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The Origin of Universal Scaling Laws in Biology

Geoffrey B. West [1]

Theoretical Division, T-8, MS B285, Los Alamos National Laboratory, Los Alamos, NM 87545, USA.

1 Introduction

It is typical of most complex systems that the only quantitative statements that can be made analytically are those relating to their scaling properties which are usually non-trivial. Life, and, in particular, its amazing diversity spanning more than 21 orders of magnitude in size, is the most complex physical system in the universe. In spite of this, biological systems obey a host of remarkably simple and systematic empirical scaling laws which relate how organismal features change with size over many orders of magnitude[1]. These include fundamental quantities like metabolic rate (the rate at which energy must be supplied in order to sustain an organism), time scales (such as lifespan and heart rate) and sizes (such as length of the aorta or height of a tree trunk). It is a remarkable fact that all of these can be expressed as power law relationships with exponents that are simple multiples of $\frac{1}{4}$ (e.g. $\frac{1}{4}$, $\frac{3}{4}$, $\frac{3}{8}$). They appear to be valid for almost all forms of life, whether it be mammalian, avian, reptilian, unicellular or plant-like. Clearly the universal character of these "laws" is telling us something important about the way life is organized and the constraints under which it has evolved. The origin of these so-called allometric scaling relationships (a term coined by Huxley) and, in particular, why the exponents are always simple multiples of $\frac{1}{4}$, have been longstanding fundamental problems in biology.

These relationships are usually expressed as power laws: $Y = Y_0 M^b$, where Y is some biological observable, Y_0 a normalization, M the mass of the organism and b a scaling exponent. Some specific examples are heart-rate ($b = \frac{1}{4}$), lifespan ($b = -\frac{1}{4}$), and the radius ($b = \frac{3}{8}$) and length ($b = \frac{1}{4}$) of *both* aortae and tree trunks. One of the best-known and fundamental of these, first detailed by Kleiber in 1932, is for basal metabolic rate. For warm-blooded organisms spanning six orders of magnitude in mass, $b = 0.75 \pm 0.1$. When extended to cold-blooded and unicellular organisms and even to plants, the same $\frac{3}{4}$-power dependence is manifested though the normalization may vary. As illustrated in Fig. 1 this has recently been extended down to the intra-cellular and molecular levels within mitochondria[3]. Thus the 3/4-power law extends over almost 27 orders of magnitude ranging from the largest mammal down to the molecular complex catalyzing metabolism at the most fundamental level!

[1] gbw@pion.lanl.gov

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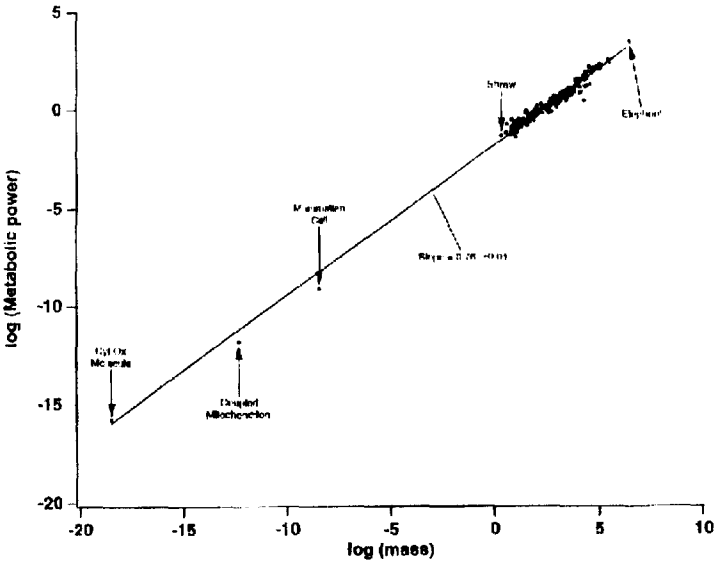

Fig. 1. Metabolic rate (in watts) as a function of mass (in Kg); the scale is logarithmic (base 10) and exemplifies the 3/4-power scaling law discovered by Kleiber. The straight line is the best fit to the metabolic rate of 289 mammals shown as a function of their mass covering 6 orders of magnitude; it has a slope of 0.76 ± 0.01. When extrapolated back a further 20 orders of magnitude it agrees well with data on a single isolated mammalian cell, its isolated mitochondrian, respiratory complex and cytochrome oxidase molecule.

