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REVIEW

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The contribution of metabolic theory to ecology

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Abstract

The metabolic theory of ecology (MTE) has been an important strand in ecology for almost a quarter of a century, renewing interest in the importance of body size and the role of energy. The core of the MTE is a hydrodynamic model of the vertebrate cardiovascular system that predicts allometric scaling of metabolic rate with exponents in the range 0.75 at infinite size to ~0.80 at more realistic sizes, though most studies using the model have assumed an exponent of 0.75. The model is broadly supported by data for resting and routine metabolic rate in ectothermic vertebrates and also a wide range of invertebrates with a circulatory system. Scaling in endotherms is influenced by additional factors, possibly associated with heat flow, and is essentially isometric in prokaryotes, unicellular eukaryotes, and diploblastic invertebrates. This suggests that the presence of any form of circulatory system, even one much simpler than the closed high-pressure system that is the basis of the model, results in allometric scaling of metabolic rate, though the value of the scaling exponent varies across taxa. The temperature sensitivity of metabolism is captured by a simple Boltzmann factor, with an assumed apparent activation energy of 0.65 eV (Q_{10} ~ 2.4). Empirical data are frequently lower than this, typically in the range 0.52–0.57 eV (Q_{10} ~ 2.0–2.2). Attempts to broaden the scope of the MTE into areas such as growth, speciation, and life-history have met with mixed success. The major use of the MTE has been to explore the consequences of the central scaling tendency for topics as diverse as migration, acoustic communication, trophic interactions, ecosystem structure, and the energetics of deep-sea or extinct taxa. Although it cannot predict absolute metabolic rates, the MTE has been an important tool for exploring how energy flow influences ecology. Its greatest potential for future use is likely to come from building energetics into ecosystem models and in exploring potential consequences of climate change. In both cases, however, it will be important to encompass the range of empirical data for both scaling and temperature sensitivity rather than the widely assumed canonical values.

KEYWORDS
allometry, climate change, energetics, growth, metabolism, model, scaling, size, temperature

INTRODUCTION

Life requires the movement of energy to power physiology and maintain structure. As ecologists, we therefore need to know how and why energy moves between organisms and their environment. The flow of energy and materials through ecosystems was a central theme for the founding fathers of ecology (Elton, 1927; Lindeman, 1942; Odum, 1968), but attention later became directed increasingly at population dynamics, ecosystem function, and most recently biological diversity (Golley, 1993; Maurer, 1999; McIntosh, 1985). That attention being once again given to energy can be attributed directly to the influence of the Metabolic Theory of Ecology (MTE) which for almost a quarter of a century has been an important strand in ecology, renewing interest in the importance of body size and the role of energy.

Here I review the strengths and limitations of the MTE as it applies to animals, summarize its wider influence in ecology to date, and discuss its potential to advance ecology in the future.

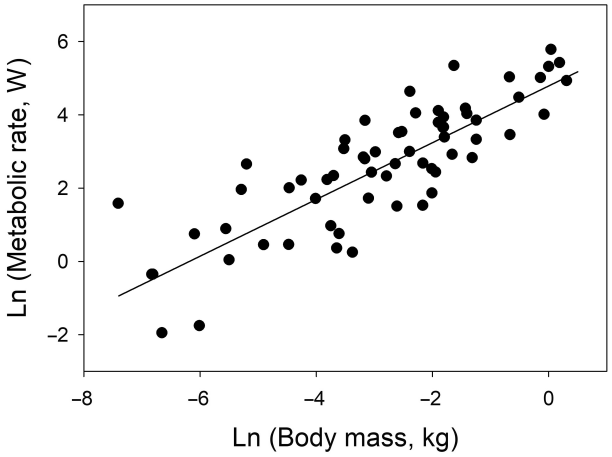

FIGURE 1 Standard metabolic rate as a function of body mass for 69 species of teleost fish (one data point per species). The line was fitted by ordinary least squares after transformation of both variables to natural logarithms and has a slope of 0.78 (see Appendix S1: Table S1 for details of the statistical analyses). Data from Clarke and Johnston (1999).

Why did we need a theory?

The importance of body size has intrigued mankind since at least as far back as Galileo (Haldane, 1926). Why is it that small animals tend to live fast and die young, while larger ones trundle on into old age? This is actually two questions, one physiological (what is the mechanism?), the other evolutionary (how did this pattern arise?). At the heart of these questions lies the relationship between energy flow and body size; Figure 1 shows an example for fish.

The key features of this relationship are that it is linear in logarithmic space, implying an underlying power law, the slope is less than unity, and there is considerable variance about the line. We might have expected that a doubling of size would lead to a doubling of oxygen demand; a slope of <1 thus suggests a constraint of some type. The variance about the line reflects ecological variety and is a measure of the extent to which evolution has been able to work around this constraint. Any complete theory of metabolic scaling must therefore explain the nature of the constraint, predict the slope of the relationship, and provide a mechanism for evolutionary variation about the central tendency.

Early attempts at a physical explanation for the scaling of metabolism were focused largely on the need to understand the food requirements of livestock. For almost a century, the guiding principle was the "surface law" (Rameaux, 1857; Sarrus & Rameaux, 1839), the basic concept of which was that the dominant function of metabolism is to maintain a relatively high internal body temperature. Since heat is lost predominantly from the body surface, resting metabolic rate should scale to body mass with an exponent of ~0.67 (the ratio of surface area to body mass) (see Appendix S1: Section S2). This was given empirical support by the classic study of domesticated dogs by Rubner (1883).

As further data accumulated, however, it became clear that scaling was typically steeper than predicted from the surface law and was also observed in ectotherms where considerations of heat flow were not relevant (Hemmingsen, 1950, 1960; Krogh, 1916; Zeuthen, 1947, 1953). Many early compilations suggested a scaling exponent for resting metabolism of ~0.73, and Kleiber (1961) suggested that this be rounded to 0.75, simply to ease computation with a slide rule. Despite the lack of any theoretical underpinning, a value of 0.75 for the scaling exponent of metabolism assumed the status of a canonical value and was often termed "Kleiber's law" (see Appendix S1: Section S4). A fascinating history of this topic in mammals is provided by Mortola (2023).

At this point, Geoffrey West, James Brown, and Brian Enquist (hereafter WBE for convenience) took up the challenge of providing a firm theoretical foundation for the scaling of metabolic rate. They developed a general hierarchical model for organism distribution systems (West et al., 1997, 2000), which yielded two closely related models, one for animals (West et al., 1997) and one for plants (Enquist et al., 2000; West et al., 1999, 2000). In this review, I will be concerned solely with the animal model, which was based on the assumption that

the key process was not heat loss through the body surface but the delivery of oxygen and nutrients to the tissues.

THE WBE MODEL

The WBE model is based on three principles. The first two are structural: the distribution system has to be space-filling with a hierarchical branching pattern (so that every cell in the body is supplied with oxygen and nutrients), and the dimensions of the final branch in the network are invariant (so capillaries are the same size in a shrew and an elephant). The third principle is an evolutionary optimization: the energy required to circulate fluid is minimized (West et al., 1997, 1999).

From these three simple assumptions, WBE built a hydrodynamic model with a fractal-like structure that predicted the observed scaling parameters for a range of structural and dynamical variables of the mammalian cardiovascular system (West et al., 1997). On the basis of this model, WBE posited a conventional power law relationship between metabolic rate and body mass:

$$B = B_0 M^b \quad (1)$$

where B is metabolic rate, M body mass and b the scaling exponent, the value of which was predicted to be ~0.75. The pre-exponential factor B_0 sets the level of metabolism; it varies, for example, between endotherms and ectotherms, between different animal groups, and with ecology. Unlike the scaling exponent, the value of B_0 cannot be predicted from the WBE model; it is a free parameter that has to be determined empirically by fitting the model to data. In other words, we cannot predict the metabolic rate of any organism from first principles; biological systems are just too complex.

West et al. (1997) noted explicitly that the predicted scaling exponent of 0.75 held only for large animals, and that the scaling would be steeper (that is, the scaling exponent would be greater) in smaller animals. A later detailed examination of the model confirmed that a scaling exponent of 0.75 was an asymptotic result that was predicted only at infinite size (Savage et al., 2008). This later study produced two important conclusions. The first was that the WBE model does not predict a pure power law, and hence the relationship between body mass and metabolic rate is curvilinear in logarithmic space (although this effect is small in relation to the variability of empirical data). The second is that subtle variations in the structure of the circulatory system affect the predicted scaling exponent. The scaling exponent would always be >0.75 but could exceed 0.80; for the typical range of body size in mammals, the exponent would be ~0.81 (Savage et al., 2008). The WBE model thus predicts a range of scaling exponents, but despite this nuanced influence of size, the WBE model is almost universally assumed to predict a scaling exponent for metabolic rate of 0.75.

Linking blood flow to metabolic rate

While the WBE model tackles the structure and dynamics of the circulatory system, there is no explicit mechanistic link to metabolic rate. It is simply assumed that one drives the other (West et al., 1997, 2000).

Consideration of the role of blood flow in determining metabolic rate goes back to the late 19th century (see Kleiber, 1961 for the early history of this idea). More recently, the question was explored by Coulson and colleagues (Coulson, 1983, 1986; Coulson et al., 1977). They contrasted the metabolic rate of a comparable cell in a small reptile and a very large mammal, where metabolic rate per unit mass differs by several orders of magnitude. A difference this great cannot be explained by changes in the concentration of nutrients or enzymes within the cell (there simply is not enough room), and cells from comparable tissues do not differ in size or structural appearance. There is little difference in the concentration of nutrients in the blood, or the fraction of nutrients and oxygen removed by the cells for each circulation of the blood. The only viable explanation for the difference in metabolic rate is a difference in the rate at which blood supplies nutrients. Critical to this explanation is that the rate of equilibration of oxygen and nutrients between cells and blood is very rapid compared with blood flow rate (Coulson, 1986).

This flow theory of metabolic rate (Coulson, 1986) thus allows us to characterize a direct mechanistic link between the dynamics of the circulatory system and cellular metabolic rate, mediated through blood circulation time. Circulation time is predicted by the WBE model to have a scaling exponent of 0.25 (West et al., 1997, 2000), which matches that suggested by Schmidt-Nielsen (1984). This in turn would lead to a scaling exponent of 0.75 for metabolic rate.

HOW GENERAL IS THE WBE MODEL?

The WBE model was developed explicitly for the vertebrate cardiovascular system, that is, a closed circulation where blood is moved by an active pump. Its predictions have been tested predominantly with empirical data for resting metabolic rate in vertebrates (usually termed

basal metabolic rate in endotherms, and standard metabolic rate in fish: Clarke, 2017).

Metabolic rate in vertebrates

The literature contains many estimates of the scaling exponent for basal or resting metabolic rate in vertebrates. These differ slightly from study to study, depending on the dataset used, the statistical model employed, and the rigor with which lower quality data are excluded. Typical estimates for the scaling of resting metabolism in different vertebrate classes are given in Table 1. Body temperature was included in the statistical model because this exerts a significant effect on metabolic rate in addition to body mass, and yields an improved fit compared with a model in body mass alone (Clarke et al., 2010; White, 2011). The data for mammals have been limited to species with body mass above 150 g; this is because in smaller species the relationship is significantly curvilinear (discussed below).

When analyzed at the level of taxonomic class, there is thus support for the canonical value of 0.75 from reptiles and larger mammals. Fish have a steeper scaling exponent, 0.83, somewhat above the wider range of values allowed by the nuanced effect of size examined by Savage et al. (2008). The data for birds are significantly shallower than predicted by the WBE model.

