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What is the status of metabolic theory one century after Pütter invented the von Bertalanffy growth curve?

Michael R. Kearney*

BioSciences4, School of BioSciences, The University of Melbourne, Victoria 3010, *Australia*

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*E-mail: m.kearney@unimelb.edu.au; Tel.:61422502210 .

ABSTRACT

Metabolic theory aims to tackle ecological and evolutionary problems by explicitly including physical principles of energy and mass exchange, thereby increasing generality and deductive power. Individual growth models (IGMs) are the fundamental basis of metabolic theory because they represent the organisational level at which energy and mass exchange processes are most tightly integrated and from which scaling patterns emerge. Unfortunately, IGMs remain a topic of great confusion and controversy about the origins of the ideas, their domain and breadth of application, their logical consistency and whether they can sufficiently capture reality. It is now 100 years since the first theoretical model of individual growth was put forward by Pütter. His insights were deep, but his model ended up being attributed to von Bertalanffy and his ideas largely forgotten. Here I review Pütter's ideas and trace their influence on existing theoretical models for growth and other aspects of metabolism, including those of von Bertalanffy, the

Dynamic Energy Budget (DEB) theory, the Gill-Oxygen Limitation Theory (GOLT) and the Ontogenetic Growth Model (OGM). I show that the von Bertalanffy and GOLT are minor modifications of Pütter's original model. I then synthesise, compare and critique the ideas of the two most-developed theories, DEB theory and the OGM, in relation to Pütter's original ideas. I formulate the Pütter, DEB and OGM models in the same structure and with the same notation to illustrate the major similarities and differences among them. I trace the confusion and controversy regarding these theories to the notions of anabolism, catabolism, assimilation and maintenance, the connections to respiration rate, and the number of parameters and state variables their models require. The OGM model has significant inconsistencies that stem from the interpretation of growth as the difference between anabolism and maintenance, and these issues seriously challenge its ability to incorporate development, reproduction and assimilation. The DEB theory is a direct extension of Pütter's ideas but with growth being the difference between assimilation and maintenance rather than anabolism and catabolism. The DEB theory makes the dynamics of Pütter's 'nutritive material' explicit as well as extending the scheme to include reproduction and development. I discuss how these three major theories for individual growth have been used to explain 'macrometabolic' patterns including the scaling of respiration, the temperature–size rule (first modelled by Pütter), and the connection to life history. Future research on the connections between theory and data in these macrometabolic topics have the greatest potential to advance the status of metabolic theory and its value for pure and applied problems in ecology and evolution.

Key words: metabolic theory, growth model, dynamic energy budget, von Bertalanffy growth, ontogenetic growth model, life history, temperature size rule, scaling.

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I. INTRODUCTION

A living thing can be thought of as a set of chemical transformations whereby food is converted into a more ordered biomass through the process of metabolism under the instruction of internally encoded information. These chemical transformations produce more biomass, i.e. growth, and underlie development and reproduction; they also only proceed if energy and materials are spent on maintenance of the system itself. The process of metabolism has been considered deeply from a physical and chemical point of view since the 1920s. Pütter (1920) developed the first theoretical growth model one century ago. Lotka (1925, pp. 14–18) emphasised how the chemical transformations of growth necessarily have a spatial structure and that the different parts must grow in harmony and maintain a suitable environment. These notions of integration and internal stability were coined ‘homeostasis’ by Cannon (1926). Despite this promising early theoretical start, the subsequent study of individual growth and other aspects of metabolism has not been particularly integrated or harmonious. In fact, it has been suggested that we presently face a crisis in our theoretical models of growth and the associated processes of development and reproduction (Marshall & White, 2018). General theories of metabolism have been proposed since Pütter’s original work, but there is no consensus on their relative merits, major misunderstandings of what they actually imply (van der Meer, 2006; Kearney & White, 2012; Maino *et al.*, 2014), and new ideas continue to be proposed (Sibly & Brown, 2020). Yet we need to consolidate a strong theory of metabolism if we are to understand life histories, population growth and the responses of species to environmental change.

(1) Individual growth and metabolic theory

The notion of 'metabolic theory', especially in ecology, has come to represent the potential power of interpreting processes at different hierarchical levels, from molecules to ecosystems, on the basis of the way individuals take up and transform energy and matter (Nisbet *et al.*, 2000; Brown *et al.*, 2004*a*). Individuals are a special organisational level in this respect because of their high degree of integration, coordination and feedback as they attempt to maintain homeostasis; individuals have a 'target' state that they head towards in a coordinated fashion when perturbed but the same cannot be said at the population, community or ecosystem level. Explicit consideration of the individual level also permits the definition of fitness and selection strength at the population level such that the evolution of life-history traits can be analysed. Much work that has been published under the umbrella of the 'metabolic theory of ecology' has shown that a variety of phenomena at scales above the individual can be seen as representing patterns manifesting at the individual level, especially the scaling of metabolic rate with size and temperature (Brown *et al.*, 2004*a*; Sibly, Brown & Kodric-Brown, 2012). This descriptive aspect of metabolic theory extends a long history of work in comparative biology (Peters, 1983). But, without an underlying theory for metabolism at the level of the individual, we are merely left with a 'metabolic pattern of ecology'.

The term 'metabolic rate' or 'metabolism' is closely associated with direct or indirect (e.g. respiration) measures of heat production by organisms. This is because heat production directly links to the use of energy for work in most organisms. But metabolism is of course much more than heat production; it includes all the processes by which an organism takes up and transforms energy and materials for growth, development, maintenance and reproduction. Individual

‘growth’ models (IGMs) aim to capture some or all of these processes at one or more life stages. The theory underlying IGMs is thus the foundation for metabolic theory in general.

The aim of this article is to review and synthesise the most notable developments in metabolic theory over the past century, especially as they relate to individual growth. I do this in the context of Pütter’s (1920) fundamental contribution to the subject which has been largely overlooked, for reasons to be explained, but which blazed an important conceptual trail. The focus of this article is on mechanistic as opposed to phenomenological models of growth and metabolism. The distinction between these approaches to modelling has been a source of confusion and controversy in metabolic theory (e.g. Cyr & Walker, 2004; White & Marshall, 2019) and so warrants further discussion.

(2) Mechanistic *versus* phenomenological models

As scientific fields mature, they inevitably wrestle with the tension between description and explanation. The study of organismal growth and metabolism is no exception. At one extreme, quantitative models may act purely as descriptions of observed patterns. In such ‘phenomenological’ models (also called ‘empirical’ or ‘correlative’ in some fields), the form of the equation and the statistically fitted parameters are not interpreted explicitly in the context of underlying processes. At the other extreme, a mechanistic model is derived from a list of assumptions (a theory) about the processes thought to underlie the phenomenon. Thus, the forms of the equations and the associated variables come directly from the theoretical assumptions, and the parameters are physically interpretable with meaningful dimensions. The distinction between empirical and phenomenological models can be fuzzy. For example, subprocesses of a generally

mechanistic model may sometimes be represented by statistical approximations, and an empirical model's functional form and parameters may be chosen with particular mechanisms in mind. Empirical models play an important pragmatic role in science but ideally one aims for theoretically based mechanistic models because they may provide greater insight, suggest new observations in a more explicit and testable fashion, make more robust predictions with the same model structure and parameters, and have greater coherence with other scientific fields. For example, it is possible to model soil temperature as a function of air temperature and rainfall using sophisticated statistical models (Horton & Corkrey, 2011) or using physical models of energy and mass transfer (Kearney *et al.*, 2014). The former, phenomenological approach requires extensive observational data at the level of the phenomenon in question (i.e. of soil temperature) to fit the parameters and only applies to conditions within the domain of the data to which it was fitted (e.g. Australia). The latter, mechanistic approach requires more specific forcing data and parameters representing the underlying processes (e.g. thermal conductivity and solar absorptivity of the soil) but its applicability is extended to wherever the underlying theory holds – in the case of a mechanistic soil temperature model it should apply anywhere on earth and even to other planets.

In the case of IGMs, as with soil temperature models, we are considering phenomena of energy and mass exchange which must obey the laws of thermodynamics. The particular way growth and reproduction is structured in organisms of course represents the action of natural selection, which has its own mechanistic formulations. But, the processes of natural selection on growth and metabolism must conform to the more fundamental thermodynamic constraints. Thus, the more explicitly we can link IGMs to the general principles of energy and mass exchange from

chemistry and physics, the greater will be the generality, the coherence with other fields, and the predictive capacity of the models (see von Bertalanffy, 1960, p. 170, for further discussion).

(3) Putting the history of metabolic theory into context

August Pütter (1879–1929) was a German physiologist best known for his ideas on the potential role of dissolved organic matter in the energy budgets of aquatic animals (Pütter, 1907, 1909; reviewed in Johnston, 1972; Jørgensen, 1976). But in 1920 he derived an equation that accurately captured the growth of many organisms on the basis of simple surface area and volume relationships (Pütter, 1920). In so doing, he provided hope that the complexities of growth at the molecular level could be captured by simpler dynamics at the whole-organism level.

In this review I will discuss some of the major ideas that have arisen since Pütter's work (Fig. 1) and argue that substantial progress has been made on the development of metabolic theory, but that this progress has been hampered by lack of a proper synthesis and comparison of existing ideas. In so doing, I will compare Pütter's theory in detail with the two existing major attempts at a mechanistic synthesis of individual growth and metabolism: Dynamic Energy Budget (DEB) Theory and the Ontogenetic Growth Model (OGM).

To facilitate this comparison, a summary of the parameters and variables of all three models is provided as online Supporting Information in Tables S1–S3 and a summary of all the major equations is provided in Table 1, using the notation conventions of Kooijman (2010) (see legend to Table 1 for explanation). As the example growth curves of Fig. 2 show, all three theories similarly predict juvenile growth under constant food intake but, as discussed in Section II, the theories have vastly different capacities to explain the web of interactions between growth,

development, reproduction and their consequences for 'macrometabolic' patterns including stoichiometry, scaling and life history. It is hoped that by consolidating the theoretical ideas on individual growth and hence metabolism over the past century, we can develop a more coordinated, efficient and integrated theoretical and empirical approach to this topic.

II. THE GROWTH MODELS

(1) Pütter's theory of growth

Pütter's equation in integrated form was:

$$L = L_\infty - (L_\infty - L_0)e^{-\dot{r}_B t} \tag{1}$$

where L is length at time t, L_∞ is the ultimate length, L_0 is the length when t is zero and $\dot{r}_B$ is a growth rate coefficient, resulting in an asymptotic growth pattern (Fig. 2A). This equation closely describes the growth of organisms as diverse as comb jellies, lobsters, fish and birds (Pütter, 1920; Kooijman, 1988) and is now famously known as the von Bertalanffy growth equation. How did Pütter derive it, and why is it named after someone else?

