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REVIEWS AND SYNTHESIS

Testing the metabolic theory of ecology

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Abstract

The metabolic theory of ecology (MTE) predicts the effects of body size and temperature on metabolism through considerations of vascular distribution networks and biochemical kinetics. MTE has also been extended to characterise processes from cellular to global levels. MTE has generated both enthusiasm and controversy across a broad range of research areas. However, most efforts that claim to validate or invalidate MTE have focused on testing predictions. We argue that critical evaluation of MTE also requires strong tests of both its theoretical foundations and simplifying assumptions. To this end, we synthesise available information and find that MTE's original derivations require additional assumptions to obtain the full scope of attendant predictions. Moreover, although some of MTE's simplifying assumptions are well supported by data, others are inconsistent with empirical tests and even more remain untested. Further, although many predictions are empirically supported on average, work remains to explain the often large variability in data. We suggest that greater effort be focused on evaluating MTE's underlying theory and simplifying assumptions to help delineate the scope of MTE, generate new theory and shed light on fundamental aspects of biological form and function.

Keywords

Allometry, fractal, kleiber curve, metabolic theory, metabolism, scaling theory, WBE model.

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INTRODUCTION

The metabolic theory of ecology (MTE) integrates cellular and global-level processes (West *et al.* 1997, 1999; Gillooly *et al.* 2001; Brown *et al.* 2004) and has been described as one of the most significant recent theories in biology (Whitfield 2004). The scope of the theory continues to expand, and MTE continues to have enormous potential as a general theory in ecology (Brown *et al.* 2004). However, despite more than a decade since the first of these seminal papers were published, controversies about the theory remain with numerous papers questioning both its theoretical foundations and empirical validity.

Given the potential of such a broad reaching theory to provide a foundation for ecological enquiry and understanding, it is paramount to critically evaluate MTE. Any theory can be evaluated at one of multiple levels: by evaluating its internal consistency, by testing the validity of its simplifying assumptions and by testing its explicit predictions. Moreover, the interest in MTE has become so widespread that many additional assumptions, predictions, extensions and corrections have been added in its application to different questions. Hence, not all tests are equivalent in their efforts to evaluate the relevance and scope of the theory. To date, the overwhelming majority of tests have evaluated model predictions instead of directly evaluating the model's internal consistency and/or its assumptions. This is partly due to lack of available data, difficulty of measurements and a lack of emphasis on this approach within the field.

In an attempt to help focus efforts on those tests that have the strongest bearing on MTE's ultimate acceptance, modification or rejection, we detail four levels of evaluation that form a continuum of tests that will ultimately help to determine to what extent this work is useful as a general theory for ecology. Coarsely, these levels represent tests of decreasing importance in this sense: If a mathematical theory is internally inconsistent, then the question of testing its predictions becomes irrelevant. If it relies on assumptions that are largely divorced from reality, one may question the value of its

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predictions (but see Friedman 1953). However, when internal consistency and simplifying assumptions are valid, testing model predictions becomes paramount. If a model is internally consistent and all assumptions are supported empirically, but the predictions do not hold, this implies the theory is incomplete and that other factors and assumptions need to be added and included. The four levels we identify are as follows:

Level 1 – Evaluating the internal consistency of the underlying derivations.

Level 2 – Evaluating the validity of the assumptions.

Level 3 – Evaluating the explicit predictions.

Level 4 – Evaluating the extended predictions.

We identify explicit predictions as those emerging directly from the theory itself, and these have been identified in the seminal papers of MTE (West *et al.* 1997, 1999; Gillooly *et al.* 2001; Brown *et al.* 2004). Extended predictions are those that emerge from model assumptions and/or explicit predictions via the incorporation of additional assumptions. The scope of the theory has expanded considerably in recent years; thus, we focus on areas that have received the greatest attention and for which there has been adequate time for evaluation.

We draw a distinction between MTE as a mechanistic result of the West, Brown and Enquist (WBE) model (West *et al.* 1997, 1999), and alternatively, MTE as an empirical scaling relationship. Many of the extended predictions we will later refer to require only: (1) that metabolic rate (B) scales approximately with mass to the ¾ power and (2) that organismal metabolic rate has a temperature dependence described by a Boltzmann–Arrhenius factor

$$B = B_0 M^{3/4} e^{-E/kT} \quad (1)$$

where E is the 'average activation energy of metabolism' (~0.6 eV), k is $8.617 \cdot 10^{-5}$ eVK^{-1} (Boltzmann constant), B_0 is the normalisation constant and T is the temperature of the organism in Kelvin (Gillooly *et al.* 2001). If eqn 1 is taken as an empirical relationship or as an assumption divorced from the causal underpinnings of the WBE model (Robinson *et al.* 1983), then many extended predictions can be considered potential support for this observed mass-temperature dependence of biological processes, rather than as support for the network optimisation arguments that serve as the crux of the WBE model (Price *et al.* 2010).