A problem here is that vertebrates typically spend only limited time operating at basal or resting level. We should be testing the WBE model by looking at the scaling of routine metabolism (that is field metabolic rate [FMR], or daily energy expenditure [DEE]). West and Brown (2005) recognized this, and Savage et al. (2008) examined FMR in the context of the WBE model, but almost all discussion (and use) of the WBE model has centered on resting or basal metabolic rate.

FMR data are available for mammals, reptiles, and birds (Table 2). Body temperature could not be included in the statistical model because vertebrate body temperature varies throughout the day, and we have daily averaged body temperature data for very few taxa. The scaling of FMR varies across groups, with exponents varying from 0.89 in reptiles to 0.66 in birds (Table 2). Data for birds and reptiles are shown in Figure 2.

Current data thus suggest that there is no single scaling exponent linking daily energy expenditure to body mass in vertebrates. At present, we have no knowledge of the scaling of daily energy expenditure for invertebrates.

A complication: Curvature of scaling

Both Savage et al. (2008) and West et al. (1997) noted that the WBE model actually predicts nonlinear scaling, with the exponent being larger (that is the scaling is steeper) at smaller sizes, though the size where this occurs was not specified.

In mammals there is a clear indication of curvature with smaller species tending to have a higher metabolic

TABLE 1 Analysis of scaling of basal or resting metabolic rate (BMR) with body mass in vertebrates.

Class	Slope	SE	*n*	p ($b = 0.75$)	p ($b = 0.81$)
Mammals (>150 g)	0.75	0.017	279	NS	$0.001 < p < 0.01$
Birds	0.64	0.025	83	<0.001	<0.001
Reptiles	0.76	0.012	155	NS	<0.001
Fish	0.83	0.023	69	<0.001	NS

Note: Data were fitted with a general linear model: Ln(BMR, in watts) as a function of Ln(body mass, in kilograms) and Tb (body temperature, in kelvins) with Class as a fixed factor. Slope is the regression coefficient for mass; SE is the standard error of the estimated slope; *n* is the number of species (one data point per species); p is the two-tailed probability from Student's t test for the difference of the estimated slope from 0.75 or 0.81; NS indicates not significant ($p > 0.05$). Full details of the statistical analysis are given in Appendix S1: Sections S5 and S12.

TABLE 2 Analysis of scaling of field metabolic rate (FMR) with body mass in vertebrates.

Class	Slope	SE	*n*	p ($b = 0.75$)	p ($b = 0.81$)
Mammals (>150 g)	0.82	0.021	76	$0.001 < p < 0.01$	NS
Birds	0.66	0.017	147	<0.001	<0.001
Reptiles	0.89	0.014	55	<0.001	<0.001

Note: Data were fitted with a general linear model: Ln(FMR, in Watts) as a function of Ln(body mass, in kilograms) and Class as a fixed factor. p is the two-tailed probability from Student's t test for the difference of estimated slope from 0.75 or 0.81; NS indicates not significant ($p > 0.05$). Full details of the data sources and statistical analysis are given in Appendix S1: Section S6.

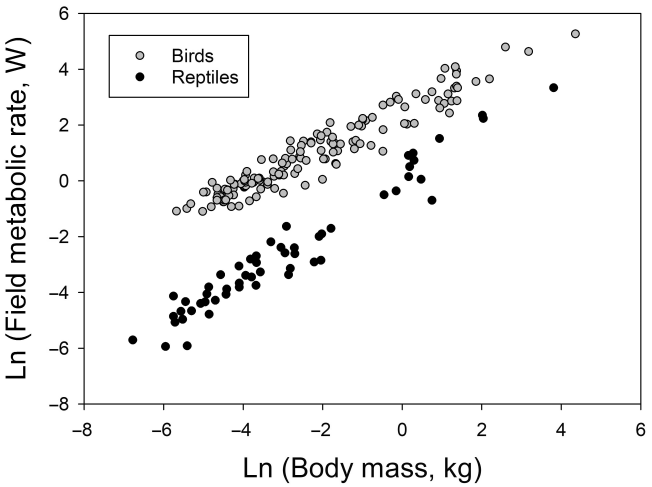

FIGURE 2 Scaling of field metabolic rate with body mass in birds and reptiles.

rate than predicted by the relationship for larger species, a pattern seen in both BMR (Clarke et al., 2010) and FMR (Hudson et al., 2013). This curvature is most evident below a body mass of 150 g, which is close to the median size of a mammal. Above 150 g scaling is effectively linear with exponents of ~0.76 for BMR and ~0.82 for FMR (see Appendix S1: Section S7).

A key aspect of this curvilinear scaling is that it is precisely opposite to that predicted by West et al. (1997), as noted by Savage et al. (2008). In later publications, however, the hydrodynamics are reconsidered and the prediction concerning curvature at smaller sizes changes. Thus West and Brown (2004, 2005) argue that smaller mammals dissipate more energy in their cardiovascular networks, and as a consequence the scaling exponent in smaller mammals "*should decrease below 3/4*" (West & Brown, 2005; italic text is verbatim). This changes the original prediction to its exact opposite, but one that now matches empirical data.

There is currently no evidence for curvature in the scaling of either BMR or FMR in birds, reptiles, or fish. Present data indicate that, among vertebrates, it is solely a feature of mammals. This suggests that explanations based on general features of the WBE model (Kolokotrones et al., 2010) cannot explain this curvature, for they would then also apply to other vertebrates where curvature is not seen.

This curvature does not invalidate the WBE model; it simply indicates that in small mammals, other factors come into play. That metabolic rates are higher in smaller species suggests the possibility that this extra factor may be heat flow, with smaller species having to increase their metabolic rate in order to maintain body temperature (Brown & Lasiewski, 1972; Clarke et al., 2010) or reduce body temperature in cold environments (Wearing & Scott, 2022; White et al., 2007). Ironically, this would bring surface area back into the picture, though it also begs the question of why curvature is not seen in birds, where body temperatures are typically higher than in mammals (Clarke & Rothery, 2008). Birds do, however, have a significantly shallower scaling than mammals, suggesting the possibility that the thermal challenges of being a small endotherm appear gradually in birds rather than below a threshold size, as in mammals. Perhaps the answer here lies in the differing nature of fur and feathers as insulation, but at present, the explanation is obscure.

Does the WBE model apply to invertebrates?

It has long been noted that the metabolic rate of invertebrates typically exhibits a similar scaling to vertebrates, with key studies establishing this being Hemmingsen (1950, 1960), Ikeda (1985), Ivleva (1980), Robinson et al. (1983), and Zeuthen (1947, 1953). Many of these invertebrate groups, however, have a very different circulatory architecture from the closed high-pressure system modeled by West et al. (1997). Invertebrate circulatory systems are highly varied (Crossley et al., 2017; Reiber & McGaw, 2009) and the obvious question is whether the scaling of the metabolic rate in invertebrates is related to differences in circulatory architecture. Data for key groups of invertebrates, classified by circulatory system structure, are summarized in Table 3.

Among metazoans, a critical divide appears to be between diploblastic and triploblastic phyla. Diploblastic phyla (Porifera, Ctenophora, Cnidaria) have just two layers of cells, each of which exchange oxygen, nutrients, and waste directly with the surrounding water. The two cell layers are separated by more or less inert tissue which provides bulk and rigidity. Although data are limited, evidence suggests that scaling in these taxa is close to isometric (that is the scaling exponent, b, is ~1: Table 3).

Triploblastic phyla have an extra mesodermal layer which is metabolically active and hence requires supplying with oxygen and nutrients; these generally have scaling exponents in the range b ~0.6 to ~0.8 (Table 3). They include insects which are unique in having oxygen delivered directly to the tissues through tracheae, each of which branches in a fractal-like pattern, whereas nutrients are delivered through an open circulation system.

Although several individual invertebrate groups have been studied in relation to the predictions of the WBE model, there have been few broad analyses of metabolic

TABLE 3 Scaling exponent for metabolic rate in selected groups of invertebrates.

Circulatory system	Taxon/group	Type	Exponent	No. taxa
None	Prokaryotes	U	1.28	165
None	Protists	U	1.00	103
None	Porifera (sponges)	D	0.92	3
None	Ctenophora	D	0.97	8
None	Cnidaria	D	0.92	13
None	Planaria	T	0.72, 0.75	2*
Clonal	Bryozoans (laminar)	T	0.99–1.19	5*
Clonal	Bryozoans (erect)	T	0.61–0.84	2*
Clonal	Ascidians	T	0.80	1*
Open circulation	Bivalve mollusks	T	0.76	77
Open circulation; oxygen supplied through tracheae	Insects	T	0.82	391
Incompletely closed circulation	Decapod crustaceans	T	0.69	35
Closed circulation (low pressure)	Nemertea	T	0.64	1*
Closed circulation (low pressure)	Polychaetes	T	0.61	17
Closed circulation (low pressure)	Echinoderms	T	0.74	85
Closed circulation (moderate to high pressure)	Cephalopod mollusks	T	0.81	39

Note: These are interspecific relationships unless otherwise indicated (*), where the exponent is a median value from intraspecific studies. Full details of these studies are given in Appendix S1: Table S9.
Abbreviations: D, diploblastic phylum; T, triploblastic phylum; U, unicells.

scaling in invertebrate groups. The most comprehensive is Glazier (2006), who suggested that in aquatic invertebrates scaling differs between planktonic and benthic forms, with plankton exhibiting scaling exponents nearer to b ~1 because of the energetic demands of staying in the water column, but benthos having scaling b ~0.7. The scaling exponents of planktonic crustacea (decapods, euphausiids, and copepods), however, typically fall in the range 0.7–0.75, which argues against this. A planktonic existence does indeed involve greater metabolic costs (Clarke & Peck, 1991) but this does not appear to affect their metabolic scaling.

Scaling in two-dimensional organisms

WBE argued that where organisms are essentially two-dimensional, the scaling exponent would be ~0.67. Two examples they suggested were flatworms and bryozoan colonies growing as a sheet across a surface.

Data for the planarians (flatworms) *Schmidtea mediterranea* and *Bipalium kewense* do not conform to this prediction, having scaling exponents of 0.75 and 0.72, respectively (Table 3). This may be because although planarians appear flat to human eyes, they nevertheless have a distinct three-dimensional structure. In planarians, oxygen is absorbed through the skin, but nutrients are distributed throughout the body via a highly divided gut. These are, however, two intraspecific studies rather than the broad interspecific analysis required to test the WBE critically (see below).

Bryozoans are clonal animals, and in many species, the growth form is laminar (that is, the bryozoan colony grows across a surface as epifauna). Hughes (1989, 2005) suggested that if the individual modules (zooids) in such a laminar colony are independent, then a complete absence of allometry would be predicted (that is an exponent ~1, a very different prediction from West et al., 1997, 2000). If, however, there is a differentiation in function across the zooids (for example for feeding, reproduction or defense), then resources would need to be moved between them, and some allometric constraints might be expected (that is an exponent <1). Most bryozoan colonies show such differentiation, with translocation between modules (Hughes, 2005). The limited data available suggest that laminar colonies have a scaling exponent ~1, whereas arborescent colonies are closer to 0.73 (Table 3).

Scaling in unicells

Eubacteria, Archaea, and unicellular eukaryotes ("protists," fungi) are phylogenetically unrelated and use a vast range of ways of gaining energy, but share a common feature in that they exchange nutrients and waste

products directly with their environment through their external cell membrane. Since metabolic rate and nutrient demand must be broadly in balance (under normal circumstances cells do not swell or shrink), this might suggest that their metabolic rate should scale with an exponent of ~0.67 (the rate of surface area to volume). Empirical data, however, indicate that scaling in these diverse organisms is essentially isometric (b ~1, Table 3). What anatomical or physiological mechanisms underpin this isometric scaling are not currently understood (DeLong et al., 2010).

