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# "Dynamic Energy Budgets in Biological Systems"

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# Dynamic Energy Budgets in Biological Systems

## Theory and Applications in Ecotoxicology

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I give estimates in this book for standard deviations for many parameter values that are obtained from experimental results, to indicate accuracy. I follow this standard procedure with some hesitation on two grounds. The first reason to doubt the usefulness is that the value of the standard deviation is rather sensitive to the stochastic part of the model, which might not be very realistic, as discussed. The second reason is that such standard deviations do not account for correlations between parameters. A small standard deviation for a parameter, therefore, does not necessarily mean that such a parameter is known accurately, an error that is easy to make.

# Chapter 2

# Individuals

From a systems analysis point of view, individuals are special because at this organization level it is relatively easy to make mass balances. This is important, because the conservation law for mass and energy is one of the few hard laws available in biology. At the cellular and at the population level it is much more difficult to measure and model mass and energy flows. It will be argued on {245} that life started as an individual in evolutionary history rather than as a particular compound, such as RNA. The individual is seen as an entity separated from the environment by physical barriers. Discussion should, therefore, start at the level of the individual.

While developing the DEB theory in the next chapter, I will present many tests against experimental data. These tests require careful interpretation of data that makes use of the material presented in this chapter, which introduces some general concepts that relate to individuals.

## 2.1 Input/output relationships

Any systems model relates inputs to a system with outputs of that system, as a function of its state. Although many formulations suggest that the output is the result of the state of the system and its input, this directional causality is, in fact, a matter of subjective interpretation. Input, state and output display simultaneous behaviour, without an objective, directional causality. The DEB model for uptake and use of energy in terms of input/output descriptions is neutral with respect to the interpretation in terms of 'supply' and 'demand'. With the 'supply' interpretation, I mean that the lead is in the feeding process, which offers an energy input to the individual. The available energy flows towards different destinations, more or less as water flows through a river delta. With the 'demand' interpretation, I mean that the lead is in some process using energy, such as maintenance and/or growth, which requires some energy intake. Food searching behaviour is then subjected to regulation processes in the sense that an animal eats what it needs. I think that in practice species span the whole range from 'supply' to 'demand' systems. A sea-anemone, for example, is a 'supply' type of animal. It is extremely flexible in terms of growth and shrinkage, which depend on feeding conditions. It can survive a broad range of food densities. Birds are examples of a 'demand' system and they can only survive

at relatively high food densities. The range of possible growth curves is thus much more restricted.

Even in the 'supply' case, growth may be regulated carefully by hormonal control systems. Growth should not proceed at a rate beyond the possibility of mobilizing energy and elementary compounds necessary to build new structures. Models that describe growth as a result of hormonal regulation should deal with the problem of what determines hormone levels. The answer invokes the individual level. The conceptual role of hormones is linked to the similarity of growth patterns despite the diversity of regulating systems. In the DEB theory, messengers such as hormones are part of the physiological machinery by which an organism regulates its growth. Their functional aspects can only be understood from other variables and compounds.

Balance equations are extremely useful for the specification of constraints for the simultaneous behaviour of input, state and output of systems. The problem of unnoticed sources of sinks can only be circumvented by precise book-keeping. The possibility of being able to formulate balance equations will turn out to be the most useful aspect of the abstract quantity 'energy', cf. {41}. The conservation law for energy was originally formulated by von Mayer [461] in 1842. Precursors of the principle of conservation of energy go back as far as Leibnitz in 1693 [118]. This law is known today as the first law of thermodynamics. The law of conservation of mass was first described in a paper by Lavoisier in 1789.

## 2.2 State variables

Many models for growth have age as a state variable. Age itself has excellent properties as a measuring-tape, because it has a relatively well defined starting point (here taken to be the start of embryogenesis and not birth, i.e. the transition from the embryonic state to the juvenile one). It can also be measured accurately. Some well-studied species only thrive on abundant food supply, which results in well-defined and repeatable size-age curves. This has motivated a description of growth in terms of age, where food is considered as an environmental variable, like temperature, rather than a description in terms of input/output relationships and energy allocation rules.

One frequently applied model was proposed by Gompertz in 1825:

$$W(t) = W_\infty (W_0/W_\infty)^{\exp(-\dot{\gamma}t)}$$

where $W(t)$ is the weight, usually the wet weight, of an individual of age $t$ and $\dot{\gamma}$ the Gompertz growth rate. The individual grows from weight $W_0$ asymptotically to weight $W_\infty$. This is essentially an age-based model, which becomes visible from a comparison of alternative ways to express it as a differential equation: $\frac{d}{dt} \ln W = -\dot{\gamma} \ln \frac{W}{W_\infty}$ or $\frac{d^2}{dt^2} \ln W = -\dot{\gamma} \frac{d}{dt} \ln W$. The first equation states that the weight-specific growth rate decreases proportionally to the logarithm of weight as a fraction of ultimate weight. (Note that the notation $\frac{d}{dt} \ln W$ suggests a dimension problem, because it looks as if the argument of a transcendental function is not dimensionless. Its mathematically equivalent notation $W^{-1} \frac{d}{dt} W$, shows that no dimension problem exists here.) It is hard to put a mechanism



Figure 2.1: These talking gouramis, *Trichopsis vittatus*, come from the same brood and therefore have the same age. They also grew up in the same aquarium. The size difference resulted from competition for a limited amount of food chunks, which amplified tiny initial size differences. This illustrates that age cannot serve as a satisfactory basis for the description of growth

growth rate decreases proportionally with the growth rate, which can be linked to a simple aging mechanism where the ability to grow fades according to a first order process. In the situation of abundant food, this model usually gives an acceptable fit. The problems with this model and similar ones become apparent when growth has been measured at different food availabilities.

Figure 2.1 shows two fish from the same brood, which have lived in the same 5 litre aquarium. Their huge size difference shows that age-based growth models are bound to fail. The mechanism behind the size difference in this case is the way of feeding, which involved a limited number of relatively big food chunks for the whole brood. Initially, the size differences were very small, but the largest animal always took priority over its smaller siblings, which amplified the size differences. Similar results apply to prokaryotes, which have a poor control over age-at-division at constant substrate density, but a high control over size-at-division [398].

Apart from empirical reasons for rejecting age as a state variable for the description of growth, it cannot play the role of an explanatory variable from a physical point of view. Something that proceeds with age, such as damage caused by free radicals, cf. {106}, can play that role. One will need an auxiliary model to show in detail how such a variable depends on age. One of the problems with the Gompertz model and related ones is that growth does not result from a difference between an uptake and a usage term. It is formulated as an intrinsic property of the organism. The environment can only affect

When feeding is conceived as input of energy, size must be one of the state variables. A large individual eats much more than a small one, so it is hard to imagine a realistic model for growth that does not have size as one of the state variables; however, many quantities can be taken to measure size. Examples are volume, wet weight, dry weight, ash-free dry weight, amount of carbon or energy etc. Originally I thought that, to some extent, they were more or less exchangeable, depending on the species. Now, I am convinced that volume is the only natural choice to measure size in the context of the present theory, where surface areas play such an important role. A volume (organism), living in another volume (environment), is bound to communicate with it over a surface area. The DEB theory makes use of the interpretation of the ratio of size and surface area in terms of length. Weights remain of considerable practical interest for several reasons, the most important ones relating to the implementation of mass balances. The relationships between size measures will be discussed in the next section.

Size alone is not sufficient to describe the process of energy uptake and cannot be used with any degree of accuracy. Energy reserves should be considered as well, even in the most simple models. There are several reasons for this.

The first one is the existence of maintenance, i.e. a continuous drain of energy necessary to keep the body going. Feeding on particles, even if these particles are molecules, implies that there are periods in which no particles arrive. The capacity of a digestive system cannot realistically be made big enough to smooth out the discrete arrival process, in order to 'pay' the steady costs for maintenance. Other costs are paid as well in the absence of any food input. Spectacular examples of prolonged action without food intake are the European, North American and New Zealand eels *Anguilla*, which cease feeding at a certain moment. Their alimentary canal even degenerates, prior to the 3000 km long journey to their breeding grounds, where they spawn. The male emperor penguin *Aptenodytes forsteri* breeds its egg in Antarctic midwinter for two months and feeds the newly hatched chick with milky secretions from the stomach without access to food. The male loses some 40% of its body weight before assistance from the female arrives.

The second argument for including storage is that individuals react slowly to changes in their feeding conditions. Again, this cannot be described realistically with the digestive system as a buffer because its relaxation time is too short.

The third argument is that well-fed individuals happen to have a different (chemical) body composition than individuals in poor feeding conditions. The type of difference depends on the species, as will be discussed later. Originally I thought that, as long as food density is constant, one can do without storage. This is why the first version of the DEB model [416], did not have energy storage. However, when growth at different food densities is compared and storage levels depend on food density, one should include storage even under these simple conditions.

Size and stored energy should play a role in even the simplest model for the uptake and use of energy. Several other state variables, such as the content of the digestive system, energy density of the blood etc., will be necessary to describe the finer details of some physiological processes, but they need not play a significant role at the population level. For the purpose of the analysis of population dynamics and the contribution of aging therein, it makes sense to introduce age as an auxiliary third state variable. It also proves



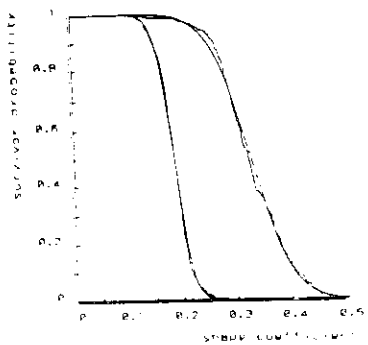


Figure 2.2: The sample survivor function (see glossary) of shape coefficients for European birds (left) and Neotropical mammals (right). The lengths include the tail for the birds, but not for mammals. Data from Bergmann and Helb [58] and Emmons and Feer [203]. The fitted survivor functions are those of the normal distribution.

necessary to distinguish life stages to catch qualitative differences in energetics, cf. [49].

## 2.3 Size and shape

### 2.3.1 Length/surface area/volume relationships

The shape that organisms can take resists any accurate description when different species are compared. For an understanding of energetics two aspects of size and shape are relevant, as will be explained later: surface areas for acquisition processes and volumes for maintenance processes. Shape defines how these measures relate to each other. Measurements of lengths and weights are usually easy to obtain in a non-destructive way, so the practical problem has to be solved of how these measurements are related to surface areas and volume.

Volume is rather difficult to measure for some species. As a first crude approximation, wet weights, $W_w$, i.e. the weight of a living organism without adhering water, can be converted to volumes, $V$, by division through a fixed specific density $[d_w]$, which is close to 1 g cm$^{-3}$. So $W_w = [d_w]V$ or $[W_w] = [d_w]$, where $[d_w]$ is here taken to be a (fixed) parameter. If an organism does not change its shape during development, an appropriately chosen length measure, $L$, can be used to obtain its volume. The length is multiplied by a fixed dimensionless shape coefficient $d_m$ and the result is raised to the third power. So $V = (d_m L)^3$. The shape coefficient, defined as volume$^{1/3}$length$^{-1}$, is specific for the particular way the length measure has been chosen. Thus the inclusion or exclusion of a tail in the length of an organism results in different shape coefficients. A simple way to obtain an approximate value for the shape coefficient belonging to length measure $L$ is on the basis of the relationship $d_m = (\frac{W_w}{[d_w]})^{1/3} L^{-1}$.

The following considerations help in getting acquainted with the shape coefficient. For a sphere of diameter $L$ and volume $L^3\pi/6$, the shape coefficient is 0.806 with respect to the diameter. For a cube with edge $L$, the shape coefficient takes the value 1, with respect to this edge. The shape coefficient for a cylinder with length $L$ and diameter $L_c$ is $(\frac{\pi}{4})^{1/3}(L/L_c)^{-2/3}$ with respect to the length.

Table 2.1: The means and coefficients of variation of shape coefficients of European birds and mammals and Neotropical mammals.

| Taxon | source | number | mean tail included | cv | mean tail excluded | cv |
|---|---|---|---|---|---|---|
| European birds | [203,105] | 418 | 0.186 | 0.14 | | |
| European mammals | [94] | 128 | 0.233 | 0.27 | 0.335 | 0.28 |
| Neotrop. mammals | [58] | 246 | 0.211 | 0.41 | 0.328 | 0.18 |

The shapes of organisms can be compared in a crude way on the basis of shape coefficients. Figure 2.2 shows the distributions of shape coefficients among European birds and Neotropical mammals, they fit the normal distribution closely. Summarizing statistics are given in table 2.1, which includes European mammals as well. Some interesting conclusions can be drawn from the comparison of shape coefficients. They have an amazingly small coefficient of variation, especially in birds including sphere-like wrens and stick-like flamingos; which probably relates to constraints for flight. Mammals have somewhat larger shape coefficients than birds, because they tend to be more spherical and this possibly relates to differences in mechanics. The larger coefficient of variation indicates that the constraints are perhaps less stringent than for birds. The spherical shape is more efficient for energetics because cooling is proportional to surface area and a sphere has the smallest surface area/volume ratio, namely $6/L_e$. When the tail is included in the length, European mammals have somewhat larger shape coefficients than Neotropical mammals, but the difference is absent when the tail is excluded. Neotropical mammals tend to have longer tails, which is probably due to the fact that most of them are tree dwellers. The temperature differences between Europe and the Neotropics do not result in mammals in Europe being more spherical to reduce cooling.

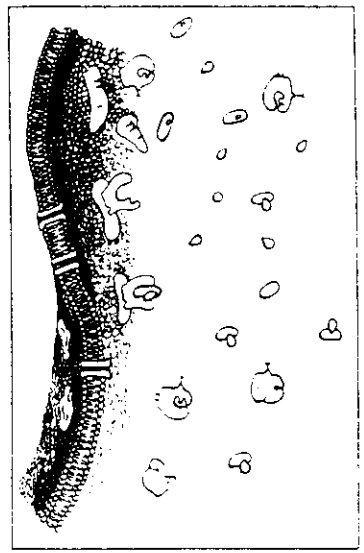
These considerations should not obscure the practical purpose of shape coefficients: to convert shape-specific length measures to volumetric lengths, i.e. cubic roots of volumes. Each parameter that has length in its dimensions is sensitive to the way that lengths have been measured (in- or excluding extremities, etc.). As long as the comparison is made between bodies of the same shape, there is no need for concern, but as soon as different shapes are compared, it is essential to convert length to volumetric length, the rationale being that a comparison made on the basis of unit volumes of organisms is made on the basis of cells.

### 2.3.2 Isomorphism

Isomorphism is an important property which applies to the majority of species on earth. It refers to conservation of shape as an individual grows in size. The shape can be any shape and the comparison is only between shapes that a single individual takes during its development. Two bodies of a different size are isomorphic if it is possible to transform one body into the other by a simple geometric scaling in three dimensional space: scaling involves only multiplication, translation and rotation. This implies, as Archimedes already knew, that if two bodies have the same shape and if a particular length takes value $L_1$ and $L_2$ in the different bodies, the ratio of their surface areas is $(L_1/L_2)^2$ and that of their volumes $(L_1/L_2)^3$, irrespective of their actual shape. It is, therefore, possible to make assertions about the surface area and the volume of the body relative to some standard, on the basis of length only. One only needs to measure the surface area or volume if absolute values are required. This property will be used extensively.



The significance of the relationship between length, surface area and volume for isomorphs does not show up in the first place, in the context of practical measurement, but for the body itself.



These relationships play an essential role in the communication between the extensive variable body size and intensive variables such as concentrations of compounds and reaction rates between compounds. Secreting organs 'know' their volume relative to body volume by the build up of the concentration of their products in the body. Each cell in the body 'knows' its volume by the ratio between its volume and the surface area of its membranes. One mechanism is that most enzymes only function if bound to a membrane, with their substrates and products in the cell volume as illustrated. The functional aspect is that the production of enzymes is a relatively slow process, a handicap if a particular transformation needs to be accelerated rapidly. Most enzymes can be conceived of as fluffy, free floating structures, with performance depending on the shape of the outer surface of the molecule and the electrical charge distribution over it.

If bound to a membrane, the outer shape of the enzyme changes into the shape required for the catalysis of the reaction specific to the enzyme. Membranes thus play a central role in cellular physiology [249,292,763]. Many pathways require a series of transformations and so involve a number of enzymes. The binding sites of these enzymes on the membrane are close to each other, so that the product of one reaction is not dispersed in the cytosol before being processed further. The product is just handed over to the neighbouring enzyme in a process called piping. Interplay between surface areas and volumes is basic to life, not only at the level of the individual, but also at the molecular level.

Most species are approximately isomorphic. It is not difficult to imagine the physiological significance of this. Process regulating substances in the body tend to have a short lifetime to cope with changes, so such substances have to be produced continuously. If some organ secretes at a rate proportional to its volume (i.e. number of cells), isomor-

phism will result in a constant concentration of the substance in the body. The way the substance exercises its influence does not have to change with changing body volume in order to obtain the same effect in isomorphs.

## Exoskeletons

Isomorphism itself poses no constraints on shape, but if organisms have a permanent exoskeleton, then stringent constraints on shape exist and as most animals with a permanent exoskeleton actually meet these constraints, it is helpful to work them out. This subsection can be skipped without loss of continuity.

A grasshopper remains isomorphic and has an exoskeleton, but it grows by moulting, thus the exoskeleton is not permanent and isomorphism poses no constraints in this case. The same holds for an organism which resembles a sphere, such as a sea urchin; it cannot have a permanent (rigid) exoskeleton, because the curvature of its surface changes during growth. A cylindrical organism that grows in length only, is not isomorphic. A cylindrical organism that grows isometrically has only its caps as a permanent exoskeleton; thus this includes only the caps, i.e. two growing disks separated by a growing distance. The permanent exoskeleton generally represents a (curved) surface in three dimensional space, which can be described in a simple way using logarithmic spirals. The idea of the logarithmic spiral or *spira mirabilis* (in the plane) goes back to Descartes' studies of *Nautilus* in 1638 and to Bernoulli in 1692. The function has been used by Thompson [713], Rudwick [616,617] and Raup [584,585] to describe the shape of brachiopods, ammonites and other molluscs. I will rephrase their work in modern mathematical terms and extend the idea a bit.

A natural starting point for a description of the isomorphic permanent exoskeleton is the mouthcurve. This is a closed curve in three dimensional space that describes the 'opening' of the permanent exoskeleton (shell). This is where the skeleton synthesizing tissue is found. The development of the exoskeleton can, in most cases, be retraced in time to an infinitesimally small beginning, giving the permanent exoskeleton just the one 'opening'. This method avoids the problem of the specification of the shape of an invisibly small object. To follow the mouth curve back in its development, we introduce a dummy variable $l$, which has the value 0 for the present mouth curve and $-\infty$ at the start of development. By placing the start of development at the origin, the test on isomorphism of the developing exoskeleton is reduced to mapping one exoskeleton to another by multiplication and rotation only (so no translation). We can always orient the exoskeleton such that the rotation is around the $x$-axis. Let $\mathbf{R}(l)$ denote the rotation matrix

$$\mathbf{R}(l) = \begin{pmatrix} 1 & 0 & 0 \\ 0 & \cos l & \sin l \\ 0 & -\sin l & \cos l \end{pmatrix}$$

The closed mouthcurve $\mathbf{m}$ at an arbitrary value for the dummy variable $l$, can be described by

$$\mathbf{m}(l) = e^{l/2\pi}\mathbf{R}(-l)\mathbf{m}(0) \tag{2.1}$$

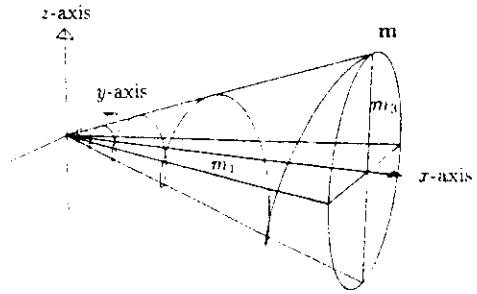


where $c$ is a constant describing how fast the mouth curve reduces in size when the exoskeleton rotates over an angle $2\pi$. If $c$ is very large, it means that the exoskeleton does not rotate during its reduction in size. Size reduction relates in a special way to the rotation rate to ensure (self) isomorphism. It follows from the requirement that for any two points $\mathbf{m}_0$ and $\mathbf{m}_1$ on the mouthcurve, the distance $\|\mathbf{m}_1(l+h) - \mathbf{m}_0(l)\|$ depends on $l$ in a way that does not involve the particular choice of points. The rotation matrix is here evaluated at argument $-l$, because most gastropods form left handed coils. For right handed coiling $l$, rather than $-l$, should be used. The mouth curve, together with the parameter $c$ determine the shape of the exoskeleton.

An arbitrary point on the mouth curve will describe a logarithmic spiral to the origin. To visualize this, it helps to realize that a simple function such as the standard circle is given by $\mathbf{f}(l) = (\sin l, \cos l)$, where the dummy variable $l$ takes values between $-\infty$ and $\infty$. A graphical representation can be obtained by plotting $\sin l$ against $\cos l$. Similarly, the logarithmic spiral with the vertex at the origin through the point $\mathbf{m}(0) \equiv (m_1, 0, m_3)$ is given by

$$\mathbf{f}(l) = e^{l/2\pi}(m_1, m_3 \sin -l, m_3 \cos -l) \tag{2.2}$$

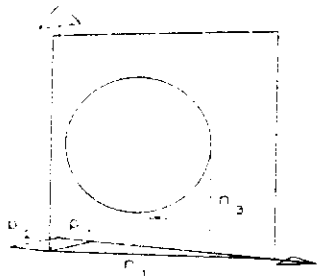
It lies on a cone around the $x$-axis with vertex at the origin, and tangent $m_3/m_1$ of the diverging angle with respect to the $x$-axis. For increasing $l$, the normalized direction vector of the spiral from the vertex, $(m_1, m_3 \sin -l, m_3 \cos -l)/\|\mathbf{m}\|$, with $\|\mathbf{m}\| = \sqrt{m_1^2 + m_3^2}$, describes a circle in the $y,z$-plane at $x$-value $m_1/\|\mathbf{m}\|$.




Until now, no explicit reference to time has been made. If the length measure of the animal follows a von Bertalanffy growth pattern, i.e. $1 - \exp\{-\delta t\}$ for $t \in (0, \infty)$, cf. {81}, the relationship $e^{l/2\pi} = 1 - \exp\{-\delta t\}$ results. So, $l = \frac{2\pi}{\ln c}\ln\{1 - \exp\{-\delta t\}\}$. I will argue on {81} that this is realistic when food density and temperature remain constant. In winter, when growth ceases in the temperate regions and calcification partially continues in molluscs, a thickening of the shell occurs, which is visible as a ridge ringing the shell. If the gradual transitions between the seasons can be neglected, these ridges will be found at $l = \frac{2\pi}{\ln c}\ln\{1 - \exp\{-\delta\tau\}\}$, $\tau = 1, 2, 3, \ldots$ when the unit of time is one growth season. In principle, this offers the possibility of determining the von Bertalanffy growth rate $\delta$ from a single shell found on the sea shore.

The mouth curve in living animals with a permanent exoskeleton frequently lies more or less in a plane, which reduces the specification of the three dimensional mouth curve to a two dimensional one, plus the specification of the plane of the mouth curve, which involves two extra parameters. The exoskeleton can always be oriented such that the plane of the mouth curve is perpendicular to the $x,y$-plane and the mouth opening is facing negative $y$-values.

Let $\mathbf{p} \equiv (p_1, p_2, 0)$ denote a point in the plane of the mouth curve, such that this plane is perpendicular to the vector $\mathbf{p}$ and $p_2 \leq 0$. (Remember that the axis of the spiral is the $x$-axis with the vertex at the origin so that the orientation of the exoskeleton is now completely fixed.) The mouth curve $\mathbf{n}$ in the plane is now measured using the point $\mathbf{p}$ as origin. If the mouth curve is exactly in a plane, a series of two coordinates suffice to describe the exoskeleton together with $c$, $p_1$ and $p_2$.



If it is not exactly in a plane, we can interpret the plane as a regression plane and still use three coordinates, where the $y$-values are taken to be small. The relationship between $\mathbf{n}$ measured in the coordinate system with the plane of the mouth curve as $x, z$-plane and $\mathbf{p}$ as origin with the original three dimensional mouth curve $\mathbf{m}$ is:

$$\mathbf{m} = \mathbf{p} + \begin{pmatrix} -p_2/\|\mathbf{p}\| & -p_1/\|\mathbf{p}\| & 0 \\ p_1/\|\mathbf{p}\| & -p_2/\|\mathbf{p}\| & 0 \\ 0 & 0 & 1 \end{pmatrix} \mathbf{n} \tag{2.3}$$

More specifically, if the mouth curve is a circle with radius $r$ and the centre point at $(q_1, 0, q_3)$, we get $\mathbf{n}(\phi) = (q_1 + r\sin\phi, 0, q_3 + r\cos\phi)$, for an arbitrary value of $\phi$ between 0 and $2\pi$. This dummy variable just scans the circle. The 6 parameters $c$, $p_1$, $p_2$, $q_1$, $q_2$ and $r$ completely fix both shape and size of all isomorphic exoskeletons with circular mouth curves. If only the shape is of interest, we can choose $r$ as the unit of distance, which leaves 5 free parameters for a full specification.

This class of morphs is too wide because it includes physically impossible shapes. The orientation of the mouth curve should be such that a mouth opening results and the shape may not 'bite' itself when walking along the spiral. This constraint can be translated into the constraint that the intersections of the exoskeleton with the $x, z$-plane should not intersect each other. The intersections of the mouth curve with the $x, z$-plane are easy to construct, given points on the mouth curve. When the point $\mathbf{m}_i \equiv (m_1, m_2, m_3)$ on the mouth curve $\mathbf{m}(0)$ spirals its way back to the vertex, it intersects the $x, z$-plane at $e^{l_i/2\pi}\mathbf{R}(l_i)\mathbf{m}_1$, with $l_i = i\pi - \arctan m_2/m_3$ for $i = 0, -1, -2, \cdots$.

The distinction Raup [584] made between a generating curve and a biological one is purely arbitrary and has neither biological nor geometric meaning: Raup raises the problem that realistic values for the parameters he uses to characterize shape tend to cluster around certain values. Schindel [634] correctly pointed out that this depends on the particular way of defining parameters, and he used the intersection of (2.1) with the $x, z$-plane to characterize shape and showed that realistic values for parameters of this curve did not cluster. Any parameterization, however, is arbitrary unless it follows the growth mechanism. This shape of permanent exoskeletons is dealt with here to show that the shape is a result of the isomorphic constraint.



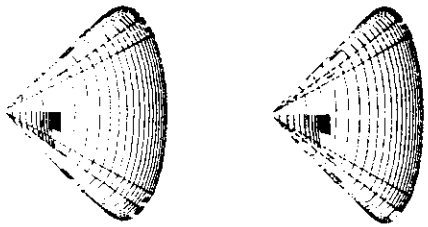
*Nautilus* has a fixed number of septa per revolution. This is to be expected as it makes a septum as soon as the end chamber in which it lives exceeds a given proportion of its body size. (The fact that the septa in subsequent revolutions frequently make contact implies that *Nautilus* somehow knows the number $\pi$.) These septa cause the shell to be no longer isomorphic in the strict sense, but to be what can be called periodically isomorphic, by which I mean that isomorphism no longer holds for any two values of $l$, but for values that differ by a certain amount. Many gastropods are sculptured at the outer surface of their shell; this sculpture is formed by the mantle curling around the shell edge. The distance from the shell edge and the height of the sculpture relates to the actual body size, the result being a shell that is also periodically isomorphic. Sculpture patterns that do not follow the mouth curve, but follow the logarithmic spirals, do not degrade isomorphism. Some shells of fully grown ammonites and gastropods have a last convolution that deviates in shape from the previous ones, showing a change in physiology related to life stage; this will be discussed later, {83,150}.

Most shapes are simple and correspond to special cases where the mouth curve lies in a plane. For $p_1 = 0$, the mouth curve lies in a plane parallel to the $x, z$-plane; shapes such as *Planorbis* and *Nautilus* result if the mouth curve is symmetrical around the $x, y$-plane. A growing sheet is obtained when $p_1 \to 0$ and $p_2 = 0$ so that the mouth curve lies in the $y, z$-plane. Age ridges can still show logarithmic spirals (in the plane), depending on the value of $c$. Figure 2.3 gives a sample of possible shapes. Although the shell of *Spirula* is internal rather than external, this does not spoil the argument.

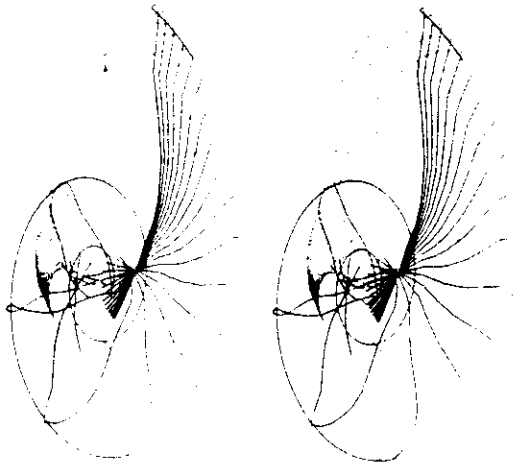
From an abstract point of view, the closed mouth curve can secrete exoskeletons to either side and no formal restrictions exist for the parameters describing their surfaces. (The biological reality is that two mouth curves are lined up and can be moved apart to let the animal interact with the environment.) Animals such as bivalves have two logarithmic spirals sharing the same mouth-curve, one turns clockwise, one anti-clockwise. Many gastropods also have a second exoskeleton, the plane-like operculum, which is so small that it easily escapes notice. Gastropods of the genera *Berthelinia*, *Julia* and *Midorigai* have two valves, much like the bivalva. As illustrated in figure 2.4, more complex shape are possible when the mouth curve is branched.

