The metabolic origins of big size in aquatic mammals

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The group of large aquatic mammals has representatives being the largest living beings on earth, surpassing the weight and size of dinosaurs. In this paper, we present some empirical evidence and a mathematical model to argue that fat accumulation in marine mammals triggers a series of metabolic events that result in these animals’ increased size. Our study starts by analysing 43 ontogenetic trajectories of species of different types and sizes. For instance, the analyses include organisms with asymptotic mass from 27 g (Taiwan field mouse) to 2.10\textsuperscript{7} g (grey whale). The available data allows us to determine all available species’ ontogenetic parameters (catabolism and anabolism constant, scaling exponent and asymptotic mass). The analyses of those data show a minimisation of catabolism and scaling exponent in marine mammals compared to other species analysed. We present a possible explanation for this, arguing that the large proportion of adipose tissue in these animals can cause this minimisation. That is because adipocytes have different scaling properties in comparison to non-adipose (typical) cells, expressed in reduced energetic demand and lower metabolism. The conclusion is that when we have an animal with a relatively large amount of adipose tissue, as is the case of aquatic mammals, the cellular metabolic rate decreases compared to other animals with the same mass but with proportionally smaller fat tissue. A final consequence of this cause-effect process is the increase of the asymptotic mass of these mammals.

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\section{I. INTRODUCTION}

The blue whale (\textit{Balaenoptera musculus}) is the largest and heaviest living being, having up to 180 tons \cite{1,2}. It is 30 times bigger than the heaviest land animal, the African elephant (\textit{Loxodonta africana}), weighing around 6 tons \cite{3,4}, and twice the size of the largest terrestrial animals that ever lived, the argentinosaurus (\textit{Argentinosaurus huinculensis}) \cite{5}, a species of Sauropoda dinosaur. This enormous difference in size between whales and terrestrial animals has to do, in part, with the reduced gravity effect in the aquatic environment \cite{6}. However, what other factors, complementary to gravity, are acting on the appearance and persistence of such large marine animals?

Some common explanations for the appearance of large animals – the so-called \textit{gigantism effect} \cite{7} – is that essential resources need to be abundant and effectively recycled and reused in a highly developed ecological infrastructure; this is a rare biological condition \cite{2}. Moreover, large animals have an advantage against predators \cite{5} and also improve the capacity to forage food \cite{7}. Nevertheless, there are also other non-trivial explanations for the phenomenon, such as genetic adaptations in the transition from land to water lifestyle \cite{8}, intense environmental pressure (the Contingency Rule \cite{10}), and the evolutionary memory to favour biomass accumulation\textsuperscript{1}. To sum up, the gigantism effect is, in fact, a multifaceted phenomenon in which each factor cited above, among others, contributes to some degree (see \cite{7}).

One trademark of marine mammals is their capacity to stock fat, especially in pinnipeds (seals), sirenians (manatees), and cetaceans (whales). The thickness of the fat layer (blubber) in cetaceans reaches 20 cm (for example, in \textit{Eschrichlius robustus}) and makes up from 15 to 55\% of the body mass \cite{12-15}. In addition to energy storage, the blubber acquired many physiological and physical functions, such as thermal insulation, aid in flotation and locomotion, and increasing swimming efficiency by smoothing the body contour \cite{13}.

Empirical evidence suggests that the way to store fat evolved from single-celled organisms (bacteria and yeasts) to specialized multi-cellular beings. While single-celled organisms developed regions accumulating lipid in droplets, the multi-cellular organisms developed their own adipose organ (in fish, amphibians, and reptiles) with the subsequent organization of subcutaneous adipose tissue (in mammals) \cite{16}. It shows that the adipocytes, cells that store fat and compose the adipose tissue, had their very evolutionary history, which occurred along with the evolution of large taxonomic groups.

Here we connect these fat properties with the metabolism and the ontogenetic scaling properties, specially using as a starting point the theories developed by

\textsuperscript{1} The biggest aquatic animals, which include the Pinnipedia (seals) and Cetacea (whales), have a common ancestor with the common hippo (\textit{Hippopotamus amphibius}) \cite{11}, one of the heaviest land animals (up to 3 tons). It suggests that marine mammals have a genetic framework and an evolutionary memory to favour biomass accumulation.
West et al. [17–20] and expanded by other researchers [21–29]. Those theories try to explain the empirical evidence that the metabolic rate \( R \) of an organism obeys an allometric scaling law with its mass \( m \) in the form [27]

\[
R = R_0 m^\beta .
\]  

(1)

This relation is known by Kleiber’s law, where \( R_0 \) is the allometric constant and \( \beta \) the scaling exponent. Empirical evidence suggests that \( \beta < 1 \), which implies that larger animals are more efficient energetically – demanding less energy per cell [15]. Kleiber’s Law is valid in inter-species context (i.e., using adult mass of different species) and in intra-species context during ontogenetic process (using time evolution of mass of a single species) [23, 28].

West et al. [17] explain such scaling properties as a transport optimisation process. Natural selection operates in the efficiency of resource distribution, generating an optimum network distribution where the calibre of the vessels is hierarchically decreased until capillaries at the lowest level of branching that are invariant. This optimum network distribution reduces energy expenditure on transportation, leading to an optimal value of \( \beta = 3/4 \) in vascular multi-cellular Metazoan [23]. The original West et al. model is derived with details in [29].

All hypotheses and models to explain transport optimization were intensively debated and improved [30] since the publication of West et al. work in the later 90s [17]. However, there is no theoretical background yet to explain why some organisms deviate to lower values of \( \beta \) from the expected values predicted by the West et al. theory. For instance, there are mathematical foundations to explain the superior-limit \( \beta \to 1 \) from microscopic interactions between non-specialized cells [29], but to our best knowledge lower \( \beta \) values have not been treated from the metabolic/scaling point-of-view.

There are substantial empirical metabolic scaling findings in virtually all taxonomic groups and in various experimental conditions/designs. In relation to theoretical studies, Glazier [30] describes four research lines to explain biological scaling: (I) surface-area hypothesis, (II) network of resource distribution hypothesis, (III) system composition hypothesis, and (IV) resource demand hypothesis. This classification helps us to organize the hypothesis, theories and experimental designs, even though these theories are not exclusionary, mainly III and IV, where our work is based.

In the present work, we try to shed some light on this discussion by combining theoretical (analytical) and experimental data, showing and explaining the small values of the metabolic scaling exponent that we observed in marine mammals (details in section (III)). More specifically, we use a careful methodology to fit curve-to-data from ontogenetic trajectories to show that aquatic mammals present a scaling exponent \( \beta \) significantly smaller than \( 3/4 \) – the value predicts by West et al. theory. We justified this trend and also the increased size in these animals by the large composition of their adipocytes. More specifically, we offer some empirical findings and a theoretical approach to argue that fat accumulation in aquatic mammals triggers a series of events that culminates in the increase in the size of these animals.

The paper is organized as follows. In Section (II), we present the ontogenetic growth model – and its parameters – on which our analyses will be based. In section (III), we present our analyses for 43 species, in which we get the ontogenetic parameters for each species. In section (IV), we discuss the role played by the fat tissue in marine mammals and present a mathematical model to demonstrate how the scaling properties of adipose cells yield a minimization of the energetic demand in such animals. In section (V) we present some biological foundation to the arguments proposed. The paper finish with final considerations in section (VI).

II. ONTOGENETIC GROWTH MODEL

The ontogenetic models, from Bertalanffy and Richards’s primordial works [31, 32] to the most advanced and contemporary studies [18, 21, 33], have been successful in describing individual organism growth. Specifically in the seminal work of West et al. [18], the authors derive the logistic shape of the temporal organism growth considering that the total energy metabolised can be used either to create new cells – the anabolism – or to maintain existing cells – the catabolism.