Conclusion: How general is the WBE model?

Unicellular organisms have no internal circulatory system and thus fall outside the scope of the WBE model. Scaling in these organisms is essentially isometric. Diploblastic invertebrates (sponges, ctenophores and cnidarians) also lack a circulatory system, and scaling in these taxa is also close to isometric ($b = 0.92$–0.97).

For triploblastic invertebrates, current evidence suggests that the presence of any form of circulatory system leads to allometric constraints with a scaling exponent <1 and usually within the range 0.6–0.8. This is broadly comparable with the predictions of the WBE model, though a few taxa (notably decapods, and polychaetes) exhibit an exponent that is below the minimum predicted value of 0.75.

The discussion so far has, however, ignored an issue that is fundamental to the use of the WBE model in ecology: what, precisely, do we mean by "metabolism"?

THE NATURE OF METABOLISM

Metabolism comprises the complex suite of regulated and coordinated reactions that take place within every living cell. WBE typically define metabolism as "*the biological processing of energy and materials*" (Brown et al., 2004). There is nothing incorrect in this and it is framed in terms familiar to ecologists. A more rigorous thermodynamic definition is *the conversion of chemical potential energy to work and heat* (Hill et al., 2008) which also avoids potential ambiguity between "metabolism" and "metabolic rate." As heat is by far the larger product, metabolic rate can be estimated directly from the dissipation of this heat, as was done by Antoine Lavoisier in his pioneering determination of the metabolic rate of a guinea pig (Holmes, 1985; Mendelsohn, 1964). Such direct calorimetry is technically demanding and so physiologists have looked for more tractable indirect measures, and settled on oxygen consumption. The rationale for this is that oxygen acts as the terminal electron acceptor in the regeneration of ATP from ADP (oxidative phosphorylation) and because ATP powers almost everything an organism does, either directly or indirectly, using oxygen consumption as an estimate of the rate of metabolism is reasonable. Indeed metabolism is often regarded as synonymous with oxygen consumption, because this is how it is usually measured.

A complication here is that some ATP is regenerated anaerobically by substrate level phosphorylation in glycolysis and some oxygen is used by the cell for processes other than ATP synthesis (Figure 3). In mammals, it is usually assumed that most oxygen use is associated with ATP regeneration (Wilson, 2013), although the production of heat by nonshivering thermogenesis (which uses oxygen but does not regenerate ATP) is important in some tissues at some times. In many invertebrates, however, a significant fraction of ATP regeneration can be anaerobic, particularly in those living in environments where oxygen availability is low or episodic.

For free-living animals in the wild, we cannot measure either heat dissipation or oxygen consumption directly. Instead, we use doubly labeled water to measure carbon dioxide production or data loggers to monitor heart rate (and increasingly other physiological variables). These techniques differ in the type and resolution

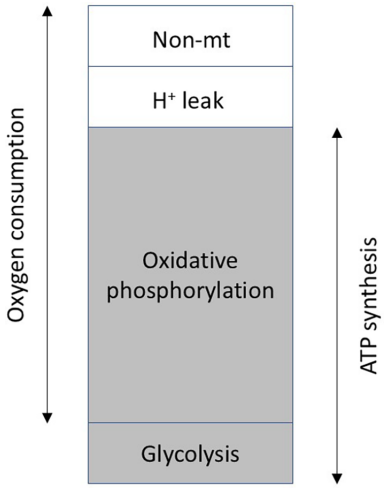

FIGURE 3 The relationship between oxygen consumption and ATP synthesis. ATP synthesis is shown in gray and oxygen consumption not associated with ATP synthesis in white. Non-mt: oxygen use not associated with mitochondrial function; H^+ leak: oxygen consumption driven by proton leak across the inner mitochondrial membrane. The sizes of the boxes are arbitrary as the relative contributions of these four processes are not fully established, and indeed may change with species and circumstances.

of information they provide, but they are both essentially proxies for a proxy, with several steps between the measurement being made and the metabolic rate being estimated. The recent development of data loggers, especially when coupled with GPS or other geolocators, does, however, offer the opportunity of gaining a far more representative picture of how organisms gain and use energy in the wild than we can build in the artificial confines of the physiologist's laboratory.

What controls metabolic rate?

The WBE model was built from the assumption that the metabolic rate of a cell, and thereby of the whole organism, is determined by the rate of delivery of nutrients and oxygen (West et al., 1997) and the flow theory of Coulson (1986) links the rate of this delivery to circulation time.

Cells are, however, not passive entities at the mercy of the supply of nutrients or oxygen, and biochemists working with in vitro systems have elucidated many subtle controls which operate within the cell to regulate its metabolic rate. Central to this is the protonmotive force, Δp, across the inner mitochondrial membrane. Any reduction in Δp resulting from an increase in ATP turnover induces a rapid increase in electron transport and hence oxygen utilization until Δp is restored. There are also feedback controls that regulate the supply of electrons from the TCA (Krebs) cycle and glycolysis, but the key point is that oxygen consumption ("metabolic rate") is a response to the ATP demand of whatever the cell is doing. In addition, cellular metabolism is linked intimately to the cardiovascular system in that use of metabolic substrates stimulates an increase in blood flow to replenish metabolite levels. These mechanisms can increase or decrease blood flow by a factor of about 10. This is ample to cope with the difference between resting (basal) metabolic rate and mean daily energy expenditure, which is typically a factor of 2–4, though it can be as large as 7–8 (Clarke, 2017).

An organism is thus an integrated whole (Suarez, 2012) with blood flow and cellular demand in balance. This integrated regulation means that variation in an individual organism's oxygen demand is dictated by the regeneration of ATP. Metabolic rate does not determine what an organism can do; what an organism is doing dictates its metabolic rate.

How does this control of metabolism at the level of the cell relate to the scaling of whole organism metabolic rate? The key here is the mean daily energy expenditure (DEE), which is the time-averaged metabolic rate of a free-ranging animal maintaining constant body mass where metabolism is fueled by food intake rather than transient depletion of reserves. This is the definition of "sustained metabolic rate" proposed by Peterson et al. (1990) and is estimated by the FMR. The absolute level of FMR is captured by the pre-exponential factor, B_0, in the WBE model and is set evolutionarily by ecology and lifestyle. This in turn influences the level of basal/resting metabolism because the anatomy and physiology required for different levels of sustained (field) metabolism have differing maintenance requirements (Clarke & Johnston, 1999; Killen et al., 2010).

Only very occasionally will an animal achieve its maximum metabolic rate, for example in a pursuit predator running down prey, or a prey organism evading a predator. The maximum metabolic rate cannot be sustained for long without exhaustion and the need for recovery. It is the long-term average daily energy expenditure that is the result of selection, and hence the focus of the WBE model. FMR varies from moment to moment, dictated by what the animal is doing, and this is accommodated by changes in blood flow, mediated through the feedback mechanisms discussed above.

The crucial point here is that the range of blood flows required by the variation in body size within broad groups such as mammals, reptiles, or fish is far greater than can be accommodated by the adjustments and feedbacks that characterize short-term variations in metabolic demand in an individual organism. This indicates that scaling over the full size range of mammals, and by extension other taxa, is determined by the structure and dynamics of the circulatory system (Coulson, 1986). This is why the WBE model is relevant only to large scales and broad groups, and not to the daily energetics of individual species; for the latter, we need different models. What is critical from the viewpoint of an individual cell is that metabolic rate is set by demand, what the animal is doing at that particular moment. Whole organism metabolic rate is almost always operating at less than full capacity, but that full capacity is determined by the cardiovascular architecture.

OXYGEN AND SIZE

A fundamental link between size and oxygen delivery to the tissues is also suggested by the relationship between organism size and oxygen availability in the environment. Chapelle and Peck (1999) showed that while the size of the smallest species in an assemblage of amphipods was pretty much the same everywhere, there was a striking relationship between the size of the largest species and the oxygen content of the water (Figure 4).

Amphipods lack hemocyanin and oxygen is carried in solution in the hemolymph. The solubility of oxygen

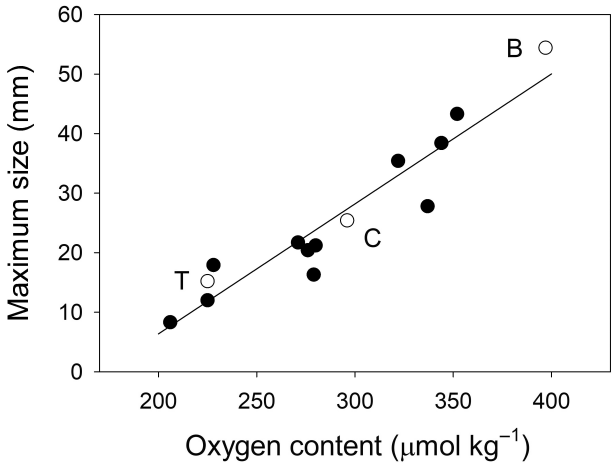

FIGURE 4 Relationship between the size of the largest species in an assemblage of amphipods and the oxygen content of the water. The line shows a least squares fit. Black symbols are for marine assemblages ranging from polar to tropical waters, and white symbols for freshwater (B, Lake Baikal, C, Caspian Sea; T, Lake Titicaca). Data from Peck and Chapelle (2003, 2004).

in water depends on temperature and salinity, but data from freshwater assemblages in Lake Baikal and the Caspian Sea, and from Lake Titicaca where the oxygen content is reduced because of its altitude, allowed the influence of oxygen to be disentangled from that of temperature (Peck & Chapelle, 2003).

A relationship between size and oxygen availability is also evident in terrestrial (air-breathing) organisms. Oxygen currently makes up ~21% of the atmosphere, but in the Carboniferous and Permian periods it reached much higher levels (>30%). At this time, there were dragonflies much larger than any known today, with wing lengths exceeding 30 cm (Dudley, 1998; Graham et al., 1995) and many terrestrial invertebrates were also larger.

There is thus a clear relationship between size and environmental oxygen availability, mediated through the anatomical systems that deliver environmental oxygen to the tissues (Harrison et al., 2010; Kaiser et al., 2007; Peck & Maddrell, 2005; Verberk et al., 2011). This provides strong support for a key assumption underpinning the WBE model.

THE INFLUENCE OF TEMPERATURE

A few years after the publication of the WBE model, Gillooly et al. (2001) added a term to capture the relationship between metabolic rate and temperature. This temperature term was a simple Boltzmann factor:

$$e^{-\frac{E}{k_B T}} \quad (2)$$

where E is an apparent activation energy (AAE, in joules), k_B is Boltzmann's constant ($1.380649 \times 10^{-23}\,\mathrm{J\,K^{-1}}$) and T is absolute (thermodynamic) temperature (in kelvins). E was taken to represent an average activation energy for "*the rate-limiting enzyme-catalysed biochemical reactions of metabolism*" (Gillooly et al., 2001; italic text is verbatim), and based on a small number of literature values was assumed to vary between 0.2 and 1.2 eV, with a mean of ~0.6 eV ($1\,\mathrm{eV} = 1.60218 \times 10^{-19}\,\mathrm{J}$; see Appendix S1: Section S11). This is actually a very wide range of temperature sensitivities, equivalent to a range of Q_{10} values from 1.31 to 5.06 at a median temperature of 20°C. In later publications (e.g., Brown et al., 2004) the predicted range of values for E was narrowed to 0.6–0.7 (equivalent to Q_{10} values of 2.25–2.57 at 20°C), with a mean of 0.65 eV.

