirical studies exploring the relationships between metabolic rate, individual growth rate and population growth rate would be extremely valuable. Our model implies that these factors should be related because reproduction covaries with both metabolism and growth, but formal tests are exceedingly rare. There are existing approaches for exploring the covariance between population growth rate, life-history traits such as growth and reproduction, and metabolism (Schuster et al., 2021) – they have just been deployed rarely. We strongly encourage others to make use of integral projection models (see Glossary) or other formal demographic tools for exploring these issues further. Such approaches make use of the sorts of data (survival, growth, reproduction) that empiricists routinely collect, and there are pre-existing packages in R for integrating these data into a demographic model (e.g. Metcalf et al., 2013; Levin et al., 2021).

The impact of density dependence

The discussion above makes an implicit, simplifying (and unrealistic) assumption that there is no density dependence (see Glossary) operating in our species. Under such circumstances, population growth rate should be maximised and so lifetime reproduction is a reasonable, if imperfect, proxy. But MacArthur (1962) and many others since have demonstrated that in constant environments and under density dependence, evolution maximises the carrying capacity (see Glossary) of the population (reviewed by Roughgarden, 1979; Boyce, 1984). So, given the reasonable assumption that density dependence is ubiquitous, we perhaps should have used carrying capacity as the focus of our optimisation procedure. Given that metabolic theory has long argued that carrying capacity should strongly and predictably covary with metabolic rate (e.g. Damuth, 1981, 1987; Brown et al., 2004; Bernhardt et al., 2018; Hatton et al., 2019), this seems like a particularly appropriate focus. But once again, we were hampered by a lack of empirical data on the topic in order to constrain the model appropriately. We know that, all else being equal, individuals with higher metabolic rates should have lower carrying capacities, so had we used carrying capacity as the optimisation parameter of our model, it would have predicted that all organisms should have an infinitely low metabolic rate. But theory has long suggested (Houston et al., 1993), and recent work demonstrates (Schuster et al., 2021), that metabolic rate affects not only the rate at which organisms use resources but also, potentially, the rate at which they access resources – in essence, individuals with higher metabolic rates can more effectively forage or capture food than individuals with lower metabolic rates. In such instances, populations with higher metabolic rates may not necessarily have lower carrying capacities and could even have higher carrying capacities. Unfortunately, there are too few studies that have formally explored the relationship between metabolic rate and carrying capacity; usually, body size is used as an imperfect proxy for metabolic rate in such studies but, of course, body size covaries with many other factors. So, until more studies examine the relationship between metabolic rate, life history and carrying capacity in a formal framework, we cannot yet use carrying capacity as our maximisation parameter (for a demonstration of how to implement the framework for metabolic studies, see Schuster et al., 2021).

All is not lost however; more recent work (Lande et al., 2009) suggests that when density dependence occurs, and environments are inconstant (the most realistic scenario in nature), both population growth rate and carrying capacity are predicted to be maximised by evolution. Hence, with the relevant caveats about the potential disconnect between lifetime reproduction and population growth rate, our fitness metric of choice is perhaps not worthless as it captures at least some of the components that evolution is likely to maximise. Our view is that our model explores life-history optimisation using an imperfect metric but it is the only one available for now. To us, this issue makes clear that we need far more estimates of the relationships between metabolic rate, life history and demographic parameters such as population growth rate and carrying capacity. Such studies exist for organisms in which measuring demographic parameters is reasonably straightforward (e.g. unicellular organisms: Malerba et al., 2018; Malerba and Marshall, 2019; Marshall et al., 2022b), but good tools also exist for multicellular organisms and we encourage their use more generally (Schuster et al., 2021). Specifically, we recommend that future studies couple manipulations of the density and metabolic rates of individuals (ideally in a response surface or even ‘cube’ design, see Cameron et al., 2019) in order to formally estimate how carrying capacity and population growth rates covary with metabolic rate.