Our goals here are as follows: first, to provide clarity and transparency regarding the assumptions and predictions of MTE and the conceptual links between different prediction levels. We hope that by drawing a distinction between these different levels of evaluation, we can help to focus effort on more direct tests of MTE's underlying theory and assumptions. At its most basic level, a model must be logically consistent. Once this consistency has been established, the next question is whether the theory is biologically useful or meaningful, which is assessed by comparing how well assumptions and predictions of different models match empirical measurements. Hence, our second goal is to evaluate MTE via stronger tests of its theoretical underpinnings. We summarise evaluations of the internal consistency of MTE and find that the original derivation of a universal ¾ scaling law is incomplete, and that a more complete derivation leads to deviations and a universal curve that is not a pure power law. We show that although many of MTE's assumptions are generally valid, other key assumptions are inconsistent with biological data, and several key assumptions remain untested. We argue that additional tests of MTE's assumptions are likely to provide fundamental insights about organismal structure and function, regardless of whether they are consistent with, or in contradiction to MTE. Finally, we briefly review a number of tests of MTE's explicit and extended predictions. In doing so, we find that the baseline of scaling proposed by MTE has strong empirical support in several cases. However, we also find that in almost all cases, there remains unexplained variation in function (e.g. metabolic rates of individuals), form (e.g. individual morphologies) and organisation (e.g. biodiversity) that cannot be explained by a single, universal scaling of mass and temperature. As we explain, the pursuit of mechanistic explanations that drive observed biological variation will require further refinement and improvement of MTE and/or the development of new theories.

Level 1: evaluating the internal consistency of the derivation of MTE

The derivations underlying any mathematically based model must be reproducible. This level of evaluation is critical as it leads to transparency between the model's assumptions, incorporated mechanisms and resulting predictions. In the case of MTE, several attempts have been made to re-derive the original model of WBE (Dodds *et al.* 2001; Kozlowski & Konarzewski 2004, 2005; Chaui-Berlinck 2006; Etienne *et al.* 2006; Apol *et al.* 2008; Savage *et al.* 2008), prompting clarifying responses in some cases from the original authors or their collaborators (Brown *et al.* 2005; Savage *et al.* 2007).

Here, we examine the internal consistency of the derivations that form the basis of the WBE theory (West *et al.* 1997, 1999) and the inclusion of temperature dependence (Gillooly *et al.* 2001). The WBE models, here denoted as Model A (for mammals) and Model B (for plants), both claim to lead to the same conclusion, that is, that metabolic rate scales with whole-organism mass to the ¾ in mammals and plants, respectively. MTE then assumes ¾ scaling and proposes, in Model C, an additional Boltzmann–Arrhenius temperature dependence. One may naturally ask: Are these models internally consistent? In other words, do the model predictions logically follow from the underlying assumptions and equations?

Evaluating Model A derivation: the ¾ allometric scaling in mammals

As originally described, Model A 'predicts structural and functional properties of vertebrate cardiovascular and respiratory systems, plant vascular systems, insect tracheal tubes, and other distribution networks' (West *et al.* 1997). However, the details of the model are mostly specific to cardiovascular systems typical in vertebrates, and the data presented to support it are primarily from mammals. Model A posits that mammals have evolved an optimal blood vessel network that both minimises energy loss through dissipation and wave reflections while also spanning the body such that capillaries are near enough to cells to deliver oxygen by diffusion (West *et al.* 1997). In this view, the mass-specific metabolic rate of different-sized organisms is the result of natural selection and follows logically from energy minimisation principles of hydrodynamics acting on hierarchical supply networks. Three assumptions to derive such a result are identified in the original paper; however, we follow Savage *et al.* (2008) in identifying both implicit and explicit assumptions (Table 1). From this hydraulic network structure and assumptions, the authors claim that the number of capillaries should scale with the ¾ power of body mass, and further, by assuming

Table 1 Model assumptions: Model's A (West *et al.* 1997), B (West *et al.* 1999) and C (Gillooly *et al.* 2001), the taxonomic group to which the model is most applicable, and their respective assumptions as referred to in the text

Model	Taxa	Assumption #	Assumption
A	Mammals	A1	The distribution network determines the scaling relationship between whole-organism metabolic rate and its mass because it both delivers the oxygen that fuels metabolic reactions and spans the body to deliver it
A	Mammals	A2	The arterial tree from the heart to the capillaries is hierarchical
A	Mammals	A3	Cylindrical vessels within the same level of the hierarchy are identical
A	Mammals	A4	The branching ratio, the number of new vessels stemming from a single parent vessel, is constant
A	Mammals	A5	The network is 'volume filling'
A	Mammals	A6	The power loss due to the flow of fluid is minimised
A	Mammals	A7	Capillary structure (length, diameter) and function are invariant across species
A	Mammals	A8	Oxygen exchange only occurs across capillaries to their surrounding tissue, not for other vessels
A	Mammals	A9	The network has a very large number of bifurcations and branching levels
B	Plants	B1	Each plant branch divides into a fixed number (usually 2) of equivalent daughter branches from trunk to petioles with no side-branching (same as A4)
B	Plants	B2	The plant has a very large number of bifurcations (same as A9)
B	Plants	B3	The lengths of branches decrease from base to petioles to satisfy 'volume filling' (same as A5)
B	Plants	B4	Elastic similarity applies uniformly to each branch (McMahon 1973)
B	Plants	B5	Tissue density is constant both within and across trees, including branches and petioles
B	Plants	B6	Branches are cylinders and do not taper within a level
B	Plants	B7	The terminal units (i.e. leaves and petioles) of plants have identical structure and metabolic rates, irrespective of plant size (same as A7)
B	Plants	B8	Resistance to water flow through the xylem network is minimised such that it does not scale with plant size (analogous to A6)
B	Plants	B9	The total number of xylem conduits does not change across branching levels in the plant
C	All Taxa	C1	The metabolic expenditures of an organism scale with supply at exchange surfaces
C	All Taxa	C2	Oxygen exchange only occurs across terminal vessels, not for other vessels
C	All Taxa	C3	Metabolic reactions are subject to the Boltzmann–Arrhenius temperature dependence
C	All Taxa	C4	The activation energy corresponds to a rate-limiting biochemical reaction or an average across reactions, e.g., the mean or mode of a unimodal distribution for activation energies across all biochemical reactions

invariance of oxygen exchange at capillaries, that metabolic rate scales with the ¾ power of body mass.