The idea can be expressed in the mathematical form

\[
R = E_c \frac{dN}{dt} + NR_c ,
\]  

(2)

where \( R \) is the metabolic rate (measured in Watts, i.e. Joules per second). The first term on the right of this equation, \( E_c dN/dt \), is the energy per time \( dt \) spend to create \( dN \) new cells, with \( E_c \) being the energy necessary to create one new cell, also called activation energy [20].

The second term on the right of Eq. (2), \( NR_c \), is the energy per time \( dt \) to maintaining the existing \( N \) cells, and \( R_c \) is the cellular metabolic rate, i.e. the energy necessary to maintain one cell.

The metabolic rate also obeys the allometric law Eq. (1), and then if \( m_c \) is the mass of a single cell and \( m = Nm_c \) is the mass of the organism, then Eq. (2) leads to

\[
\frac{dm}{dt} = Am^\beta - Bm .
\]  

(3)

Here, we introduce

\[
A \equiv \left( \frac{R_0 m_c}{E_c} \right) ,
\]  

(4)

2 The activation energy \( E_c \) is a meaningful but also controversial variable. There is a paucity of empirical observations and any well-established experimental design to measure it.
namely the anabolism constant (measured in grams$^{-1/3}$time$^{-1}$), which defines the rate at which the mass is incorporated into the organism. Note that $A \propto 1/E_c$, which means that this parameter is a measure of the energy necessary to create a new cell. We also introduce

$$B \equiv \frac{R_c}{E_c},$$

namely the catabolism constant (measured in unit of frequency 1/time), which defines the rate at which the organism uses the energy for vital demands. Eq. (5) expresses that the catabolism can also be understood as the relation between cellular metabolic rate and activation energy.

Eliminating $E_c$ in Eqs. (5) and (4) yields

$$R_c \propto \frac{B}{A},$$

that means cellular metabolic rate can be inferred by the anabolism-catabolism relation. The anabolism constant $A$ is invariant among species of the same taxonomy group, since it depends only on scaling invariant parameters$^3$. This fact, together with Eq. (6), suggest that inside the same taxonomy group the cellular metabolic rate can be understood solely by the catabolism constant, i.e. $R_c \propto B$.

Equation (3), in turn, has as solution

$$m(t) = \left[ \frac{A}{B} + \left( \frac{m_0}{B} - \frac{A}{B} \right) e^{B(\beta - 1)t} \right]^{\frac{1}{\beta}},$$

where $m_0$ is the initial mass of the organism. This solution diverges for $t$ sufficiently large when $\beta > 1$ and $B > 0$, or when $\beta < 1$ and $B < 0$. However, for $\beta < 1$ (sublinear regime) and $B > 0$, which is in agreement with biological systems$^{[13]}$, this solution converges to

$$m(t \gg 1) \equiv M = \left( \frac{A}{B} \right)^{\frac{1}{\beta}}.$$

Here we define $M$ as the mature (asymptotic) mass of the organism. For the special case that $\beta < 1$ and $B \approx 0^+$, and according to the Eq. (7), the mass growth initially as power law (given by $m(t) \sim t^{1/\beta}$) and then saturates to $M$$^{[20]}$. More details about the solution of this model are presented in Appendix A.

### III. EMPIRICAL ANALYSES

The empirical base used in this work consists 43 ontogenetic trajectories collect from the literature, including organisms with asymptotic mass ranging from 27g

(Taiwan field mouse) to 2$ \cdot$ 10$^7$g (grey whale). The data and the methodology, described in Supplemental Materials (B) and (E), allow us to determine the ontogenetic (macroscopic) parameters ($A$, $B$, $\beta$ and $M$) for all included species.

The first finding that we can obtain after estimating the four ontogenetic parameters are the strong correlations between their numeric values, as suggested by the results presented in Fig. (1). One can see that all analysed species, independently of their type or size, rigorously obey Eq. (5). From this result we can infer that any change in one of these parameters – for instance caused by adaptation – automatically reverberates in the other parameters, always maintaining the constraints imposed by Eq. (5).

However, when sub-groups of ontogenetic parameters are analysed, some particularities can be observed, especially concerning marine mammals. For instance, there is an evident differentiation between the marine mammals and the other analysed species in respect to the anabolism-catabolism relation. One can see in Fig (2) that there is a strong linear dependence between $A$ and $B$ (and the slope is related to $R_c$, cf. Eq. (6)), which implies that animals with higher anabolism also tend to have higher catabolism. However, marine mammals break this rule, maintaining their catabolism ($B$) much lower than it should be (according to this linear rule).

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$^3$ However, $A$ must to differ between distinct taxonomic groups, since it depends directly on the allometric constant $R_0$, which varies between taxonomic groups.
Figure 2: The catabolism constant \( B \) as a function of the anabolism constant \( A \) (in a log-log plot). The data suggest a strong linear relationship between these constants, and the slope is related to the cellular metabolic rate \( (R_c) \), conform Eq. (6). However, the marine mammals (blue points) break this rule, presenting a much lower catabolism \( (B) \) than should be (according to this rule). The rainbow trout was the animal with the lowest anabolism and catabolism constants among the analysed species, while the male Taiwanese wood rat is the species with the highest numerical value for these metabolic constants.

This result, together with Eqs. (5) and (6), suggests that these mammals have lower cellular metabolic rates.

The catabolism reduction in marine mammals is also accompanied by a lower scaling exponent \( \beta \) among these animals. The scaling exponent of the considered terrestrial mammals lies in the interval \( 0.70 < \beta < 0.99 \). However, the scaling exponent for the analysed marine mammals is \( \beta \approx 0.5 \), that is much lower than the other species. Fig. (3) shows that these large mammals differ from the other species in terms of both \( \beta \) and \( B \). Marine mammals are the biggest animals among the analysed species and, at the same time, the ones with lowest \( \beta \) and \( B \) values. It suggests that these large mammals’ metabolism is relatively slower compared to the other analysed species.

Extreme cases

To finalise this section, we would like to discuss two species of our database that exhibit extreme values of the anabolism and catabolism constants, i.e. the rainbow trout and the Taiwan field mouse. The intention is to show that the organism’s energetic demand – its metabolism – reflects directly in the values of \( A \), \( B \) and \( \beta \).

The rainbow trout (Oncorhynchus mykiss), which lives in the Pongokepuk River, a low-temperature region in Alaska [34], is the animal with the lowest anabolism and catabolism constants among the analysed species (see Fig. (2)). It is valid to say that ectothermic animals, like this one, have metabolism directly influenced by temperature [35]. That means that the slower metabolism of this fish (small \( B \) and \( A \), simultaneously) is associated with the lower temperatures in its environment. Moreover, this species exhibits a smaller scaling exponent than the other non-marine mammal’s species (\( \beta \approx 0.69 \)).

In contrast, the male Taiwan field mouse (Apodemus semotus) is the species with the highest value of the anabolism and catabolism constants (see Fig. (2)), which is evidence of high metabolism. In fact, this species reaches sexual maturity very quickly (in 25 days after being born) [36], expressing a high growth rate (related to anabolism), demanding a high metabolism. Moreover, this animal exhibits a larger value of the scaling exponent (\( \beta = 0.99 \)), which is also compatible with this rodent’s high metabolic demand.

These extreme cases reveal to be consistent with the assumption that the energetic demand of an organism...
We argue that the large proportion of adipose tissue in catabolism and the scaling exponent in marine mammals. Evidence, especially regarding the minimisation of forces, the increase of adipose tissue in these mammals’ bodies, due to benefits of the fat blubber. But, most important in the context of this work is that the large proportion of adipose tissue reflects the storage and demand for energy \cite{37,38}. Moreover, adipose tissue has a structural function \cite{14} and a scaling response, once that blubber thickness and body fat-mass composition scales with body mass \cite{12}.