The Boltzmann factor has its origin in statistical mechanics and describes the fraction of particles (atoms, molecules) that exceed a specified energy E at temperature T. This simple idea explains the marked sensitivity of elementary reactions to temperature and the activation energy of a simple (elementary) reaction is now formally *defined* as its temperature sensitivity (Laidler, 1981, 1984). The Boltzmann factor is what links reaction rate to temperature in the Arrhenius equation:

$$k(T) = Ae^{-\frac{E}{k_B T}} \quad (3)$$

where $k(T)$ is the reaction rate at temperature T, k_B is Boltzmann's constant and A is a pre-exponential factor which captures the fraction of colliding molecules that have the correct orientation to react.

The enzyme-catalyzed reactions of physiology are very different from the simple reactions modeled by the Arrhenius equation: substrate binds to an enzyme, passing through an unstable transition state before being converted to products which are then released. Moreover, this takes place in an extremely crowded cellular environment where there is essentially no bulk water. Binding and release are mediated through the continuous internal movement of the enzyme molecule at the active site and are the rate-limiting steps as they involve the reorganization of many weak bonds; they typically have a time constant in the range 10^{-3}–10^{-6} s. In contrast, the reactions by which substrates are converted to products are extremely fast (10^{-12}–10^{-15} s, the timescale of molecular vibrations). It is now well established that quantum processes can play an important role in enzyme catalysis, and that thermal motion within the protein brings the

reacting centers close enough for these quantum processes to operate (Klinman, 2013; Klinman & Kohen, 2013). The Arrhenius equation cannot capture the complex processes involved in enzyme catalysis; instead, we must use transition-state theory (Evans & Polanyi, 1935; Eyring, 1935) or, for analysis over a wider temperature range, a full quantum-mechanical treatment (Brookes, 2017).

Both the Arrhenius and transition state models predict a linear relationship between reaction rate (log-transformed) and inverse temperature, and such linearity is usually assumed when fitting small datasets. When detailed data are available, however, the relationship can often be seen to be curvilinear (Knies & Kingsolver, 2010).

Capturing the downturn in enzyme performance at high temperatures

Historically, physiologists fitting statistical models to enzyme activity data have concentrated on the rising part of the relationship, over the "physiological temperature range" (roughly 0–40°C). In doing so they have ignored the decrease in activity on the warmer side of the optimum temperature, usually ascribing this to some form of protein denaturation (e.g., Ratkowsky et al., 2005). This decline in activity, however, starts long before irreversible thermal denaturation exerts any detectable influence on activity (Thomas & Scopes, 1998; see also Morowitz, 1978). Clearly something is missing from our picture of the thermal sensitivity of physiological activity and recently a number of models have been proposed to capture the full thermal behavior of enzyme activity without involving denaturation (Arroyo et al., 2022; Daniel et al., 2007; Daniel & Danson, 2010; Hobbs et al., 2013). These models are discussed by Clarke (2017) and Michaletz and Garen (2024) (see Appendix S1: Section S11).

Temperature and metabolic rate

The use of a Boltzmann factor parameterized from enzyme data to describe the relationship between organism physiological rates and temperature assumes that it is meaningful to scale up from the temperature sensitivity of a simple chemical reaction to the thermal behavior of a whole organism. It also carries the implicit assumption that temperature determines metabolic rate.

A subtle but important point here is that the fundamental temperature sensitivity of a physiological process can be determined only when that process is operating at maximum capacity; if it is not doing so, it is being limited by something else. A nice example of the importance of this subtlety is Bernacchi et al. (2001), who used transgenic tobacco plants containing only 10% of the normal concentration of Rubisco to determine the inherent thermal sensitivity of photosynthesis in vivo. The low concentration of Rubisco meant that the enzyme was operating at maximum capacity, and the temperature sensitivity determined was substantially different from previous estimates made under nonsaturating conditions.

The inherent thermal sensitivity of ATP regeneration by oxidative phosphorylation can be estimated from state 3 respiration in isolated mitochondria, this being a measure of the maximum rate at which ATP can be regenerated. This has an across-species Q_{10} of 2.11 (AAE = 0.55 eV) (Figure 5). Our best current estimate of the inherent temperature sensitivity of ATP regeneration (and hence oxygen consumption) is thus distinctly lower than the canonical value of 0.65 eV proposed by Gillooly et al. (2001). Ketchum and Nakamoto (1998) determined a Q_{10} of 2.30 (AAE 0.61 eV) for an isolated membrane preparation of ATP synthase from *Escherichia coli*, suggesting that ATP synthase activity is not the rate-limiting step in oxidative phosphorylation.

In mammalian cells, however, mitochondria typically operate well below maximum capacity (Wilson, 2013). In other words, under normal circumstances, the metabolic rate is not limited by temperature, and the relationship of oxygen consumption with temperature is therefore indirect. The rise in oxygen consumption we see when cell temperature increases follows because the rate of a number of cell processes changes when temperature changes, and they then require more (or less) ATP. A problem is that we do not know with any certainty what these processes are, though they are likely to include gene

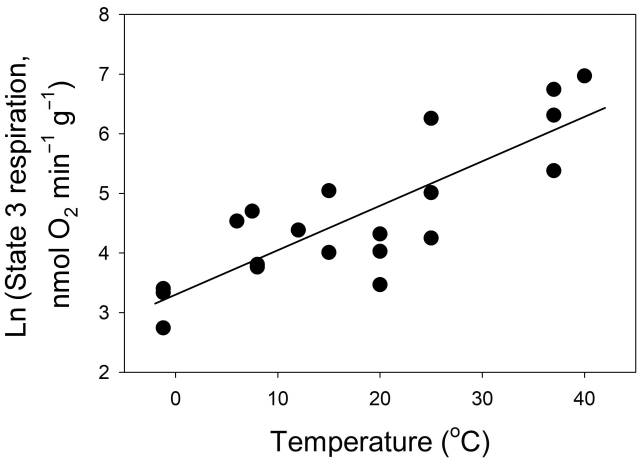

FIGURE 5 Mitochondrial state 3 respiration and temperature (from Clarke & Pörtner, 2010). Note that the respiration data have been expressed per gram of mitochondrial protein to correct for the varying number of mitochondria in the different assays.

expression, protein synthesis, ion regulation, and membrane turnover. What we observe in the whole organism is thus an integration of the oxygen demand from a suite of cellular and organismal-level processes that change when temperature does, and this relationship can only be described statistically (Clarke, 2004; Clarke & Fraser, 2004).

Intermediary metabolism is highly conserved across life on Earth, and hence its thermal sensitivity might also be expected to be. The relationship of whole organism metabolic rate with temperature can be estimated from a statistical model that allows the scaling of metabolism with body mass and temperature to be fitted simultaneously, and any interactions to be assessed. Apparent temperature sensitivities for different classes of vertebrates vary from a Q_{10} of 3.14 in mammals to 2.01 in fish and amphibians (Table 4). The apparent temperature sensitivities differed significantly between classes, but this was driven by the high value for mammals; if mammals are excluded from the statistical model, then there is no significant difference in temperature sensitivity between classes. This qualitative pattern is similar to that of Clarke (2017) and White et al. (2006), though the exact values of the exponents differ because of small differences in the datasets and statistical analysis. The all-vertebrate data, whether mammals are included or not, exhibit a temperature sensitivity of resting metabolism distinctly lower (being equivalent to an AAE of 0.55–0.57 eV) than the value assumed by the MTE (0.65 eV). Why mammals should be so different is not clear, although phylogenetic effects may be involved (see discussion below).

Data for invertebrates are few, but median Q_{10} values for arthropods, echinoderms, and mollusks range from 1.41 to 2.91 (Clarke, 2017). Within-species Q_{10} values for fish, reptiles, echinoderms, insects, and crustaceans range from 2.21 to 2.65 (Clarke, 2017).

Fitting an exponential (Q_{10}) or Arrhenius model provides a simple statistical description of temperature sensitivity averaged over the range of body temperatures for which data are available. These two models are not linearly related (see Appendix S1: Section S11). They do, however, provide closely similar fits (Figure 6) and are effectively indistinguishable over most of the physiological temperature range; even above 30°C, the difference is trivial in comparison with the variance in the data. The two models explain essentially identical fractions of the variance and fit the same number of parameters, so the choice between them is entirely arbitrary.

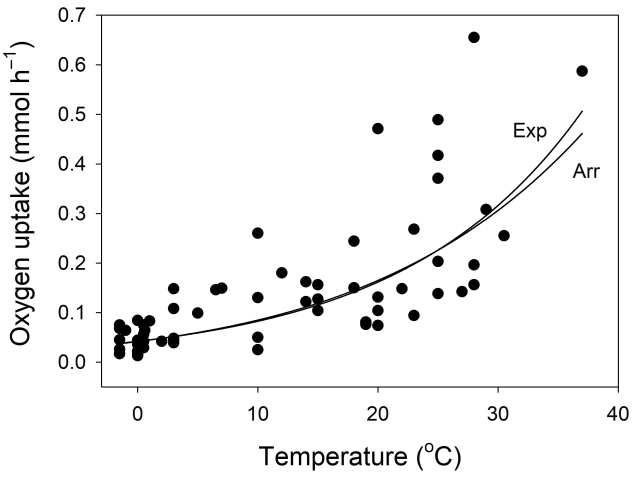

FIGURE 6 Resting metabolic rate in teleost fish as a function of temperature (data from Clarke & Johnston, 1999, here plotted in original measurement units rather than converted to energy). The two lines show the Q_{10} (exponential: Exp) and Arrhenius (Arr) models. Data were fitted with a general linear model to allow for the effects of body mass and temperature simultaneously (see Appendix S1: Table S2). As the model was fitted after log transformation of the metabolic rate and body mass data, when plotted in linear space, as here, the fitted models do not fall in the middle of the data.

TABLE 4 Apparent temperature sensitivities of vertebrate basal or resting metabolic rate.

Class	*n*	Slope	SE	Q_{10}	Tb range	AAE (eV)
Mammals (>150 g)	639	0.115	0.012	3.14	30.5–40.7	0.85
Birds	83	0.074	0.025	2.09	36.0–42.0	0.55
Reptiles	155	0.087	0.009	2.39	16.0–40.0	0.64
Amphibians	160	0.070	0.010	2.01	10.0–30.0	0.52
Fish	69	0.070	0.010	2.01	−1.50–37.0	0.52
All vertebrates	1101	0.078	0.004	2.17	−1.50–42.0	0.57
All vertebrates excluding mammals	462	0.074	0.005	2.09	−1.50–42.0	0.55

Note: Analysis with a general linear model: Ln(BMR, in watts) as a function of Ln(body mass, in kilograms) and Tb (body temperature, in kelvins) with Class as a fixed factor. Slope is the regression coefficient for temperature; SE is the standard error of the estimated slope; *n* is the number of species (one data point per species); Q_{10} is the temperature sensitivity determined from the slope; Tb range is the range of body temperatures for that class; AAE is the apparent activation energy calculated for a median body temperature of 20°C. Data from Clarke (2017). Full details of the statistical analysis are given in Appendix S1.

Temperature sensitivity of daily energy expenditure

The temperature sensitivity of resting or basal metabolic rate appears to be fairly similar across many taxa. However, BMR comprises only a fraction of routine metabolism (typically about 10%–25% in vertebrates: Clarke, 2017). Day-to-day energy flux is dominated by processes for which we have no data on temperature sensitivity. Indeed, it is perfectly possible that in many terrestrial organisms, behavioral thermoregulation and trade-offs mean that an individual's average daily energy expenditure is largely independent of environmental temperature. Marine ectotherms, however, will always have a body temperature close to that of the seawater in which they live.