There have been three thorough re-considerations of the Lagrange optimisation method utilised in this derivation (Dodds *et al.* 2001; Apol *et al.* 2008; Savage *et al.* 2008). Dodds *et al.* (2001) argued that the area-preserving branching of conduit diameters and volume-filling decay of conduit lengths cannot be derived from the model as originally described. Hence, they concluded that ¾ scaling could not be derived based on hydraulic optimisation principles as stated in West *et al.* (1997). Similarly, Apol *et al.* (2008) concluded that full optimisation of the WBE model leads to either an invariant relationship between metabolic rate and mass or, given relaxed assumptions, isometric scaling between metabolic rate and mass. Savage *et al.* (2008) concluded that although the mathematics underlying the original model derivation are consistent, they rely on unstated assumptions and predict ¾ scaling in the asymptotic limit of an 'infinite' network. Savage *et al.* (2008) find that finite size corrections for realistic sized mammals yield a theoretical prediction of approximately 0.81 for the scaling of metabolic rate with mass. What should we make of these efforts and conflicting claims? The acceptance of proofs is generally the result of thorough examination by the research community at large. Importantly, such a process has occurred for the West *et al.* (1997) theory over the past 10+ years, yet the community at large has not reached a consensus as to whether the theory is or is not logically consistent. Instead of parsing the intent of the original formulation of the theory, we propose the following consensus summary.

Summary

All three re-evaluations demonstrate that a Lagrange optimisation scheme for minimising energy loss utilising pipe flow resistance (i.e. Poiseuille or dissipative) leads to the scaling of $B \sim M$ with a logarithmic correction in mass. Furthermore, it is not currently known how to construct a well-posed Lagrange optimisation scheme for globally minimising energy loss for pulsatile flow resistance for the whole network (see the appendices of Dodds *et al.* 2001 and Apol *et al.* 2008). Instead, if most of the energy loss in distributing resources within a pulsatile flow network is due to wave reflections at junctions, then principles of impedance matching can be used to derive the scaling of vessel radii, leading to area-preserving branching. Given area-preserving branching within a fractal network and additional assumptions on the scaling of vessel length, it is possible to derive the relationship $B \sim M^{3/4}$ in the limit of infinite body and network size and ignoring all other forms of energy loss for these large vessels such as turbulence or blockages (Etienne *et al.* 2006; Savage *et al.* 2008).

Evaluating Model B derivation: the ¾ allometric scaling for plants

The original derivation of ¾ scaling of metabolic rate in plants is strictly based on geometric and mechanical constraints for the external branching network (West *et al.* 1999). In the WBE theory for plants, the imposition of hydrodynamic optimisation through natural selection within the internal conduit network is only used to predict a scaling law for conduit tapering that is theorised to have evolved to minimise hydrodynamic resistance along flow paths (see Intermediate Tests – Explicit Predictions).

Summary

Using the simplifying assumptions detailed in Table 1, one can successfully derive the prediction that the number of petioles in a plant

scales with mass to the ¾ power, again in the limit of an infinitely sized plant (see Savage *et al.* 2008; Price *et al.* 2010), thus we regard the derivation of the plant model as internally consistent. The rationale for the scaling follows the same logic as the original West *et al.* (1997) derivation for cardiovascular systems. Hence, given area-preserving and volume filling of external plant branches, there should be a predicted ¾ relationship between the number of terminal units and individual size. However, here, the scaling of the number of petioles with plant mass is not the result of an optimisation principle for plant hydraulics, but rather optimisation for collecting homogeneous resources (volume filling) and for biomechanical stability (area-preserving branching, McMahon & Kronauer 1976).

Evaluating Model C derivation: the Boltzmann–Arrhenius temperature dependence

Building from assumptions C1–C4 (Table 1), Gillooly *et al.* (2001) arrived at an equation for the mass-temperature dependence of metabolic rate, $B = B_0 M^{3/4} e^{-E/kT}$ (see eqn 1) that includes a ¾ scaling dependence on mass and a Boltzmann–Arrhenius dependence on temperature. Note that if temperature varies or activation energies differ, then this relationship must be viewed as an approximation because of the well-known problem of averaging nonlinear functions:

$$\left\langle e^{-E_i/(kT)} \right\rangle \neq e^{-\langle E_i \rangle / k \langle T \rangle} \quad (2)$$

where < > denotes the average of a quantity.

