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COMMUNICATIONS ARISING

Ontogenetic growth

Modelling universality and scaling

Understanding the allocation of metabolic energy between the sustenance of an organism and its growth is an important issue in ecology. West *et al.*[1] have built on their earlier attempts to explain the exponent for the allometric scaling of metabolism and have derived a single, parameterless universal curve that describes the growth of many species. Here we show that the universal curve arises from general considerations that are independent of the specific allometric model used by West *et al.* and that the data do not distinguish between a 3/4 or 2/3 exponent in the relationship between metabolic rate and mass scaling.

von Bertalanffy[2] (see his equation (5)) considered a general equation of the form $dm/dt = a_\alpha m^\alpha - b_\alpha m^\beta$, where m is the body's mass at time t, and α and β are unspecified exponents, and a_α and b_α are positive coefficients. The equation considered by West *et al.*[1] is a special case, with $\alpha = 3/4$ and $\beta = 1$, whereas von Bertalanffy studied the case with $\alpha = 2/3$ and $\beta = 1$. Such an equation can be cast in a scaling form: $dm/dt = m^\alpha f(m/M)$, where M provides a scale for the organism's mass and is usually chosen to be the asymptotic maximum body size. The scaling function f approaches a constant value for a small argument — when $m(t)$ is small compared with M, relatively little energy is required to sustain the organism and virtually all of the metabolic energy is funnelled into growth processes. The requirement that $dm/dt = 0$ when m reaches the value M leads to the condition that $f(1) = 0$.

Moreover, the initial condition of the mass at $t = 0$ being equal to the birth mass, m_0, must be satisfied. For von Bertalanffy's equation[2], $f(x) = a_\alpha(1 - x^{1-\alpha})$, with $a_\alpha = b_\alpha M^{1-\alpha}$.

We have analysed the data of West *et al.*[1] on the cow, hen and guppy to assess whether it is possible to discriminate between the two choices for α. We find that their equation (5) (which is a special case of equation (6) of ref. 2), written in the form

$$(m/M)^{1-\alpha} = 1 - [1 - (m_0/M)^{1-\alpha}]e^{-\gamma_\alpha t} \qquad (1)$$

or, equivalently, as

$$r = 1 - e^{-\tau} \qquad (2)$$

(with the dimensionless mass ratio $r = (m/M)^{1-\alpha}$ and the dimensionless time $\tau = -\ln[[1 - (m_0/M)^{1-\alpha}]e^{-\gamma_\alpha t}]$), fits the observations equally well for both values of α (Fig. 1).

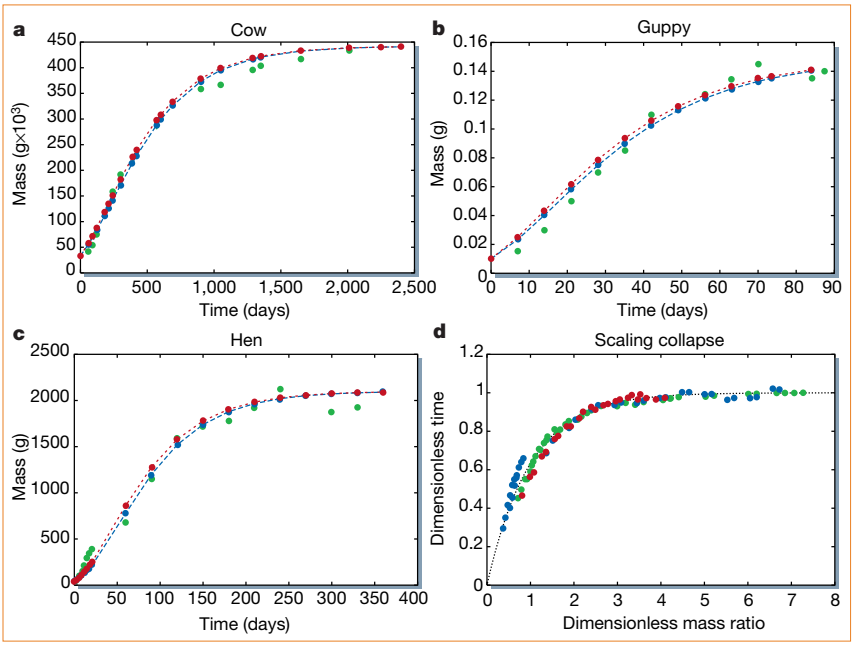

Figure 1 Growth curves for three different organisms and the collapse of mass scaling. **a–c,** Growth curves for **a**, cow; **b**, guppy; and **c**, chicken. Green, empirical data; blue, best fit obtained by West *et al.*[1] (that is, $\alpha = 3/4$); red, plot of equation (1), with $\alpha = 2/3$ and the values of M and γ as obtained in ref. 1. **d,** Scaling collapse. The universal growth curve (equation (2); dotted line) is derived from data from the three species (green, cow; blue, hen; red, guppy) for both $\alpha = 3/4$ and $\alpha = 2/3$.