Kleiber's law implies that the power required to sustain unit mass of an organism, *decreases* with size. Thus, to support one gram of a mouse requires three times the power needed for a dog and nine times that for an elephant! In this sense it is clearly more efficient to be large. It is instructive to compare this with mechanical engines, which do indeed scale isometrically. For example, over nearly six orders of magnitude the power output of internal combustion engines scales linearly with mass ($b = 1$) while their revolution rate scales as $M^{\frac{1}{3}}$. The reason why $b = \frac{3}{4}$ for biological systems rather than $b = 1$, reflecting the fact that mammals are built from essentially the same "fundamental" cellular building blocks, or a naive surface-to-volume relationship, $b = \frac{2}{3}$, has been sought for decades.

One of the most intriguing consequences of these scaling laws is the emergence of invariant quantities governing longevity[1]. For many organisms lifespan increases like $M^{\frac{1}{4}}$ whereas heart-rate decreases like $M^{-\frac{1}{4}}$, so, for example, the number of heart-beats in a life-time is the same for all mammals (about 1.5×10^9). At the molecular level this implies that the number of turnovers of the molecular respiratory complex per cell during a typical lifetime is invariant ($\approx 10^{16}$)[3]. Similarly, since specific metabolic rate also decreases like $M^{-\frac{1}{4}}$, the total energy needed to support a given mass of an organism during its life-time is also the same for all mammals (about 300KCals/gm). So, large organisms live slower and longer. Understanding these scaling laws and, in particular, the magnitude of the invariant quantities would provide significant insight into the origin of

aging and mortality. As a first step, however, one needs to understand the scaling laws of metabolism and the sustenance of life.

To summarize: Allometric scaling laws are special because they express a systematic universal simplicity in the most complex of all complex systems; furthermore, they exhibit one of the rare examples of quantitative laws in biology. Their origin and universality present a major challenge and are suggestive of a set of fundamental principles at work.

Recently, we have proposed a general model for the origin of these universal quarter-power scaling laws[4]. It is based on the observation that complex structures are constrained, and ultimately limited, by the rate at which essential resources that sustain them can be supplied. The model accounts, in a well-defined quantitative testable fashion, for the fact that the scaling exponent for almost all biological phenomena is a simple multiple of $\frac{1}{4}$ and, in particular, accounts for the $\frac{3}{4}$-scaling exponent for metabolic rate. It is based on the idea that, to supply the huge number of local fundamental units of an organism (in most cases, the cells), a linear, branching hierarchical transport network is required as manifested, for example, in the circulatory and respiratory systems of mammals or the vascular system of plants and trees. Although designed with these systems in mind, we anticipate that the model should also apply to less well-understood systems such as the intracellular transport system. Indeed, the fact that scaling persists down to the molecular level strongly suggests that the same mechanism and principles that govern organismal scaling are at work inside the cell. It is important, therefore, that such principles are sufficiently general that they are not sensitive to details of specific taxa. We propose the following three basic general principles for the design of network transport systems:

i) In order for the network to supply the entire volume of the organism, a space-filling hierarchical branching pattern is required;
ii) the final branch of the network, where nutrients are exchanged (e.g. the capillary of the circulatory system or the petiole of a tree), is a size-invariant unit; and
iii) organisms have evolved so that the energy required to sustain them is minimized.