Pütter conceived the rate of change in volume of biomass $V = L^3$ at a given moment as the balance between a surface-related build-up (anabolic) process with coefficient $\{\dot{k}_a\}$ and a volume-related breakdown (catabolic) process with coefficient $[\dot{k}_C]$, i.e.

$$\frac{dV}{dt} = \{\dot{k}_a\}V^{2/3} - [\dot{k}_C]V. \tag{2}$$

Pütter emphasised that it was not necessary to know which surface the build-up process was dependent upon (e.g. gut, gills, intracellular features, etc.), and that the proportionality constant $\{\dot{k}_a\}$ may therefore have to be indirectly determined. The breakdown process was assumed to be directly proportional to the volume of tissue produced, according to the proportionality constant $[\dot{k}_C]$. At the start of growth, the build-up term $\{\dot{k}_a\}V^{2/3}$ is necessarily larger than the breakdown

term $[\dot{k}_C]V$, and becomes increasingly so for a while in absolute terms (Fig. 2B). But, because the breakdown term increases faster with size than does the build-up term, growth rate ultimately tends to zero (Fig. 2C) and growth is asymptotic (Fig. 2A).

The ultimate size the organism reaches, L_∞, emerges from this equation at this steady-state point. That is, if $\frac{dV}{dt}$ is set to zero and the equation is rearranged, L_∞ can be conceived as the ratio of the anabolic and catabolic coefficients (Table 1). Pütter emphasised that 'length' in his model was not empirical but a more abstract concept of the cubic root of the of the volume, and he connected it to observed lengths L_w and wet masses (W_w) (Fig. 2D) by a simple conversion

$$W_w \cong V = L^3 = \delta_W {L_w}^3 \tag{3}$$

where δ_W represents the mass of a unit-length (e.g. 1 cm) individual and thus $L = \delta_W^{1/3} L_w$ under the assumption that density is one cubic cm per gram and that the organism does not change shape in the chosen length measure as it grows (isomorphy).

The anabolic coefficient $\{\dot{k}_a\}$ (lower case 'a' to distinguish anabolism from assimilation) was regarded by Pütter as controlling the concentration of nutritive materials available for anabolic reactions, i.e. for maintenance processes and the work (overhead costs) involved in growth. To capture this concept, Pütter used the metaphor of water pouring into a container with an outlet hole. The height of the water in the container represents the steady-state concentration of nutrients for growth. The flow to maintenance and overhead costs $[\dot{k}_M]$ offsets the available concentration for the anabolic processes and, in the metaphor, represents the size of the outlet hole. The height of the container can be thought of as being proportional to the maximum possible value of $\{\dot{k}_a\}$, i.e. $\{\dot{k}_{am}\}$. However, this may be reduced to a lower maximum level according to a limitation of the form

$$\{\dot{k}_a\} = \{\dot{k}_{am}\}\frac{[\dot{k}_N]}{[\dot{k}_N] + [\dot{k}_M]} \tag{4}$$

where $[\dot{k}_N]$ is a nutrient diffusion coefficient controlling how fast the nutritive materials move to the site of anabolic reactions. As $[\dot{k}_N]$ becomes very large, $\frac{[\dot{k}_N]}{[\dot{k}_N]+[\dot{k}_M]}$ approaches 1, which metaphorically represents water overflowing from the brim of the container, and $\{\dot{k}_a\}$ becomes $\{\dot{k}_{am}\}$. But at sufficiently lower nutrient diffusion rates, or with higher maintenance or overhead costs, the maximum concentration of growth materials will decrease, i.e. there will be a lower water level in the container. Thus, the concentration of nutrients supplying growth drops and the breakdown rate per volume $[\dot{k}_C]$ stays constant, so the organism will reach a lower ultimate size L_∞ than the maximum possible size $L_m = \frac{\{\dot{k}_{am}\}}{[\dot{k}_C]}$.

Finally, by substituting equation (4) into equation (2) and integrating, L can be specified as a function of time as in equation (1), thus deriving the 'von Bertalanffy' growth equation where the growth rate parameter $\dot{r}_B = [\dot{k}_C]/3$ and is therefore interpreted as being driven by volume-specific catabolic rate. Note that Pütter actually wrote equation (1) as $L = L_\infty - L_\infty \alpha e^{-\frac{c}{L_\infty}t}$ with $\alpha = 1 - L/L_\infty$ and implying $c = \{\dot{k}_{am}\}/3$. However, he later erroneously defined $\frac{c}{L_\infty}$ as equal to $\{\dot{k}_{am}\} - [\dot{k}_C]$ (clearly a dimensional error) and then proceeded to estimate $\{\dot{k}_{am}\}$ and $[\dot{k}_C]$ from data on the basis of ultimate physical lengths [not the abstracted dimension of equation (3)] and estimates of the parameter c. von Bertalanffy (1934) was aware of this problem and used it as part of his general dismissal of Pütter's theoretical interpretation.

(2) von Bertalanffy's contribution

The Austrian biologist Ludwig von Bertalanffy (1901–1972) made important contributions to theoretical biology, including a series of papers about organismal growth (e.g. von Bertalanffy, 1934, 1938, 1957, 1960). He was the first to define an organism as an open, stationary chemical system (von Bertalanffy, 1950) – one that heads to a time-independent (steady) state where the net inputs and outputs are balanced on a macro-scale, even though there is continued exchange on a micro-scale. That is, it grows to a final size determined by the underlying chemical reactions that determine the inputs and outputs of material. Following Pütter, von Bertalanffy ascribed inputs and outputs to the processes of anabolism and catabolism, where the fraction breaking down is a fixed proportion of the total amount and construction is proportional to 'some surface'. However, von Bertalanffy focused on mass as a state variable whereas Pütter focused on volumes and surface areas.

Despite the strong similarities in reasoning, von Bertalanffy (1938) only acknowledged Pütter for his equation $\frac{dW}{dt} = \{\dot{H}\}s - [\dot{k}]W$, where W is mass $[\dot{k}]$ is a constant for destruction per unit mass, $\{\dot{H}\}$ is a constant representing the synthesised mass per surface area and s is the surface area. This 'fictitious' surface responsible for growth was considered by von Bertalanffy to be unknown and not directly determinable, but proportional to $W^{2/3}$. Whereas Pütter's notion of length was an abstraction (cube root of volume), von Bertalanffy's length was actual. But, because von Bertalanffy worked with an abstract surface area, he included conversions from real length to abstract surface area.

Pütter's growth equation became known by von Bertalanffy's name because of Beverton & Holt's (1957) influential book on fish population dynamics. They emphasised the power of equation (1) for characterising fish growth curves and inaccurately attributed it to von

Bertalanffy (1934, 1938). A retrospective reading of von Bertalanffy's work makes it clear how his ideas are derived from those of Pütter, and he did acknowledge Pütter in his 1934 paper (in German). But it is also easy to understand how Beverton & Holt (1957) missed the often subtle referencing by von Bertalanffy, especially in his English publications, to Pütter's work. Subsequently, von Bertalanffy also referred to Pütter's model as the 'Bertalanffy model' (von Bertalanffy, 1960).

von Bertalanffy generalised the Pütter model to be flexible on the exponents by which the anabolic and catabolic terms scale with mass, such that

$$\frac{dW}{dt} = aW^c - bW^d \tag{5}$$

where *a* and *b* are the anabolic and catabolic constants and *c* and *d* are their respective scaling exponents with body mass (von Bertalanffy, 1957).

(3) The Gill-Oxygen Limitation Theory

The Gill-Oxygen Limitation Theory (GOLT) of Pauly (1979, 2010) represents a more mechanistically-explicit version of Pütter and von Bertalanffy's ideas about the balance between anabolism and catabolism. Pauly took the position that mass was a primary state variable and length was subsidiary, as did von Bertalanffy. Pauly emphasised that von Bertalanffy's assumptions imply a link between anabolism and surface area. His main contribution was to argue that, for water-breathers, it was the surface involved in oxygen uptake that was limiting, i.e. the gills, since oxygen cannot be stored in large quantities. Pauly then demonstrated that gill surface area scales with mass with an allometric exponent and argued that, as fish and other water-breathers grow, their demand for oxygen ultimately outstrips the supply. Pauly used the general form of the von Bertalanffy model [equation (5)] for his models. This is because gill

surface area has a variable scaling with mass, whereby the mass scaling exponent increases with fish size, and so the value of the exponent *c* in equation (5) was allowed to vary accordingly. However, the underlying logic of the GOLT is the same as Pütter's, with an explicit focus on gills and oxygen.

(4) Dynamic Energy Budget theory

Dynamic Energy Budget (DEB) theory was developed by Kooijman (1986*a*,*b*, 1993, 2000, 2010), and it can be seen as a natural extension of Pütter's ideas to include food dependence, body condition, development, reproduction and embryonic growth while explicitly adhering to energy and mass conservation. In both theories, the input for growth is related to a surface and this is balanced against something that is proportional to a volume. In DEB theory this volume is called the 'structural volume' V and is the part of the biomass being built and maintained through the process of growth. Similarly to Pütter's theory, physical length L_w is related to the structural linear dimension L by the conversion parameter δ_L, so $L_w = V^{1/3}/\delta_L = L/\delta_L$. Length does not play a role in the theory *per se* but, conceptually, L is always interpreted as a ratio of volume to surface area in the case of isomorphy.

Both Pütter's theory and DEB theory include the notion of the 'mobilisation' of the nutritive materials for the processes of growth and maintenance. Pütter thought of the nutritive material in terms of its concentration, driven by a diffusion process. But he was not explicit about these dynamics nor about the amount of this nutritive material in his final growth model; he simply estimated his nutrition-related parameters $\{\dot{k}_{am}\}$ and $\{\dot{k}_N\}$ from data. As a result, his model has a single state variable, V. By contrast, DEB theory makes the nutritive material an additional state variable E, called the 'reserve', and models the dynamics of its accumulation and mobilisation in

relation to the structure. In addition, DEB theory uses the concept of a nutritive density, i.e. the amount of reserve per volume of structure $[E] = E/V$, rather than Pütter's idea of a concentration. Thus, the wet mass W_w is not simply the linear dimension (structural length) cubed but is the combination of the mass of reserve plus the mass of the structure:

$$W_w = d_V L^3 + E\frac{w_E}{\bar{\mu}_E} \tag{6}$$

where d_V is the density of structure (typically that of water, i.e. 1 g cm^{-3}), w_E is the molecular mass of reserve (grams per mole) and $\bar{\mu}_E$ is the chemical potential of reserve (joules per mole). Reserve is conceptualised as stored polymers (fats, carbohydrates and lipids) in 'blobs' within individual cells that must be accessed by the structure from the surface of the blobs at the subcellular level. Thus, the surface area interface of the reserve with the structure is a fundamental geometrical driver of the dynamics in DEB theory *via* the process of reserve mobilisation. The reserve dynamics of DEB theory makes the assumption that the interface between reserve and structure scales as $E/V^{1/3}$, based on arguments that isomorphy is strongly selected at the cellular level to minimise regulatory complexity as size changes (for further details, see Section 1.2.2 in Kooijman, 2010). The mobilisation rate has the symbol $\dot{p}_C$, with the C standing for catabolism, because the reserve is being broken down, and is a function of the density of the reserve $[E]$; the greater the reserve density, the faster it is mobilised because there is more reserve surface area relative to structure. It also depends on the inherent 'conductance' of the organism from an energetic point of view, represented by the parameter $\dot{v}$ (length per time), which can be thought of as representing the density of pores (or enzymes) at the interface between the reserve and structure.

