CHAPTER 12
The Metabolic Theory of Ecology

Often and often it happens that our physical knowledge is inadequate to explain the mechanical working of the organism; the phenomena are superlatively complex, the procedure is involved and entangled, and the investigation has occupied but a few short lives of men.

D'Arcy Wentworth Thompson

We have seen in previous chapters how temperature affects any process involving a movement of energy, and thereby all of physiology. Given that physiologists have studied the influence of temperature on many cellular processes, and done so for a long time, it is perhaps surprising that until recently no general theory of thermal physiology had emerged. Despite a good understanding of how temperature affects many individual processes, we had no synthetic theory that linked all of these results into a single coherent picture of how organisms deal with temperature.

And then in the late 1990s, two ecologists from the University of New Mexico, Jim Brown and Brian Enquist, together with a physicist from the nearby Los Alamos Laboratory, Geoffrey West, decided to take a close look at metabolism. Their initial work was concerned with how metabolic rate varies with body size, and they developed a model (or models, as there is more than one) based upon the architecture of distribution systems in plants and animals. Soon afterwards, in collaboration with Jamie Gillooly, they added a temperature term to the model to derive the fundamental equation of the Metabolic Theory of Ecology (MTE). From this basic theory, the originators, together with a series of students and collaborators, have examined the implications of the MTE for a wide range of ecological processes and patterns.

This theory has generated enormous controversy. Because it makes explicit recognition of the role of temperature in governing the flow of energy and materials through organisms, it has the potential to form a general theory of thermal ecology of wide applicability. It therefore deserves close scrutiny from any physiologist or ecologist interested in temperature. Before examining the theory in detail, however, it is helpful to establish the historical context.

12.1 The influence of size: scaling

Ask someone to describe a mouse or an elephant and it is almost certain that they will mention its size. Size, and especially size relative to ourselves, is a key feature of any object. We also know intuitively that size influences shape; an elephant does not look like a huge mouse, nor does a shrew resemble a small buffalo. These contrasts in shape are less marked in water, where a large fish can look pretty similar to a small fish. This difference between land and water has much to do with the need for terrestrial animals to support their own weight against gravity. These observations introduce a topic that has been central to the debate about the relationship between metabolic rate and size: geometric similarity or size invariance.

Two objects of the same general form show geometric similarity if the ratio of two corresponding linear measurements (say length or breadth) are the same. This concept goes back to the Ancient Greek geometers, and quite likely as far back as the
(the Richards function). These equations are essentially descriptive (what physicists call phenomenological), although some were based on physiological arguments. In particular Ludwig von Bertalanffy argued, based on Pütter's original work, that growth resulted from a balance between catabolic processes utilising reserves and anabolic processes synthesising new tissues. Assuming that anabolism (synthesis) was limited by the surface area over which the organism gains nutrients whereas catabolism was a function of body mass, von Bertalanffy suggested that \( \alpha = 2/3 \) (this being the ratio of surface area to body mass) and, on the basis that anabolic processes occur throughout the body, \( \beta = 1.7. \) This equation can be solved analytically and in mass terms it is:

\[
M_t = M_\infty \left(1 - e^{-K(t-t_0)}\right)^3
\]

Here \( M_t \) is the mass at time \( t \), \( M_\infty \) is the asymptotic mass and \( t_0 \) is the hypothetical time at which mass is zero. \( K \), the von Bertalanffy growth coefficient, defines the rate at which the growth curve approaches \( M_\infty \). In a more complex version of his model, von Bertalanffy recognised a series of metabolic and growth rate 'types' defined by different scaling of metabolic rate with mass. The basic model has also been modified to describe species where growth is seasonal. An alternative physiological interpretation came from Michael Reiss who proposed that the first term \( (am^\alpha) \) represented the assimilation and the second \( (bm^\beta) \) the metabolic costs of existence.

The von Bertalanffy growth equation has long been popular with biologists interested in growth. This is despite the assumed exponents being shown to be unrepresentative of the real world: for example an early comparative study of fish growth suggested that the best estimate of \( \alpha \) was 0.59 (rather than 2/3) and \( \beta = 0.83 \) (rather than 1). Recently ecologists have revisited the problem, attempting to build new growth models from physiological principles. Before we look at these we need to establish the key energetic features of growth, and how these are affected by temperature.

### 13.2 The energetics of growth

Growth requires both raw materials and energy. The raw materials include amino acids for proteins, simple sugars for polysaccharides, purines and pyrimidines for nucleic acids and fatty acids for lipids. The energy required to synthesise macromolecules from monomers is supplied by ATP or GTP. The nutrients that provide both the raw materials for synthesis and substrates for the regeneration of ATP by intermediary metabolism are carried to where they are required by the circulatory system. Once the nutrients reach the cell they are used either as raw material or to regenerate ATP (Figure 13.2).
treatment of the energetics of growth requires knowledge of both pathways. This has long been recognized by microbial ecologists, who routinely consider the role of entropy and heat dissipation in bacterial growth, but by relatively few ecologists concerned with the growth of plants, invertebrates or vertebrates.