On the dangers of overparameterisation

> *With four parameters I can fit an elephant, and with five I can make him wiggle his trunk.*
>
> Johnny von Neumann (Dyson, 2004)

Our life-history model for metabolic scaling is, of course, a gross simplification of reality. In addition to using an imperfect fitness metric, it ignores, for example, the possibility that the scaling of metabolic rate may not be a strict power function (e.g. Moran and Wells, 2007; Seymour et al., 2013) and that it may change through life stages (e.g. Glazier, 2005; Killen et al., 2007). If the cost of reproduction is assumed to include only the cost of synthesising gametes, then our model almost certainly overestimates reproductive output (Kearney and Jusup, 2023). Although our model is explicit in terms of mass and energy, it fails to capture the vast complexity of metabolism. It provides an adequate fit to growth data (White et al., 2022), but other growth models provide similarly adequate fits to data, which makes congruence with observations alone a poor criterion for evaluation (Marshall and White, 2019b). Our model also assumes that the amount of energy available for allocation is unconstrained, and does not respond to changes in, for example, food availability – although this is unlikely to affect the optimisation outcome, it still represents a gross oversimplification.

It is tempting to add new parameters to address the limitations of a model. For example, one could address some of the above shortcomings by introducing a term that alters allocation to

production when food is restricted, and a term that introduces phase shifts or curvature in scaling patterns, and so on. Ultimately, we are reluctant to do so, for fear of overparameterisation: just as it is possible to fit an elephant with only four (complex) parameters (Fig. 1A), our model already has multiple free parameters that imbue it with considerable flexibility (Fig. 1B). Rather than continually elaborate the model to accommodate exceptions, we instead take the view that deviations from model predictions can guide empirical research programmes (e.g. Kearney and Enriquez-Urzelai, 2022). Specifically, we propose that a more fruitful way to explore the utility of our approach is to test the key assumptions of the model as it stands, as well as the novel predictions that emerge from it. Such an approach has the benefit that, if the empirical programmes ultimately demonstrate that our model is fatally flawed, the empirical work will provide new data that will help the field move forward.