Adipose cells respond differently to scale in comparison to other cells. While the mass of non-adipose (typical) cells, say \(m_c^{(n)}\), is scale invariant \cite{22,40}, some empirical studies have shown that the mass of the adipose cells, say \(m_c^{(ad)}\), increase with the organism’s size \(M\) by the form

\[
m_c^{(ad)} \sim M^\alpha,
\]

as suggested by the data shown in Fig. (5). Note that a scaling property between adipose cells volume and body mass is evident, and consequently, adipose cells mass and body mass also scale if we consider cellular density to be invariant \cite{22}, which means \(\alpha > 0\). However the value of this scaling exponent cannot be precisely determined. Savage et al. \cite{22} argue that \(\alpha = 1 - \beta\) in order to obey Kleiber’s law (see supplementary material (C)). Nevertheless, we will see that the value predicted by them does not agree with the data.

Other important scale difference between adipose and non-adipose cells is concerning the cellular metabolic rate. While in non-adipose cells their cellular metabolic rate, namely \(R_c^{(n)}\), decrease with the organism mass by the form \(R_c^{(n)} = m_c^{(n)} R_0 M^{-\beta - 1}\) (from Eq. (1)), the adipocytes maintain scaling invariant its cellular metabolic rate, namely \(R_c^{(ad)}\) \cite{22}, that is \(R_c^{(ad)} \sim M^0\). These properties are organized in Table I. In this way, the typical cells present scaling invariant mass but their cellular metabolic rate decrease with the size of the organism. However, the adipocytes does the opposite: they increase their mass with the size of the organism and maintain scale-invariant their energy demand and consequently their cellular metabolic rate.

The consequence of these properties, as will be shown by the mathematical model presented in the following subsection, is that when we have an animal with a relatively large amount of adipose tissue, as is the case of marine mammals, the cellular metabolic rate decreases compared to other animals with the same mass but with proportionally smaller fat tissue. Having said that, we hypothesise that the metabolic minimisation in these fat-rich animals results from the very accumulation of fat. That is because fat tissue has reduced energetic demand.

Table I: Scaling properties of adipose and non-adipose (typical) cells.

| cell mass | typical cells | adipose cells |
|-----------|---------------|---------------|
| \(m_c^{(n)}\) | \(m_c^{(n)} \sim M^\alpha\) | \(m_c^{(ad)} \sim M^\alpha\) |
| cellular metabolic rate | \(R_c^{(n)} = m_c^{(n)} R_0 M^{-\beta - 1}\) | \(R_c^{(ad)} \sim M^0\) |

IV. METABOLISM AND THE FAT STORES IN MARINE MAMMALS

Next we describe our explanation the empirical evidence, especially regarding the minimisation of catabolism and the scaling exponent in marine mammals. We argue that the large proportion of adipose tissue in these animals can be the cause of this minimisation. We present a mathematical model to justify this argument. The processes involved in this hypothesis are summarized in Fig. (4). A final consequence of this causal process is the increase of the asymptotic mass of these mammals.

The evolutionary pressure in the marine environmental forces the increase of adipose tissue in these mammals’
mass and polar bear, the data suggest that the scaling exponent value $\alpha$ is not well established. For instance, in the case of Pond & Mattack (1986)’s work [11] and polar bear [12], the data suggest $\alpha = 0.35$ (in red and blue, respectively). However, Morgado et al.’s data suggest $\alpha = 0.29$ (gray line). The data for fin whales (Balaenoptera physalus) is also presented (orange dot). The data of Pond & Mattacks (1985) [43] is in respect to adipocytes in intra-abdominal, superficial and intermuscular depots, in species of the orders: Rodentia (3 species), Felidae (1 species), Canidae (1 species), Artiodactyla (4 species).

and lower metabolism, which is expressed by smaller values of $B$ and $\beta$ values, and in the increase of the body mass $M$, conform presented in previous section. This will be better explained by the mathematical model that will be presented now.

### A. Mathematical Model

For an adult organism (that is $dN/dt = 0$), the metabolic rate can be written (from Eq. (2)) as

$$R = R^{(n)} + R^{(ad)} = N_n R_c^{(n)} + N_{ad} R_c^{(ad)}, \quad (10)$$

where $R^{(n)}$, $N_n$ and $R_c^{(n)}$ (total metabolic rate, number of cells and cellular metabolic rate, respectively) are quantities related to non-adipose (typical) cells, and $R^{(ad)}$, $N_{ad}$ and $R_c^{(ad)}$ are quantities related to adipocytes. Of course, we are considering an idealized situation that the organism is composed only by these two kinds of cells. If that is the case, then the total number of cells and the total mass must obey $N = N_n + N_{ad}$ and $M = M_n + M_{ad}$, respectively. If $m_n^{(n)}$ and $m_{ad}^{(n)}$ are the cellular mass of the non-adipose and adipose cells, respectively, then $M_n = N_n \cdot m_n^{(n)}$ and $M_{ad} = N_{ad} \cdot m_{ad}^{(n)}$. Consequently, Eq (10) yields

$$R = M_n \frac{R_c^{(n)}}{m^{(n)}} + M_{ad} \frac{R_c^{(ad)}}{m_{ad}^{(ad)}}. \quad (11)$$

Using the scaling properties of these cells (see table [1]), the equation above yields

$$R = M_n R_0 M^{\beta_{-1}} + \text{cte} \cdot M_{ad} M^{-\alpha}. \quad (12)$$

Let’s now introduce the parameter $\lambda$ which represent the proportion of fat mass in the whole organism body. In this sense, we consider, by hypotheses, that

$$M_{ad} = \lambda M, \quad (13)$$

and

$$M_{n} = (1 - \lambda) M. \quad (14)$$

This parameter lies between $0 \leq \lambda \leq 1$, and these extreme cases means: $\lambda = 0$, an organism with no fat tissue; and $\lambda = 1$, an (unrealistic) organism formed purely by fat tissue. A real organism must be something in between these two extreme situations. For instance, in the case of Balaenoptera physalus, that has around 30% of the total body mass composed by fat tissue [15], we would have $\lambda = 0.3$.

Introducing such hypothesis in Eq. (12) yields

$$\frac{R}{M} = (1 - \lambda) R_0 M^{\beta_{-1}} + \text{cte} \cdot \lambda M^{-\alpha}. \quad (15)$$

In the appendix [13] we show that $R/M \sim R/N$ for $M$ sufficiently large. Also, if we identify the organism cellular metabolic rate as $R_c = R/N$, one has from the above equation that

$$R_c(\lambda) \sim R_0 M^{\beta_{-1}} - \lambda \left(R_0 M^{\beta_{-1}} - \text{cte} \cdot M^{-\alpha}\right). \quad (16)$$

That is, we write the cellular metabolic rate of the organism as a function of the proportion of fat tissue: $R_c = R_c(\lambda)$. Note that the total absence of adipocytes...
B. Connection with the catabolism, scaling exponent and organism size

From Eq. (5) and considering only adults organism (and then the creation energy $E_c$ does not plays any rule) then $B \sim R_c$. That is, the catabolism constant is, in fact, directly related to the cellular metabolic rate, and it is valid the same properties that came with the result (16). In this way, when $\alpha > 1 - \beta$, the catabolism decrease with the increasing of the proportion of adipose tissue ($\lambda$). In summary, as a hypothesis to explain the data presented in the previous section, we suggest that the bigger proportion of fat in marine mammals, caused by evolutionary pressure, is one of the components responsible for minimising their catabolism ($B \rightarrow 0$).

We can also infer about the scaling exponent $\beta$ and the organism size with such results. As we saw in the previous section, any change in the ontogenetic parameters ($A$, $B$, $\beta$, and $M$) must be done constrained to the relation (8). By the mathematical model proposed here, we saw that the reduction of cellular metabolic rate, and consequently the decrease of $B$, is followed by i) the reduction of $\beta$, or ii) the increase of $M$, or both, in order to maintain constrained to the relation (8).