Just as the height of Pütter's metaphorical container links to the maximum possible concentration of nutritive materials via $\{\dot{k}_{am}\}$, DEB theory has a maximum reserve density $[E_m] = \{\dot{p}_{Am}\}/\ \dot{v}$

where $\{\dot{p}_{Am}\}$ is the maximum potential assimilation rate per surface area of structure. The realised specific assimilation is an explicit function of the food available

$$\{\dot{p}_A\} = \{\dot{p}_{Am}\}\frac{X}{X+K} \tag{7}$$

where X is food density, K is the half-saturation constant and $f = \frac{X}{X+K}$ is thus a scaled Holling's type II functional response (Holling, 1959). There is a clear parallel between equation (7) from DEB theory and equation (4) from Pütter. But, in DEB theory, the density of nutritive material is directly related to assimilation and hence to food intake whereas in Pütter's model it is related to basal metabolism and a diffusion coefficient.

The rate of change of the reserve density in DEB theory was derived by Kooijman (2010, p. 37) to be

$$\frac{d[E]}{dt} = [\dot{p}_A] - \frac{\dot{v}[E]}{L} = \frac{\{\dot{p}_{Am}\}f - \dot{v}[E]}{L}. \tag{8}$$

Thus, the change in the reserve density depends on the input from assimilation minus what leaves the reserve *via* mobilisation. From equation (8), if the reserve density is not changing and f is 1, it is apparent that the steady-state value is $\frac{\{\dot{p}_{Am}\}}{\dot{v}} = [E_m]$, as stated above.

The rate of mobilisation of reserve per unit of structure is

$$[\dot{p}_C] = [\dot{p}_A] - \frac{d[E]}{dt} - [E]\dot{r} = \frac{[E]\dot{v}}{L} - [E]\dot{r}. \tag{9}$$

In equation (9), $\dot{r}$ is the growth rate of structure (see below) and the term $[E]\dot{r}$ accounts for dilution by growth. Equations (7–9) thus specify the reserve dynamics in DEB theory.

The dynamics of structure V in DEB theory, is

$$\frac{dV}{dt} = \frac{f\{\dot{p}_{Am}\}}{[E]+[E_G]}V^{2/3} - \frac{[\dot{p}_M]}{[E]+[E_G]}V = V\dot{r} \tag{10}$$

where $[E_G]$ is the energetic cost of making a unit volume of structure (tissue biomass energy content plus the energy needed to build the tissue), and $[\dot{p}_M]$ is the energetic cost to maintain a unit of structure per time. [Note that here and in the remainder of this section the DEB state variable of maturity, and the associated process of reproduction and parameter κ, is excluded for a clearer comparison with Putter, but see Table 1.] This is analogous to equation (2) from Pütter, especially when his factor $\{\dot{k}_{Am}\}$ is adjusted by $\frac{[\dot{k}_N]}{[\dot{k}_N]+[\dot{k}_M]}$ which is equivalent to f (Table 1). However, the surface-related process in DEB theory is assimilation rather than anabolism and the volume-related process is maintenance rather than catabolism. Also, both the assimilation and the maintenance terms have the additional state variable, reserve density $[E]$, in their denominators, thereby linking growth rate to nutritional condition (growth rate slows as condition declines). Locomotion costs are not explicit in DEB theory but, in the simplest cases, can be subsumed into the maintenance costs and/or food assimilation efficiency.

It is apparent from equation (10) that assimilation scales more slowly with structure (to the 2/3 power) than does maintenance (to the power 1). Thus, eventually maintenance takes all the mobilised reserve flux and growth ceases. It is also apparent from equation (10) that, when the rate of change in growth is zero, the ultimate length $L_\infty = \frac{f\{\dot{p}_{Am}\}}{[\dot{p}_M]}$, i.e. the ultimate size reflects the balance between assimilation and maintenance, not anabolism and catabolism which was the case in Pütter's theory.

The growth parameter $\dot{r}$ (not to be confused with $\dot{r}_B$) is defined as

$$\dot{r} = \frac{\dot{v}[E]/L - [\dot{p}_M]}{[E] + [E_G]} = \frac{f[\dot{p}_{Am}] - [\dot{p}_M]}{[E] + [E_G]} \tag{11}$$

with $\frac{[E]}{[E_m]}$ converging to $f = \frac{X}{X+K}$ under the assumption of constant food. Thus, the growth rate at constant food is a result of the difference between the nutritive material coming in *via* assimilation and that being lost to somatic maintenance, mediated by the reserve.

Finally, equation (10) in integrated form with respect to length becomes

$$L = L_\infty - (L_\infty - L_b)e^{-\dot{r}_B t} \tag{12}$$

with L_b being the length at birth and the growth rate parameter $\dot{r}_B$ is interpreted as

$$\dot{r}_B = \frac{[\dot{p}_M]}{3([E]+[E_G])}. \tag{7}$$

Thus, the standard DEB model simplifies to 'von Bertalanffy' growth under constant food [i.e. equation (1), Pütter's original equation], with the physiological interpretation of the von Bertalanffy growth rate parameter as being directly proportional to structural maintenance costs and inversely proportional to the sum of the current amount of reserve per unit structure and the cost of making a unit of structure. DEB models can also handle the effect of varying food through time on growth because they are explicit about reserves.

(5) The Ontogenetic Growth Model

One would expect processes of metabolism, including growth, to be driven by relationships between surface areas and volumes, such that scaling relationships involve powers of one third, as assumed by Pütter and in DEB theory. The major problem regarding this expectation is the frequently observed quarter-power scaling relationships of metabolic rates, and associated physiological and life-history rates, with body mass. The Ontogenetic Growth Model (OGM) of West, Brown & Enquist (2001) attempted to resolve this paradox by building on the idea that the delivery of resources through supply networks drives quarter-power metabolic scaling (West *et al.*, 2001; but see also Banavar *et al.*, 2010).

Like the Pütter model, the OGM uses mass as a state variable (i.e. $W = d_V V$). Its equation for growth rate is of the same mathematical form as that used by Pütter and von Bertalanffy. Like DEB theory, it uses the notion of maintenance rather than catabolism as the loss term in the growth model but, as explained further below, it considers growth rate to be determined by the difference between anabolic and maintenance rates.

The OGM assumes that the scaling relationship between anabolic rate and mass is the same as that for respiration and mass, i.e. 3/4, and it assumes that maintenance is directly proportional to body mass. Thus

$$\frac{dW}{dt} = aW^{3/4} - bW. \tag{14}$$

In this equation, a was interpreted by West *et al.* (2001) as $\frac{\{\dot{p}_O\}W_{cell}}{E_{cell}}$, where W_{cell} and E_{cell} are the mass per cell and energy requirements for cell synthesis, respectively, and $\{\dot{p}_O\}$ is a coefficient defining the supply of limiting resources through a network from a central point (with dimensions of energy × time^{-1} × mass$^{-3/4}$; the symbol *O* represents oxygen for reasons explained below). Defining mass-specific synthesis (overhead) cost of growth as $[E_g] = \frac{E_{cell}}{W_{cell}}$, the parameter b is a maintenance coefficient to be interpreted as $\frac{[\dot{p}_M]}{[E_g]}$, where $[\dot{p}_M]$ is the maintenance rate per unit mass. Thus, equation 14 can be rewritten as

$$\frac{dW}{dt} = \frac{\{\dot{p}_O\}}{[E_g]}W^{3/4} - \frac{[\dot{p}_M]}{[E_g]}W \tag{15}$$

in parallel to Pütter's model [equation (2)] and the DEB model [equation (10)]. The ratio of a to b simplifies to $\frac{\{\dot{p}_O\}}{[\dot{p}_M]}$ and the ultimate mass reached is $W_\infty = \left(\frac{\{\dot{p}_O\}}{[\dot{p}_M]}\right)^4$. Thus, as in DEB theory, maximum size reflects the ratio of a quantity that is available for growth and maintenance to a quantity that is required for maintenance. It was argued by West *et al.* (2001) that one can, in

principle, measure the mass, synthesis cost and maintenance cost of an individual cell of a species to obtain estimates of W_{cell}, E_{cell} and $[\dot{p}_M]$, respectively (but see Makarieva, Gorshkov & Li, 2004), and that $\{\dot{p}_O\}$ could be obtained by measurements of respiration.

Following the criticism that explicit consideration of the overhead cost of growth was omitted from the OGM (with E_{cell} erroneously equated to energy content of a cell) (Makarieva *et al.*, 2004; van der Meer, 2006), it was modified by Hou *et al.* (2008; see also Moses *et al.*, 2008) whereby the total flow of assimilated energy from food was split between storage (i.e. a reserve, E) and the total metabolic rate. The total metabolic rate was defined as the 'resting costs' $\{\dot{p}_O\}W^{3/4}$ multiplied by the activity scope parameter F (f was used in the original publication). The resting costs were then split into synthesis (overhead) costs and maintenance costs so that the input term equation (14) [and equation (15)] must be reinterpreted as representing only the energy dissipated in the work done to build the tissue, i.e. an anabolic process. Hou *et al.* (2008) also added the work of digestion ('specific dynamic action') to the maintenance term of their model. The dynamics of assimilation and reserve mobilisation were not specified; assimilation was simply computed on a demand basis from the activity costs and the energy content of biomass $[E_c]$ [i.e. a parameter representing West *et al.*'s (2001) original interpretation of $[E_g]$]. Hou, Bolt & Bergman (2011) subsequently added a new parameter f (β in the original publication) as a food restriction factor by which $\{\dot{p}_O\}$ was multiplied. Overall, these changes of interpretation of $\{\dot{p}_O\}$ imply that, in the OGM, the network dynamics are constraining the delivery of non-storable metabolites (namely oxygen) required for the overhead costs of growth and digestion, and for the maintenance costs of the soma, and not the flow of the somatic building blocks and associated chemical energy to the cells. It is not clear whether the network dynamics were also thought by the authors to limit activity costs $F\{\dot{p}_O\}W^{3/4}$, although this is the

implication (but see Darveau *et al.*, 2002). It is also not clear how their original loss term in equation (15), which represents maintenance costs, can be interpreted in the context of the scaling of resting metabolic rate with $W^{3/4}$, and at this point the whole scheme must be regarded as logically inconsistent.

Finally, it should be noted that two contributors to the OGM, Sibly & Brown (2020), recently criticised its assumption of maintenance costs scaling in direct proportion to mass and abandoned the network-based explanation for quarter-power scaling. They endeavoured to reformulate the OGM such that maintenance scales with mass$^{3/4}$, and supply (i.e. assimilation) is network constrained but with a capacity that scales with mass$^{2/3}$. They therefore arrived at a case of the generalised von Bertalanffy model, equation (5)**,** with c = 2/3 and d = 3/4 based on empirical rather than theoretical argument (see points made by West, Enquist & Brown, 2002). However, their conceptual reasoning about the underlying physiological processes regarding developed and growing cells results in a violation of mass conservation (Kearney & Kooijman, in press).

(6) What about reproduction, development and embryos?

Any comprehensive theory of growth and metabolism should of course incorporate reproduction and development as well as embryonic growth. None of these processes were considered by Pütter, von Bertalanffy or Pauly, but they have been fully included in DEB theory and partly included the OGM.