Scaling laws arise from the interplay between the physical and geometric constraints implicit in these three principles. Below we show that they imply that these networks must typically be fractal-like structures with self-similar properties dominated by area-preserving branching. Quarter-power scaling then follows even though the various transport systems considered, and the pumps that drive them, have quite different characteristics. A particularly salient feature of these networks is that their hydrodynamic resistance decreases with size, typically like $M^{-3/4}$. This explains why less power is required to sustain a unit mass of a larger animal. Our model leads to a plethora of scaling laws not only *between* organisms (allometry) but also *within* a given organism itself (e.g. it successfully predicts the length of, and speed of blood flow in the aorta relative to that of a capillary). In addition, the model puts potentially severe physical constraints on the structural design of organisms.

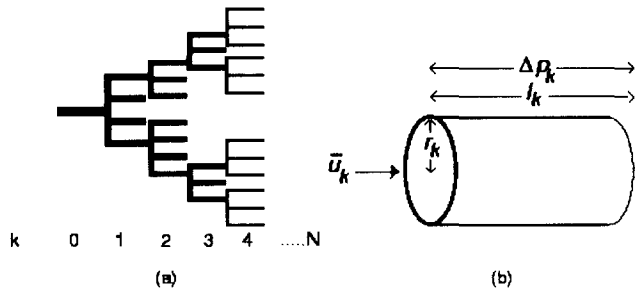

Fig. 2. (a) Topological representation of such networks, where k specifies the order of the level, beginning with the aorta ($k = 0$) and ending with the capillary ($k = N$); (b) Parameters of a typical tube at the kth level.

2 The Model

2.1 General Description and Terminology

The model should be viewed as an idealized representation of typical biological network systems in that we assume, for example, that all vessels have cylindrical symmetry and that turbulence and non-linear effects at junctions do not play a crucial role in the fluid flow. Otherwise, the model is quite realistic, incorporating all important aspects of these systems; it can be used as a point of departure for more detailed analyses and refined versions.

All systems can be described by a branching network in which the sizes of tubes regularly decrease (Fig. 2). A familiar example is exhibited by the vertebrate circulatory and respiratory systems. Although all of the systems considered and the fluids in them are physically quite different, we shall show that they give rise to essentially the same scaling laws. To be specific we shall concentrate on the cardiovascular system, later briefly discussing the extension to other specific systems.

The network is composed of N branchings arranged hierarchically beginning with the aorta (level 0) and ending with the capillaries (level N). The length of a kth level branch will be denoted by l_k, its radius by r_k, and the pressure drop across it by Δp_k. The volume rate of flow is $\dot{Q}_k = \pi r_k^2 \bar{u}_k$ where $\bar{u}_k$ is the velocity averaged over the cross-section and, if necessary, over time. Since fluid is conserved as it flows through the network

$$\dot{Q}_0 = N_k \dot{Q}_k = N_k \pi r_k^2 \bar{u}_k = N_N \pi r_N^2 \bar{u}_N \tag{1}$$

which holds for any k. Since oxygen and/or nutrients are transported by the fluid for metabolism, $\dot{Q}_0 \propto B$. Now, from (ii) above, the terminal units (capillaries) are invariant, i.e. r_N, l_N, and $\bar{u}_N$ are independent of M. Thus, if $B \propto M^a$ then $\dot{Q}_0 \propto M^a$. Eq. (1) then predicts $N_N \propto M^a$. Thus, if $a = 3/4$, this implies that the number of cells serviced by a single capillary increases with M indicative of the increased efficiency of larger systems.