Summary

We regard the temperature component of the MTE derivation as internally consistent, with the caveat that efforts to approximate metabolic rate in terms of a single energy of activation within and across species will not capture all of the variability in the scaling relationships. For example, even if the *average* activation energy remains the same between species of different sizes, systematic differences in the *distribution* of energies of activation across species can lead to deviations from predictions. Moreover, metabolic rate is an integrative process, and mechanistic models of the relationship between biological rates and temperature (e.g. photosynthesis in C3 plants; Farquhar *et al.* 1980) do not necessarily yield a strict Boltzmann–Arrhenius dependence on temperature.

Level 2: evaluating MTE's simplifying assumptions

The measurements required to evaluate many of MTE's assumptions involve determining the dimensions and properties of physical networks and rates of fluid flow and oxygen exchange. However, in some cases, the scope of measurement necessary has precluded extensive tests, for example, capillaries in a mammal can number in the billions. Here, we describe efforts to evaluate the biological validity of different assumptions in the MTE theory, utilising the same notation for assumptions for Model A (**A1–A9**), Model B (**B1–B9**) and Model C (**C1–C4**) (Table 1).

Evaluating Model A assumptions: allometric scaling in mammals

The central assumption (**A1**) that forms the core of the evolutionary optimisation argument underlying the WBE model is that natural selection has acted to shape the structure and fluid dynamics of distribution networks leading to minimisation of energy expenditure (**A6**) (West *et al.* 1997). For example, mammals have a direct energetic cost for pumping blood from the heart, so minimising this required energy allows more available energy for other activities important to fitness.

West, Brown and Enquist assumes that vascular trees are hierarchical (**A2**), which is universally acknowledged as valid across most levels within mammals. Furthermore, WBE assumes that vessels within the same level of the arterial tree are identical (**A3**), with the same number of new daughter vessels stemming from each parent vessel (**A4**). Further, the length of vessels should decrease in such a way that the network is volume filling at each level of the hierarchy (**A5**). Explicitly, the 'volume filling' assumption means that $N_k l_k^3 = N_{k+1} l_{k+1}^3$, where k and $k + 1$ denote levels in the hierarchy, N_k and N_{k+1} are the number of vessels in each level and l_k and l_{k+1} are the lengths of vessels in each level. Evaluating the geometry of conduits and branches at the whole network level within and across species can be quite challenging empirically. Moreover, actual cardiovascular networks in mammals are not simple hierarchies but rather mixed hierarchies, with larger vessels possessing 'side-branching' vessels at a range of levels (Tokunaga 1984; Kassab *et al.* 1994). Side-branching does not necessarily invalidate the results of a purely hierarchical fractal-branching model provided the branches retain the same self-similar structure of the main branch (Turcotte *et al.* 1998).

Analysis of biological network structure data is limited. First, most current published reports on branching networks do not report the variability in conduit dimensions within a given level of a branching hierarchy. Hence, assumption (**A3**) remains largely untested and warrants follow-up study. Next, the average branching ratio is assumed to be constant and independent of the branching level (**A4**). In reality, the average branching ratio can exhibit considerable variability and is also confounded by side-branching (Kassab *et al.* 1993; Jiang *et al.* 1994). More recently, a compilation of network data (Huo & Kassab 2012) included summary network statistics using a Horton–Strahler ordering scheme (Horton 1945; Strahler 1957). The length ratio of vessels (**A5**) is shown to deviate significantly from volume filling in an analysis of human, pig, dog, cat and rat vascular networks (Huo & Kassab 2012).

Model A assumes that the only site of transfer of metabolites from network to tissues is across membranes of the terminal units, for example, capillaries in mammals (**A8**). These terminal units are assumed to be invariant in their size and physical properties (**A7**). This requirement is not that exchange surfaces be exactly the same in organisms of different sizes, but rather that their properties be statistically invariant with respect to organism size. For example, for mammals this would imply that the size of capillaries and their biomechanical properties do not systematically change going from mice to elephants. Such invariance is assumed to be both geometric, that is, physical dimensions, and functional, that is, mechanical, dynamical and/or bio-energetic properties. Data compilations for mammals, however, suggest a systematic increase in capillary dimensions with mammal size, albeit weakly, i.e., with a scaling exponent of approximately 1/12 (Dawson 2001, 2003). Finally, the network must have a very large number of bifurcations for the predicted ¾ scaling to hold (**A9**), a limitation recognised in the original publication and one which has been shown, theoretically, to lead to different scaling exponents in cases where all other assumptions are met for a finite size network (Savage *et al.* 2008).

Summary

We conclude that while some of model A's assumptions are consistent with real vascular networks, the empirical data suggest that mammalian vascular networks by and large do not conform to the strict assumptions of the model. It remains to be determined what an 'average' mammalian vascular network looks like, and if the geometry of that network changes systematically with mammal size (in ways other than those already mentioned).

Evaluating Model B assumptions: allometric scaling for plants

Model B assumes that conduit lengths increase from terminal units towards the trunk in such a way that 'space filling' is preserved at each order of the network (West *et al.* 1999). Conventional definitions of volume (or space) filling imply that points within the volume are embedded within a 3D geometric space, within a constrained distance of one another and/or some source point. However here, as above, volume filling (**B3**) means that the sum of the service volumes with radius equal to the length of conduits will be constant for all conduits of a given order, specifically $N_k l_k^3 = N_{k+1} l_{k+1}^3$, without explicit consideration of the space in which the conduits are embedded. In practice, this requirement implies a particular form of change for conduit lengths after each bifurcation, for example, the ratio of daughter to parent branch lengths is $l_{k+1}/l_k \sim 0.794$ for $n = 2$. Again, there is no consideration of side-branching, so it is very difficult to evaluate this assumption in practice.