### 2.3.3 Changing shapes

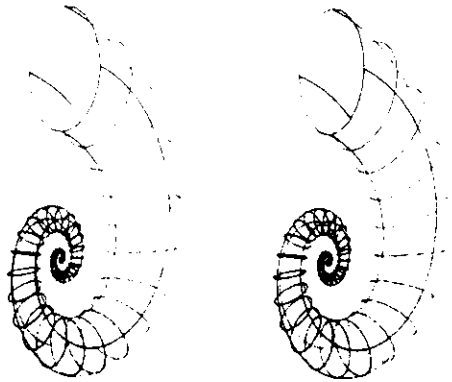
Huxley [345] described how certain parts of the body can change in size relative to the whole body, cf. {252}. He used allometric functions to describe this change and pointed to the problem that if some parts change in an allometric way, other parts can not. From an energetics point of view, the change in relative size of some extremities is not very important. The total volume is of interest because of maintenance processes, and certain surface areas for acquisition processes. The fact that wings, for instance, have a delayed development in birds is of little relevance to whole body growth. The basic problem is in the relationship between the size measure and the volume that has to be maintained. Reserve materials allocated to reproduction contribute substantially to the trunk length of the larvacean *Oikopleura*, but do not require maintenance. I will show on {147} how such



*Patella*, $c \to \infty$, $p_2 = 0$

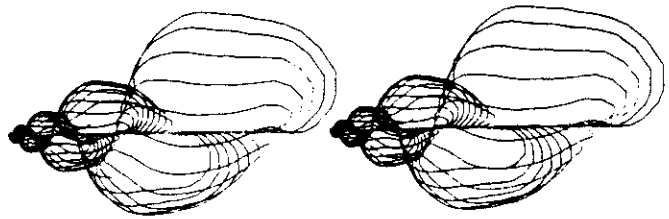


*Nautilus*, $c = 3$, $p_1 = 0$, $p_2 \to 0$

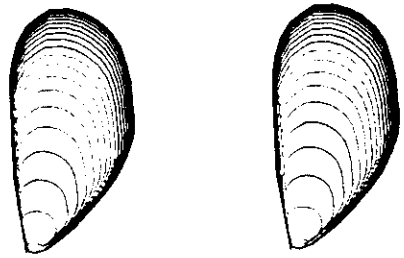


*Spirula*, $c = 5$, $p_1 = 0$, $p_2 \to 0$





*Lymnaea*, $c = 2$, $p_1 = 0$, $p_2 \to 0$



*Mytilus*, $c = 10^4$, $p_1 \to 0$, $p_2 = 0$

*Ensis*, $c = 10^5$, $p_1 \to 0$, $p_2 = 0$

Figure 2.3: A sample of possible shapes of isomorphs with permanent exoskeletons. The mouth curves are shown at equal steps for the dummy argument (*Lymnaea*, *Spirula*) or for time. Illuminate well and evenly to obtain the stereo effect. Hold your head about 50 cm from the page with the axis that connects your eyes exactly parallel to that for the figures. Do not focus at first on the page but on an imaginary point far behind the page. Try to merge both middle images of the four you should see this way. Then focus on the merged image. If this fails, try stereo glasses. If the grey is in front, rather than at the background, you are looking with your right eye to the left picture. Prevent this with a sheet of paper placed between your eyes and the page. About 10% of people actually look with one eye only and thus fail to see depth. If necessary, test this by raising one finger in front of your nose and counting the number of raised fingers that you see while focusing at infinity.

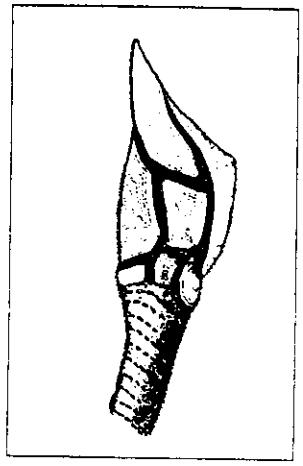


Figure 2.4: The goose barnacle (*Scalpellum scalpellum*) has an exoskeleton with a large number of components, each belonging to the family (2.1); it is an example of a branched mouth curve. Tetrahedrons provide an example of permanent exoskeletons with three branching points in the mouth curve and cubes with eight. If the (branched) mouth curve is a globular network, the exoskeleton can even resemble a sphere.

Some species such as echinoderms and some insects change shape over different life-stages. Some of these changes do not give problems because food intake is sometimes restricted to one stage only. If the shape changes considerably during development, and if volume has been chosen as the basis for size comparisons, the surface area related processes should be corrected for these changes in shape. A convenient way to do this is by means of the dimensionless shape correction function $\mathcal{M}(V)$, which stands for the actual surface area relative to the isomorphic one for a body with volume $V$, where a particular shape has been chosen as the reference. The derivation of this function will be illustrated for two important examples that will occur throughout the book: filamentous hyphae of fungi and rod-shaped bacteria. These organisms are both very important from a biological point of view and they serve to illustrate the important notion of 0D- and 1D-isomorphs.

If a filament can be conceived as a cylinder with variable length, and thus variable volume $V$, but a fixed diameter $L_\phi$, its surface area equals $A(V) = 4VL_\phi^{-1}$ if the caps are excluded. Suppose now that the cylinder grows isomorphically from the start. The surface area of the isomorphic cylinder, i.e. a cylinder that has a diameter proportional to its length, is proportional to $V^{2/3}$. The constant of proportionality depends on the choice of a reference volume, say $V_d$. The isomorphic surface area is thus $A_d(V/V_d)^{2/3}$, where $A_d$ denotes the surface area of a cylinder with volume $V_d$. So the shape correction function for filaments becomes

$$\mathcal{M}(V) = \frac{4VL_\phi^{-1}}{4V_dL_\phi^{-1}(V/V_d)^{2/3}} = (V/V_d)^{1/3} \qquad (2.4)$$

It is not essential that the cross section through a filament is circular, it can be any shape, as long as it does not change during growth. The important aspect is that growth is isomorphic in one direction. So it must be possible to the orient the body such that the direction of growth is along the $x$-axis, while no growth occurs along the $y$- and $z$-axes. The different body sizes can be obtained by multiplication of the $x$-axis by some scalar $l$. By doing so, both the surface area and the volume are



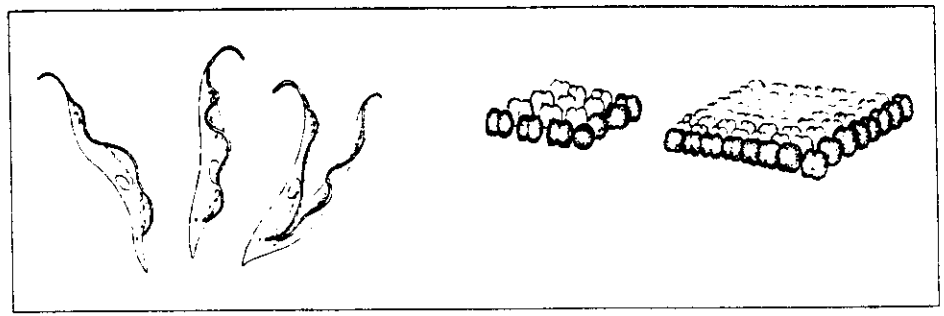
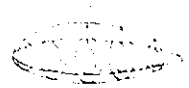


Figure 2.5: Left: The kinetoplastid *Trypanosoma* does not grow in the longitudinal direction, along which it divides. The change in shape is like a 2D-isomorph. Right: The blue-green bacterial colony *Merismopedia* is only one cell layer thick. Although this sheet also grows in two dimensions, it is a 1D-isomorph. The arrangement of the cells requires an almost perfect synchronization of the cell cycli.

multiplied by $l$, so $A = A_dV/V_d$, if the surface area at $V = 0$ is negligibly small. Division by the isomorphic surface area $A_d(V/V_d)^{2/3}$ results in the shape correction function for filaments. Filaments are therefore 1D-isomorphs, while the isomorphs of the previous subsection are in fact 3D-isomorphs.



Several unicellulars divide longitudinally, such as members of several classes of the phylum *Zoomastigina* (*Opalinita*, *Retortamonadida*, *Choanomastigotes*, *Kinetoplastida*) and some filamentous bacteria (spirochetes [333]). The notorius *Trypanosoma*, which cause sleeping sickness, are among these unicellulars; see figure 2.5. Some are filamentous, but do in some respects just the opposite of the above mentioned filaments: they grow in diameter rather than length. To illustrate the concept of the shape correction function, I will derive here the shape correction function for this growth pattern, assuming that growth perpendicular to the longitudinal axis of the body is isomorphic and that no growth occurs in the longitudinal direction.

Thus it is possible to orient the body with its axis of no growth along the $x$-axis and multiply both the $y$- and $z$-axes by some scalar to obtain different body sizes. There are no restrictions in shape, as long as growth in the $y,z$-plane is isomorphic and for each $x,y$-value there are a limited number of $z$-values. The body need not be rotationally symmetric, it can taper towards its ends. Multiplying the $y$- and $z$-axis by some value $l$ results in a multiplication of the surface area of the body by $l$ and of the volume by $l^2$. (To see this, one should realize that surface area can be written as $A = \int_0^{L_d} L_c(x)\,dx$, where $L_c(x)$ denotes the circumference of the cross section through the body at $x$, and $L_d$ the length of the body in the longitudinal direction. Multiplying the $y$- and $z$-axes by some value $l$ results in a multiplication of $L_c(x)$ with $l$, while $L_d$ remains untouched. Likewise, volume can be written as $V = \int_0^{L_d} A_c(x)\,dx$, where $A_c(x)$ denotes the surface area of the cross section at $x$, which is multiplied by $l^2$.) For some reference volume $V_d$ for $l = 1$, we

have thus $l = \sqrt{V/V_d}$, or $A = A_d\sqrt{V/V_d}$, where $A_d$ denotes the surface area of a body with volume $V_d$. The surface area of a 3D-isomorph is $A_d(V/V_d)^{2/3}$, so that the shape correction function for 2D-isomorphs is

$$\mathcal{M}(V) = A_d\sqrt{V/V_d}(A_d(V/V_d)^{2/3})^{-1} = (V_d/V)^{1/6} \tag{2.5}$$

This correction function for 2D-isomorphs *decreases* with the cubic root of a length measure, while for 1D-isomorphs (filaments) it *increases* with a length measure.

A subtlety of this reasoning can be illustrated by sheets, i.e. flat bodies that only grow in two dimensions, with a constant, but small, height. The archaebacterium *Methanoplanus* fits this description.
Several colonies, such as the sulphur bacterium *Thiopedia*, the blue-green bacterium *Merismopedia* and the green alga *Pediastrum* also fall into this category; see figure 2.5. How sheets grow in two dimensions does not matter: they may change wildly in shape during growth. Height must be small to neglect the contribution of the sides to the total surface area. The surface area of the sheet relates to its volume as $A(V) = 2VL_h^{-1}$, where $L_h$ denotes the height of the sheet and the factor 2 accounts for the upper and lower surface area of the sheet. Division by the isomorphic surface area $A(V_d)(V/V_d)^{2/3}$ gives $\mathcal{M}(V) = (V/V_d)^{1/3}$, as for filaments, i.e. 1D-isomorphs. This may come as a surprise since sheets have much in common with 2D-isomorphs, where height $L_h$ plays the same role as longitudinal length $L_d$. The important difference is that for $x = 0$ and $x = L_h$, there are infinitely many $z$-values for appropriately chosen $y$-values, a case that has been excluded for 2D-isomorphs. This suggests an obvious route for mixtures of 1D- and 2D-isomorphs: thick sheets that grow isomorphically in two dimensions. So the upper and lower surface areas behave as a 1D-isomorph, while the sides behave as a 2D-isomorph.

This conclusion invites a examination of the contribution of the caps in filaments. This can best be done via the introduction of biofilms, conceived as super-organisms, which resemble sheets, but in some ways they do the opposite; they grow in height rather than in the direction of the sheet, but the increase in surface area is negligibly small. A biofilm on a plane can be conceived formally as a 0D-isomorph. Its surface area is just $A_d$, while that of a 3D-isomorph is still $A_d(V/V_d)^{2/3}$, which leads to the shape correction function for 0D-isomorphs

$$\mathcal{M}(V) = (V_d/V)^{2/3} \tag{2.6}$$

Films relate to sheets as 1D-isomorphs relate to 2D-isomorphs. Films frequently occur in combination with 1D-isomorphs, as will be shown.

Cooper [137] argues that at constant substrate density *Escherichia* grows in length only, while the diameter-length ratio at division remains constant for different substrate densities.
(For the use of the term 'density', see the remark under (3.3).) This mode of growth and division is typical for most rod-shaped bacteria, and most bacteria are rod-shaped. Shape and volume at division, at a given substrate density, are selected as a reference. The cell



then has, say, length $L_d$, diameter $\delta L_d$, surface area $A_d$ and volume $V_d$. The fraction $\delta$ is known as the aspect ratio of a cylinder. The index $d$ will be used to indicate length, surface area and volume at division at a given substrate density. The shape of the rod shaped bacterium is idealized by a cylinder with hemispheres at both ends and, in contrast to a filament, the caps are now included. Length at division is $L_d = \left(\frac{4V_d}{(1-\delta/3)\delta^2\pi}\right)^{1/3}$, making length $L = \frac{\delta}{3}\left(\frac{4V_d}{(1-\delta/3)\delta^2\pi}\right)^{1/3} + \frac{4V}{\pi\delta^2}\left(\frac{(1-\delta/3)\delta^2\pi}{4V_d}\right)^{2/3}$. Surface area becomes $A = L_d^2\frac{\pi}{3}\delta^2 + \frac{4V}{\delta L_d}$. The surface area of an isomorphically growing rod equals $A_d(V/V_d)^{2/3}$. The shape correction function is the ratio of these surface area's. If volume, rather than length, is used as an argument the sought, dimensionless, correction function becomes

$$\mathcal{M}(V) = \frac{\delta}{3}\left(\frac{V_d}{V}\right)^{2/3} + \left(1 - \frac{\delta}{3}\right)\left(\frac{V}{V_d}\right)^{1/3} \tag{2.7}$$

When $\delta = 0.6$, the shape just after division is a sphere as in cocci, so this is the upper boundary for aspect ratio $\delta$. This value is obtained by equating the volume of a cylinder to that of a sphere with the same diameter. When $\delta \to 0$, the shape tends to a filament.

The shape correction function for rods can now be conceived as a weighted sum of those for a 0D- and a 1D-isomorph, with a simple geometric interpretation of the weight coefficients. A cylinder with blunt caps has the shape correction function

$$\mathcal{M}(V) = \frac{\delta}{\delta+2}\left(\frac{V_d}{V}\right)^{2/3} + \frac{2}{\delta+2}\left(\frac{V}{V_d}\right)^{1/3} \tag{2.8}$$

which is again a weighted sum of correction functions for 0D- and 1D-isomorphs. For the aspect ratio $\delta \to \infty$, the shape can become arbitrary close to a 0D-isomorph. The exact geometry of the caps is thus of less importance for surface area/volume relationships. Rods are examples of static mixtures of a 0D- and a 1D-isomorph, i.e. the weight coefficients do not depend on volume. Crusts are examples of dynamic mixtures and will be discussed on {145}.
The table right summarizes the shape correction functions for isomorphs of different dimensions. The power of the scaled volumes has an odd relationship with the dimension of isomorphy. Mixtures of 1D- and 0D- or 2D-isomorphs can resemble 3D-isomorphs, depending the weight coefficients and the range of values for the scaled volume.

| Dim | $\mathcal{M}(V)$ |
|---|---|
| 0 | $(V/V_d)^{-2/3}$ |
| 1 | $(V/V_d)^{1/3}$ |
| 2 | $(V/V_d)^{-1/6}$ |
| 3 | $(V/V_d)^{0}$ |

## 2.3.4 Weight/volume relationships

In the discussion about shape coefficients, {21}, the crude relationship $W_w = [d_w]V$ was used to relate wet weight to structural biovolume. This mapping in fact assumes homeostasis, see {38}, without a decomposition of the organism into a structural and a storage

component. Almost all of the literature is based on this relationship or the similar one for dry weights: $W_d = [d_d]V$.

For some purposes in energetics, such relationships between volumes and weights are far too crude. One needs a more refined definition of size to distinguish structural body volume from energy reserves. The necessity of making this distinction originates, among other things, from the quantification of metabolic costs. These costs are not paid for reserve materials: this is most obvious for freshly laid bird eggs. Such eggs are composed almost entirely of reserve materials and use practically no oxygen, as will be discussed later, {84,103}.

Some convenient size measures, such as weight, suffer more from the contribution of reserves than others. For example, energy allocated to reproduction, but temporarily stored in a buffer, will contribute to weight, but not to structural body volume. Energy reserves replace water in many aquatic species [553,788], but in the human species, for instance, energy reserves are often (painfully) visible. Energy reserves generally contribute more to dry weight than to wet weight [248]. While wet weight is usually easier to measure and can be obtained in a non-destructive way, dry weight has a closer link to chemical composition and mass balance implementations. I will show on {192} how to separate structural body mass from reserves and determine the relative abundances of the main elements for both categories on the basis of dry weight.

The relationship between wet weight $W_w$ and dry weight $W_d$ and structural body volume, $V$, non-allocated energy reserves $E$, and energy reserves allocated to reproduction $E_R$ is

$$W_w = [d_{wv}]V + [d_{we}](E + E_R)/[E_m] \quad (2.9)$$

$$W_d = [d_{dv}]V + [d_{de}](E + E_R)/[E_m] \quad (2.10)$$

where $[E_m]$ denotes the maximum non-allocated reserve energy density as discussed in the next chapter. Its occurrence here is just to obtain the dimension weight volume$^{-1}$ for the density $[d_{we}]$ and it is part of the tactic to avoid measurement of energies if not strictly required. If food is *ad libitum*, the energy reserve $E$ will be found to evolve to $[E_m]V$ in the DEB theory, so that the energy reserves will then contribute $[d_{we}]V$ to wet weight. Under this condition weight is thus proportional to volume, apart from the possible contribution of reserves allocated to reproduction. If energy reserves replace water and the specific density of the energy reserves equals that of water, we have $[d_{we}] = 0$. If their specific density is less than that of water because of a high lipid content, for instance, $[d_{we}]$ can be negative if the reserves still replace water. The conversion coefficients $[d_{**}]$ have fixed values, due to homeostasis for the structural biomass and the reserves, see {38}.

Although this relationship between weight and structural biovolume is more accurate than a mere proportionality, it is by no means 'exact' and it depends again on the species. The gut contents of earthworms, shell of molluscs, exoskeleton of crustaceans and calcareous skeleton of corals do not require maintenance and for this reason they should be excluded from biovolume and weight. In the finer details, all species pose specific problems for the interpretation of size measurement. The contribution of inorganic salts to the dry weight of small marine invertebrates is frequently substantial. Figure 2.6 illustrates the interpretation problem in the measurement of ash-free dry weight in relation to length in



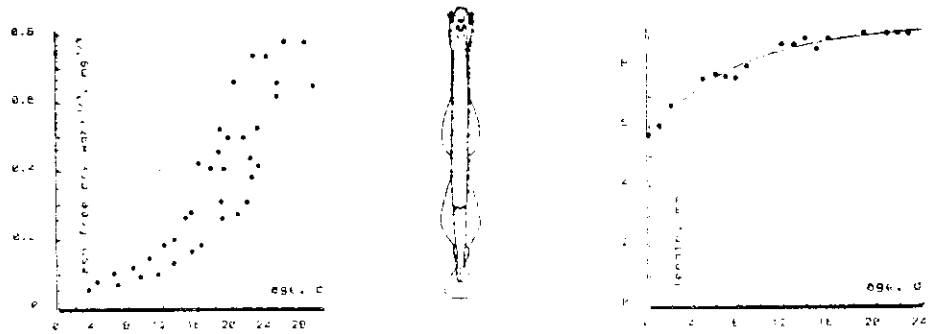


Figure 2.6: The ash-free dry weight and the length of the cheatognat *Sagitta hispida*. Data from Reeve [589,588]. The curve through the lengths is $L(t) = L_\infty - (L_\infty - L_0)\exp\{-\dot{r}t\}$.

cheatognats. Length measurements follow the expected growth pattern closely at abundant food, while the description of weight seems to require an *ad hoc* reasoning. Although quickly said, this is an important argument in the use of measurements within a theoretical context: if an explanation that is not species-specific competes with a specific one, the first explanation should be preferred if the arguments are otherwise equally convincing. Since energy reserves contribute to weight and are sensitive to feeding conditions, weights usually show much more scatter, in comparison to length measurements. This is illustrated in Figure 2.7.

The determination of the size of an embryo is complicated by the extensive system of membranes the embryo develops in order to mobilize stored energy and materials and the decrease in water content during development [766]. In some species, the embryo can be separated from 'external' yolk. As long as external yolk is abundant, the energy reserves of the embryo without that yolk, if present at all, will on the basis of DEB theory turn out to be a fixed fraction of wet and dry weight, so that the embryo volume is proportional to weight. Uncertainty about the proportionality factor will hamper the comparison of parameter values between the embryonic stage and the post-embryonic one.

The aqueous fraction of an organism is of importance in relation to the kinetics of toxicants. The aqueous weight is the difference between wet weight and dry weight, so $W_a = W_w - W_d$. It can be written as $W_a = [d_{wa}]V$, for

$$[d_{wa}] = [d_{wv}] - [d_{dv}] + ([d_{we}] - [d_{de}])\frac{E + E_R}{[E_m]V} \quad (2.11)$$

The contribution of the last term, which stands for that of the reserves to the volume-water weight conversion, is probably small in most cases. The volume occupied by water is $V_a = W_a/d_a$, where $d_a$ stands for the specific density of water, which is close to 1 g cm$^{-3}$. The aqueous fraction of structural body volume is thus $V_a/V = [d_{wa}]/d_a$ and typically takes values between 0.7 and 0.9.

It is possible to use variations in weight relative to some measure of length to indicate

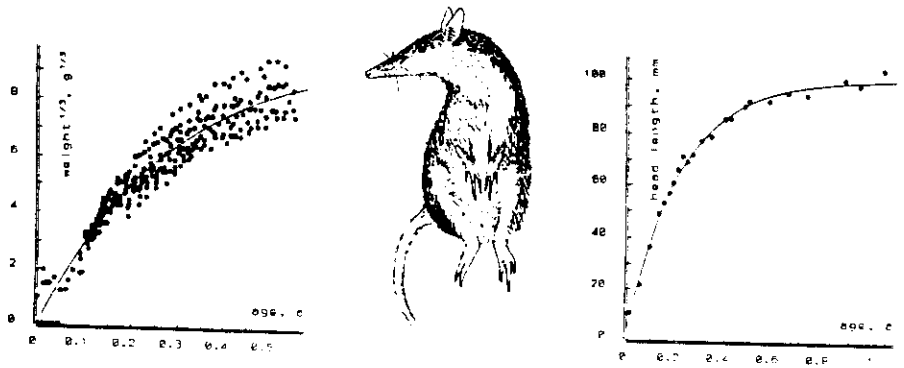


Figure 2.7: The weight to the power 1/3 and the head length of the long-nosed bandicoot *Perameles nasuta*. Data from Lyne [444]. The curves are again $L(t) = L_\infty - (L_\infty - L_0)\exp\{-\dot{\gamma}t\}$.

variations in energy reserves. This has been done for birds [552], and fish. There exists a series of coefficients to indicate the nutritional condition of fish, e.g. (weight in g)×(length in cm)$^{-1}$, which is sometimes used with a factor 1, 10 or 100. It is known as the condition factor, Hile's formula or the ponderal index [7,240,318,343].

The problems energy reserves pose for the finer details of the definition of size are not restricted to weights. They also affect the relationship between total volume and structural volume, in a way comparable to wet weights for species that do not interchange water for energy reserves. I see structural volume and energy reserves as rather abstract quantities that define the state of an organism. DEB theory specifies how the behaviour of the organism depends on its state. In addition to that, we need theories that relate these abstract quantities to things we can measure in order to substantiate the claim that DEB theory is about the living world. This subsection presents such an auxiliary theory of how weights (things we can measure) relate to the (abstract) state variables structural volume and energy reserves. This mapping rests on a key concept of DEB theory: homeostasis. An intimate relationship thus exists between the (DEB) theory that is based upon abstract quantities and the auxiliary theory that relates abstract quantities to measurements. Later, {103,192}, I will show that this relationship is even more intimate for the auxiliary theory that relates respiration measurements to (abstract) energy fluxes. The reason for discussing the relationship between weight and structural volume and energy reserves in a chapter prior to the developing the DEB theory is to stress the distinction between 'core' theory of abstract variables and auxiliary theories of relationships with measurements. If the core theory is no longer useful, the auxiliary theories should be thrown away automatically. However, it is possible to change to other auxiliary theories, without changing the core theory.

The relationship between volume, reserves and mass will be worked out further in the section on mass-energy coupling, {192}.



### 2.3.5 C-mole/volume relationships

The microbiological tradition is to express the relative abundances of the elements hydrogen, oxygen and nitrogen in dry biomass relative to that of carbon and to conceive the combined compound so expressed as a kind of abstract 'molecule' that can be counted and written as $CH_{n_{HW}}O_{n_{OW}}N_{n_{NW}}$. For each C-atom in dry biomass, there are typically $n_{HW} = 1.8$ H-atoms, $n_{OW} = 0.5$ O-atoms and $n_{NW} = 0.2$ N-atoms for a randomly chosen micro-organism [608]. This gives a 'molecular weight' of $w_W = 24.6$ g mol$^{-1}$, which can be used to convert dry weights into what are called 'C-moles'. The relative abundances of elements in biomass-derived sediments largely remain unaltered on a geological time scale, apart from the excretion of water. In the geological literature the Redfield ratio C:N:P = 105:15:1 is popular [587], or for silica bearing organisms such as diatoms, radiolarians, silico-flagelates and (some) sponges C:Si:N:P = 105:40:15:1. This literature usually excludes hydrogen and oxygen, because their abundances in biomass-derived sediments change considerably during geological time. Other bulk elements in organisms are S, Cl, Na, Mg, K and Ca, while some 14 other trace elements play an essential role, as reviewed by Fraústo da Silva and Williams [231]. The ash that remains when dry biomass is burnt away is rich in these elements. Since ash-weight is typically some 5% of dry weight, I will here include the first four most abundant elements only, but the inclusion of more elements is straightforward. As stated before, some taxa require special attention on this point.

As for weight densities, the chemical composition of biomass cannot be taken constant for most purposes in energetics, {192}. If a 'molecule' of structural biomass is denoted by $CH_{n_{HV}}O_{n_{OV}}N_{n_{NV}}$ and of energy reserves by $CH_{n_{HE}}O_{n_{OE}}N_{n_{NE}}$, the relative abundances in dry biomass are for $[E] \equiv E/V$ given by

$$n_{*W} = \frac{n_{*V}[d_{mv}] + n_{*E}[d_{me}][E]/[E_m]}{[d_{mv}] + [d_{me}][E]/[E_m]} \tag{2.12}$$

where $*$ stands for $H$, $O$ or $N$ and $[d_{mv}]$ and $[d_{me}]$ denote the conversion coefficients from structural biovolume and energy volume to C-mole. These conversion coefficients have simple relationships with those from volume to dry weight, because the 'molar weight' of structural biovolume and energy reserves are given by

$$\begin{aligned} w_V &\simeq [d_{dv}]/[d_{mv}] = 12 + n_{HV} + 16n_{OV} + 14n_{NV} \quad \text{gram mol}^{-1} \\ w_E &\simeq [d_{de}]/[d_{me}] = 12 + n_{HE} + 16n_{OE} + 14n_{NE} \quad \text{gram mol}^{-1} \end{aligned}$$

since the contribution of the other elements to dry weight is negligibly small. The problem of uncovering the relative abundances $n_{*V}$ and $n_{*E}$ from measurements of $n_{*W}$ will be discussed on {192}.

As is standard in the microbiological literature, the concept of C-mole will be extended to (simple) substrates, the difference from an ordinary mole being that it always has at most 1 C-atom. For reasons of consistency of notation, substrate density $X_0$ will be expressed on a volume per volume basis, while $d_{mx}X_0$ gives substrate as C-mole per volume. The same strategy will be used for products that are produced by micro-organisms: $[d_{mp}]$ converts volume of product into mole of product.

## 2.4 Homeostasis

The compounds that cells use to drive metabolism require enzymes for their chemical transformation. Compounds that react spontaneously are excluded. In this way cells achieve full control over all transformations, because they synthesize enzymes, consisting of protein, themselves. No reaction runs without the assistance of enzymes. The properties of enzymes depend on their micro-environment. So homeostasis, i.e. a constant chemical composition, is essential for full control. Changes in the environment in terms of resource availability, both spatial and temporal, require the formation of reserve pools to ensure a continuous supply of essential compounds for metabolism. This implies a deviation from homeostasis. The cells solution to this problem is to make use of polymers that are not soluble. In this way these reserves do not change the osmotic value. In many cases cells encapsulate the polymers in membranes, to reduce interference even further, at the same time increasing access, as many cellular activities are membrane bound.

Reserve materials can be distinguished from materials of the structural biomass by a change in relative abundance if resource levels change. This defining property breaks down in case of extreme starvation when structural materials are degraded as well as when reserves are exhausted. An example of this is the break down of muscle tissue in mammals such as ourselves, which must be considered as structural material. The distinction between reserves and structural materials is meant to accommodate the fact that some materials are more mobile than others. DEB theory builds on a two-way classification and in fact assumes homeostasis implicitly for structural biomass and reserve separately, via two other assumptions. Homeostasis is assumed for the structural materials because the volume-specific energy costs for growth are assumed to be constant, as explained in the next chapter. The assumption that the energy content of reserve materials is just proportional to the amount of reserve material, without any labels relating to their composition, in fact implies the assumption of homeostasis for reserve materials as well, and because the amount of reserves can change relative to the structural materials, the chemical composition of the whole body can change. That is, it can change in a particular way. This is a consequence of the choice of energy as a state variable rather than the complete catalogue of all compounds.

Storage and structural compounds differ in the way in which they are non-permanent in organisms. Storage materials are continuously used and replenished, while structural materials, and in particular proteins, are subjected to continuous degradation and reconstruction. Most proteins (enzymes) have a fragile, tertiary structure, which results in very short mean, functional lifetimes. Energy costs for protein turnover are included in maintenance costs. The DEB model assumes no maintenance for energy reserves.

The two-way classification of compounds into permanent (structural biomass) and dynamic (reserves) will doubtlessly prove to be too simplistic on biochemical grounds. It is, however, a considerable improvement on the one-way classification, which is standard at present, in the field considered in this book. The consequences of a two-way classification for the interpretation of measurements and for the evaluation of population dynamical properties are complicated enough.

The reserve dynamics within the DEB model will work out such that homeostasis applies for the whole organism (including structural biomass and reserves) from birth to death, if food density does not change and reserves are at equilibrium. Realistic or not, any attempt to deviate from this property will soon break down with insurmountable problems of tying measurements of body size to the abstract variables structural biomass and reserves. This, of course, would degrade the testability of such a theory and so its usefulness.



### 2.4.1 Storage materials

Storage material can be classified into several categories: see table 2.2. These categories do not point to separate dynamics. Carbohydrates can be transformed into fats, for instance. Most compounds have a dual function as a reserve pool for both energy and elementary compounds for anabolic processes. For example, proteins stores supply energy, amino acids and nitrogen. Ribosomal RNA (rRNA) catalyzes protein synthesis. In rapidly growing cells such as those of bacteria in rich media, rRNA makes up to 80% of the dry weight, while the relative abundance in slowly growing cells is much less. For this reason, it should be included in the storage material. I will show how this point of view leads to realistic descriptions for peptide elongation rates, {250}, and growth rate related changes in the relative abundance of nitrogen, {192}. There is no requirement that storage compounds be inert.

Waxes can be transformed into fats (triglycerides) and play a role in buoyancy e.g. of zooplankton in the sea [55]. By increasing their fat/wax ratio, they can ascend to the surface layers, which offer different food types (phytoplankton), temperatures and currents. Since surface layers frequently flow in directions other than deeper ones, they can travel the earth by just changing their fat/wax ratio and stepping from one current into another. Wax ester biosynthesis may provide a mechanism for rapidly elaborating lipid stores from amino acid precursors [627].

Unsaturated lipids, which have one or more double bonds in the hydrocarbon chain, are particularly abundant in cold water species, compared with saturated lipids. This possibly represents a homeo-viscous adaptation [654].