For simplicity, we have chosen M from Table 1 of ref. 1. We determined $\gamma_\alpha = a_\alpha(1-\alpha)/M^{1-\alpha}$ using the values of $a_{3/4}$ from the same table and $a_{2/3}$ for the cow, hen and guppy to be 0.62, 0.67 and 0.064, respectively, to ensure that $\gamma_{2/3} = \gamma_{3/4}$ for simplicity. (We have not tried to adjust the value of M and γ to achieve a better fit because that is beside the point here.) Also, an equally good fit of the universal curve is obtained (Fig. 1d) for the three species with both values of α. Furthermore, the form of the universal curve (equation (2)) is independent of the value of α. Thus, the existence of a universal curve indicates nothing about the value of α.

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West et al. *reply* — Of the equations that have been used to describe ontogenetic growth in terms of the rate of increase in mass, m, as a function of time, t, most are merely statistical descriptions with no mechanistic basis. Our model[1] is derived from fundamental biological and physical principles and relates growth to metabolic power at the cellular level. It is based on the allocation of resources to the maintenance and replacement of existing tissue and the production of new tissue, with the whole-body metabolic rate $B = N_c B_c + E_c(dN_c/dt)$, where B_c is the cellular metabolic rate, E_c is the energy needed to create a cell, and N_c is the total number of cells. As $m = N_c m_c$, where m_c is the average cell mass, this gives

$$dm/dt = am^\alpha - bm^\beta \qquad (1)$$

where $a = B_0 m_c/E_c$, $b = B_c/E_c$, $\beta = 1$ and α *is* the allometric exponent for B ($\equiv B_0 m^\alpha$), taken to be 3/4 in accordance with a large body of data and with theoretical arguments[2,3]. The asymptotic mass, for which $dm/dt = 0$, is predicted to be $M = (B_0 m_c/B_c)^4$.

Equation (1) reflects the diminishing capacity of fractal-like distribution networks to supply resources as body size increases. It has no 'arbitrary' parameters; all exponents and coefficients are derived from measured

fundamental quantities that are not directly related to growth, such as m_c, E_c, B_c and B_0.

Equations of the form (1) were originally proposed by von Bertalanffy[4], who suggested that growth rate is the difference between anabolic rate, B_a (biomass production, scaling as $m^{2/3}$), and catabolic rate, B_c (biomass breakdown, scaling as m): $dm/dt \propto B_a - B_c$. This case, which is considered by Banavar *et al.*, corresponds to $\alpha = 2/3$, $\beta = 1$, with a, b (and M) being 'arbitrary' parameters determined by fitting growth data. No explanation or derivation is given for any of these parameters, so the authors' version of equation (1) is simply a curve-fitting statistical description.

The assertion that $\alpha = 2/3$ by Banavar *et al.* is at odds with their earlier theoretical argument[2] for a 3/4 exponent for B. In any case, von Bertalanffy's explanation (and that of Banavar *et al.*) for the origin of equation (1) cannot be correct, as both B_a and B_c scale as $m^{3/4}$, leading to $dm/dt \propto m^{3/4}$, or $m \propto t^4$ for all times.

To reveal the universality of growth that is implied by equation (1), we showed that, by plotting $r \equiv (m/M)^{1/4}$ against $\tau \equiv at/M^{1/4} - \ln[1-(m_0/M)^{1/4}]$, all organisms conform to a predicted universal curve, $1 - e^{-\tau}$. Banavar *et al.* observe that a similar plot can be generated by using an unrealistic $\alpha = 2/3$, rather than $\alpha = 3/4$. Most data on ontogenetic growth are not of sufficient quality to distinguish between the two: we recognized this and made no claim that $\alpha = 3/4$ is a better fit than $\alpha = 2/3$. However, the statement by Banavar *et al.* that this curve is independent of α is misleading because r and τ depend explicitly on α, so the scaling curve cannot be constructed without knowing its value, as well as the values of a and b. (Indeed, Banavar *et al.* use our values based on a 3/4 power.)

Banavar and colleagues' comment misses our central point that, because equation (1) is derived from fundamental principles concerning how growth is fuelled by metabolic power at the cellular level, many important quantities can be understood quantitatively. For example, our model elegantly interprets r as the proportion of total lifetime metabolic energy that is devoted to maintenance and other activities.