The assumption of elastic similarity (**B4**) stems from the model of McMahon (1973), McMahon & Kronauer (1976) and is of importance in many of the explicit predictions in plants as it is critical in deriving the scaling of heartwood and sapwood fractions, etc. For plants, the assumption of area-preserving branching was based on collection of the scaling of limb radii (e.g. Horn 2000). The interpretation of assumption **B7** is that the photosynthetic properties of leaves of small plants and shrubs are statistically equivalent to the leaves of large trees. Assumption **B8** implies that plants have evolved to minimise resistance to water flow through the xylem network, leading to the prediction that whole-plant resistance does not scale with plant size, which we consider further below (Explicit Prediction Model B, Vessel Tapering).

Some of Model B's assumptions have been shown to be empirically incorrect. With few exceptions, canopy branches rarely bifurcate symmetrically (**B1**); elastic-self-similarity rarely holds true across all levels of branching architecture (**B4**) (Niklas 1992, 1994a, 1995; Swenson & Enquist 2008); the material properties of stems (e.g. bulk density) differ as a function of stem size and location within plant canopies (**B5**); the majority of woody stems taper along the lengths of individual stems (**B6**); and conduits that function solely in water transport but not in mechanical support of the plant are consistent with Murray's law (McCulloh *et al.* 2003, 2004). Regarding **B7**, data for plants are sparse and conflicting. Analyses within specific genera (*Quercus*) suggest an allometric relationship between leaf size and leaf xylem dimensions (Coomes *et al.* 2008), while additional work on a broad spectrum of leaf networks suggests that many geometric properties of leaf networks are invariant with leaf size (Price *et al.* 2011). The number of xylem elements is known to vary throughout the plant (**B9**). Recent work that incorporates variable conduit number on theoretical predictions makes a number of alternative scaling predictions, for example, predicting that vessels taper more quickly than as predicted in the original WBE model for plants (Savage *et al.* 2010).

Summary

Empirical data provide limited support for the assumptions of Model B. That said, the model is an admittedly coarse-grained theory and does not attempt to capture the observed variability in all of these plant traits. Therefore, the degree to which these deviations change model predictions needs to be quantified across taxa and habitats. Future efforts to quantify the magnitude of variability in these traits and their influence, or lack thereof, on macroscopic scaling properties will therefore be important irrespective of its bearing on MTE.

Evaluating Model C assumptions: temperature dependence of metabolic rate

The assumption that metabolic expenditures scale with oxygen supply (**C1**) is an alternative way of stating that metabolic rate scales with the number and surface area of invariant terminal units (Gillooly *et al.* 2001). With respect to the assumption that oxygen or carbon dioxide supply only occurs at terminal units (**C2**), the transmural transfer of oxygen does occur exclusively in the capillaries, so for mammals this seems a reasonable assumption. Similarly, in plants with non-photosynthetic stem tissue, this seems a reasonable assumption. However, a large number of plants (herbs, succulents, etc.) have photosynthetic stem tissue. In this case, if the photosynthetic surface area scales linearly with the number of terminal units, all of the scaling relationships will still hold. This may be valid because stem surface area is predicted to scale as $M^{3/4}$, and similar to the number of terminal units (Price & Enquist 2006).

The Boltzmann temperature dependence assumed by Gillooly *et al.* (2001) (**C3**) implies that the natural logarithm of metabolic rate varies linearly and negatively with inverse absolute temperature (usually referred to as an Arrhenius plot). This relationship has a physical basis in reaction kinetics, where a Boltzmann term captures the change with temperature in the probability that a molecule exceeds a threshold kinetic energy and thus participates in the reaction. It thus affords a first-order, albeit approximate, description of the thermal behaviour of reaction rates of simple molecules in dilute aqueous solution. The cell is of course a very different environment, being highly concentrated and structurally partitioned with complex membrane structures. Furthermore, the interaction between enzymes and their substrates or cofactors is complex; capturing this full complexity requires sophisticated kinetic models (see Farquhar *et al.* 1980 for one example of how temperature affects photosynthetic rates in C3 plants). All of this implies that a simple Boltzmann correction is likely to be a simplification of temperature sensitivity of whole-organism metabolic rate. The key question is whether this simplification is valid or misses important processes.

The consumption of oxygen, which is how biologists usually measure metabolic rate, is essentially a measure of the electron flow needed to maintain the proton motive force across the mitochondrial inner membrane. So it could be argued that despite the complexity of the cell, metabolic rate can be regarded, to a first approximation, as either electron transport activity or Adenosine triphosphate (ATP) synthase activity. If either of these processes has a single rate-determining step, then the thermal behaviour of this step would dictate the temperature sensitivity of overall metabolic rate

(Gillooly *et al.* 2001). However, note that the assumption of an exponential dependence of reaction rates is violated within plants, where photosynthesis includes components which have a Boltzmann dependence on temperature but when convoluted yield a more complex relationship (Farquhar *et al.* 1980).