THE CENTRAL EQUATION OF THE METABOLIC THEORY OF ECOLOGY

Combining the Boltzmann factor with the scaling relationship derived from the WBE model produced what has become known as the central equation of the Metabolic Theory of Ecology (Brown et al., 2004):

$$B = B_0 M^b e^{-\frac{E}{k_B T}} \quad (4)$$

Here B is metabolic rate, M body mass, and T is body temperature. The pre-exponential factor B_0 sets the level of metabolism, b is the scaling exponent, k_B is Boltzmann's constant and the apparent activation energy E captures the temperature sensitivity of metabolism.

This was not the first equation linking a scaling relationship for metabolic rate and body mass with an exponential temperature term. This had been done previously by Robinson et al. (1983); their equation was:

$$V = aM^b e^{cT} \quad (5)$$

Here V is the mass-specific oxygen consumption, M is body mass, (so $V = B/M$), T is temperature and a, b and c are constants whose values are estimated by fitting to data. This differs from the MTE equation in two aspects: the scaling exponent (b) is fitted empirically rather than being defined by theory and the exponential temperature factor is a Berthelot rather than Arrhenius model (see Appendix S1: Section S11). Several subsequent analyses of metabolic rate, notably for marine invertebrates, also used a combination of a power law to capture the scaling with mass and an exponential factor for the variation with temperature (see for example Ikeda, 1985). These equations were all conceived as statistical models, and were typically fitted as multiple regressions using least-squares. They differ from the central equation of the MTE in that all parameters are free, and the statistical analysis allows for the significance of interactions to be assessed.

Incorporating stoichiometry

The MTE central Equation (4) is concerned solely with energy, but organisms also require nutrients. Among the macronutrients, N is essential for building proteins, and P is needed for membranes and nucleic acids, together with the molecules that move chemical potential energy around the cell. In addition, a range of ions (protons, alkaline earths, and halogens) are used to store energy temporarily across membranes or in signaling, and transition metals are key to the structure and operation of some enzymes, notably those involved in transferring nutrients or energy (Fraústo da Silva & Williams, 2001).

It has long been recognized that the growth of terrestrial plants and aquatic phytoplankton depends critically on the availability of N and P (Redfield, 1958). In animals, a shortage of a specific element or an imbalance in the influx of others can exert significant influence on performance (Elser et al., 2006; Sterner & Elser, 2002). Particular attention has been directed at the ratio of macronutrients in relation to trophic level (Clarke, 2008) and the relationship of P content to growth rate (Elser et al., 1996, 2000). Allen and Gillooly (2009) and Kaspari (2012) have explored ways of incorporating nutrient dynamics into the MTE, but this is tough to do and has yet to develop a significant body of work.

EXTENDING THE MTE

The central equation of the MTE deals with only one aspect of an organism's energy flow, albeit an important one: the dissipation of energy in respiration. When fitted to data for basal or resting metabolic rate, as it typically is, then it deals with an even more restricted aspect, namely the cost of staying alive. All other things an organism must do, such as obtain resources, grow, move about, or reproduce, are not included. When fitted to data for mean daily energy expenditure (as estimated by FMR), then the MTE central equation becomes a more relevant measure of energy use.

It is this central equation that defines the fundamental scope of the MTE, which is the dissipation of energy through respiration. The theory has been used, however, to explore a wide range of physiological and ecological processes. The underlying assumption here is that since

ATP supplies, directly or indirectly, the energy for everything an organism does, the scaling of respiration may be reflected in other aspects of an organism's physiology or ecology. These extended predictions have typically used the MTE scaling relationship assuming only that metabolic rate scales with mass to the three-quarter power ($b = 0.75$) and a temperature dependence equivalent to an AAE of 0.65 eV ($Q_{10} = \sim 2.4$). A few studies have, however, explored the importance of variability in the scaling parameters (see, e.g., Dell et al., 2011, 2014).

Two important areas of ecology where attempts have been made to formally include the MTE in broader models are growth and biological diversity.

MTE and growth

One of the first, and most influential, developments of the MTE was the ontogenetic growth model proposed by West et al. (2001). This model is a simple conservation of energy equation whereby the total metabolic rate of an organism, B, is the sum of energy devoted to *the creation of new tissue* (growth) and the energy required for *the maintenance of existing tissue* (West et al., 2001; italic text is verbatim):

$$B = E_c \frac{dm}{dt} + B_m m \quad (6)$$

Here m is the mass at time t, E_c the energetic cost of creating a unit of biomass and B_m the metabolic cost of maintaining unit biomass. In the original paper this equation was expressed in terms of a representative individual cell, scaled up by the number of cells in the organism. The presentation here follows later formulations of the model, expressed in terms of body mass. This equation is balanced at the whole-organism level if (and only if) the B_m term includes all the broader costs of staying alive, including activities such as foraging and territorial defense as well as the organism-level costs of maintenance such as circulation, neural activity and immune function.

West et al. (2001) defined B, *the incoming rate of energy flow* as the *average resting metabolic rate of the organism*, and the metabolic cost of producing new tissue, E_c, as the *energy content of mammalian tissue*. This parameterization is flawed thermodynamically: metabolic rate is a measure of energy dissipated to the environment as heat, and this energy cannot also be invested in new tissue without contravening the First Law of Thermodynamics (the conservation of energy: Clarke, 2019; Makarieva et al., 2004). In this context it is the thermodynamic definition of metabolism as the conversion of chemical potential energy to heat that helps to avoid ambiguity.

In a growing organism, an anabolic pathway provides the raw material (monomers) from which new tissue is built, while a catabolic pathway provides the ATP required to construct new tissue from these monomers. Simple calculation shows that in a typical protein, >99% of the energy comes from the monomers (food or reserves) and <1% comes from ATP (metabolism) (Clarke, 2019). A complete model of the energy flow in growth must include both the anabolic and catabolic pathways. West et al. (2001) confuse the two pathways and also fail to recognize the energy overhead of synthesizing new tissue (the cost of growth: Clarke, 2019; Parry, 1983).

A later paper (Moses et al., 2008) tackled some of the errors in the original formulation of the model, redefining B rather ambiguously as "*the rate of metabolic energy assimilation*." A further more radical revision (Hou et al., 2008) is essentially a simplified version of the balanced energy budget equation developed many years previously during the International Biological Program (Petrusewicz & Macfadyen, 1970; Winberg, 1956), though expressed in dynamic terms rather than as a discrete time difference model. Despite these modifications, it is the original model that has attracted most attention and use, suggesting that its thermodynamic flaws are not widely recognized.

It has long been known that maximum growth rate scales with adult body size, with an exponent in vertebrates of b ~0.75 (Case, 1978; Hatton et al., 2019). This is perhaps not surprising, as the same cardiovascular system supplies the cells with both oxygen to power metabolism and nutrients to fuel growth. Growth has, however, yet to be successfully incorporated into the MTE. The use of energy for growth in an individual organism is currently handled better by the traditional balanced energy equation (Clarke, 2013; Petrusewicz & Macfadyen, 1970) or dynamic energy budget theory (Kooijman, 2010). For broad-scale comparisons across vertebrate species of differing body mass, a scaling exponent for growth of ~0.75 is a useful empirical result. For invertebrates, the situation is complicated by metamorphosis and shape change during ontogeny (Hirst et al., 2014; Hirst & Forster, 2013) and we lack a general framework for invertebrate growth that might be incorporated into ecosystem models.

MTE and biological diversity

The MTE has also been extended to explain global patterns of plant and animal diversity (sometimes referred to as the "metabolic theory of biodiversity":

Stegen et al., 2009). The key idea is that metabolic rate influences the generation of mutations though oxidative damage to the genetic material. This was then coupled to the idea of energy equivalence (Damuth, 1987) to predict that species richness should vary directly with body temperature (Allen et al., 2002). Evidence for energy equivalence remains equivocal, and Allen et al. (2007) subsequently recast the theory, abandoning the dependence on energy equivalence in favor of a conceptual model linked to the "more individuals mechanism" (larger populations buffer species against extinction: Lande, 1993).

A link between metabolic rate and speciation rate had been suggested previously (Martin, 1995; Martin & Palumbi, 1993) and both attempts to link the MTE to biological diversity center on the role of temperature inducing more mutations through "*the generally faster biological rates observed at higher temperatures*" (Brown et al., 2004; italic text is verbatim). The number of neutral mutations accumulated may indicate the period of time since divergence, but it is not simply the accumulation of mutations that drives speciation. Indeed, we do not know which, or how many, genes are involved in postzygotic isolation, although we might assume that as the number of mutations increases, so do the chances of genetic incompatibilities in hybrids between lineages in the process of allopatric speciation (Coyne & Orr, 2004).

Both models linking the MTE to biological diversity predict a positive relationship between richness and temperature. They also have two simple predictions for patterns of diversity at the global scale:

1. Where temperature remains constant over large spatial scales, then species richness in ectotherms should also be uniform.
2. The narrow range of body temperatures in endotherms means that birds and mammals should show greatly reduced patterns in richness compared with ectotherms.

Neither of these predictions is upheld. In the deep sea, temperature is remarkably uniform over vast spatial scales. The temperature of the global ocean is <4°C below ~1000 m depth, and the deep-sea is uniformly cold, typically <2°C, making cold water the largest habitat by volume on Earth. These low temperatures are caused by the generation of oxygen-rich cold bottom water at high latitudes (principally in the Antarctic) which spreads equatorward, cooling and ventilating the deep sea. Despite this uniform temperature in the deep sea, there are steep latitudinal clines in the diversity of gastropod and bivalve mollusks, isopods, nematodes, and foraminifera, though the patterns differ across the various groups and are far less distinct in the southern hemisphere (Culver & Buzas, 2000; Lambshead et al., 2000; Rex et al., 2000, 2005) (Figure 7). Furthermore, in many shallow water marine invertebrates, the correlation between species richness and environmental temperature is poor or nonexistent (Clarke, 2009). Lastly, both birds and mammals show very strong latitudinal clines in richness (Jenkins et al., 2013). A number of authors have found almost no support for the postulated relationship between environmental temperature and species richness (Algar et al., 2007; Hawkins et al., 2007; Latimer, 2007) or diversification rate (Liu et al., 2023; Orton et al., 2019; Soria-Carrasco & Castresana, 2012; Tietje et al., 2022). Some support has, however, been reported for fish (Wright et al., 2011) and mammals (Rolland et al., 2014).

MTE AND PLANTS

In parallel with the animal model that is the focus of this paper, WBE also proposed a model for vascular plants (Enquist et al., 2000; West et al., 1997, 1999, 2000). Vascular plants have a very different mechanism from animals for obtaining nutrients and moving these about the body: the leaves that are the site of photosynthesis are at the distal ends of the delivery network for water and mineral nutrients (the xylem), whereas gases (CO_2 and O_2) are exchanged directly with the atmosphere and reach the cells by diffusion. Sugars, amino acids, and other metabolic products of photosynthesis are then transported from the leaves by the phloem. An additional constraint in plants is the need to counter mechanical stresses such as bending in the wind and for support against gravity (Farnsworth & Niklas, 1995; Niklas & Spatz, 2006, 2012; Price et al., 2010, 2022).

The xylem that carries water and nutrients is comprised of tubes that run continuously from roots to leaves. WBE developed a model in which the xylem vessels taper and which allows for the strengthening of the tissues in response to mechanical stresses. It is thus more realistic than earlier simple pipe models (Shinozaki et al., 1964). The WBE model predicts scaling exponents for a wide variety of structural features and an exponent of 0.75 for "metabolic rate" (Enquist et al., 2000; West et al., 1999).

