The origin of allometric scaling laws in biology

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Abstract

The empirical rules relating metabolic rate and body size are described in terms of (i) a scaling exponent, which refers to the ratio of the fractional change in metabolic rate to a change in body size, (ii) a proportionality constant, which describes the rate of energy expenditure in an organism of unit mass. This article integrates the chemiosmotic theory of energy transduction with the methods of quantum statistics to propose a molecular mechanism which, in sharp contrast to competing models, explains both the variation in scaling exponents and the taxon-specific differences in proportionality constants. The new model is universal in the sense that it applies to unicellular organisms, plants and animals.

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1. Introduction

Two fundamental elements that characterize behavioral and physiological properties of an organism are its body size, and its metabolic rate, that is its rate of energy expenditure. Empirical studies which go back to Kleiber (1961), with extensions and analyses by several authors, see for example Brody (1945), Hemmingsen (1960), have delineated an allometric relation between the basal metabolic rate, that is, the rate of energy expenditure of a fasted individual under thermoneutral conditions, and body size, that is, the body mass of an organism at the adult state. These empirical rules are expressed in terms of two quantities: (i) a scaling exponent, which refers to the ratio of the fractional change in metabolic rate to a change in body size; (ii) a normalizing coefficient, which describes the rate of energy expenditure in an organism of unit mass. The scaling exponent is known to assume a range of values which is dependent on body size, phylogenetic status and the degree of activity of the organism. Large mammals are typically described by a 3/4-power scaling rule, however, among small mammals and several species of birds, a 2/3-power scaling is observed (Dodds et al., 2001; White et al., 2003). The normalizing coefficient, sometimes called the proportionality constant, also assumes a large range of values. This variation, however, is primarily dependent on phylogenetic status. Values of the proportionality constant increase from unicellular organisms to plants and animals (Hemmingsen, 1960). The values also vary between ectotherms and homeotherms. For example, the metabolic rate of a 1 kg homeotherm is typically 30 times higher than a similarly sized ectotherm.

Metabolic activity within organisms has its origin in processes of energy transduction localized in biomembranes—the plasma membrane in unicells, the thylakoid membrane of chloroplasts and the inner membrane of mitochondria. The chemiosmotic theory of energy transduction within biomembranes, as proposed by Mitchell (1966), postulates that metabolic activity is due to the coupling between two bioenergetic processes, the transfer of electrons between redox centers within the biomembrane and the phosphorylation of ADP to form ATP.

Processes of energy transduction can be analysed either on the microscopic scale, where the interactions of the...
electrons and protons with atoms and molecules within the biomembrane are considered, or on the macroscopic scale where we only consider the behavior of matter in bulk and deny that the system consists of molecular components.

The models that have been proposed to analyse the relation between metabolic activity and body size have focused almost exclusively on processes defined at the macroscopic level, see, for example, McMahon (1973), Banavar et al. (2002) and West et al. (1997). The predictions derived in these studies pertain uniquely to the scaling exponents. These models have not succeeded in explaining the large variation in proportionality constants that characterize different groups of organisms, for example, ectotherms and endotherms. This failure derives partly from the macroscopic scale on which these models are described, and the fact that proportionality constants are to a large extent determined by the microscopic molecular dynamics of metabolic regulation (Hochachka and Somero, 2000, Chapter 7).

This article is concerned with explaining both the variation in scaling exponents and the differences in normalizing coefficients that describe the empirical laws. Accordingly, the model we propose will be based on energy transduction on the microscopic molecular scale, namely: the coupling of two molecular motors—the redox reaction and ADP phosphorylation.

Our model rests on the assumption that metabolic energy generated by the redox reactions—the primary energy source—can be stored in terms of coherent vibrational modes of the energy transducing membranes in bacteria, chloroplasts and mitochondria. The significance of biomembranes and the collective excitations of molecular groups as a mechanism for storage of metabolic energy was first articulated by Frohlich (1968) (see also Tuszynski and Kurzynski, 2003, Chapter 3). This mechanism for linking the energy from electron transport to phosphorylation constitutes a fundamental element in our theory.

Biomembranes can be viewed as a bilayer of complex lipids embedded with proteins (Singer and Nicholson, 1972). Each lipid molecule or protein has a preferred surface area. Consequently, the membrane, unlike the surface of a fluid, can be considered inextensible. Thus, in our model of the metabolic activity of the cell, we will consider the membrane to be a relatively rigid system whose molecular groups can undergo collective modes of vibration due to the energy fed into the system from the redox reactions. We will assume that these molecular excitations are quantized, and by appealing to the quantum theory of solids, we will derive certain relations between the metabolic energy generated by the cell and the metabolic cycle time, that is, the mean turn over time for the oxidation–reduction reactions that provide the primary energy source. These analytic results will then be invoked to determine relations between cellular metabolic rate and cell size.

Now, organismic metabolic rates are driven by the rates of energy expenditure within internal organs; whereas organ and tissue metabolic rates represent the sum of cellular metabolic rates, see Krebs (1950) and Suarez and Darveau (2005). These observations will be invoked to extend the allometric relations derived for unicells to relations that apply to multicellular organisms.

A class of models to derive the allometric relations in terms of quantum statistics was initially developed in Demetrius (2003). This study did not address inter-specific variations in the proportionality constants. The model focused primarily on explaining the 2/3 and 3/4 scaling exponents documented for birds and mammals. The present study subsumes this earlier effort. The mechanism of energy storage in terms of collective modes of vibration of molecular groups provides a conceptual framework which we will invoke to build on our original study. The model we will now develop is applicable to unicells, plants and animals, and explains the variation in both scaling exponents and proportionality constants which characterize the allometric laws.

The main achievement of this new model is the derivation of the following scaling rule relating metabolic rate, \( P \), with body size, \( W \), namely

\[
P = \gamma C \Delta p W^\beta.
\]  

The terms described in Eq. (1) will now be defined and elucidated. We will also present a synopsis of the main predictions of this new theory of metabolic activity. We first distinguish between the scaling exponent and the normalizing constants.

The scaling exponent: This quantity, denoted by \( \beta \), is a function of metabolic efficiency, the extent to which the electron transport chain is coupled to ADP phosphorylation.