In DEB theory, reproduction is captured by adding an allocation parameter, κ which is a constant that splits the mobilised reserve flux into $\kappa\dot{p}_C$ which goes to growth and maintenance, and $(1-\kappa)\dot{p}_C$ which goes to reproduction (the ‘kappa rule’), so that equation (10) becomes

$$\frac{dV}{dt} = \frac{\kappa f\{\dot{p}_{Am}\}}{\kappa[E] + [E_G]} V^{2/3} - \frac{[\dot{p}_M]}{\kappa[E] + [E_G]} V \tag{16}$$

and ultimate length then becomes $L_\infty = \frac{\kappa f\{\dot{p}_{Am}\}}{[\dot{p}_M]}$.

An immature animal spends the $(1-\kappa)\dot{p}_C$ flux of energy from the reserve, not on reproduction, but on 'maturation', i.e. the metabolic work required for development and general preparation for reproduction. This introduces a new state variable, maturity E_H, which should formally be considered in the dimension 'information' but is quantified in practice as cumulative energy investment. The formal incorporation of information into biology and physics in general is an important work in progress (Davies, 2019) but it is more tractable to work with energy as a dimension because the energetic costs of development are readily measured (Tyler, 1935). The threshold level of maturation investment when an embryo transitions to a juvenile (i.e. when feeding starts) is the parameter E_H^b (at 'birth') and maturity investment plateaus at a value E_H^p (at 'puberty'), after which any surplus energy from the $(1-\kappa)\dot{p}_C$ goes to a reproduction buffer E_R and is periodically dispensed to the environment as eggs (and other reproductive material) with efficiency κ_R ($1-\kappa_R$ being dissipated). Maturity level is maintained at a cost directly proportional to E_H defined by the 'maturity maintenance rate coefficient' $\dot{k}_J$. Thus, in the standard DEB model, the processes of reproduction and development are included with the addition of two new state variables and five new parameters. Also, in the standard model, the onset of reproduction does not alter growth trajectory, assimilation rate, respiration rate or food intake, consistent with empirical data (Kooijman, 2010, pp. 47–49).

In the OGM, reproduction was handled differently for those that reproduce after reaching their maximum size (determinate growth) and those that reproduce while still growing (indeterminate growth) (West *et al.*, 2001; Charnov, Turner & Winemiller, 2001). For determinate growers, the

required energy is derived from increases in metabolic scope and hence in feeding post maturation. For indeterminate growers, reproduction costs were added to the maintenance parameter $[\dot{p}_M]$ with investment of energy of eggs E_R proportional to body size *via* an allocation term λ and a spawning period t_s. This alters ultimate size to be $W_\infty = \left(\frac{f\{\dot{p}_O\}}{[\dot{p}_M]+\lambda/t_s}\right)^4$ once the organism matures and thus causes a switch in the growth trajectory post maturation (West *et al.*, 2001). The indeterminate grower reproduction scheme arises from West *et al.*'s (2001) error of equating energy content of tissue with metabolic work to make tissue. In the reformulation (Hou *et al.*, 2008), λ can only apply to the overhead costs of reproduction and not to the biomass allocated to eggs (this problem propagates into Charnov *et al.*, 2001). Reproduction was not revisited in Hou *et al.* (2008) but, presumably, the egg energy would come from the stored energy. Thus, in both determinate and indeterminate growers, reproduction must involve an increase in assimilation, and hence of food intake, under the OGM. The dynamics of reproduction under this theory are therefore constrained solely by feeding, which must increase in mature animals in proportion to reproductive output.

Zuo *et al*. (2011) attempted to incorporate development into the OGM by arguing that the flux of energy originally seen as fuelling the overhead costs of growth could be allocated between growth and development. They argued that maximum size was reached when development was complete and that this could be at different body sizes depending on whether the temperature responses of growth and development differed. Developmental state was defined as $\mu' = t/t_{dev}$ where t_{dev} is total developmental time (at some reference temperature). Thus, development, and its end point, were only defined in the dimension of time and no parameters were introduced explicitly to account for developmental costs. It is unclear how the earlier definition of maximum size as $W_\infty = \left(\frac{\{\dot{p}_O\}}{[\dot{p}_M]}\right)^4$ (West *et al.*, 2001) can be consistent with a partitioning of growth costs

also to include development costs, or how development and reproduction are related (especially for indeterminate growers). The inclusion of development under the full OGM scheme has been argued to produce conceptual and empirical inconsistencies (Makarieva *et al.*, 2004; Makarieva, Gorshkov & Li, 2009; Zuo *et al.*, 2009). Hence, the integration of development in the OGM is problematic at present.

Embryonic growth does not follow equation (1), as acknowledged by Pütter (1920), but proceeds at a faster rate. In DEB theory, the dynamics of embryonic growth are captured by the reserve dynamics (Kooijman, 1986*b*) and the inclusion of maturity as a state variable (Kooijman, 1993). The organism is initialised as almost pure reserve (the egg) and thus has a very high reserve density. It has all the same dynamics as a juvenile [equations (8, 9, 11 & 15)] except that it does not feed (f and hence $\dot{p}_A$ is zero). By contrast, embryonic growth was incorporated into the OGM by simply deleting the maintenance term of equation (14) on the argument that it is negligibly small for embryos (Gillooly *et al.*, 2002). This renders the model phenomenological, and hence the incorporation of embryonic development in the OGM is also problematic.

III. COMPARISON AND SYNTHESIS

This review shows that Pütter's (1920) model was the first general and comprehensive attempt at a mechanistic theory of whole-organism growth. The ideas of von Bertalanffy and the GOLT were nuanced restatements of Pütter's ideas. However, two new mechanistic theories of metabolism have since emerged – the DEB theory based on the notion of reserve and its dynamics, and the OGM based on the notion of branching supply networks. The DEB theory builds directly from Pütter's concepts whereas the OGM derives from von Bertalanffy's generalisation of Pütter's model, with a theoretical justification for the exponents (Fig. 1). The

fundamental equations (Table 1), parameters and variables (Tables S1–S3) are collated here using the same notation and arrangement, permitting a general comparison and synthesis (Table 2). I do this first with respect to ontogenetic growth and then consider how other aspects of whole-organism metabolism are included as well as assessing the relative numbers of parameters and state variables. I then consider how 'macrometabolic' patterns in the scaling of metabolic rate, the temperature–size rule and life histories relate to these theories.

(1) Interpretation of individual growth

Growth is typically modelled as the difference between a gain term and a loss term, and the Pütter, DEB and OGM theories have very different interpretations of gain and loss with respect to the notions of anabolism, catabolism, assimilation and maintenance (Table 1). These differences of interpretation have not always been made clear.

Pütter's 'gain' term was interpreted as anabolism. But he incorporated maintenance into anabolism [equation (4), see also Table 1], as represented by the hole in his metaphorical bucket of nutritive materials. He interpreted maintenance as the 'basal metabolism' and thus his anabolic gain term represents materials available for the growth process after the basal metabolic costs are subtracted from assimilated food, assimilation being implicit in the model. These basal costs were regarded by Pütter to include growth overheads in addition to general maintenance (and, presumably, digestion overheads), hence Pütter's argument that the anabolic term should be proportional to the basal metabolic rate. Pütter's loss term was catabolism – a breakdown of the material built, as reflected in nitrogenous waste production – and was not connected to the basal metabolism.

In the OGM, the gain term was equated to basal metabolic rate, with the network argument (West, Brown & Enquist, 1997) used to justify the ¾ power scaling of this term. In the first version of the OGM (West *et al.*, 2001), the gain term represented the total energy going into the production of biomass, whereas the loss term was attributed to maintenance rather than catabolism. West *et al.* (2001) neglected the overhead costs of growth from their estimate of the cost of tissue production, as was later acknowledged (Hou *et al.*, 2008). But it is clear from their reasoning about reproduction that West *et al.* (2001) initially meant for the gain term to represent the total biomass production rate, in parallel to Pütter's model. Later, however, they revised their gain term to include only the growth overhead costs (Hou *et al.*, 2008). Thus, in the new formulation, the growth potential was defined entirely in terms of metabolic work, being the difference between total basal metabolic rate and maintenance costs. Energy content of tissue was accounted for secondarily by the demand-driven assimilation term as described above. This is most clearly illustrated in the magnitude of the energy flows of the Pütter model compared to the OGM in Fig. 2B, C. Throughout the development of the OGM there has therefore been confusion about the connections between basal metabolism, maintenance costs and growth overheads, culminating in the abandonment of the entire scheme by Sibly & Brown (2020) on the basis that maintenance costs do not scale proportionately with mass.

The DEB model differs from both Pütter and the OGM in specifying the gain term as assimilation and the loss term as maintenance (see also Winberg, 1960). However, by including the concept of reserve and its dynamics, the DEB model made explicit the missing step in Pütter's model between assimilation and the flow of nutritive materials to the growth process. In the DEB model, anabolic and catabolic processes are included in the gain term in that food is broken down (catabolic) and then converted into reserve (anabolic) which, in turn is broken

down (catabolic) in the process of reserve mobilisation and converted into structure (anabolic). The basal metabolism is thus interpreted as having contributions from the overhead cost of growth (and assimilation if digesting), as well as from the loss term due to the maintenance costs of the existing structure and maturity level.

Finally, in all three models, there is also the notion of a functional response that cuts the growth rate (and maximum possible size) down from its maximum, but with different interpretations (Table 1): in the Pütter model it is the diffusion rate of nutritive materials balanced by maintenance costs whereas in the DEB model and the OGM it is the food availability. However, the OGM as originally stated does not directly link to food as an environmental forcing variable but assumes that feeding meets demands [but see Hou *et al.* (2011) for an attempt to include food restriction in the OGM].

(2) Beyond growth

Individual metabolism is a network of strongly interacting processes that include growth and maintenance but also feeding, assimilation, storage, reproduction and development (and ageing, which is not considered here), and these processes change throughout ontogeny. Pütter and von Bertalanffy focused on growth but never explicitly considered feeding, assimilation, reproduction and development. The DEB theory for growth incorporated feeding, assimilation and reproduction from the beginning (Kooijman, 1986*a*), including how these processes transition between embryo and juvenile (Kooijman, 1986*b*), and later incorporated development explicitly (Kooijman, 1993). The inclusion of reserve as a state variable in DEB theory allowed embryo dynamics to be captured (Kooijman, 1986*b*, 2009), as well as the pupal phases of holometabolic insects (Llandres *et al.*, 2015). The OGM came from a strong focus on a

mechanism for the scaling of respiration (West *et al.*, 1997) and the translation of this mechanism to growth (West *et al.*, 2001), with other processes added on later (Hou *et al.*, 2008, 2011; Zuo *et al.*, 2011). However, inconsistencies in the way growth costs were handled during the development of the OGM, and the limited accounting for development and storage (reserve) dynamics under varying food levels, as discussed above, have resulted in an incomplete theory (Table 2).