Defining metabolic rate for a plant, however, is not straightforward. This is because a plant's metabolism comprises two distinct flows of energy that are not directly coupled: a conversion of light energy to chemical potential energy in sugars (photosynthesis) and the use of this potential energy to fuel growth and a diverse secondary metabolism (respiration). If a plant has sufficient water, at any instant the rate of photosynthesis is controlled principally by a complex interaction between light

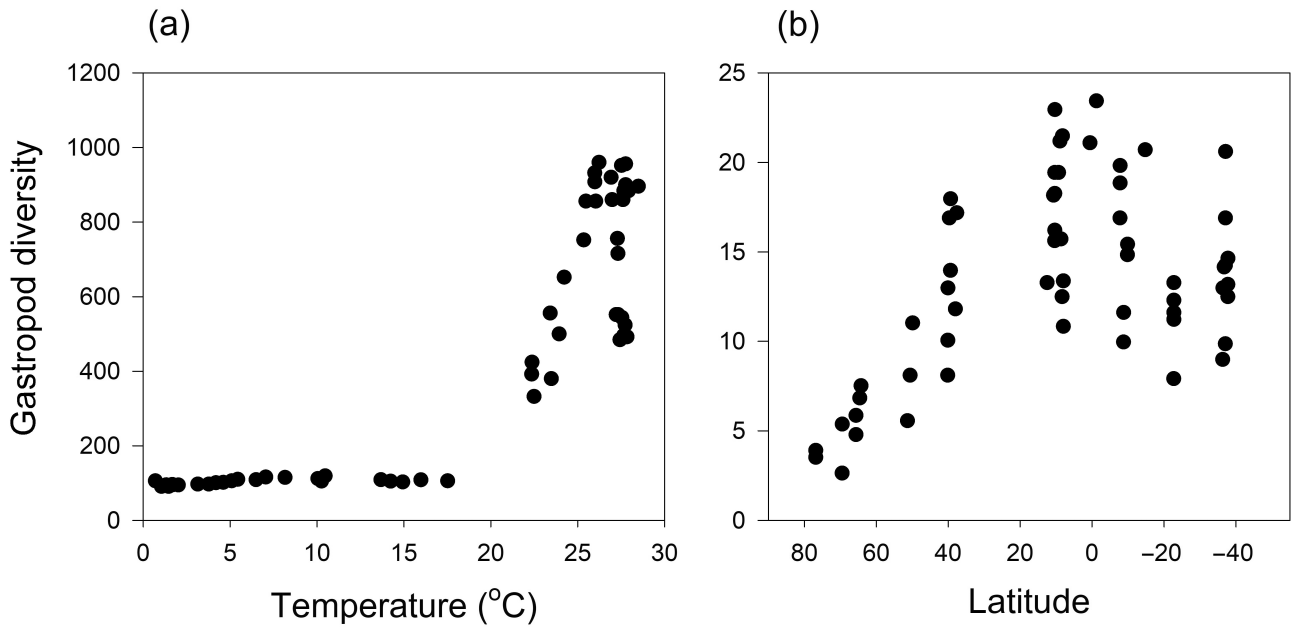

FIGURE 7 Species richness and temperature in gastropod mollusks. (a) Relationship between richness and mean environmental temperature for shallow water gastropods on the Atlantic continental shelf of North America (from Clarke, 2009). The relationship is markedly biphasic: There is a small increase in richness with temperature up to about 20°C, and a very steep relationship thereafter. (b) Relationship between gastropod richness (here as Sanders–Hurlbert expected number of species for 50 individuals) and latitude in the deep sea. There is a steep latitudinal cline in the northern hemisphere despite an essentially uniform seawater temperature, and a nonsignificant relationship in the southern hemisphere (from Rex et al., 2005).

climate, CO_2 concentration, temperature, and wind speed (Xiong & Nadal, 2020). On broader temporal and spatial scales, however, plant growth is limited by water and nutrient availability (especially N and P). As with the WBE animal model, the WBE plant model is concerned with patterns on the broadest scale rather than particular species or individual plants.

WBE are explicit in defining metabolic rate as the "*gross rate of photosynthesis*," which they take to be proportional to the rate of water flow through the xylem (West et al., 1999). The WBE plant model is thus dealing with a quite different aspect of metabolism (conversion of light energy to chemical potential energy) from the animal model (conversion of chemical potential energy to heat). However, West et al. (1999) also argue that "*it is reasonable to assume that growth rate ... is directly proportional to metabolic rate*." WBE do not include terms for the relationship between the supply of water and nutrients in the xylem and photosynthesis, or between growth rate and "metabolism"; it is simply assumed that they are linked mechanistically.

A direct test of the WBE model is the scaling of xylem flow, and this had already been shown to have a scaling exponent of ~0.75 (Enquist et al., 1998). Subsequently, Meinzer et al. (2005) found scaling exponents for xylem water flow of ~0.69 in angiosperms and ~0.74 in gymnosperms (conifers). Xylem water flow is not easy to measure, and so the WBE plant model has also been tested by examining the scaling of dark respiration rate and growth rate (annual biomass accumulation) (Enquist et al., 1999). Such tests are indirect because there are many metabolic steps between the process being measured and the xylem flow that is the focus of the WBE model. Niklas and Enquist (2001) found that annual biomass production scaled with an exponent of ~0.75. Data for dark respiration, however, are more mixed: Reich et al. (2006) found a scaling exponent of ~1, but the plants were mostly seedlings. Mori et al. (2010) used a much wider range of species and sizes, and showed that the scaling of dark respiration was curvilinear, with the exponent being ~1 at small size (and ~1.2 if below-ground material was included in the mass term), but nearer to 0.75 at larger size.

The WBE plant model has been used as a basis to develop broader models for plant growth and carbon flux at large spatial scales, and has proved valuable for modeling carbon flux through forests and ecosystems (Enquist et al., 2007, 2009).

Status as theory

Everything that an organism does involves the movement of energy. That ecology is fundamentally metabolic is thus not a theory in itself; it follows directly from

thermodynamics. Peters (1983) argued that the statistical (allometric) relationship between metabolic rate and body size alone constituted a meaningful ecological theory. It does not. Without the physical understanding of the mechanism that underpins the observed relationship, we cannot recognize those organisms or circumstances to which it does and does not apply. An allometric relationship by itself is limited in its usefulness; as with any pattern in ecology, its value lies in pointing to mechanism. The MTE provides such a mechanism for the scaling of metabolic rate with body mass. While empirical data are varied in the extent to which they support the WBE model, it remains the most widely accepted physical explanation for the scaling of metabolic rate in organisms with a circulatory system.

A good theory has to be built from sound first principles, be internally consistent, and able to explain past observations; it must also be predictive and thus be open to testing. The mathematical challenges of hydrodynamics are formidable, and at present there is no consensus as to whether the WBE model is internally consistent or not (Price et al., 2012). Analysis of the assumptions underpinning the model has shown that these are generally met in the mammalian vascular system, although the requirement of volume-filling may not be met fully (Huo & Kassab, 2012) and the assumption that the size of capillaries is invariant across mammals has long been known to be incorrect (Dawson, 2001, 2003, 2010; Gehr et al., 1981). The extent to which small variations in the architecture of the circulatory system affect the predicted scaling exponent was analyzed in detail by Savage et al. (2008).

Like all useful models, the WBE model is a simplification of reality, but in reducing the problem to its basics, it provides a simple equation that captures important features of energy flow within organisms. The WBE model does not have generality across all living organisms because it does not apply to unicells or diploblastic invertebrates (Table 3). This is not a problem as long as the model is not applied to domains for which it is not relevant (for example microbial or protist metabolism).

The second part of the central MTE equation is a Boltzmann factor used to capture the correlation between metabolic rate and temperature. The Boltzmann factor is a useful statistical description of an integrative measure over a limited temperature range, but unlike the WBE model, it is not mechanistic. This difference has important consequences for any predictive use of the MTE.

Reception

One immediate response to the publication of the WBE model was to spur the development of a suite of alternative models. Of particular interest are those of Banavar et al. (2002, 2010) who showed that a scaling exponent of 0.75 emerges from a much simpler analysis of distribution systems with no assumptions about energy minimization. Other models are based on the interplay between resource capture through surfaces and its utilization by bulk tissue (Glazier, 2005; Kooijman, 2010). Since all of these models describe the same well-established empirical pattern, deciding between them is not a question of comparing divergent predictions with nature, but of considering their physical realism, underlying assumptions, and internal consistency.

There was also a flurry of papers arguing that the predictions of the WBE model that underpins the MTE were not matched by empirical data, particularly for mammals and birds. This would not necessarily invalidate the WBE model, as sometimes argued; it could simply indicate that additional factors are at work in endotherms, possibly the need to maintain a relatively high internal body temperature. The energy flow associated with this has been modeled (Ballesteros et al., 2018; Kwak et al., 2016; Roberts et al., 2010); to be general, any such model will need to predict both the curvilinear scaling of mammals and the shallower scaling in birds, but this has yet to be achieved.

Other critiques pointed to the heterogeneity of scaling when analysis is undertaken at ever lower taxonomic levels. This is to be expected, and was recognized by WBE, for as scaling analyses become ever more fine-grained, other factors that influence metabolic rate may come to dominate the observed pattern. Analyses showing this are Clarke and Johnston (1999) for fish, Chown et al. (2007) for insects, Hudson et al. (2013) for mammals, and Giancarli et al. (2023) for birds.

This raises the issue of the role of phylogeny in the scaling of metabolic rate: if there is significant heterogeneity in the scaling of different lineages, this needs to be allowed for in the statistical analysis to avoid a biased estimate of the scaling parameters (Freckleton et al., 2002). An analysis of scaling in fish suggested that different orders exhibit similar scaling of metabolic rate but different levels, associated with ecology (Clarke & Johnston, 1999). In mammals, there is a strong phylogenetic signal in metabolic rate and body temperature, but the estimated scaling depended on the statistical model used to allow for phylogenetic relatedness (Clarke et al., 2010). Generally, allowing for phylogeny had a small impact on the estimate of the mass scaling parameter (Duncan et al., 2007; Sieg et al., 2009; White et al., 2009), though this depended on the phylogenetic tree used (Symonds & Elgar, 2002) and assumptions concerning branch length (Clarke et al., 2010). This emphasizes that phylogenetic analyses come with their own set of

assumptions (Freckleton, 2009). Allowing for phylogeny did, however, reduce the estimated temperature sensitivity of BMR in mammals to a value similar to that of other vertebrates (Clarke et al., 2010).

The WBE model explicitly concerns a central tendency, and its predictions are for broad patterns that emerge at large scales rather than fine-grained or single-species analyses. For this reason, most MTE-related studies of scaling have ignored phylogenetic variability in looking for the broadest patterns.

The impact of the MTE on the wider field of ecology

Perhaps the most significant contribution of the MTE has been to direct attention back to the importance of energy in ecology and to renew interest in the role of body size. The most influential statement of the implications of the MTE for ecology in general is that of Brown et al. (2004), who describe the theory as:

> ...a mechanistic, quantitative, synthetic framework that characterises the effects of body size and temperature on the metabolism of individual organisms, and ... the effects of metabolism of individual organisms on the pools and flows of energy and matter in populations, communities, and ecosystems.

They also surveyed the range of ecological topics to which they felt the MTE central equation could be applied. These included individual growth, survival and mortality, and at the population or ecosystem level, biomass, production, population density, interspecific interactions, trophic dynamics and species richness. In other words, pretty much all of ecology.