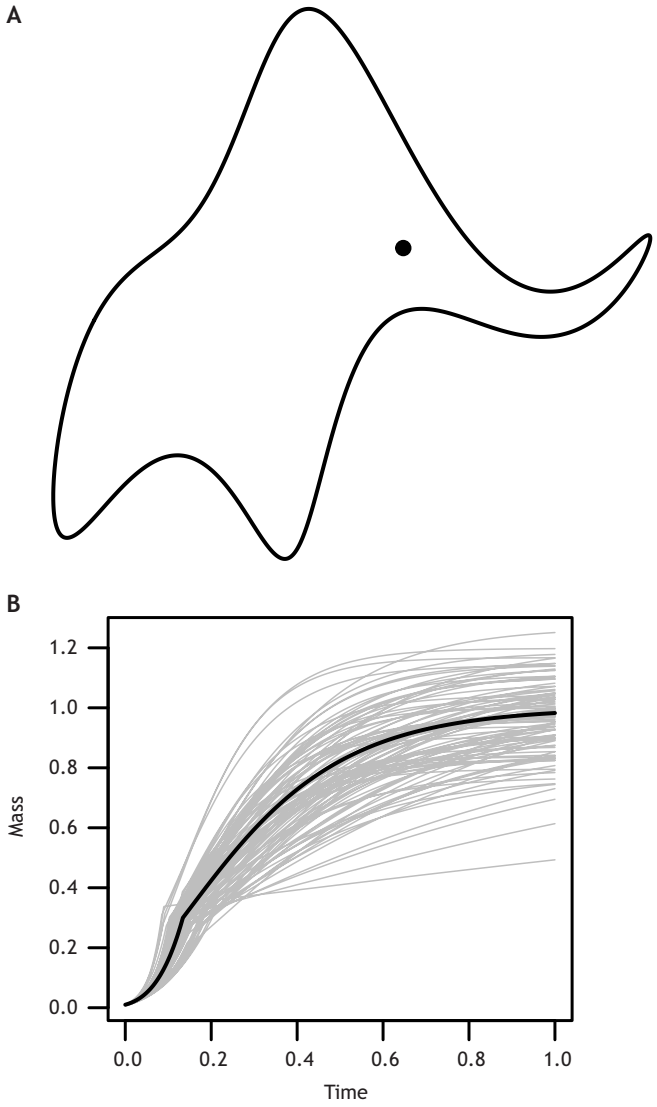

Fig. 1. Free parameters allow for very flexible fitting. (A) A drawing of an elephant made by fitting four (complex) parameters, as demonstrated by Mayer et al. (2010) and redrawn here using a modification of code provided by Neil Gunther (https://perfdynamics.blogspot.com/2011/06/winking-pink-elephant.html). (B) The black line shows the shape of a growth curve derived from Eqn 1 (see Box 1) for an animal growing from an initial mass of M_0=1, using arbitrary parameters (f=0.4, C_m=1, a_{E_T}=100, b_{E_T}=0.75, M_{Mat}=30, M=100, b_{E_R}=1.13). The grey lines show the range of growth curves generated when six of these parameters (M_0, f, a_{E_T}, b_{E_T}, M_{Mat}, M, b_{E_R}) are varied randomly around these values using normal deviates with a coefficient of variation of 10%.

Moving forward: untested assumptions and testable predictions

Our model, like all models, makes a number of simplifying assumptions. In the following paragraphs, we identify the tests of the model that we view as the most valuable, including tests of model assumptions and tests of model predictions.

The scaling of production

A key parameter in the model is f, the fraction of energy expenditure that is allocated to the metabolic work of production (growth+reproduction). Our model assumes that the scaling exponents of total metabolic rate and maintenance metabolic rate (see Glossary) are identical, such that f is independent of size. This is in stark contrast to other models that assume that the maintenance metabolism scales isometrically (e.g. von Bertalanffy, 1957; West et al., 2001; Hou et al., 2008; Kooijman, 2010; Pauly, 2010; Pauly and Cheung, 2017; Kearney, 2021) – which we have previously argued is inappropriate (Marshall and White, 2019a,b). There is some evidence that metabolic rate scales allometrically with mass for non-growing juvenile animals maintained on a maintenance ration (e.g. Elliott, 1976), which supports our assumption that the scaling exponents of total and maintenance metabolic rate are the same, but estimating maintenance metabolism directly is problematic. It seems very unlikely to us that animals on a maintenance ration will differ from growing animals only in their energy allocation to growth, and so measurements of animals under these conditions may not be relevant to our model (see Metcalfe et al., 2023, for a list of the types of metabolic rates most frequently measured by scientists; and see the Glossary for definitions of those types most relevant to the present model: basal metabolic rate, resting metabolic rate, routine metabolic rate, and standard metabolic rate). It is possible, however, to estimate the parameter f indirectly by comparing the scaling of pre-maturation growth rate with body size with the scaling exponent of metabolic rate. Assuming that C_m is independent of size, then the assumption of equal scaling exponents for total and maintenance metabolic rates is supported if the scaling exponents of pre-maturation growth rate and metabolic rate are the same, and if pre-maturation growth rate scales isometrically with metabolic rate. Testing this assumption in reproductively mature animals is more complex, because it requires estimation of all costs associated with reproduction, and gamete production alone represents only the lower bound of total reproduction costs (e.g. White et al., 2023).

The relationship between longevity and metabolism

The second assumption of our model that should be explored is the relationship between metabolic rate and longevity. Metabolic theory (Brown et al., 2004; Kooijman, 2010; see Glossary) and life-history theory (Ricklefs and Wikelski, 2002; Speakman, 2005; see Glossary) both predict that lifespan should be inversely related to metabolic rate, and we verified this negative relationship in our recent study (White et al., 2022). Invocation of this inverse relationship is central to our optimisation process, but the precise form of the relationship between metabolic rate and lifespan, and the influence of other life-history traits – particularly growth and reproduction, independent of size and metabolism – on this relationship requires further empirical exploration. Although among-species comparative studies have been

useful, within-species relationships may yield different conclusions (Speakman, 2005) that are also more relevant, and we particularly encourage examination of the within-species relationship between metabolism and lifespan.

Emergent predictors of metabolic scaling: reproduction scaling and size at maturity

One of the most powerful tests of a model is the examination of novel predictions that emerge from it, as opposed to testing the model against the observations it was conceived to explain. Optimal values of the metabolic scaling exponent are predicted to increase as the scaling exponent of reproduction increases, but the relationship between these is predicted to be less than 1:1 (White et al., 2022). Although large databases of metabolic rate and reproduction data are available (e.g. Marshall and White, 2019b; Marshall et al., 2022a; White et al., 2022), the overlap between these is surprisingly poor, and empirical work is likely to be necessary to generate data to test this prediction. Our model also predicts that animals that mature at a small size relative to their maximum size will have lower metabolic scaling exponents than animals that mature at a relatively large size (White et al., 2022), and this prediction should also be amenable to testing.