Food restriction is another important process that a general metabolic theory should be able to capture. Pütter considered the effect of constant food restriction through its influence on the nutrient diffusion coefficient [$\dot{k}_N$], resulting in a smaller maximum size. In DEB theory, and the OGM (under the formulation of Hou *et al.*, 2011), a similar role is played by reductions in $\{\dot{p}_{Am}\}$ and $\{\dot{p}_O\}$, respectively, *via* the functional response factor f, resulting in smaller maximum sizes. Thus, in DEB theory the reduced maximum size is interpreted as direct consequence of the change in food itself and its effect on assimilation, whereas in the OGM it is due to a correlated change in the processes of respiration. Under extreme starvation, the standard DEB model results in degradation of structure to pay for maintenance but alternative formulations involve the redirection of reproduction buffer or energy for maturation to maintenance (i.e. effectively *via* changes in κ) (Kooijman, 2010, pp. 115–122).

Of the three theories, only the DEB theory handles fluctuating food availability, and this is because it includes reserve as a state variable. Feeding modules of varying sophistication can be incorporated, including digestive processes and stomachs (Kooijman, 2010, pp. 25–36, 114–124 and 261–279) but, at a minimum, the incorporation of variable food involves the addition of two parameters: $\{\dot{p}_{Xm}\}$ which is the maximum structural surface area-specific feeding rate and κ_X which is the conversion efficiency of assimilated food. The additional forcing variable X is the

food availability, which can be expressed per environmental surface area (two-dimensional environments) or volume (three-dimensional environments). In addition, the 'synthesising unit' construct of DEB theory allows multiple limiting substrates to be incorporated into food-limitation models (Kooijman, 1998; Kooijman, Andersen & Kooi, 2004).

(3) Parameter richness and accessibility

An important practical consideration of models from any theory is the number of parameters they require and how easily they are obtained, especially as more processes are included. As summarised in Tables S1–S3, Pütter's theory has one state variable, five core parameters and one auxiliary parameter (auxiliary parameters are not required for the theory but for linking to observations). For the processes covered in the present paper, the DEB theory has four state variables, ten core parameters and five auxiliary parameters whereas the OGM has four state variables, ten core parameters and three auxiliary parameters. The initial version of the OGM had fewer parameters than the original DEB model but, as the OGM has evolved to include the same number of processes, it has converged on the number of parameters and state variables in the DEB model, despite claims to the contrary (e.g. Brown *et al.*, 2004*b*; Zuo *et al.*, 2009; Marquet *et al.*, 2014). The differences in parameter and variable counts among the different models are thus a simple reflection of the number of processes considered, with approximately one parameter per process.

Regarding the availability of parameter estimates, the growth rate parameter of the Pütter model has been estimated for many species (Kooijman, 1988, p. 199; Froese & Pauly, 1994) but there are no accurate estimates of the core parameters. For the DEB theory, estimates of all core parameters are now available for over 2300 animal species (Marques *et al.*, 2018). For the OGM,

compilations of some core parameter estimates have been made for a number of taxa, with a focus on $\{\dot{p}_O\}$, $[\dot{p}_M]$, $[E_g]$, $[E_c]$, f and F (West *et al.*, 2001; Hou *et al.*, 2008, 2011) but there are few estimates of reproduction parameters.

(4) Mass balance and stoichiometry

A formal metabolic theory should allow both mass and energy balances to be computed based on the interacting sub-processes. All the metabolic theories discussed here are closely tied to empirical patterns of respiration, which connects to metabolism *via* indirect calorimetry – i.e. the flows of oxygen, carbon dioxide, water and nitrogenous waste in relation to heat production. Moreover, the sum of all the mass flows of different molecules in the metabolism could in principle be related to the flows of individual elements, and so connect directly with the field of ecological stoichiometry (Sterner & Elser, 2002).

Pütter's model and the OGM are not stoichiometrically explicit, and mass conservation is considered only in terms of total biomass. Discussions of stoichiometry in relation to OGM-based metabolic theory have emphasised empirical body-size scaling patterns in elemental turnover (Peters, 1983) and verbal arguments have been made about why this might be tied to the metabolic rate (Brown *et al.*, 2004*a*; Allen & Gillooly, 2009). But such ideas have never been formalised other than through phenomenological equations invoking theoretically justified scaling exponents. However, the strategy in DEB theory of dealing with pools (reserve, structure) of constant chemical composition allows the mass to be balanced on the basis of chemical elements (Kooijman *et al.*, 2004). It can thus make a direct mechanistic link to indirect calorimetry and to stoichiometry.

The capacity of DEB theory explicitly to connect individual growth and metabolism to stoichiometry has been overlooked by many authors, including those calling for such a synthesis. For example, Allen & Gillooly (2009) reviewed the literature on the metabolic theory of ecology and ecological stoichiometry and remarked (p. 380) that the models and data they considered showed how "individual energetics and biomass stoichiometry are inextricably linked with each other". They lamented that there was not a stronger connection between the two fields and discussed how this could be achieved. It is thus worth briefly explaining how a way of formally connecting energy, mass and elemental flows had already been proposed within DEB theory (Kooijman, 2000; Kooijman *et al.*, 2004).

First, the composition of the 'organic' $\mathcal{O}$ aspect of the biomass, i.e. food X, structure V, reserve E, reproduction buffer E_R and 'product' P (i.e. faeces for an animal), can be specified as a matrix in terms of C-moles n (i.e. moles of molecules per moles of carbon) for a set of common elements, minimally carbon C, hydrogen H, oxygen O and nitrogen N:

$$\boldsymbol{n}_{\mathcal{O}} = \begin{pmatrix} n_{CX} & n_{CV} & n_{CE} & n_{CP} \\ n_{HX} & n_{HV} & n_{HE} & n_{HP} \\ n_{OX} & n_{OV} & n_{OE} & n_{OP} \\ n_{NX} & n_{NV} & n_{NE} & n_{NP} \end{pmatrix}. \tag{17}$$

The same can be done for the 'mineral' $\mathcal{M}$ component of the biomass flows, i.e. CO_2, H_2O, O_2 and the organism-specific nitrogenous waste:

$$\boldsymbol{n}_{\mathcal{M}} = \begin{pmatrix} n_{CC} & n_{CH} & n_{CO} & n_{CN} \\ n_{HC} & n_{HH} & n_{HO} & n_{HN} \\ n_{OC} & n_{OH} & n_{OO} & n_{ON} \\ n_{NC} & n_{NH} & n_{NO} & n_{NN} \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 & n_{CN} \\ 0 & 2 & 0 & n_{HN} \\ 2 & 1 & 2 & n_{ON} \\ 0 & 0 & 0 & n_{NN} \end{pmatrix}. \tag{18}$$

The complete mass balance can be summarised as follows, where $\dot{J}$ represents a mass flux (mol/time):

$$\begin{pmatrix} 1 & 0 & 0 & n_{CN} \\ 0 & 2 & 0 & n_{HN} \\ 2 & 1 & 2 & n_{ON} \\ 0 & 0 & 0 & n_{NN} \end{pmatrix} \begin{pmatrix} \dot{j}_C \\ \dot{j}_H \\ \dot{j}_O \\ \dot{j}_N \end{pmatrix} + \begin{pmatrix} n_{CX} & n_{CV} & n_{CE} & n_{CP} \\ n_{HX} & n_{HV} & n_{HE} & n_{HP} \\ n_{OX} & n_{OV} & n_{OE} & n_{OP} \\ n_{NX} & n_{NV} & n_{NE} & n_{NP} \end{pmatrix} \begin{pmatrix} \dot{j}_X \\ \dot{j}_V \\ \dot{j}_E + \dot{j}_{E_R} \\ \dot{j}_P \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \qquad (19)$$

where the fluxes of elements associated with both the mineral $\dot{\boldsymbol{J}}_{\mathcal{M}}$ and organic $\dot{\boldsymbol{J}}_{\mathcal{O}}$ fluxes must sum to zero according to the conservation law, i.e. $\boldsymbol{n}_{\mathcal{M}}\dot{\boldsymbol{J}}_{\mathcal{M}} + \boldsymbol{n}_{\mathcal{O}}\dot{\boldsymbol{J}}_{\mathcal{O}} = \boldsymbol{0}$.

The only remaining step is to connect the organic mass flows to the energy flows from assimilation $\dot{p}_A$, growth $\dot{p}_G$ and the other dissipative processes associated with maintenance, maturation and reproduction $\dot{p}_D$ *via* the multiplication

$$\begin{pmatrix} \dot{j}_X \\ \dot{j}_V \\ \dot{j}_E + \dot{j}_{E_R} \\ \dot{j}_P \end{pmatrix} = \begin{pmatrix} -\eta_{\mathrm{XA}} & 0 & 0 \\ 0 & 0 & \eta_{\mathrm{VG}} \\ \bar{\mu}_{\mathrm{E}}^{-1} & -\bar{\mu}_{\mathrm{E}}^{-1} & -\bar{\mu}_{\mathrm{E}}^{-1} \\ \eta_{\mathrm{PA}} & \eta_{\mathrm{PD}} & \eta_{\mathrm{PG}} \end{pmatrix} \begin{pmatrix} \dot{p}_A \\ \dot{p}_D \\ \dot{p}_G \end{pmatrix}, \qquad (20)$$

where the η terms are mass/energy conversion parameters and $\bar{\mu}_E$ is the chemical potential of reserve discussed earlier. Thus, if the mineral and organic stoichiometric composition is known and the organic fluxes are computed as above *via* the DEB model energetic fluxes associated with assimilation, growth and dissipation, the mineral fluxes can be computed as

$$\dot{\boldsymbol{J}}_{\mathcal{M}} = \begin{pmatrix} \dot{j}_C \\ \dot{j}_H \\ \dot{j}_O \\ \dot{j}_N \end{pmatrix} = \boldsymbol{n}_{\mathcal{M}}^{-1}\boldsymbol{n}_{\mathcal{O}}\dot{\boldsymbol{J}}_{\mathcal{O}}. \qquad (21)$$

The total respiration rate in terms of CO_2 (i.e. $\dot{j}_C$) or O_2 (i.e. $\dot{j}_O$) then emerges as a weighted sum of the assimilation, growth and dissipation processes, which will vary through time depending on ontogenetic and feeding state. So too does metabolic water $\dot{j}_H$ and nitrogenous waste $\dot{j}_N$ production. Thus, in the DEB theory, the respiration rate is a secondary outcome, rather than a

primary driver. In the OGM, by contrast, the empirically observed metabolic rate is a primary and given driver, linked directly to the anabolic flux.

Although organisms show a striking degree of 'stoichiometric invariance' (Allen & Gillooly, 2009), they may vary in their composition as a function of diet. In DEB theory, the reserve concept allows explicit consideration of fluctuating food (the 'dynamic' part of DEB theory) and its effect on body condition if the elemental compositions of reserve and structure differ and their relative amounts change. The introduction of multiple reserves in DEB theory together with the innovation of the 'synthesizing unit' dynamics (Kooijman, 1998) can also permit the consideration of multiple limiting nutrients (Schouten, Vesk & Kearney, in press) and ultimately link to food chains and ecosystem dynamics (Nisbet *et al.*, 2000; Kuijper *et al.*, 2004; Kearney *et al.*, 2010; Sperfeld *et al.*, 2017). These features of current metabolic theory are not widely appreciated in mainstream research on ecological stoichiometry but have great potential to unify ecological studies of nutrition, stoichiometry and energetics (Sperfeld *et al.*, 2017). Empirical tests of this aspect of DEB theory will be potent tests of its performance. These could include assessments of the expected allometric patterns the theory predicts for e.g. metabolic water and nitrogenous waste production under different circumstances (e.g. food limitation), as well as consistency checks against detailed stochiometric accounting during different phases of ontogenetic growth.