To test the generality of assumption **C4**, it is necessary to measure the mean and distribution of activation energies across metabolic reactions within an organism and across species (Dell *et al.* 2011). On the one hand, if metabolic reactions all occur in series, a single rate-limiting step and activation energy must drive metabolic rate (Savage & West 2006). On the other hand, if metabolic reactions occur in parallel, the measured activation energy will represent an average over biochemical reactions, many of which are shared across taxa (Savage & West 2006).

In reality, organisms have biochemical reactions that occur both in series and in parallel (and that include feedbacks) such that the activation energy for metabolic rate must represent an average over some subset of metabolic reactions. If activation energies of different biochemical reactions differ by physiological processes across species, this can create differing temperature responses. Moreover, variability in the temperature response across species can be partly measured by the higher order moments (e.g. variance or skewness) of the overall distribution of activation energies across species. Recent analysis reveals a systematic right skew in the distribution of activation energies, and thus that the median is systematically lower than the mean (Dell *et al.* 2011).

Clarke (2004) and Clarke & Fraser (2004) have argued that temperature does not drive metabolism directly and mechanistically through a single rate-limiting step. They argue that the rate of oxygen utilisation is not source driven by temperature but instead sink driven by the demand for ATP. Rather, they posit that the cell has a series of feedback controls that regulate the supply of electrons to the electron transport chain, and also there are higher level whole-organism controls on metabolic rate. From this perspective, when temperature changes, the rates of the various processes comprising metabolic rate (protein turnover, membrane turnover, ion pumps and so on) change, and this changes the requirement for ATP (Clarke 2004; Clarke & Fraser 2004; Savage & West 2006).

Summary

The Boltzmann–Arrhenius model matches empirical data for how biological rates increase with temperature up to some peak temperature, T_{pk}. The mean activation energy is around 0.6–0.7 eV, but there is significant variation around this mean with biologically meaningful interpretation, such as the thermal life-dinner principle (Dell *et al.* 2011). Ignoring the effects of enzymes and averages across aggregate reactions may be reasonable when looking over large temperature ranges (> 10 °C) where the exponential effects of Boltzmann–Arrhenius dynamics would dominate. Over narrower ranges of temperature, however, these other effects may be of similar magnitude to the Boltzmann–Arrhenius function and thus be important to include. Developing those models and introducing additional assumptions is an important area of future research. Investigating the mechanisms and assumptions behind variation in activation energies is also an important future direction. Finally, it may also be important to extend MTE to include Ratkowsky *et al.* (2005) or Johnson & Lewin (1946) type models that describe the decline of biological rates at high temperatures.

Level 3: evaluating MTE's explicit predictions

Explicit prediction Model A, area-preserving branching

West *et al.* (1997) predicts that area-preserving branching dominates the network, transitioning to area-increasing branching (Murray's law) at a fixed number of levels before the terminal units are reached. For a branching ratio of $n = 2$, the location of this transition has been approximated to occur for conduits of approximately 1 *mm* in diameter for mammalian systems. However, to pinpoint the exact nature and location of this transition requires a detailed hydrodynamic calculation that likely requires numerical simulations. In a strictly symmetrical hierarchical tree, this results in a mathematical relationship between the dimensions of branches before and after a bifurcation event such that in a bifurcating tree, the ratio of parent to daughter branch radius is area-preserving, $r_k/r_{k+1} \approx \sqrt{2}$ ('square law'). As fluid approaches the sites of exchange to drive metabolism, this value should switch to a specific type of area-increasing branching known as Murray's law (Murray 1926). More generally, area preserving requires the following: $N_i r_i^2 = N_j r_j^2$, while Murray's law requires $N_i r_i^3 = N_j r_j^3$, where r_i and r_j represent the radii of vessels at level i and j of the network respectively. Huo & Kassab (2012) examined data on the ratio of daughter to parent branch radius from around 20 animal studies including pigs, rats and mice. In mammalian vascular systems with many branch orders (generations), they found support for the squared-law to cubed-law transition predicted by WBE. However, the agreement in lower order systems was weaker.

Summary

There is support for the trend of a transition from squared-law to cubed-law diameter scaling predicted by WBE in vascular trees with large numbers of branching generations.

Explicit prediction Models A and B, metabolic rate scaling

The scaling of metabolic rate with body mass has been a subject of considerable interest (Kleiber 1932; Hemmingsen 1950). A full review of the literature on how well the MTE prediction is supported by data is beyond the scope of this review (see e.g., Glazier 2005, 2010). A few issues are worth considering, however, in any attempt to derive a general model that applies across taxa. For example, the empirical data from several clades including mammals (Dodds *et al.* 2001; Clarke *et al.* 2010; Kolokotrones *et al.* 2010), plants (Reich *et al.* 2006; Mori *et al.* 2010) and insects (Chown *et al.* 2007) indicate nonlinearity of the log–log relationship. Moreover, the curvature differs depending on taxonomic group, convex in mammals with small mammals exhibiting higher metabolic rates than predicted, and concave in small insects and plants, with data indicating lower rates than predicted. In addition, there is considerable debate as to the value of fitted slopes in empirical size-metabolism data, with some studies finding values closer to 2/3 (Dodds *et al.* 2001; White & Seymour 2003) and some finding values closer to ¾ (Savage *et al.* 2004). These differences can be explained, in part, by the curvilinearity of the scaling relationship and the body mass range of the data (Dodds *et al.* 2001; Kozlowski & Konarzewski 2005; Clarke *et al.* 2010; Kolokotrones *et al.* 2010).