The analytic dependence of \( \beta \) on metabolic efficiency, denoted by \( \mu \), will be shown to be contingent on the mechanism by which energy is transduced within redox centers in the biomembrane. The mechanism will be shown to be largely dependent on cell size. Cell size shows a large variation within all taxa. However, cell size differences between plants and animals are largely determined by the size of their energy transducing organelles. Chloroplasts, the energy transducing organelle in plants, by virtue of their evolutionary origin, are generally much larger than mitochondria. Consequently, large cell size will be typical of plants and relatively small cell size characteristic of animals. Hence, the functional dependence of \( \beta \) on \( \mu \) will differ between green plants and animals.

We will show that for green plants, \( \beta \) is given by

\[
\beta = \frac{2\mu - 1}{\mu}.
\]  

We will also show that for animals and most prokaryotes, the scaling exponent will be given by

\[
\beta = \frac{4\mu - 1}{4\mu}.
\]
The *minimal* value for the scaling exponent $\beta$ is defined by the condition where the rate of heat production of the organism is matched by the rate at which heat is dissipated through its body surface. Euclidean surface area rules entail that there is a unique minimal scaling exponent, which is given by $\beta = 2/3$.

Maximal values for the scaling exponent will be achieved when the metabolic efficiency $\mu = 1$. Hence, when the exponent is given by Eq. (2), $\beta = 1$. We therefore predict that the scaling exponent for green plants will satisfy the relation

$$2/3 \leq \beta \leq 1.$$

However, when the exponent is given by Eq. (3), we have $\beta = 3/4$. We now predict that the scaling exponent for animals will satisfy the bounds

$$2/3 \leq \beta \leq 3/4.$$

The actual scaling exponents achieved by different organisms will depend on evolutionary constraints. These constraints can be derived by appealing to directionality theory, an analytic model of evolution based on demographic entropy as the measure of Darwinian fitness, Demetrius (1997). Directionality theory distinguishes between equilibrium species, that is, populations which spend the greater part of their evolutionary history in the stationary growth phase, and opportunistic species, that is organisms, which are subject to irregular fluctuations in population numbers. According to this theory, equilibrium species (which include perennial plants and large animals) will be characterized by physiological states which maximize the basal metabolic rate; whereas opportunistic species (annual plants, small animals) will be described by states which minimize the basal metabolic rate.

The predictions which derive from this analysis can be annotated as follows:

(A) In green plants, equilibrium species, typically perennials, will be described by a scaling exponent $\beta = 1$, whereas opportunistic species, typically annuals will be defined by $\beta = 2/3$.

(B) In animals, equilibrium species, typically large organisms, will have a scaling exponent $\beta = 3/4$, whereas opportunistic species, typically small organisms, will have a scaling exponent $\beta = 2/3$.

The *proportionality constant:* This parameter involves three elements:

(i) *A measure of transport efficiency:* This quantity, denoted by $\gamma$, describes the efficiency with which energy is transported between the different cells and tissues within the organism. This measure of transport efficiency depends on (a) the nature and composition of the nutrients, organic and inorganic, that the organism uses, (b) the geometry of the internal branching network within the organism.

(ii) *Bioenergetic parameters.* These quantities, the proton conductance, $C$, and the electrochemical proton gradient, $\Delta p$, characterize the dynamics of proton transport within and across the energy transducing membranes.

(a) The proton conductance can be expressed in terms of an activation free energy $\Delta \tilde{p}$, the absolute temperature $T$ and the gas constant $R$ (Garlid et al., 1989). We have

$$C = C_0 \exp \left( -\frac{\Delta \tilde{p}}{RT} \right).$$

where $C_0$ is a numerical constant.

(b) The electrochemical proton gradient is given in terms of $\Delta \psi$, the membrane potential, that is, the electrical potential difference between the phases separated by the biomembrane, and $\Delta \textrm{pH}$, the pH difference between the two phases on either side of the membrane, see Nicholls and Ferguson (2002).

We have

$$\Delta p = \Delta \psi - \frac{2 \cdot 3RT}{F} \Delta \textrm{pH}. \quad (5)$$

Here $F$ denotes Faraday’s constant.

There exists no quantitative data on transport efficiency. However, a large body of data on proton conductance and the electrochemical proton gradient exists. Comparative studies of proton conductance in various organisms have delineated certain distinct patterns: proton conductance is greater in endothermal tissues than in homologous tissues of ectotherms; proton conductance is lower in tissues of larger mammals than homologous tissues of smaller mammals (Brand et al., 1991; Porter and Brand, 1993). Experimental studies of metabolic activity in mitochondria indicates that the proton conductance of the inner membrane is potential dependent, increasing rapidly for certain range of values for $\Delta p$ (Nicholls, 1974). Hence, within organisms, the proton conductance and the electrochemical proton gradient will be positively correlated.

The predictions which emerge by integrating these empirical observations and experimental studies with Eq. (1) can be described as follows:

(C) The metabolic rate of endotherms will be greater than that of equivalent size ectotherms when compared at constant body temperature.

(D) Tissues in larger mammals should have a lower in vitro metabolic rate than homologous tissues in smaller mammals.

The technical arguments that support the predictions described by items (A)–(D), are developed in Sections 2–5. Section 2 gives an account of the chemiosmotic theory of energy transduction. Section 3 appeals to the methods of quantum statistics to derive, for models of unicellular
organisms, a relation between metabolic energy and the cycle time of the redox reactions. In Section 4 we give the rationale for the mathematical methods used in Section 3 by giving a historical account of the origins of quantum theory. The relation between metabolic energy and cycle time derived in Section 3 is invoked in Section 5 to obtain a relation between metabolic rate and cell size. This allometric relation is then extended to determine a general expression relating metabolic rate and body size which pertains to multicellular organisms. The species variation in scaling exponents and the taxon-specific differences in proportionality constants are explained in Section 6. Sections 7 and 8 deal with empirical and comparative issues, respectively. In Section 7 we provide empirical support for the predictions of the new theory. Section 8 contrasts the predictive and explanatory range of quantum metabolism with the fractal network model (Brown et al., 2004) which has recently been proposed as an explanation of the allometric laws for metabolic rate. Brown et al. (2004) contend that their model represents a framework for understanding the relation between metabolic activity and size at various levels of biological organization—macromolecules, organisms, populations, ecosystems—thus providing a basis for a metabolic theory of ecology. Our comparative analysis of the two models suggests that quantum metabolism may have stronger claims as a conceptual framework for a metabolic theory that applies across all scales of biological organization.