The amount of storage materials depends on the feeding conditions in the (recent) past, cf. {72}. Storage density, i.e. the amount of storage material per unit volume of structural biomass, tends to be proportional to the volumetric length for different species, if conditions of food (substrate) abundance are compared, as explained on {218} and tested empirically on {224}. This means that the maximum storage density of bacteria is small. Under conditions of nitrogen limitation for instance, bacteria can become loaded with energy storage materials such as polyphosphate or polyhydroxybutyrate, depending on the species. This property is used in biological plastic production and phosphate removal from sewage water. Intracellular lipids can accumulate up to some 70% of the cell dry weight in oleaginous yeasts, such as *Apiotrichum* [582,785]. This property is used in the industrial production of lipids. The excess storage is due to the uncoupling of energy and mass fluxes in bacteria and these conditions have been excluded from the present analysis. Only situations of energy limitation are dealt with.

Table 2.2: Some frequently used storage materials in heterotrophs.

| | |
|---|---|
| ***phosphates*** | |
| pyrophosphate | bacteria |
| polyphosphate | bacteria (*Azotobacter*) |
| ***carbohydrates*** | |
| $\beta$-1.3-glucans | |
| leucosin | *Chrysomonadida, Prymnesiida* |
| chrysolaminarin | *Chrysomonadida* |
| paramylon | *Euglenida* |
| $\alpha$-1.4-glucans | |
| starch | *Cryptomonadida, Dinoflagellida, Volvocida* |
| glycogen | blue green bacteria, protozoa, yeasts, molluscs |
| amylopectin | *Eucoccidiida, Trichotomatida, Entodiniomorphida* |
| trehalose | fungi, yeasts |
| ***lipoids*** | |
| poly $\beta$ hydroxybutyrate | bacteria |
| triglyceride | oleaginous yeasts, most heterotrophs |
| wax | sea water animals |
| ***proteins*** | |
| ovalbumin | most heterotrophs |
| casein | egg-white protein |
| ferritin | milk protein (mammals) |
| cyanophycine | iron storage in spleen (mammals) |
| phycocyanin | blue green bacteria |
| | blue green bacteria |
| ***ribosomal*** RNA | all organisms |



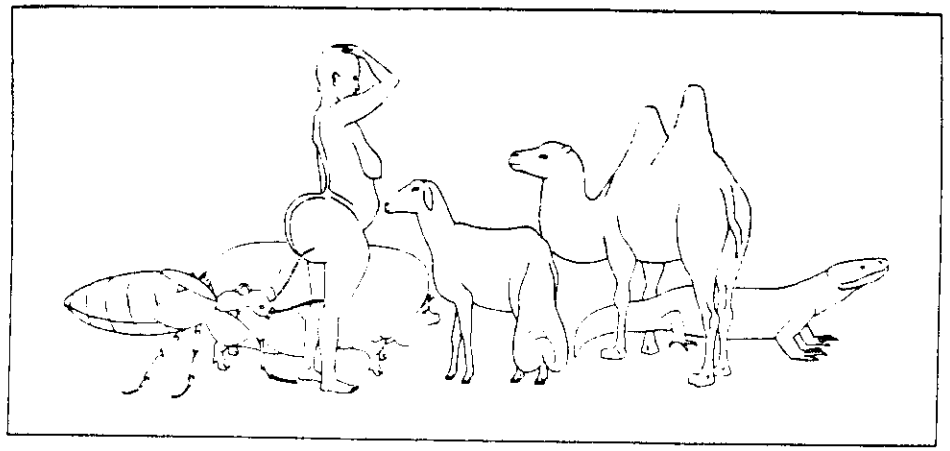


Figure 2.8: Some storage deposits are really eye-catching.

### 2.4.2 Storage deposits

Lipids, in vertebrates, are stored in cell vacuoles in specialized adipose tissue, which occurs in rather well defined surface areas of the body. The cells themselves are part of the structural biomass, but the contents of the vacuole is part of the reserves. In molluscs specialized glycogen storage cells are found in the foot [308]. The areas for storage deposits are usually found scattered over the body and therefore appear to be an integral part of the structural body mass, unless super-abundant: see figure 2.8. The occurrence of massive deposits is usually in preparation for a poor feeding season. The rodent *Glis glis* is called the 'edible doormouse', because of its excessive lipid deposits just prior to dormancy, {131}. (Stewed in honey and wine, doormice were a gourmet meal for the ancient Romans.)

In most invertebrate groups, storage deposits do not occur in specialized tissues, but only in the cells themselves at an amount that relates to requirements. So reproductive organs tend to be rich in storage products. The mesoglea of sea anemones, for instance, has mobile cells that are rich in glycogen and lipid, called 'glycocytes', which migrate to sites of demand during gametogenesis and directly transfer the stored materials to e.g. developing oocytes [654]. Glycogen that is stored for long-term typically occurs in rosettes and for short-term in particles [322,654].

## 2.5 Energy

Energy fluxes through living systems are difficult to measure and even more difficult to interpret. Let me mention briefly some of the problems.

Although it is possible to measure the thermodynamic energy content of food through complete combustion, this only shows that the organism cannot gain more energy from food, since combustion is not complete. Food is degraded to a variety of elementary

efficiency. The difference between the energy content of food and faeces is just an upper boundary for the influx, because there are energy losses in the digestion process. Part of this difference is never actually used by the organism, but is used by e.g. the gut flora. Another part becomes lost by enhanced respiration coupled to digestion, especially of proteins, called 'specific dynamic action' or 'heat increment of feeding'.

Growth involves an energy investment, which is partially preserved in new biomass. On top of the energy content of the newly formed biomass, energy has been invested to give it its structure. Part of this energy is lost during growth and can be measured as dissipating heat. This heat can be considered as an overhead of the growth process. The energy that is fixed in new biomass is partly present as energy bearing compounds. Cells are highly structured objects and the information contained in their structure is not measured by bomb calometry.

Thermodynamics of irreversible or nonequilibrium processes offers a framework to pinpoint the problem, cf. [281,421] for instance. While bomb calometry measures enthalpy, Gibbs free energy is the more useful concept to quantify the energy performance of individuals. Enthalpy and Gibbs free energy are coupled via the concept of entropy: the enthalpy of a system equals its Gibbs free energy minus the entropy times the absolute temperature. This basic relationship was formulated by Gibbs in 1878. The direct quantification of entropy requires the complete specification of the biochemical machinery, which is exactly what we try to avoid. (Dörr [177], for instance, gives an entropy reduction of 0.05 eV $\simeq$ 5 kJ mol$^{-1}$ associated with the spatial fixation of one single amino acid group of a chain molecule at 25 °C.) Gibbs relationship can be used to measure entropy indirectly in simple systems such as micro-organisms growing on well defined substrates via enthalpy and free energy. Since such free energies for micro-organisms are measured at the population level, a detailed discussion is postponed till {201}. Although this discussion opens the way to determine the entropy of living systems, I did not yet attemp to obtain numerical estimates, unfortunately. Existing ideas still range from entropy values larger than that of substrate (succinic acid) [42] to very low values [434].

All these problems about the measurement and interpretation of energy hamper direct experimental testing of assumptions about energy flows. It is possible, however, to circumvent this problem to some extent in a stunningly simple way: by not measuring energies! By refraining from direct testing of assumptions about energies, one would think that theories about energy fluxes are not testable and, therefore, useless. The consequences of such assumptions for quantities that do not represent energies, however, are testable. Many testable consequences are presented and actually tested in this book. Tests on consequences of assumptions on energy are weaker tests, which becomes apparent as soon as one or more consequences are found to be not realistic enough. It can be quite a puzzle to identify which of the assumptions about energy is the least useful one. The procedure, however, allows one to include overheads in parameter values without the obligation to take the complete machinery apart for all species.

Despite the difficulties in interpretation of energy fluxes, many attempts have been made to measure them. A relatively successful method is through the measurement of respiration. One such empirical relationship is given by Brafield and Llewellyn [85] for aquatic animals:



heat loss in J = (11.16 mg $O_2$ cons.) + (2.62 mg $CO_2$ prod.) − (9.41 mg $NH_3$ prod.)
Blaxter [73] advises replacement of the last term by −(5.93 mg N) − (3.39 mg $CH_4$) for mammals and by −(1.2 mg N) for birds. The justification of these conversions to energy rests on the idea of homeostasis. This makes the relationship between energy fluxes and gas exchange to some extent species-specific. The ratio between carbon dioxide production and oxygen consumption on a molar basis is known as the respiration quotient (RQ). Complete combustion of fat gives a respiration coefficient of 0.71, starch gives 1.0 and meat protein gives 0.82 [324]. The respiration coefficient thus gives (partial) information about the compounds that are combusted. If the composition of the combusted material remains the same, so that the respiration coefficient is constant, the oxygen consumption rate is proportional to the energy used.

Von Bertalanffy [64] related the respiration rate to the rate of anabolism. I cannot follow this reasoning. At first sight, synthesis processes are reducing by nature, which makes catabolism a better candidate for seeking a relationship with respiration. In the standard static budget studies, respiration rates are identified with routine metabolic costs. Routine metabolic costs are a lump sum including the maintenance of concentration gradients across membranes, protein turnover, regulation, transport (blood circulation, muscle tonus), and an average level of movement. The Scope For Growth (SFG) concept rests on this identification. The idea behind this concept is that energy contained in faeces and the energy equivalent of respiration are subtracted from energy derived from food, the remainder being available for growth [46]. In the DEB model, where energy derived from food is added to the reserves, the most natural candidate for a relationship with respiration is the rate at which the reserves are used.

This interpretation is also not completely free of problems, even if the respiration measurements are done on animals that are not digesting at the time. Some of the energy used from the reserves is not lost, but is fixed in the structural biovolume. This introduces some double counting. However, it seems realistic to assume that this flow is small in comparison to the overheads of the anabolic processes. This is a rather crucial point in the interpretation of respiration rates. Although respiration rates are measured over short periods (typically a couple of minutes) and the actual growth of the body is absolutely negligible, the energy invested in the growth process is by no means negligibly small. Parry [531] estimates the cost of growth between 17 and 29% of the metabolism of an 'average' ectotherm population. The respiration rate includes routine metabolic costs as well as costs for growth [619]. This interpretation is, therefore, incompatible with the SFG concept. Since the DEB model does not use respiration rates as a primary variable, the interpretation problems concerning respiration rates only play a role in testing the model.

In the next chapter, I will argue that routine metabolic costs are proportional to structural biovolume, {76}, heating costs to surface area, {78}, and growth costs to volume increase, {80}. I will show that these assumptions result in a respiration rate that is a weighted sum of surface area and volume in steady state conditions for the reserves. This is, for all practical purposes, numerically indistinguishable from the well known Kleibers rule, that takes respiration to be proportional to weight to the power 0.75 or length to the power 9/4; see figure 2.9. There are three major improvements in comparison to Kleibers rule: this model does not suffer from dimensional problems, it provides an explanation

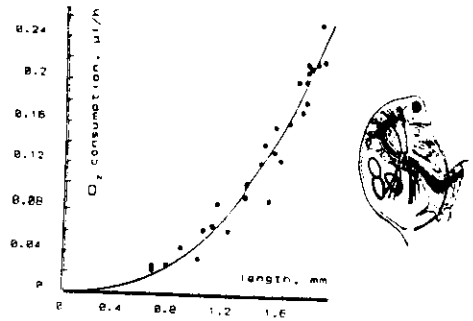



Figure 2.9: The respiration rate of *Daphnia pulex* with few eggs at 20 °C as a function of length. Data from Richman [595]. The DEB model based curve $0.0336L^2 + 0.01845L^3$ as well as the standard allometric curve $0.0516L^{2.437}$ have been plotted on top of each other, but they are so similar that this is hardly visible. Looking hard, you will notice that the line width varies a little.

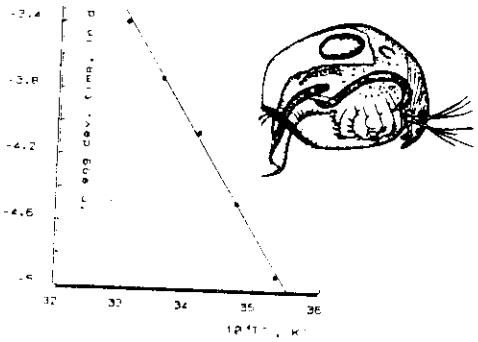


Figure 2.10: The Arrhenius plot for the development time for eggs of the waterflea *Chydorus sphaericus*, i.e. the time between egg laying and hatching. Data from Meyers [482].

rather than a description and it accommodates species that deviate from Kleibers rule, such as endotherms. This will be discussed later in somewhat more detail, {103}.

## 2.6 Temperature

All physiological rates depend on the temperature of the body. For a species-specific range of temperatures, the description proposed by Arrhenius in 1889, see e.g. [260], usually fits well:

$$\dot{k}(T) = \dot{k}(T_1) \exp\left\{\frac{T_A}{T_1} - \frac{T_A}{T}\right\} \tag{2.13}$$

where $T$ is the absolute temperature (in Kelvin), $T_1$ a chosen reference temperature, $T_A$ a parameter known as the Arrhenius temperature and $\dot{k}$ a (physiological) reaction rate. So, when $\ln \dot{k}$ is plotted against $T^{-1}$, a straight line results with slope $T_A$; see figure 2.10.

Arrhenius based this formulation on the van't Hoff equation for the temperature coefficient of the equilibrium constant and amounts to $\dot{k}(T) = \dot{k}(\infty) \exp\{\frac{-E_a}{RT}\}$, where $\dot{k}(\infty)$ is known as the frequency factor, $R$ is the gas constant 8.31441 J K$^{-1}$ mol$^{-1}$, and $E_a$ is called the activation energy. Justification rests on the collision frequency which obeys the law of mass action, i.e. it is proportional to the product of the concentrations of the reactants. The Boltzmann factor $\exp\{\frac{-E_a}{RT}\}$ stands for the fraction of molecules that



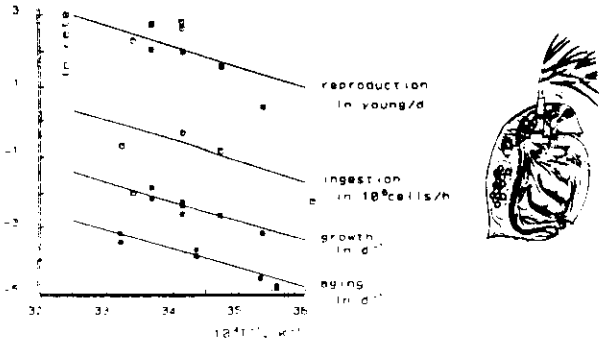



Figure 2.11: The Arrhenius plot for reproduction, ingestion, von Bertalanffy growth and Weibull aging of *Daphnia magna*; from [415]. The Arrhenius temperature is 6400 K. ◇ males, □ females. Food: the algae *Scenedesmus subspicatus* (open symbols) or *Chlorella pyrenoidosa* (filled symbols). The ingestion and reproduction rates refer to 4 mm individuals.

the critical energy $E_a$ to react.

Eyring [260] studied the thermodynamical basis of the Arrhenius relationship in more detail. He came to the conclusion that this relationship is approximate for bimolecular reactions in the gas phase. His absolute rate theory for chemical reactions proposes a more accurate description where the reaction rate is proportional to the absolute temperature times the Boltzmann factor. This description, however, is still approximate [260,320].

The step from a single reaction between two types of particles in the gas phase to physiological rates where many compounds are involved and gas kinetics do not apply is, of course, enormous. If, however, each reaction depended in a different way on temperature, cells would have a hard time coordinating the different processes when the temperature fluctuated. The Arrhenius relationship seems to describe the effect of temperature on physiological rates with acceptable accuracy in the range of relevant temperatures. Due to the somewhat nebulous application of thermodynamics to describe how physiological rates depend on temperature, I prefer to work with the Arrhenius temperature, rather than the activation energy. I even refrain from the improvement offered by Eyring's theory, because the small correction does not balance the increase in complexity of the interpretation of the parameters for biological applications.

Figure 2.11 shows that the Arrhenius temperatures for different rates in a single species are practically the same, which again points to the regulation problem an individual would experience, if they were different. Obviously, animals cannot respire more without eating more.

In chemistry, activation energy is known to differ widely between different reactions. Processes such as the incorporation of $^{14}$C-leucine into protein by membrane-bound rat-liver ribosomes have an activation energy of 180 kJ mol$^{-1}$ in the range 8-20 °C and 67 kJ mol$^{-1}$ in the range 22-37 °C. The difference is due to a phase transition of the membrane lipids, [723] after [10]. Many biochemical reactions seem to have an activation energy in this range [680]. This supports the idea that the value of activation energy is a constraint for functional enzymes in cells.

Table 2.3 gives Arrhenius temperatures for several species. The mean Arrhenius tem-

development of 35 species [790] and the von Bertalanffy growth of 250 species [410]. The value is in the upper range of values usually applied. This is due to the fact that many experiments do not allow for an adaptation period. The problem is that many enzymes are changed a little when temperature changes. This takes time, depending on species and body size. Without an adaptation period, the performance of enzymes adapted to one temperature is measured at another temperature, which usually results in a lower Arrhenius temperature being measured.

At low temperatures, the actual rate of interest is usually lower than expected on the basis of (2.13). If the organism survives, it usually remains in a kind of resting phase, until the temperature comes up again. For many sea water species, this lower boundary is between 0 and 10 °C, but for terrestrial species it can be much higher; caterpillars of the large-blue butterfly *Maculinea rebeli*, for instance, cease growth below 14 °C [200]. The lower boundary of the temperature tolerance range frequently sets boundaries for geographical distribution. Reef building corals only occur in waters where the temperature never drops below 18 °C.

At high temperatures, the organism usually dies. At 27 °C, *Daphnia magna* grows very fast, but at 29 °C, it dies almost instantaneously. The tolerance range is sharply defined at the upper boundary. Nisbet [509] gives upper temperature limits for 46 species of protozoa, ranging from 33 to 58 °C. The width of the tolerance range depends on the species; many endotherms have an extremely small one. Thermophilic bacteria and organisms living in deep ocean thermal vents thrive at temperatures of 100 °C or more.

Sharpe [644,651] proposed a quantitative formulation for the reduction of rates at low and high temperatures, on the basis of the idea that the rate is controlled by an enzyme that has an inactive configuration at low and high temperatures. The reaction to these two inactive configurations is taken to be reversible with rates depending on temperature in the same way as the reaction that is catalized by the enzyme, however the Arrhenius temperatures might differ. This means that the reaction rate has to be multiplied by the fraction of enzyme that is in its active state, which is assumed to be at its equilibrium value. This fraction turns out to be

$$\left(1+\exp\left\{\frac{T_{A,L}}{T}-\frac{T_{A,L}}{T_L}\right\}+\exp\left\{\frac{T_{A,H}}{T_H}-\frac{T_{A,H}}{T}\right\}\right)^{-1} \tag{2.14}$$

where $T_L$ and $T_H$ relate to the lower and upper boundaries of the tolerance range and $T_{A,L}$ and $T_{A,H}$ are the Arrhenius temperatures for the rate of decrease at both boundaries. All are taken to be positive and all have dimension temperature. We usually find $T_{A,H} \gg T_{A,L}$. The fraction of enzyme that is active is close to 1 between $T_L$ and $T_H$ for realistic values of these four temperatures.

Many extinctions are thought to be related to changes in temperature. This is the conclusion of an extensive study by Prothero, Berggren and others [571] on the change in fauna during the middle-late Eocene (40–41 Ma ago). This can most easily be understood if the ambient temperature makes excursions outside the tolerance range of a species. If a leading species in a food chain is a victim, many species that depend on it will follow. The wide variety of indirect effects of changes in temperature complicate a detailed analysis



Table 2.3: Arrhenius temperatures as calculated from literature data on the growth of ectothermic organisms. The values for the mouse cells are obtained from Pirt [557]. The other values were obtained using linear regressions.

| species | range (°C) | $T_A$ (K) | type of data | source |
|---|---|---|---|---|
| *Escherichia coli* | 23–37 | 6590 | pop. growth | [491] |
| *Escherichia coli* | 26–37 | 5031 | pop. growth | [350] |
| *Escherichia coli* | 12–26 | 14388 | pop. growth | [350] |
| Psychrophilic pseudomonad | 12–30 | 6339 | pop. growth | [350] |
| Psychrophilic pseudomonad | 2–12 | 11973 | pop. growth | [350] |
| *Klebsiella aerogenes* | 20–40 | 7159 | pop. growth | [722] |
| *Aspergillus nidulans* | 20–37 | 7043 | pop. growth | [724] |
| 9 species of algae | 13.5–39 | 6842 | pop. growth | [264] |
| mouse tissue cells | 31–38 | 13834 | pop. growth | [751] |
| *Nais variabilis* | 14–29 | 9380 | pop. growth | [366] |
| *Pleurobrachia pileus* | 5–20 | 10000 | Bert. growth | [275] |
| *Mya arenaria* | 7–15 | 13000 | Bert. growth | [18] |
| *Daphnia magna* | 10–26.5 | 6400 | Bert. growth | [410] |
| *Ceriodaphnia reticulata* | 20–26.5 | 6400 | Bert. growth | [410] |
| *Calliopius laeviusculus* | 6.5–15 | 11400 | Bert. growth | [151] |
| *Perna canaliculus* | 7–17 | 5530 | lin. growth | [317] |
| *Mytilus edulis* | 6.5–18 | 8460 | lin. growth larvae | [679] |
| *Cardium edule* & *C.glaucum* | 10–30 | 8400 | lin. growth larvae | [385] |
| *Scophthalmus maximum* | 8–15 | 15000 | lin. growth larvae | [360] |
| 25 species of fish | 6–29 | 11190 | embryonic period | [467] |
| *Brachionus calyciflorus* | 15–25 | 7800 | embryonic period | [282] |
| *Chydorus sphaericus* | 10–30 | 6600 | embryonic period | [482] |
| *Canthocampus staphylinus* | 3–12 | 10000 | embryonic period | [629] |
| *Moraria mrazeki* | 7–16.2 | 13000 | embryonic period | [629] |

Table 2.4: The von Bertalanffy growth rate for the waterfleas *Ceriodaphnia reticulata* and *Daphnia magna*, reared at different temperatures in the laboratory both having abundant food. The length at birth is 0.3 and 0.8 mm respectively.

| | *Ceriodaphnia reticulata* | | | | *Daphnia magna* | | | |
|---|---|---|---|---|---|---|---|---|
| temp | growth rate | s.d. | ultimate length | s.d. | growth rate | s.d. | ultimate length | s.d |
| °C | $a^{-1}$ | $a^{-1}$ | mm | mm | $a^{-1}$ | $a^{-1}$ | mm | mm |
| 10 | | | | | 15.3 | 1.4 | 4.16 | 0.16 |
| 15 | 20.4 | 4.0 | 1.14 | 0.11 | 25.9 | 1.3 | 4.27 | 0.06 |
| 20 | 49.3 | 3.3 | 1.04 | 0.09 | 38.7 | 2.2 | 4.44 | 0.09 |
| 24 | 57.3 | 2.6 | 1.06 | 0.01 | 44.5 | 1.8 | 4.51 | 0.06 |
| 26.5 | 74.1 | 4.4 | 0.95 | 0.02 | 53.3 | 2.2 | 4.29 | 0.06 |

geographic limitations for lizards set by temperature, if feeding during daytime is only possible when temperature is in the tolerance range, which leads to constraints on ectotherm energy budgets.

As a first approximation it is realistic to assume that all physiological rates are affected by temperature, so that a change in temperature amounts to a simple transformation of time. Accelerations, such as the aging acceleration that will be introduced on {107}, must thus be corrected for temperature differences by application of the squared factor, so $\dot{k}(T) = \dot{k}(T_1)\exp\{-2T_A(T_1^{-1} - T^{-1})\}$. It will be argued, {81}, that ultimate size results from a ratio of two rates, so it should not depend on the temperature if all rates are affected in the same way. Table 2.4 confirms this for two species of daphnids cultured under well standardized conditions and abundant food [410]. This is consistent with the observation by Beverton, see appendix to [126], that the walleye *Stizostedion vitreum* matures at 2 years at the southern end of its range in Texas and at 7 or 8 years in northern Canada, while the size at maturation of this fish is the same throughout its range.

Ultimate sizes are, however, frequently found to decrease with increasing temperature. The reason is usually that the feeding rate increases with temperature, so at higher temperatures, food supplies are likely to become limited, which reduces ultimate size. I will discuss this phenomenon in more detail in relation to the Bergmann rule, {132}. For a study of the effects of temperature on size, it is essential to test for the equality of food density. This requires special precautions.

A common way to correct for temperature differences in physiology is on the basis of $Q_{10}$ values, known as van't Hoff coefficients. The $Q_{10}$ is the factor that should be applied to rates for every 10 °C increase in temperature: $\dot{k}(T) = \dot{k}(T_1)Q_{10}^{(T-T_1)/10}$. The relationship with the Arrhenius temperature is thus $Q_{10} = \exp\{\frac{10T_A}{TT_1}\}$. Because the range of relevant temperatures is only from about 0 to 40 °C, the two ways to correct for temperature differences are indistinguishable for practical purposes. If the reference temperature is 20 °C, or $T_1 = 293$ K, $Q_{10}$ varies from 3.49 to 2.98 over the full temperature range for $T_A = 10000$ K.



## 2.7 Life-stages

Three life-stages are to be distinguished: embryo, juvenile and adult. The triggers for transition from one stage to another and details of the different stages will be discussed later, {97}. This section serves to introduce the stages.

The first stage is the embryonic one, which is defined as a state early in the development of the individual, when no food is ingested. The embryo relies on stored energy supplies. Freshly laid eggs consist, almost entirely, of stored energy, and for all practical purposes, the initial volume of the embryo can realistically be assumed to be negligibly small. At this stage it hardly respires, i.e. it uses no oxygen and does not produce carbon dioxide. (The shells of bird eggs initially produce a little carbon dioxide [77,294].) In many species, this is a resting stage. Although the egg exchanges gas and water with the environment, it is a rather closed system. Foetal development represents an exception, where the mother provides the embryo with reserve material, such as in the placentals and some species of velvet worm *Peripatus*. Complicated intermediates between reproduction by eggs and foetuses exist in fishes [781,782], reptiles and amphibians [71,555,684]. The evolutionary transition from egg to foetus occured many times independently. From the viewpoint of energetics, foetuses are embryos because they are not taking food. The digestive system is not functional and the embryo does not have a direct impact on food supplies in an ecological sense. The crucial difference from an energetics point of view is the supply of energy to the embryo. In lecithotrophic species, nutrients are provided by the yolk of the ovum, whereas in matrotrophic species nutrients are provided by the mother as the foetus grows, not just in vitellogenesis. The fact that eggs are kept in the body (viviparity) or deposited into the environment (oviparity) is of no importance. (The difference is of importance in a wider evolutionary setting, of course.) As in eggs, a number of species of mammal have a developmental delay just after fertilization, called diapause [656].

The second stage is the juvenile one, in which food is taken but as yet resources are not allocated to the reproductive process. In some species, the developing juvenile takes a sequence of types of food or sizes of food particles. Most herbivores, for instance, initially require protein rich diets which provide nitrogen for growth, cf. {60}. Some species, such as *Oikopleura*, seem to skip the juvenile stage. It does not feed as a larva, a condition known as lecithotrophic, and it starts allocating energy to reproduction at the same moment as feeding. The larva is a morphologically defined stage, rather than an energy defined stage. If the larva feeds, it is here treated as a juvenile, if not, it is considered to be an embryo. So, the tadpole of the mouth-breeding frog *Rheobatrachus*, which develops into a frog within the stomach of the parent, should for energy purposes be classified as an embryo, because it does not feed. Parthenogenetic aphids have a spectacular mode of reproduction: embryos producing new embryos [383] cf. {171}. Since aphids are oviviparous, females carry daughters and grand daughters at the same time. The juvenile stage is lacking and

the embryo stage overlaps with the adult one.

The word mammal refers to the fact that the young usually receive milk from the mother during the first stage after birth, called the baby stage. The length of the baby stage varies considerably. If adequate food is available, the guinea-pig *Cavia* can do without milk [656]. At weaning the young experience a dramatic change in diet and frequently the growth rate drops substantially. Few biochemical conversions are required for milk to become building blocks for new tissue. The baby, therefore, represents a transition stage between embryo and juvenile. The baby stage relates to the diet in the first instance, cf. {60}, and not directly to a stage in energetic development, such as embryo and juvenile. This can best be illustrated by the stoat *Mustela erminea*. Although blind for some 35–45 days, the female offspring reaches sexual maturity when only 42–56 days of age, before they are weaned. Copulation occurs whilst they are still in the nest [384,656].

Asexually propagating unicellulars take food from their environment, though they do not reproduce in a way comparable to the production of eggs or young by most multicellulars. For this reason, I treat them as juveniles in this energetics classification of stages. Although I realize that this does not fit into standard biological nomenclature, it is a logical consequence of the present delineations. I do not know of better terms to indicate energy defined stages, which points to the absence of literature dealing with the individual-based energetics of both micro-organisms and multicellulars. This book will show that both groups share enough features to try to place them into one theoretical framework. Some multicellulars, such as some annelids and triclads, propagate also by division. Some of them sport sexual reproduction as well, causing the distinction between both groups to become less sharp and the present approach perhaps more amenable. Some authors think that ciliates stem from multicellulars that have lost their cellular boundaries. This feature is standard in fungi and acellular slime molds. Some bacteria have multicellular tendencies [650]. So no sharp separation exists between unicellulars and multicellulars.

The eukaryotic cell cycle is usually partitioned into the interphase and the mitotic phase, which is here taken to be infinitesimally short. The interphase is further partitioned into the first gapphase, the synthesis phase (of DNA) and the second gapphase. Most cell components are made continuously through the interphase, so that this distinction is less relevant for energetics. The second gapphase is usually negligibly short in prokaryotes. Since the synthesis phase is initiated upon exceeding a certain cell size, size at division depends on growth conditions and affects the population growth rate. These phenomena will be discussed in detail.

Holo-metabolic insects are unique in having a pupal stage between the juvenile and adult one. It closely resembles the embryonic stage from an energetics point of view, cf. {151}. Pupae do not take food and start the synthesis of (adult) tissue from tiny imaginal disks. A comparable situation occurs in phyla such as echinoderms, bryozoans, sipunculans and echiurans, where the adult stage develops from a few undifferentiated cells of the morphologically totally different larva. Williamson [771] gave intriguing arguments for interpreting this transition, called cataclysmic metamorphosis, as evidence that the larval stage has been acquired later in phylogeny from, sometimes, unrelated taxa. In some cases, the larval tissues are resorbed, so converted to storage materials, in other cases the new stage develops independently. When *Luidia sarsi* steps off its bipinnaria larva as a tiny starfish, the relatively large larva swims actively for another 3 months, [702] in [771]. Some jelly fishes (Scyphomedusae) alternate between an asexual stage, small sessile polyps, and a sexual stage, large free swimming medusae. Many parasitic trematods push this alternation of generations into the extreme. From an energetic perspective, the sequence embryo, juvenile is followed by a new sequence, embryo, juvenile, adult, with different values for energy parameters for the two sequences. The coupling between parameter values is discussed on {217}.