In order to describe the network we need to determine how the branching ratios and radii and lengths of the tubes change throughout the network. To do so introduce scale factors

$\beta_k \equiv r_{k+1}/r_k$, $\gamma_k \equiv l_{k+1}/l_k$ and $n_k = N_{k+1}/N_k$. Below we show that space-filling (our first principle) requires $\gamma_k = \gamma$, independent of k, and that minimization of energy (our third principle) leads to both $\beta_k = \beta$ and $n_k = n$, independent of k. An important exception to the latter is a crucial modification for pulsatile systems, which will be discussed below. If β_k, γ_k, and n_k are all independent of k, then the network is a conventional self-similar fractal. Before proving these it is worth noting that, with $n_k = n$, the number of branches increases geometrically from level 0 to level N, ($N_k = n^k$). Furthermore, since $N_N = n^N$, $N \propto \ln M$. Thus, in going from a mouse to a whale, an increase in mass by a factor of 10^7, the number of branchings from aorta to capillary increases by only about 70%.

2.2 *Space-filling and Volume-preserving Branching*

Our first postulate expresses the notion that a space-filling network is a natural structure for ensuring that all cells are serviced by capillaries. The organism is composed of many groups of cells, "service volumes", v_N, which are supplied by a single capillary. The network must branch so as to reach all such service volumes. The total volume to be filled, or serviced, is given by $V = N_N v_N$. For a network with a large number of branchings, N, complete space-filling implies that this same volume is filled by analogous volumes throughout the network, v_k, defined by branches at any level k. Since $r_k \ll l_k$, $v_k \propto l_k^3$, so space-filling constrains only branch lengths, l_k. Complete space-filling implies that the volume filled, or serviced, does not depend on the level used to estimate it. Thus, $V \approx N_k v_k \propto N_k l_k^3$, independent of k. We assume its validity throughout the network although it becomes less realistic for small values of k (or N). This leads to $\gamma_k^3 \equiv (l_{k+1}/l_k)^3 \approx N_k/N_{k+1} = 1/n$, so that $\gamma_k \approx n^{-1/3} \equiv \gamma$, independent of k. Note that this can be straightforwardly generalized to d-dimensions giving $\gamma \approx n^{-1/d}$. This result will be taken to be a general property of all systems that we consider.

2.3 *Area-preserving Branching and Self-similar Fractals*

Below we shall show how an analogous result for β_k can be derived from dynamical considerations based on the energy minimization principle. For many systems this leads to area-preserving branching, meaning that the sum of the cross-sectional areas of the daughter branches is equal to that of the parent: $\pi r_k^2 = n\pi r_{k+1}^2$. Thus, $\beta_k \equiv r_{k+1}/r_k = n^{-1/2} \equiv \beta$, independent of k. Proving this is somewhat technical, especially for the circulatory system, so before doing so we shall first explore its consequences and show how it is a key ingredient in deriving $\frac{1}{4}$-power scaling. Once we show that β_k, γ_k, and n_k are all independent of k, then we have proven that the network is a conventional self-similar fractal. This has been tested empirically for plant systems using variants of the "box-counting" method by measuring the length of the boundary of their images at different resolutions. This should be related by a power relationship whose exponent defines the fractal dimension. Although there is some variation in the data depending on details of age and growth conditions, the observations do indeed support a self-similar fractal structure.

2.4 *Derivation of the 3/4-Power Exponent*

To derive allometric relations we need to relate the scaling of vessel size within an organism to its total mass. A natural vehicle for this is the total volume of fluid in the network, V_b. Below we shall show that the network that minimizes energy (our third principle) not only has area-preserving branching but also requires $V_b \propto M$. It is straightforward to derive $V_b \approx V_N(\gamma\beta^2)^{-N}/(1-n\gamma\beta^2)$. Since $V_N \propto M^0$, this gives $(\gamma\beta^2)^{-N} \propto M$. Now recall that, if $B \propto M^a$, then the number of capillaries, $N_N \propto M^a$ leading to

$$a = -\frac{\ln n}{\ln(\gamma\beta^2)} \tag{2}$$

When area-preserving, $\beta = n^{-1/2}$, is combined with space-filling, $\gamma = n^{-1/3}$, this gives $a = 3/4$ (*independent* of the value of the branching ratio, n). Consequently, $B \propto M^{3/4}$.