Revisiting the balanced energy budget we explored in Chapter 4, we can express the total energy cost of synthesising new somatic tissue, $E_s$, as:

$$E_s = P_s + R_e$$

where $P_s$ is the chemical potential energy sequestered in the new tissue and $R_e$ is the metabolic cost of synthesising that tissue. $R_e$ is termed variously the metabolic (or sometimes calorimetric) cost of growth or the metabolic overhead of synthesising new tissue. A better term might be the thermodynamic cost of growth because of its basis in entropy, although practical measures of $R_e$ inevitably include metabolic costs additional to those associated with bond formation. If we assume that $R_e$ is a constant proportion of $P_s$ (and we express both in the same units) then we can calculate a dimensionless fractional cost of growth, $c$:

$$c = \frac{R_e}{P_s}$$

and hence

$$E_s = P_s + cP_s$$

Because $R_e$ is part of the total metabolic rate measured during growth, it is not easy to determine. While we can estimate a minimum cost of synthesising a protein from the ATP requirement for peptide bond synthesis, this ignores a large number of associated costs, such as RNA processing, transport within the cell, post-translational modification of the newly synthesised proteins, the recycling of proteins that fold incorrectly and so on. We can estimate an overall cost of growth, however, from the relationship between new tissue production and respiration. If we assume that the cost of growth, $R_e$, is additional to the costs of tissue maintenance and also the general activity required in existence, then (after controlling for the effects of mass on both growth rate and metabolic rate) we would expect a positive linear relationship between the rate of growth and metabolic rate. The slope of this relationship reflects the thermodynamic cost of growth (a higher cost leading to a steeper slope). Data for a range of aquatic organisms suggests a value for $c$ of around 0.32 (Figure 13.3).

It is this metabolic cost of growth that underpins the widely observed correlation between respiration and production at the population level. Two

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**Figure 13.3** The cost of growth. A (left panel): a simple conceptual model showing the relationship between metabolic rate and growth rate for an individual organism. BMR: basal metabolic rate. The slope of the relationship between metabolic rate and growth rate reflects the thermodynamic cost of growth; a higher cost results in a steeper line. B (right panel): across-species relationship between rate of growth (production) and metabolic rate. The line is a least-squares regression with a slope of 0.32.
comprehensive studies have shown linear relationships between production and respiration in a range of animal populations over seven orders of magnitude of annual production. The relationships for endotherms and ectotherms exhibited similar slopes but were offset (with endotherms having a higher respiration rate for a given production rate). While measurements at a population level will be affected by a range of additional factors, such as the respiratory costs of non-growing individuals, the general result is precisely that to be expected from the thermodynamics of growth.

It is clear immediately that a simple estimate of the chemical potential energy content of new tissue underestimates the actual energy needed for growth by leaving out the metabolic cost of assembling that tissue. Equally a measure of energy dissipated during growth (for example by estimating metabolic rate by oxygen consumption) misses entirely the energy retained in the new tissue. The relationship between these two measures is shown schematically in Figure 13.4.

The best estimate of $c$ is $0.33$ (Table 13.1). This exceeds the cost of peptide synthesis by a factor of about 3, indicating the importance of the associated costs. There is also an indication from work on isolated fish hepatocytes that the cost varies with the rate of protein synthesis, which suggests that while the basic cost of assembling a protein does not change (that is, the ATP cost for synthesis of peptide bonds is invariant), the associated costs can. At present we do not know which of these associated costs changes, or why.

13.3 Temperature and growth

Growth at the cellular level involves a complex and carefully regulated sequence of chemical, mechanical and diffusional events. In eukaryotes this involves transcription of messenger RNA (mRNA) in the nucleus, modification of the mRNA (removal of introns), transfer of the mRNA to the ribosome in the cytoplasm, elongation of the new peptide, post-translational modification and often transport of the new protein to where it is needed. As we saw previously in discussing metabolism, the temperature dependence of such a complex system cannot be predicted theoretically; all we can do is examine the effect of temperature empirically and hope to describe this with relatively simple statistics.