For instance, Eq. (8) can be written as

$$\beta = 1 - \frac{\log(A/B)}{\log M}.$$  \hfill (17)

Note then that if we decrease $B$ maintaining $M$ and $A$ fixed, then $\beta$ must diminish. On the other hand, Eq. (8) can also be written as

$$M \sim B^{\frac{1}{1-\beta}}.$$  \hfill (18)

for $A$ fixed. Note then that by this equation, if $\beta < 1$, then a decrease in $B$ implies that $M$ increases. Therefore, the minimisation of $B$, maintaining $\beta$ fixed, implies automatically in the organism mass increasing (given the constraint imposed by Eq. (3)).

In conclusion, the $B$ minimization (due to the increase in fat tissue) is automatically followed by the decrease of $\beta$ and also by the increase in the organism size $M$. The scheme (4) shows the sequence of events that are chained thanks to evolutionary pressure for fat acquisition.

Alternatively, we can interpret the Eq. (8) from the evolutionary terminology. It has well described the tradeoffs where a biological attribute has a compensatory effect on others. So, Eq. (15) suggests the mandatory effect of $B$ on $M$ (from the very particular adipocyte metabolism impacting all adipose tissue) and a secondary effect of $B$ on $\beta$ (from the lower oxygen demand by adipocytes impacting in all adipose tissue). It is a phenomenon with microscopic origins, where adipocytes have their own evolutionary specialization, allowing new functional assignments on a higher scale (adipose tissue), reverberating in macroscopic attributes.

To sum up, one can conclude by the model that the accumulation of fat leads to a decrease in the cellular metabolic rate (due to the scaling properties of adipocytes) and decreasing in catabolism ($B$). Finally, as an answer to the constraint given by Eq. (8), the organism can either decrease the scale constant ($\beta$) or increase the size ($M$), or both.
V. BIOLOGICAL FOUNDATIONS

In this section, we propose some biological foundations to explain the metabolic-catabolic minimization and the gigantism effect in aquatic mammals.

A. Scaling exponent and cell oxygen consumption

It is crucial to speculate or think about what the minimization of $\beta$ means in biological terms. Darveau et al. [45] have shown a positive relationship between oxygen consumption and the value of the scaling exponent. It suggests that a smaller value of $\beta$ in those fat-rich mammals may be a consequence of the reduction of per-cell oxygen consumption. This reduction, in turn, is caused by their metabolism reduction given by the scaling properties of the adipose cells (see schematic diagram in Fig. 4).

B. Genes related to fat accumulation in adipocytes

Positive selection has been detected in some genes (belonging to the ACSL gene family) of cetaceans during their adaptation to the aquatic environment, which implied an association with enhanced triglycerides synthesis and thickened blubber [13]. Such genes increase the ability to capture free fatty acids and triglycerides in the circulation, which accelerate the increase of the adipocytes size. In this way, the adipocytes size expansion is directly linked to the amount of fat accumulated in these cells. Moreover, some specific genes related to fat synthesis and production can be found in a common ancestor of fat-accumulating marine mammals, indicating a strong adaptive selection in such genes [13]. Since blubber composition is made mainly by adipocytes, and Cetacean health can be diagnosed by blubber thickness, such genes could be crucial to these animals.

C. Leptin and adipocyte

In terrestrial mammals, adipose tissue acquired the property of secreting hormones; leptin, a hormone produced and secreted by adipocytes, is strongly related to adipocyte hypertrophy, as seen in rats and humans [17]. Interestingly, in addition to finding significant leptin gene expression in the blubber of seals, an unusual and significant expression of leptin was also observed in their lungs [48]. These findings suggest that maybe leptin promotes the energy balance in the lungs, and not only on the adipose tissue. In the previous section, we saw that the decrease in the oxygen consumption could be associated with the metabolism minimization. Currently, there is evidence pointing to adaptations where leptin started to participate in the respiratory processes of marine mammals, including whales, mainly in conditions of lower oxygen supply (hypoxia) [49]. To sum up, the leptin participation in respiratory control suggests that the respiratory system also contributes to minimizing metabolism, but the literature still lacks experimental evidence and mechanistic explanations.

D. Fishes and muscles

Usually, fishes have 50-60% of their biomass composition of muscle, and this trend is contrary to marine mammals. They are ectothermic, and their metabolism is strongly influenced by environmental variations of the temperature, with a positive relationship between temperature and body mass [35]. Moreover, some tilapia strains are also selected to be thermo-resistant to environmental variations [50]. Since most of the data from tilapia used in this study come from zootechnical studies - that is, in optimal growth environments (with optimal temperature) - we were interested to see some response to this artificial scenario in our numerical results. However, our analysis (not shown in this study) did not indicate any effect of these artificial improvements in $\beta$ values, but yes in the parameters $A$ and $B$, and consequently in $M$ (from Eq. [8]). It suggests that $\beta$ has solid genetic and evolutionary determinants, but is not susceptible to environmental pressure.

The linear trend between $A$ and $B$ (Fig. 2) confirms that fast-developing animals (and shorter lifespan) are directly affected by both parameters. In fishes, a higher anabolism constant could be related to higher biomass muscle synthesis with faster maturity age. On the other hand, a higher catabolism constant could be related to a higher energetic cost to muscle maintenance. Muscle tissue has biological properties very different from adipose tissue, with higher mitochondrial concentration (reflects the higher metabolic activity) and in fishes, where they employ almost all muscle body mass to contract and to swim, this higher energetic demand to maintain muscle function is proportionally higher. So these zootechnical improvements in tilapia seem to respond mainly in $A$, reflecting in $B$ (given Eq. [8] see Fig. 2) with more conservative values to $\beta$.

VI. CONCLUSION

We are conscious that the animal’s size is a multiplicative effect of a series of factors. However, we offer in this work some empirical findings and a theoretical approach to argue that fat accumulation in aquatic mammals triggers a series of events that increase the size of these animals.

The pieces of evidence shown and presented here suggest that when we have an animal with a relatively large amount of adipose tissue, as is the case of marine mammals, the cellular metabolic rate decreases compared to...
other animals with the same mass but with proportionally smaller fat tissue. That is due to the different scaling properties of the adipose cell in comparison to the non-adipose cells. It implies that fat tissue has reduced energetic demand and lower metabolism, which is expressed by smaller values of $B$ and $\beta$ values, and in the increase of the body mass $M$. That is the case for marine mammals. It suggests that the metabolic minimization in these fat-rich animals results from the very accumulation of fat. As a consequence of these cause-effect mechanisms and given the constraint imposed by the relation (8), we have the emergency of those big animals.

In conclusion, one can say that the large proportion of fat tissue in marine mammals has the following hypothetical consequences: i) slower metabolism (characterized by smaller values of the catabolism constant $\beta$); ii) reduced scaling exponent $\beta$, which may be related to the reduction in per-cell oxygen consumption; and iii) increase in the organism’s mass $M$.

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VII. REFERENCES
A. B. Herman, W. H. Woodruff, and G. B. West, “Scaling of number, size, and metabolic rate of cells with body size in mammals,” *Proceedings of the National Academy of Sciences of the United States of America*, vol. 104, no. 11, pp. 4718–4723, 2007.

[23] M. E. Moses, C. Hou, W. H. Woodruff, G. B. West, J. C. Nekola, W. Zuo, and J. H. Brown, “Revisiting a model of ontogenetic growth: Estimating model parameters from theory and data,” *American Naturalist*, vol. 171, no. 5, pp. 632–645, 2008.

[24] J. P. DeLong, J. G. Okie, M. E. Moses, R. M. Sibly, and J. H. Brown, “Shifts in metabolic scaling, production, and efficiency across major evolutionary transitions of life,” *Proceedings of the National Academy of Sciences of the United States of America*, vol. 107, no. 29, pp. 12941–12945, 2010.