Many other scaling laws follow. For example, for the aorta, $r_0 = \beta^{-N} r_N = N_N^{1/2} r_N$ and $l_0 = \gamma^{-N} r_N = N_N^{1/3} l_N$, yielding $r_0 \propto M^{3/8}$ and $l_0 \propto M^{1/4}$. Notice that γ and β play a dual scaling role: they not only determine how quantities scale from the aorta to the capillary within an organism, but also how they scale between organisms. Although these allometric scaling results are in good agreement with data there are some problems. The first is that, for humans where $N_N \approx 10^{10}$[7], the above gives $r_0/r_N \approx 10^5$, in disagreement with the observed value of 10^4. The second is more serious: area-preserving branching implies that the fluid velocity remains constant throughout the network, i.e. $u_0 = u_k = u_N$. This may not be serious for plants but would be disastrous (and obviously wrong!) for mammals. Indeed, for the efficient transfer of oxygen and nutrients across the walls of capillaries and into cells, blood, which leaves the heart at over $100cm/sec$, must slow down to less than $1cm/sec$ by the time it reaches the capillaries. These, and other problems, are solved by considering the dynamics of these systems utilizing the energy minimization principle to which we now turn.

3 Minimization; Energy Loss and Impedance

We now examine the dynamics of these systems and, in particular, the consequences of the assumption that biological networks have evolved to minimize energy dissipation. First consider the simpler problem of non-pulsatile flow. For steady laminar flow of a Newtonian fluid, the resistance of a single tube is given by the well-known Poiseuille formula: $R_k = 8\mu l_k/\pi r_k^4$, where μ is the viscosity of the fluid. Ignoring effects such as turbulence and non-linearities at junctions, which are expected to be small, gives for the total resistance of the network, $Z = \sum_{k=0}^{N} R_k/N_k$, from which it follows that $Z \propto N_N^{-1} \propto M^{-a}$ so the total resistance *decreases* with size. It is in this sense that a larger organism is more efficient. This leads to two important scaling laws: blood pressure, $\Delta p = Q_0 Z$, and aortic blood velocity must both be independent of body size, in agreement with data[1,7]. Neither of these depends on detailed knowledge of n, β, or γ or, therefore, a. They are both quite surprising and counter-intuitive. After all, the aorta of a whale has a radius of $30cm$ whereas that of a shrew is barely visible at $0.01cm$, yet both sustain the same pressure and blood velocity!

In spite of these successes we still have the problem that area-preserving implies that blood does not slow down in going from the aorta to the capillary. To explain this we turn to the energy minimization principle. The basic idea is that the trial-and-error feedback implicit in evolutionary adaptation has led to network transport systems that, on the average, minimize the energy required to run them. Thus, to sustain a given metabolic rate in an organism of fixed mass, M, with a given volume of blood, V_b, the cardiac output must be minimized subject to a space-filling geometry. To enforce such a constraint it is natural to use the classic method of Lagrange multipliers. Consider then the cardiac output, $W(r_k, l_k, n_k, M)$, as a function of all relevant variables characterizing the network. We need to minimize the auxiliary function

$$F(r_k, l_k, n_k) = W(r_k, l_k, n_k, M) + \lambda V_b(r_k, l_k, n_k, M) + \sum_{k=0}^{N} \lambda_k N_k l_k^3 + \lambda_M M \tag{3}$$