Price et al. (2012) noted that within less than a decade, the MTE had been combined with information theory (Harte et al., 2008), life-history theory (Brown & Sibly, 2006; Charnov & Gillooly, 2004), resource limitation models (Allen & Gillooly, 2009; Elser et al., 2010; Hammond & Niklas, 2012; Lichstein et al., 2007; Niklas et al., 2005), neutral theory (Allen & Gillooly, 2006; O'Dwyer et al., 2009; Stegen et al., 2009), food web theory (Gillooly et al., 2006), predator–prey models (Brose et al., 2006; Vasseur & McCann, 2005; Weitz & Levin, 2006), and models of forest structure and dynamics (Enquist et al., 2009; West et al., 2009). This extensive body of work has yielded predictions on processes ranging from molecular evolution to food web structure. More recently, Humphries and McCann (2014) surveyed the areas of ecology where the MTE had achieved its greatest impact, as judged by a bibliometric network analysis. They suggested that for animal ecology these were the wider field of scaling, the role of temperature in patterns of biological diversity, trophic interactions within food webs, and evolutionary trade-offs. The impact of the MTE across these diverse fields of inquiry has not been equal, but the sheer range of topics is testament to a profound impact.

The key MTE papers continue to be well cited (Appendix S1: Figures S16 and S17). Citation rates do not, however, necessarily equate to intellectual influence in the sense of changing how we view the world. While a detailed review of the extensive work that has used the MTE is beyond the scope of this paper, some broad themes are worth comment.

One theme has been to build on the MTE with models exploring different aspects of ecology. The initial developments of the WBE model were to model growth (West et al., 2001) and patterns of biological diversity (Allen et al., 2002) but as discussed above, both of these have significant deficiencies. More successful were Savage et al. (2004) and Sibly et al. (2013) who examined the consequences of metabolic scaling for population dynamics. Dell et al. (2014) modeled predator–prey interactions, assuming an Arrhenius temperature dependence for most of the biological rates involved, and developed an extended theory that predicted a thermal dependency of equilibrium population density.

Perhaps because of the limitations of the MTE in estimating absolute metabolic rate, most studies using the MTE central equation have been concerned solely with the consequences of the scaling central tendency, almost always assuming the canonical values of 0.75 for the scaling exponent and 0.65 eV for the temperature sensitivity (but see Dell et al., 2011, 2014 for notable exceptions). This approach bypasses the nuances and details of the underlying WBE model. This may be because the mathematics is seen as formidable, or it is assumed that the problem has been solved, or perhaps because approximately three-quarters scaling is observed so widely that its mechanistic or evolutionary underpinning is of no immediate interest.

An example of this approach is the use of the MTE to explore the energetics of acoustic communication (Gillooly & Ophir, 2010; Ophir et al., 2010). Many animals use sound to communicate, and this can be energetically expensive. Sound is produced by muscular vibration, and Gillooly and Ophir (2010) assume that the frequency of the muscular vibration is directly proportional to metabolic rate, with the latter predicted from the MTE central equation. The model predicted a linear relationship between the dominant frequency of the sound produced and body mass, with a slope of −0.25.

Data for anuran amphibians (frogs and toads) are shown in Figure 8. The fitted slope is −0.22, the CIs around which include the predicted value of −0.25. Gillooly and Ophir (2010) fitted a relationship to combined data for insects, crustaceans, fish, anurans, reptiles, birds, and mammals. Although this mixes animals with very different sound production mechanisms, and with that sound transmitted through media with very differing physical properties (air and water), the overall line also had a slope of −0.21. While this might indicate strong support for the model based on metabolic rate, sound frequency is also determined by the size of the vocal apparatus, with larger muscles having intrinsically lower vibration frequencies. This means that there is a strong relationship between the dominant frequency and body size, as was found for 136 species of frog by Gingras et al. (2013). In both these models, there is an inverse relationship between body size and dominant frequency (larger species produce lower sounds). The choice between them thus depends on their underlying physical realism and internal consistency. Does the energy available from metabolism dictate the frequency of the sound produced, or does metabolic rate reflect the energy cost of producing that sound?

A second example comes from modeling movement across the landscape. Hein et al. (2012) examined the possible relationship between energetics and maximum migration distance, assuming that the size dependence of the relevant mechanical variables and the energy provided by metabolism all followed the canonical values assumed by the MTE. The modeling inevitably involved a number of simplifying assumptions but suggested a positive relationship between migration distance and body mass for walking mammals and swimming fish, although the relationship was more complex for flying organisms (for which insects and birds were combined). A recent bioenergetic model for animal dispersal not involving the assumptions of the MTE is Wilkinson et al. (2025). Again, the choice between these competing explanations depends on the realism of the physical mechanism on which they are based.

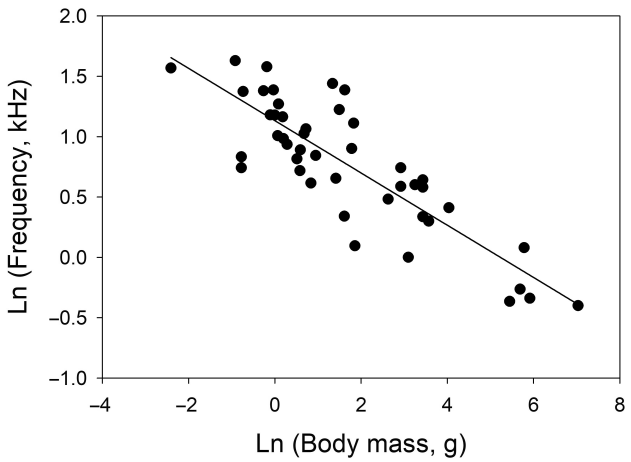

FIGURE 8 Relationship between the dominant frequency (in hertz) and body mass (in grams) in anurans. The fitted line has a slope of −0.217 (SE 0.021). The slope is −0.221 if body temperature is included in the statistical model, but the effect of temperature is not significant ($p = 0.126$). Data from Gillooly and Ophir (2010).

A simple energetic explanation for the ecology of movement may also prove to be incomplete. Thus it has long been recognized that the home range of an animal must in some way reflect its energetic needs, and a seminal early study suggested that in mammals home range scaled with body mass with a similar exponent to BMR (McNab, 1963). Subsequent work, however, pointed to scaling exponents ~1, albeit with a powerful influence from the mode of feeding (Lindstedt et al., 1986; Ofstad et al., 2016; Swihart et al., 1988). The home range, however, has to provide energy not just for BMR (maintenance) but for everything that the animal is doing on a day-to-day basis, and Boratyński (2019) demonstrated that the size of the home range is related also to the maximum aerobic capacity. Energetics of movement are also affected by behavioral choices (Berti et al., 2025) and individual differences (Laforge et al., 2025). Metabolism alone is insufficient as an explanation (Carbone et al., 2005; Jetz et al., 2004; Tamburello et al., 2015) and the MTE can at best point to only part of the answer.

A final illustration of the strengths and limitations of the MTE comes from the way in which the theory has been used to model the energetics of animals from habitats that are difficult to sample, such as the deep sea, or extinct organisms where energetics can only be inferred from morphological or anatomical data. The key problem here is that the MTE cannot predict the metabolic rate of an organism from its body mass and temperature alone; the pre-exponential constant that defines the intensity of metabolic rate has to be determined by fitting the WBE model to empirical data. But for understanding the energetic relationship between an organism and its environment, it is the absolute metabolic rate that we need to know.

McClain et al. (2012) used the general form of the MTE to explore broad scale ecological patterns in deep-sea benthos in relation to factors such as depth and particulate carbon flux, but used available direct measures of metabolic rate in deep-sea benthos to parameterize the model. Interestingly, they found that the empirical mass scaling exponent (0.80) and temperature sensitivity (0.47 eV) differed from the canonical MTE values. In a subsequent study, McClain et al. (2020) used

an extensive database of metabolic rates in shallow water benthos to parameterize the MTE model and were able to demonstrate marked differences in metabolic rate across benthic invertebrate phyla.

Extinct organisms pose a more difficult problem, in that there are no data at all with which to parameterize an MTE model or estimate absolute metabolic rate. Glazier et al. (2012) used the number and size of eye facets to estimate the scaling of metabolic rate with mass in nine species of Devonian trilobite. The inference of metabolic rate here relies on the relationship between eye facet size and number with body mass established for living insects (Chown et al., 2007). The estimated scaling exponents for the trilobites ranged from 0.74 to 1.1, with pelagic species generally having higher exponents than benthic species. But we still do not know what the absolute metabolic rate of a trilobite was, only how it may have varied with size.

Of all extinct organisms, it is the metabolic status of ancient reptiles that has attracted most attention because of the intense interest in the evolution of endothermy in the ancestors of birds and mammals. Endothermy can be inferred from a number of anatomical features, and body temperature can be estimated from skeletal isotopes, but we cannot estimate the absolute metabolic rate of a mammal-like reptile or a dinosaur other than by reference to living organisms that might provide a valid comparison. Thus Clarke (2013) used living reptiles as a comparison, coupled with body temperature estimates from skeletal isotopes, to demonstrate that many inferred dinosaur ecologies were perfectly feasible for an animal with a metabolism akin to that of a warm reptile.

One subtle consequence of the MTE has been an increasing tendency to view metabolism as a fixed attribute, whereby a species can be assigned a value for its "metabolism." In reality metabolism is dynamic, varying continually as the animal goes about its daily business. The important features of metabolism in relation to ecology are:

1. Metabolism is a cost; energy dissipated in metabolism has to be met from food or reserves.
2. Metabolic rate is determined by what an organism is doing, not the other way around.
3. Maintenance metabolic rate (BMR or SMR) varies between individuals, with consequences for ecologically important processes such as growth (Auer et al., 2017; Hawkins et al., 1986; Hawkins & Day, 1996; Norin & Metcalfe, 2019; Steyermark, 2002).
4. In most cases, the ecologically relevant measure is not BMR but the average daily energy expenditure, though the maximum metabolic rate can limit an organism's performance.
5. Metabolic rate varies between species, reflecting their lifestyle (Clarke & Johnston, 1999; Killen et al., 2010).

We can capture broad trends in metabolism across species in simple patterns such as scaling relationships such as that encapsulated in the MTE, but we must not lose sight of the evolutionary variability; these differences matter ecologically.

OUTSTANDING PROBLEMS AND FUTURE PROSPECTS

We now know that the scaling of metabolic rate with body mass in many organisms falls within the range 0.6–0.8 (Table 3). The WBE model provided the first general mechanistic explanation for this scaling, though it is notable that some invertebrate groups exhibit scaling exponents that are well below the minimum prediction of the WBE model (0.75).

While the WBE model provides a mechanistic explanation for the scaling of metabolic rate in organisms with a circulatory system, it predicts only a broad central tendency. Empirical data (Table 3) indicate that evolution and ecology can loosen allometric constraints to some degree; all that is needed is for the fitness benefits to outweigh the metabolic costs. How and why this occurs are intriguing problems yet to be tackled, and to achieve meaningful generalizations we need to examine physiological processes over as wide a range of taxa as we can (Krogh, 1929). It would therefore be valuable to strengthen our current picture of metabolic scaling in invertebrates in relation to the diversity of their circulatory systems. In this context, the biggest gaps in our current knowledge are robust estimates of metabolic scaling in polychaetes, gastropod mollusks, and planaria (flatworms).

One extra factor that is likely to be important here is cell size. Because a cell exchanges oxygen and nutrients through its external cell membrane, cell size (and hence its surface area to volume ratio) might influence its ability to take up resources and thereby limit the maximum metabolic rate that the cell can sustain, a constraint that could well be strongly evident in muscle cells. A consequence of this would then be that whole organism metabolic scaling could be affected by the fraction of the body that is muscle, and whether growth is achieved predominantly through an increase in cell number or cell size. This topic has been explored by Glazier (2022), Kozłowski et al. (2003, 2020), and Savage et al. (2007), but has not been incorporated into a refinement of the WBE model.

Outstanding problems

Despite the widespread uptake and use of the MTE, some outstanding problems remain. These include the special case of endotherms, the role of energy minimization, and the nature of the relationship between metabolic rate and temperature.