[25] F. L. Ribeiro, R. V. Dos Santos, and A. S. Mata, “Fractal dimension and universality in avascular tumor growth,” *Physical Review E*, vol. 95, no. 4, pp. 1–9, 2017.

[26] M. Kleiber, “Metabolic turnover rate: A physiological meaning of the metabolic rate per unit body weight,” *Journal of Theoretical Biology*, vol. 53, no. 1, pp. 199–204, 1975.

[27] W. Zuo, M. E. Moses, C. Hou, W. H. Woodruff, G. B. West, and J. H. Brown, “Response to comments on "Energy uptake and allocation during ontogeny,"” *Science*, vol. 325, no. 5945, 2009.

[28] F. L. Ribeiro and W. R. Pereira, “A Gentle Introduction To Scaling Relations in Biological Systems,” *Revista Brasileira de Ensino de Fisica*, vol. 44, 2022.

[29] D. S. Glazier, “Metabolic scaling in complex living systems,” *Systems*, vol. 2, no. 4, pp. 451–540, 2014.

[30] L. von Bertalanffy, “Quantitative laws in metabolism and growth,” *The Quarterly Review of Biology*, vol. 32, no. 3, pp. 217–231, 1957.

[31] F. J. Richards, “A flexible growth function for empirical use,” *J Exp Bot*, vol. 10, no. 2, pp. 290–301, 1959.

[32] F. L. Ribeiro, R. V. Dos Santos, and A. S. Mata, “Fractal dimension and universality in avascular tumor growth,” *Physical Review E*, vol. 95, no. 4, 2017.

[33] T. N. W. R. A. MacDonald, R., Lisac, M. J., “Age, weight and length statistics of Togiak River Drainage Resident Fish Species, Togiak National Wildlife Refuge, Alaska, 1993-1995,” 1996.

[34] A. Audzijonyte, S. A. Richards, R. D. Stuart-Smith, G. Pecl, G. J. Edgar, N. S. Barrett, N. Payne, and J. L. Blanchard, “Fish body sizes change with temperature but not all species shrink with warming,” *Nature Ecology and Evolution*, vol. 4, no. 6, pp. 809–814, 2020.

[35] L.-k. Lin, T. Nishino, and S. Shiraishi, “Postnatal Growth and Development of the Formosan Wood Mouse,” *Journal of the Mammalogical Society of Japan*, vol. 18, no. 1, pp. 1–18, 1993.

[36] G. E. Davis, M. F. Baumgartner, P. J. Corkeron, J. Bell, C. Berchok, J. M. Bonnell, J. Bort Thornton, S. Brault, G. A. Buchanan, D. M. Cholewiak, C. W. Clark, J. Delarue, L. T. Hatch, H. Klink, S. D. Kraus, B. Martin, D. K. Mellinger, H. Moors-Murphy, S. Nieukirk, D. P. Nowacek, S. E. Parks, D. Parry, N. Pegg, A. J. Read, A. N. Rice, D. Rischo, A. Scott, M. S. Soldevilla, K. M. Stafford, J. E. Stanistreet, E. Summers, S. Todd, and S. M. Van Parijs, “Exploring movement patterns and changing distributions of baleen whales in the western North Atlantic using a decade of passive acoustic data,” *Global Change Biology*, vol. 26, no. 9, pp. 4812–4840, 2020.

[37] J. Groff, P. Virtue, P. D. Nichols, P. Eisenmann, C. A. Waugh, and S. Bengtson Nash, “Interannual variability in the lipid and fatty acid profiles of east Australia-migrating humpback whales (Megaptera novaeangliae) across a 10-year timeline,” *Scientific Reports*, vol. 10, no. 1, pp. 1–14, 2020.

[38] J. Castrillon, W. Huston, and S. Bengtson Nash, “The blubber adipocyte index: A nondestructive biomarker of adiposity in humpback whales (Megaptera novaeangliae),” *Ecology and Evolution*, vol. 7, no. 14, pp. 5131–5139, 2017.

[39] Y. H. M. Chan and W. F. Marshall, “Scaling properties of cell and organelle size,” *Organogenesis*, vol. 6, no. 2, pp. 88–96, 2010.

[40] C. M. Pond and C. A. Mattacks, “Allometry of the cellular structure of intraorbital adipose tissue in cetotherian mammals,” *Journal of Zoology*, vol. 209, no. 1, pp. 35–42, 1986.

[41] C. M. Pond, C. A. Mattacks, R. H. Colby’, and M. A. Ramsay, “The anatomy, chemical composition, and metabolism of adipose tissue in wild polar bears (Ursus maritimus),” tech. rep.

[42] C. M. Pond and C. A. Mattacks, “The distribution, cellular structure, and metabolism of adipose tissue in the fin whale, Balaenoptera physalus,” tech. rep.

[43] E. Morgado, C. Ocqueteau, M. Cury, L. Becker, U. Gonzalez, L. Muxica, and B. Gunther, “Three-dimensional morphometry of mammalian cells II. Areas, volumes, and area-volume ratios,” *Archivos de Biologia y Medicina Experimentales*, vol. 23, no. 1, pp. 21–27, 1990.

[44] C. A. Darveau, R. K. Suarez, R. D. Andrews, and P. W. Hochachka, “Allometric cascade as a unifying principle of body mass effects on metabolism,” *Nature*, vol. 417, no. 6885, pp. 166–170, 2002.

[45] D. Derois, M. ten Doeschate, A. C. Brownlow, N. J. Davison, and D. Luseeau, “Toward New Ecologically Relevant Markers of Health for Cetaceans,” *Frontiers in Marine Science*, vol. 7, no. May, pp. 1–8, 2020.

[46] M. Maffei, J. Halaas, E. Ravussin, R. E. Pratley, G. H. Lee, Y. Zhang, H. Fei, S. Kim, R. Lallone, S. Ranganathan, P. A. Kern, and J. M. Friedman, “Leptin levels in human and rodent: Measurement of plasma leptin and ob RNA in obese and weight-reduced subjects,” *Nature Medicine*, vol. 1, no. 11, pp. 1155–1161, 1995.

[47] J. A. Hammond, K. A. Bennett, M. J. Walton, and A. J. Hall, “Molecular cloning and expression of leptin in gray and harbor seal blubber, bone marrow, and lung and its potential role in marine mammal respiratory physiology,” *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, vol. 289, no. 5 P2-58, pp. 545–553, 2005.

[48] L. Yu, W. Jin, X. Zhang, D. Wang, J. Song Zheng, G. Yang, S. xia Xu, S. Cho, and Y. ping Zhang, “Evidence for positive selection on the leptin gene in cetacea and pinnipedia,” *PLoS ONE*, vol. 6, no. 10, 2011.

[49] V. B. dos Santos, E. A. Mareco, and M. D. P. Silva,
“Curvas de crescimento de linhagens de tilapias do Nilo (Oreochromis niloticus) cultivadas em diferentes temperaturas,” Acta Scientiarum - Animal Sciences, vol. 35, no. 4, pp. 323–326, 2013.

[51] B. B. C. T. Cabella, A. S. A. Martinez, and F. Ribeiro, “Data collapse, scaling functions, and analytical solutions of generalized growth models,” Physical Review E, vol. 83, p. 061902, jun 2011.

[52] B. B. C. T. Cabella, F. Ribeiro, and A. S. A. Martinez, “Effective carrying capacity and analytical solution of a particular case of the Richards-like two-species population dynamics model,” Physica A: Statistical Mechanics and its Applications, vol. 391, pp. 1281–1286, feb 2012.

[53] R. Froese, “Cube law, condition factor and weight-length relationships: History, meta-analysis and recommendations,” Journal of Applied Ichthyology, vol. 22, no. 4, pp. 241–253, 2006.

