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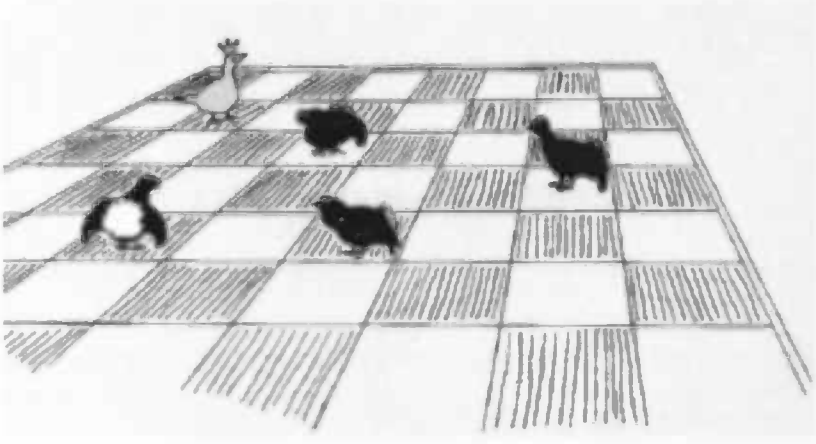
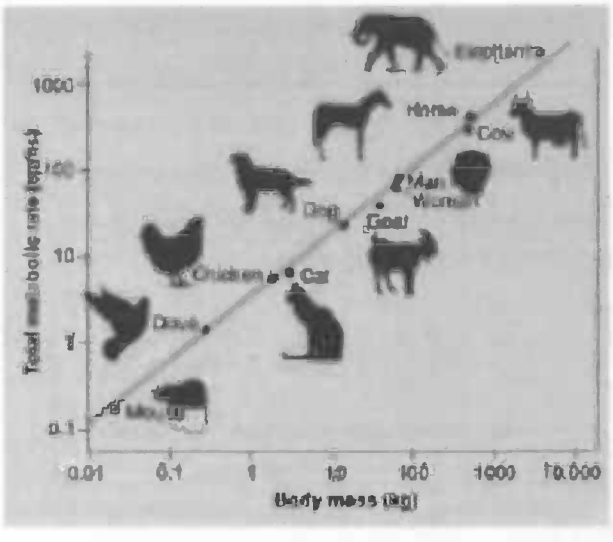
LIFE HISTORY APPROACH FOR ALLOMETRIC SCALING AND GROWTH





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LIFE HISTORY APPROACH FOR ALLOMETRIC SCALING AND GROWTH

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Introduction

During the development of this project, models for ontogenetic growth were compiled and analyzed in the light of a more general model that share rescaling laws. It has been proposed that such models, apparently, share an invariant allometric scaling exponent. Therefore, I tried to find out whether this allometric scaling exponent parameter is related to growth rate and limiting size or size at maturity. As a life history trait, it might be constrained by trade offs, instead of being an "universal invariant" as proposed in a model by West, Brown and Enquist (2001, hereafter called WBE's model) in the Metabolic Theory in Ecology.

1. Allometric Scaling Law

Size may provide indications about differences between species regarding to its ecology, reproductive activities, evolutionary progress, development etc (Bonner, 1965). Allometry provides one way to understand and compare those differences, and refers to the structural or functional change with growth; such changes can be dimensional (how a part of an organism is related with its total body size) or physiological (how is the respiration rate in a juvenile with respect to an adult). In addition, scaling is a transformation that allows us to quantify the consequences of those changes and allows us to do comparisons within and among species. According to Calder (1984), almost all characteristics in organisms vary predictably with body size. It has been proposed, that different aspects of organisms such as physiology, growth and life history, which vary from plants to animals, are matter of scaling. This is what we nowadays call allometric scaling laws.

a. b.

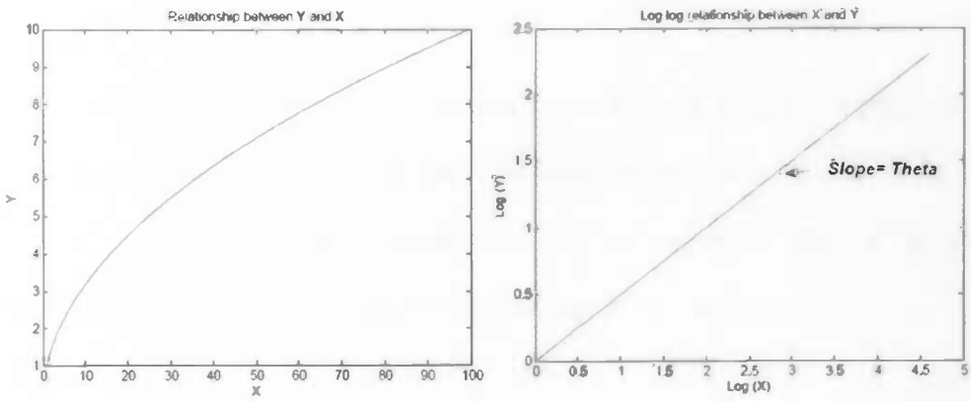


Figure 1 (a) Relationship between Y and X for $1 > \theta > 0$. **(b)** Log-log plot of the relationship between Y and X where the slope of the line is the theta value (scaling exponent).

The first description relating growth and allometry was by Huxley in 1932. He stated that the size of a certain part Y, is related to some standard, X (namely the whole body, the rest of the body without Y, or a standard part chosen by convenience), according to the formula

$$Y = bX^{\theta} \tag{1.1}$$

Where b and θ are constants. The constant b represents the value of Y when $X=1$. The exponent θ is considered the proportion of specific growth rates of Y and X (Huxley, 1950), in a log-log scale (Fig. 1a). Therefore, this relationship is better understood when logarithm is taken on both sides of the equation, thus obtaining a straight line where the slope corresponds to the value of the exponent θ (Fig. 1b)

$$log(Y) = log(b) + \theta log(X) \tag{1.2}$$

Throughout the course of the years, assuming that there is a single "universal" value for θ, its estimated value has changed across different studies. For example, von Bertalanffy

(1957) studied two fundamental aspects of living organisms: metabolic rate and growth. His original equation takes the following form:

$$Y = bX^{2/3} \tag{1.3}$$

Where Y is metabolic rate, b is a constant and X is body weight. He proposed that the value of θ should be *2/3* based on the relationship between area/volume of the organism according to the following explanation. If the organism can be geometrically approximated as a sphere, its area *(A)* and volume *(V)* are defined by:

$$A = 4\pi r^2 \tag{1.4}$$

$$V = \frac{4}{3}\pi r^3 \tag{1.5}$$

Where r is the mean radius. It is known that volume multiplied by density is proportional to weight; therefore, weight X is proportional to volume. From equation (1.5) we get volume is of the order of r^3, hence the weight is also of the order of r^3. Solving for r, we get that $r \sim X^{1/3}$, hence $Y \sim X^{2/3}$.

On the other hand, the relationship between metabolic rate and body size is based on the surface rule, which states that the heat output takes place through the body surface. Hence, the metabolic rate must be proportional in the same amount to compensate for the heat loss. Von Bertalanffy's model aimed to show connections between metabolism and growth. He stated that animal growth is the result of a counteraction of synthesis and destruction processes. For example, anabolism and catabolism of the building material of the body. Growth can be achieved only if the rate of building up is higher than the rate of breaking down. A simple model describing this process is:

$$\frac{dX}{dt} = aX^{2/3} - \rho X \tag{1.6}$$

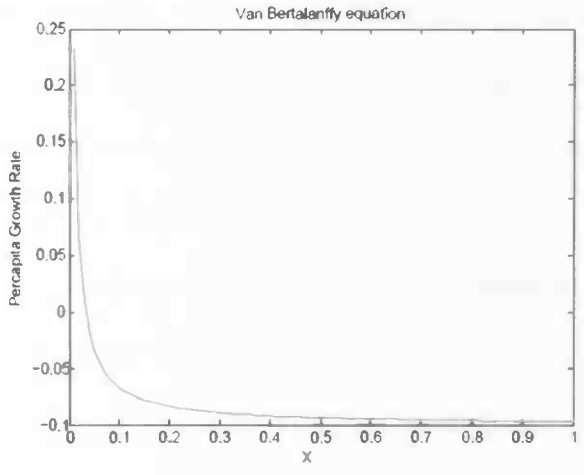


Figure 2. The per capita growth rate for the von Bertalanffy equation initially decreases very fast and then asymptotically slows until vanishing

Here the change of body weight X is given by the difference between the processes of building up and breaking down; a and ρ are constants of anabolism and catabolism respectively. The exponent, as explained before, reflects the geometrical constraints of the surface/volume ratio (von Bertalanffy, 1957). For convenience it is possible to rewrite the equation (1.6) in the following form:

$$\frac{dX}{dt} = -\rho X\left[1-\left(\frac{X}{K}\right)^{-1/3}\right] \tag{1.7}$$

K is limiting size defined as a ratio between anabolic and catabolic rates $K=(a/\rho)^3$.