2. Energy transduction in biomembranes

Organisms require a continual supply of energy to synthesize molecules and to organize these molecules into the intricate fabric of cells and tissues that define the living state. Energy transduction within cells is primarily associated with enzyme complexes that are localized in a particular class of biomembrane: the plasma membrane in simple prokaryotic cells, the inner membrane of mitochondria and the thylakoid membrane of chloroplasts. These membranes have a common evolutionary origin. Thus the mechanism of ATP synthesis and ion transport associated with these diverse membranes are similar, despite the different nature of their primary energy sources, see, for example, Harold (1986).

According to the chemiosmotic theory, the energy released in oxidations is coupled by proton translocation across the biomembrane to ADP phosphorylation. The model of energy transduction we now consider is universal in the sense that it is applicable to all aerobic organisms. The energy transformation involves the interconversion of three forms of free energy, see Nicholls and Ferguson (2002) and Harris (1995).

1. The redox potential difference, that is, the actual redox potential between the donor and acceptor couples in the electron transfer chain.

2. The proton motive force which describes the free energy stored in the membrane electrochemical proton gradients.

3. The phophorylation potential for ATP synthesis.

The proton circuit which describes the coupling of the electron transport chain with ADP phosphorylation by means of the proton flux, denoted by \( J \), is schematically represented by Fig. 1.

The transfer of electrons is accomplished by a series of redox centers. It is described by a cyclic scalar process (oxidized–reduced–oxidized form) which induces a vectorial process characterized by a net movement of protons from one molecular center to another. The transit time of this cyclic process of energy transfer determines the total metabolic flux, that is, the number of proton charges released by the redox reactions. This transit time, which we will call the metabolic cycle time, denoted by \( \tau \), will play a fundamental role in our model.

The electron transport between redox centers is coupled to the outward pumping of protons across the membrane, thus producing an electrochemical gradient, called the proton motive force, \( \Delta p \) (dimension: mV). Let \( C \) (dimension: \( n \) mol H\(^+\) per unit time, per mg protein, per mV) denote the proton conductance of the membrane and \( J \) (dimension: charge per unit time) denote the proton current induced by the electromotive force. We can now apply Ohm’s law to the proton circuit and obtain \( J = C \Delta p \).

The energy generated per cycle will be given by \( E = J \tau \); where \( \tau \) denotes the cycle time, the mean turn over time for the redox reactions. We can rescale this quantity using the counting index, Avogadro’s number \( N_A \), the number of atomic mass units per mole, by writing \( g = J/N_A \). We now obtain

\[
E = g \tau.
\]

We will exploit this characterization of the metabolic energy of the molecule, to determine the total metabolic energy generated by the downhill electron transfer during one cycle of the redox reaction.

3. Quantum metabolism

The basic information on the structure of biomembranes derives from the work of Singer and Nicholson (1972).

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(For context, a brief diagram illustrating the proton circuit linking the electron transport chain with ADP phosphorylation is shown.)
These studies, which have led to the fluid-mosaic model, describe the membrane as a sheet-like structure with a thickness of about $10^{-6}$ cm. These structures, which consist of lipid–protein complexes, are non-covalent aggregates: the constituent proteins, which are embedded in the phospholipid bilayer, are held together by many cooperative non-covalent interactions (Fig. 2).

The proteins embedded in the membrane have extraordinary dipolar properties and may be expected to exhibit oscillations. Fröhlich (1968) has proposed that if the primary energy source is sufficiently large, then long range Coulomb interactions will cause the energy to be shared among the molecular groups embedded in the membrane. We postulate this mechanism of energy storage as availing the energy transfer step linking oxidations and ATP synthesis.

In evaluating the metabolic energy of the system, which we assume is determined by the molecular oscillators, we let $N$ denote the number of molecular groups in the membrane. The system has $3N$ degrees of freedom corresponding to the $3N$ coordinates which are necessary to specify the location of the embedded proteins. In view of the long range Coulomb interactions between the molecules in the membrane, the molecular groups will not vibrate independently of each other but will be subject to coupled vibrations. Hence the molecular oscillations of the system can be described in terms of $3N$ normal modes of vibration, each with characteristic frequency $\omega_1, \omega_2, \ldots, \omega_{3N}$. The molecular vibrations can therefore be considered equivalent to $3N$ harmonic oscillators. We will consider these molecular oscillators to be collective properties of the membrane as a whole and we will compute the metabolic energy, a property which comes from the vibrations of the lattice into which the molecular groups are bound together, in terms of quantum theory.

The quantum hypothesis means that the energy of the harmonic oscillator is quantized according to the equation $E_n = n\hbar\omega$, where $\omega$ is the fundamental frequency of the oscillator and $\hbar$ is Planck’s constant. This equation asserts that the oscillator may only have discrete energies, $\hbar\omega, 2\hbar\omega, 3\hbar\omega \ldots$ above the zero point level. According to the quantum hypothesis, each independent mode of oscillation of the membrane as a whole should be ascribed an average energy

$$E(\omega) = \frac{\hbar\omega}{\exp(\hbar\omega/\gamma t) - 1},$$

where $\omega$ is the frequency of vibration of a mode.

The total metabolic energy generated by the redox reactions and stored in the membrane will be given by

$$u = \sum_{k=1}^{3N} \frac{\hbar\omega_k}{\exp[\hbar\omega_k/\gamma t] - 1}. \quad (6)$$

We will derive an approximate value for this energy by ignoring the discrete structure of the membrane, and considering the system as a homogeneous elastic medium. In order to determine the total metabolic energy in the context of this model, we need to calculate the different standing wave patterns generated by the vibrations of the molecular groups.

Now, the number of standing waves in an enclosure with wave vectors in the range $\omega$ to $\omega + d\omega$ is determined by the geometry of the system. It is proportional to volume of the enclosure and to $\omega^3 d\omega$. Hence for elastic waves, the density of modes will be given by

$$f(\omega) d\omega = \frac{3 v_0^2}{2 \pi c^3} \omega^3 d\omega.$$

Here, $v$ is the volume of the membrane and $c$ the velocity of propagation of waves within the membrane.

We can now use this expression for the density of modes to derive an approximate value for the total metabolic energy, $u$. We have

$$u = \int_0^{\omega_{\text{max}}} \frac{3 \hbar v}{2 \pi c^3} \exp(\hbar\omega/\gamma t) - 1. \quad (7)$$

Here $\omega_{\text{max}}$ denotes the maximum frequency.