[54] G. Vikingsson, J. Sigurjonssoon, T. Gunnlaugsson, W. A. Watkins, and M. K. E. “On the relationship between weight, length and girth dimensions in fin and se whales caught off Iceland,” An underwater acoustic survey for sperm whales (Physeter catodon) and other cetaceans in the southeast caribbean,” Rep. Int. Whal. Commn, no. 38, pp. 323–326, 1988.

[55] T. Gunnlaugsson, G. A. Vikingsson, S. D. Halldórsson, B. Elvarsson, T. Haug, and C. Lydersen, “Body mass, muscle, blubber and visceral fat content and their seasonal, spatial and temporal variability in North Atlantic common minke whales,” Journal of Oceanic Research and Management, vol. 21, no. 1, pp. 50–70, 2020.

[56] R. S. Amaral, V. M. da Silva, and F. C. Rosas, “Body weight/length relationship and mass estimation using morphometric measurements in Amazonian manatees Trichechus inunguis (Mammalia: Sirenia),” Marine Biodiversity Records, vol. 3, no. 2011, 2010.

[57] S. Agbayani, S. M. Fortune, and A. W. Trites, “Growth and development of North Pacific gray whales (Eschrichtius robustus),” Journal of Mammalogy, vol. 101, no. 3, pp. 742–754, 2020.

[58] M. I. da Chacon, J. O., Bezerra e Silva, J. W., Nobre, “Ensaiio sobre o cultivo de fêmeas albinas de tilápia do Nilo, Oreochromis niloticus (L., 1766),” Ciência Agron., Fortaleza, vol. 23, no. (1/2), pp. 1–8, 1992.

[59] Q. Han, M. Zhang, C. Guo, X. Zhou, B. Li, and Y. Wang, “Density Effect on Postnatal Growth of Laboratory-Bred Yangzte Vole (Microtus fortis calamorum),” Pakistan Journal of Zoology, vol. 49, no. 6, 2017.

[60] Y. Crispel, O. Katz, D. Ben-Yosef, and Z. Hochberg, “Effects of breastfeeding on body composition and maturational tempo in the rat,” BMC Medicine, vol. 11, no. 1, p. 114, 2013.

[61] J. Kolding, L. Haug, and S. Stefansson, “Effect of ambient oxygen on growth and reproduction in Nile tilapia (Oreochromis niloticus),” Canadian Journal of Fisheries and Aquatic Sciences, vol. 65, no. 7, pp. 1413–1424, 2008.

[62] C. C. Frasier, “An explanation of the relationship between mass, metabolic rate and characteristic length for placental mammals,” PeerJ, vol. 2015, no. 9, 2015.

[63] S. D. Yi, J. D. Noh, P. Minmhagen, M. Y. Song, T. S. Chon, and B. J. Kim, “Human bipedalism and body-mass index,” Scientific Reports, vol. 7, no. 1, pp. 1–8, 2017.

[64] T. E. Essington, J. F. Kitchell, and C. J. Walters, “The von Bertalanffy growth function, bioenergetics, and the consumption rates of fish,” Canadian Journal of Fisheries and Aquatic Sciences, vol. 58, no. 11, pp. 2129–2138, 2001.

[65] C. Guiot, P. G. Degiorgis, P. P. Delsanto, P. Gabriele, and T. S. Deisboeck, “Does tumor growth follow a "universal law"?,” Journal of theoretical biology, vol. 225, pp. 147–51, nov 2003.

[66] V. M. Savage, A. B. Herman, G. B. West, and K. Leu, “Using Fractal Geometry and Universal Growth Curves as Diagnostics for Comparing Tumor Vasculature and Metabolic Rate With Healthy Tissue and for Predicting Responses to Drug Therapies,” Discrete and continuous dynamical systems. Series B, vol. 18, pp. 1077–1108, jun 2013.

[67] X.-P. Han, Q. Hao, B.-H. Wang, and T. Zhou, “Origin of the Scaling Law in Human Mobility: Hierarchical Organization of Traffic Systems,” Physical Review E - Statistical, Nonlinear and Soft Matter Physics, vol. 83, no. 3 Pt 2, p. 036117, 2011.

[68] R. Fernández-Salvador, R. García-Perea, and J. Ventura, “Reproduction and postnatal growth of the Cabrera vole, Microtus cabrerae , in captivity,” Canadian Journal of Zoology, vol. 79, no. 11, pp. 2080–2085, 2001.

[69] F. Oliveira, “Livro traz história improvável da expansão global do Airbnb,” pp. 1–3, 2017.

[70] M. Ježek, K. Stípek, T. Kušta, J. Červený, and J. Vicha, “Reproductive and morphometric characteristics of wild boar (Sus scrofa) in the Czech Republic,” Journal of Forest Science, vol. 57, no. 7, pp. 285–292, 2011.

[71] I. L. Boyd, T. Arnbo, and M. A. Fedak, “Biomass and energy consumption of the South Georgia populations of Southern elephant seals. In: Le Boeuf, B.J., Laws, R.M. (eds.). Elephant seals: population, ecology, behaviour and physiology, Berkeley, University of California Press, 98–117.,” in Proceedings of the elephant seal symposium (R. Le Boeuf, B.J., Laws, ed.), pp. 98–117, 1994.

[72] F. A. P. Colares, “Estudo de modelos n−lineares de crescimento em peixe−boi marinho Trichechus manatus manatus e peixe−boi-Amazoniano Trichechus inunguis (Mammalia: Sirenia) em cativeiro,” 2002.

[73] P. Mendoza, J. Velasquez, J. Sanchez, L. Davila, D. Loja, R. Rovers, and C. Vilchez, “Growth curve of Amazonian manatee (Trichechus inunguis) in captivity,” Aquatic Mammals, vol. 45, no. 4, pp. 389–397, 2019.

[74] A. T. Silva, J. W. B., Machado, J. R., Nobre, M. I. da S., Bezerra, “Policultivo da carpa comum, Cyprinus carpio (L., 1758) vt. comunnis, com macho da tilápia do Nilo, Oreochromis niloticus (L., 1766), alimentados com esterco de codorna, Nothura maculosa L. e milho, Zea mays L.,” Ciência Agron., Fortaleza, vol. 23, no. 1/2, p. 1130121, 1992.

[75] S. R. Silva, J. W. B., Bezerra, A. T., Sobrinho, A. C., Pereira, “Cultivo da tilápia do Nilo, Oreochromis niloticus (L., 1766), com manejo da densidade de estocagem e do peso dos peixes na sexagem,” Ciência Agron., Fortaleza, vol. 23, no. 1/2, pp. 75–83, 1992.

[76] V. Medri, G. V. Pereira, and J. H. Leonhardt, “Growth of Nile tilapia Oreochromis niloticus fed with different levels of alcohol yeast.,” Revista brasileira de biologia, vol. 60, no. 1, pp. 113–121, 2000.
Appendix A: Ontogenetic Models

It is presented in this supplementary material section the ontogenetic models that were used to describe the temporal dynamics of mass, length and energy of the organisms analysed, and consequently the ontogenetic parameters used. It is discussed here the Bertalanffy-Richards model, which describes the time evolution of the organism mass; the length-weight relationship, which is the empirical evidence that the organism’s mass and length obey a power law relation; version of the Bertalanffy-Richards Model for the time evolution of length; and finally, the West-Banavar model, which describes the time evolution of the organisms’ maintenance energy.

1. Bertalanffy-Richards Model

The Bertalanffy-Richards Model [31, 32], which was presented in the main part of the paper (see Eq. (3)) describes the time evolution of the organism’s mass [26, 51, 52]. The solution of this model if given by Eq. (7), and it is written in terms of the ontogenetic parameters $A, B, m_0,$ and $\beta$, whose biological means was discussed in the main text.