We further contend that the implication made by Banavar *et al.* that their equation $dm/dt = m^{\alpha} f(m/M)$ is the most general form of the growth equation is also misleading. The function f depends on several variables, including m, M, B, m_c, E_c, B_c, cell growth and lifetime, time to maturity, and so on. Without a specific mechanistic model, why should f depend only on m/M, and what sets the fundamental timescale for growth? Our equation (1) answers these and other questions. It contains, derives and predicts many fundamental biological and physical variables that capture the essential features of ontogenetic growth, yet it yields an extraordinarily simple universal equation.

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COMMUNICATIONS ARISING

Climate change

Regional warming and malaria resurgence

Disease outbreaks are known to be often influenced by local weather, but how changes in disease trends might be affected by long-term global warming is more difficult to establish. In a study of malaria in the African highlands, Hay *et al.*[1] found no significant change in long-term climate at four locations where malaria incidence has been increasing since 1976. We contend, however, that their conclusions are likely to be flawed by their inappropriate use of a global climate data set. Moreover, the absence of a historical climate signal allows no inference to be drawn about the impact of future climate change on malaria in the region.

The findings of Hay *et al.*[1] are based on interpolation to four locations, from a 0.5°-resolution gridded climate data set[2,3], within an area of large altitudinal contrasts and sparse, geographically dispersed historical climate data. In such a region, these gridded climate anomalies are often based on data outside a particular grid cell, and their interpolation to specific sites ignores local elevational dependencies. The mean site altitudes used by Hay *et al.* (1,693 m, 1,819 m, 1,893 m and 2,031 m), compared with those of the actual input weather-station sites (506 m, 1,110 m, 1,312 m, 1,515 m, 1,624 m and 1,635 m), differ on average by 575 m, which corresponds to a temperature deviation of 3 °C. The sparse weather stations also range over a wide expanse of 5° latitude and 7° longitude. This climate data set is appropriate for up-scaling to African regions, but not for down-scaling to specific area locations; it cannot therefore support the type of analysis carried out by Hay *et al.*

Hay *et al.* focus on climate trends, but 'climate change' also applies to changes in variability. In regression analysis, a trend in covariates is not necessary; a change in variance can yield larger or more frequent responses. In the African highland, increases in the magnitude or frequency of malaria epidemics are most closely associated with short-term climate anomalies[4–6]. Because of the existence of critical climate thresholds, the association between change in malaria incidence and change in climate can be biologically meaningful, even without 'significant' climate change.

Based on an understanding of the limitations of the gridded climate data set[3] and on an examination of individual station data[7–9], we calculate that in the east African region encompassing the four study sites there was a mean warming trend of 0.15 °C per decade during 1970–98, aggregated across the 320 0.5° grid boxes (Fig. 1). This regional warming tells us little, however, about climate trends at specific sites[9], as these data[2,3] are not designed to reveal such information.

In contrast to Hay *et al.*, we have identified regional warming trends in east Africa that parallel rising trends in malaria inci-

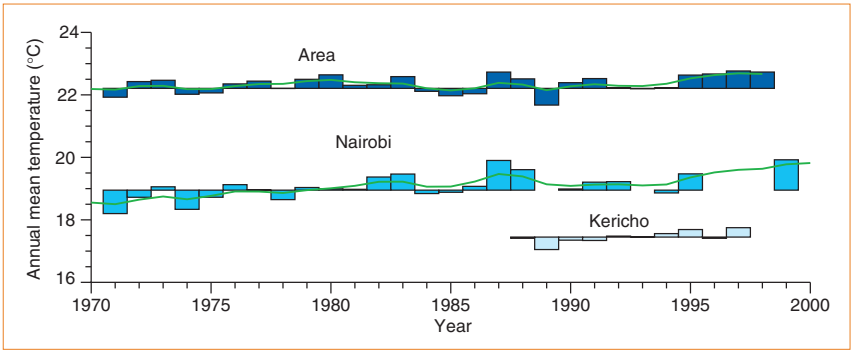

Figure 1 Annual mean temperature for Nairobi airport (WMO 63741, 1.3° S, 36.9° E, 1,624 m) and Kericho (0.37° S, 35.35° E, 2,031m) are plotted as bars to show deviations from the averages for 1961–90 (19.0 °C) and 1988–97 (17.4 °C), respectively. These station records are complemented with a large-area average from a 0.5°-gridded time-series defined by 4° S, 4° N, 28° E, 38° E (refs 2,3). The area-averaged annual mean temperature is plotted (top) as bars that display deviations from the 1961–90 average (22.2 °C). Time series for Nairobi airport and area-averaged data are also plotted after smoothing with a 10-year gaussian filter to emphasize changes on decadal timescales. In addition to the observed temperature trends, note the marked altitudinal dependency in temperatures.