Although the mammalian circulatory system was the basis for the development of the WBE animal model, it is striking that the two endotherm groups are where the predictions of the model are less successful: mammals exhibit marked curvilinear scaling and birds very shallow scaling. It is tempting to conclude that this may reflect the importance of heat flow, but as yet there is no general theory of how the twin constraints of maintaining a high internal body temperature and the architecture of the circulatory system interact in determining the scaling of resting metabolic rate with size in these two groups. Such a refinement would go a long way to increasing the generality of the WBE model and hence the MTE.

There is now strong evidence for a direct link between the structure of the circulatory system and metabolic rate. Simpler models developed after WBE have suggested that three-quarter power scaling emerges from the necessary design of a distribution system alone, leaving unresolved the question of whether energy minimization invoked in the WBE model is actually a significant factor.

The final problem is that we do not know what the fundamental thermal sensitivity of oxygen utilization by cells or organisms actually is. Electron transport and oxidative phosphorylation (the process of regenerating ATP) comprise a tightly integrated series of reactions that include enzyme catalysis, physical diffusion, quantum processes, and mechanical work, many of which have differing inherent thermal sensitivity (Clarke, 2017). Perhaps the best insight here comes from a comparative study of state 3 respiration (a measure of the maximum rate at which ATP can be regenerated) in vertebrate mitochondria, which exhibits an across-species Q_{10} of 2.11 (equivalent to an AAE of 0.56 eV) (Figure 5). However, while important in a physiological context, this is not directly relevant to the MTE or ecology in general, as mitochondria usually operate well below maximal level, and the rate of ATP regeneration is set by factors other than temperature.

Furthermore, we do not yet have a clear understanding of what physiological processes change, and to what extent, when cell temperature changes. Oxygen consumption varies because the rate of a number of cell processes alters in response to a shift in temperature. What we observe in the whole organism is thus an integration of the oxygen demand of a suite of cellular and organismal level processes which change when temperature does, and the relationship of this to temperature can only be described statistically (Clarke, 2004; Clarke & Fraser, 2004). Because the temperature component of the MTE is based on empirical observation, albeit one that appears to be remarkably uniform across the domains of life, this limits the confidence with which it can be used predictively. It remains a key problem in ecology and physiology that we do not have a full understanding of the relationship between temperature and organism metabolism. In a time of rapid global change, this is an important gap in our knowledge.

MTE and future ecological questions

The two most pressing environmental questions of the moment are the ecological consequences of climate change and the widespread loss of biological diversity. A direct link between metabolic rate and species richness has proved to be elusive, and it is unlikely that the MTE will have anything to contribute to understanding or reversing the loss of diversity. For example, in the ocean, there have been major shifts in the distribution of marine organisms (Beaugrand et al., 2002; Perry et al., 2005), even though the large thermal capacity of water means that the change in temperature has been relatively small in physiological terms. The lesson here is that climate change is having a dramatic impact on the distribution of marine organisms, but these are effected through mechanisms such as changes in oceanography, dispersal, and species interactions more than by direct impacts on physiology (Pinsky et al., 2020).

Temperature changes in terrestrial habitats have been greater, and the impact of climate change on global processes such as carbon cycling means that there is an urgent need to understand how these processes are changing. The reservoir of carbon in soil exceeds that in terrestrial biota and atmosphere combined, with much of this concentrated in boreal and northern polar latitudes (Scharlemann et al., 2014). The release of carbon to the atmosphere through respiration of soil organisms is critical to climate change but is not well understood. Two approaches have been either to build models of the soil ecosystem to explore how this might respond to climate forcing or to determine the temperature sensitivity of soil respiration empirically.

The first challenge to building a model is the complexity of soil respiration, which emanates from a wide range of organisms with very different physiologies, many of which will have scaling properties different from those modeled by the MTE (notably bacteria,

protists and fungi). To handle this complexity, current models of carbon cycling typically utilize a range of living carbon pools, several litter pools, and typically three (fast, slow, and passive) soil carbon pools (Friedlingstein et al., 2006).

The second challenge is the nature of soil temperature. In general, surface soil temperatures are determined by the air temperature, though this will be moderated by the nature of the overlying vegetation. The extent of daily and seasonal temperature variations diminishes rapidly with depth, and at about 10–15 m depth, soil temperature is broadly equal to the annual mean air temperature. Below this depth, soil temperature may rise slowly, driven by geothermal heat flux. The flux of thermal energy through soil, and hence soil temperature, is influenced greatly by factors such as soil porosity and water content. These factors together make it difficult to model "soil temperature" in a simple manner for incorporation into an ecosystem model. Kirschbaum (2006) commented that despite considerable work, there was no consensus on the temperature dependence of organic matter decomposition. A simple exponential (Q_{10}) relationship is usually assumed, and current ecosystem models use Q_{10} values in the range 1.5–2 (Meyer et al., 2018).

A carbon cycle model based explicitly on the MTE is that of Allen et al. (2005), who built a general carbon flux model with three pools of carbon (autotrophs, heterotrophs, and a labile carbon pool). The metabolic processes removing carbon from the atmosphere (photosynthesis) or releasing it back (respiration) were modeled using the MTE, assuming temperature dependencies of 0.32 eV for photosynthesis and 0.65 eV for respiration. The model predicted a large (four orders of magnitude) difference in carbon turnover between aquatic systems dominated by phytoplankton and terrestrial systems dominated by trees and suggested a strong temperature sensitivity (an AAE of 0.79 eV) to the turnover of labile C in soils.

The use of the MTE to model ecosystem dynamics is a continually developing field (Stark et al., 2025). A challenge for a full ecosystem model is that it must include organisms that exhibit a variety of scaling, both isometric and allometric, and also allow for the difference in temperature sensitivity of production and respiration processes in the various taxa. A difficulty here is that some of these parameters are not fully established. For example, some studies have reported temperature coefficients for photosynthetic production and respiration that match the values assumed by the MTE (Regaudie-de-Gioux & Duarte, 2012), while others have found quite different values (Garcia-Corral et al., 2017; Huete-Ortega et al., 2012; Johnson et al., 2009; Marañón, 2008; Meehan, 2006).

If we are to predict the response of ecosystems to climate change, the temperature sensitivities of the key processes are perhaps the most critical of all parameters to have firmly established. While Allen et al. (2005) assumed the canonical MTE values in building their model of carbon cycling, empirical estimates of the temperature sensitivity of soil respiration vary widely, with published values of Q_{10} ranging from ~1 to >20, depending on the temperature range used and the temporal scale over which the estimates are made (Gu et al., 2008). Kirschbaum (2006) showed that Q_{10} values for soil respiration themselves varied inversely with temperature, and that estimates from laboratory incubations tended to be higher than those estimated from field soil warming experiments or from natural seasonal variations. Yvon-Durocher et al. (2012) compared the temperature sensitivity of respiration in soils estimated from seasonal change (mean AAE = 0.65, Q_{10} ~ 2.42) and by comparing soils from different latitudes, and hence different mean temperatures (mean AAE = 0.32, Q_{10} ~ 1.54). These two estimates are not strictly comparable because whereas the seasonal change is exhibited by a single community (albeit one where the active components may vary over the year), soil communities from different latitudes will differ greatly in a wide range of factors, including species composition.

Hamdi et al. (2013) compiled 112 Q_{10} values for soil respiration from 47 studies and showed that these fell almost entirely in the range 1.2–4.8. There were eight values >5 with a maximum >150. The median value was 2.5, which is higher than the 1.4 suggested by Mahecha et al. (2010) as a global value (Figure 9) (see also Appendix S1: Section S12).

Confining analyses to the implications of a central tendency sacrifices detail for simplicity but takes no account of two key features of scaling evident in the empirical data: that scaling relationships in nature are varied and that there is considerable ecological and evolutionary variation about the central tendency (Figure 1). Modeling ecosystem responses to climate change will need sophisticated ecosystem models, but key to meaningful predictions will be getting the parameterization correct, and also allowing for the variability evident in nature (Alcaraz, 2016). Not all organisms follow the MTE central tendency, and parameterizing models with this alone could lead to unreliable conclusions. In the sea, temperature changes slowly, and most organisms have a body temperature very close to that of the water in which they live. On land, this is not the case, and many organisms have sophisticated behavioral mechanisms that allow them to exert a degree of control over their body temperature. This makes a simple relationship between body temperature and energetics unrealistic, and recent

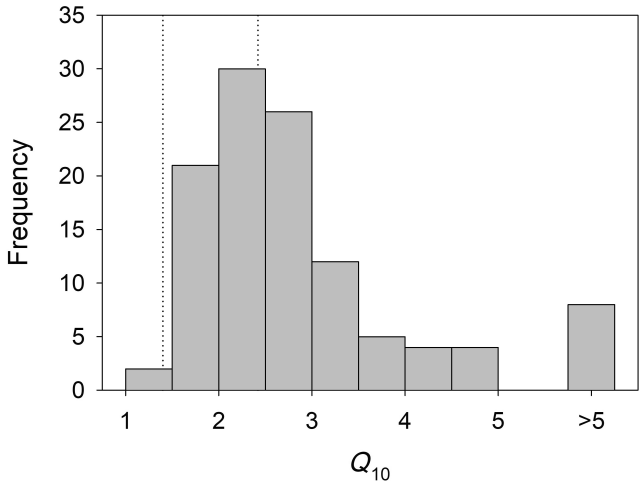

FIGURE 9 Frequency histogram of estimates of the temperature sensitivity (Q_{10}) of soil respiration; data ($n = 112$) are from 57 studies collated by Hamdi et al. (2013). The dotted lines show the global value of 1.40 proposed by Mahecha et al. (2010) and the canonical metabolic theory of ecology (MTE) value of 2.42 (apparent activation energy [AAE] = 0.65 eV).

studies have shown that behavioral thermoregulation may allow some terrestrial ectotherms to offset some of the effects of climate warming (Kearney et al., 2009). While organisms are undoubtedly shifting their range and their phenology in response to climate change, the mechanisms are more subtle and complex than can be modeled with simple equations such as the central equation of the MTE.

A final evolutionary comment

The enormous amount of work on the influence of body size on physiology and ecology has quantified, but not explained, an important physiological question: why do the cells of small and large species differ so much in their metabolic rates? From the perspective of a small lizard, the lower metabolic rate of an analogous cell in a Komodo dragon can be explained by the constraints imposed by the architecture of the circulatory system, as captured by the WBE model. There are also well-understood energetic and ecological benefits that come from a large size, which provide a clear rationale for the widespread evolutionary tendency for lineages to evolve large size (Cope's Rule: Baker et al., 2015; Hone & Benton, 2005).

But if we reverse the perspective, things become less clear. How is it that a cell in a large monitor lizard can remain viable with an intrinsic metabolic rate so much lower than in a small lizard? What are the metabolic or fitness costs that prevent the small lizard from lowering its maintenance costs per cell to match that of the monitor lizard or Komodo dragon? In an energy-limited world, selection will surely act to reduce maintenance costs as far as possible without compromising viability or fitness, but we have yet to identify what it is about cells from smaller organisms that means they are so much more costly to maintain. Scaling models such as the MTE describe this difference, but they do not explain it. At the core of the relationship between metabolic rate and size lies a profound, but as yet unanswered, evolutionary question.

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CONFLICT OF INTEREST STATEMENT

The author declares no conflicts of interest.

DATA AVAILABILITY STATEMENT

Metabolic data (Clarke & Johnston, 2025) are available from the UK Polar Data Centre at https://doi.org/10.5285/8D9FFD4A-16C0-4D48-9FC9-02988676EF78. Sources of all data are given in Appendix S1.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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