Summary

The empirical data indicate that pure ¾ scaling does not hold across the full size range for mammals, plants or insects, but that it is a

reasonably accurate approximation across certain size ranges, especially for organisms of very large size.

Explicit prediction Model B, gross morphology of plants

Given the assumptions of local branching invariants (including elastic similarity), one can derive predictions for the scaling of gross morphological characteristics such as the allometric interdependence of height, diameter (e.g. plant stem), surface area and mass (West *et al.* 1999).

A wealth of empirical data and several reviews of this area have been published (Niklas 1994a, 1995; Henry & Aarssen 1999; Price *et al.* 2007, 2009), which indicates that plant morphological allometry is highly variable, and influenced by factors such as growth form, functional group, competition, sex and nutrient availability. Recent analyses suggest that the central tendencies of scaling exponents for morphological relationships across a range of taxa do not coincide with the predictions of Model B (Price *et al.* 2009). Instead, the variation in morphological scaling are better described by a more relaxed model in which network geometry remains fractal, but is not constrained to take on particular universal values (Price *et al.* 2007). Moreover, comparison of scaling models, utilising a hierarchical Bayesian framework, shows that there exists statistical support for species-specific parameterisations of morphology, even when accounting for added model complexity (Price *et al.* 2009).

Summary

Empirical data do not support the predictions of universal morphological scaling. There is evidence, instead, of allometric covariation in which scaling exponents for plant morphology covary systematically together (Price *et al.* 2007; Price & Weitz 2012). The mechanisms underlying allometric covariation represent an important target for future research. For example, direct assessment of scaling ratios for radii and length at the branch level should provide stronger tests of connections among gross morphological features.

Explicit prediction Model B, vessel tapering

The WBE model of plants predicts that conduit radii should increase in cross-sectional radius moving from petiole to trunk. The increase in conduit radii had long been observed and was described, nearly 100 years ago, as Sanio's laws (Bailey & Shepard 1915). However, WBE argued that to equalise hydraulic resistance across paths, the increase in cross-sectional radius should be a power law. The lower bound of the scaling exponent of tapering profiles was then derived, with a prediction that plants should evolve conduit tapering profiles that approach this lower bound.

Summary

The empirical examination of such tapering exponents from tip-to-trunk profiles of trees have been shown to be in qualitative agreement with theory (Weitz *et al.* 2006; Mencuccini & Holtta 2007; Coomes *et al.* 2008; Savage *et al.* 2010). That is, tapering profiles can be well approximated by a power law of distance from petiole (or of branch). However, there is no evidence of a single universal tapering exponent (e.g. see Mencuccini & Holtta 2007), and recent theory predicts the value of the exponent more accurately than the original model (Savage *et al.* 2010).

Explicit prediction Model C, Temperature dependence of metabolic rate

Measurements of the thermal dependence of whole-organism metabolism (typically resting metabolism or basal metabolic rate, BMR) have shown that the temperature sensitivity of BMR, both within and across species, can be approximately described by a Boltzmann relationship with a mean activation energy in the range of 0.6–0.7 eV. However, these data are also frequently well approximated by a power law (typically linear) or Q10 relationship (Clarke & Johnston 1999). A recent analysis finds that across a huge diversity of data, the Boltzmann model provides the best statistical description of these alternatives, but also that several alternative models also provide good fits for most temperature responses (Dell *et al.* 2011). Consequently, the choice of which functional form to use depends on the particular system and temperature range being studied as well as on the conventions within that specific field.

Summary

The MTE relationship, which is based on the Boltzmann model, has a biochemical basis and matches empirical data as well, or better than the proposed alternatives of a power law or Q10 relationship.

Level 4: evaluating MTE's extended predictions

In recent years, the domain of MTE has been extended considerably by combining MTE with other theoretical frameworks designed to address questions beyond its original domain of organismal biology. Some of these extensions use allometric predictions (i.e. eqn 1) to parameterise models, while others extend the domain of MTE considerably. Because these extensions touch on so many areas of biology, and because many of them are recent, they have not been well evaluated by the community at large. Therefore, in the Supplementary Information, we constrain our discussion to a few core areas that have received considerable attention: ontogenetic growth, tree size–abundance distributions and biodiversity gradients.

DISCUSSION

> '… all models are wrong, but some are useful.' - George E.P. Box

Many of the key ideas currently underlying MTE have a long history. The idea that the amount of surface area available for thermal exchange might constrain metabolic rate dates at least to Rubner (1883). The links between fractal geometry, scale invariance and natural design owes much to the insights of D'Arcy Thompson (1917) and Mandelbrot (1977). The use of a simplified pipe model to describe the scaling properties of plants is almost 50 years old (Shinozaki *et al.* 1964). The use of normalisation by mass to uncover life-history traits was extensively explored in a series of seminal works on the importance of scale in biology during the 1980s (Peters 1983; Calder 1984; Schmidt-Nielsen 1984). Work on the relationship between size, metabolism and a suite of allometric traits in plants was advanced by Niklas (1994b,a). Temperature has long been known to influence metabolism at multiple scales of ecological organisation from individuals to ecosystems (Rosenzweig 1968).