Afterwards, in 1997, WBE with a mechanistic model gave a new interpretation to the parameter θ in equation (1.1). Here Y is a dependent variable, which can be metabolic rate, developmental time, population growth rate etc., and it is correlated with the body mass X, through two coefficients: b, that is a constant characteristic for each organism, and θ that is the allometric (scaling) exponent:

$$Y = bX^{3/4} \tag{1.8}$$

The underling rules for this value of $\theta=3/4$, according to WBE's model are based on the transport system of the organism, that branches to supply materials to all parts of it. Three assumptions are made for the derivation of their allometric law. First, that a fractal branching network fills the space. Second, the last branch level should be size invariant. And third, the energy to supply all materials through the entire organism must be minimized (West. *et al.*, 1997).

Subsequently, West *et al.*, (2001) used the fractal network model to describe ontogenetic growth. They started from the basic principle for allocation of metabolic energy between maintenance of the existing tissue and the creation of new one. This is equivalent to the following mathematical expression:

$$\frac{dX}{dt} = \left(\frac{X_c}{E_c}\right)B - \left(\frac{B_c}{E_c}\right)X \qquad (1.9)$$

Here X is body size, X_c is the mass of a cell, E_c is the energy to create a new cell, $B=B_oX^{3/4}$ where B_o is a constant specific for a taxon, and B_c is metabolic rate of the cell. Calling $a=(X_c/E_c)$ and $\rho=(B_c/E_c)$, then rewriting we get,

$$\frac{dX}{dt} = aX^{3/4} - \rho X \qquad (1.10)$$

Hence, the ¾ exponent describes the overall allometric growth of X from birth to reproductive maturity and it is related to the scaling of total terminal units in the network. The network constrains the total number of supply units to scale differently from the total number of cells supplied. Therefore an imbalance between supply and demand is generated, which at the end limits growth.

Since maximal size is given by $dX/dt = 0$

$$K = \left(a/b\right)^4 \tag{1.11}$$

Then, equation (1.10) can be written as,

$$\frac{dX}{dt} = -\rho X\left[1-\left(\frac{X}{K}\right)^{1/4}\right] \tag{1.12}$$

Where $\rho=(B_o/E_c)$, K is limiting size and X is the body mass which can be interpreted as in equation (1.7). Integrating equation (1.12) taking X_0 as mass at birth $(t=0)$, and rescaling mass with respect to K, and time with respect to ρ we get, (West. *et al.*, 2001):

$$\left(\frac{X}{K}\right)^{1/4} = 1-\left[1-\left(\frac{X_0}{K}\right)^{1/4}\right]e^{-\rho t/4X^{1/4}} \tag{1.13}$$

With this equation, they made a plot of the dimensionless mass ratio $r = 1-R = (X/K)^{1/4}$ vs. dimensionless time variable $\tau = \left(\rho t/4X^{1/4}\right) - \ln\left[1-(X_0/K)^{1/4}\right]$ for 13 different species. In this way, they claim that ontogeny is determined by the same scaling exponent of metabolism because they rescaled the fitted curves assuming θ = ¾ perfectly to the universal invariant $r=e^{-\tau}$.

The ¾ scaling exponent theory has been supported by empirical data in many studies. The evidences for the value of ¾ come from studies in both animals and plants. Charnov (1993) studied characteristics that do not change in the life history of animals and plants that lead to the same exponent ¾. Enquist *et al.* (1999) and Price *et al.* (2006) studied the allometric scaling in plants. They reassumed the ¾ exponent in photosynthetic area in plants with minimal branching as well as in vascular plants. However, the morphological

dimension (height, spread, etc) in plants with minimal branching scales with different exponents from those with fractal-like external branching. (Enquist *et al.*, 1999; Price and Enquist, 2006).

Savage *et al.* (2004) analyzed a large database of basal metabolic rate, field metabolic rate, and maximal metabolic rate in different species of mammals. These are supposed to scale to the power of ¾ with body size. Although in some cases deviations from this exponent were found, they were attributed to certain effects between elevated metabolic rates and measurement errors in body masses in some species (Savage *et al.*, 2004).

Etienne *et al.* (2006) (Hereafter called EAO's) made a redesign of the WBE's model. In that study, they make a formulation deriving the scaling exponent in a more straightforward and clear way. WBE's model and EAO's model differ in two ways: WBE's suggest that in the fractal-like network, the cross-sectional area preservation combined with preservation of $N_k l_k^3$ (N identical pipes at k^{th}= level of branching with length l_k) optimizes the efficiency of the network. That is why WBE argue "that the allometric scaling with the exponent *3/4* reflects that the organisms have evolved so that the energy required to sustain them is minimized." Instead, EAO's model do not use an energy minimization principle to support that the number of vessels in the last branch does not depend on the body size, but keep the area preservation principle. In addition, EAO's model does not require a fractal-like network in order to obtain the scaling exponent *3/4*. Based on more general assumptions than stated by WBE, they leave open the possibility for incorporating different assumptions and finding the corresponding scaling exponent for a given system (Etienne *et al.*, 2006).

Despite the data supporting the WBE's model theory, many researchers are sceptical about the invariance of these exponents since they have found evidences against the theory. It is the case, especially when patterns following the scaling law are examined in a narrower range, both within particular taxonomic groups or ecosystems, and even across taxonomic groups. Dodds *et al.* (2001) made a re-evaluation of the scaling hypothesis, using the same equation (1.1). They re-examined empirical data available for metabolic rates of endotherms as well as the theoretical justifications for $\theta = 2/3$. They constructed two types of hypothesis tests to determine whether $\theta = 2/3$ or $\theta = 3/4$ should be rejected by the available data. By analyzing the correlations of the residuals from the best fitting line, it was possible to determine quantitatively which values of θ are compatible with the data. They did not find, however, compelling evidence of a simple scaling law for metabolic rate, and if it were to exist, they did not find convincing evidence why the exponent should be $\theta = 3/4$ (Dodds *et al.*, 2001).

Later on, in a review of the WBE's model, it was pointed out that according to it, animals' body structure cannot have a broad range of sizes, because in large animals the blood volume would exceed the body volume. Additionally, many features of the plant vascular system, insect tracheal system, vertebrate lung, or vertebrate cardiovascular system, do not follow the assumptions of the model. They pinpoint the flaw in the model regarding the size- invariance of the final branch. The model assumes that blood flow should be proportional to the metabolic rate. Because metabolic rate is proportional to the total number of capillaries, it must scale with the exponent θ. According to Kozlowski & Konarzewski (2004): "Unless the metabolic rate exponent equals one, the number of

capillaries must scale allometrically to satisfy the assumption that blood flow through all capillaries should be proportional to the metabolic rate. At the same time the capillaries must scale isometrically to make possible to compose a body with spheres having size-invariant radii, which is required for a space-filling fractal" (Kozlowski and Konarzewski, 2004; Kozlowski and Konarzewski, 2005)

In 2006, a study by Glazier on metabolic rate of pelagic animals found that during ontogeny, their size often scales isometrically with the body mass, instead of allometrically. This pattern was found in five different phyla. When comparing benthic species with pelagic species within phyla, benthic species scale with an exponent of 0.744 whereas pelagic species was 0.947. The same pattern of isometric scaling also can be seen when comparing pelagic larvae and benthic adults. There are two possible theories to explain the phenomenon; 1) High energy costs for swimming and stay floating, or 2) high energy costs for rapid growth rate and reproduction in response to selection pressure (predation) in open water.

These finding suggest that metabolic scaling represents an adaptative strategy that has evolved in the context of multiple physical, chemical and ecological constraints, rather than a universal physiological structure resulting in an invariant law with $\theta=3/4$ (Glazier, 2006).