Given that the anabolism and catabolism constants, $A$ and $B$ respectively, are related one each other by the constraint given by Eq. (8), it is possible to eliminate one of this parameters in the solution if we have available the organism’s saturation mass $M$. In this way it is possible to write the solution of this model in at least two way, depending on the type of parameter to be investigated. The first one is writing the solution in an explicit form of the catabolism constant $B$, and then eliminating the de- pence with the anabolism constant $A$. That is, inserting the Eq. (8) to the solution (7), which yields

$$m(t) = M \left[ 1 + \left( \frac{m_0}{M} \right)^{1-\beta} - 1 \right] e^{B(\beta-1)t} \left[ 1 - \beta \right]. \quad (A1)$$

In this case we can say that $m(t) = m(t|M, B, m_0, \beta)$, expliciting the depentence of the solution to these four ontogenetic parameters.

Alternatively, maintaining explicit the anabolism constant by eliminating the catabolism constat, one gets

$$m(t) = M \left[ 1 + \left( \frac{m_0}{M} \right)^{1-\beta} - 1 \right] e^{A(\beta-1)t} \left[ 1 - \beta \right]. \quad (A2)$$

In this case one can write that $m(t) = m(t|M, A, m_0, \beta)$. Of course, these two ways of writing the model’s solution only works when the saturation mass of the organism is available, which happens when $\beta < 1$. 
2. Length-weight relationship

The next model is in fact the empirical evidence of the power-law relation between mass and length of organisms: the so-called length-weight relationship LWR \[53\]. That is, the mass \(m\) of the organism scales with its linear body length \(l\) as

\[ m = al^b, \quad (A3) \]

where \(a\) and \(b\) are parameters of this model which can be obtained by data-fitting. The relation \[(A3)\] is commonly and traditionally described in fisheries experimental data \[53\], and nowadays it is applied for many purposes, for example, to predict biomass in large marine mammals \[54, 55, 56, 57\], due to difficulties in experimental measurements.

The empirical parameters \(a\) and \(b\) carry biological information, that could be any biotic \[58, 59, 60\] or abiotic effect \[34, 61\], or a meaningful taxonomic information \[62, 63\].

3. Length time evolution

The next model is the junction of LWR and the Bertalanffy-Richards model, which results in the length time evolution, which also obey a Betalananffy-Richards dynamics.

To demonstrate this consider firstly the derivative of the LWR \[(A3)\], that is

\[ \frac{dm}{dt} = abl^{b-1} \frac{dl}{dt}. \quad (A4) \]

Parallel we can insert \[(A3)\] into \[(A3)\] to get

\[ \frac{dm}{dt} = A(al)^b - Bal^b. \quad (A5) \]

Equaling these two last equations, one gets the ODE

\[ \frac{dl}{dt} = Cl^\delta - Dl, \quad (A6) \]

which shows that the length \(l\) is also governed by a Betertalanffy-Richards dynamics, where

\[ C \equiv \frac{Aa^{b-1}}{b}, \quad (A7) \]

\[ D \equiv \frac{B}{b}, \quad (A8) \]

and

\[ \delta \equiv b(\beta - 1) + 1. \quad (A9) \]

In fact, Essington et al. \[61\] present some empirical evidences that length \(l\) could be modelled by the Bertalanffy-Richards model. We coined Essington et al. relationships these three relationships \[(A7), (A8)\] and \[(A9)\], where the parameters related to length can be explained in metabolic terms.

The solution of the ODE \[(A6)\] has the same logistic-like behavior that emerges during ontogenetic body mass growth, that is

\[ l(t) = L \left[ 1 + \left( \frac{l_0}{L} \right)^{1-\delta} - 1 \right] e^{D(\delta-1)t} \quad (A10) \]

with \(L \equiv l(t \to \infty)\) been the saturation/assimptotic length, given by

\[ L = \left( \frac{C}{D} \right)^{\frac{1}{\delta}} \quad (A11) \]

or symply

\[ L = \left( \frac{M}{a} \right)^{\frac{1}{\beta}}. \quad (A12) \]

The condition for the saturation to happen is that \(0 < \delta < 1\).

4. Energy allocated to maintanence

The last model to be presented is based on the seminal work of West et al. \[18\], which describe the temporal evolution of the energy maintenance of the organism (see also \[26, 65, 66\]). Using the idea that the energy in an organism \((R)\) is used for maintence \((NR_c)\) and growth \(E_c \frac{dN}{dt}\), conform described by Eq. \((2)\), then one can show that the ration between maintence energy and total energy, say

\[ r = \frac{\text{maintenance energy}}{\text{total energy}}, \quad (A13) \]

can be written (using the definition presented in section \((III)\) as

\[ r(t) = \left( \frac{m(t)}{M} \right)^{1-\beta}. \quad (A14) \]

Then, comparing this result with Eq. \[(A2)\], one gets

\[ r(t) = 1 + \left( \frac{m_0}{M} \right)^{1-\beta} - 1 \right] e^{\frac{A(\beta-1)}{M}t}, \quad (A15) \]
The quantity $r(t)$ reveals universal properties, been approximately the same for a huge amount of species, as demonstrated in the original paper and in other works [26, 65, 66]. We use this properties of universality in the present work as additional information to calibrate the methods and to get better estimated values for the ontogenetic parameters. More detail in the next section.

The $\beta$ exponent in (A15) was relaxed by Banavar et al [21], where it gave rise to doubts in the universality ideas suggested by West et al [18]. Moreover, they suggested a function acting in biomass saturation. Because this improvements helped us to make more accurate fit-to-data, we coined equation (A15) as West-Banavar (W-B) model.

**Appendix B: Getting metabolic parameters with the fitting curve with data and the models**

This supplementary material section presents the methodology used to determine the ontogenetic parameters’ values from the data. Those data are available on a sheet that contain mass ($m$), length ($l$) and time ($t$) for 43 ontogenetic trajectories. The data were extracted from the literature and can be classified into four groups:

- Terrestrial small-size mammals: Yangtze vole (*Microtus fortis calamorum*) [67], Sprague-Dawley rat (*Rattus rattus*) [60], Taiwan field mouse (*Apodemus semotus*) [66], Cabrera vole (*Microtus cabrerae*) [68].

- Terrestrial medium-size mammals: anglo nubian goat (*Ovis aries*) [69], wild boar (*Sus scrofa*) [70].

- Marine large-size mammals: gray whale (*Eschrichtius robustus*) [57], southern elephant seal (*Mirounga leonina*) [71], Amazonian manatee (*Trichechus inunguis*) [72, 73], Antillean manatee (*Trichechus manatus manatus*) [72].

- Fishes: rainbow trout (*Oncorhynchus mykiss*) [34], Nile tilapia (*Oreochromis niloticus*) [55, 74, 75, 76, 61].

One example of such available data is the one presented in table 1, which shows the time evolution of mass and length in the gray whale (*Eschrichtius robustus*). The methodology that will be described here compares those data with the ontogenetic growth equations (A1), (A2), (A3), (A10), and (A15). The intention is to extract, for each species, the ontogenetic parameters’ values that best describe, simultaneously, all these ontogenetic growth equations.

Some ontogenetic parameters are obtained by direct curve-fitting; others, however, can be obtained (or estimated) directly from the data, as is the case of parameters that govern the initial dynamics, as $m_0$ and $l_0$, and the ones that govern the saturation, as $M$ and $L$. We can use the empirical values of these parameters as a constraint to estimate the other ontogenetic parameters values with more precision.

The schema drawn in Fig. 7 describes the methodology used to fit the data simultaneously with all the five ontogenetic growth curves. The idea is to adjust the values of the parameters interactively with all growth curves in a recurrently way. This is done step by step until the values converge. The flowchart presented in this figure shows the order in which the growth curves are used, and the order in which the parameters are updated.