In order to evaluate Eq. (7), we will exploit certain arguments which have been developed in solid state physics for determining the heat capacity of lattice crystalline solids, see, for example, Mandl (1988).

The value of the integral in Eq. (7) depends critically on the ratio $\hbar\omega_0/\gamma t$. We will evaluate Eq. (7) under two constraints on the product $\gamma t$. We will distinguish between two limiting cases: $\gamma t \gg \hbar\omega_0$ and $\gamma t \ll \hbar\omega_0$. The metabolic energy per molecule, $\gamma t$, is determined by the proton motive force, $\Delta p$, and the metabolic cycle time. We will assume that the range of variation in $\gamma t$ is determined by species and taxon-specific variation in $\tau$. Accordingly, we will characterize the limiting conditions $\gamma t \gg \hbar\omega_0$ and $\gamma t \ll \hbar\omega_0$, in terms of limiting constraints on the cycle time $\tau$.

Large cycle time: This constraint is equivalent to the condition $\gamma t \gg \hbar\omega_0$. When this condition prevails, the exponent $\hbar\omega_0/\gamma t$ in Eq. (7) is always much less than 1. The integral given by (7) can now be simplified, and

Fig. 2. Fluid-mosaic model of membrane structure, showing protein molecules, lipid molecules and lipid bilayer (adapted from Singer and Nicholson, 1972).
modes from zero to \( u \).

We obtain from Eq. (12), the relation

\[
\frac{v}{2\pi^2 c^3} \delta \omega_{max} = 2N.
\]

Thus, we obtain from Eq. (8) that

\[
u = 2NgT.
\]

Eq. (9) asserts that the total metabolic energy, \( u \), is a product of \( gT \), the metabolic energy associated with the oscillation of a single molecular group, and \( 2N \), the total number of modes available for energy storage. This equation indicates that, in the limit of large cycle time, the total metabolic energy per cycle scales linearly with cycle time.

In Section 5, we will appeal to Eq. (9) to show that in systems described by large cycle time, the cellular metabolic rate is isometric to cell size. This result will then be extended to explain departure from isometry due to the metabolic inefficiency of the cell.

Small cycle time: When this constraint prevails, we have \( gt \ll \hbar \omega_0 \), and the approximation made in deriving (9) is no longer valid. We will now introduce the dimensionless variable, \( x = \hbar \omega_0 / gT \), and obtain

\[
u = \frac{3v}{2\pi^2 h c^3} (gT)^4 \int_0^{\omega_{max}} \frac{x^3}{e^x - 1} \, dx.
\]

This upper limit of integration is \( x_{max} = \hbar \omega_{max} / gT \). In view of the constraint on \( g \), the upper limit of integration is large relative to 1. Hence, the integrand is exponentially small at the upper limit. We may therefore write for Eq. (10),

\[
u = \frac{3v}{2\pi^2 h c^3} (gT)^4 \int_0^\infty \frac{x^3}{e^x - 1} \, dx.
\]

Since the integral in the above equation takes the value \( \pi^4/15 \), we obtain

\[
u = \left( \frac{\pi^4 v}{10h c^3} \right) T^4.
\]

We obtain from Eq. (12), the relation

\[
u = aT^4.
\]

Here \( a = kq^4 \) and \( k = \pi^2 v / 10h c^3 \).

Eq. (13) asserts that in systems defined by a small cycle time, the metabolic energy generated by the redox reactions scales with cycle time to the fourth power. In Section 4, we will use Eq. (13) to show in this class of systems that the cellular metabolic rate will now scale to the 3/4 power of cell size. As in the case involving large cycle time, this scaling relation will also be extended to explain deviations from the 3/4 rule due to metabolic inefficiency of the cell.

4. Quantum metabolism and the Stephan–Boltzmann law

The general expression for the metabolic energy generated per cycle is given by Eq. (7). The relation given by Eqs. (9) and (13) are limiting cases of the general expression obtained by imposing constraints on the cycle time. Eq. (9) describes a linear dependence of metabolic energy on cycle time. This relation arises when the cycle time is large. Eq. (13) describes a fourth power dependence on cycle time, a relation which is obtained when the cycle time is small. This fourth power relation is analogous to the Stephan–Boltzmann law for electromagnetic radiation, which asserts that the energy density of radiation, denoted by \( e \), is given by

\[ e = \sigma T^4. \]

Here \( T \) denotes the absolute temperature and \( \sigma \) a proportionality constant. The formal correspondence between Eqs. (13) and (14) suggests a mechanistic congruence between the biological process of energy transduction in biomembranes and the physical process of electromagnetic radiation. We will now explore the basis for this congruence and discuss its implications. We will do this by providing an account of the origin of quantum theory, and the application of this theory to the analysis of heat capacity of solids. Our analysis will draw from the historical and conceptual account given in Longair (2004).

The empirical law relating electromagnetic radiation and temperature was discovered by Stephan in 1879. The first attempt to give an explanation of the radiation law was made by Boltzmann in 1884. Boltzmann’s model, which was based on classical thermodynamic arguments, yielded an explanation of the scaling exponent in the radiation law. However, the thermodynamic methods were unable to explain the proportionality constant \( \sigma \). In Boltzmann’s analysis, the proportionality constant simply appeared as a constant of integration without any evident physical interpretation.

An explanation of both the scaling exponent and the proportionality constant was later achieved by Max Planck who addressed the problem by treating radiation as a gas of photons. In 1900 Planck proposed a model in which the radiation in a metallic cavity was analysed in terms of oscillations of these photons. The critical assumption in Planck’s model was the quantization principle: the energy that can be stored by an oscillator with frequency \( \nu \) can only be integral multiples of a basic energy unit, which is proportional to the characteristic frequency of the oscillator.