2. Growth rate and models

Size depends on the ability to gather and process food, the proportion of acquired energy used for maintenance and the allocation of surplus energy to growth and reproduction which changes dynamically through (Kozlowski and Gawelczyk, 2002)

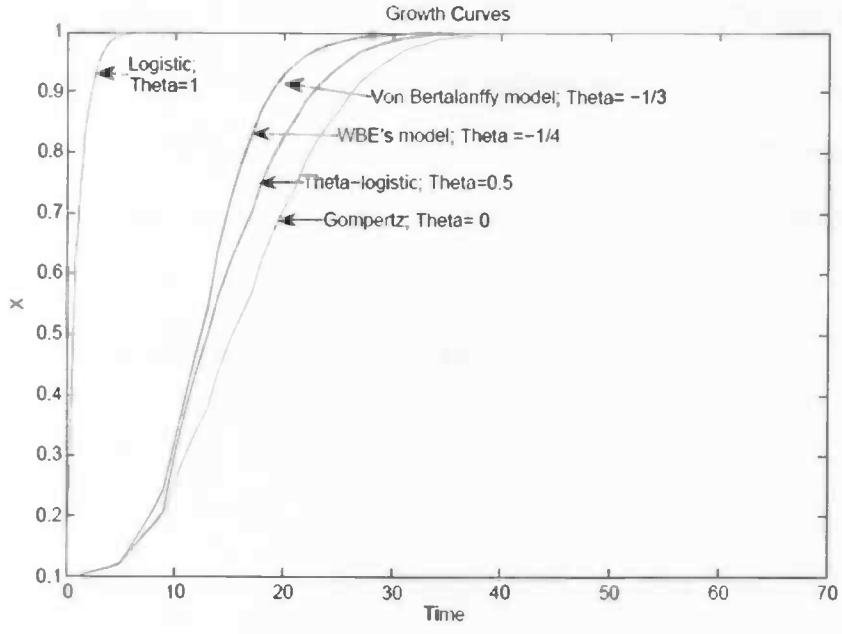


Figure 3. Growth curves: Here von Bertalanffy's model and WBE's model follow almost exactly the same trend of the curve regardless of the different values for theta, as well as the other curves that follow the same shape.

On the other hand, growth of an organism is a multiplicative process in which cell number and cell volume increase. Under unlimited source of nutrition conditions, growth proceeds in an exponential shape. However, in reality this shape is continuously deadened as size and age increase. Growth rate decreases greatly when the maturity (first reproduction event) starts (Brett, 1979). There are several models to describe the growth rate, not only in individual organisms but also in populations (Fig. 3).

2.1. General Model for Growth

Because the growth function is a very important issue in biology, and it has been described in many ways (Henle *et al.*, 2004), a generalization has been proposed by de Vladar (2006).

This generalization that define the model has the peculiarity that when changing the values of the parameters ρ, θ we can recreate the curves of the growth models like exponential, logistic, θ-logistic, Gompertz, Von Bertalanffy, potential growth, among others. Because of the generalization, all growth functions can be rescaled in the same way.

One class of solutions resembles von Bertalanffy and WBE equations.

$$\frac{dX}{dt} = -\rho X\left(1-\left(\frac{X}{K}\right)^{-\theta}\right) \tag{1.14}$$

Where ρ is a term related with catabolic rate, and here I interpret it as cellular death rate, θ is the allometric exponent.

Suppose that an individual achieves the maximal size at K. Integrating equation (1.14), and rearranging we get:

$$\left(\frac{X}{K}\right)^{-\theta} = 1-\left[\left(\frac{X_{(0)}}{K}\right)^{-\theta}-1\right]e^{-\rho t} \tag{1.15}$$

Defining the new-scaled variables as:

$$\begin{aligned} \chi &= \left(\frac{X}{K}\right)^{-\theta} \\ \tau &= \rho t - \log\left[\left(\frac{X}{K}\right)^{-\theta}-1\right] \end{aligned} \tag{1.16}$$

we find that all the equations of growth named above, obey a general scaling law (Fig. 4). And the expression is valid for all combinations of values of the parameters θ and ρ (de Vladar, 2006). Therefore, following the re-definitions of the equation (1.16) and substituting in the second part of equation (1.16) we obtain in the universal curve:

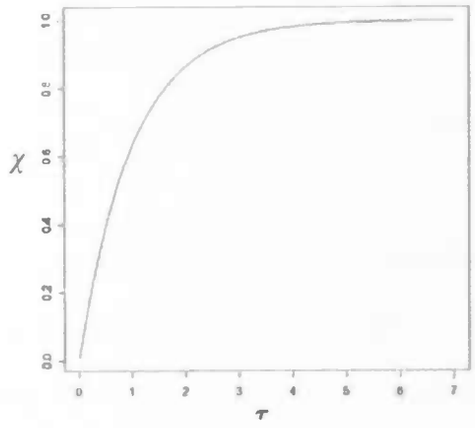


Figure 4. Universal curve following the equation(1.17). χ is the rescaled mass and τ is the rescaled time as indicate equation (1.16). Irrespective of the special choices of ρ, θ and K, the curve shows a monotonically increase saturing at X=1

$$\chi = 1 - e^{-\tau} \tag{1.17}$$

The scaling variables proposed by WBE's model are special cases of equation (1.16) when we use θ=*-1/4*. The same holds for the von Bertalanffy equation when we use θ=*-1/3*. Equation is plotted in Fig 4.

3. Life history theory

The different organisms living on the Earth manage to survive and reproduce thanks to different strategies. Those strategies consist of distinct relationships between age, size, mortality, reproductive performance etc. Life history theory deals directly with the fundamental principles of resource and energy allocation to determine how these relationships are. This theory allows understanding how natural selection works. In order to allow natural selection to act, two conditions are necessary: (1) Genotypic factor; the heritable variability for a trait determines whether there will be a response to selection. (2) Phenotypic factor; that determines fitness and which may vary among individuals.

This variation in fitness among individuals allows evolution, and fitness to increase. The evolution of life history traits and their variabilities determine the interaction dynamics of a population. The theory at the same time, analyses the causes for differences in fitness among life history invariants, and it predicts the phenotype at equilibrium.

Life history theory predicts that individuals of a species are able to make limited shifts in reproductive strategies in response to the prevailing environments. Depending on abundance of resources and possible individual lifetime, individuals can, either consciously or not, shift their reproductive strategy in one direction or the other, in order to take advantage of the available resources, or to compensate for resource scarcity or uncertainty (Brommer, 2000; Stearns, 1992).

This theory formulates its arguments by constraining relationships of traits like birth, age and size at maturity, number and size of offspring, growth, reproductive investment, length of life, etc. Such traits combine to determine individual fitness and reflect different allocation of resources to competing life functions like growth, maintenance, and reproduction. The resources available in a particular environment are finite for any organism. Time, effort, and energy used for one purpose diminish the time, effort, and energy potentially used for another. The connection of constraining relationships and the allocation of resources involves trade-offs that counterbalance both to get the better outcome (Stearns, 1992).

Originally, this theory was focused on reproductive traits. Lately, the role of growth in life history theory has become more important. By definition, the optimal life history

strategy is the one that maximizes lifetime reproduction and it is determined at the same time by maximizing age-specific survival and reproduction. For most organisms, though, size determines both: survival and fecundity. The growth rate defines the relationship between age and size. Hence, it is an important factor to take into consideration in life history theory. In order to understand why specific growth rates evolve, we also need to understand which traits trade-off with growth rate and how they interact under given selective pressures, with respect to size and time constrains (Arendt, 1997).

The study of life history strategies and their optimization often refer to measurement of fitness. Two fitness measures are r and R_0. Each one of these two measures is defined as exponential growth rate on a continuous time basis(r), and lifetime reproductive success in discrete time (R_0) (Mylius and Diekmann, 1995).

The exponential growth rate in the discrete time basis:

$$X_{(t+1)} = r_{(t)} X_{(t)}. \tag{1.18}$$

If there are two populations with $r_1 < r_2$, selection will favour the one with the greater value of r because it would grow much faster than the one with small value of r. Therefore, certain value to r_{max} should exist, which is an optimal strategy that cannot be displaced by any other mutant. This is what is called Evolutionarily Stable State (ESS).

On the other hand, as soon as some density regulation mechanisms in the population are taken into account it is required that $r=1$, in order to get $X_{(t+1)} = X_{(t)}$ which means that the population will have the same value as in the time before.

The other fitness measure,R_0 is a function of survival *(S)* and fecundity *(l)*. The total lifetime reproductive success in the population is given by:

$$R_0 = \sum_{x=1}^{\infty} S_{(x)} l_{(x)} \tag{1.19}$$

and R_0 =1 when the population has reached the carrying capacity. Maximizing R_0 is a way to get an ESS between the life history traits (for example survival and fecundity), which are functions or other underlying relationships of traits as well. The value where R_0 is optimal varies according to the traits that trade-off (Bulmer, 1994; Charnov, 1993; Mylius and Diekmann, 1995)

In order to explain variation in lifetime reproductive success across the species, several models have been employed, using a set of assumptions like growth, fecundity, mortality etc. Some authors reject the idea to use of growth equations to model this fitness since such growth equations imply certain resource allocation in different stages of life (Fig. 5, (Charnov, 1993; Day and Taylor, 1997).