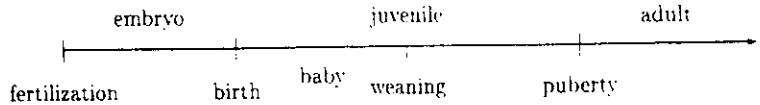


The third stage is the adult one, which allocates energy to the reproduction process. The switch from the juvenile to the adult stage, puberty, is here taken to be infinitesimally short. The actual length differs from species to species and behavioural changes are also involved. The energy flow to reproduction is continuous and usually quite slow, while reproduction itself is almost instantaneous. This can be modelled by the introduction of a buffer, which is emptied or partly emptied upon reproduction. The energy flow in females is usually larger than that in males, and differs considerably from species to species.

Most animal taxa have two sexes, male and female, but even within a set of related taxa, an amazing variety of implementations can occur. Some species of mollusc and annelid for instance, are hermaphrodite, being male and female at the same time; some species of fish and shrimp for instance, are male during one part of their life and female during another part; some have very similar sexes while other species show substantial differences between male and female: see figure 2.12. The male can be bigger than the female, as in many mammals, especially sea elephants, or the reverse can occur, as in spiders. Males of some fish, rotifers and some echiurans are very tiny, compared to the female, and parasitize in or on the female or do not feed at all. The latter group combines the embryo stage with the adult one, not unlike aphids. As will be explained in the chapter on the comparison of species on {217}, differences in ultimate size reflect differences in values for energy parameters. Parameter values, however, are tied to each other, because it is not possible to grow rapidly without eating a lot (in the long run). Differences in energy budgets between sexes are here treated in the same way as differences between species.

In some species a senile stage exists, where reproduction diminishes or even ceases. This relates to the process of aging and is discussed on {105}. An argument is presented for why this stage cannot be considered as a natural next stage within the context of DEB theory.

The summary of the nomenclature used here reads:



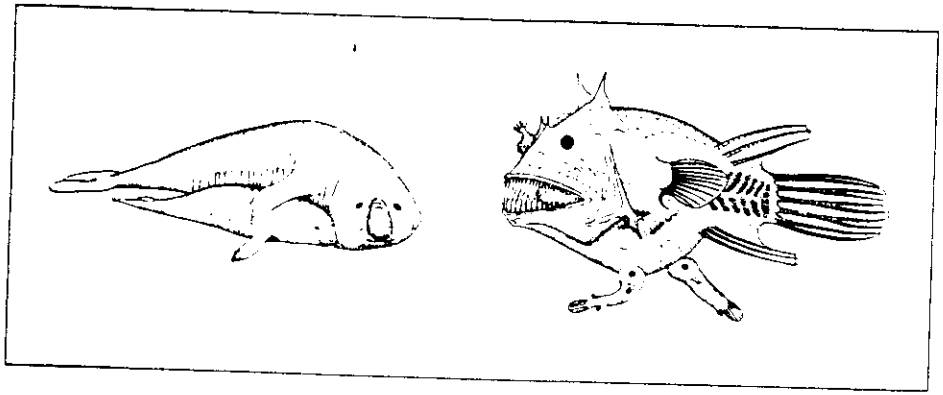


Figure 2.12: Sexual dimorphy can be extreme. The male of the southern sea elephant *Mirounga leonina* is ten times as heavy as the female, while the parasitic males of the angler fish *Haplophryne mollis* are just pustules on female's belly.

# Chapter 3

# Energy acquisition and use

This chapter discusses the mechanistic basis of different processes which together constitute the Dynamic Energy Budget (DEB) model. The next chapter will summarize and simplify the model and evaluate consequences at the individual level. Tests against experimental data are presented during the discussion to examine the realism of the model formulations, and also to develop a feeling for the numerical behaviour of the model elements. The next chapter presents additional tests that involve combinations of processes. The sequential nature of human language does not do justice to the many interrelationships of the processes. These interrelationships are what makes the DEB model more than just a collection of independent sub-models. I have chosen here to follow the fate of food, ending up with production processes and aging. This order fits 'supply' systems, but for 'demand' systems, another order may be more natural. The relationships between the different processes is schematically summarized in figure 3.1.

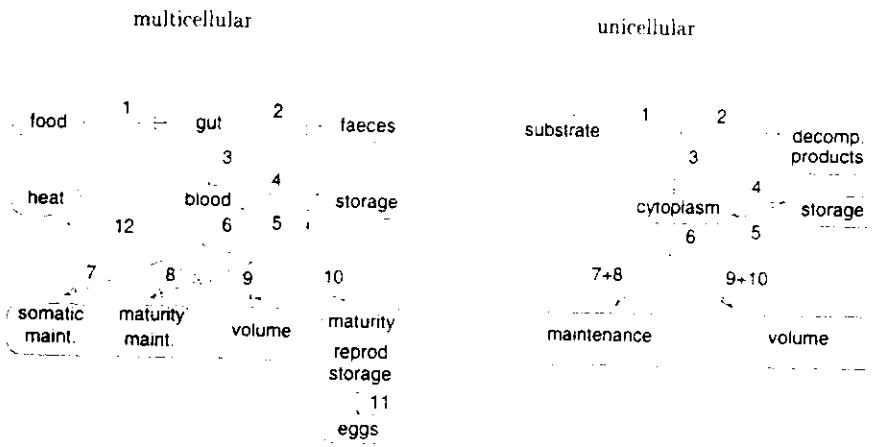
The details and logic of the energy flows will be discussed in this chapter, and a brief introduction will be given below.

Food is ingested by an animal, transformed into faeces and egested. Energy derived from food is taken up via the blood, which has a low capacity for energy but a high transportation rate. Blood exchanges energy with the storage, and delivers energy to somatic and reproductive tissues. A fixed part, $\kappa$, of the utilization rate, i.e. the energy delivered by the blood, is used for (somatic) maintenance plus growth, the rest for development and/or reproduction. The decision rule for this fork is called the $\kappa$-rule. Maintenance has priority over growth, so growth ceases if all energy available for maintenance plus growth is used for maintenance. Energy used for development in embryos and juveniles is similarly partitioned into maintenance of a certain degree of maturation and an increase in the degree of maturity. The energy spent on increasing the degree of maturity in juveniles is allocated to reproduction in adults.

Substrate is taken up and processed by unicellulars (including prokaryotes) in a way conceptually comparable to food by animals, although defecation and utilization share partly the same machinery to mobilize energy. The coupling between mass and energy fluxes, particularly relevant to micro-organisms, is discussed on [192]

Figure 3.1: Energy fluxes through a heterotroph. The rounded boxes indicate sources or sinks. Rates 3, 7, 8, 9 and 10 also contribute a bit to heating, but this is not indicated in order to simplify the scheme.

| | | | |
|---|---|---|---|
| 1 ingestion (uptake) | 2 defecation | 3 assimilation | 4 demobilization |
| 5 mobilization | 6 utilization | 7 maintenance | 8 maturation maintenance |
| 9 growth investment | 10 maturation | 11 reproduction | 12 heating (endotherms only) |




## 3.1 Feeding

Feeding is part of the behavioural repertoire and, therefore, notoriously erratic compared with other processes involved in energetics. The three main factors that determine feeding rates are body size, food availability and temperature. If different types of food are available, many factors determine preferences, e.g. relative abundances, size and searching patterns, which relate to experience and nutritional aspects. For some species it is sensible to express food availability per surface area of environment, for others food per volume makes more sense, and intermediates also exist. Body size of the organism and spatial heterogeneity of the environment hold the keys to the classification. Food availability for krill, which feed on algae, is best expressed in terms of biomass or biovolume per volume of water, because this links up with processes that determine filtering rates. The spatial scale at which algal densities differ is large with respect to the body size of the krill. Baleen whales, which feed on krill, are intermediate between surface and volume feeders because some dive below the top layer, where most algae and krill are located, and sweep the entire column to the surface; so it does not matter where the krill is in the column. Cows and lions are typically surface feeders and food availability is most appropriately expressed in terms of biomass per surface area.

These considerations refer to the relevance of the dimensions of the environment for feeding, be it surface or volume. The next section discusses the relevance of the size of the organism for feeding. The significance of food density returns in the section on functional response.



### 3.1.1 Feeding methods

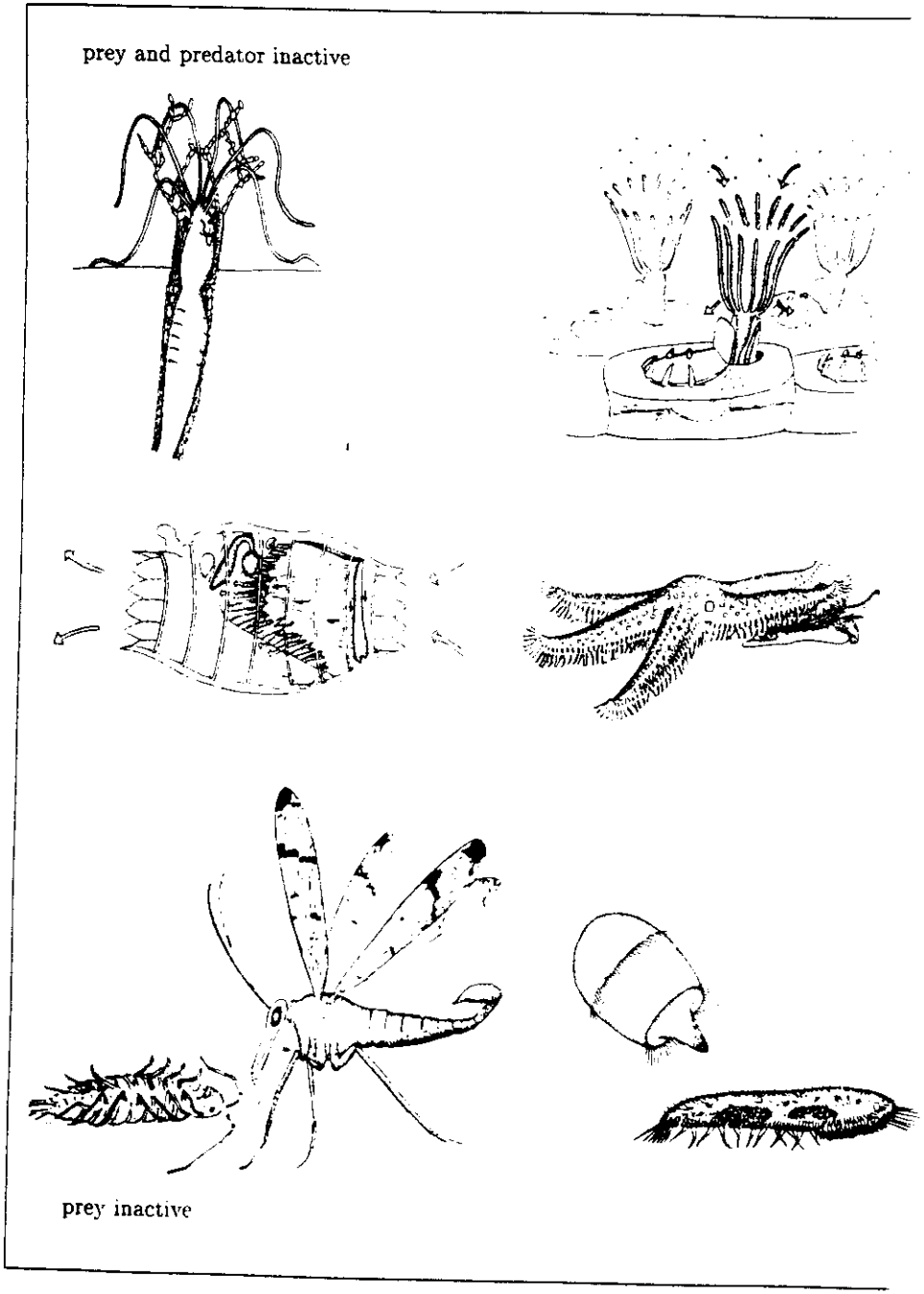
The methods organisms utilize to get their meal are numerous; some sit and wait for the food to pass by, others search actively. Figure 3.2 illustrates a small sample of methods, roughly classified with respect to active movements by prey and predator. The food items can be very small with respect to the body size of the individual and rather evenly distributed over the environment, or it can occur in a few big chunks. This section mentions briefly some feeding strategies and explains why feeding rates tend to be proportional to the surface area, when a small individual is compared to a large one of the same species. (Comparisons between species will be made in a separate chapter, {217}.)

Bacteria, floating freely in water, are transported even by the smallest current, which implies that the current relative to the cell wall is effectively nil. Thus bacteria must obtain substrates through diffusion, {141}, or attach to hard surfaces (films) or each other (flocs) to profit from convection, which can be a much faster process. Some species develop more flagellae at low substrate densities, which probably reduces diffusion limitation (Dijkhuizen, pers. com.). Uptake rate is directly proportional to surface area, when the carriers that bind substrate and transport it into the cell have a constant frequency per unit surface area of the cell membrane [5.114]. *Arthrobacter* changes from a rod shape into a small coccus at low substrate densities to improve its surface area to volume ratio. Caulobacters do the same by enhancing the development of stalks under those conditions [560].

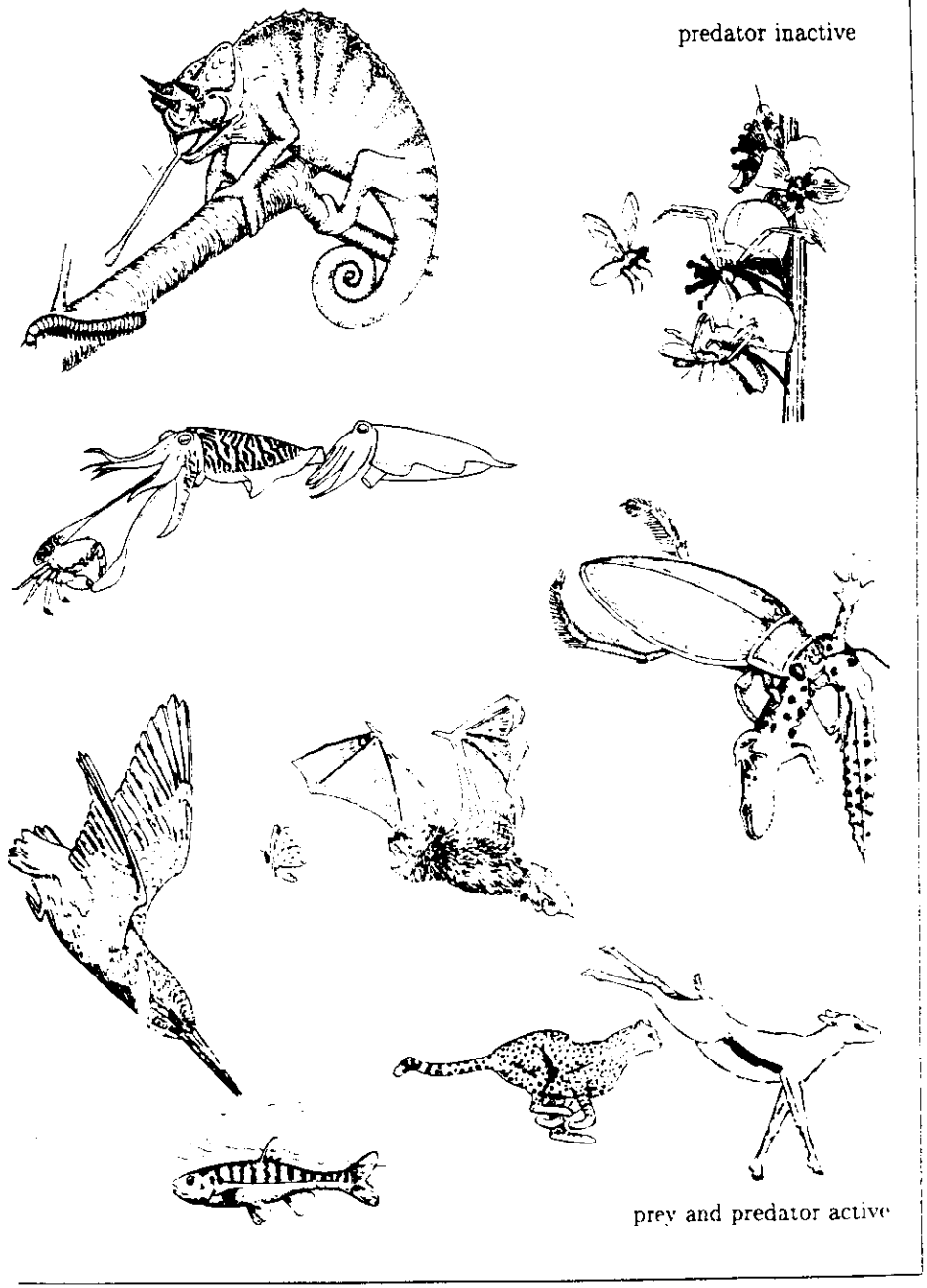
Some fungi, slime molds and bacteria glide over or through the substrate releasing enzymes and collecting elementary compounds via diffusion. Upon arrival at the cell surface, the compounds are taken up actively. The bakers' yeast *Saccharomyces cerevisiae* typically lives as a free floating, budding unicellular, but under nitrogen starvation it can switch to a filamentous multicellular phase, which can penetrate solids [329]. Many protozoans engulf particles (a process known as phagocytosis) with their outer membrane (again a surface), encapsulate them into a feeding vacuole and digest them via fusion with bodies that contain enzymes (lysosomes). Such organisms are usually also able to take up dissolved organic material, which is much easier to quantify. In giant cells, such as the Antarctic foraminiferan *Notodendrodes*, the uptake rate can be measured directly and is found to be proportional to surface area [161]. Ciliates use a specialized part of their surface for feeding, which is called the 'cytostome': isomorphic growth here makes feeding rate proportional to surface area again.

Marine polychaetes, sea anemones, sea lilies and other species that feed on blind prey are rather apathetic. Sea lilies simply orient their arms perpendicular to an existing current (if mild) at an exposed edge of a reef and take small zooplankters by grasping them one by one with many tiny feet. The arms form a rather closed fan in mild currents, so the active

Figure 3.2: A small sample of feeding methods classified with respect to the moving activities of prey and predator.


prey and predator inactive
prey inactive




predator inactive
prey and predator active

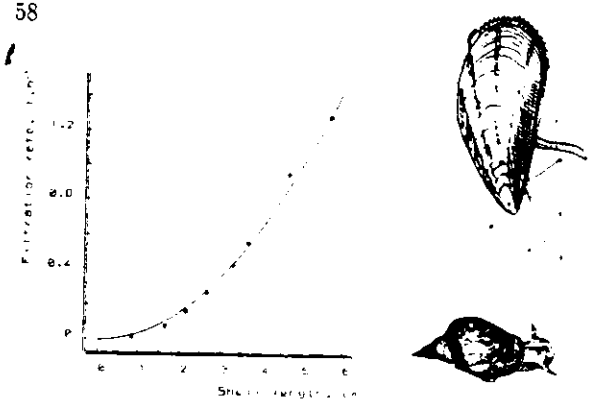


Figure 3.3: Filtration rate as function of shell length, $L$, of the blue mussel *Mytilus edulis* at constant food density ($40 \times 10^6$ cells $l^{-1}$ *Dunaliella marina*) at 12 °C. Data from Winter [774]. The least squares fitted curve is $\{\dot{F}\}L^2$, with $\{\dot{F}\} = 0.041$ (s.d. $6.75 \times 10^{-4}$) $l\,h^{-1}\,cm^{-2}$.

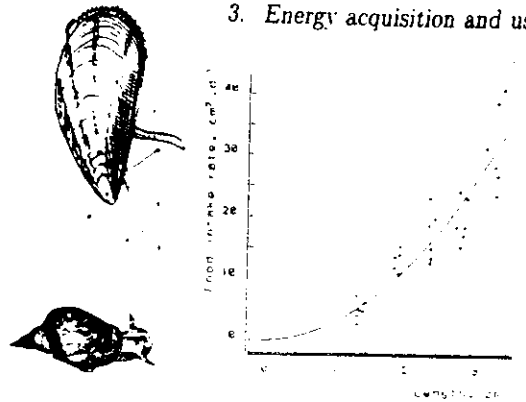


Figure 3.4: Lettuce intake as a function of shell length, $L$, in the pond snail *Lymnaea stagnalis* at 20 °C [788]. The weighted least squares fitted curve is $\{\dot{I}\}L^2$, with $\{\dot{I}\} = 2.81$ (s.d. 0.093) $cm^2\,d^{-1}\,cm^{-2}$.

area is proportional to the surface area of the animal. Sea-gooseberries stick plankters to the side branches of their two tentacles using cells which are among the most complex in the animal kingdom. Since the length of the side branches as well as the tentacles are proportional to the length of the animal, the encounter probability is proportional to a surface area.

Filter feeders, such as daphnids, copepods and larvaceans, generate water currents of a strength that is proportional to their surface area [100], because the flapping frequency of their limbs or tails is about the same for small and large individuals [565], and the current is proportional to the surface area of these extremities. (Allometric regressions of currents gave a proportionality with length to the power 1.74 [90], or 1.77 [196] in daphnids. In view of the scatter, they are in good agreement to a proportionality with squared length.) The ingestion rate is proportional the current, so to squared length. Allometric regressions of ingestion rates resulted in a proportionality with length to the power 2.2 [468], 1 [566], 2.4–3 [163], 2.4 [529] in daphnids. This wide range of values illustrates the limited degree of replicatability of these type of measurements. This is partly due to the inherent variability of the feeding process, and partly to the technical complications of measurement. Feeding rate depends on food density, as will be discussed, {63}, while most measurement methods make use of changes of food densities so that the feeding rate changes during measurement. Figure 3.11 illustrates results obtained with an advanced technique that circumvents this problem [209].

The details of the filtering process differ from group to group. Larvaceans are filterers in the strict sense, they remove the big particles first with a coarse filter and collect the small ones with a fine mesh. The collected particles are transported to the mouth in a mucous stream generated by a special organ, the endostyle. Copepods take their minute



food particles out of the water, one by one with grasping movements [732]. Daphnids exploit centrifugal force and collect them in a groove. Ciliates, bryozoans, brachiopods, bivalves and ascidians generate currents not by flapping extremities, but by beating cilia on part of their surface area. The ciliated part is a fixed portion of the total surface area [227], and this again results in a filtering rate proportional to squared length; see figure 3.3.

Some surface feeding animals, such as crab spiders, trapdoor spiders, mantis, scorpion fish and frogs, lay an ambush for their prey, who will be snatched as soon as they arrive within reach, i.e. within a distance that is proportional to the length of a leg or jaw or tongue. The catching probability is proportional to the surface area of the predatory isomorphs. When aiming at prey having rather keen eye sight, they must hide or apply camouflage.

Many animals search actively for their meal, be it plant or animal, dead or alive. The standard cruising rate of surface feeders tends to be proportional to their length, because the energy investment in movement as part of the maintenance costs tends to be proportional to volume, while the energy costs for transport are proportional to surface area; see {63}. Proportionality of cruising rate to length also occurs if limb movement frequency is more or less constant [570]. The width of the path searched for food by cows or snails is proportional to length if head movements perpendicular to the walking direction scale isomorphically. So feeding rate is again proportional to surface area, which is illustrated in figure 3.4 for the pond snail.

The duration of a dive for the sperm whale *Physeter macrocephalus*, which primarily feeds on squid, is proportional to its length, as is well known to the whalers [752]. This can be understood, since respiration rate of this endotherm is about proportional to surface area, as I will argue on {103}, and the amount of reserve oxygen proportional to volume on the basis of a homeostasis argument. It is not really obvious how this translates into feeding rate, if at all; large individuals tend to feed on large prey, which tend occur less frequently than small prey and depends on depth. Moreover, time investment in hunting can depend on size as well. If the daily swimming distance during hunting would to be independent of size, the searched water volume is about proportional to surface area for a volume feeder such as the sperm whale. If the total volume of squid per volume of water is about constant, this would imply that feeding rate is about proportional to surface area.

The amount of food parent birds feed per nestling relates to the requirements of the nestling, which is proportional to surface area; figure 3.5 illustrates this for chickadees. This is only possible if the nestlings can make their needs clear to the parents, by crying louder.

Catching devices, such as spider or pteropod webs and larvacean filter houses [13], have effective surface areas that are proportional to the surface area of the owner.

All these different feeding processes relate to surface areas in comparisons between different body sizes within a species at a constant low food density. At high food densities, the encounter probabilities are no longer rate limiting, but digestion and other food processing activities involving other surface areas, for example the mouth opening and the gut wall. The gradual switch in the leading processes becomes apparent in the functional response, i.e. the ingestion rate as function of food density, {63}.

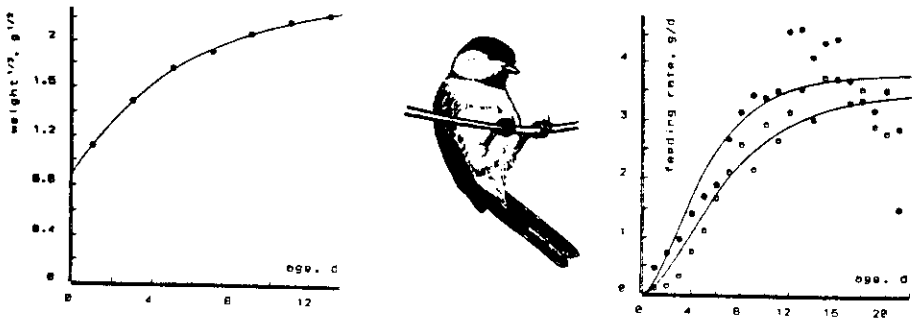



Figure 3.5: The von Bertalanffy growth curve applies to the black-capped chickadee, *Parus atricapillus* (left figure, data from Kluyver [392,671]. Brood size was a modest 5.) The amount of food fed per male (•) or female (○) nestling in the closely related mountain chickadee, *P. gambeli*, is proportional to weight$^{2/3}$ (right figure), as might be expected for individuals that grow in a von Bertalanffy way. Data from Grundel [277,671]. The last five data points were not included in the fit, because of transition to independent food gathering behaviour.

### 3.1.2 Selection

Details of growth and reproduction patterns can only be understood in relation to selection of food items and choice of diet. The reverse relationship holds as well, especially for 'demand' systems. I will, therefore, mention some aspects briefly.

Many species change their diet during development in relation to their shifting needs with an emphasis on protein synthesis during the juvenile period and on maintenance during the adult one. Mammals live on milk during the baby stage, cf. {50}. The male emperor penguin *Aptenodytes* and mouth-brooding frog *Rhinoderma darwinii* provide their young initially with a secretions from the stomach. Plant eating ducks live on insects during the first period after hatching. The first hatching tadpoles of the alpine salamander *Salamandra atra* live on their siblings inside the mother, where they are also supported by blood from her reproductive organs, and the 1-4 winners leave the mother when fully developed. The same type of prenatal cannibalism seems to occur in the coelacanth *Latimeria* [715], and several sharks (sand tiger sharks *Odontaspidae*, mackerel sharks *Lamnidae*, thresher sharks *Alopiidae* [581]). Some species of poison dart frog *Dendrobates* feed their offspring with unfertilized eggs in the water-filled leaf axils of bromeliads, high up in the trees [187,188]. Many juvenile holo-metabolic insects live on different types of food than adults. Many wasps, for instance, are carnivorous when juvenile, while they feed on nectar as adults. Prickleback fish change from being carnivorous to being herbivorous at some stage during development [167].

Some species select for different food items in different seasons apart from changes in relative abundances of the different food sources. This is because of the tight coupling between feeding and digestion. The bearded tit *Panurus biarmicus* is a spectacular example; it lives on the seed of *Typha* and *Phragmites* from September to March and on insects in summer [676,754]. This change in diet comes with an adaptation of the stomach which is much more muscular in winter [...]



verted to summer conditions, the bearded tit is unable to survive on seeds. The example is remarkable because the bearded tit stays in the same habitat over the seasons. Many temperate birds change habitats over the seasons. Divers, for instance, inhabit fresh water tundra lakes during the breeding season and the open ocean during winter. Such species also change prey, of course, but the change is usually not as drastic as the one from insects to seeds.

When offered different food items, individuals can select for type and size. Shelbourne [652] reports that the mean length of *Oikopleura* eaten by plaice larvae increased with the size of the larvae. Copepods appear to select the larger algal cells [695]. Daphnids do not collect very small particles, < 0.9 $\mu$m cross-section [266], or large ones, > 27 and > 71 $\mu$m, the latter values were measured for daphnids of length 1 and 3 mm respectively [112]. Kersting and Holterman [381] found no size-selectivity between 15-105 (and probably 165) $\mu m^3$ for daphnids. Selection is rarely found in daphnids [601], or in mussels [226,767].

The relationship between feeding rates and diet composition gives a clue as to which processes actually set the upper limits to the ingestion rate. An indication that the maximum ingestion rate is determined by the digestion rate comes from the observation that the maximum ingestion rate of copepods feeding on diatoms expressed as amount of carbon is independent of the size of the diatom cells, provided that the chemical composition of the cells is similar [235]. The maximum ingestion rate is inversely related to protein, nitrogen and carbon content fed to the copepod *Acartia tonsa* [338]. The observation that the maximum ingestion rate is independent of cell size on the basis of ingested volume [247], points to the capacity of gut volume as the limiting factor.

These remarks should make it clear that the quantitative details of the feeding process cannot be understood without some understanding of the fate of the food. This involves the digestion process in the first place, but a whole sequence of other processes follow. Regulation of (maximum) ingestion depends by definition on the need in 'demand' systems, which is especially easy to observe in species that lose the ability to grow, such as birds and mammals. Temporarily elevated food intake can be observed in birds preparing for migration or reproduction or in mammals preparing for hibernation or in pregnant mammals [731]. For simplicity's sake, these phenomena will not be modelled explicitly.

Prokaryotes show a diversity and adaptability of metabolic pathways which is huge in comparison to that of eukaryotes. Many bacteria, for example, are able to synthesize all the amino acids they require, but will only do so if they are not available from the environment. The fungus *Aspergillus niger* only feeds on cellulose if no compounds are available that are easier to decompose. Another example is growth on glucose limited media. Figure 3.6 illustrates that prolonged exposure to limiting amounts of glucose eventually results in substantially improved uptake of glucose from the environment. The difference can amount to a factor of 1000. The outer membrane is adapted to this specialized task and may jeopardize a rapid change to other substrates. This adaptation process takes many cell division cycles, as is obvious from the measurement of population growth rates, which itself takes quite a few division cycles.

The relationship between food quality and physiological performance is taken up again

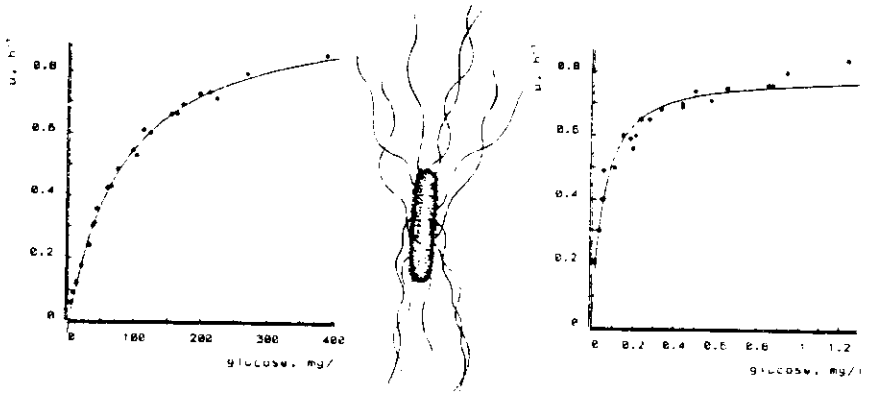


Figure 3.6: The population growth rate of *Escherichia coli* on glucose limited media. Schulze and Lipe's culture [645], left, had been exposed to glucose limitation just prior to the experiment, while that of Senn [648], right, had been pre-adapted for a period of three months.