The effect of extrinsic mortality on metabolic scaling

Perhaps the most unexpected outcome of our modelling approach is the predicted negative relationship between extrinsic mortality (in essence, the sum of mortality sources that are not phenotype specific) and the optimal scaling exponent of metabolic rate (White et al., 2022). This relationship has the potential to explain ecological effects on metabolic scaling (e.g. Glazier, 2005, 2006; Killen et al., 2010; White and Kearney, 2013, 2014). For example, our prediction is supported by the observation that amphipods living with fish predators have lower scaling exponents than those in predator-free environments (Glazier et al., 2011). Further empirical validation of the link between extrinsic mortality and metabolic scaling is required, and could be obtained by measuring metabolic scaling after evolution following multiple generations of elevated extrinsic mortality (e.g. Stearns et al., 2000; Chen and Maklakov, 2012; Wootton et al., 2021; Roy and Arlinghaus, 2022).

Thoughts on optimality and the adaptationist programme

Our pursuit of an optimality approach to explaining metabolic scaling is built upon two findings. First, the observation that the scaling of fecundity is often hyperallometric (i.e. has a scaling exponent greater than 1) (Barneche et al., 2018; Marshall et al., 2022a). And second, the demonstration that allometric scaling of metabolic rate is unlikely to arise as a consequence of drift under a genetic constraint (White et al., 2019). The first finding raised fundamental questions about how to model growth (Marshall and White, 2019b), and the second finding implicated selection as being important for the scaling of metabolic rate. Although the importance of the first finding is now clear, in hindsight we underestimated the importance of the second finding, which eliminated one possible non-adaptive explanation for metabolic scaling, but other non-adaptive explanations, such as the drift barrier hypothesis (Lynch, 2022), require further consideration.

The drift barrier hypothesis proposes that larger animals experience reduced selection on deleterious mutations because they have longer generation times, leading to reduced mass-specific growth rates in larger organisms. Our comparative analysis shows that slower-growing species have lower metabolic rates (White et al., 2022), providing support for one of the putative links between elevated deleterious mutation rates, slow growth and low metabolic rates in larger species. The drift barrier hypothesis represents an alternative, non-adaptive explanation for metabolic scaling, which would be supported if larger animals exhibit more deleterious mutations in protein-coding genes, and enzymes with lower mass-specific catalytic capacity (Harrison et al., 2022). It could be tested by manipulating effective population size over generations in the lab, which is predicted to result in changes in the load of deleterious mutations, enzyme catalytic capacity and organismal growth and metabolic rates (Harrison et al., 2022). We raise the drift barrier hypothesis here not only because it is worthy of future investigation but also because it serves as a reminder of the importance of considering non-adaptive hypotheses for the origin of biological patterns (Gould et al., 1979; Lynch, 2007, 2022).

Conclusion

> *The first principle is that you must not fool yourself – and you are the easiest person to fool.*
>
> Richard P. Feynman (1998)

We have argued that our model provides an imperfect but useful explanation for the evolution of metabolic scaling. Most importantly, we hope that the model does what good models should – identify priorities for empirical research. We think that producing estimates of within-species relationships between metabolic rate, generation time, population growth rate and carrying capacity represents the next important step for the field.

We do not regard the modelling approach we have taken as the only viable approach to studying the co-evolution of metabolism and life history. However, we do regard it as a useful starting point for exploration of this issue, and we suggest that the work we have proposed in the preceding sections will be helpful in moving the field forward. We conclude our Commentary by cautioning ourselves, and the proponents of existing and future models, against being beguiled by the congruence between data and a model designed to fit those data. Rather than risk fooling oneself, we advocate for rigorous empirical testing of model assumptions, explicit statements of testable predictions and openness about the limitations of any given approach.

Although the task is daunting, providing empirical estimates of our model assumptions has never been more possible – the field is vibrant and growing, and technological innovations continue to provide more powerful techniques. So, on this centenary of JEB, we are optimistic about the next 100 years of research in this area.

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Competing interests

The authors declare no competing or financial interests.

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