Planck’s quantum hypothesis is now recognized as a fundamental principle which applies not only to photons in an electromagnetic field, but also to phonons in crystalline...
solids, electrons in metals, spin waves in ferromagnets. The fundamental nature of the hypothesis was evidently recognized by Einstein, who in 1907 invoked quantum theory to study the heat capacity of lattice vibrations of crystalline solids. Einstein realized that Planck’s quantization of radiation oscillators can also be applied to material oscillators, that is, the vibrations of atoms in a solid. In contrast to Planck’s radiation oscillators, which can have all possible frequencies, the material oscillators considered in Einstein’s model was assumed to have only one frequency, which is the characteristic frequency of the substance. This assumption restricted the range of applicability of the model, and failed to explain the heat capacity of a solid at low temperature. The reasons for the discrepancy at very low temperatures was ultimately elucidated by Debye in 1912. This new model postulates that the material oscillators are not actually independent of the oscillations of the others, but are coupled to them because of the forces between the molecules. The methods introduced by Debye have now become canonical in the quantum theory of solids.

Quantum metabolism is a molecular biological application of Planck’s quantization hypothesis. Quantum metabolism assumes that Planck’s quantization of radiation oscillators can also be applied to the vibration of large molecular groups embedded in the membrane of energy transducing organelles in biological cells. In contrast to Planck’s radiation oscillators, which can assume an infinite number of frequencies, the biological oscillators are assumed to be described by a narrow band of frequencies. This restriction derives from the long range Coulomb interactions between the oscillating units in the membrane.

In addition to these differences in the nature of the oscillators, there exist fundamental differences between Planck’s model of electromagnetic radiation and the model of energy transduction in biomembranes.

Electromagnetic radiation is generated by the energy of photons. Radiant energy is due to the random thermal motion of the individual photons. Hence, radiant energy at thermal equilibrium will depend on temperature. The fundamental unit of energy in this class of models is

$$E = k_B T,$$

the typical thermal energy per molecule. Here, $k_B$ is Boltzmann’s constant and $T$ the absolute temperature.

Metabolism is generated by the energy released by the electrons and protons as the particles are transferred from donor to acceptor states within the energy transducing membrane. This process occurs by quantum tunnelling (Devault, 1980; Page et al., 1999) and is essentially isothermal. Metabolic energy is due to the coherent excitation of the molecular groups. This excitation is driven by the downhill flow of electrons within the electron transport chain. The generation of metabolic energy is thus a non-equilibrium process. The energy at steady state will therefore depend on the mean turn over time of the oxidation–reduction reaction, that is the metabolic cycle time, $\tau$. The fundamental unit of energy in this class of models is now given by

$$E = g \tau,$$

the typical metabolic energy per particle. Here, $g$ is a function of the proton conductance and the proton motive force.

The derivation of Eqs. (9) and (13) proceeds from the notion that physical processes at thermal equilibrium, where temperature is the organizing variable, and biological processes at non-equilibrium steady states, where cycle time is the mediating parameter, can be addressed within the same mathematical formalism. This observation has an analytical basis. It issues from the following mathematical fact: the growth rate parameter in population dynamics satisfies a variational principle which is formally analogous to the minimization of the free energy in thermodynamic systems (Demetrius, 1983; Arnold et al., 1994). This analytical fact implies a formal correspondence between certain fundamental thermodynamic variables and the metabolic parameters which we now describe in Table 1.

The methods we have solicited to derive Eqs. (9) and (13) are an application of this general principle.

### 5. The scaling relations

The expressions for the metabolic energy $u$ given by Eqs. (9) and (13) pertain uniquely to the dynamics of the electron transport process, that is, the energy donating system within a single cell. Let $\tilde{u}$ denote the metabolic energy associated with ADP phosphorylation, the energy accepting process and let $\mu$ denote the metabolic efficiency. It is represented by the ratio: rate of ADP phosphorylation/rate of electron transport.

Hence

$$\log \tilde{u} = \mu.$$

The expression $u$ denotes the energy generated by the electron transport process. This is given in the large cycle time limit, by Eq. (9), and the small cycle time limit, by Eq. (13).

The metabolic efficiency $\mu$ can be expressed in the form

$$\mu = q Z.$$

Here, $q$ with $0 \leq q \leq 1$ is a measure of the degree of coupling between the electron transport process and ADP phosphorylation; $q = 1$ and 0 for completely coupled and uncoupled systems, respectively. The quantity $Z$ is a stochiometric parameter, which is equal to the ATP/electron flux ratio.

### Table 1

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Now at the steady state, the energy \( \tilde{u} \), associated with ADP phosphorylation, is given by \( \tilde{u} = \tilde{S} T \), where \( \tilde{S} \) denotes the thermodynamic entropy of the cell and \( T \) denotes the absolute temperature.

Now the entropy \( \tilde{S} \) is an extensive quantity which will be proportional to the total volume of the cell. Assuming that the density of the cell is uniform, we obtain, since the temperature is an intensive parameter, \( \tilde{u} = \rho W_c \), where \( W_c \) denotes cell size and \( \rho \), a proportionality constant. We will consider \( W_c \) as a measure of metabolic mass and write \( \rho = 1 \).

We can now appeal to Eqs. (9) and (13) to derive an expression for the metabolic rate in the two regimes, large and small cycle times, respectively.

**Large cycle time**: The expression for the total metabolic energy generated by the electron transfer process is given by Eq. (9). We will assume that \( N \) the number of molecular groups in the membrane scales linearly with cell volume, which we assume scales linearly with cell size \( W_c \). Hence \( u = k_1 g W_c \). Accordingly, the metabolic energy due to the coupling between the electron transfer and ADP phosphorylation is given by \( \tilde{u} = (k_1 g W_c)^{\mu} \). By appealing to the thermodynamic characterization of \( \tilde{u} \) as a function of cell size, we now obtain an expression for the metabolic cycle time \( \tau_c \), namely

\[
\tau_c = \left( \frac{1}{k_1 g} \right) W_c^{(1-\mu)/\mu}.
\]

The metabolic rate \( P_c \) of the cell is given by \( P_c = \tilde{u} / \tau_c \), which yields

\[
P_c = k_1 g W_c^{(2\mu-1)/\mu}.
\]

When \( \mu = 1 \), we obtain for the cycle time \( \tau_c \), and the metabolic rate \( P_c \), the expressions

\[
\tau_c = \frac{1}{k_1 g}, \quad P_c = k_1 g W_c.
\]

Hence, when \( \mu = 1 \), the cycle time is independent of cell size, and is determined uniquely by the proton conductance and the proton motive force, whereas the metabolic rate is isometric to cell size.