### 3.1.3 Feeding and movement costs

As feeding methods are rather species-specific, costs for feeding will also be species-specific, if they contribute substantially to the energy budget. I will argue here that costs for feeding and movements that are part of the routine repertoire are usually insignificant with respect to the total energy budget. For this reason this subsection does not do justice to the voluminous amount of work that has been done on the energetics of movements, a field that is of considerable interest in other contexts. Alexander [9] has recently given a most readable and entertaining introduction to the subject of energetics and biomechanics of animal movement. Differences in respiration between active and non-active individuals give a measure for the energy costs of activity. The resting metabolic rate is a measure that excludes active movement. The standard or basal metabolic rate includes a low level of movement only. The field metabolic rate is the daily energy expenditure for free ranging individuals. Karasov [371] found that the field metabolic rate is about twice the standard metabolic rate for several species of mammal, and that the costs for locomotion ranges from 2–15% of the field metabolic rate. Mammals are amoung the more active species. The respiration rate associated with filtering in animals such as larvaceans and ascidians was found to be less than 2% of the total oxygen consumption [223]. The circumstance that energy investment into feeding is generally small, makes it unattractive to introduce many parameters to describe this investment. Feeding costs can be accommodated in two ways within the DEB theory without introduction of new parameters, and this subsection aims to explore to what extent this accommodation is realistic.

The first way is when the feeding costs are proportional to the feeding rate. They then show up as a reduction of the energy gain per unit of food. One can, however, argue that feeding costs per unit of food should increase with decreasing food density, because of the increased effort to extract it from the environment. This type of costs can only be accommodated without complicating the model structure if these costs cancel against an



increased digestion efficiency, due to the increased gut residence time, cf. {247}.

The second way to accommodate feeding costs without complicating the model structure is when the feeding costs are independent of the feeding rate and proportional to body volume. They then show up as part of the maintenance costs, cf. {76}. This argument can be used to understand that feeding rates for some species tend to be proportional to surface area if transportation costs are also proportional to surface area, so that cruising rate is proportional to length, {59}. In this case feeding costs can be combined with costs for other types of movement that are part of the routine repertoire. A fixed (but generally small) fraction of the maintenance costs then relates to movement.

Schmidt-Nielsen [638] calculated 0.65 ml $O_2\,cm^{-2}\,km^{-1}$ to be the surface area-specific transportation costs for swimming salmon, on the basis of Brett's work [91]. (He found that transportation costs are proportional to weight to the power 0.746, but respiration was not linear with speed. No check was made for anaerobic metabolism of the salmon. Schmidt-Nielsen obtained, for a variety of fish, a power of 0.7, but 0.67 also fits well.) Fedak and Seeherman [213] found that the surface area-specific transportation costs for walking birds, mammals and lizards tend to be about 5.39 ml $O_2\,cm^{-2}\,km^{-1} \simeq 0.03$ Wh $cm^{-2}\,km^{-1}$. (They actually report that transportation costs are proportional to weight to the power 0.72 as the best fitting allometric relationship, but the scatter is such that 0.67 fits as well.) This is consistent with data from Taylor *et al.* [703] and implies that the costs for swimming are some 12% of the costs for running. Their data also indicate that the costs for flying are between swimming and running and amount to some 1.87 ml $O_2\,cm^{-2}\,km^{-1}$.

The energy costs of swimming are frequently taken to be proportional to squared speed on sound mechanical grounds [422], which questions the usefulness of the above mentioned costs and comparisons because the costs of transportation become dependent on speed. If the inter-species relationship that speed scales with the square root of volumetric length, see {223}, also applies to inter-species comparisons, the transportation costs are proportional to volume if the travelling time is independent of size.

The energy required for walking and running is found to be proportional to velocity for a wide diversity of terrestrial animals including mammals, birds, lizards, amphibians, crustaceans and insects [244]. This is quite a relief, as otherwise temperature would be a significant variable, to mention just one problem, affecting rates in a different way and making movements a complicated variable to handle at the population and the community level; the energy costs for walking or running a certain distance are independent of speed and just proportional to distance.

### 3.1.4 Functional response

The feeding or ingestion rate, $\dot{I}$, of an organism as a function of food or substrate density, $X$, expressed as number of items per surface area or volume, is described well by the hyperbolic functional response

$$\dot{I} = f\dot{I}_m \quad \text{with } f \equiv \frac{X}{K+X} \tag{3.1}$$

where $K$ is known as the saturation coefficient or Michaelis constant, i.e. the density at which food intake is half the maximum value, and $\dot{I}_m$ the maximum ingestion rate. This

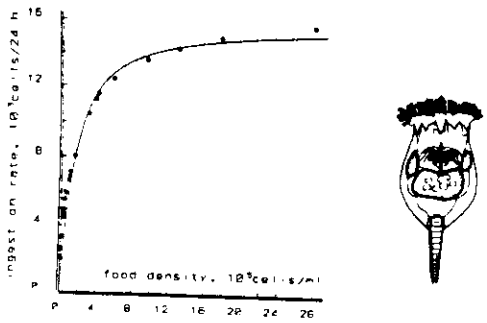



Figure 3.7: The ingestion rate, $\dot{I}$ of an individual (female) rotifer *Brachionus rubens*, feeding on the green alga *Chlorella* as a function of food density, $X$, at 20 °C. Data from Pilarska [554]. The curve is the hyperbola $\dot{I} = \dot{I}_m \frac{X}{K+X}$, with $\dot{I}_m$ = 15.97 (s.d. 0.81) $10^5$cells d$^{-1}$ and $K$ = 1.47 (s.d. 0.26) $10^5$cells d$^{-1}$. The stippled curve allows for an additive error in the measurement of the algal density of 34750 cells ml$^{-1}$.

functional response has been proposed by Holling [332] as type II, and is illustrated in figure 3.7. It applies to ciliates feeding on organic particles (phagocytosis), algae filtering daphnids, mantis catching flies, substrate uptake by bacteria, or the enzyme mediated transformation of substrates. Although these processes differ considerably in detail, some common principle gives rise to the hyperbolic function. This can be explained on the basis of a simple model for feeding, that will be generalized subsequently.

Suppose that the handling of a particle takes a certain time $\tau$ and that particles arriving during handling are ignored. ('Handling' is used here in a wide sense, for feeding animals it might refer to the act of catching and eating as well as to decomposing the particles in the gut or the transfer of products across the gut wall.) Suppose further, that particles do not interfere with each other. So the number of particles arriving in a unit of time is Poisson distributed with a parameter proportional to the particle density, $X$, say $\dot{F}X$. Here $\dot{F}$ relates to a filtering rate or the speed of an animal relative to prey particles, a rate that is taken to depend on mean particle density only, and not on particle density at a particular moment. The time between subsequent arrivals, $\underline{t}_i$, is then exponentially distributed, with mean $(\dot{F}X)^{-1}$. The time between the end of a handling period and the next arrival is again exponentially distributed with mean $(\dot{F}X)^{-1}$. (To see this, one should make use of a defining property for an exponentially distributed variable $\underline{y}$, that $\underline{y}$ and $\underline{y}|\underline{y} > y$ are identically distributed, i.e. $\varphi_{\underline{y}|\underline{y}>y}(t+y) = \varphi_{\underline{y}}(t)$.) The time required to eat $N$ particles is thus given by $\underline{t} = N\tau + \sum_{i=1}^{N} \underline{t}_i$ if one starts observations at a randomly chosen arrival of a particle. The mean ingestion rate, $\dot{I} = N/\mathcal{E}\underline{t}$, is thus $\dot{I} = (\tau + (\dot{F}X)^{-1})^{-1} = \tau^{-1}X((\tau\dot{F})^{-1} + X)^{-1}$, which is hyperbolic in the density $X$. The saturation coefficient is inverse to the product of the handling time and the filtering (or searching) rate, i.e. $K = (\tau\dot{F})^{-1}$. The maximum ingestion rate is inverse to the handling time. (The ingestion rate is here taken to be the ratio of a fixed number of particles eaten and the measured time it takes the animal to do this. If the feeding period is fixed, rather than the number of particles eaten, the mean ingestion rate might, in principle, deviate from the hyperbolic function. Moreover, we make sure that the particle density does not change during the observation period.)

This derivation can be generalized in different ways without changing the model. Each arriving particle can have an attribute that stands for the catching probability. The $i$-th



particle has some fixed probability $p_i$ of being caught upon encounting an animal, if the animal is not busy handling particles, and probability 0 if it is. It is not essential that the handling time is the same for all particles; this can be conceived as a second attribute attached to each particle, but it must be independent of food density. The condition of zero catching probability when the animal is busy can be relaxed. Metz and van Batenburg [476,477] and Heijmans [301], tied catching probability to satiation, which is thought to relate to gut content in mantis. An essential condition for hyperbolic functional responses is that catching probability equals zero if satiation (gut content) is maximal.

Another generalization is from one server, i.e. the individual handling the particles, to a large but fixed number of identical servers handling particles simultaneously, but without interfering with each other. The term 'server' stems from an extensive theory of applied probability calculus, known as queueing theory, that deals with this type of problem. See for instance [625,662]. Think of a server as an active site (enzyme molecules) in a membrane, of particles as substrate molecules and of catching as adsorption. If $\theta$ stands for the fraction of busy servers, then the change of this fraction due to arrivals is given by $\frac{d}{dt}\theta = \dot{k}_a X(1-\theta)$, where the adsorption rate constant, $\dot{k}_a$, plays exactly the same role as the filtering or searching rate $\dot{F}$. The change of the fraction of busy servers due to termination of handling is proportional to the number of busy servers, so it is given by $\frac{d}{dt}\theta = \dot{k}_d\theta$, where the desorption rate constant, $\dot{k}_d$, is just inverse to the mean handling time $\tau$. In equilibrium, the fraction $\theta$ does not change, so $\dot{k}_a X(1-\theta) = \dot{k}_d\theta$, or $\theta = X/(K+X)$, with $K = \dot{k}_d/\dot{k}_a$. We assume here that the absorption and desorption process is rapid with respect to the changes in the particle density $X$.

The fraction of occupied sites as a function of the density of adsorbable particles (i.e. partial pressure in gas), is called the adsorption isotherm in physical chemistry. If the sites operate independently, as here, and so give rise to a hyperbolic function, this isotherm is called the Langmuir isotherm [24]. The adsorption rate of particles is found easily by substituting the Langmuir isotherm into the change of the busy fraction of servers:

$$\dot{k}_a X(1-\theta) = \dot{k}_a X\left(1 - \frac{X}{K+X}\right) = \frac{\dot{k}_d X}{K+X}$$

So the adsorption rate depends hyperbolically on the particle density in equilibrium. The saturation coefficient has the interpretation of the ratio of the desorption and the adsorption rate constants and the maximum adsorption rate of particles equals the number of servers times the desorption rate. If the desorbed particles are transformed with respect to the adsorbed ones, the process stands for an enzyme mediated transformation of substrate into product. The simple kinetics discussed here are called Michaelis-Menten kinetics. The condition of constant particle density can be somewhat relaxed: if the total number of particles, $N$, is really large with respect to the number of servers (a condition formulated by Briggs and Haldane [92]), or if the rate of product formation $\dot{k}_d$ is really small (a condition formulated by Michaelis and Menten [483]), or if $K$ times the number of servers is really small with respect to $(K+N)^2$, (a more general condition formulated by Segel [647]), the reaction still follows Michaelis–Menten kinetics.

Although the details of feeding and adsorption processes differ considerably, from a more abstract point of view the mechanisms are closely related. What is essential is that

a busy period exists and that, if more servers are around, they operate identically and independently.

It is entirely possible that the hyperbolic response also arises from completely different mechanisms. A most interesting property of the hyperbolic function is that it is the only one with a finite number of parameters that maps into itself. For instance, an exponential function of an exponential function is not again an exponential function. A polynomial (of degree higher than one) of a polynomial is also a polynomial, but it is of an increasingly higher degree if the mapping is repeated over and over again. The hyperbolic function of a hyperbolic function is also a hyperbolic function. (Note that the linear response function is a special case of the hyperbolic one.) In a metabolic pathway each product serves as a substrate for the next step. Neither the cell nor the modeller needs to know the exact number of intermediate steps to relate the production rate to the original substrate density, if and only if the functional responses of the subsequent intermediate steps are of the hyperbolic type. If, during evolution, an extra step is inserted in a metabolic pathway the performance of the whole chain does not change in functional form. This is a crucial point because each pathway has to be integrated with other pathways to ensure the proper functioning of the individual as a whole. If an insert in a metabolic pathway simultaneously required a qualitative change in regulation at a higher level, the probability of its occurrence during the evolutionary process would be remote.

A most useful property of the hyperbolic functional response is that it has only two parameters which serve as simple scaling factors on the food density and ingestion rate axis. So if food density is expressed in terms of the saturation coefficient, and ingestion rate in terms of maximum ingestion rate, the functional response no longer has dimensions or parameters.

Filter feeders, such as rotifers, daphnids and mussels, reduce filtering rate with increasing food density [226,565,603,604], rather than maintaining a constant rate, which would imply the rejection of some food particles. They reduce the rate by such an amount that no rejection occurs due to handling (processing) of particles. If all incoming water is swept clear, the filtering rate is found from $\dot{F}(X) = \dot{I}/X$, which reaches a maximum if no food is around (temporarily), so that $\dot{F}_m = \{\dot{I}_m\}V^{2/3}/K$, and approaches zero for high food densities. The braces stand for 'surface area-specific', thus $\{\dot{I}_m\} \equiv \dot{I}_m V^{-2/3}$, stands for the maximum surface area-specific ingestion rate, which is considered as a parameter that depends on the composition of the diet. An alternative interpretation of the saturation coefficient in this case would be $K = \dot{I}_m/\dot{F}_m = \{\dot{I}_m\}/\{\dot{F}_m\}$, which is independent of the size of the animal, as long as only intraspecific comparisons are made. It combines the maximum capacity for food searching behaviour, only relevant at low food densities, with the maximum capacity for food processing, which is only relevant at high food densities.

Mean ingestion rate for an isomorph of volume $V$ at food density $X$ thus amounts to

$$\dot{I} = \{\dot{I}_m\} f V^{2/3} \text{ with } f \equiv \frac{X}{K + X} \tag{3.2}$$

where $\{\dot{I}_m\}$ stands for the maximum surface area-specific ingestion rate, expressed in volumetric length. When starved animals are fed, they often ingest at a higher rate for some time [753], but this is usually a fast process which will be neglected here. Starved daphnids



for instance are able to fill their guts within 7.5 minutes [247].

The ingestion rate, or substrate uptake rate for filaments and rods are found from (3.2) by multiplication of $\{\dot{I}_m\}$ with the shape correction function (2.4) or (2.7), which leads to

$$\begin{array}{rcll} \dot{I} & = & [\dot{I}_m] f V & \text{for filaments} \\ \dot{I} & = & [\dot{I}_m] f \left(\frac{s}{3} V_d + \left(1 - \frac{s}{3}\right) V\right) & \text{for rods} \end{array} \tag{3.3}$$

for $[\dot{I}_m] \equiv \{\dot{I}_m\} V_d^{-1/3}$. Since food for rods and filaments in cultures usually consists of a simple organic compound, it is standard to quantify uptake rate in gram or mole, rather than in volume as is done here. The choice of unit is free and to some extent arbitrary, the present one being motivated by the study of food chains, see {212}, where the conversion from food to structural biomass urges symmetry. Likewise, I will use the term 'substrate density', rather than 'substrate concentration' to stress the relationship with food density and to cover insoluble substrates as well.

An important source of deviations from the hyperbolic functional response will be discussed on {144}.

### 3.1.5 Food deposits and claims

Any description of the feeding process that is not species-specific can only be roughly approximative at best. In this subsection I want to point briefly to some important types of feeding behaviour that are likely to cause deviations from the hyperbolic functional response: stocking food and claiming resources via a territory. The importance of these types of behaviour is at the population level, where the effect is strongly stabilizing for two reasons. The first is that the predator lives on deposits if prey is rare, which lifts the pressure on the prey population under those conditions. The second one is that high prey densities in the good season do not directly result in an increase in predator density. This also reduces the predation pressure during the bleak seasons. Although the quantitative details will not be worked out here because of species-specificity, I want to point to this behaviour as an introduction to other smoothing phenomena that will be covered. The DEB model differs from almost all other models in dealing with such phenomena, so these remarks serve to point to the necessity of including smoothing phenomena in realistic models.

Many food deposits relate to survival during winter, frequently in combination with dormancy, cf. {131}. In the German, Dutch and Scandinavian languages, the word 'hamster' is the stem of a verb for stocking of food in preparation for adverse conditions. This rodent is famous for the huge piles of maize it stocks in autumn. The English language has selected the squirrel for this purpose. This type of behaviour is much more widespread, for example in jays; cf. figure 3.8.

Many species defend territories just prior to and during the reproductive season. Birds do it most loudly. The size of the territories depends on bird as well as food density. One of the obvious functions of this behaviour is to claim a sufficient amount of food for the peak demand when the young grow up. The behaviour of stocking and reclaiming of food typically fits 'demand' systems and is less likely to be found in 'supply' systems.

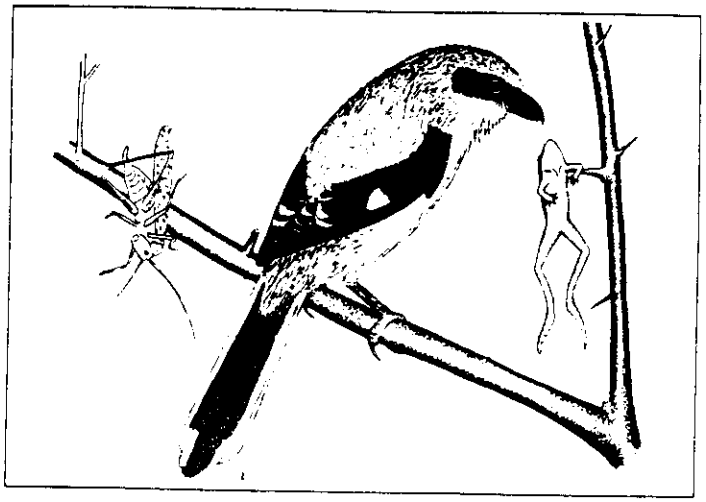


Figure 3.8: The great grey shrike *Lanius excubitor*, also known as the 'nine-killer' in Dutch, hoards throughout the year, possibly to guard against bad luck when hunting. Many other shrikes do this as well.

## 3.2 Digestion

Details of the digestion process are discussed on {247} because they do not bear directly on the specification of the DEB model. Logic of arguments requires, however, that some aspects of the digestion process should be discussed here.

### 3.2.1 Smoothing and satiation

The capacity of the stomach/gut volume is specific to a species. It depends strongly on type of food specialized on. Fish feeding on plankters, i.e. many small constantly available particles, have a low capacity, while fish such as the swallower, that feed on rare big chunks of food, have high capacities. It may wait weeks for new chunks of food; see figure 3.9. The stomach/gut volume, which is still 'environment' rather than animal, is used to smooth out fluctuations in nutritional input to the organism. Organisms attempt to run their metabolic processes under controlled and constant conditions. Food in the digestive tract and reserves inside the organism together make it possible for regulation mechanisms to ensure homeostasis. Growth, reproductive effort and the like do not depend directly on food availability but on the internal state of the organism. This even holds, to some extent, for those following the 'supply' strategy, where energy reserves are the key variable. These reserves rapidly follow the feeding conditions.

If the food in the stomach, $X_s$, follows a simple first order process, the change of stomach contents is

$$\frac{d}{dt}X_s = \{\dot{I}_m\}V^{2/3}\left(\frac{X}{K+X} - \frac{X_s}{[X_{sm}]V}\right) \tag{3.4}$$



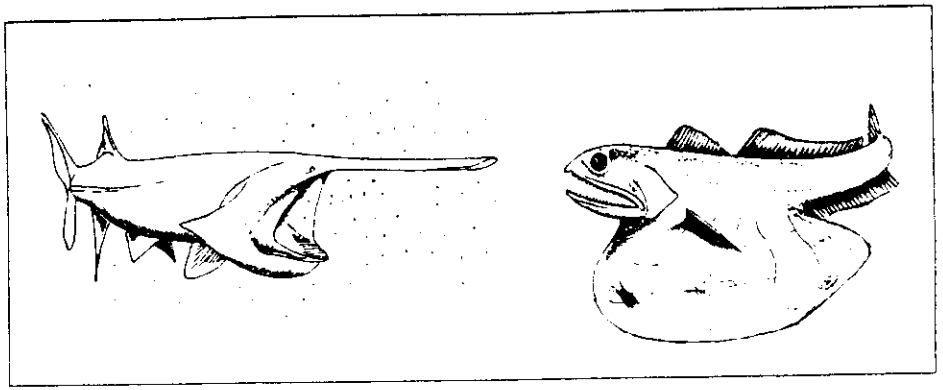


Figure 3.9: The 2 m paddlefish *Polyodon spathula* feeds on tiny plankters, while the 18 cm black swallower *Chiasmodon niger* can swallow fish bigger than itself. They illustrate extremes in buffer capacities of the stomach.

where $[X_{sm}]$ is the maximum food capacity density of the stomach. The derivation is as follows. A first order process here means that the change in stomach contents can be written as $\frac{d}{dt}X_s = \dot{I} - \dot{\alpha}X_s$, where the proportionality constant $\dot{\alpha}$ is independent of the input, given in (3.2). Since food density is the only variable in the input, $\dot{\alpha}$ must be independent of food density $X$, and thus of scaled functional response $f$. If food density is high, stomach content converges to its maximum capacity $\dot{I}_m/\dot{\alpha} = \{\dot{I}_m\}V^{2/3}\dot{\alpha}^{-1}$. The assumption of isomorphism implies that the maximum storage capacity of the stomach is proportional to the volume of the individual. This means that we can write it as $[X_{sm}]V$, where $[X_{sm}]$ is some constant, independent of food density and body volume. This allows one to express $\dot{\alpha}$ in terms of $[X_{sm}]$, which results in (3.4).

The mean residence time in the stomach is thus $t_s = V^{1/3}[X_{sm}]/\{\dot{I}_m\}$, and so it is proportional to length and independent of the ingestion rate. First order dynamics implies complete mixing of food particles in the stomach, which is unlikely if fermentation occurs. This is because the residence time of each particle is then exponentially distributed, so a fraction $1 - \exp\{-1\} = 0.63$ of the particles stays less time in the stomach than the mean residence time, and a fraction $1 - \exp\{-\frac{1}{2}\} = 0.39$ less than half the mean residence time. This means incomplete, as well as 'too complete', and thus wasteful fermentation.

The extreme opposite of complete mixing is plug flow, where the variation in residence times between the particles is nil in the ideal case. Pure plug flow is not an option for a stomach, because this excludes smoothing. These conflicting demands probably separated the tasks of smoothing for the stomach and digestion for the gut to some extent. Most vertebrates do little more than create an acid environment in the stomach to promote protein fermentation, while actual uptake is via the gut. Plug flow of food in the gut, $X_g$, can be described by

$$\frac{d}{dt}X_g(t) = t_s^{-1}(X_s(t) - X_s(t - t_g)) \tag{3.5}$$

... and $t_s$ the mean stomach residence time. This

equation follows directly from the principle of plug flow. The first term $t_s^{-1}X_s(t)$, stands for the influx from the stomach and follows from (3.4). The second one stands for the outflux, which equals the influx with a delay of $t_g$. Substitution of (3.4) and (3.2) gives $\frac{d}{dt}X_g(t) = \dot{I}(t) - \dot{I}(t - t_g) + \frac{d}{dt}X_s(t - t_g) - \frac{d}{dt}X_s(t)$. Since $0 \leq X_s \leq [X_{sm}]V$, $\frac{d}{dt}X_s \to 0$ if $[X_{sm}] \to 0$. So the dynamics of food in the gut reduces to $\frac{d}{dt}X_g(t) = \dot{I}(t) - \dot{I}(t - t_g)$ for animals without a stomach.

Some species feed in meals, rather than continuously, even if food is constantly available. They only feed when 'hungry' [178]. Stomach filling can be used to link feeding with satiation. From (3.4) it follows that the amount of food in the stomach tends to $X_s^* = f[X_{sm}]V$, if feeding is continuous and food density is constant. Suppose that feeding starts at a rate given by (3.2) as soon as food in the stomach is less than $x_{s0}X_s^*$, for some value of the dimensionless factor $x_{s0}$ between 0 and 1, and feeding ceases as soon as food in the stomach exceeds $x_{s1}X_s^*$, for some value of $x_{s1} > x_{s0}$. The mean ingestion rate is still of the type (3.2), where $\{\dot{I}_m\}$ now has the interpretation of the *mean* maximum surface area-specific ingestion rate, not the one during feeding. A consequence of this on/off switching of the feeding behaviour is that the periods of feeding and fasting are proportional to a length measure. This matter is taken up again on {121}.

### 3.2.2 Gut residence time

The volume of the digestive tract is proportional to the whole body volume in strict isomorphs. This has been found for e.g. ruminant and nonruminant mammals [162] ($\simeq 11\%$) and for daphnids [209] ($\simeq 2.5\%$ if the whole space in the carapace is included). If the animal keeps its gut filled to maximum capacity, $[X_{gm}]V$ say, and if the volume reduction due to digestion is not substantial, this gives a simple relationship between gut residence time of food particles $t_g$, ingestion rates $\dot{I}$, and body volume $V$:

$$t_g = [X_{gm}]V/\dot{I} = \frac{V^{1/3}[X_{gm}]}{f\{\dot{I}_m\}} \tag{3.6}$$

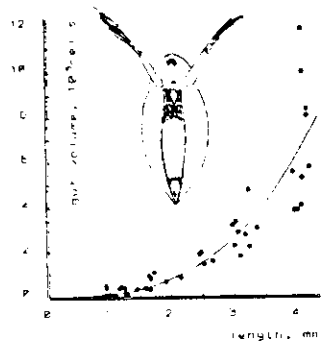
This is exactly what has been found for daphnids [209], see figure 3.10, and mussels [286]. Copepods [127] and carnivorous fish [358] seem to empty their gut at low food densities, which leads to a gut residence time of $V^{1/3}[X_{gm}]/\{\dot{I}_m\}$, if the throughput is at maximum rate.

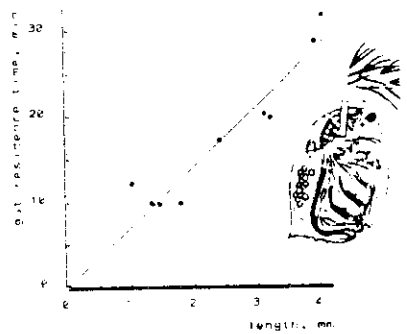
Since ingestion rate, (3.2), is proportional to squared length, the gut residence time is proportional to length for isomorphs. For filaments such as worms, which have a fixed diameter, ingestion rate is proportional to cubed length, (3.3), so gut residence time is independent of body volume.

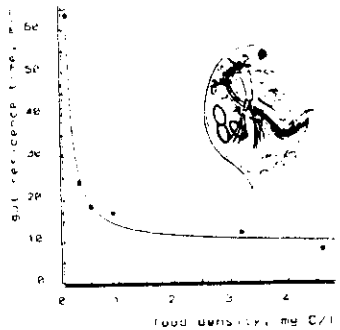
Daphnids are translucent, which offers the possibility of studying the progress of digestion as a function of body length.



Figure 3.10: Gut volume is proportional to cubed length (right) and gut residence time is proportional to length (lower left), while the latter depends hyperbolically on food density (lower right), as illustrated for daphnids. The first two figures relate to *D. magna* feeding on the green alga *Scenedesmus* at 20 °C. Data from Evers and Kooijman [209]. The third one relates to *D. pulex* of 2 mm feeding on the diatom *Nitzschia actinastroides* at 15 °C. Data from Geller [247].







The photograph of *D. magna* on the right shows the sharp transition between the chlorophyll of the green algae and the brown-black digestion products, which is typical for high ingestion rates. The relative position of this transition point depends on the ingestion rate, but not on the body length. Even in this respect daphnids are isomorphic. At low ingestion rates, the gut looks brown from mouth to anus. The paired digestive caecum is clearly visible just behind the mouth.

## 3.3 Assimilation

In animal physiology it is standard to call the enthalpy of ingested food the 'gross' energy intake [441]. It is used to quantify the energy potential for the individual. In microbial physiology and biochemistry [37], the more appropriate free energy content of consumed substrate is used for the same purpose, cf. {201}. The difference obviously relates to the poor thermodynamical definition of food of complex chemical nature. The term 'digestible' energy is used for gross energy minus energy in faeces. Then comes 'metabolisable' energy, which is taken to be digestable energy minus energy in urine and in released methane gas, followed by 'net' energy, which is metabolisable energy minus energy lost in heat increment of feeding. The term 'assimilated' energy will here be the free energy intake minus free energy in faeces and in all losses in relation to digestion. The energy in urine is treated somewhat differently and tied to the process of maintenance, cf. {77}.

The assimilation efficiency of food is here taken to be independent of the feeding rate. This makes the assimilation rate proportional to the ingestion rate, which seems to be realistic, cf. figure 7.1. I will discuss later the consistency of this simple assumption with more detailed models for enzymatic digestion, {247}. The conversion efficiency from food into assimilated energy is written as $\{\dot{A}_m\}/\{\dot{I}_m\}$, where $\{\dot{A}_m\}$ is a diet-specific parameter standing for the maximum surface area-specific assimilation rate. This notation may seem clumsy, but the advantage is that the assimilated energy that comes in at food density $X$ is now given by $\{\dot{A}_m\} f V^{2/3}$, where $f = X/(K + X)$ and $V$ the body volume. It does not involve the parameter $\{\dot{I}_m\}$ in the notation, which turns out to be useful in the discussion of processes of energy allocation in the next few sections.

The conversion from substrate to energy in bacteria is substantially more efficient under aerobic (oxygen rich) conditions than under anaerobic ones, while metabolic costs are not affected by oxygen availability [417]. This means that the parameter $\{\dot{A}_m\}$ and not $\{\dot{I}_m\}$ is of direct relevance to the internal machinery, cf. {201}.

## 3.4 Storage dynamics

Energy crossing the gut wall enters the blood or body fluid and is usually circulated through the body rapidly. It therefore does not matter where in the gut uptake takes place. Residence time in the digestive tract is usually short compared to that in the energy reserves, which means that for most practical purposes, the effect of digestion can simply be summarized as a conversion of ingested food, $\dot{I} = \{\dot{I}_m\} f V^{2/3}$, into (assimilated) energy, $\dot{A} = \{\dot{A}_m\} f V^{2/3}$. Blood has a low uptake capacity for energy (or nutrient), but a high transportation rate; it is pumped through the body many times an hour. The changes of energy in blood, $E_{bl}$, and in reserves, $E$, are coupled by $\frac{d}{dt}E_{bl} = \dot{A} - \frac{d}{dt}E - \dot{C}$ where $\dot{C}$ denotes the energy consumed by the body tissues and is called the utilization or catabolic rate. The change of energy reserves can be positive or negative. Since the energy capacity of blood is small, the change of energy in blood cannot have a significant impact on the whole body. It therefore seems safe to assume that $\frac{d}{dt}E_{bl} \simeq 0$, which means that $\frac{d}{dt}E = \dot{A} - \dot{C}$ as a first approximation.