The constants, λ, λ_k and λ_M are the Lagrange multipliers. Since $B \propto Q_0$ and $W = \dot{Q}_0^2 Z$ this is tantamount to minimizing the total resistance, Z. By demanding $\partial F/\partial l_k = \partial F/\partial r_k = \partial F/\partial n_k = 0$, one obtains $\beta_k = n^{-1/3}$ with $n_k = n$, independent of k. This corresponds to area-*increasing* branching and solves the problem of slowing blood down in the capillaries: Eq. (1) gives $\bar{u}_N/\bar{u}_0 = (n\beta^2)^{-N} = N_N^{-1/3}$. For humans, $N_N \approx 10^{10}$ so $\bar{u}_N/\bar{u}_0 \approx 10^{-3}$, which is in reasonable agreement with data [7]. This result, $\beta_k = n^{-1/3}$ [8], however, does not give $a = 3/4$ when used in Eq. (2). Minimizing F with respect to M (i.e., $\partial F/\partial M = 0$) gives $V_b \propto M$, which is just what is needed to derive Eq. (2). We now show that incorporating pulsatile flow not only solves all of these problems, giving the correct scaling relations (e.g., $a = 3/4$ and $\bar{u}_N/\bar{u}_0 \propto M^0$), but also gives the correct magnitude for $\bar{u}_N/\bar{u}_0$.

A detailed treatment of pulsatile flow is complicated. Here, we present a highly condensed version that contains the essential features needed for the scaling problem. Most importantly, blood vessels are no longer taken to be rigid but are allowed to expand and contract elastically as the pulse wave propagates along them. The classic Poiseuille resistance of the rigid tube, is thereby generalized to a complex impedance, Z, signifying the possibility of attenuated wave propagation[7,9]. Both Z and the dispersion relation that determines the wave velocity, c, are derived by solving the Navier-Stokes equation for the fluid coupled to the Navier equations for the vessel wall by appropriate boundary conditions. In the linearized, incompressible fluid, thin wall approximation, this problem can be solved analytically to give

$$\left(\frac{c}{c_0}\right)^2 \approx -\frac{J_2(i^{3/2}\alpha)}{J_0(i^{3/2}\alpha)} \qquad \text{and} \qquad Z \approx \frac{c_0^2 \rho}{\pi r^2 c} \tag{4}$$

Here $\alpha \equiv (2\pi\nu\rho/\mu)^{1/2} r$ is the dimensionless Womersley number [9] and $c_0 \equiv (Eh/2\rho r)^{1/2}$, the classic Korteweg-Moens velocity; ν is the frequency, ρ the blood density and E the modulus of elasticity for the vessel wall whose thickness is h. In general, both c and Z are complex functions of ν. Let us examine the consequences of these formulae as blood flows through progressively smaller tubes forcing α to decrease. The crucial point is that the character of the wave depends critically on whether $|\alpha|$ is $<$ or $>$ 1 reflecting

the behaviour of the Bessel functions in Eq. (4). In humans, typical values of α range from around 15 in the aorta to 5 in the arteries, 0.04 in the arterioles to about 0.005 in capillaries. Furthermore, since the volume of blood, $V_b \propto M$, we expect the volume of the heart, V_H, to scale likewise. Now, the overall volume flow-rate, $Q_0 = V_H \nu$, so heart-rate, ν, scales as $M^{-1/4}$, in good agreement with data. Consequently, $\alpha \propto M^{1/4}$, so, in the smallest mammals $|\alpha|$ is barely larger than 1 even in their aorta.

(i) For large tubes, where α is large (> 1), Eq. (4) gives $c = c_0$, the classic Korteweg-Moens velocity. Numerically this gives $c \approx 580 cm/sec$ in good agreement with measurements[7]. Since this is purely real, the wave suffers neither attenuation nor dispersion, reflecting the fact that, in this regime, viscosity plays almost no role. The corresponding impedance is given by $Z = \rho c_0/\pi r^2$: its r-dependence has dramatically changed from r^{-4} to r^{-2}. Using this in Eq. (3) to minimize energy loss now leads to an area-preserving law at the junctions, so $\beta_k = n^{-1/2}$. Physically this ensures that when pulse waves traveling in a vessel come to a branch point no energy is reflected back; it is the exact analog of impedance matching at the junctions of electrical transmission lines [7].