The insight of MTE was to build on these early foundations and to propose a unified theoretical framework, with roots in the theory of evolution by natural selection as well as physical principles. The promise of MTE was that a model with relatively few parameters, that are also biologically intuitive, could explain a substantial amount of variability in biological rates and states. Has this promise come to fruition? At the very least, MTE has served to energise the field and to refocus efforts on the use of biological scaling as a theoretical and empirical methodology. At the most, it provides a coarse-grained theory for the origin of metabolic scaling phenomena across disparate taxa, the impacts of which are potentially far reaching as evidenced by the numerous extensions that have been developed thus far. For example, MTE has recently been combined with information theory (Harte *et al.* 2008), life-history theory (Charnov & Gillooly 2004; Brown & Sibly 2006), the neutral theory of biodiversity (O'Dwyer *et al.* 2009), resource limitation models (Niklas *et al.* 2005; Lichstein *et al.* 2007; Allen & Gillooly 2009; Elser *et al.* 2010; Hammond & Niklas 2012), Kimura's and Hubbell's neutral theory (Allen *et al.* 2006; Stegen *et al.* 2009), food web theory (Gillooly *et al.* 2006); predator–prey models (Vasseur & McCann 2005; Brose *et al.* 2006; Weitz & Levin 2006), and models of forest structure and dynamics (Enquist *et al.* 2009; West *et al.* 2009) to yield predictions on a suite of additional processes ranging from molecular evolution to food web structure.

Although these extensions are exciting, several lines of evidence suggest that some may reach beyond the foundations on which they rest, and represent, in some cases, new bodies of theory rather than confirmations of MTE. First and foremost, greater efforts have been expended in testing the predictions of MTE than in rigorous examination of its basic assumptions and structure. The available evidence indicates that many of the core MTE predictions, such as ¾ scaling of metabolic rate with mass, are not universal as previously believed and considerable variation across mammals and plants in network geometry remains unexplained. The reasons for these differences in predictions may have, at their root, the fact that structural and physiological assumptions of MTE differ from those in the biological system of interest. Moreover, some principles likely need to be modified or added to accurately capture the primary drivers behind the evolution of vascular networks and organismal metabolism.

An attractive aspect and strength of MTE is the number of predictions it makes at so many levels of organisation, a rarity in ecology. Indeed while we have highlighted ways in which MTE has fallen short in making accurate predictions, we also emphasise the breadth of areas within which the theory comes close. With this in mind, we advocate the use of tests that examine multiple predictions within or across levels of organisation simultaneously when comparing models. Such tests are more informative than tests of a single prediction or set of predictions that are expected to covary. Moreover, as the theory is coarse grained, it is intended to describe the central tendency across many orders of magnitude. Tests examining the scaling of form or function within a single, or handful of species, while certainly helpful in facilitating meta-analyses, cannot alone validate or invalidate them (although some extensions, such as ontogenetic growth, require them, e.g. Sears *et al.* 2012), but can add support, help identify deviations and be used along with tests of assumptions to bolster or revise models. Variability within certain taxonomic or functional groups that depart from model assumptions has long been acknowledged by the primary authors (West *et al.* 1999; Enquist *et al.* 2000). What is currently being determined through numerous studies appearing in the literature is whether these groups represent the exception, or the rule, or whether modifications of the theory can account for them.

There are those who view empirical data on biological rates and physical dimensions as, at least partially, a means to test whether MTE is correct. When alternative theories exist, models and predictions should be compared to see which gives the most accurate and robust results. Moreover, it is often argued that MTE provides a baseline or null model, analogous to physical models, such as ideal gas laws, which rarely are completely accurate for real systems, but are insightful and important nevertheless. For skeptics, exceptions to MTE for even single species or single biological scaling relationships can be taken as evidence that the entire formalism underlying MTE is faulty. A more constructive interpretation of this failure is that MTE's assumptions are not met in some cases of interest, which means that the theory is limited in scope rather than wholly invalid, or that it is 'right for the wrong reasons' – that the basic and useful ¾ power laws stem from different processes and dynamics than those assumed by MTE. The question of importance then becomes a question of determining the limits of the theory. However, there are those who consider the theoretical foundations either too technical or irrelevant to the question of whether predictions (based on empirical laws) can be used, in practice, to simplify and explain seemingly complex patterns in ecology. For them, the internal consistency of the theory has no bearing so long as predictions provide some information gain over alternative theories (which in many cases do not exist).

We hope that this article serves to clarify and contextualise the aims and expectations of both groups by suggesting best practices to evaluate when MTE can or cannot be used to further our understanding of the complex ecological world around us, and to spur further research on MTE's basic mechanisms and assumptions, not only on MTE's predictions.

Summary

We argue that there does not yet exist a complete, universal and causal theory that builds from network geometry and energy minimisation to individual, species, community, ecosystem and global-level patterns. Whilst all models are necessarily incomplete approximations of reality, we believe the time is ripe for a new wave of empirical tests and the development of theories that emphasise the central role of body size, metabolism and temperature as highlighted by MTE and others (Peters 1983; Calder 1984; Schmidt-Nielsen 1984; Kooijman 1993; Niklas 1994a; Brown & West 2000).

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AUTHORSHIP

CAP, JSW and VMS conceived the study. All authors contributed to the writing of the manuscript.

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