It is important to stress that the studies presented in the main text of this work are built by the knowledge of only four ontogenetic parameters: $A$, $B$, $M$ and $\beta$. To support the hypotheses proposed, it is crucial to have good accuracy in these parameters values for all analysed species. That is why this methodology uses five growth equations (Eqs. (A1), (A2), (A3), (A10), and (A15)) and, consequently, other ontogenetic parameters.

For instance, only the adjust (fit) of the model with mass and time data is enough to the get what we need (i.e. $A, B, M$ and $\beta$). However, if we restrict to this

| Age (years) | Age (days) | Length (cm) | Weight (g) |
|------------|------------|-------------|------------|
| 0          | -          | -           | -          |
| 1          | 365        | 851         | 6019000    |
| 2          | 730        | 922         | 7618000    |
| 3          | 1095       | 981         | 9155000    |
| 4          | 1460       | 1031        | 10590000   |
| 5          | 1825       | 1072        | 11901000   |
| 6          | 2190       | 1107        | 13077000   |
| 7          | 2555       | 1136        | 14119000   |
| 8          | 2920       | 1160        | 15033000   |
| 9          | 3285       | 1181        | 15828000   |
| 10         | 3650       | 1198        | 16515000   |
| 11         | 4015       | 1212        | 17105000   |
| 12         | 4380       | 1224        | 17611000   |
| 13         | 4745       | 1234        | 18042000   |
| 14         | 5110       | 1243        | 18408000   |
| 15         | 5475       | 1250        | 18719000   |
| 16         | 5840       | 1256        | 18982000   |
| 17         | 6205       | 1261        | 19205000   |
| 18         | 6570       | 1265        | 19393000   |
| 19         | 6935       | 1269        | 19551000   |
| 20         | 7300       | 1271        | 19685000   |
| 25         | 9125       | 1280        | 20094000   |
| 30         | 10950      | 1284        | 20266000   |
| 35         | 12775      | 1286        | 20338000   |
| 40         | 14600      | 1286        | 20368000   |

Table II: Data of mass, length and time for gray whale (*Eschrichtius robustus*) got from [57]. Similar data for others 42 ontogenetic trajectories are also available for our analyses.
model’s data analysis only, the parameters’ values obtained will be very noisy and imprecise. On the other hand, using this fit together and constrained to other growth equations allows us to get better and more precise values for the parameters (for instance, using \( a, b, \) and \( L \) from the mass-length relation). In fact, the final values obtained have an \( r^2 \) always higher than 0.87 for all analysed species.

Appendix C: Strategies of Savage et al.

The Kleiber’s Law \([1]\) and the relations between organism and cell properties (given by: \( R = NR_c \) and \( M = Nm_c \)) implies that the ratio between cellular metabolic rate \( R_c \) and cellular mass \( m_c \) must obey the scaling

\[
\frac{R_c}{m_c} = R_0 M^{\beta - 1}. \tag{C1}
\]

Based on this, Savage et al.\([22]\) namely two possible scaling scenarios the cell must obey. They are:

- **strategy 1**: cellular average mass remains fixed - scaling invariant -, but the cellular metabolic rate must be scaling dependent. That is
  \[
  m_c \sim M^0 \text{ and } R_c \sim M^{\beta - 1}. \tag{C2}
  \]
  This strategy is followed by typical cells;

- **strategy 2**: cellular metabolic rate remains fixed, but then cellular mass must be scaling dependent. That is
  \[
  m_c \sim M^{1-\beta} \text{ and } R_c \sim M^0. \tag{C3}
  \]

These strategies reflect the fact that cellular metabolic rate and cellular mass cannot both remain scale-invariant simultaneously.

Appendix D: Relation between \( R_c = R/N \) and \( R/M \)

In this supplementary material section we will show that when we have organism formed by two types of cell: typical and adipose cells, then \( R/N \sim R/M \) when \( M \) is sufficiently large. Identify such relation is important because in the mathematical model presented in section \([IV A]\) we gets \( R/M \), however the goal is to find a expression for \( R_c = R/N \). If we were considering an organism composed only of typical cells, the relationship between \( R/M \) and \( R/N \) is direct, but this is not exactly the case when we have an organism with a significant amount of adipocytes, which have different scaling properties.

First of all note that the total number of cells (the sum of the number of typical cells and the number of adipose cells) can be write as

\[
N = N^{(n)} + N^{(ad)} = \frac{M_n}{m_c^{(n)}} + \frac{M_{ad}}{m_c^{(ad)}}. \tag{D1}
\]

And from the hypotheses \([13]\) and \([14]\),

\[
N \sim (1 - \lambda)M + \lambda \cdot \text{cte} \cdot M^{1-\alpha}, \tag{D2}
\]

which give us how \( N \) scales with the organism mass when it is composed by a proportion \( \lambda \) of fat tissue. Note that for \( \lambda = 0 \) (organism composed only by typical cells), simply \( N \sim M \). However for any fixed \( \lambda \) one has that

\[
N \sim M + \text{cte} \cdot M^{1-\alpha}, \tag{D3}
\]

But note that if \( \alpha > 0 \) (which is supported by the empirical data, see Fig. \([5]\)), then the second tern on the right of this equation above is always smaller than the first. Then, for \( M \) sufficiently large only the first term on the right of this equation plays the rule, that is \( N \sim M \) for \( M \) sufficiently large and for individuals composed by both typical and adipose cells. In this way,

\[
R_c = \frac{R}{N} \sim \frac{R}{M}, \tag{D4}
\]

as we wanted to demonstrate.

Appendix E: Data Sheets

In this supplementary material section is attached all the data (and the respective references) used in the present work.

In the file \([\text{link}]\) it is presented all ontogenic trajectories used. That includes mass, length, and age for 42 species.

In the file \([\text{link}]\) It presents all the data related to adipocytes used in this work. It includes adipocyte volume, body mass, and the references in which the data was taken.
Gray Whale (*Eschrichtius robustus*)

Second ontogenetic phase

\[
m(t) = M \left( 1 + \left( \frac{M(t)}{M} \right)^{1-\beta} \right)^{\frac{1}{\beta}}
\]

Bertalanffy-Richards equation (in B terms)

\[
l(t) = L \left( 1 + \left( \frac{l(t)}{L} \right)^{1-\beta} \right)^{\frac{1}{\beta}}
\]

West-Banavar equation

\[
r(t) = \frac{m(t)}{M} \left( 1 + \left( \frac{m(t)}{M} \right)^{1-\beta} \right)^{1-\beta}
\]

Experimental data

| Age (days) | Length (cm) | Weight (g) |
|-----------|-------------|------------|
| 365,00    | 851,00      | 6.019.000,00 |
| 730,00    | 922,00      | 7.618.000,00 |
| 1.095,00  | 981,00      | 9.155.000,00 |
| 1.460,00  | 1.031,00    | 10.590.000,00 |
| 1.825,00  | 1.107,00    | 11.901.000,00 |
| 2.190,00  | 1.136,00    | 13.077.000,00 |
| 2.555,00  | 1.160,00    | 14.119.000,00 |
| 2.920,00  | 1.181,00    | 15.033.000,00 |
| 3.285,00  | 1.212,00    | 15.828.000,00 |
| 3.650,00  | 1.224,00    | 16.515.000,00 |

**Figure 7**: Diagram showing how we proceed with the data and models to get the values of the ontogenetic parameters (*A*, *B*, *β*, *M*, *L*, *a*, and *b*) for a specific species. The gray whale data was used in this particular example. The tick arrows show how the data are inserted into the models, while the thin arrows show how the ontogenetic parameters’ values are recurrently obtained and feedback the other models. This process is fulfilled in all 42 ontogenetic trajectories available in the database.

**A = 4,00; B = 0,0009; β = 0,50; L = 1286,00 cm; M = 20.700.000,00 g; a = 0,01; b = 2,95**