The reserve density, $[E] \equiv E/V$, is assumed to follow simple first order dynamics

$$\frac{d}{dt}[E] = \frac{\{\dot{A}_m\}}{V^{1/3}}\left(f - \frac{[E]}{[E_m]}\right) \tag{3.7}$$

where $[E_m]$ is the maximum energy reserve density. Its derivation is completely analogous to (3.4). A first order process for the reserve density means that it can be written as $\frac{d}{dt}[E] = \dot{A}/V - \dot{\alpha}[E]$, where the proportionality constant $\dot{\alpha}$ is independent of food density $X$. At high food density, the reserve density converges to its maximum $\dot{A}_m(V\dot{\alpha})^{-1} = \{\dot{A}_m\}V^{-1/3}\dot{\alpha}^{-1}$. Because of the homeostasis assumption for energy reserves, the maximum capacity must be independent of body volume, so it can be written as a constant $[E_m]$, independent of both food density and body volume. This allows one to express $\dot{\alpha}$ in terms of the maximum capacity $[E_m]$, which gives (3.7).

An essential difference between stomach and reserves dynamics is that the first is in absolute quantities, because it relates to bulk transport, while the latter is in densities because it relates to molecular phenomena. (One cannot simply divide by body volume in (3.4) to turn to densities because body volume depends on time. One should, therefore, correct for growth to observe the mass conservation law.) Note that the requirement of homeostasis for energy density overrules the interpretation of reserve dynamics in terms of a simple mechanism where reserve 'molecules' react with the catabolic machinery at a rate given by the law of mass action. (Due to the concept of homeostasis, the density of the catabolic machinery is constant.) The organism has to adjust the reaction rate between reserves and the catabolic machinery during growth to preserve homeostasis. These adjustments are small, as long as dilution of energy density by growth is small with respect to the use of energy, i.e. if $\frac{d}{dt}\ln V \ll V^{-1/3}\{\dot{A}_m\}[E_m]^{-1}$. In practice, this condition is always fulfilled, which is not surprising because growth can only be high if the use of energy is really high. This naive picture of the mechanism can be made much more realistic without disturbing the first order kinetics.

Since inflow of energy is over a surface area and use over a volume, use of energy density is inversely proportional to length. This too corresponds closely with processes at the molecular level. Since energy reserves should not interfere with osmolarity, they are formed from insoluble polymers which are frequently further separated from the body fluid by membranes and confined to particular surface areas of the body, both macroscopically, e.g. around the gut, and microscopically. The bigger the body, the less accessible the energy reserves expressed as density. Many consequences of these extremely simple dynamics for the reserves will be tested against observations in this book. Direct testing is hampered by the problem of measuring energy fluxes inside organisms. Tests on the basis of respiration rates are probably the most direct ones feasible. Some auxiliary theory has to be developed first.

A consequence of the assumption of a first order dynamics for energy reserves is that the utilization rate must obey

$$\dot{C} = \dot{A} - \frac{d}{dt}([E]V) = \dot{A} - V\frac{d}{dt}[E] - [E]\frac{d}{dt}V = [E](\dot{v}V^{2/3} - \frac{d}{dt}V) \tag{3.8}$$

where $\dot{v} \equiv \{\dot{A}_m\}/[E_m]$; as $\dot{v}$ will show up time and again, I have given it a name, *energy conductance*, as a result of one of many discussions with Roger Nisbet. Its dimension is length per time and stands for the ratio of the maximum surface area-specific assimilation rate and the maximum volume-specific reserve energy density. The inverse, $\dot{v}^{-1}$, has the interpretation of a resistance. It is remarkable that the biological use of conductance measures seems to be restricted to plant physiology [362,511]. An important property of utilization rate is that it does not depend directly on the assimilation rate and, therefore, not on food density. It only depends on the volume of the organism and energy reserve.

The storage residence time in (3.7) is thus $V^{1/3}[E_m]/\{\dot{A}_m\}$, which must be large with respect to that of the stomach, $V^{1/3}[X_{sm}]/\{\dot{I}_m\}$ and the gut, $V^{1/3}[X_{gm}]/\{\dot{I}_m\}$, to justify neglect of the smoothing effect of the digestive tract.

If the energy reserve capacity, $[E_m]$, is extremely small, the dynamics of the reserves degenerates to $[E] = f[E_m]$, while both $[E]$ and $[E_m]$ tend to 0. The utilization rate then becomes $\dot{C} = \{\dot{A}_m\} f V^{2/3}$. This case has been studied by Metz and Diekmann [479].

## 3.5 The $\kappa$-rule for allocation

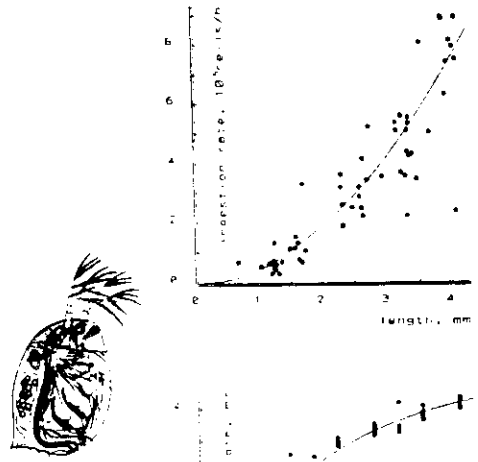
Some animals, such as birds, reproduce after having obtained their final size. Others, such as daphnids continue growth after onset of reproduction. *Daphnia magna* starts reproducing at a length of 2.5 mm, while its ultimate size is 5 to 6 mm, if well-fed. This means an increase of well over a factor 8 in volume during the reproductive period. Figure 3.11 illustrates a basic problem for energy allocation rules that such animals pose. It becomes visible as soon as one realizes that a considerable amount of energy is invested in reproductive output. The volume of young produced exceeds $\frac{1}{4}$ of that of the mother each day, or 80% of the utilization rate [595]. The problem is that growth is not retarded in animals crossing the 2.5 mm barrier; they also do not feed much more but they simply follow the surface area rule with a fixed proportionality constant at constant food densities. It seems unlikely that they digest their food much more efficiently, so where does the energy allocated to reproduction come from?

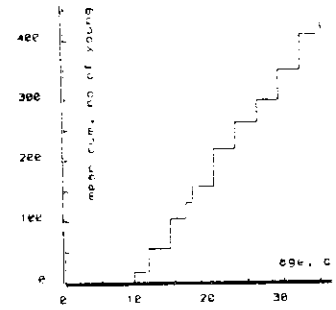
A solution to this problem can be found in development. Juvenile animals have to mature and become more complex. They have to develop new organs and install regulation systems. Increase in size (somatic growth) of the adult does not include an increase in complexity. The energy no longer spent on development in adults is spent on reproduction. Growth continues smoothly at the transition from development to reproduction. This suggests the '$\kappa$-rule' : a fixed proportion $\kappa$ of energy utilized from the reserves is spent on growth plus maintenance, the remaining portion, $1-\kappa$ on development plus reproduction. The background and rationale of the $\kappa$-rule is as follows.

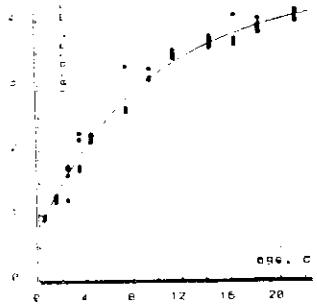
At separated sites along the path that blood follows, somatic cells and ovary cells pick up energy. The only information the cells have is the energy content of the blood and body size, cf. {23}. They do not have information about each others activities in a direct way. This also holds for the mechanism by which energy is added to or taken from energy reserves. The organism only has information on the energy density of the blood, and on size, but not on which cells removed energy from the blood. This is why the parameter



Figure 3.11: Ingestion in the waterflea *Daphnia magna* as a function of body length at 20 °C and abundant food (right), its reproduction (below) and body length (below right) as functions of age. Comparison of the quadratic feeding curve (right) and the von Bertalanffy growth curve (below right) leads to the question: where did the substantial reproductive energy come from in animals crossing the 2.5 mm barrier? The answer leads to the $\kappa$-rule. Original data and from [209].







$\kappa$ does not show up in the dynamics of energy density. The activity of all carriers which remove energy from the body fluid and transport it across the cell membrane depends, in the same way, on the energy density of the fluid. Both somatic cells and ovary cells may use the same carriers, but the concentration in their membranes may differ so that $\kappa$ may differ from the ratio of ovary and body weight. This concentration of active carriers is controlled, e.g. by hormones, and depends on age, size and environment. Once in a somatic cell, energy is first used for maintenance, the rest is used for growth. This makes maintenance and growth compete directly, while development and reproduction compete with growth plus maintenance at a higher level. The $\kappa$-rule makes growth and development parallel processes that interfere only indirectly, as has been discussed by Bernardo [60], for instance.

If conditions are poor, the system can block allocation to reproduction, while maintenance and growth continue to compete in the same way. This will be discussed further in the next chapter. I will show that Huxley's allometric model for relative growth closely links up with the $\kappa$-rule on {252}.

It is important to realize that although the fraction of utilized energy spent on maintenance plus growth remains constant, the absolute size of the flow tends to increase during development at constant food densities, as does the energy flow to maintenance plus growth.

The $\kappa$-rule solves quite a few problems from which other allocation rules suffer. Al-

though it is generally true that reproduction is maximal when growth ceases, a simple allocation shift from growth to reproduction leaves similarity of growth between different sexes unexplained, since the reproductive effort of males is usually much less than that of females. The $\kappa$-rule implies that size control is the same for males and females and for organisms such as yeasts and ciliates which do not spend energy on reproduction, but do grow in a way that is comparable to species that reproduce; see figure 1.1. A strong support for the $\kappa$-rule comes from situations where the value for $\kappa$ is changed to a new fixed value. Such a simple change affects reproduction as well as growth and so food intake in a very special way. Parasites such as the trematod *Schistosoma* in snails harvest all energy to reproduction and increase $\kappa$ to maximize the energy flow they can consume, as will be discussed on {243}. Parasite induced gigantism, coupled to a reduction of the reproductive output, is also known from trematod infested chaetognats [499], for instance. The daily light cycle also affects the value for $\kappa$ in snails; see {128}. The effect of some toxic compounds can be understood as an effect on $\kappa$, as will be discussed in the chapter on ecotoxicity, {282}. I will show how the $\kappa$-rule can be derived from a number of other assumptions that lend themselves to direct experimental testing on {119}.

## 3.6 Maintenance

Maintenance stands for a collection of processes necessary to 'stay alive'. More precisely, maintenance energy is defined as the (mean) energy requirement of an organism, excluding the production processes of growth, reproduction and development. Maintenance costs are species-specific and depend on the size of the organism and on body temperature. Maintenance processes include the maintenance of concentration gradients across membranes, the turnover of structural body proteins, a certain (mean) level of muscle tension and movement, and the (continuous) production of hairs, feathers, scales. I do not include heating of endotherms in maintenance for convenience, although it is a process necessary to stay alive and will be treated accordingly. As explained in the discussion on the $\kappa$-rule, {74}, development is excluded from maintenance, as it relates (partly) to a type of production process. The maintenance part of development is referred to as maturity maintenance, and will be discussed in the section on development, {97}. To distinguish maturation maintenance from other maintenance costs, the latter will be called costs for somatic maintenance, if necessary.

The notion of maintenance costs for advanced taxa is probably as old as man himself. Duclaux [186] was the first to recognize in 1898 that maintenance costs should be separated from production costs to understand the energetics of micro-organisms. The next reference to maintenance costs for micro-organisms stems from Sherris *et al.* [653] in 1957, in relation to motility. In the early 1960s maintenance costs for micro-organisms received considerable attention [310,380,455,466,533,556].

As is customary, I use the term 'metabolism' or 'respiration' to cover non-maintenance processes as well. The realization that respiration includes growth leads, I think, to the solution of a long standing problem: the acceptance that maintenance energy is proportional to biovolume, while metabolism or respiration is about proportional to volume to the power $\frac{3}{4}$. I will discuss this further in the section on respiration, {103}.



The idea that maintenance costs are proportional to biovolume is simple and rests on homeostasis: a metazoan of twice the volume of a conspecific has twice as many cells, which each use a fixed amount of energy for maintenance. A unicellular of twice the original volume has twice as many proteins to turn over. Bacteria, which grow in length only, have a surface area that is a linear function of cell volume. The energy spent on concentration gradients, which is coupled to membranes is, therefore, proportional to volume. Protein turnover seems to be low in prokaryotes [397]. Eukaryotic unicellular isomorphs are filled with membranes, and this ties the energy costs for concentration gradients to volume. (The argument for membrane-bound food uptake works out differently in isomorphs, because feeding involves only the outer membrane directly.) Working with mammals, Porter and Brand [567] argued that proton leak in mitochondria represents 25% of the basal respiration in isolated hepatocytes and may contribute significantly to the standard metabolic rate of the whole animal.

The energy costs for movement are also taken to be proportional to volume if averaged over a sufficiently long period. Costs for muscle tension in isomorphs are likely to be proportional to volume, because they involve a certain energy investment per unit volume of muscle. In the section on feeding, I discussed briefly the energy involved in movement, {63}, which has a standard level that includes feeding. This can safely be assumed to be a small fraction of the total maintenance costs. Sustained powered movement such as in migration requires special treatment. Such activities involve temporarily enhanced metabolism and feeding. The occasional burst of powered movement hardly contributes to the general level of maintenance energy requirements. Sustained voluntary powered movement seems to be restricted to humans and even this seems of little help in getting rid of weight!

Energy lost in excretion products is here included in maintenance costs, because the excretion of nitrogen is the most important component. This flux is tied to protein turnover, the costs of which are also included in maintenance. Products directly derived from food can also be excreted. These products are linked to the feeding process and should, therefore, show up in the value for $\{\dot{A}_m\}$. Such a partitioning of products complicates the analysis of excretion fluxes and the practical significance is limited because the energy flux involved in excretion is usually very small. Microbial product formation is discussed on {189}.

Maintenance costs are here taken to be independent of the growth rate. Tempest and Neijssel [707] argued that the concentration gradients of potassium and glutamic acid can involve a substantial energy requirement in prokaryotes. However, the concentrations of these compounds vary markedly with growth rate so that this energy drain is not taken to be part of maintenance here, but as part of the overhead costs of the growth process. The high costs for potassium gradients is at odds with Ling's association-induction hypothesis [434], which states a.o. that virtually all $K^+$ in living cells exists in an absorbed state. The mechanism is via a liquid crystal type of structure for the cytoplasm [102].

Some species have specific maintenance costs, such as daphnids which produce moults every other day at 20 °C. The synthesis of new moults occurs in the intermoult period and is a continuous and slow process. The moults tend to be thicker in the larger sizes. The exact costs are difficult to pin down, because some of the weight refers to inorganic

compounds, which might be free of energy cost. Larvaceans produce new feeding houses every 2 hours at 23 °C [214], and this contributes substantially to organic matter fluxes in oceans [11,12,157]. These costs are taken to be proportional to volume. The inclusion of costs for moults and houses in maintenance costs is motivated by the observation that these rates do not depend on feeding rate [214,407], but only on temperature. Euryhaline fishes have to invest energy for osmoregulation in waters that are not iso-osmotic. The cichlid *Oreochromis niloticus* is iso-osmotic at 11.6 ppt and 29% of the respiration rate at 30 ppt can be linked to osmoregulation [780]. Similar results have been obtained for brook trout *Salvelinus fontinalis* [237].

The maintenance costs $\dot{M}$, are thus taken to be proportional to volume

$$\dot{M} = [\dot{M}]V \tag{3.9}$$

and the volume-specific costs for maintenance, $[\dot{M}]$, can be partitioned into a variety of processes that together are responsible for these costs.

As stated on {39}, no maintenance costs are paid over reserves. The empirical justification can most easily be illustrated by the absence of respiration in freshly laid eggs, which consist almost entirely of reserves; see figure 3.14. Note, however, that costs for turnover of reserves are covered by overheads in assimilation and utilization. Although the difference between turnover costs for reserves and structural biomass is subtle, eggs show that the turnover costs for reserves are not equivalent with maintenance for reserves, since they do not respire when freshly laid.

## 3.7 Homeothermy

Heat comes free as a side product of all uses of energy, cf. {201}. In ectotherms, this heat simply dissipates without increasing the body temperature above that of the environment to any noticeable amount as long as the temperature is sufficiently low. If the environmental temperature is high, as in incubated bird eggs just prior to hatching, metabolic rates are high as well, releasing a lot more free energy in the form of heat and increasing the body temperature even further, cf. {135}. This is called a positive feedback in cybernetics. The rate of heat dissipation obviously depends on the degree of insulation and is directly related to surface area. A small number of species, known as endotherms, use energy for the purpose of keeping their body temperature at a predetermined high level, 34 °C in monothremes, 37 °C in most mammals, 39 °C in non-passerine birds, 41 °C in passerine birds. Mammals and birds change from ectotherms to endotherms during the first few days of their juvenile stage. Some species temporarily return to the ectothermic state or partly so in the night (kolibries) or during hibernation (rodents, bats) or torpor (tenrecs, cf. {131}). Not all parts of the body are kept at the target temperature, especially not the extremities. The naked mole rat *Heterocephalus glaber* (see figure 3.12) has a body temperature that is almost equal to that of the environment [441] and actually behaves as an ectotherm. Huddling in the nest of this colonial species plays an important role in thermoregulation [778]. Many ectotherms can approach the state of homeothermy under favourable conditions by walking from shady to sunny places, and back, in an appropriate



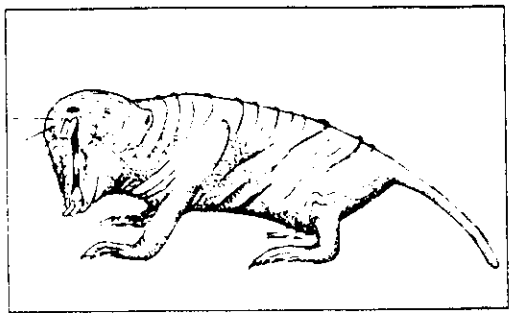


Figure 3.12: The naked mole rat *Heterocephalus glaber* (30 gram) is one of the few mammals that are essentially ectothermic. They live underground in colonies of some 60 individuals. The single breeding female suppresses reproductive development of all 'frequent working' females and of most 'infrequent working' females, a social system that reminds us of termites [442].

way. In an extensive study of 82 species of desert lizards from three continents, Pianka [551] found that body temperature $T_b$ relates to ambient air temperature $T_e$ as

$$T_b = 311.8 + (1 - \beta)(T_e - 311.8)$$

where $\beta$ stands for the species-specific thermoregulatory capacity, spanning the full range from perfect regulation, $\beta = 1$ for active diurnal heliothermic species, to no regulation, $\beta = 0$ for nocturnal thigmothermic species. The target temperature of 311.8 K or 38.8 °C varied somewhat between the different sub-groups and is remarkably close to that of mammals. Other species can raise their temperature over 10 °C above that of the environment (bumble bees, moths, tuna fish, mackerel shark). These examples do make clear that energy investment into heating is species-specific and that the regulation of body temperature is a different problem.

The 'advantages' of homeothermy are that enzymes can be used that have a narrow tolerance range for temperatures and that activity can be maintained at a high level independent of environmental temperature. At low temperatures ectotherms are easy prey for endotherms. Development and reproduction are enhanced, which opens niches in areas with short growing seasons that are closed to ectotherms. The costs depend on the environmental temperature, insulation and body size. If temperature is high and/or insulation is excellent and/or body size is large, there may be hardly any additional costs for heating; the range of temperatures for which this applies is called the thermo-neutral zone. The costs for heating, $\dot{H}$, due to losses by convection or conduction can be written as

$$\dot{H} = \{\dot{H}\}V^{2/3} \tag{3.10}$$

Heat loss is not only proportional to surface area but, according to Newton, also to the temperature difference between body and environment. This is incorporated in the concept of thermal conductance $\{\dot{H}\}/(T_e - T_b)$, where $T_e$ and $T_b$ denote the temperature of the environment and the body. It is about 5.43 $J\,cm^{-2}\,h^{-1}\,°C^{-1}$ in birds and 7.4-9.86 $J\,cm^{-2}\,h^{-1}\,°C^{-1}$ in mammals, as calculated from [311]. The unit $cm^{-2}$ refers to volumetric squared length, not to real surface areas which involve shape. The values represent crude means in still air. The thermal conductance is roughly proportional to the square root of wind speed

This is a simplified presentation. Birds and mammals moult at least twice a year, to replace their hair and feathers which suffer from wear, and change the thick winter coat for the thin summer one. Cat owners can easily observe that when their pet is sitting in the warm sun, it will pull its hair into tufts, especially behind the ears, to facilitate heat loss. Many species have control over blood flow through extremities to regulate temperature. People living in temperate regions are familiar with the change in the shape of birds in winter to almost perfect spheres. This increases insulation and generates heat from the associated tension of the feather muscles. These phenomena point to the variability of thermal conductance.

There are also other sources of heat exchange, through ingoing and outgoing radiation and cooling through evaporation. Radiation can be modulated by changes in colour, which chameleons and tree frogs apply to regulate body temperature [441]. Evaporation obviously depends on humidity and temperature. For animals that do not sweat, evaporation is tied to respiration and occurs via the lungs. Most non-sweaters pant when hot and lose heat by enhanced evaporation from the mouth cavity. A detailed discussion of heat balances would involve a considerable number of coefficients [492,677], and would obscure the main line of reasoning. I will, therefore, refrain from giving these details. It is important to realize that all these processes are proportional to surface area, and so affect the heating rate $\{\dot{H}\}$ and in particular its relationship with the temperature difference between body and environment.

## 3.8 Growth

Growth can now be derived on the basis of (3.8), (3.9), (3.10) and the $\kappa$-rule; see {74}. The $\kappa$-rule states that

$$\kappa \dot{C} = [G]\frac{d}{dt}V + \dot{M} + \dot{H} \tag{3.11}$$

where $[G]$ denotes the volume-specific costs for growth, which are taken as fixed in view of homeostasis of the structural biomass. These costs thus include all types of overhead costs, not just the costs for synthesis. There are no costs for heating for ectotherms, so $\dot{H} = 0$ for them. Substitution of (3.8), (3.9) and (3.10) gives

$$\frac{d}{dt}V = \dot{r}\frac{V^{2/3}[E]/[E_m] - V^{2/3}(V_h/V_m)^{1/3} - V/V_m^{1/3}}{[E]/[E_m] + g} \tag{3.12}$$

Note that growth does not depend on food density directly. It only depends on reserve density and body volume. The energy parameters combine in the compound parameters $V_h$, $V_m$, $g$ and $\dot{r}$. The compound parameters will appear frequently in the sequel, so they are best introduced here. To aid memory, it is useful to give them names.

The *maintenance rate constant* $\dot{m} \equiv [\dot{M}]/[G]$ was introduced by Marr [455] and publicized by Pirt [556], and stands for the ratio of costs for maintenance and biovolume synthesis. It has dimension time$^{-1}$. It remains hidden here in the maximum volume $V_m$, but it will frequently play an independent role.



The quantity $g \equiv [G]/\kappa[E_m]$ is called the (energy) *investment ratio* and stands for the costs for new biovolume relative to the maximum potentially available energy for growth plus maintenance. It is dimensionless.

$V_m \equiv (\frac{\kappa}{g\dot{m}})^3 = (\kappa\{\dot{A}_m\}/[\dot{M}])^3$ stands for the *maximum volume* ectotherms can reach. (Endotherms cannot reach this volume because they loose energy through heating.) The comparison of species is based on this relationship between maximum volume and energy budget parameters and is the core of the relationship between body size and physiological variables together with the invariance property of the DEB model, to be discussed later, {217}.

The *heating volume* $V_h \equiv (\{\dot{H}\}/[\dot{M}])^3$ stands for the reduction in volume endotherms experience due to the energy costs for heating. It can be treated as a simple parameter as long as the environmental temperature remains constant. If the temperature changes slowly relative to the growth rate, the heating volume just a function of time. If environmental temperature changes rapidly, body temperature can be taken to be constant again while the effect contributes to the stochastic nature of the growth process, cf. {121}. Note that (3.12) shows that the existence of a heating volume is not an extra assumption, but a consequence of the volume-bound maintenance costs and the surface area-bound input and heating costs.

If food density $X$ and, therefore, the scaled functional response $f$ are constant, and if the initial energy density equals $[E] = f[E_m]$, energy density will not change. Volumetric length as a function of time since hatching where $V(0) \equiv V_b$, can then be solved from (3.12)

$$\frac{d}{dt}V^{1/3} = \frac{\dot{r}}{3(f+g)}\left(f - (V_h/V_m)^{1/3} - (V/V_m)^{1/3}\right) \tag{3.13}$$

$$V^{1/3}(t) = V_\infty^{1/3} - (V_\infty^{1/3} - V_b^{1/3})\exp\{-t\dot{\gamma}\} \quad \text{or} \tag{3.14}$$

$$t(V) = \frac{1}{\dot{\gamma}}\ln\frac{V_\infty^{1/3} - V_b^{1/3}}{V_\infty^{1/3} - V^{1/3}} \tag{3.15}$$

I will follow tradition and call this curve the von Bertalanffy growth curve despite its earlier origin and von Bertalanffy's contribution of introducing allometry, which I reject; see {12}. The von Bertalanffy growth rate equals

$$\dot{\gamma} \equiv (3/\dot{m} + 3fV_m^{1/3}/\dot{r})^{-1} \tag{3.16}$$

and the ultimate volumetric length

$$V_\infty^{1/3} \equiv fV_m^{1/3} - V_h^{1/3} \tag{3.17}$$

Time $t$ in (3.14) is measured from hatching or birth. (Note that time and age are not the same.) The von Bertalanffy growth curve results for isomorphs at constant food density and temperature and has been fitted successfully to the data of some 270 species from many different phyla; see table 6.2 and [410]. The gain in insight since Pütter's original formulation in 1920 is in the interpretation of the parameters in terms of underlying processes. It appears that heating costs do not affect the von Bertalanffy growth rate $\dot{\gamma}$.

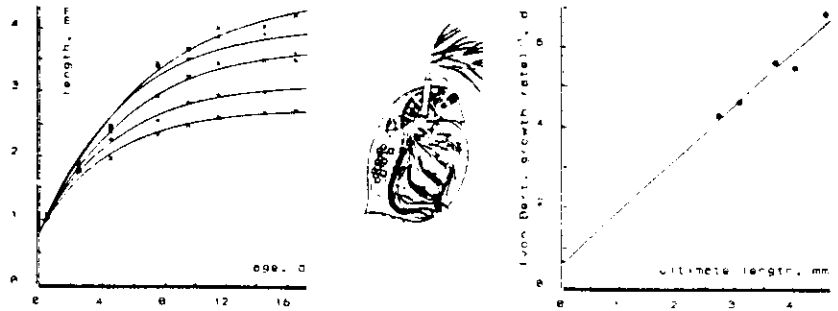


Figure 3.13: The left figure shows the length-at-age data of the waterflea *Daphnia magna* for various densities of the green alga *Chlorella* at 20 °C with von Bertalanffy growth curves. Data from [407]. The inverses of the estimated von Bertalanffy growth rates have been plotted against estimated ultimate lengths (right). The expected relationship is $\dot{r}^{-1} = 3/\dot{m} + 3\dot{d}_m L_\infty/\dot{v}$. The least squares fitted line gives estimates for $\dot{v}/\dot{d}_m$ of 2.29 mm d$^{-1}$ and for $\dot{m}$ of 4.78 d$^{-1}$, both of which seem to be too high in comparison with other species. Frequent moulting may contribute to the maintenance costs and so to the high estimate for the maintenance rate coefficient $\dot{m}$.

Being a rate, high temperature does elevate it, of course. Food density affects both the von Bertalanffy growth rate and the ultimate volume. The inverse of the von Bertalanffy growth rate is a linear function of the ultimate volumetric length: see figure 3.13. This is consistent with the Pütter's original formulation, which took this rate to be inversely proportional to ultimate length, as has been proposed again by Gallucci and Quinn [242].

The requirement that food density is constant for a von Bertalanffy curve can be relaxed if food is abundant. This is due to the hyperbolic functional response. As long as food density is higher than 4 times the saturation coefficient, food intake is higher than 80% of the maximum possible food intake, which makes it hardly distinguishable from maximum food intake. Since most birds and mammals have a number of behavioural traits aimed at guaranteed adequate food availability, they appear to have a fixed volume-age relationship. This explains the popularity of age-based models for growth in 'demand' systems. In the next chapter I will discuss deviations from the von Bertalanffy growth curve that can be understood in the context of the present theory.

In contrast, at low food densities, fluctuations in food density soon induce deviations from the von Bertalanffy curve. This phenomenon will be discussed further in the section on genetics and parameter variation, {112}.

Maximum volumetric length is reached at prolonged exposure to high food densities, where $f = 1$, which gives $V_\infty^{1/3} = V_m^{1/3} - V_h^{1/3}$. If the juvenile period ends upon exceeding volume $V_p$, the length of this period is $t(V_p)$ at constant food density, as given in (3.15).

In the discussion on population dynamics, it will become important to distinguish time, $t$, from age, $a$. The age at the end of the juvenile period, so at puberty, is thus $a_p = a_b + t(V_p)$, if $a_b$ stands for the age at birth and fertilization initializes age.

Growth ceases, i.e. $\frac{d}{dt}V = 0$, if $[E] = (\{\dot{H}\} + [\dot{M}]V^{1/3})/\kappa\dot{v}$. If the energy density drops even further, some organisms, such as protozoa and coelenterates, shrink. Even animals



with a skeleton, such as shrews of the genus *Sorex*, can exhibit a geographically varying winter size depression, known as the Dehnel phenomenon [250]. Molluscs seem be to able to reduce shell size [179]. Some animals only deviate from the $\kappa$-rule in situations of prolonged starvation, that is, they still follow first order dynamics for the use of energy reserves, pay maintenance (and heating) costs, the rest being spent on development and/or reproduction, whereas others deviate from first order dynamics for the utilization of energy, (3.7). These species only pay maintenance (and heating) costs, so

$$\frac{d}{dt}[E] = f\{\dot{A}_m\}V^{-1/3} - [\dot{M}] - \{\dot{H}\}V^{-1/3} \tag{3.18}$$

where $V$ remains fixed. At constant food density, thus constant energy uptake rate, this dynamics implies that energy density either increases to the no-growth boundary or decreases to zero. Pond snails are a beautiful example of a species that follows both strategies for energy expenditure, depending on day length. When in a long day/short night cycle, they reproduce continuously, but they cease to do so in a short day/long night rhythm. This will be discussed further in the next chapter, {128}. Although food availability does not influence growth directly, it does so indirectly via reserve energy. Moreover, the maximum surface area-specific assimilation rate $\{\dot{A}_m\}$, and so energy conductance $\dot{v}$, relate to the food-energy conversion. Many herbivores, such as chickens, eat animal products in the early juvenile period to gain nitrogen, which they need for the synthesis of proteins. They experience a shift in diet during development. Mammals feed milk to their offspring, this needs little conversion and induces growth rates that cannot be reached with their later diet. Growth curves show a sharp kink at weaning.