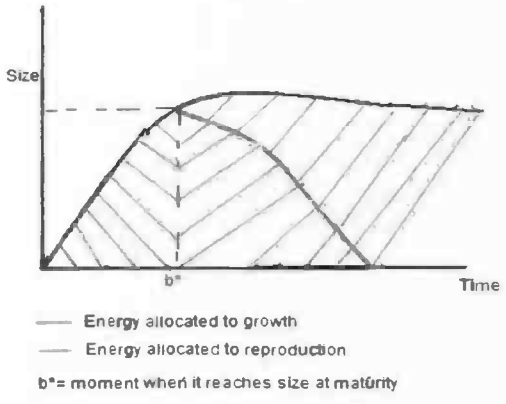


Figure 5. Growth curve implying energy allocated to different traits. (---) Energy allocated to growth during first stages of life. When size at maturity b^*, is reached, (---) energy is reallocated to reproduction, and every time less to growth.

4. Research Questions

The current debate about the invariance of the scaling law, its use in ecology, ontogenetic growth and other fields of biology, has motivated me to find the answer to the following questions: **Is the scaling parameter really a universal exponent of the allometric scaling law with respect to growth?** A second research question to be addressed is whether **the value of this exponent is really ¾ as proposed in the Metabolic Theory, or if it is a "free parameter" that can tentatively be a product of life history optimization?**

A third question to be addressed is: **Are there correlations implying trade offs between the parameters describing growth (namely, θ, ρ and K)?** This could imply that resource or energy allocation and/or physiological or another constrains, are leading to an ESS perhaps predicting some kind of invariance between θ and ρ.

5. Methodology

5.1. Simulations

In order to answer these questions above-mentioned three analyses were performed.

1. With a bootstrapping technique, I measured the robustness of the universality assumptions, employing the following algorithm:

I. A set of growth curves X_θ were generated with a randomly selected value of θ from a uniform distribution in the interval (0, 1), and randomly selected values of ρ and K from a gamma distribution $\Gamma(1,3)$ for both parameters.

II. The simulated growth function was rescaled, with respect to a universal exponent θ_u. Calling this function χ_θ, the distance between χ_θ and χ_U: $r^2 = \sum_1^n (\chi_U - \chi_\theta)^2$ was calculated, where χ_U is the invariant curve (1.17), resulting from the proper scaling with the theta value selected in step 1.

III. A simulation of 10000 repetitions was run from steps I to III rescaling systematically to different hypothetical exponents, and calculated the expected average deviation $\bar{r}^2$ was calculated, together with the confidence interval for r.

The gamma distribution for K value was chosen because many studies support the idea of the body size distribution is usually skewed to right. Such studies include: (Caughley, 1987; Gardezi and da Silva, 1999; Kozlowski and Gawelczyk, 2002; Maurer *et al.*, 1992), among others. According to optimization models, each species has a separate optimum due to energetic properties and mortality rates, and the distributions of the body size reflect the distributions of such optimal values. The gamma distribution for ρ was chosen

since it is known that there is a proportional relationship between this parameter and maximum size in an organism (Charnov, 1993; Stearns, 1992) , which in my case is represented by the K value. I assumed a uniform distribution for the parameter θ because there is no information about the real distribution of this parameter. Hence, to avoid biases in the calculations I assumed this uninformative distribution.

5.2. Estimating parameters from data

In order to determine empirically the distributions of parameters ρ, θ:

1. I re-analyzed the data used by West *et al.* (2001) and gathered further data from several published sources (Hobbs *et al.*, 2007; Leigh, 1992; Ojanguren and Brana, 2003; Sakata and Setoyama, 1997) The data was found using Web of Science electronic database using the keywords ("Ontogenetic growth", "Body Mass vs Time", "Body Mass vs Age", "Size" and "Time") and then contacting the authors of the publications to request the data. Some of them provided unpublished data.

2. I estimated the parameters from data using the function NLS (Nonlinear Least squares) in R program.

3. The deviation of each curve from the collected data was compared with the theoretical expectations determined in point 1.

4. Furthermore, I empirically studied the correlations between the estimated parameters ρ, θ, K in order to identify whether there were patterns indicative of trade-offs. Since each of the fitted curves gave estimated values $\hat{\rho}, \hat{\theta}, \hat{K}$ I got as many estimated points as curves used in the previous analysis.

Superfamily	Family	Species
Ceboidae	Cebinae Calliniconinae	*Callicebus moloch, Cebus paella* *Callimico goeldi*
Cercopithecidae	Cercopithecinae	*Erytrocebus patas, Cercocebus albigena, Cercopithecus ascanis, Cercocebus cephus, Cercopithecus mitis, Ceropithecus hamlyni, Cercopithecus neglectus, Cercopithecus torquatus athys, Cercopithecus torquatus torquatus Cercopithecus aethiops, Macaca artoides, Macaca fuscata.*
	Colobinae	*Colobus guereza*
Hominoidea	Hylobatidae	*Hylobates syndactilus, Hylobates lar, Hylobates moloch*
	Ponginae	*Pan panicus, Pan troglodites, Gorilla gorilla*

Table 1 Primates taxonomy used to group the data collected (Myers.P, 1999).

5.2.1. Gathering data

I wrote to 12 authors in order to collect data, out of which 6 kindly provided the information. All the individuals of the species that I analyzed were captive animals. The collected data includes: 24 species of primates, 4 species of fish, 3 species of birds, 2 species of rat, 1 species of fungus, 1 species of rabbit, 1 species of pig, 1 species of cow, 1 species of shrew, 1 species of shrimp, 1 species of guinea pig. The number of individuals per specie varies between 1 and 21. In total I analyzed data from 178 individuals.

I grouped the primate species taxonomically following Myers (1999) (Table 1):

5.2.2. Estimation of the parameters

The estimation of the parameters ρ, θ was performance based on Nonlinear Least Squares (NLS). Due to its sensitivity to initial conditions and the non linearity of the equation used, it was necessary to adjust the method in special ways, described as follows.

Considering the following general equation for growth equation (1.14):

The solution of equation (1.14) was used to estimate the parameters θ and ρ with the NLS:

$$X = K\left(1 - e^{-\rho t + B}\right)^{1/\theta} \tag{1.20}$$

Where X is the size, X_0 is the initial size in grams (g) , K is the maximum size, t is time in days, ρ is the cell death rate which has units of time^{-1}, and it indicates how fast the asymptote is approached. B is get as a function of the other parameters: I constrained B in this way in order to make easier the estimation to the NLS model, and is defined as:

$$B = \left(\left[\frac{X_0}{K}\right]^{\theta} - 1\right) \tag{1.21}$$

K was estimated selecting the last point of size if it was the maximal or from the average of the last 2 or 3 points.

5.2.3. Nested Estimation (NEM)

This method is based on the alternated estimation of parameters. First I fixed the parameter ρ, to estimate the parameter θ, and then I used the value obtained in this step as a fixed parameter to estimate the parameter ρ both using equation (1.20). Trying to

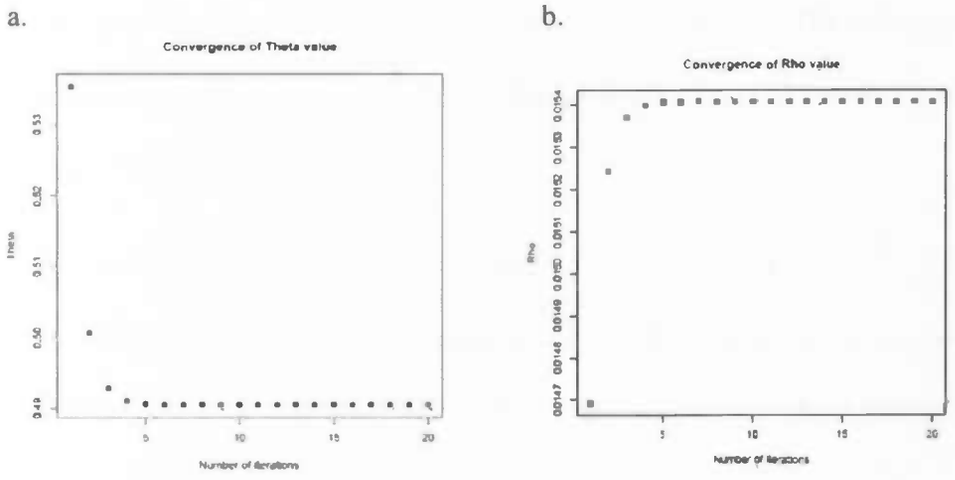


Figure 6. Estimation of (a) θ and (b) ρ, at each step in the iteration. The estimates converged in less than 10 points in several monitored cases, like this arbitrary example of Table 1.

estimate θ directly from equation (1.20) did not work, therefore I employed a transformation to facilitate the convergence in the NLS estimation.