One step in the chain where the temperature sensitivity has been studied in some detail is protein synthesis and an early study is shown in Figure 13.5. Here protein synthesis in isolated hepatocytes from

<table>
<thead>
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<th>Study</th>
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<tr>
<td>1</td>
<td>0.12</td>
<td>Estimated for the marine copepod Calanus hyperboreus growing rapidly with growth fuelled from lipid reserves; calculated from the observed partial growth efficiency of 89%</td>
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<tr>
<td>2</td>
<td>0.25-0.43</td>
<td>Derived from studies of a variety of domesticated and cultured vertebrates, taking extreme values of the partial growth efficiency to be 70% and 80%</td>
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<tr>
<td>3</td>
<td>0.32</td>
<td>Estimated from the slope of the relationship between metabolic rate and growth rate in the amphibian Bufo bufo</td>
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<td>4</td>
<td>0.33</td>
<td>A consensus value derived from studies of fish and aquatic invertebrates</td>
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<tr>
<td>5</td>
<td>0.32</td>
<td>Estimated from growth of the garter snake Thamnophis sirtalis. Original measure 1.67 kJ g$^{-1}$ (wet mass) converted assuming a water content of 75% and a tissue energy content of 20.7 kJ g$^{-1}$</td>
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<tr>
<td>6</td>
<td>0.51</td>
<td>Estimated from the slope of the relationship between metabolic rate and growth rate in the timber rattlesnake, Crotalus horridus</td>
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mark a genuine threshold between mesophile and thermophile bacteria, and the temperature range from 42 °C to 60 °C has been termed the mesophile-thermophile gap.

13.5 Modelling growth

The growth of an organism is such an important feature of its interaction with its environment and other organisms that capturing the main features in a simple manner is critical to ecosystem modelling. As with any model, the aim is to capture the key elements of growth in as few parameters as possible without leaving out anything important. The enormous variation in observed growth rates of all organisms might suggest that there are a large number of factors at work, and that capturing the main drivers in a simple model would be difficult. Two models (strictly they are groups of models) that have attracted much recent attention are the dynamic energy budget and the ontogenetic growth model. These two models differ greatly in their aims, structure and formulation.

13.5.1 The dynamic energy budget

The dynamic energy budget (DEB) tracks the energy content of an organism through its life cycle from embryo to adult. As we saw in Chapter 4 it can describe growth well, but its disadvantage for many ecologists is that the data needed to estimate the core variables and parameters are not always available. DEB models are thermodynamically rigorous and comprehensive, but their applicability can be limited by our lack of knowledge of the system being modelled.

While a powerful tool for modelling the energetics of an individual species and capturing how this varies across its range, or might alter as climate changes, DEB models are not always the most useful way to capture general features of the growth of whole assemblages, such as is needed for ecosystem models. For this we need more general models.

13.5.2 The ontogenetic growth model

The ontogenetic growth model, OGM, was developed by Geoffrey West, James Brown and Brian Enquist, and is a development of the Metabolic Theory of Ecology, MTE, which we explored in Chapter 12. The OGM starts with the assumption that the total metabolic rate of an organism is the sum of energy devoted to growth and the energy required to maintain existing biomass:

$$ B = E_a \frac{dm}{dt} + B_m m $$
where \( B \) is the total metabolic rate (\( W \)), \( m \) the body mass at time \( t \), \( E_m \) the energy required to synthesise new tissue (\( J \ g^{-1} \)) and \( B_m \), the energy required to maintain a unit of biomass (\( W \ g^{-1} \)). Allowing for the scaling of metabolic rate as formulated in the MTE (where \( b_1 \) is a coefficient that varies with taxon and metabolic level and has to be derived empirically):

\[
B = b_1 m^\alpha
\]

and rearranging produces an equation identical in form to the Pütter equation:

\[
\frac{dm}{dt} = am^\alpha - bm
\]

where \( a = \frac{bE_m}{E_n} \), \( b = \frac{b_n}{E_n} \) and \( \alpha = 0.75 \) as predicted by the MTE. While the OGM is a simple Pütter equation, the authors argue that it is derived more rigorously than any previous growth model with coefficients that are related directly to physiological variables.

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\text{the incoming rate of energy flow and quantified as the average resting metabolic rate of the whole organism at time } t, \text{ and } B_m \text{ is defined as the power needed to sustain the organism in all of its activities. This introduces an unfortunate ambiguity, because it would seem to imply that } B \text{ is equivalent to basal or resting metabolic rate, BMR, and } B_m \text{ equivalent to daily energy expenditure, DEE. In a typical mammal DEE exceeds BMR by a factor of about } 3-4, \text{ and yet the OGM equation requires } B > B_m. \text{ We saw in Chapter 8 that resting metabolic rate is a measure of energy dissipation. While this dissipated energy includes the thermodynamic cost of growth (\( R \) in Figure 13.4), it does not include the chemical potential energy retained in the new tissue (\( P \) in Figure 13.4); indeed it cannot without contravening the conservation of energy. In the OGM, however, the cost of producing new tissue \( E_m \) is estimated from the energy content of mammalian tissue, which is a measure of \( P \) rather than \( R \) (Figure 13.4). Neither is it clear how the OGM allows for metabolic expenditure required for daily activity (since \( B \) is defined as resting metabolic rate). The lack of clarity over definitions and appropriate estimation makes it very difficult to evaluate the model further.}