**Small cycle time**: The expression for the total metabolic energy generated by the electron transfer process is now given by Eq. (13). By appealing to the argument described in the large cycle time limit, we now obtain for the metabolic cycle time, the expression

\[
\tau_c = \left( \frac{1}{k_1 g} \right) W_c^{1/4\mu}.
\]

Here \( a \) is the proportionality constant given by \( a = k_2 g^a \).

The total metabolic rate, \( P_c \), of the cell now becomes

\[
P_c = k_2^{1/4} g W_c^{(4\mu-1)/4\mu}.
\]

When \( \mu = 1 \), we now have

\[
\tau_c = \left( \frac{1}{k_2^{1/4} g} \right) W_c^{1/4}, \quad P_c = k_2^{1/4} g W_c^{1/4}.
\]

The metabolic cycle time in this case satisfies the 1/4 rule, and the metabolic rate, the 3/4 rule.

### 5.1. Multicellular organisms

The preceding expression for the metabolic rate, which pertains to a single cell, can be extended to multicellular organisms. Our analysis rests on the following multilevel scaling hypothesis: organ and tissue metabolic rates represent the sum of cellular metabolic rates, see, for example, Suarez and Darveau (2005).

We will assume that the metabolic rate \( P_c \) of a unicellular organism is given by

\[
P_c = z_c W_c^\beta.
\]

A multicellular organism of size \( W \) can be considered an aggregate of closely packed identical cells. The metabolic rate \( \tilde{P}_c \) of a cell in this multicellular aggregate will, on account of the changes in the normalizing coefficient induced by the interaction between the cells, differ from the metabolic rate \( P_c \) of a free living cell. We write

\[
\tilde{P}_c = \tilde{z}_c W_c^\beta.
\]

Here, \( \beta \) is the scaling exponent of the cell in the free living state, and \( \tilde{z}_c \) the effective normalizing coefficient, the proportionality constant that describes the cell considered as an element in a multicellular aggregate. The number \( \tilde{z}_c \) will be determined by the total number of cells, \( N_c \), in the aggregate, and the scaling exponent \( \beta \).

Assuming the multilevel scaling hypothesis, the proportionality constant \( \tilde{z}_c \), (provided we impose continuity constraints on \( \beta \), and \( N_c \)) will be given by

\[
\tilde{z}_c = z_c (N_c^\beta / N_c).
\]

Hence \( \tilde{P}_c = z_c (N_c^\beta / N_c) W_c^\beta \).

Now the total metabolic rate \( P \) of the multicellular organism will be given by \( P = \gamma N_c \tilde{P}_c \), where \( \gamma \) denotes the efficiency with which energy is transported between the different cells and tissues within the organism. Since \( N_c \sim W/W_c \), we conclude that

\[
P = \gamma \alpha W^\beta,
\]

where \( \alpha = C \Delta p \).

### 6. The scaling exponents and the normalization parameter

We will now exploit Eq. (21) to generate a series of predictions regarding the metabolic activity of plants and animals, and also to specify the range of values assumed by the scaling exponents and the proportionality constant.

In deriving the predictions of the model, we will appeal to a series of empirical studies—based on metabolic activity in mitochondria—of correlations between the different bioenergetic parameters, the proton conductance, \( C \), the proton motive force, \( \Delta p \), and the proton current, \( \Delta \rho \).

Nicholls (1974) has investigated the relation between proton conductance and \( \Delta p \), and shown that it is potential dependent, increasing rapidly when \( \Delta p \) is greater than
6.1. Scaling exponents

There exist two analytic expressions for the scaling exponent as a function of metabolic efficiency, \( \mu \), namely \( \beta = (2\mu - 1)/\mu \) and \( \beta = (4\mu - 1)/4\mu \). It is important to specify the class of organisms to which these different expressions for \( \beta \) refer.

We note that the condition \( \beta = (2\mu - 1)/\mu \) occurs when the relation \( g_t \gg h_o \). This constraint prevails for very large values of \( \tau \). Such values for the metabolic cycle time will be characteristic of systems with large cell size, typically, green plants whose cells contain chloroplasts as energy transducing organelles.

The condition \( \beta = (4\mu - 1)/4\mu \) obtains when the relation \( g_t \ll h_o \) holds. This condition will occur when the metabolic cycle time \( \tau \) is small—a situation which is typical of systems with relatively small cell size. Accordingly, this constraint on \( \beta \) will be characteristic of animals.

We will also appeal to certain results in directionality theory, an analytic model of evolution based on the concept entropy as a measure of Darwinian fitness (Demetrius, 1997).

Directionality theory distinguishes between equilibrium and opportunistic species and predicts that (i) in equilibrium species evolution will result in a unidirectional increase in entropy; (ii) in opportunistic species, evolution will result in a unidirectional decrease in entropy, when population size is large, and random non-directional change in entropy when population size is small. Evolutionary entropy is known to be positively correlated with metabolic rate, hence the theory implies that equilibrium species will be described by conditions which maximize metabolic rate. Opportunistic species will be described by states which minimize metabolic rate, when population size is large and states which show large variations in metabolic rate when population size is small.

Among green plants, perennials are equilibrium species, whereas annuals are opportunistic. In animal populations, body size is a rough indication of the condition, equilibrium or opportunistic. Large animals are equilibrium species, whereas small animals are opportunistic.

We will now apply these evolutionary principles to predict the scaling exponents which will characterize plants and animals.

(i) The case \( \beta = (2\mu - 1)/\mu \): The minimal value for the scaling exponent is given by \( \beta = 2/3 \). This corresponds to the situation where the organism’s rate of heat production is matched to the rate at which heat is dissipated through its body surface. Maximal values for \( \beta \) is given when \( \mu = 1 \), and we have \( \beta = 1 \).

We predict, in view of our earlier discussion, that perennial plants (equilibrium species) will be described by the scaling exponent \( \beta = 1 \), whereas annual plants (opportunistic species) will be described by \( \beta = 2/3 \).

(ii) \( \beta = (4\mu - 1)/4\mu \). The minimal value for the basal metabolic rate is also \( \beta = 2/3 \). The maximal value obtains when \( \mu = 1 \), and we have \( \beta = 3/4 \). Hence, large mammals and large birds (equilibrium species) will be described by \( \beta = 3/4 \), whereas small mammals and small birds (opportunistic species) will be described by \( \beta = 2/3 \).