If I substitute $X=1/y$ in equation (1.20) obtain

$$\frac{1}{y}\frac{dy}{dt} = \rho\left(1-\left(Ky\right)^{\theta}\right) \tag{1.22}$$

From the data it is possible to calculate the left hand side of the equation, where X is the data, that is the mass of the individual at time t, then,

$$\frac{1}{y}\frac{\Delta y}{\Delta t} \simeq \left(\frac{1}{X_{t-1}} - \frac{1}{X_t}\right)X_t \tag{1.23}$$

In the right hand side of the equation (1.22), fixing the parameters ρ and K using the NLS, we can estimate the parameter θ, and use this estimation as an initial condition to estimate parameter ρ, this time directly from equation (1.20).

Iterating this procedure gives a robust estimation of $\hat{\theta}$ and $\hat{\rho}$. The convergence of the estimators is linear, and fast (less than 10 steps in all successful cases). I made, for the sake of safety, 20 iterations (Fig 6).

5.2.4. Log-log Estimation Method (LEM)

This method is based on a Taylor's expansion at the first part of the curve equation (1.20) Then I performed a Log transform of first stages of growth, that is the data until the inflexion point in the curve.

When time is small in equation (1.20) is possible to expand the equation and get:

$$X \approx K\left[1-(1-\rho t)e^{B}\right]^{1/\theta} \approx K\left(\rho t e^{B}\right)^{1/\theta}. \tag{1.24}$$

Taking the logarithm in both sides

$$log\, X = log[\, K\left(\rho t e^{B}\right)^{1/\theta}], \tag{1.25}$$

and rearranging:

$$\log(X) = A + \frac{1}{\theta}\log(t) \tag{1.26}$$

Where $A = \log(Ke^{B/\theta}\rho^{1/\theta})$

Then using a linear model I estimate the value of θ, as the inverse of the slope. Therefore I include this theta value as a fixed parameter with equation (1.20) and estimated the parameter ρ, using NLS. This procedure does not require iterated estimations as in NEM.

a. b.

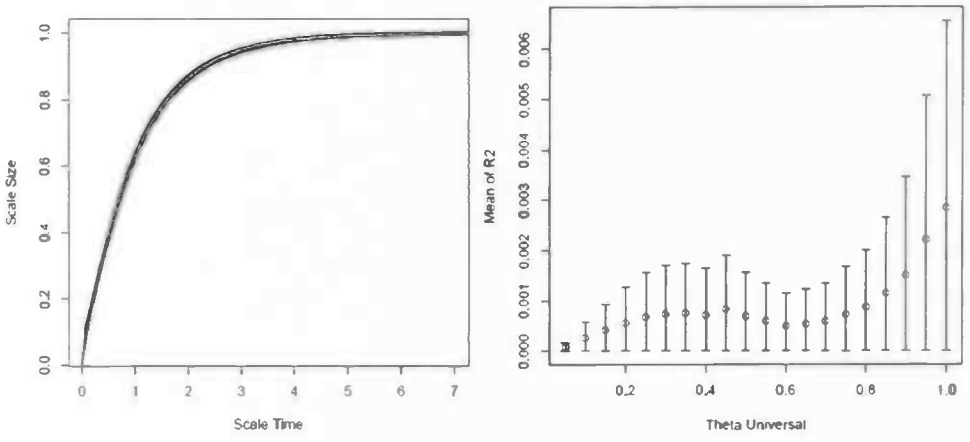


Figure 7. (a) 100000 rescaled curves with a universal θ exponent chosen from an interval [0,1]. ——: Universal curve. **(b)** Plot of the mean deviation with respect to hypothetical values of rescaling, the bars indicate the 95% confidence interval

6. Results

6.1. From simulation

I ran a simulation where a total of 100000 growth curves were created in total (Fig.7a). I generated a set of curves with random choices of θ, and then I wanted to verify whether there is a better fit with a specific exponent, so I allowed this "universal" exponent to vary in an interval [0,1], rescaling the same set of generated curves and calculated the mean deviation and the confidence interval (Fig. 7b).

In order to see whether the methods induced some kind of correlation between the parameters, I ran another shorter simulation with the purpose of comparing the two algorithms used to estimate the parameters, namely LEM and NEM. As show in Fig. 8, the LEM induces a correlation between the parameters ρ, θ, whereas the NEM does not induce any kind of correlation.

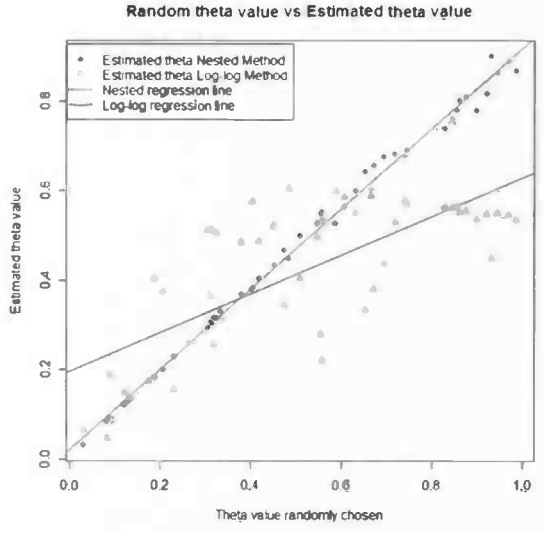


Figure 8. Correlation between the theta value ramdomly chosen and the theta value estimated with both methods. NEM = ■, LEM= □ The trend lines and the dispersion around it show that NEM is more accurate and precise that LEM. LEM is reliable only for very small values $\theta \in (0, 0.4)$.

According to simulations, the NEM estimations are more accurate and precise than LEM. Fig. 8 shows how accurate and precise is the NEM since its regression line has a statistically significant slope of 1. In contrast, the LEM even though is accurate for small theta values, seems to overestimate values in the range 0.4 until 0.6 and underestimate big values of theta.

6.2. Data Analysis

All the collected data was analyzed with the NEM. Those data which either could not be estimated because the NLS did not converge, or had a non significant estimation were re-analyzed with the LEM.

a. b.

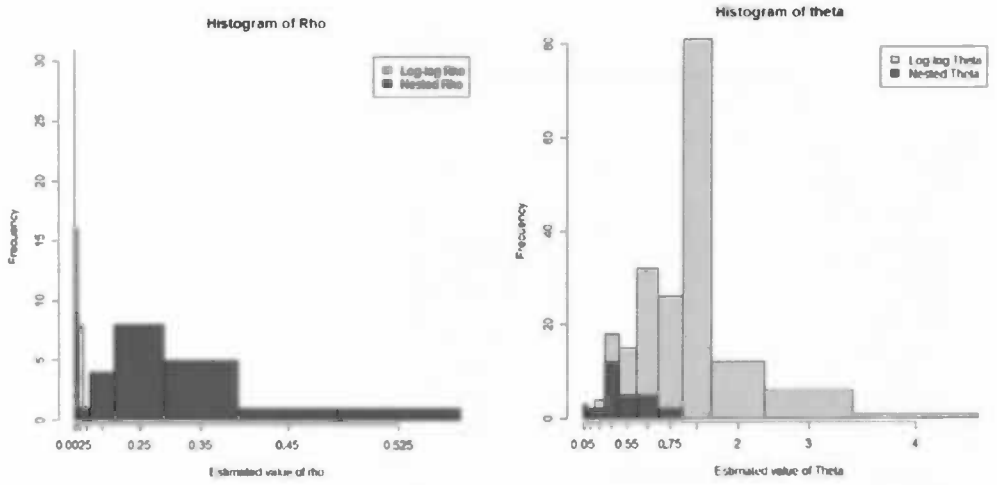


Figure 9. Histograms of the estimated parameters from data. (a) Histogram of Rho with the two estimation methods, mean (NEM Rho= 0.011891, LEM Rho=0.00815898). (b) Histogram of Theta with the two estimation methods, mean (NEM theta=0.46352, LEM theta=1.030459).
(■ = NEM, ■ = LEM)

a. b.