(ii) For small tubes where $|\alpha| < 1$ the role of viscosity becomes increasingly important until it eventually dominates the flow; Eq. (4) gives $c \approx \frac{1}{4} i^{1/2} \alpha c_0 \to 0$, in quantitative agreement with observation [7]. Because this has a significant imaginary part, the traveling wave is heavily damped, leaving an almost steady oscillatory flow whose impedance is given by the original Poiseuille formula; i.e., the r^{-4} behaviour is restored! Thus, from our previous argument, for large k, corresponding to small vessels, $\beta_k = n^{-1/3}$: area-preserving is lost and blood is forced to slow down.

Thus β_k is *not* independent of k but rather has a step-like behaviour which is well supported by data for the total cross-sectional area of the vascular bed. Because most of the blood resides in the large vessels whereas most of the resistance is in the small ones this behaviour solves the problem of having blood slow down in the capillaries while maintaining the success of the various scaling laws. Though considerably more involved, the derivation of scaling laws based on β_k, derived using the "exact" expression for Z, Eq. (4), in the minimization constraint, Eq. (3), leads to essentially the same results as before but without the attendant problems. In addition, quantitative agreement with values of lengths and velocities is excellent throughout the network. The crossover from one behaviour of β_k to the other occurs where the wave and Poiseuille impedances are comparable in size. The number of generations where Poisseuille flow dominates turns out to be invariant: roughly 15. For humans, with $n = 3$ (the approximate effective empirical value [7]), Poiseuille flow becomes appreciable after about 7 branchings, whereas in a mouse after only 2-3. Interestingly, a mammal not much smaller than a shrew ($M = 3$g) could not support a pulse wave beyond the aorta! This may well be the the lower limit on the size of a mammal for, if impedances cannot be matched, the energy dissipated is no longer minimal.

3.1 Some Consequences and Extensions

Time scales in the network typically vary like $M^{1/4}$. We have already seen that heart-rate scales this way and it is straightforward to show that blood circulation time does likewise. It is tempting to relate this to life-span, which also scales like $M^{1/4}$. It seems

unlikely that this is a coincidence. It is more likely that dissipation in the network and the subsequent production of entropy plays a critical role in determining lifetime and aging. The seeds of destruction may very well be built into the very system designed to sustain life!

The model can be generalized to organisms living in d-dimensions. The only significant change is that, since the network must now fill a d-dimensional volume, γ generalizes to $\gamma = n^{-1/d}$. Repeating the previous derivation leads, as before, to Eq. (2) from which we obtain $a = 1/(1 + 1/d) = d/(d + 1)$. This shows that the "3" in 3/4 represents the dimensionality of space that most organisms live in; two-dimensional organisms, such as flatworms, might therefore be expected to have $a = 2/3$. Note the "1" in $(d + 1)$ arises from $2 \times 1/2$, the "1/2" coming from the area-preserving exponent in β and the "2" from converting this from radii to cross-sectional areas.

Other analogous network systems can be analyzed in a similar fashion. For example, in the mammalian respiratory system, apart from obtaining many analogous scaling laws, we derive the surprising result that the total surface area of the lung $A_L \propto M^{11/12}$, thereby explaining Weibel's paradox [1], that it scales with a higher exponent $(11/12 \approx 0.92)$ than the 3/4 seemingly needed to supply oxygen.