Animals that have non-permanent exoskeletons (arthropods, insects) have to moult to grow. The rapid increase in size during the brief period between two moults, relates to uptake of water or air, not to synthesis of new structural biomass, which is a slow process occurring during the intermoult period. This minor deviation from the DEB model (see figure 3.11 and [409]) relates more to size measures than to model structure.

### 3.8.1 Embryonic growth

The DEB model takes the bold view that the only essential difference between embryos and juveniles is that the former do not feed. Although information on parameter values is still sparse, it indicates that no (drastic) changes of values occur at the transition from the embryonic to the juvenile state. I will first discuss eggs, which do not take up energy from the environment. (See [113] for an excellent introduction to eggs, with beautiful photographs.) Subsequently, I will deal with foetuses, which obtain energy reserves from the mother during development.

The idea is that the dynamics for growth, (3.12), and reserve density, (3.7), also apply to embryos in eggs in absence of food intake. The scaled functional response is thus taken to be $f = 0$. The dynamics for the reserve density then reduces to

$$\frac{d}{dt}[E] = -\dot{v}[E]V^{-1/3} \tag{3.19}$$

initial volume is practically nil, so $V(0) = 0$. This makes the energy density infinitely e, so $[E](0) = \infty$. The (absolute) initial energy is a certain amount, $[E](0)V(0) = E_0$, h, however, is not considered to be a free parameter. Its value is determined from the lition of the energy reserves at hatching. Hatching occurs at age $a_b$, say, and initial gy density $[E_b]$, so $[E](a_b) = [E_b]$. The just-born juvenile still needs some energy rves to cope with its metabolic needs. If all utilized energy is used for maintenance atching, a lower boundary for reserve energy density follows from $[\dot{M}]V_b = \dot{v}[E_b]V_b^{2/3}$, ıg $[E_b] = [\dot{M}]V_b^{1/3}/\dot{v}$.

f food density is constant, the energy density will change from the one at hatching, ed $[E_b]$, to $f[E_m]$ in juveniles. If energy density at hatching is about equal to $f[E_m]$, growth curve will follow a von Bertalanffy curve. For initial energy densities less ı $f[E_m]$, growth will be retarded compared to the von Bertalanffy growth curve; the osite holds for initial densities larger than $f[E_m]$. Although the deviation from the von talanffy growth curve will not last long, because the relaxation time for energy density :oportional to length, which is small at hatching, it is tempting to take the initial energy sity to be equal to that of the mother at egg laying. This results in von Bertalanffy wth at constant food density even just after hatching, and it does not require additional ameters.

Tests on the realism of the initial condition that $[E_b]$ equals $[E]$ of the mother at laying are conflicting for daphnids. The triglycerides component of energy density is ble as a yellow colour and as droplets. I have observed that well-fed, yellow mothers *Daphnia magna* give birth to yellow offspring, and poorly fed, glassy mothers give h to glassy offspring. This is consistent with observations of Tessier *et al.* [710]. er observations by Tessier as well as by Lisette Enserink, however, indicate an inverse ıtionship between food density and energy reserves at hatching. An increase of energy estment per offspring can also result in larger offspring rather than an increased reserve ısity at hatching. Large bodied offspring at low food availability has been described for terrestrial isopod *Armadillium vulgare* [96]. Because of the relationship with energy its for egg production, and so with reproduction rate, this response to resource depletion s implications for population dynamics. It can be viewed as a mechanism that aims to sure adequate food supply for the existing individuals. The condition that energy density hatching equals that of the mother at egg formation is made here for reasons of simplicity d theoretical elegance. No theoretical barriers exist for other formulations within the ntext of the DEB theory. Such formulations are likely to involve species-specific empirical optimization arguments, however, which I have tried to avoid as much as possible.

Embryo development provides excellent opportunities to test the model for the dy- mics of energy reserves, because of the huge change of energy density, which avoids the thological conditions starving individuals face. As embryos do not feed, data on their velopment do not suffer from a major source of scatter.

The goodness of fit is remarkable, as illustrated in figure 3.14, where data on weight, lk and respiration have been fitted simultaneously by Cor Zonneveld [790]. The total mber of parameters is 5 excluding, or 7 including, respiration. As will be discussed later,





this way. The examples are representative of the data collected in table 3.1, which gives parameter estimates of some 40 species of snails, fish, amphibians, reptiles and birds. The model tends to underestimate embryo weight and respiration rate in the early phases of development. This is partly due to deviations in isomorphism, the contributions of extra-embryonic membranes (both in weight and in the mobilization of energy reserves), and the loss of water content during development. The estimates for the altricial birds such as the parrot *Agapornis* should be treated with some reservations, because neglected acceleration due to temperature increase during development substantially affects the estimates, as discussed on {135}.

The values for the energy conductance $\dot{v}$, as given in table 3.1, are in accordance with the average value for post-embryonic development, as given on {224}, which indicates that no major changes in energy parameters occur at birth. The maintenance rate constant $\dot{m}$ for reptiles and birds is about 0.08 $d^{-1}$ at 30 °C, implying that the energy required to maintain tissue during 12 days at 30 °C is about equal to the energy necessary to synthesize the tissue from the reserves. The maintenance rate constant for fresh water species seems to be much higher, ranging from 0.3 to 2.3 $d^{-1}$. Data from Smith [670] on the rainbow trout *Salmo irideus*, now called *S. gairdneri*, result in 1.8 $d^{-1}$ and figure 3.13 gives over 10 $d^{-1}$ for the waterflea *Daphnia magna* at 30 °C. The costs for osmosis might contribute to these high maintenance costs, as has been suggested on {78}.

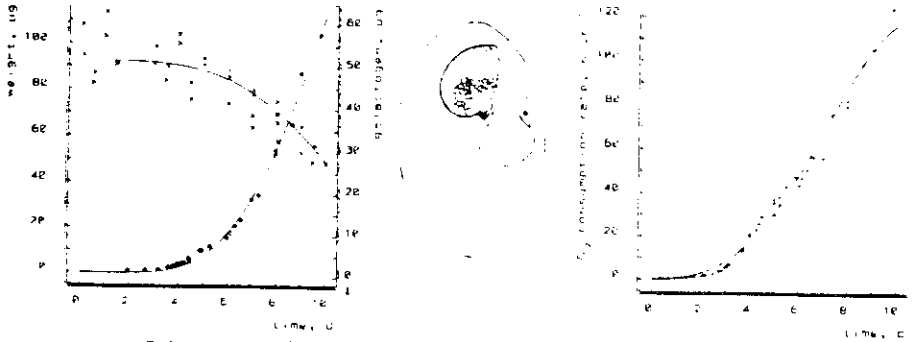
Table 3.1 shows that about half of the reserves are used during embryonic development. The deviating values for altricial birds are artifacts, due to the mentioned acceleration of development by increasing temperatures. Congdon *et al.* [133] observed that the turtles *Chrysemus picta* and *Emydoidea blandingi* have 0.38 of the initial reserves at birth. Respiration measurements on sea birds by Pettit *et al.* [545] indicate values that are somewhat above the ones reported in the table. The extremely small value for the soft shelled turtle, see also figure 3.14, relates to the fact that these turtles wait for the right conditions to hatch, where they have to run the gauntlet as a cohort at night from the beach to the water, where a variety of predators are waiting for them.

The general pattern of embryo development in eggs is characterized by unrestricted fast development during the first part of the incubation period (once it has started the process) due to unlimited energy supply, at a rate that would be impossible to reach if the animal had to refill reserves by feeding. This period is followed by a retardation of development due to the increasing depletion of energy reserves. Due to the goodness of fit of the model in species that do not possess shells, retardation is unlikely to be due to limitation of gas diffusion across the shell, as has been frequently suggested for birds [579]. Such a limitation also fails to explain why respiration declines in some species after its peak value, here beautifully illustrated with the turtle data.

Large eggs, so large initial energy supplies, thus result in short incubation times if eggs of one species are compared. Crested penguins, *Eudyptes*, are known for egg dimorphism [749]; see figure 3.15. They first lay a small egg and, some days later a 1.5 times bigger one. As predicted by the DEB model, the bigger one hatches first, if fertile, in which case the parents cease incubating the smaller egg, because they are only able to raise one chick. They continue to incubate the small egg only if the big one fails to hatch. This is probably [illegible] of loss of eggs

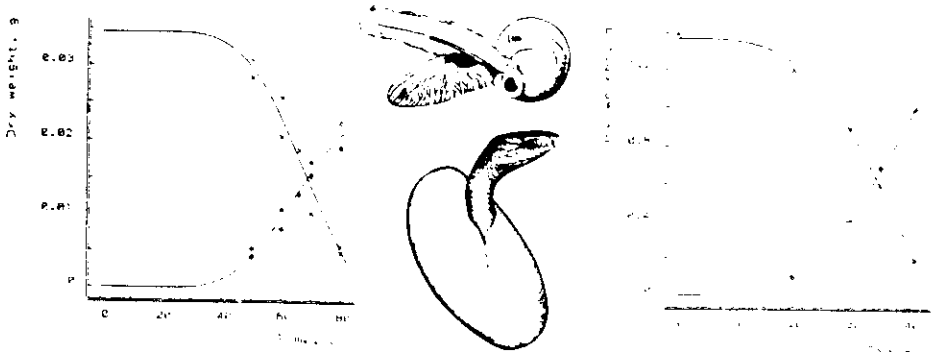
Figure 3.14: Yolk-free embryo weight (○), yolk weight (×), and respiration rate (+) during embryo development, and fits on the basis of the DEB model. Data sources are indicated.

pond snail *Lymnaea stagnalis* [335]

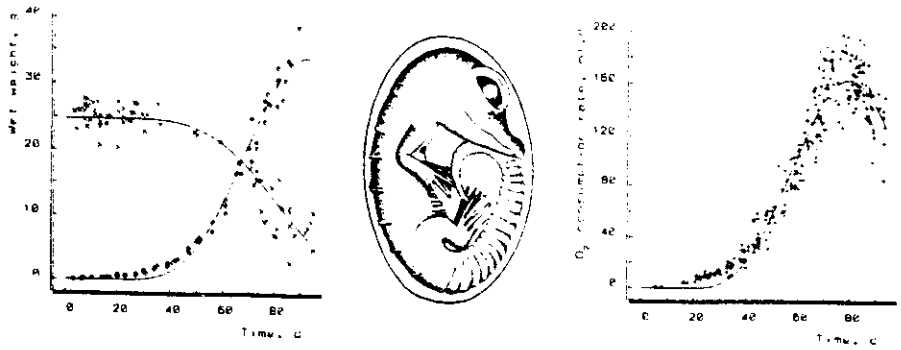


sea trout *Salmo trutta* [273]

American racer *Coluber constrictor* [523]

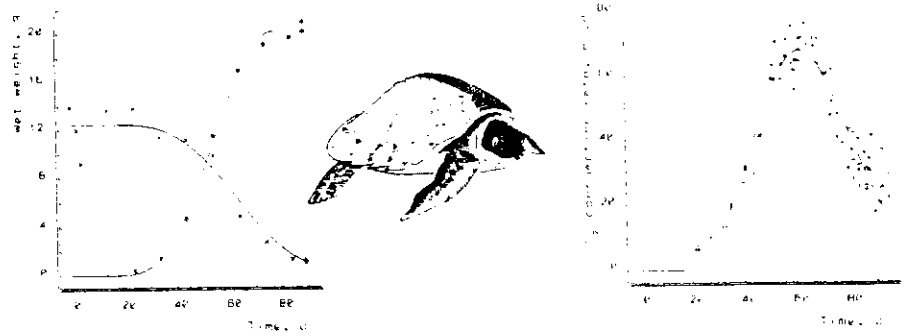


Australian crocodile *Crocodylus johnstoni* [452,765]





New Guinea soft-shelled turtle *Carettochelys insculpta* [755]



Laysan albatross *Diomedea immutabilis* [544]

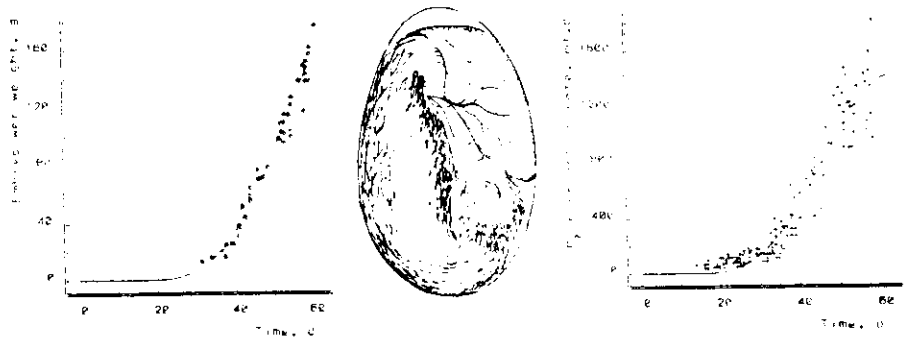


Table 3.1: Survey of re-analyzed egg data, and parameter values standardized to a temperature of 30 °C, taken from [790]. *1* Whitehead, pers. comm., 1989 ; *2* Thompson, pers. comm., 1989; 'galac.', stands for galactogen content.

| species | temp °C | type of data | $v_{30}$ mm d$^{-1}$ | $m_{30}$ d$^{-1}$ | $E_b/E_0$ | reference |
|---|---|---|---|---|---|---|
| *Lymnaea stagnalis* | 23 | ED, galac, O | 0.80 | 2.3 | 0.55 | [335] |
| *Salmo trutta* | 10 | ED, YD | 3.0 | 0.31 | 0.37 | [273] |
| *Rana pipiens* | 20 | EW, O | 2.5 | | 0.87 | [25] |
| *Crocodylus johnstoni* | 30 | EW, YW | 1.9 | 0.060 | 0.31 | [452] |
| | 29, 31 | O | | | | [765] |
| *Crocodylus porosus* | 30 | EW, YW | 2.7 | 0.023 | 0.19 | [756] |
| | 30 | O | | | | *1* |
| *Alligator mississippiensis* | 30 | EW, YW | 2.7 | | 0.34 | [160] |
| | 30 | O | | | | [716] |
| *Chelydra serpentina* | 29 | ED, YD | 1.9 | | 0.35 | [523] |
| | 29 | O | | | | [251] |
| *Carettochelys insculpta* | 30 | EW, YW, O | 1.9 | 0.040 | 0.08 | [755] |
| *Emydura macquarii* | 30 | EW, O | 1.6 | 0.14 | 0.35 | [716] |
| *Caretta caretta* | 28-30 | EW, O | 3.0 | | 0.65 | [3.2] |
| *Chelonia mydas* | 28-30 | EW, O | 3.0 | | 0.57 | [3.2] |
| *Amphisbolurus barbatus* | 29 | ED, YD | 0.92 | 0.063 | 0.47 | [524] |
| *Coluber constrictor* | 29 | ED, YD | 1.1 | | 0.69 | [525] |
| *Sphenodon punctatus* | 20 | HM, O | 0.85 | 0.062 | 0.25 | *2* |
| *Gallus domesticus* | 39 | EW, O, C | 3.2 | 0.039 | 0.34 | [610] |
| *Gallus domesticus* | 38 | EW, C | 3.1 | | 0.52 | [77] |
| *Lespoa ocellata* | 31 | EE, YE, O | 1.7 | 0.031 | 0.55 | [743] |
| *Pelecanus occidentalis* | 36.5 | EW, O | 3.2 | 0.10 | 0.77 | [38] |
| *Anous stolidus* | 35 | EW, O | 2.0 | 0.11 | 0.59 | [546] |
| *Anous tenuirostris* | 35 | EW, O | 1.8 | 0.20 | 0.59 | [546] |
| *Diomedea immutabilis* | 35 | EW, O | 2.5 | 0.069 | 0.57 | [544] |
| *Diomedea nigripes* | 35 | EW, O | 2.5 | 0.039 | 0.58 | [544] |
| *Puffinus pacificus* | 38 | EW, O | 0.92 | 0.084 | 0.61 | [4] |
| *Pterodroma hypoleuca* | 31 | EW, O | 1.9 | | 0.20 | [544] |
| *Larus argentatus* | 38 | EW, C | 2.7 | 0.15 | 0.56 | [182] |
| *Gygis alba* | 35 | EW, O | 1.1 | | 0.53 | [543] |
| *Anas platyrhynchos* | 37.5 | EW | 2.5 | 0.16 | 0.67 | [569] |
| | 37.5 | O | | | | [372] |
| *Anser anser* | 37.5 | EW | 4.1 | 0.039 | 0.23 | [609] |
| | 37.5 | O | | | | [741] |
| *Coturnix coturnix* | 37.5 | EW, O | 1.7 | | 0.49 | [741] |
| *Agapornis personata* | 36 | EW, O | 0.8 | | 0.79 | [107] |
| *Agapornis roseicollis* | 36 | EW, O | 0.81 | | 0.81 | [107] |
| *Troglodytes aedon* | 38 | EW, O | 1.1 | | 0.82 | [378] |
| *Columba livia* | 38 | EW | 2.7 | | 0.86 | [373] |
| | 37.5 | O | | | | [741] |

EW: Embryo Wet weight; YW: Yolk Wet weight; ED: Embryo Dry weight
EE: Embryo Energy content; YE: Yolk Energy content; YD: Yolk Dry weight
O: Oxygen consumption rate; C: Carbon dioxide production rate; HW: Hatchling Wet weight



Figure 3.15: Egg dimorphism occurs standard in crested penguins (genus *Eudyptes*). The small egg is laid first, but it hatches later than the big one, which is 1.5 times as heavy. The DEB theory explains why the large egg requires a shorter incubation period. The illustration shows the Snares crested penguin *E. atratus*.

(aggression [749]), which occurs in this species.

Incubation periods only decrease for increasing egg size if the structural biomass of the hatchling is constant. The incubation period is found to increase with egg size in some beetle species, lizards and marine invertebrates [128,206,660]. In these cases, however, the structural biomass at hatching also increased with egg size. This is again consistent with the DEB theory, although it does not explanain the variation in egg sizes.

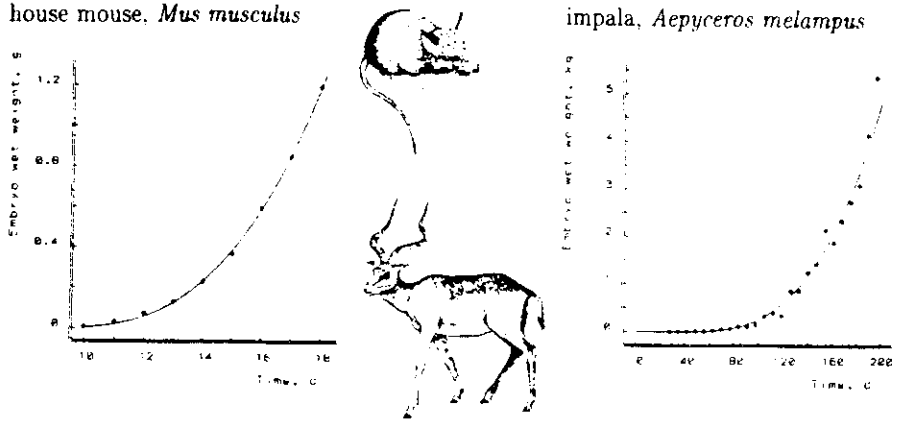
Foetal development differs from that in eggs in that energy reserves are supplied continuously via the placenta. The feeding and digestion processes are not involved. Otherwise, foetal development is taken to be identical to egg development, with initial reserves that can be taken to be infinitely large, for practical purposes. At birth, the neonate receives an amount of reserves from the mother, such that the reserve density of the neonate equals that of the mother. So the approximation $[E] = \infty$ for the foetus can be made for the whole gestation period and the dynamics of the reserve density (3.19) no longer applies, because the foetus lives on the reserves of the mother. In other words: unlike eggs, the development of foetuses is not restricted by energy reserves. Initially the egg and foetus develop in the same way, but the foetus keeps developing at an unrestricted rate till the end of the gestation time, while the development of the egg becomes retarded, due to depletion of the reserves. The approximation $[E] = \infty$ reduces the growth equation (3.12) to

$$\frac{d}{dt}V = \dot{v}V^{2/3} \quad \text{so} \tag{3.20}$$

$$V(t) = (\dot{v}t/3)^3 \tag{3.21}$$

This growth curve was proposed by Huggett and Widdas [341] in 1951. Payne and Wheeler [536] explained it by assuming that the growth rate is determined by the rate at which nutrients are supplied to the foetus across a surface that remains in proportion to the total

Figure 3.16: Foetal weight development in mammals.
house mouse, *Mus musculus* impala, *Aepyceros melampus*



surface area of the foetus itself. This is consistent with the DEB model, which gives the energy interpretation of the single parameter.

The fit is again excellent; see figure 3.16. It is representative for the data collected in table 3.2 taken from [790]. A time lag for the start of foetal growth has to be incorporated, and this delay may be related to the development of the placenta, which possibly depends on body volume as well. The long delay for the grey seal *Halichoerus* probably relates to timing with the seasons to ensure adequate food supply for the developing juvenile. Variations in weight at birth are primarily due to variations in gestation period, not in foetal growth rate. For comparative purposes, energy conductance $\dot{v}$ is converted to 30 °C, on the assumption that the Arrhenius temperature is $T_A = 10200$ K and the body temperature is 37 °C for all mammals in the table. This is a rather crude conversion because the cat, for instance, has a body temperature of 38.6 °C. Weights were converted to volumes using a specific density of $[d_w] = 1$ g cm$^{-3}$.

One might expect that precocial development is rapid, resulting in advanced development at birth and, therefore, comes with a high value for the energy conductance. The values collected in table 3.2, however, do not seem to have an obvious relationship with altricial-precocial rankings. The precocial guinea-pig and alpaca as well as the altricial humans have relatively low values for the energy conductance. The altricial-precocial ranking seems to relate only to the relative volume at birth $V_b/V_m$.

## Egg costs

The embryo thus develops from state $(a, [E], V) = (0, \infty, 0)$ to state $(a_b, [E_b], V_b)$. The costs for growth and maintenance together with $\kappa$ determine the energy costs for an egg, $E_0$. These costs and the incubation time thus follow from specifications at hatching. This back reasoning is necessary because the initial volume is taken to be infinitesimally small, which makes the initial reserve density infinitely large.

The derivation of the costs for an egg is a bit technical, I am afraid, due to the nonlinearity of the dynamics. We will need the costs to go from an energy flux allocated



Table 3.2: The estimated energy conductance, $\dot{v}$, and its value corrected for a temperature of 30 °C, and the time lag for the start of development, $t_l$, for mammalian embryos.

| Species (race) | $\dot{v}$ mm d$^{-1}$ | (cv) | $\dot{v}_{30}$ mm d$^{-1}$ | $t_l$ d | (cv) | reference |
|---|---|---|---|---|---|---|
| *Homo sapiens* | | | 0.84 | | | |
| males | 0.180 | (0.3) | | 26.8 | (2.0) | [758] |
| females | 0.179 | (0.4) | | 26.5 | (2.9) | |
| *Oryctolagus cuniculus* | 0.560 | (0.9) | 2.6 | 10.7 | (1.5) | [432] |
| small litters | 0.602 | (1.5) | | 11.5 | (2.4) | [34] |
| large litters | 0.571 | (1.5) | | 11.5 | (2.4) | [34] |
| | 0.504 | (5.6) | | 10.4 | (10) | [36] |
| *Lepus americanus* | 0.573 | (3.1) | 2.7 | 13.1 | (4.2) | [79] |
| *Cavia porcellus* | 0.269 | (3.3) | 1.1 | 15.7 | (8.3) | [180] |
| | 0.239 | (2.3) | | | | [346] |
| *Cricetus auratus* | 0.570 | (2.1) | 2.6 | 9.29 | (1.3) | [573] |
| *Mus musculus* | 0.333 | (0.1) | 1.5 | 8.45 | (0.1) | [448] |
| *Rattus norvegicus* | | | 2.5 | | | |
| wistar | 0.487 | (0.5) | | 11.4 | (0.3) | [217] |
| albino | 0.531 | (0.8) | | 12.2 | (0.5) | [688] |
| | 0.525 | (0.2) | | 11.8 | (0.2) | [341] |
| albino | 0.568 | (3.3) | | 12.7 | (2.1) | [16] |
| albino | 0.542 | (3.1) | | 12.4 | (2.0) | [222] |
| *Clethrionomys glareolus* | 0.374 | (9.3) | 1.8 | 8.29 | (11) | [140] |
| *Aepyceros melampus* | 0.316 | (1.2) | 1.4 | 39.4 | (3.8) | [210] |
| *Odocoileus virginianus* | 0.296 | (6.7) | 1.3 | 34.9 | (28) | [605] |
| | 0.274 | (1.6) | | 25.1 | (8.5) | [733] |
| *Dama dama* | 0.345 | (6.4) | 1.7 | 9.94 | (46) | [21] |
| *Cervus canadensis* | 0.336 | (3.1) | 1.5 | 24.9 | (19) | [494] |
| *Lama pacus* | 0.120 | (7.6) | 0.56 | 7.47 | (83) | [218] |
| *Ovis aries* | | | 1.9 | | | |
| welsh | 0.482 | (5.6) | | 43.9 | (12) | [341] |
| merino | 0.341 | (8.6) | | 14.9 | (71) | [450] |
| | 0.346 | (4.6) | | 15.2 | (32) | |
| | 0.433 | (4.4) | | 33.3 | (13) | [130] |
| karakul | 0.436 | (3.7) | | 31.6 | (13) | [193] |
| | 0.403 | (2.6) | | 27.5 | (8.2) | [365] |
| hampshire × | 0.382 | (3.5) | | 20.4 | (7.9) | [775] |
| *Capra hircus* | 0.339 | (6.5) | 1.7 | 24.3 | (29) | [199] |
| | 0.365 | (4.5) | | 31.3 | (14) | [35] |
| *Bos taurus* | 0.475 | (2.6) | 2.3 | 59.5 | (7.5) | [776] |
| *Equus caballus* | 0.370 | (11) | 1.8 | 37.0 | (81) | [481] |
| *Sus scrofa* | 0.266 | (0.6) | | 4.73 | (12) | [750] |
| Yorkshire | 0.283 | (0.9) | | 5.49 | (16) | [729] |
| Large white | 0.383 | (1.3) | | 23.6 | (4.2) | [562] |
| Essex | 0.321 | (4.8) | | 14.1 | (30) | |
| *Felix catus* | 0.371 | (1.2) | 1.8 | 18.8 | (2.3) | [139] |
| *Pipistrellus pipistrellus* | | | 0.97 | | | |
| 1978 | 0.237 | (1.9) | | 9.95 | (2.9) | [575] |
| 1979 | 0.181 | (3.5) | | 13.7 | (4.7) | |
| *Halichoerus grypus* | 0.375 | (10) | 1.8 | 145 | (9.2) | [314] |

to reproduction to a reproductive rate. You will not miss a lot if you skip the rest of this section, if you are ready to accept the result that egg costs do not involve any new parameters. Costs for breeding by the parent are not included in this derivation.

The first step to derive the costs for an egg is to get rid of a number of parameters by turning to the dimensionless variables scaled energy density $e = [E]/[E_m]$, scaled volumetric length $l = (V/V_m)^{1/3}$ and scaled time $\tau = t\dot{m}$. Substitution into (3.19) and (3.12), reduces the coupled differential equations to

$$\frac{d}{d\tau}e = -g\frac{e}{l} \text{ and } \frac{d}{d\tau}l = \frac{g}{3}\frac{e-l}{e+g} \tag{3.22}$$

The ratio of these equations gives the Bernoulli equation

$$\frac{dl}{de} = -\frac{l}{3e}\frac{e-l}{e+g} \text{ or } \frac{dx}{de} = \frac{ex-1}{3e(e+g)} \tag{3.23}$$

where $x \equiv l^{-1}$ is only introduced because the resulting equation in $x$ is of a solvable linear first order with variable coefficients. Its solution is

$$x(e) = v(e)\left(\int_{e_b}^{e}\frac{-de_1}{3(e_1+g)e_1v(e_1)} + x(e_b)\right) \tag{3.24}$$

$$\text{with } v(e) = \exp\{\int_{e_b}^{e}\frac{de_1}{3(g+e_1)}\} = \left(\frac{g+e}{g+e_b}\right)^{1/3}$$

Substitution of $l = x^{-1}$ gives

$$\frac{1}{l} = \left(\frac{g+e}{g+e_b}\right)^{1/3}\left(\frac{1}{l_b} - \frac{(g+e_b)^{1/3}}{3g^{4/3}}\int_{\frac{e_b}{e_b+g}}^{\frac{e}{e+g}} s^{-1}(1-s)^{1/3}\,ds\right) \tag{3.25}$$

Assume that the condition at hatching is fixed at $e_b$ and $l_b$ and let $l \to 0$ and $e \to \infty$ such that $[E_m]V_m e l^3 = E_0$, say, which has the interpretation of the energy reserves in a freshly laid egg. Solving $E_0$ gives for $\epsilon_0 \equiv \frac{E_0}{[E_m]V_m}$

$$\epsilon_0 = \left(\frac{1}{l_b(g+e_b)^{1/3}} - \frac{B_{\frac{e}{e_b+e}}(\frac{1}{3},0)}{3g^{4/3}}\right)^{-3} \tag{3.26}$$

where $B_x(a,b) \equiv \int_0^x y^{a-1}(1-y)^{b-1}\,dy$ is the incomplete beta function. Its two term Taylor expansion in $[G]$ around the point $[G] = 0$ gives

$$\epsilon_0 \simeq \frac{2^6\epsilon_b^4}{(4\epsilon_b/l_b-1)^3} + \frac{4\epsilon_b/l_b - 16/7}{(4\epsilon_b/l_b-1)^4}g2^6\epsilon_b^3 \tag{3.27}$$

### Incubation time

The incubation time can be found by separating variables in (3.22) and substituting in (3.25). After some transformation, the result is

$$a_b = \frac{3}{\dot{m}}\int_0^{x_b}\frac{dx}{(1-x)x^{2/3}(\alpha - B_x(\frac{4}{3},0) + B_x(\frac{1}{3},0))} \tag{3.28}$$



where $x_b \equiv \frac{g}{\epsilon_b+g}$ and $\alpha \equiv 3gx_b^{1/3}/l_b$. Its two term-Taylor expansion in $[G]$ around the point $[G] = 0$ gives after tedious calculation

$$a_b \simeq \frac{3\sqrt{2}}{\dot{m}}u^3\left(\frac{\epsilon_b}{g} + \frac{1}{4} - \frac{9}{28}u^4\right)\left(\frac{1}{2}\ln\frac{u^2+u\sqrt{2}+1}{u^2-u\sqrt{2}+1} + \arctan\frac{u\sqrt{2}}{1-u^2}\right) + \frac{9}{7\dot{m}}(u^4 + \ln\{1+u^4\}) \tag{3.29}$$

where $u$ stands for $(4\epsilon_b/l_b - 1)^{-1/4}$. I owe you an apology for writing out such a threatful expression; the essence, however, is that no new parameters show up and that (3.29) can readily be implemented in computer code.