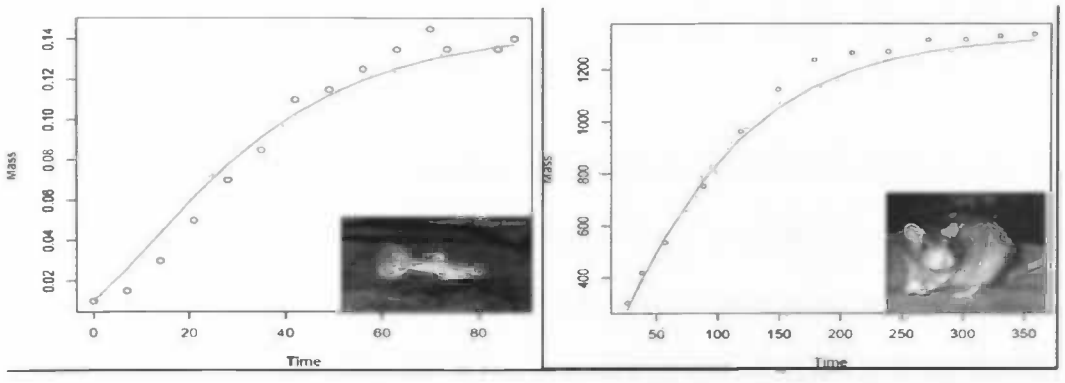
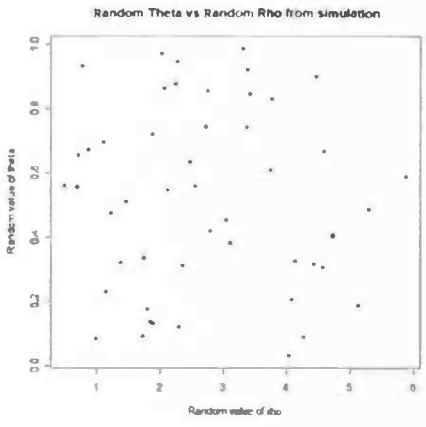
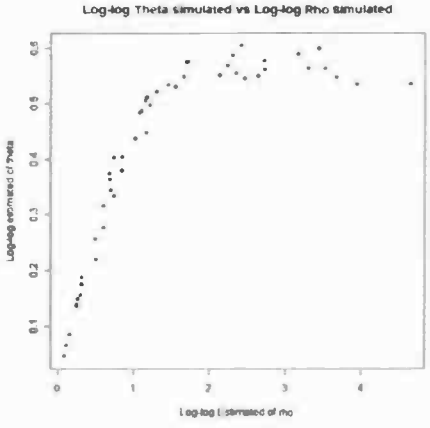


Figure 10. Examples of the estimation in two species. (a) Estimation for *Lebiscus reticulatus* (Guppy) using NEM. (b) Estimation for *Rattus novergicus* (Rat) using LEM.

a.



b.



c.

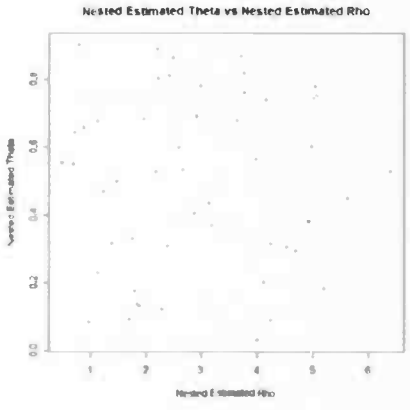


Figure 11. The parameters θ and ρ from simulations. **(a)** The parameters, randomly, chosen, as explained in section 5.1., did not have any correlation at the beginning of the simulation.**(b)** After the estimation a correlation between the parameters $\hat{\theta}, \hat{\rho}$ is induced by LEM. **(c)** After the estimation any correlation between the parameters $\hat{\theta}, \hat{\rho}$ is induced by NEM.

When both estimations are compared (Fig. 10), seems to be that LEM fits better the data in the first part of the curve, however, from Fig. 9 it is possible to say only when the value of theta was small the method provided a good estimation, but there was an overestimation when the value was above 0.4, and an underestimation when the value was above 0.6.

This could be due to the fact that the method was defined as a Taylor's expansion taking only the linear part to estimate the parameter, but might be possible that the high order terms of the polynomial had a significant effect and therefore deviated the estimation.

Also Fig 11., shows the correlation between parameters $\hat{\theta}, \hat{\rho}$ induced by the method. Because of the bias in the LEM and the accuracy of the NEM the discussion and conclusion are drawn from the NEM.

6.2.1. Correlations between parameters: ρ, θ and K

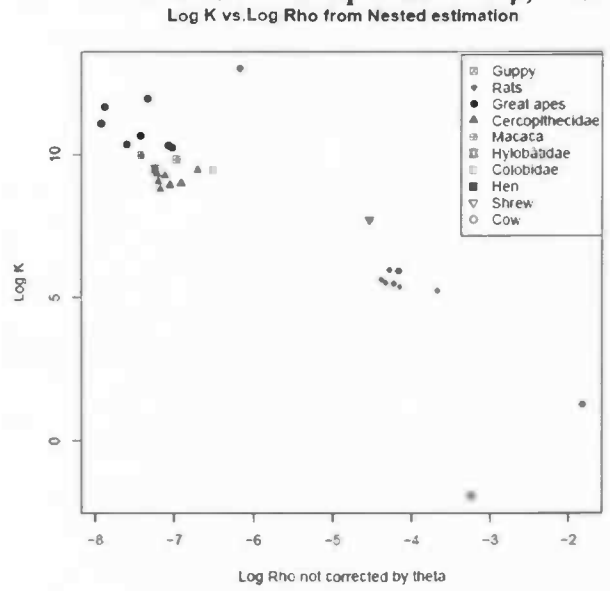


Figure 12. Estimated value of $\hat{\rho}$ and $\hat{K}$. There is an inverse relationship between these two parameters that could be interpreted in that the smaller animals reach maximal size than bigger animals. The plot is in log-log scale.

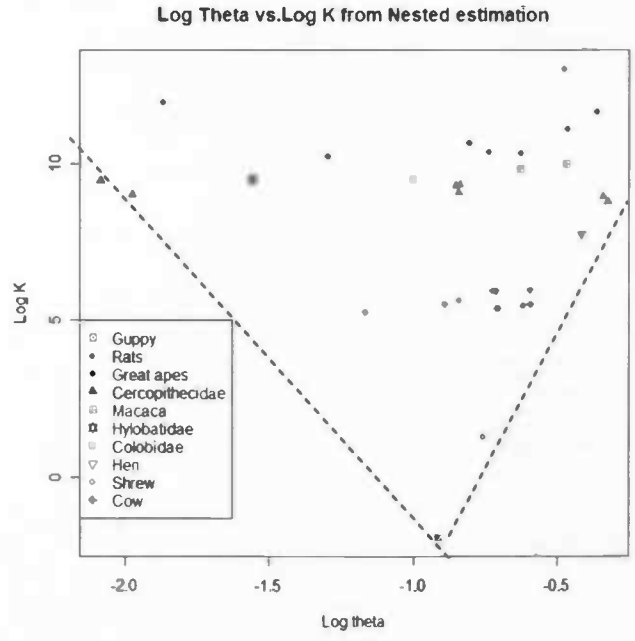


Figure 13. Estimated value of θ and K. There is a possible relationship: The bigger the maximal size, the larger the variance of the θ value, as indicated on the broken lines, that are not actual estimations but they are for guiding the view of the reader. The plot is in log-log scale.

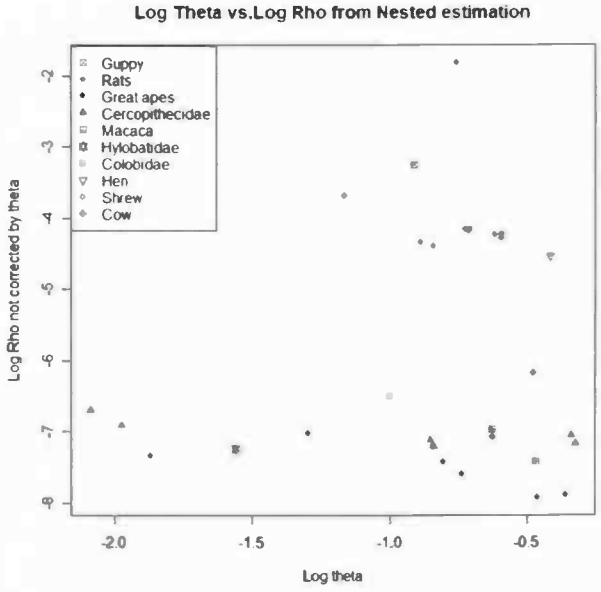


Figure 14. Estimated values of θ and ρ. The Plot indicates an inverse relationship that the one obtained for θ and K. The plot is in log-log scale.

6.2.2. Species plotted at once following the same universal curve.

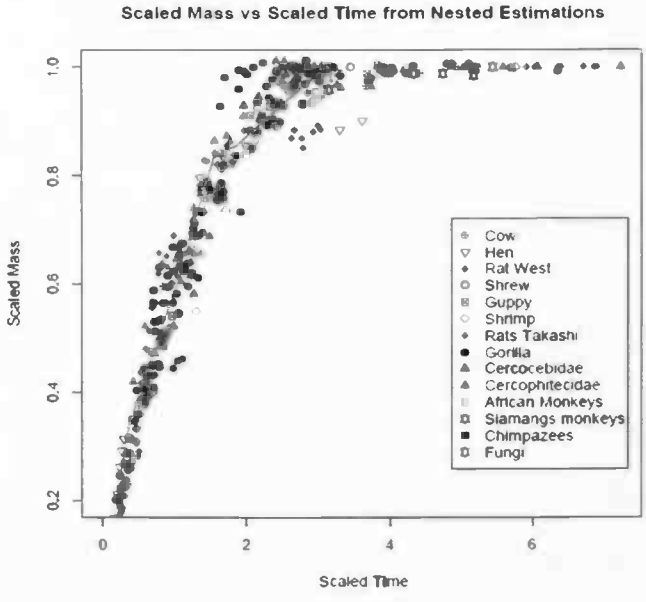


Figure 15. Plot of scaled time vs scaled mass with 27 individuals belonging to 14 species. The plot fit the universal curve equation **(1.17)**, although there is some scattering possible, caused by measurement errors.