6.2. The normalization coefficient

The normalizing coefficient \( z \) is given by

\[ z = \gamma \Delta p \]

where \( C \) denotes the proton conductance, \( \Delta p \) the proton motive force, and \( \gamma \) a measure of the efficiency of the nutrient transport within the organism.

The effect of proton conductance: Proton conductance is given by \( C = C_o \exp(-\Delta \tilde{p}_i/RT) \). Proton conductance is highly dependent on the degree of polyunsaturation of membrane phospholipids: the more polyunsaturated the mitochondrial membrane, the larger the proton conductance. Empirical studies indicate a large variation in phospholipid composition between and within species. Two cases of significant interest can be itemized as follows (Hulbert and Else, 2000; Hulbert, 2005; Brookes et al., 1998):

(a) Membrane bilayers of endotherms are more polyunsaturated than those of similar-sized ectotherms.
(b) Membrane bilayers of tissues of small mammals are highly polyunsaturated, while in large mammals a decreased membrane polyunsaturation with increased body size exists.

Since proton conductance is positively correlated with proton current, we can appeal to (a) and (b) and the expression for metabolic rate given by Eq. (1) to predict the following:

(I) The metabolic rate of endotherms will be greater than that of equivalent size ectotherms when compared at constant body temperature.

(II) Tissues in large mammals should have a lower in vitro metabolic rate than the homologous tissues in small mammals.

The effect of the electrochemical proton gradient: The electrochemical proton gradient is given by \( \Delta p = \Delta \tilde{p} - (2.3RT/F)\Delta \tilde{p} H \). Empirical studies of \( \Delta p \) show that this
quantity also varies between and within species. The extent of this variation among unicellular organisms, plants and animals, can be illustrated by representative values of the membrane potential $\Delta \psi$, and the concentration difference $\Delta p\mathrm{H}$, for bacteria, mitochondria and chloroplasts (Fox, 1982), see Table 2.

The apparent anomalies in the values for $\Delta p\mathrm{H}$ are due to structural differences in the three membrane systems: The inner membrane of mitochondria and bacteria contain the energy transducing proteins, resulting in negative values for $\Delta p\mathrm{H}$. In chloroplasts, however, these proteins are located in a third membrane system encapsulated within the outer membrane. The structural constraints imply that $\Delta p$ will be positive in the membrane of bacteria and mitochondria but negative for the thylakoid membrane in chloroplasts.

We can infer from the differences in absolute value for the proton motive force, $\Delta p$, between bacteria, mitochondria and chloroplasts, and the positive correlation between proton current and proton conductance, the following general prediction.

(III) Unicellular organisms, plant and animal tissues should be characterized, respectively, by increasing values for the metabolic rate when evaluated at the same temperature.

7. Empirical considerations

The predictions we have described in the preceding section specifies certain explicit relations between the metabolic activity of plants and animals, and their scaling exponents and proportionality constant. We will now consider the empirical support for these predictions.

7.1. Scaling exponents

The empirical studies of the scaling exponents which we will invoke in our analysis are the recent reviews given by Dodds et al. (2001), White and Seymour (2003) and Glazier (2005), which pertain mainly to animals, and the recent study by Reich et al. (2006) which refers to plants. These studies indicate that for animals, $\beta$ ranges from $2/3$ to $3/4$, with small animals satisfying the $2/3$ rule and larger animals the $3/4$ rule. The plants studied in Reich et al. (2005) are perennials. The scaling exponent for these organisms satisfy $\beta = 1$. These observations are all consistent with the predictions of the theory.

It is important to emphasize that the theory predicts that annual plants, since they are opportunistic species, should be described by scaling exponents $\beta = 2/3$. Hence, the theory makes an explicit prediction which can be evaluated by studies of metabolism–size relationship in green plants.

7.2. The proportionality constant

Comparative studies of the metabolic rate of organisms of equivalent body size provide a framework for evaluating predictions pertaining to the proportionality constants.

Hulbert and Else (2000) have made extensive comparative studies of the metabolic rate of mammals and reptiles of equivalent body size. These studies are consistent with the prediction that the metabolic rate of endotherms will be greater than that of equivalent size ectotherms when compared at the same body temperature. Porter and Brand (1993) have made comparative studies of the metabolic rate of homologous tissues of large and small mammals. These investigations are consistent with the prediction that tissues in large mammals should have a lower in vitro metabolic rate than homologous tissues in small mammals.

Hemmingen (1960) has drawn from empirical studies of metabolism of unicellular organisms, poikilotherms and homeotherms to produce the following equations relating basal metabolic rate, $P$, to body size, $W$:

\[ P(\text{unicellular organisms}) = 0.0176W^{0.756}, \]
\[ P(\text{poikilotherms}) = 0.144W^{0.738}, \]
\[ P(\text{homeotherms}) = 4.1044W^{0.739}. \]

A graphical description of the data is given in Fig. 3.

In this comparative study, the metabolic rate is measured in cubic centimeters of $O_2$ per hour and $W$ in grams. The data for unicellular and ectothermic organisms are corrected for $20^\circ C$ while the data for homeotherms (birds and mammals) are corrected for $25^\circ C$.

![Fig. 3. Standard metabolic rates of homeotherms, poikilotherms and unicells. Taken from Peters (1983).](image-url)
The difference in the proportionality constant for unicellular organisms, poikilotherms and homeotherms, described in Fig. 3, are consistent with the prediction that

(i) animal tissues have larger metabolic rate than unicellular organisms; (ii) homeotherms have larger metabolic rate than similar-sized ectotherms.

8. Discussion

The metabolic rate of an organism, that is, the rate at which energy is being transformed from nutrient into energy required for movement, growth and heat production, is constrained by the size of the organism and regulated by three main factors:

(a) Metabolic efficiency, the extent to which the electron transfer process is coupled to ADP phosphorylation.

(b) Membrane composition—the degree of unsaturation of the phospholipid constituents in the membrane.

(c) The structure of the circulatory network by which nutrients are transported within the cells and tissues of the organism.

This article has analysed the dependence of metabolic rate on these three factors by integrating the chemiosmotic theory of energy transduction with the methods of quantum statistics. This study, which we call smotic theory of energy transduction with the methods of quantum statistics, has given rise to a general analytic framework for explaining parameters entails that quantum metabolism provides an empirical relation between metabolic rate, \( P \), and body size, \( W \), namely

\[
P = \gamma \exp \left( \frac{-\Delta \mu}{RT} \right) \Delta p W^\beta.
\]

The expression incorporates both a scaling exponent and a proportionality constant:

The scaling exponent \( \beta \), is expressed in terms of a single parameter, the metabolic efficiency \( \mu \), the degree to which the electron transport chain is coupled to ADP phosphorylation. In the case of plants, \( \beta = (2\mu - 1)/\mu \); for animals and most unicells, \( \beta = (4\mu - 1)/\mu \).