The original OGM took no account of temperature, but a temperature term was introduced subsequently; as with the MTE itself, this was a simple Boltzmann factor. The implications of incorporating temperature were explored for the growth of juvenile stages well below adult size, where it could be assumed that maintenance costs are negligible. The model described embryonic development of a range of organisms well, and the fit was improved further by including a term of stoichiometry (specifically C:P ratio, which captures, in part the concentration of RNA in the cell). The Boltzmann factor was applied to both terms in the OGM equation, which meant that adult mass was independent of temperature. In nature, however, there is a striking relationship between body size and temperature, which we explore below. This indicates that the relationship between temperature and growth is more subtle and complex than was captured in the OGM by a simple Boltzmann factor.

A subsequent revised version of the OGM redefined some terms. In this version \( B \) is described as the rate of energy assimilation, which is quite different from its definition in the original OGM as the rate of energy dissipation. More importantly \( E_m \) is now defined as the total metabolic work the organism expends to create biomass from preformed organic molecules, and explicitly does not include the energy content of the new tissue. This is a clear definition of the thermodynamic cost of growth (\( R \) in Figure 13.4) and marks a fundamental shift in the conceptual basis for \( E_m \) from the original OGM. Their estimates of \( E_m \) are derived by comparing metabolic rate with growth rate in embryos and juveniles of vertebrates, when maintenance costs are assumed to be small. Estimates of \( E_m \) for juveniles ranged from 4.0–7.5 \( \text{kJ g}^{-1} \), based on tissue growth as wet mass. Conversion to dry mass yields values that exceed previous estimates of the thermodynamic cost of growth (Table 13.1) by up to four-fold.

The model was revised further by Chen Hou and colleagues, and this version (the extended OGM) distinguishes explicitly between the use of assimilated food to provide energy for maintenance, energy for activity, energy for synthesis and the raw materials for new tissue. This model thus has a structure identical to the balanced energy budget developed in the early part of the twentieth century.
where $B$ is the total metabolic rate ($W$), $m$ the body mass at time $t$, $E_m$ the energy required to synthesise new tissue (J g$^{-1}$) and $B_m$ the energy required to maintain a unit of biomass (W g$^{-1}$). Allowing for the scaling of metabolic rate as formulated in the MTE (where $b_3$ is a coefficient that varies with taxon and metabolic level and has to be derived empirically):

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and rearranging produces an equation identical in form to the Pütter equation:

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$$B = b_0 m^n$$

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(see Chapter 4), though it also has the valuable development of including scaling. The extended OGM model captures the growth of endothermic vertebrates very well, but the authors acknowledge that it has yet to be tested in ectothermic vertebrates or invertebrates.

An important aspect of the OGM is that it suggests that beneath the variety of growth trajectories observed in nature lies a 'universal growth model'. This arises when a dimensionless mass ratio, \( r \), is plotted as a function of a dimensionless time variable, \( \tau \), where

\[
r = \left( \frac{m}{M} \right)^{\frac{1}{4}}
\]

and

\[
\tau = \frac{a}{4M^{\frac{1}{4}}} \ln \left[ 1 - \left( \frac{m_0}{M} \right)^{\frac{1}{4}} \right]
\]

Here \( m_0 \) is the mass at birth and \( M \) the maximum body mass (other variables are as defined above). The universal growth curve is then:

\[
r = e^{-\tau}
\]

This captures the growth rate of a variety of vertebrates very well, including both endotherms and ectotherms and also species with determinate or indeterminate growth (Figure 13.9). Although one species of invertebrate (the planktonic mysid shrimp *Mysis mixta*) was included, subsequent more extensive analyses suggested that the universal growth model was not a good description of growth in organisms where the life history includes a fundamental change in morphology between larval and adult stages, such as insects and aquatic invertebrates.

The obvious question is why, if the logical foundation and parameterisation of the OGM is unclear, should the universal growth model derived from it describe vertebrate growth data so well? The answer would appear to be that the form of the universal growth model is dictated principally by the scaling parameters in the OGM. Variations in the growth and metabolic variables have much less impact, as suggested by the tightness of the data about the model. If so, this points to the importance of the structure of the cardiovascular system in determining not just the scaling of metabolism in vertebrates, but also of growth. This is perhaps not so surprising because the same cardiovascular system supplies the tissue with oxygen for metabolism and raw material for growth. It may also explain why the universal growth model is not so good at capturing the growth of invertebrates, with their very different circulatory systems.

![Figure 13.9](image_url)

*Figure 13.9* The universal growth model, UGM. A (left panel): UGM fitted to data for vertebrate growth. B (right panel): UGM fitted to data for aquatic invertebrates. In both cases the raw growth data are shown as circles and the fitted UGM as a solid line.