7. Discussion

Previous statistical work showed that the form of the universal curve is independent from any specific allometric exponent. (Banavar *et al.*, 2002; de Vladar, 2006). Furthermore, this result is confirmed with my simulations (Fig. 7a.) that show that regardless of the exponent with which the body size (Mass) is rescaled, the fit to the universal curve is virtually perfect. Moreover, Fig.7b shows that the deviation from the universal curve is very small, and lies in the range of 10^{-4} to $6x10^{-3}$. This is consistent with the fact that there might be not a value of θ, contrary to WBE's model. These results also support the idea of Banavar *et al.* (2002) that the universal curve can arise from general considerations independently from a specific allometric exponent in the relationship between metabolic rate and mass scaling. In addition, Fig. 9b shows a broad distribution of theta values which can lead to think that such allometric exponent is not only a single value.

7.1. The limiting size K and cell death rate ρ in growth

As explained before, K in the model is the maximum size that given organism can attain, and ρ is the cellular death rate. Results in Fig. 12, shows a negative relationship between those two parameters. It is known that growing bigger takes more time; hence, larger animals most likely have longer immature periods.

It can be argued that most likely bigger animals could take more time to reach limiting size since it could lead to higher initial fecundity, longer life expectancy, and longer periods of parental care which would decrease the instantaneous juvenile mortality rate of the offspring. On the other hand, small animals tend to have shorter pregnancy and, also shorter parental care periods which could lead to have more reproductive events,

compensating in that way the fact of growing faster until reach limiting size. But of course, not all these correlations can be attributed to body size, also body plan, evolutionary history of the species and the ecology might play an important role (Calder, 1984; Stearns, 1992).

According to Kozlowski (2006) also found a reciprocal relationship between mortality and size, and explained that it can be a source of found variability of life histories. Size strongly depends on mortality, (how big an organism could grows, often depend on the mortality selection pressure it has upon). But on the other hand, mortality often is size-dependent; The bigger the animal the less direct mortality pressure it has, but in the mean time it reaches this point, the probability to die is high (Kozlowski, 2006).

In this study most of the species are mammals and birds, both of which have determinate growth. According to (Gaillard *et al.*, 1989), a longer period as immature is associated with long life and low fecundity. When patterns of parental investment for mammals are take into account the biggest animals tend to mature earlier and had higher reproductive rates than smallest animals. It also confirms the importance of take into account ecological constrains and conditions.

For indeterminate growth organisms, The benefits of grow faster result in an increases in either survival or reproductive output associated with increased body size in older individuals their body size (Calder., 1984; Charnov, 1993; Stearns and Koella, 1986)

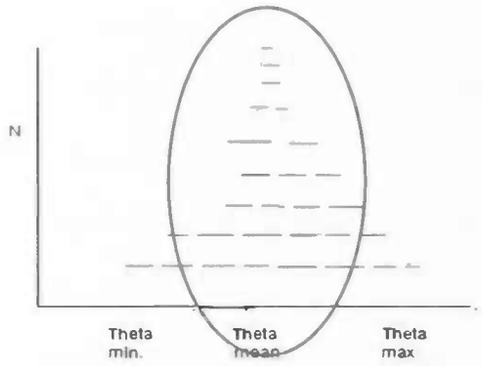


Figure 16. Overrepresentation due to a bias in the number of individuals: Where few species with many individuals, many species with few or only one individual form the shape of the graph.

7.2. Limiting size K and θ exponent in growth

The results in Fig. 13 suggest a trend in the variance with respect to the parameter K. Thus the bigger the maximal size, the greater the variance in the value of θ. However, one has to be aware of the potential bias due to the under representation of the species in the plot, since there were several cases with only one individual of the species, as is shown in Fig.16. Further analysis could be performed, like categorizing by K value and weight every category with its variance. This could give an idea whether this tendency is true or not.

7.3 The θ scaling exponent and its relationship with growth

From the histogram in Fig.9b, we can see that θ it is not constrained to a single value. It rather shows a broad distribution. The histogram also shows that for LEM, the values are biased. We can also see this from the slope of the regression line differs significantly from 1. The values between 0.4 and 0.6 are overestimated and underestimated for values bigger than 0.6 (Fig.8).

Also, over representations of the species further bias our distribution, since the number of curves (N= 130) analyzed with this method was bigger than those of NEM (N= 38). Hence the density of values in the interval 0.6 - 0.8 can be due to the fact of having data of many species with only one individual, and data of few species with many individuals, as show Fig.16.

According to WBE's model the parameter $\theta=3/4$ is derived from fundamental biological and physical principles, and explains how growth is fuelled by metabolic power at the cellular level. The scaling exponent is related to the capacity of the fractal-like network to distribute the resources through the body and its capacity diminishes as body size increases hence WBE's model also assume that organisms have evolved to minimize the energy required to maintain such network. On the other hand, they claim as well that most of the growth data can not discriminate between scaling exponent values $\theta=¾$ or $\theta=⅔$ (West *et al.*, 2002). From my results, this affirmation can be extended to not only $\theta=¾$ or $\theta=⅔$ but also to any value, since all values in a broad range give a fit that is almost perfect.

According to EAO's model, the preservation of the area across the hierarchical levels of the network correspond to $N_k l^3_k$ which is the total number of vessels multiplied by volume of each one, and its preservation is geometrical instead of a biological property, since the summation of the volumes around the vessels remains constant. Etienne *et al.* assumed, maximal efficiency corresponding to preservation of the summation of total vessels instead minimization of energy principle. The total number of vessels depends on the radius and the length of the capillaries, which is determined as well by the allometric

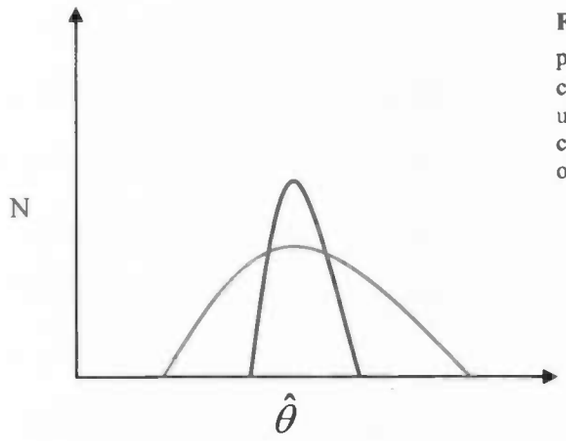


Figure 17. Possible relaxation of selection pressure in captivity. $\hat{\theta}$ is the value which could be modeled by selection processes, but under a controlled environment such processes could be relaxed and allows a broad distribution of θ.

exponent θ. Following this reasoning one should expect a small variance among individuals, of the same species, since the variations in N_k and l_k are expected to be small. In contrast, Fig. 13 shows a large variance suggesting that physiological constrains might not be the unique determining variables for the value of the scaling exponent.

EAO's and WBE's are mechanistic models that predict just one value of the allometric exponent based on optimization principles, and also assume that ontogenetic development is determined directly by allometry. From Fig 9b it is possible to say that there is a great variability and deviation from a single value, as the hypothesis of predicted by these models.

As I said before, the data I employed was from individuals in captivity. An invariant and universal assumption of a physiologically determined scaling exponent should not affect its value, even under different ecological scenarios. On the other hand, if the scaling exponent and the other parameters involved in ontogenetic growth are governed by life

history traits, captivity could blur possible trade offs between such traits, since they were in a controlled environment where energy (in nature usually spent on activities such as foraging for food, competing for mates and shelter etc.) can be re-allocated and let selection pressures forces to relax (Fig. 16) and allows a broad distribution of this parameter, as the one found in (Fig.9a,b).

Most of the models until now assume that ontogeny is determined directly by allometry; however the results obtained in this project suggest that it might not be the case. Despite the fact that metabolism has an important contribution on ontogenetic growth, the results showed that such a contribution might be allometric, but without assuming that ontogeny follows the same allometric scaling exponent between metabolism and body mass.