The proportionality constant involves three elements:

(a) \( \gamma \); a measure of the efficiency of the circulatory network that transports energy and nutrients between cells and tissues within the organism network;

(b) \( C \); the proton conductance, which is given by \( C = \exp((-\Delta \mu)/RT) \), where \( \Delta \mu \) is the activation free energy and \( T \), the temperature;

(c) \( \Delta p \); the proton motive force.

The dependence of the proportionality constant on these parameters entails that quantum metabolism provides an analytical framework for explaining

(i) The large variation in scaling exponents that characterizes different organisms.

(ii) The differences in metabolic rate of ectotherms and endotherms of the same body size and the same temperature.

(iii) The differences in metabolic rate between homologous tissues of large and small organisms.

(iv) The dependence of temperature on metabolic rate.

The problem of explaining in mechanistic terms the empirical relations between metabolic rate and body size has generated a large literature, as the recent reviews in Agutter and Wheatley (2004) and Glazier (2005) indicate. A model which has been given considerable attention, partly because of the boldness of its claims, is the fractal branching network model proposed by West et al. (1997).

In view of the various claims made for the universality of the model, it is of some interest to contrast the fractal network model with quantum metabolism in order to situate their relative explanatory and predictive status.

The fractal model, in sharp contrast to quantum metabolism, deals with energy transformation at the macroscopic level. The model ignores both the effect of metabolic efficiency, and the effect of membrane composition on metabolic activity. The relation between metabolic rate and body size in the context of this model is determined primarily by the geometry of the circulatory network that transports nutrients to the different cells and tissues that constitute the organism. The model contends, that metabolism is regulated by body size and temperature, and postulates that the allometric relation is given by

\[
P = c \exp[-E/k_B T] W^{3/4},
\]

where \( E \) is an activation free energy, \( k_B \) is Boltzmann’s constant. The Boltzmann factor is said to specify how temperature affects the rate of reaction by changing the proportion of molecules with sufficient energy to surmount certain energy barriers. The quantity \( c \) is a proportionality constant.

Eq. (23) does not result from a single underlying tenet, but aggregates two classes of models. The first class, see West et al. (1997), postulates the relation

\[
P = a W^{3/4}
\]

and contends that the scaling exponent \( 3/4 \) is the result of the architecture of the circulatory networks that control the flow of metabolites within the organism. The analytical derivation of Eq. (24) has generated some controversy, see, for example, Alexander (1992), Dodds (2001), Hochachka and Somero (2002) and Makarieva et al. (2005).

The second class of models, see Gillooly et al. (2001), simply posits a Boltzmann factor

\[
\exp[-E/k_B T]
\]

to reflect the empirical fact that metabolic rate is a temperature-dependent property.

The Boltzmann factor can be considered as an attempt to offer a biological interpretation for the proportionality constant \( a \) in Eq. (24), a point which has been underscored
in the critique of Clarke and Fraser (2004). The Boltzmann factor is not a consequence of the fractal constraints which are assumed to be a fundamental property underlying metabolic activity.

It is nevertheless claimed by Brown et al. (2004) that Eq. (23), in spite of its hybrid origins, constitutes a mechanistic basis for a metabolic theory of ecology. This bold statement has been criticized by several authors on the grounds that the model does not offer a coherent mechanistic basis for the scaling rules. However, the default in mechanistic understanding, as argued in Cyr and Walker (2004), does not deter from the possible predictive capability of Eq. (23). For this reason, it is of some interest to assess the potential predictive power of the fractal network model in spite of the many reservations raised regarding its mathematical consistency and explanatory power.

In this regard, we note that Eq. (23) ignores factors such as metabolic efficiency, a parameter which regulates the scaling exponent, and membrane composition, a fundamental property of metabolic activity. The neglect of metabolic efficiency entails that the model is unable to address the following empirical observations:

(i) Large mammals are characterized by a 3/4-power scaling law, however, small mammals and many species of birds are described by a 2/3-power scaling (Dodds et al., 2001; White and Seymour, 2003).

(ii) The scaling exponents of basal metabolic rate, which reflects the lowest need of energy achieved under thermoneutral conditions, and maximal metabolic rate, the rate achieved under conditions of heavy exercise, differ (Weibel et al., 2004).

The neglect of membrane composition entails difficulty in explaining another family of basic data sets.

(i) Endotherms have a higher metabolic rate than ectotherms of the same body size and the same temperature (Hulbert and Else, 2000).

(ii) Tissues in a larger animal has a lower in vivo metabolic rate than the same homologous tissue in a smaller animal (Porter and Brand, 1993).

The inability to account for these two classes of basic data sets, in addition to the failure of the model to explain the scaling relation for plants and unicells, casts doubt on the claim that Eq. (23) may provide a basis for a metabolic theory of ecology. With respect to this claim, it is of interest to cite the observation made by Tilman et al. (2004) that many different ecological roles can be performed by organisms of similar size and temperature. These ecological roles determine coexistence, abundance and ecosystem dynamics. However, Eq. (23) is unable to account for the large differences in metabolic rate which are observed in organisms of the same body size and at the same temperature.

The allometric relation derived from quantum metabolism is given by Eq. (22). This model incorporates the fundamental factors that determine metabolic activity, namely metabolic efficiency, membrane composition and the structure of the circulatory network. Quantum metabolism explains the large variation in both the scaling exponents and the proportionality constants, and thus accounts for the large differences in metabolic rate which are observed in organisms of the same body size and at the same temperature. Moreover, Eq. (22) subsumes Eq. (23).

In models which pertain to metabolism in animal organisms, the former reduces to the latter when the metabolic efficiency \( \mu = 1 \) and when the proton motive force \( \Delta p = 1 \).

The derivation of Eq. (22) from the molecular dynamics of electrons and protons, its predictive range and the fundamental nature of the explanation it provides suggest that quantum metabolism, rather than the fractal network model, may have a stronger claim as providing a basis for a metabolic theory of ecology.

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