(5) The scaling of metabolic rate

An understanding of the scaling of metabolic rate with size, especially the hypoallometric 3/4 power scaling pattern, has been a holy grail in comparative physiology for many years (Kleiber, 1932). Pütter (1920) had already emphasised that his theory implied a 2/3 scaling relationship

between basal metabolism and body size and that it would thus not apply to all species. von Bertalanffy went further and grouped animal growth responses into three distinct types according to whether respiration was: (*i*) surface proportional (e.g. fish, mammals): (*ii*) mass proportional (e.g. insects); and (*iii*) somewhere in between (e.g. snails) (von Bertalanffy, 1957). This is what led him to an allometrically generalised version of Pütter's model (equation 5).

The first major paper on DEB theory aimed to explain the scaling pattern of metabolic rate by distinguishing between ontogenetic and interspecific patterns of metabolic rate (Kooijman, 1986*a*). The ontogenetic pattern was explained by the decreasing contribution of growth overhead costs with size, a mechanism subsequently used to explain half-power scaling of respiration in two-dimensional organisms (White *et al.*, 2011). The interspecific scaling was attributed to a null expectation from the theory that species of larger size have proportionally more reserve. This emerges from the fact that the assimilation parameter is the only extensive parameter of the three that determine maximum length, i.e. $L_m = \frac{\kappa\{\dot{p}_{Am}\}}{[\dot{p}_M]}$; because maximum reserve density $[E_m] = \frac{\{\dot{p}_{Am}\}}{\dot{v}}$ and $\dot{v}$ is also an intensive parameter, it follows that $[E_m]$ increases with size if selection acts solely on size. The increased reserve density with length dilutes the metabolic rate per total biomass because reserve does not require maintenance. The departures from 'von Bertalanffy' growth were subsequently explained due to different forms of 'metabolic acceleration' (Kooijman, 2014). Moreover, although the DEB and OGM predict curvature in the scaling of metabolic rate, it is in the wrong direction to what has been reported empirically in mammals (Kolokotrones *et al.*, 2010).

As already mentioned, the OGM argued that the scaling of the supply network (circulatory system) and its effect on the supply of non-storable metabolites (namely oxygen) was what caused respiration to scale hypoallometrically. Thus, both the DEB theory and the OGM (West *et*

al., 1997, 2001) predicted 3/4 power scaling of metabolic rate but through different mechanisms. There is still great potential for empirical investigations into the different theoretical expectations of the two theories (Kearney & White, 2012). The theories may well be complementary in some respects (Maino *et al.*, 2014) and, although network limitations may not always exist or occur in the manner expected (Aitkenhead *et al.*, 2020), they may become limiting in certain situations such as low oxygen (Verberk & Bilton, 2011) and may be empirically tied to organismal shape changes (Hirst, Glazier & Atkinson, 2014).

(6) Temperature–size rule

Another macrometabolic pattern is for body size to decline as a plastic response to rearing temperature under benign conditions – the 'Temperature–Size Rule' (TSR) (Atkinson, 1994). Pütter (1920) noted that many species, including foraminifera, paramecium and fish, grow larger in the cold. This pattern typically is explained by proposing differential temperature sensitivity to the parameters in the growth equation. Pütter (1920) proposed that parameters interpreted as reaction coefficients should have an exponential response to temperature whereas diffusion constants should have a linear response. He regarded the nutrient supply parameter [$\dot{k}_N$] [see equation (4)] as a diffusion constant and the maintenance parameter [$\dot{k}_M$] [see equation (4)] as well as the catabolic rate parameter [$\dot{k}_C$] [see equation (2)] as reaction coefficients. Thus, the anabolic response should have a lower temperature sensitivity than the catabolic response. von Bertalanffy proposed Pütter's hypothesis as his own (von Bertalanffy, 1960, p. 216). As a consequence, Pütter's major role in the history of the temperature–size rule has gone unnoticed (but see Taylor, 1962).

Pütter's notion that anabolism and catabolism have different temperature sensitives has not survived empirical analysis (e.g. Angilletta, Steury & Sears, 2004). An alternative avenue of investigation, first proposed by van der Have and de Jong (1996) and then restated within the OGM (Zuo *et al.*, 2011; Pettersen *et al.*, 2019; but see also Forster, Hirst & Woodward, 2011), is to assume different temperature sensitivities for the rates of development and growth. This assumption was most recently shown to provide a powerful empirical explanation for the TSR rule in respect to offspring size (Pettersen *et al.*, 2019). However, due to the incomplete incorporation of development into the OGM, as discussed above, these empirical findings do not help with the theoretical assessment of the OGM.

The TSR has not been considered formally within the DEB theory and in this sense presents an important challenge. The thermal sensitivities of rates of different metabolic processes are not assumed to vary under benign body temperature ranges because of the regulatory complexities (and extra parameters) resulting from temperature dependence of yield coefficients in metabolic pathways (Kooijman, 2010). However, because the maturity state variable is conceptually information, rather than mass or energy, it could be expected to depart from other processes in its thermal sensitivity. Further experimental investigation of the TSR in a DEB context is a promising way to test fundamental aspects of the theory, especially in regard to the maturity concept.

(7) Life history and evolution

Life histories are the outcome of natural selection acting to optimise reproductive success within the constraints imposed by energy, mass and time budgets (Stearns, 1992; Roff, 2002). These constraints involve trade-offs necessitated by the laws of thermodynamics and the specific

architecture of metabolism. Most life-history analyses have been done on the basis of optimisation arguments without an underlying metabolic framework (Stearns, 1992; e.g. Kozłowski, 1992; Charnov, 1993; Roff, 2002). However, the physical and chemical constraints on growth, maintenance, development and reproduction assumed in a metabolic theory should capture those life-history patterns that are driven primarily by constraints. In this way, the theory should provide null expectations for life-history patterns and covariations in much the same way that the physics of load bearing can predict constraints on limb shape in animals.

Neither Pütter nor von Bertalanffy discussed life-history patterns in relation to metabolic theory but they have been considered in the DEB and OGM theories. DEB theory makes predictions of life-history constraints in part through the way that growth, maintenance, maturation and reproduction relate to ultimate size *via* the kappa rule for allocation. For example, all else being equal, a decrease in the value of κ itself would simultaneously decrease size, shorten development and maturation times and increase reproduction allocation, but the smaller overall size would result in a lower absolute reproductive output if size at birth remained constant. The other source of life-history constraints relates to the notion that extensive parameters (size-dependent) covary as a null expectation while intensive parameters do not (Kooijman, 2010, p. 300). For example, the extensive parameters $\{\dot{p}_{Am}\}$, E_H^b and E_H^p would be expected to covary so that size at birth, size at maturation and ultimate size also co-vary; selection on size at one of these three life stages would cause correlated responses in the other two unless there is differential selection acting across life stages. A general analysis of the evolution of life histories under DEB theory is provided by Lika & Kooijman (2003). Life-history evolution under the OGM has been studied by Charnov *et al*. (2001) in the context of optimality analyses and more recently by Burger, Hou & Brown (2019), but there has been no assessment of the OGM and

life-history variation in the context of extensive and intensive parameters, and Burger *et al*. (2019) did not put their work into context with previous DEB work on life history. Life-history phenomena may be a potent testing ground for ideas in metabolic theory because they represent the outcome of the full network of interactions of metabolic processes. For example, Marshall & White (2019) pointed out that reproduction scales hyperallometrically in some species, and challenged existing growth theories on this basis. They emphasised the optimality argument of Kozłowski & Wiegert (1986), Kozłowski (1992) and Kozłowski & Teriokhin (1999) whereby declining rate of growth reflects a smooth, increasing investment in reproduction. Kozłowski, Konarzewski & Czarnoleski (2020) recently argued that selection on life history drives the patterns of scaling in metabolic rate. There is scope to explore such patterns and hypotheses more fully within the existing metabolic theories of growth (Kearney, 2019). In particular, the DEB theory provides a unique perspective through its clear distinction between null expectations from a 'physics only' perspective with respect to scaling (or 'covariation') rules, and because it adds more explicit internal constraints for optimality analyses *via* the kappa rule and the resultant connections between growth, development, reproduction and ultimate size.

IV. CONCLUSIONS

(1) Although whole-organism metabolic theory has a rich and productive history, it has progressed in a disorganised and disconnected manner. Some of the most fundamental concepts stem from Pütter's original contribution 100 years ago (Fig. 1). However, the subsequent development of the field has been hampered by failures properly to acknowledge or account for existing ideas when proposing new ones.

(2) This review aimed to consolidate our current understanding of metabolic theory so that the research program can develop in a more coordinated and coherent fashion. A summary of the major outcomes is provided in Table 2. The analysis has singled out two major mechanistic theories for individual growth that have emerged since Pütter: DEB theory and the OGM. A comparison of these models revealed that their similar mathematical structure for asymptotic growth is underlain by major conceptual differences in the interpretation of anabolism, catabolism, maintenance and assimilation.

(3) Serious problems with the OGM arise from its conceptual interpretation of the supply term in the growth equation as anabolism, and the loss term as maintenance. This interpretation arises from the theory's emphasis on networks limiting the supply of non-storable metabolites, and the notion that the empirically observed metabolic rate is a primary driver of growth as opposed to a secondary outcome. Thus, the OGM explicitly considers only the energy flows associated with metabolic work, i.e. overheads and maintenance, and not tissue content. This has prevented a clear connection to assimilation, reproduction and stoichiometry, and led to logical inconsistencies in the treatment of maintenance. Moreover, the OGM has not adequately incorporated development in embryos or juveniles.

(4) By contrast, the DEB theory was shown to be a natural expansion upon Pütter's original concepts. This is especially by making the 'nutritive materials' explicit with their own state variable, the 'reserve', which mediates between the assimilated food and the energy and matter used for growth. However, a major conceptual difference is the shift from an anabolism/catabolism framework to an assimilation/maintenance framework, where there are both anabolic and catabolic aspects to the processes of assimilation and growth. Because DEB theory is built on the assumption of stoichiometrically invariant pools of biomass, it interprets

the empirically observed metabolic rate as a secondary outcome and permits stoichiometrically explicit mass balancing. Reproduction and development are incorporated by a partitioning of flow out of the reserve, with a fixed fraction going to growth and maintenance and the remainder allocated to maturation plus reproduction, preserving 'von Bertalanffy' growth trajectories in isomorphic organisms. Embryonic development is then captured simply through an absence of assimilation and an initial state of almost pure reserve.