From equations (1.6) from von Bertalanffy's model and (1.10) from WBE's model we know the value of the scaling exponent for metabolism, however it is rather arbitrary to assume the same value of the exponent in a process that although requires energy from metabolism does not depend entirely on the same constraints. During the early stages growth requires more energy from metabolism than last stages of growth; therefore it should not be so straightforward to think that ontogeny is determined directly by metabolism as proposed by all the models until now.

Future research

In order to know how important is the value of the allometric exponent and its contribution to ontogeny, it would be worth to perform a model selection test, and

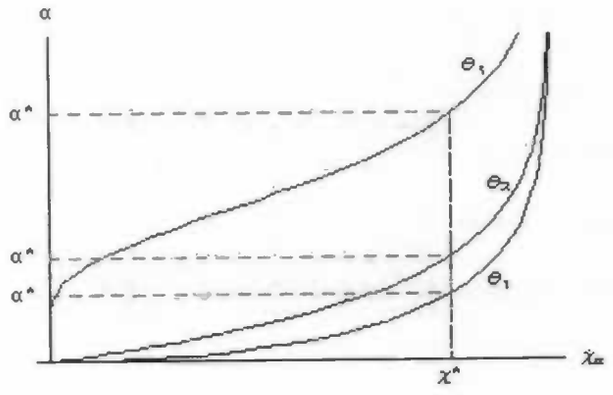


Figure 18. Rescaled size at maturity (X_∞/K) versus rescaled time at maturity $(\rho\alpha)$. X^* arbitrary value of size at maturity. α^* is the time at what such size is reached. In this example, $\theta_3<\theta_2<\theta_1$

following the idea of ontogenetic growth is not determined by allometry, considering equation (1.6) it is possible to fix the value of θ either ¾ or ⅔, and in addition vary the value of the exponent in the second term like this:

$$\frac{dX}{dt} = aX^{\theta} - \rho X^{\upsilon} \tag{1.27}$$

where υ in the second term of the equation can helps to determine as well growth and the evaluate how better it improves the fit in the estimation of the other parameters.

Another analysis that could be implemented is to compare the estimated exponent from ontogenetic fits with the exponent from an optimal calculated from the optimization of time at maturity like this:

Taking α as time at maturity from equation (1.20) and X_α is size at maturity, we get:

$$\alpha = -\frac{B}{\rho} + \left(\frac{1}{\rho}\right) * Log\left[1 - \left(\frac{X_\alpha}{K}\right)\right]^{\theta} \tag{1.28}$$

If fitness in equation (1.18) is maximized using it is a function of the energy allocated to reproduction, it gives a relationship between the parameters K, θ, and ρ. Fig. 18 shows curves that only depend on θ, since time at maturity and size at maturity are rescaled. It

also shows how α varies: the smallest the value of theta, the less time takes to reach size at maturity. Considering trade offs between K and ρ gives distinct α for a given size at maturity and θ. Thus if calculate from data of other studies a similar graph, might be possible to compare the value of θ from life history with the value of θ from ontogeny and then find out the contribution of allometry in growth.

8. Conclusions

With the results of this project the following conclusions can be drawn:

- With respect to the first and second question about the universality of the parameter θ I did not find evidence that supports such universality, neither from the simulations nor from the data, since I obtained a broad range of θ values. Also the simulations showed that universal fits are not indicative of such universality in the scaling exponent.
- With respect to the third question about the possible relations between the parameters:

  - There is no apparent relation between the parameters θ and ρ. However, because individuals were kept in captivity, any existing life history constraints could be relaxed because the individuals grew in controlled environments.

  - There seems to be tendency between parameters θ and K: the bigger the individual the larger the variance in the value of θ. This observation should have a deeper analysis and more data, since the amount of points I employed does not suffice to draw this conclusion with statistical robustness.

  - There is a relation between the parameters ρ and K, where the bigger the maximal size, the smallest the value of ρ, possible explained by ecological aspects

of the specie, as reported before by (Calder, 1984; Gaillard *et al.*, 1989; Kozlowski, 2006).

- It is assumed that ontogeny is directly influenced by metabolism, but according to the results in Fig 9b. this might be not the case. Instead the possibility that life history traits determine the allometric relation to size, rather metabolism and physiology, is open.

Acknowledgments:

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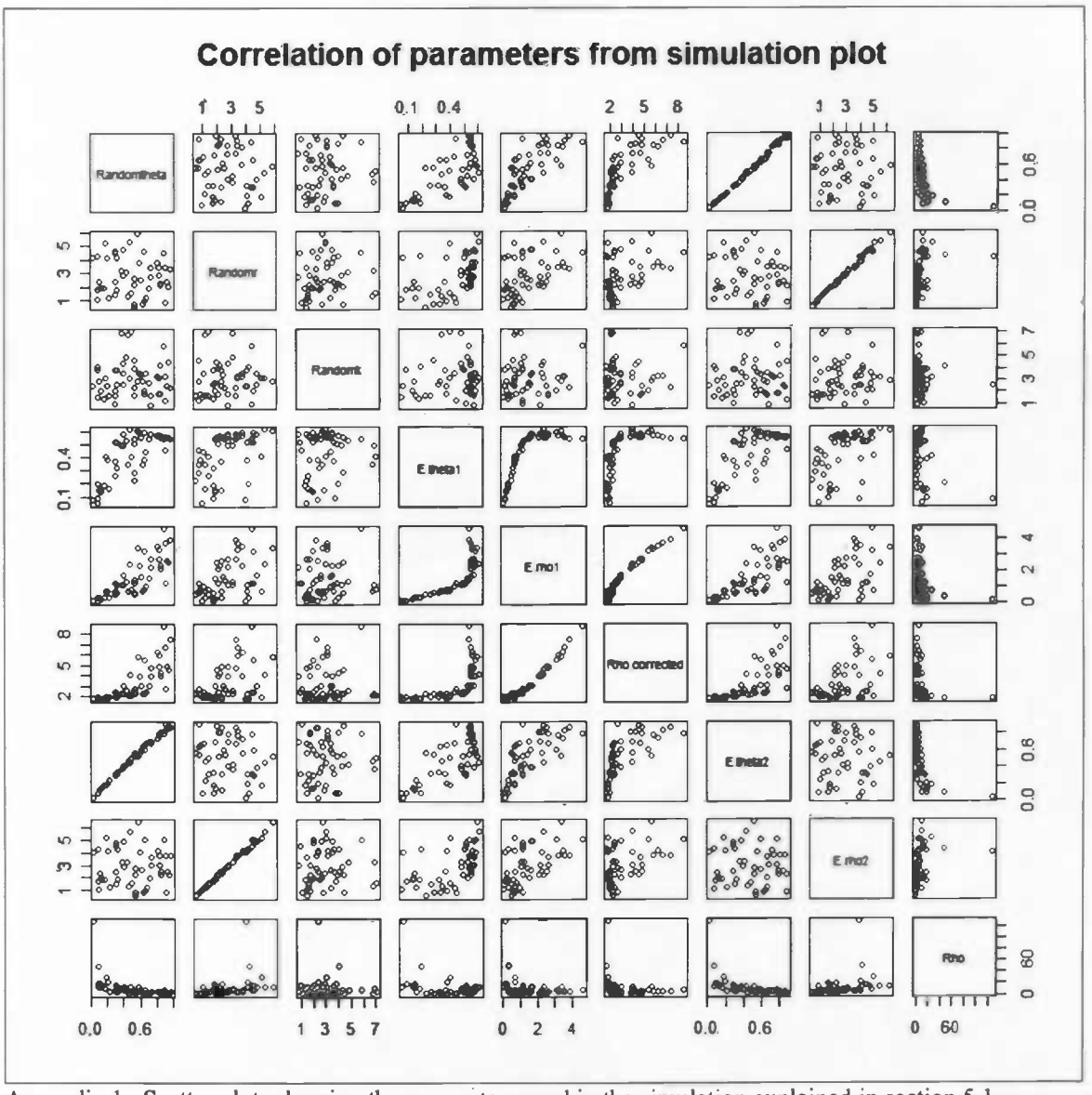
Appendix



Appendix 1. Scatter plots showing the parameters used in the simulation explained in section 5.1. From upper left to lower right (chosen θ, chosen ρ, chosen K, estimated θ, ρ from LEM, estimated θ, ρ from NEM). Randomtheta is the value of θ chosen at random, randomr is the value of ρ chosen at random, and randomk is the value of K chosen at random as explained in section 5.1. Estimated θ, ρ values from LEM as explained in section 5.2.4. And estimated θ, ρ values from NEM as explained in section 5.2.3. According to equation (1.20) the value of ρ is correlated with the value of θ so it should be corrected, Rhocorrected and Rho are the values of ρ divided by the value of θ in order to correct it, from LEM and NEM respectively. In the report, some of them were used to explain and discuss in more detailed my results.