Plant vascular systems are essentially a sheath of tubes, tightly bundled together as in an electrical cable. Many of them are non-conducting and effectively form the heartwood which gives biomechanical stability [5] to branches, ultimately leading to area-preserving branching. Since hydrodynamic resistance of uniform conducting tubes increases linearly with length, resource supply to apical meristems and forest canopies would be seriously limited; if not circumvented, the evolution of trees and other plant forms would be severely constrained to ground cover. This problem is solved in the model by allowing vessels to have a small uniform taper. Minimizing the vessel resistance determines the magnitude of this taper which turns out to be quite small: for example, over 12 orders of magnitude variation in plant mass, the radius of the vessel in the trunk, or stem, is predicted to change by only about 60%, in agreement with observation. Remarkably, this taper has the consequence that the vessel resistance is *independent of length*, thereby equalizing resource supply to all leaves, especially those on the most distal branches of the tallest trees. This invariance of vessel resistance coupled with area-preserving branching and a space-filling network leads to many scaling laws including the 3/4-power scaling for metabolic rate. Detailed scaling predictions for conductivity, pressure gradients, fluid velocity, and relative amount of heartwood are all in excellent agreement with data. As an added bonus we can also show why the maximum height of a tree is of the order of 100m rather than 1m or 1000m. This follows because the taper cannot continue indefinitely; the trunk, whose size is, to some extent, constrained biomechanically, simply cannot contain all conducting vessels if it is allowed to grow without bound. This therefore provides an explanation from fundamental principles why the size of trees is limited and how that size is related to basic parameters which depend on both mechanical and hydrodynamic constraints.

It is also possible to use the model to calculate the fractal dimension of plants, D, as defined by the box-counting method. As long as the resolution is not too fine then the model predicts $D = 3/2$, in excellent agreement with data [6]. Amusingly, measurements have been repeated on roots grown between glass plates separated by only 3mm in order to minimize their disturbance. This leads to $D \approx 1.3$ significantly different from 1.5. However,

by growing roots in this way, the network has been restricted to 2-dimensions rather than 3. If the calculation is now repeated in 2-dimensions the model predicts $d = 4/3$! This is indirect evidence of the space-filling requirement for these networks.

4 Conclusions

The paradigm and principles expressed by the general model leads to a novel way of exploring these types of complex systems and understanding their scaling properties. On the one hand it is, by necessity, a "zeroth order model" embodying many of the essential features of biological systems and, as such, can serve as a point of departure for more detailed, possibly more realistic analyses. On the other, its success inevitably suggests applications to other interesting and challenging problems and situations. Obvious areas deserving serious investigation include intracellular transport, aging and longevity, and the extension to ecological environments. Quarter-power scaling has been observed in all of these regimes so it is natural to explore the application of our ideas to them. The success of the model should be viewed as a beginning rather than an end.

References

[1] K. Schmidt-Nielsen, *Scaling; Why is Animal Size so Important?*, Cambridge University Press, Cambridge, (1984); W. A. Calder III, *Size, Function and Life History*, Harvard University Press, Cambridge, (1984).

[2] B. J. Enquist, J. H. Brown and G. B. West, *Nature* **395**, 163 (1998).

[3] W. H. Woodruff, G. B. West and J. H. Brown, to be published.

[4] G. B. West, J. H. Brown, and B. J. Enquist, *Science* **276**, 122 (1997).

[5] A. G. Greenhill, *Proc. Cam. Phil. Soc.* **4**, 65, 1881; T. A. McMahon, *Science* **179**, 1201, (1973); T. A. McMahon and R. E. Kronauer, *J. Theo. Biol.* **59**, 443 (1976).

[6] J. A. Tatsumi *et al*, *Ann. Bot.* **64**, *499 (1989)*; D. R. Morse *et al*, *Nature* **314**, 731 (1985); A. H. Fitter and T. R. Strickland, *Func. Eco.* **6**, *632 (1992)*.

[7] Y. C. Fung, *Biodynamics*, Springer-Verlag, New York, (1984); C. G. Caro et al., *The Mechanics of Circulation*, Oxford University Press, Oxford, (1978).

[8] This is often referred to as Murray's law, (see C. D. Murray, *Proc. Nat. Acad. Sci.* **12**, 207, (1926)) though it is mentioned earlier by D'Arcy W. Thompson, *On Growth and Form*, Cambridge University Press, Cambridge, (1942).

[9] J. R. Womersley, *Phil. Mag.* **46** (Series 7), 199, (1955); *J. Physiol.* **127**, 553, (1955).