(5) A theory should be logically consistent, adequate to explain the desired range of phenomena, and coherent with existing related theory (including basic physics and chemistry). In this context, the inescapable conclusion from this review is that we currently have only one viable mechanistic theory of metabolism at present – the DEB theory. The founder of that theory has provocatively suggested there can be no simpler alternatives (Kooijman, 2020). Future research on this topic must therefore either prove this assertion wrong, or further explore how the DEB scheme may explain metabolic phenomena. If the former proves possible then research should focus on ways to test the alternative assumptions. Most tests of metabolic theory have used existing data that were not collected with the specific goal of testing the theoretical assumptions in convincing ways (Kearney & White, 2012). Yet, there is a very rich array of empirical phenomena to explore in the context of formal metabolic theory, especially in the area of stoichiometric and life-history phenomena. In this way we can progress towards the vision of a metabolic theory of ecology (Nisbet *et al.*, 2000; Brown *et al.*, 2004*a*).

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Author contributions: M.R.K. conceived the ideas, conducted the literature review, designed and created the figures and tables and wrote the article.

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VII. SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. The variables and parameters of Pütter's growth model.

Table S2. The variables and parameters of the standard model of the Dynamic Energy Budget theory.

Table S3. The variables and parameters of the Ontogenetic Growth Model.

Appendix S1. R code used to calculate the graphs plotted in Fig. 2.

Table 1. A general comparison of the core equations of the three major theoretical models of growth: the 'von Bertalanffy' model as first developed by Pütter, the Dynamic Energy Budget (DEB) theory and the Ontogenetic Growth Model (OGM). Reproduction for indeterminate growers is included in the equation for ultimate physical wet mass for the OGM. Note that lower and upper case 'a' refer to anabolism and assimilation, respectively, lower and upper case 'g' refer to growth overhead costs and total growth costs, respectively, and lower and upper case 'm' refer to maximum and maintenance, respectively. See Tables S1–S3 for definitions of variables and parameters used in this table. In this review I follow the notation of Kooijman (2010), indicating rates by using a dot above the symbol, volume-specific quantities with square brackets and surface area-specific (and network-specific) quantities with curly brackets.

	Pütter (aka von Bertalanffy)	**DEB**	**OGM**
Key publication(s)	Pütter (1920)	Kooijman (1986*a*, 2010)	West *et al.* (2001); Hou *et al.* (2008, 2011)
Key innovations	surface to volume relation	reserve dynamics, maturation	supply network constraints
Growth interpretation	anabolism–catabolism	assimilation–maintenance	anabolism–maintenance
Growth equation (no reproduction)	$\frac{dV}{dt} = f\{\dot{k}_{am}\}V^{2/3} - [\dot{k}_C]V$	$\frac{dV}{dt} = \frac{f\{\dot{p}_{Am}\}V^{2/3} - [\dot{p}_M]V}{[E] + [E_G]}$	$\frac{dW}{dt} = \frac{f\{\dot{p}_O\}W^{\frac{3}{4}} - [\dot{p}_M]W}{[E_g]}$
Scaled functional response	$f = \frac{[\dot{k}_N]}{[\dot{k}_N] + [\dot{k}_M]}$	$f = \frac{X}{X + K}$	$f = \frac{X}{X + K}$
Growth incorporating reproduction/maturation in volume/mass	–	$\frac{dV}{dt} = \frac{\kappa f\{\dot{p}_{Am}\}V^{2/3} - [\dot{p}_M]V}{\kappa[E] + [E_G]}$	$\frac{dW}{dt} = \frac{f\{\dot{p}_O\}W^{3/4} - ([\dot{p}_M] + \lambda/t_s)W}{[E_g]}$
Growth parameter (in length)	$\dot{r}_B = \frac{[\dot{k}_C]}{3}$	$\dot{r}_B = \frac{[\dot{p}_M]}{3(\kappa[E] + [E_G])}$	$\dot{r}_B = \frac{[\dot{p}_M] + \lambda/t_s}{4[E_g]}$

Ultimate volumetric length	$L_\infty = \frac{f\{\dot{k}_{am}\}}{[\dot{k}_C]}$	$L_\infty = \frac{\kappa f\{\dot{p}_{Am}\}}{[\dot{p}_M]}$	$L_\infty = \left(\frac{f\{\dot{p}_O\}}{[\dot{p}_M] + \lambda/t_s}\right)^{4/3}$
Ultimate physical wet mass	$W_{w,\infty} = \delta_W {L_\infty}^3$	$W_{w,\infty} = d_V {L_\infty}^3 + E_m \frac{w_E}{\bar{\mu}_E}$	$W_{w,\infty} = \left(\frac{f\{\dot{p}_O\}}{[\dot{p}_M] + \lambda/t_s}\right)^4$
Physical length	$L_w = \frac{L}{{\delta_W}^{1/3}}$	$L_w = \frac{L}{\delta_L}$	$L_w = \frac{L}{{\delta_W}^{1/3}}$
Growth curve in volumetric length	$L = L_\infty - (L_\infty - L_0)e^{-\dot{r}_B t}$	$L = L_\infty - (L_\infty - L_b)e^{-\dot{r}_B t}$	$L = \sqrt[3]{W_\infty \left(1 - (1 - \left(\frac{W_b}{W_\infty}\right)^{1/4})e^{-\dot{r}_B t}\right)^4}$

Table 2. Summary of the nature of mechanistic individual growth models including their conceptual basis, applicability, consistency and major strengths and limitations. GOLT, Gill-Oxygen Limitation Theory; DEB, Dynamic Energy Budget Theory; OGM, Ontogenetic Growth Model. For the DEB theory, surface area and volume refer to that of the 'structure' (see Section II.4).

	Pütter	**von Bertalanffy**	**GOLT**	**DEB**	**OGM**
Core processes	anabolism, catabolism	anabolism, catabolism	anabolism, catabolism	assimilation, maintenance, maturation, growth, reproduction, ageing	anabolism, maintenance
Core internal constraints	surface area-dependent assimilation and anabolism, volume-dependent breakdown	surface area-dependent assimilation and anabolism, volume-dependent breakdown	(gill) surface area-dependent uptake, mass-dependent breakdown	surface area-dependent assimilation, surface area to volume ratio-dependent reserve mobilisation, volume-dependent maintenance	distribution network-dependent anabolic rate, mass-dependent maintenance
Potential environmental forcing	constant food, temperature	constant food, temperature	dissolved oxygen, temperature	dynamic food (and other resources), temperature, food composition, water	constant food, temperaure
Role of reserves	implicit, mechanistic role, static	absent	absent	explicit state variable, dynamic	static output
Role of respiration	implicit outcome, given	implicit outcome, given	explicit constraint, given	explicit outcome (all core processes contribute), mechanistically derived	explicit constraint, given
Stoichiometry	absent	absent	absent	explicit	absent
Food limitation	static	absent	absent	dynamic	static
Reproduction	absent	absent	absent	explicit	unresolved logical issues
Development and embryonic growth	absent	absent	absent	explicit	unresolved logical issues

Major advance or strength	conceptual pioneer	wide uptake, conceptual expansion	specific limiting mechanism	integrated across whole life cycle, in dynamic environments, stoichiometrically explicit	wide uptake, specific limiting mechanism
Major limitations	only applicable to growth under constant food	only applicable to growth under constant food	only relevant to water-breathers	level of abstraction of state variables, requires more targeted testing of realism	only applicable to growth in mass under constant food

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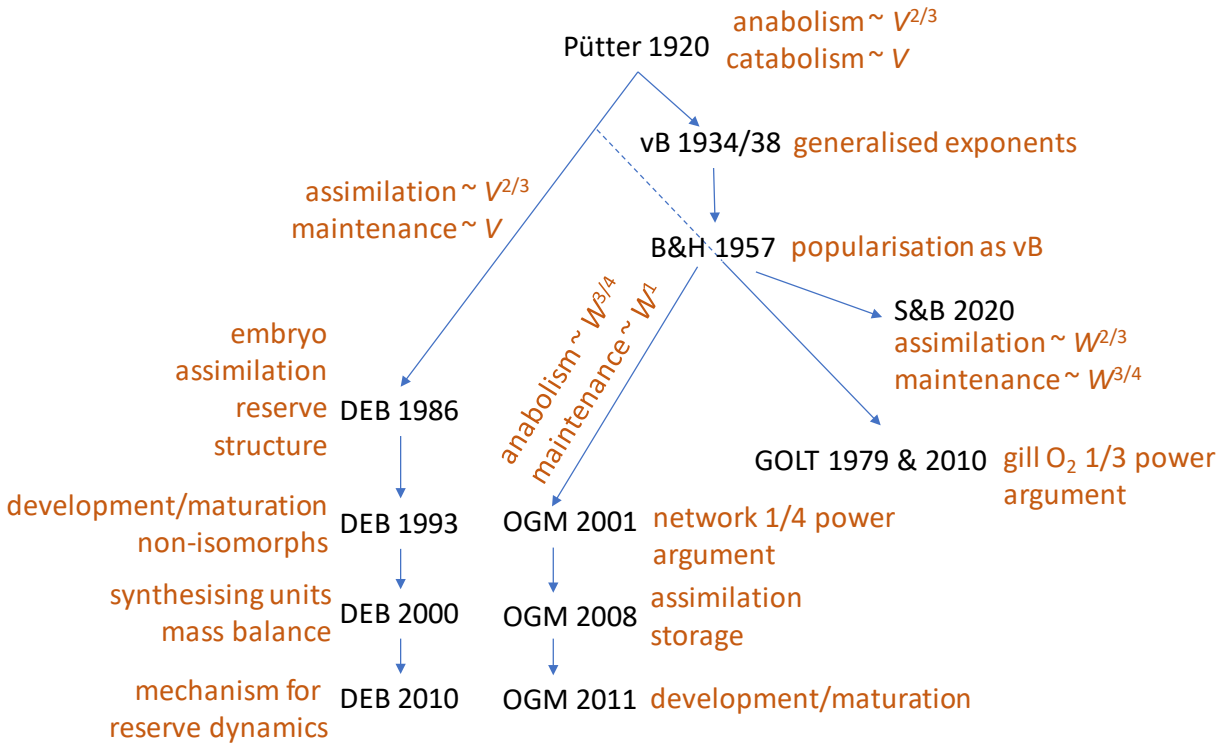

Fig. 1. Major developments in the theory of organismal growth and metabolism, starting with the ideas of Pütter and leading to the development of Dynamic Energy Budget theory (DEB) on one branch, and on the other *via* von Bertalanffy (vB) and Beverton and Holt (B&H) to the Gill Oxygen and Limitation Tolerance hypothesis (GOLT), the Ontogenetic Growth Model (OGM) and Sibly & Brown (S&B). Important conceptual innovations are indicated in orange. The GOLT model was based on von Bertalanffy's work but derives in fact from Pütter. V is volume and W is mass.

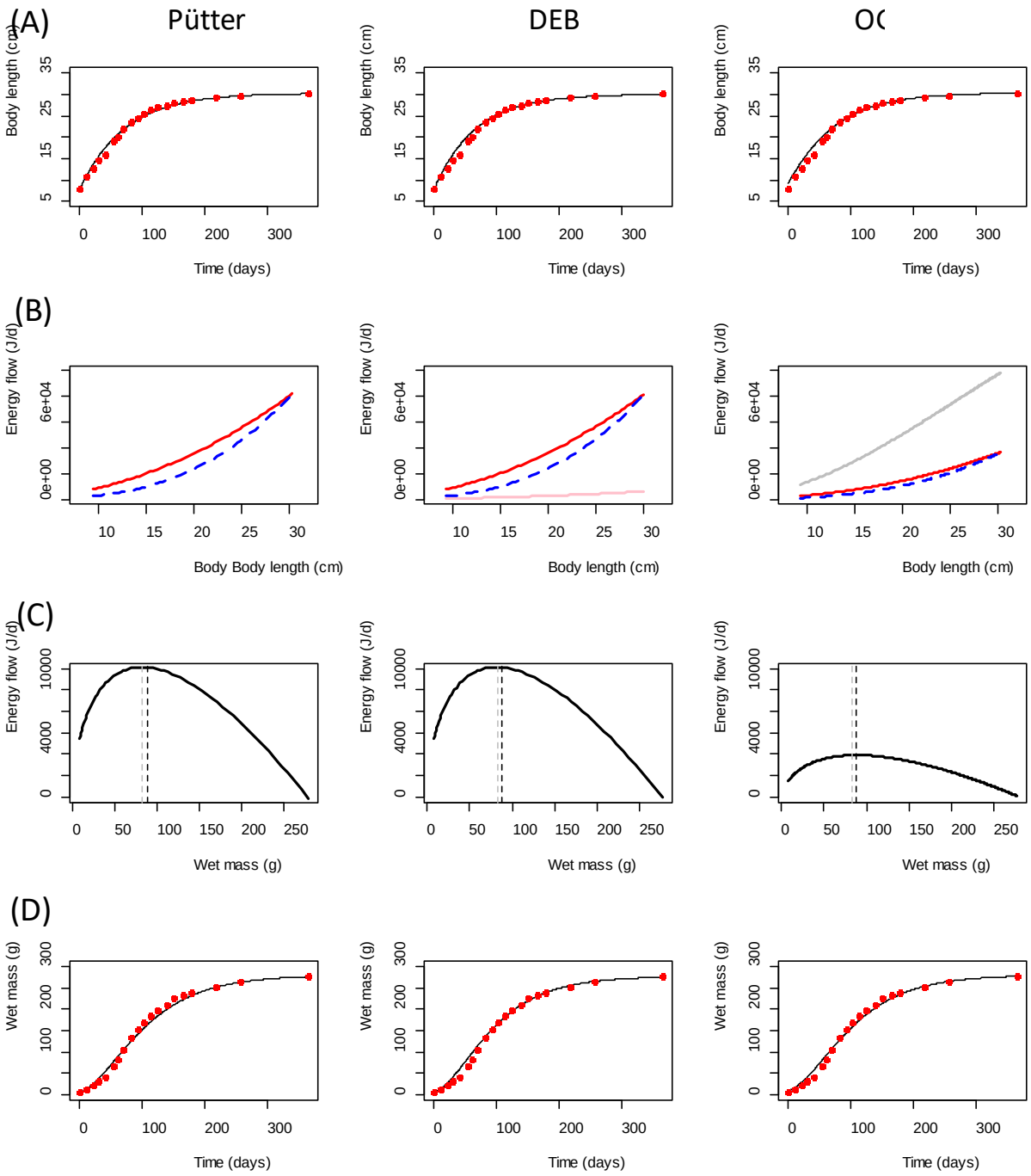

Fig. 2. Growth outcomes of the Pütter, Dynamic Energy Budget (DEB) and Ontogenetic Growth Model (OGM) for the rat, *Rattus norvegicus*: (A) Observed (red dots) and predicted (black line) body length *versus* time. (B) Energy input (red) and energy sink (blue) *versus* body length for

each model, representing anabolism/catabolism in the Pütter model, assimilation/maintenance in the DEB model and anabolism/maintenance in the OGM. Energy to reproduction is also indicated for the DEB model (pink line) and assimilated energy (grey line) for the OGM. (C) Energy flow to growth *versus* body mass, with grey vertical broken lines showing where Pütter/DEB models reach a maximum and black vertical broken lines where the OGM reaches a maximum. (D) Observed (red dots) and predicted (black line) wet mass *versus* time. Although each approach captures the growth pattern well under constant food intake, the underlying theoretical bases are very different and vary substantially in their ability to capture the dynamics of growth and reproduction across different stages of the life cycle and in variable environments. The R code used to compute these curves is provided in Appendix S1.

Table S1. The variables and parameters of Pütter's growth model.

	Symbol	Dimension	Interpretation
State variables	$V = L^3$	volume	total volume = cube of volumetric length $L \approx$ total mass (assuming 1 cm^3 = 1 g)
Core parameters	$\{\dot{k}_{am}\}$	mass $time^{-1}$ $volume^{-2/3}$	maximum specific nutrient concentration for anabolism (build-up) rate
	$[\dot{k}_C]$	mass $time^{-1}$ $volume^{-1}$	specific catabolic rate
	$[\dot{k}_N]$	mass $time^{-1}$ $volume^{-1}$	specific nutrient diffusion coefficient
	$[\dot{k}_M]$	mass $time^{-1}$ $volume^{-1}$	specific basal metabolic rate
	L_0	length	initial volumetric length
Compound parameters	$f = \frac{[\dot{k}_N]}{[\dot{k}_N] + [\dot{k}_M]}$	–	nutrient concentration functional response
	$\{\dot{k}_a\} = f\{\dot{k}_{am}\}$	mass $time^{-1}$ $length^{-2}$	specific nutrient concentration-adjusted anabolic (build-up) rate
	$L_\infty = \frac{f\{\dot{k}_{am}\}}{[\dot{k}_C]}$	length	ultimate volumetric length
	$W_{w,\infty} = \delta_W {L_\infty}^3$	mass	ultimate wet mass
	$\dot{r}_B = \frac{[\dot{k}_C]}{3}$	mass $time^{-1}$ $length^{-3}$	relative (von Bertalanffy) growth rate coefficient
	$L_w = \frac{L}{{\delta_W}^{1/3}}$	length	physical (measured) length
Auxiliary parameters	δ_W	$volume^{1/3}$ $mass^{-1}$	volumetric length to wet mass conversion coefficient

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Table S2. The variables and parameters of the standard model of the Dynamic Energy Budget theory.

	Symbol	Dimension	Interpretation
State variables	V	volume	structural volume
	$[E]$	energy volume^{-1}	reserve density
	E_H	energy	maturity
	E_R	energy	reproduction buffer
Forcing variables	X	energy volume^{-1} (or surface^{-1})	food energy density per unit environmental volume or surface area
Core parameters	$\{\dot{p}_{Am}\}$	energy time^{-1} volume$^{-2/3}$	maximum specific assimilation rate
	$[\dot{p}_M]$	energy time^{-1} length^{-3}	specific maintenance rate
	$\dot{v}$	volume$^{1/3}$ time^{-1}	energy conductance
	κ	–	energy allocation parameter (growth/maintenance *versus* maturation/reproduction)
	κ_R	–	reproduction efficiency (determines overhead costs)
	E_H^b	energy	threshold maturity at birth
	E_H^p	energy	threshold maturity at puberty
	$[E_G]$	energy volume^{-1}	specific cost (biomass content and overhead) of growth
	$\dot{k}_J$	time^{-1}	maturity maintenance rate coefficient
	K	energy volume^{-1} (or surface^{-1})	food density half-saturation constant

Compound parameters	$f = \frac{X}{X+K}$	–	food functional response
	$\{\dot{p}_A\} = f\{\dot{p}_{Am}\}$	energy time^{-1} volume$^{-2/3}$	specific food-adjusted assimilation rate
	$[E_m] = \frac{\{\dot{p}_{Am}\}}{\dot{v}}$	energy volume^{-1}	maximum reserve density
	$L_\infty = \frac{f\kappa\{\dot{p}_{am}\}}{[\dot{p}_M]}$	volume$^{-1/3}$	ultimate structural length
	$W_{w,\infty} = d_V L_\infty{}^3 + E_m \frac{w_E}{\bar{\mu}_E}$	mass	ultimate wet mass
	$L_w = \frac{L}{\delta_L}$	length	physical (measured) length
	$\dot{r} = \frac{f[\dot{p}_{Am}] - [\dot{p}_M]}{[E] + [E_G]}$	time^{-1}	growth rate of structure
	$\dot{r}_B = \frac{[\dot{p}_M]}{3(\kappa[E] + [E_G])}$	energy time^{-1} volume^{-1}	relative (von Bertalanffy) growth rate coefficient
	L_b	length	length at birth
Auxiliary parameters	δ_L	volume$^{1/3}$ length^{-1}	structural length (L) to physical length (L_w) conversion coefficient
	d_V	mass volume^{-1}	structural density
	L_w	Length	physical (measured) length
	w_E	weight mole^{-1}	molecular mass of reserve

$\bar{\mu}_E$	energy mole^{-1}	chemical potential of reserve

Table S3. The variables and parameters of the Ontogenetic Growth Model.

	Symbol	Dimension	Interpretation
State variables	W	mass	wet mass
	E	energy	stored energy (total biomass content)
	E_R	energy	energy to egg
	μ'	–	relative development
Forcing variables	X	energy volume^{-1} (or surface^{-1})	food energy density per unit environmental volume or surface area
Core parameters	$\{\dot{p}_O\}$	energy time^{-1} mass$^{-3/4}$	specific anabolic rate
	$[\dot{p}_M]$	energy time^{-1} mass^{-1}	specific maintenance rate
	$[E_g]$	energy mass^{-1}	specific overhead cost of growth
	$[E_c]$	energy mass^{-1}	specific energy content of tissue
	F	–	activity scope (multiplier on $\{\dot{p}_O\}$ for determining assimilation)
	λ	–	energy allocation parameter (growth *versus* reproduction overheads)
	t_s	time	time at spawning (at a reference temperature)
	t_{dev}	time	time at completion of development (at a reference temperature)
	W_b	mass	mass at birth
	K	energy volume^{-1} (or surface^{-1})	
Compound parameters	$f = \frac{X}{X+K}$	–	food restriction factor

	$W_{w,\infty} = \left(\frac{f\{\dot{p}_O\}}{[\dot{p}_M] + \lambda/t_s}\right)^4$	mass	ultimate mass
	$L_\infty = \left(\frac{f\{\dot{p}_O\}}{[\dot{p}_M] + \lambda/t_s}\right)^{4/3}$	length	ultimate volumetric length
	$\dot{r}_B = \frac{[\dot{p}_M] + \lambda/t_s}{4[E_g]}$	energy time^{-1} mass^{-1}	relative (von Bertalanffy) growth rate coefficient
	$a = \frac{\{\dot{p}_O\}}{[E_c]}$	energy time^{-1} mass$^{-3/4}$	anabolic coefficient
	$b = \frac{[\dot{p}_M]}{[E_g]}$	energy time^{-1} mass^{-1}	maintenance coefficient
Auxiliary parameters	δ_W	volume$^{1/3}$ mass^{-1}	volumetric length to wet mass conversion coefficient
	L_W	length	physical (measured) length
	d_V	mass volume^{-1}	density of total body

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