### Foetal costs and gestation time

The energy costs for the production of a neonate is found by the addition of costs for development, growth and maintenance plus energy reserves at birth, i.e. $[E_b]V_b$. Expressed as a fraction of the maximum energy capacity of an adult, these costs are

$$\epsilon_0 = (\int_0^{a_b}\dot{C}(t)\,dt + [E_b]V_b)([E_m]V_m)^{-1}$$

Substitution of the $\kappa$-rule, $\kappa\dot{C} = [G]\frac{d}{dt}V + [\dot{M}]V$, and the growth curve (3.21) results in

$$\epsilon_0 = l_b^3(g + \epsilon_b + l_b 3/4) \tag{3.30}$$

This expression does not include the costs for the placenta. These costs can easily be taken into account if they happen to be proportional to that of the rest of the foetuses; see {100}.

Gestation time (excluding any time lag) is

$$a_b = 3l_b/g\dot{m} = 3V_b^{1/3}/\dot{r} \tag{3.31}$$

## 3.8.2 Growth for non-isomorphs

The above derivation assumes isomorphism, but it can easily be extended to include changing shapes. The surface areas of organisms that change shape, such as filaments and rods, have to be corrected for this change by multiplying parameters containing surface area, $\{\dot{I}_m\}$ and $\{\dot{A}_m\}$ and thus $\dot{v}$ and $V_m$, by the shape correction function $\mathcal{M}(V)$. These organisms are ectothermic, so $\{H\} = 0$. For filaments, the shape correction function (2.4) transforms the change of energy density (3.7) and the growth rate (3.12) into

$$\frac{d}{dt}[E] = [\dot{A}_m](f - [E]/[E_m]) \tag{3.32}$$

$$\frac{d}{dt}V = \dot{\nu}\frac{[E]/[E_m] - (V_d/V_m)^{1/3}}{g + [E]/[E_m]}V \tag{3.33}$$

where $V_d$ is the volume at division, and $V_m$ is defined by $V_m^{1/3} = \frac{\dot{r}}{g\dot{m}}$. The length-specific energy conductance $\dot{\nu}$ is just an abbreviation for $\dot{\nu} \equiv \dot{v}V_d^{-1/3}$. It has dimension time$^{-1}$. Likewise, the notation $\{\dot{A}_m\} \equiv \{\dot{A}_m\}V_m^{-1/3}$ is introduced. If substrate density $X$ and,

therefore, the scaled functional response $f$ are constant long enough, energy density tends to $[E] = f[E_m]$ and volume as a function of time since division becomes for $V(0) = V_d/2$

$$V(t) = \frac{1}{2}V_d \exp\{t\dot{\gamma}_f\} \quad \text{or} \tag{3.34}$$

$$t(V) = \dot{\gamma}_f^{-1} \ln\{2V/V_d\} \tag{3.35}$$

with $\dot{\gamma}_f \equiv \dot{v}\frac{f-(V_d/V_m)^{1/3}}{f+g}$. The time taken to grow from $V_d/2$ to $V_d$ is thus $t(V_d) = \dot{\gamma}_f^{-1} \ln 2$.

Exponential growth can be expected if the surface area at which nutrients are taken up is proportional to volume. For filaments, this happens when the total surface area, or a fixed fraction of it, is involved. If uptake only takes place at tips, the number of tips should increase with total filament length to ensure exponential growth. This has been found for the fungi *Fusarium* [725], and *Penicillium* [506,558], which do not divide: see figure 3.17. The ascomycetous fungus *Neurospora* does not branch this way [201]; it has a mycelium that grows like a crust, see {145}.

Exponential growth of individuals should not be confused with that of populations. As will be discussed in the chapter on population dynamics, all populations grow exponentially at resource densities that are constant long enough, whatever the growth pattern of individuals. This is due to the simple fact that the progeny repeats the growth/reproduction behaviour of the parents. Only for filaments it is unnecessary to distinguish between the individual and the population level. This is a characteristic property of exponential growth of individuals and will be discussed on {162}.

The same derivation for growth can be made for rods on the basis of the shape correction function (2.7):

$$\frac{d}{dt}[E] = [\dot{A}_m]\left(\frac{\delta}{3}\frac{V_d}{V} + 1 - \frac{\delta}{3}\right)\left(f - \frac{[E]}{[E_m]}\right) \tag{3.36}$$

$$\frac{d}{dt}V = \dot{v}\frac{\delta V_d}{3V_\infty}\frac{[E]/[E_m]}{g + [E]/[E_m]}(V_\infty - V) \tag{3.37}$$

where $V_\infty \equiv V_d\frac{\delta}{3}(\frac{[E_m]}{[E_j]}(\frac{V_d}{V_m})^{1/3} - 1 + \frac{\delta}{3})^{-1}$ and, as before, $V_m^{1/3} \equiv \frac{\dot{v}}{g\dot{m}}$. If substrate density $X$ and, therefore, the scaled functional response $f$ are constant long enough, energy density tends to $[E] = f[E_m]$ and volume as a function of time since division becomes

$$V(t) = V_\infty - (V_\infty - V_d/2)\exp\{-t\dot{\gamma}_r\} \tag{3.38}$$

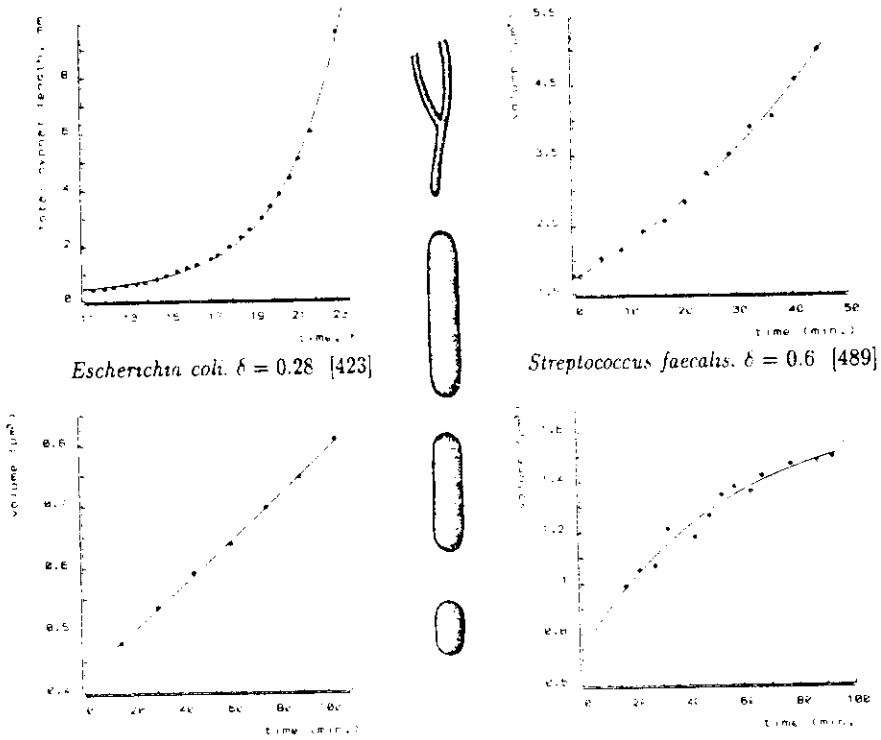
where $\dot{\gamma}_r \equiv \frac{V_d f \dot{v} g/3}{V_\infty(f+g)}$. The interpretation of $V_\infty$ depends on its value.

- If $V_\infty = \infty$, i.e. if $f(1-\delta/3) = (V_d/V_m)^{1/3}$, the volume of rods grows linearly at rate $\frac{\dot{v}f}{f+g}V_d\frac{\delta}{3}$. This is frequently found empirically [29].
- If $0 < V_\infty < \infty$, $V_\infty$ is the ultimate volume if the cell ceased to divide but continued to grow. For these values, $V(t)$ is a convex function and is of the same type as $V(t)^{1/3}$ for isomorphs, (3.14). Note that volume, and thus cubed length, grows skewly S-shaped for isomorphs. When $V_\infty$ is positive, the cell will only be able to divide when



Figure 3.17: DEB-based growth curves for cells of filaments and rods. The larger the aspect ratio, $\delta$, the more the growth curve turns from the exponential to the satiation type, reflecting the different surface area to volume relationships.

*Fusarium graminearum*, $\delta = 0$ [725] *Bacillus cereus*, $\delta = 0.2$ [131]



*Escherichia coli*, $\delta = 0.28$ [423] *Streptococcus faecalis*, $\delta = 0.6$ [489]

- If $\delta = 0$, $V_\infty = 0$ and the rod behaves as a filament, which grows exponentially.
- For $V_\infty < 0$, $V(t)$ is a concave function, tending to an exponential one. The cell no longer has an ultimate size if it ceased to divide. $V_\infty$ is then no longer interpretated as ultimate size, but this does not invalidate the equations.

The shape of the growth curve, convex, linear or concave, thus depends on substrate density and the aspect ratio. Figure 3.17 illustrates the perfect fit of growth curves (3.38) with only three parameters: volume at 'birth', $V_d/2$, ultimate volume, $V_\infty$, and growth rate, $\dot{\gamma}_r$. The figure beautifully reveals the effect of the aspect ratio; the larger the aspect ratio, the more important the effect of the caps, so a change from 1D-isomorphic behaviour to a 0D-isomorphic behaviour.

The transition from $V_d/2$ to $V_\infty$ at constant substrate density is found from

(3.38):

$$t(V) = \frac{(f+g)V_\infty}{f\dot{v}V_d\delta/3} \ln \frac{V_\infty - V_d/2}{V_\infty - V} \tag{3.39}$$

At the end of the cell cycle, the cell has to synthesize extra cell wall material. Since the cell grows in length only, the growth of surface material is directly tied to that of cytoplasm material. Straightforward geometry shows that the change in surface area $A$ is given by $\frac{d}{dt}A = (16\pi\frac{1-\delta/3}{\delta V_d})^{1/3}\frac{d}{dt}V$. So the energy costs for growth can be partitioned as $[G] = [G_V] + \{G_A\}(16\pi\frac{1-\delta/3}{\delta V_d})^{1/3}$, where $\{G_A\}$ denotes the energy costs for the material in a unit surface area of cell wall and $[G_V]$ that for the material in a unit volume of cytoplasm. For reasons of symmetry, it is more elegant to work with $[G_A] \equiv \{G_A\}V_d^{-1/3}$ rather than $\{G_A\}$. The dimensions of $[G_V]$ and $[G_A]$ are then the same: energy per volume. At the end of the cell cycle, when cell volume is twice the initial volume, the surface material should still increase from $A(V_d)$ to $2A(V_d/2) = (1+\delta/3)A(V_d)$. This takes time, of course. If all incoming energy not spent on maintenance is used for the synthesis of this material, the change in surface area is given by $\frac{d}{dt}A = \frac{\dot{v}}{g_A}(fA - V_d/V_m^{1/3})$, where $g_A \equiv [G_A]/\kappa[E_m]$. So $A(t) = (A(0) - V_d/fV_m^{1/3})\exp\{tf\dot{v}/g_A\} + V_d/fV_m^{1/3}$. The time it takes for the surface area to reach $(1+\delta/3)V_d^{2/3}$, starting from $A(0) = V_d^{2/3}$, equals

$$t_A = \frac{g_A}{f\dot{v}}\left(\ln 2 + \ln \frac{V_\infty - V_d/2}{V_\infty - V_d}\right) \tag{3.40}$$

For the time interval between subsequent divisions, $t(V_d)$ must be added, giving

$$t_d = \frac{g_A}{f\dot{v}} \ln 2 + \left(\frac{g_A}{f\dot{v}} + \frac{(f+g)V_\infty}{f\dot{v}V_d\delta/3}\right) \ln \frac{V_\infty - V_d/2}{V_\infty - V_d} \tag{3.41}$$

The extra time for cell wall synthesis at the caps does not play a role for filaments, as their caps are comparatively small. It also does not play a significant role in unicellular eukaryotic isomorphs, because they do not have cell walls to begin with. The cell volume is full of membranes in these organisms, so the amount of membranes at the end of the cell cycle does not need to increase as abruptly as in bacteria, where the outer membrane and cell wall (if present) are the only surfaces. Comparable delays occur in ciliates for instance, where the cell mouth does not function during and around cell division.

Cooper [138] and Koch [398] argued that weight increase of bacterial cells is always of the exponential type, apart from minor contributions of cell wall, DNA, etc. If the activity of the carriers for substrate uptake is constant during the cell cycle, an implication of this model is that carriers should be produced at a rate proportional to the growth rate, and consequently to cell volume rather than to surface area. This would increase the number of carriers per unit of surface area of active membrane during the cell cycle. At the end of the cell cycle the number of carriers per unit of surface area should (instantaneously) drop by a factor of $(1+\delta/3)^{-1}$ due to the production of new membrane without carriers that separates the daughter cells. This factor amounts to $5/6 = 0.83$ for cocci and 1 for 1D-isomorphs. The factor stands for the ratio of the surface area of a body with volume $V_d$ and two times the surface area of a body with volume $V_d/2$; so it is $2^{-1/3} = 0.79$ for



3D-isomorphs and $2^{-1/2} = 0.71$ for 2D-isomorphs. To my knowledge, such a reduction has never been demonstrated. The carrier density is assumed to be constant in the DEB theory. If the carrier density in the membrane is constant in case of exponential growth (in non-1D-isomorphs), the carrier activity should increase during the cell cycle. This requires the loss of homeostasis and/or complex regulation of carrier activity. In the DEB theory, the carrier activity is constant during the cell cycle. Although exponential growth of the cell seems an attractively simple model at first sight, theory to tie the growth rate to nutrient levels no longer comes naturally for such an extreme 'demand' type of system. Moreover, phenomena such as the small cell size in oligotrophic oceans, the growth of stalks in *Caulobacter*, the removal of disused DNA need other explanations than given in this book. Another point is of course, that if bacteria increase their weight exponentially, they would deviate from unicellular eukaryotes in this respect, where exponential growth is obviously untenable, cf. figure 1.1. The problem should then be adressed of what makes prokaryotes fundamentally different from eukaryotes in terms of energetics.

## 3.9 Development

Now that growth has been specified, the utilization rate for isomorphs can be evaluated from (3.8) and (3.12). It amounts to

$$\dot{C} = \frac{g[E]}{g + [E]/[E_m]}(\dot{v}V^{2/3} + \dot{m}V_b^{1/3}V^{2/3} + \dot{m}V) \tag{3.42}$$

Energy allocation to development is $(1-\kappa)\dot{C}$. Comparison of growth and reproduction at different food levels points to a problem: the volume at the first appearance of eggs in the broodpouch of daphnids seems to be independent of food density. It appears to be almost fixed; see figure 3.18. Let this volume be called $V_p$, where subscript $p$ refers to puberty (transition juvenile/adult). The same holds for the volume at hatching, $V_b$, say, where subscript $b$ refers to birth (transition embryo/juvenile). The problem is that the total energy investment in development depends on food density. Indeed, if feeding conditions are so poor that the ultimate volume is less than $V_p$, the cumulated energy investment into development becomes infinitely large, if the organism survives long enough. This seems to be highly unrealistic.

Horst Thieme [711] proposed a solution to this problem: split the energy allocated to development into two fluxes, the increase of the state of maturity and the maintenance of a certain degree of maturity. For a special choice of the maturity maintenance costs the total energy investment into the increase of the state of maturity does not depend on food density for ectotherms. This can be seen most easily from (3.11), when both sides are multiplied by $(1-\kappa)/\kappa$ to obtain the investment into development

$$(1-\kappa)\dot{C} = \frac{1-\kappa}{\kappa}\dot{M} + \frac{1-\kappa}{\kappa}[G]\frac{d}{dt}V \tag{3.43}$$

for juvenile ectotherms ($V < V_p$ and $\dot{H} = 0$). If the first term of the right hand side corresponds to maturity maintenance costs, the second one for the increase of the state of

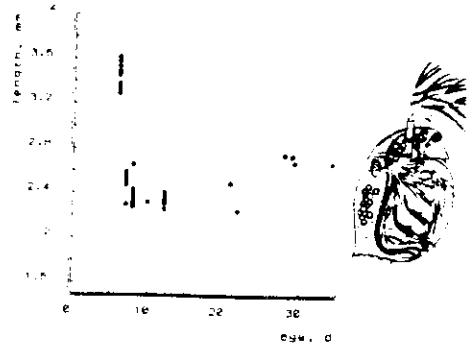


Figure 3.18: The carapace length of the daphnid *Daphnia magna* at 20 °C for 5 different food levels at the moment of egg deposition in the brood pouch. Data from Baltus [32]. The data points for short juvenile periods correspond with high food density and growth rate. They are difficult to interpret because length increase is only possible at moulting in daphnids.

maturity only depends on size, not on food density. Since the individual does not become more complex after attaining size $V_p$, the energy flow to maintain a certain degree of maturity must then be $\min\{V, V_p\}[\dot{M}]\frac{1-\kappa}{\kappa}$. It can be thought to relate to the maintenance of regulating mechanisms and of concentration gradients, such as those found in *Hydra*, that are responsible for the maintenance of the head/foot differentiation [258].

It took me quite a while to accept the existence of maturity maintenance as inevitable. Although the concept sounds a little esoteric, there are two hard observations in support of its existence. The first one concerns an experiment where food density is held constant at two levels, just below and above the food density that gives an ultimate size $V_\infty = V_p$. For ectotherms, such as daphnids, (3.17) implies that this food density is found from $f = (V_p/V_m)^{1/3} \equiv l_p$, so $X = Kl_p/(1-l_p)$. If maturity maintenance did not exist, animals kept at the lower food density would never reproduce, while those at the higher food density would reproduce at a rate that might be substantial, depending on $\kappa$. Substitution of $V = V_p$ into (3.11) shows that the energy investment into development and/or reproduction tends to $\frac{1-\kappa}{\kappa}[\dot{M}]V_p$, which amounts to $4[\dot{M}]V_p$ for $\kappa = 0.2$, which is realistic for daphnids. This substantial difference in reproductive output as a result of a tiny difference in feeding rates has never been observed.

The second observation that points to the existence of maturity maintenance concerns pond snails, where the day/night cycle affects the fraction of utilized energy spent on maintenance plus growth [788], such that $\kappa$ at equal day/equal night, $\kappa_{md}$, is larger than that at long day/short night, $\kappa_{ld}$. Apart from the apparent effects on growth and reproduction rates, volume at the transition to adulthood is also affected. If the cumulated energy investment into the increase of maturity does not depend on the value for $\kappa$ and if the maturity maintenance costs are $\frac{1-\kappa}{\kappa}\dot{M}$, the expected effect is $\frac{V_{p,ld}}{V_{p,md}} = \frac{\kappa_{ld}(1-\kappa_{md})}{\kappa_{md}(1-\kappa_{ld})}$, which is consistent with the observations on the coupling of growth and reproduction investments to size at puberty [788]. Some species, such as birds, only reproduce well after the growth period. The giant petrel wanders seven years over Antarctic waters before it starts to breed for the first time. From a mathematical point of view, growth is asymptotic, so it is possible to choose $V_p$ to be so close to $V_\infty$ that the desired result is described adequately. This must be rejected, however, because it seems most unrealistic to have a model where decision rules depend on such small differences in volume in a world that is full of scatter.



The introduction of costs for maintaining a certain degree of maturity solves this problem, because the model is then energy-structured as well as size-structured. A transition from embryo to juvenile and from juvenile to adult occurs if the cumulative investment to increase the state of maturity exceeds specified amounts. If growth has almost ceased, this cumulative investment increases linearly, it therefore has no asymptote. The rate of increase of cumulated investment can be substantial, even if body size hardly increases, so this rule causes no problems for species that separate growth and reproduction in time.

There is, however, a problem connected with this introduction of maturity maintenance: it is hard to see why it should have just the value $\frac{1-\kappa}{\kappa}\dot{M}$; it is a fact that it produces the observed fixed-volume transition in daphnids and pond snails, but one would like to understand why. One solution might be to interpret $\frac{1-\kappa}{\kappa}$ as the basic parameter and try to explain why the relative allocation to development plus reproduction takes a value that relates to the costs of development. I still find this an unsatisfactory point in the theory. Of course, it is possible to introduce a free parameter for the maturity maintenance costs and use volume at first maturation for the estimation of its value, which then proves to be close to $\dot{M}_d = \frac{1-\kappa}{\kappa}\dot{M}$, because this value produces a volume that is independent of food density. If this free parameter has a different value, variations in volume at first maturation will result when food density varies, as has been observed for some species, according to the review by Bernardo [60]. Its introduction has the serious drawback that evaluation of the length of incubation and juvenile period become cumbersome, which causes problems especially at the population level. The fixed size transition should then be replaced by a fixed cumulative energy transition.

Note that growth and development are parallel processes in the DEB model, which links up beautifully with the concepts of acceleration and retardation of developmental phenomena such as sexual maturity [267]. These concepts are used to describe relative rates of development in species that are similar in other respects.

In embryos and juveniles, the energy spent on somatic maintenance and the maintenance of a certain degree of maturation can be combined, because both can be taken proportional to volume. The difference between the two only shows up in adults that still increase in size. Somatic maintenance remains proportional to size, while maturation maintenance stays constant at constant temperature. The same holds for the energy spent on growth and the increase of the degree of maturity. In embryos and juveniles, they can be combined, because both are taken proportional to volume increase. This means that for non-adults the $\kappa$-rule is not of quantitative relevance, and the model simplifies to the one for micro-organisms with respect to the use of energy.

Whether or not unicellulars and particularly prokaryotes invest in cell differentiation during the cell cycle is still open to debate. Dworkin [192] gives a review of development in prokaryotes and points to the striking similarities between myxobacteria and cellular slime molds and *Actinomyceta* and some fungi. A most useful aspect of the $\kappa$-rule is that this matter need not first be resolved, because this investment only shows up in the parameter values and not in the model structure. As stated in the introduction to this chapter, the energy invested in development according to the $\kappa$-rule can only be deduced from the transition to the adult state in metazoans. The utilization rate for rods can be obtained in the same way as for isomorphs: application of the shape correction function (2.7) to $\dot{r}$

(3.42). This amounts to

$$\dot{C} = \frac{g[E]}{g + [E]/[E_m]} \left( \dot{\nu} \frac{\delta}{3} V_d + \left( \dot{m} + \dot{\nu} \left( 1 - \frac{\delta}{3} \right) \right) V \right) \tag{3.44}$$

e utilization rate for filaments can be found by application of the shape correction ction (2.4) to $\dot{v}$ in (3.42), which leads to

$$\dot{C} = \frac{g[E]}{g + [E]/[E_m]} (\dot{m} + \dot{\nu}) V \tag{3.45}$$

can also be obtained by letting $\delta \to 0$ in (3.44). Since both growth and maintenance are proportional to volume for filaments, the utilization rate is also proportional to volume.

# 10 Propagation

ganisms can achieve an increase in numbers in many ways. Sea anemones can split off ot tissue that can grow into a new individual. This is not unlike the strategy of budding asts. Colonial species usually have several ways of propagating. Fungi have intricate ual reproduction patterns involving more than two sexes. Under harsh conditions some imals can switch from parthenogenic to sexual reproduction, others develop spores or ner resting phases. It would not be difficult to fill a book with descriptions of all the ssibilities. I will confine the discussion to the two most common modes of propagation: egg and foetus or vegetatively, via division.

## 10.1 Reproduction

ergy allocation to reproduction equals the allocation to development plus reproduction nus the costs to maintain the state of maturity

$$(1 - \kappa)\dot{C} - \frac{1 - \kappa}{\kappa}[M]V_p \tag{3.46}$$

his is a continuous energy investment. The costs for egg (or foetus) development are lly determined, as has been discussed in the section on embryonic growth, {83}. The sts for the production of an egg can be written as $E_0/q$, where the dimensionless factor $q$ tween 0 and 1 relates to the overhead involved in the conversion from the reserve energy the mother to the initial energy available for the embryo. Since these types of energy serves are chemically related, the overhead is likely to be small in most cases so that $q$ close to 1. This might seem an odd way to introduce this overhead, but $q$ can also be terpreted as an egg survival probability, which can be further modulated by predation id toxic compounds, as discussed in later chapters. This is practical because egg survival frequently governed by different processes than survival of later stages. Substitution of ilization rate (3.42) into (3.46) leads to a mean reproduction rate for ectotherms of

$$\dot{R} = \frac{q}{\cdot\cdot}(1 - \kappa) \left( \frac{g[E]/[E_m]}{\cdot\cdot} (\dot{\nu} V^{2/3} + \dot{m} V) - g \dot{m} V_p \right) \tag{3.47}$$





where the relative energy costs for embryo development $e_0$ are given in (3.26). Under no-growth conditions, i.e. when $\frac{[E]}{[E_m]} \leq (\frac{V}{V_m})^{1/3}$, individuals can no longer follow the $\kappa$-rule, because the allocation to maintenance would no longer be sufficient. Maintenance has priority over all other expenses. Individuals that still follow the storage dynamics (3.8) under no-growth conditions, must reproduce at mean rate $(\dot{C} - \dot{M} - \frac{1-\kappa}{\kappa}[M]V_p)q/E_0$, so

$$\dot{R} = \frac{q}{e_0 V_m} g \dot{m} \left( \frac{[E]}{[E_m]} V_m^{-1/3} V^{2/3} - \kappa V - (1 - \kappa) V_p \right)_+ \tag{3.48}$$

At the border of the no-growth condition, i.e. when $\frac{[E]}{[E_m]} = (\frac{V}{V_m})^{1/3}$, both expressions for the reproduction rate are equal, so there is no discontinuity for changing energy reserves.

At constant food density where $[E] = f[E_m]$, the reproduction rate is according to (3.47) proportional to

$$\dot{R} \propto V^{2/3} + \frac{\dot{m}}{\dot{v}} V - \frac{g + f}{f} \frac{\dot{m}}{\dot{v}} V_p \tag{3.49}$$

where the third term is just a constant. Comparison of reproduction rates for different body sizes thus involves three compound parameters, i.e. the proportionality constant, the parameter $\dot{m}/\dot{v}$ and the third term, if all individuals experience the same food density for a long enough time. Figure 3.19 illustrates that this relationship is realistic, but that the notorius scatter for reproduction data is so large that access to the parameter $\dot{m}/\dot{v}$ is poor. The fits have been based on guestimates for the maintenance rate coefficient, $\dot{m} = 0.011$ d$^{-1}$, and the energy conductance, $\dot{v} = 0.433$ mm d$^{-1}$ at 20 °C. Note that if the independent variable is a length measure rather than structural body volume, the shape coefficient $d_m = V^{1/3} L^{-1}$ has to be introduced since the guestimate for the energy conductance is expressed in volumetric length. For some length measure $L$, we have

$$\dot{R} \propto L^2 + \frac{\dot{m}}{\dot{v}} d_m L^3 - \frac{g + f}{f} \frac{\dot{m}}{\dot{v}} d_m L_p^3 \tag{3.50}$$

The practical significance of this remark is in the comparison between species, which will be discussed later, {217}. The main reason for the substantial scatter in reproduction data is that they are usually collected from the field, where food densities are not constant, and where spatial heterogeneities, social interactions, etc., are common.

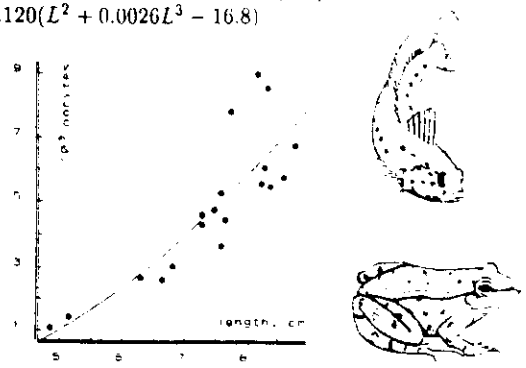
The reproduction rate of spirorbid polychaetes has been found to be roughly proportional to body weight [312]. On the assumption by Strathmann and Strathmann [694] that reproduction rate is proportional to ovary size and that ovary size is proportial to body size (an argument that rests on isomorphy), the reproduction rate is also expected to be proportional to body weight. They observed that reproduction rate tends to scale with body weight to the power somewhat less than one for several other marine invertebrate species, and used their observation to identify a constraint on body size for brooding inside the body cavity. The DEB theory gives no direct support for this constraint; an allometric regression of reproduction rate against body weight would result in a scaling parameter between 2/3 and 1, probably close to 1, depending on parameter values.

The maximum (mean) reproduction rate for ectotherms of maximum volume $V_m = (\dot{v}/g\dot{m})^3$ amounts to

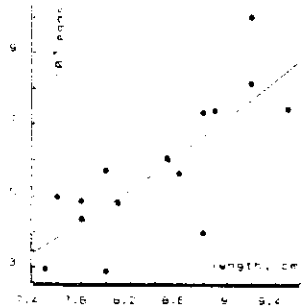
$$\dot{R}_m = \frac{q}{e_0}(1 - \kappa) g \dot{m} (1 - V_p/V_m) \tag{3.51}$$

Figure 3.19: Reproduction rate as a function of body length for two randomly selected species. The data sources and DEB-based curves are indicated. The parameter that is multiplied by $L^3$ in both fits has been guestimated on the basis of common values for the maintenance rate coefficient and the energy conductance, with a shape coefficient of $d_m = 0.1$ for the goby and of $d_m = 0.5$ for the frog. Both the other parameters represent least squares estimates.

rock goby *Gobius paganellus* [487]
$0.120(L^2 + 0.0026L^3 - 16.8)$



green frog *Rana esculenta* [280]
$0.124(L^2 + 0.0128L^3 - 32.5)$



All these expressions only refer to mean reproduction rates. Individuals are discrete units, which implies the existence of a buffer, where the energy allocated to reproduction is collected and converted to eggs at the moment of reproduction. The translation of reproduction rate into number of eggs in figure 3.19 assumes that this accumulation is over a period of one year. The energy content of the buffer is denoted by $E_R$.

Some species reproduce when enough energy for a single egg has been accumulated, others wait longer and produce a large clutch. There is considerable variation in the way the reproduction buffer is handled. If the reproduction buffer is used completely, the size of the clutch equals the ratio of the buffer content and the energy costs for one young, $qE_R/E_0$, where $E_0$ is given in (3.26). This resets the buffer. So after reproduction $E_R = 0$ and further accumulation continues from there. That is to say, if the bit of energy that was not sufficient to build the last egg will be lost. Fractional eggs do not exist. In the chapter on population dynamics, {171,207}, I will show that this uninteresting detail substantially affects dynamics at low population growth rates, which occur most frequently in nature. If food is abundant, the population will evolve rapidly to a situation in which food per individual is sparse and reproduction low if harvesting processes do not prevent this.

The strategies for handling this buffer are species-specific and are affected by environmental variables. Most species are able to synchronize the moment of reproduction with seasonal cycles such that food availability just matches the demand of the offspring. Clutch size in birds typically relates to food supply during a two-month period prior to egg laying and tends to decrease if breeding is postponed in the season [475]. The laying date is determined by a rapid increase in food supply. Since feeding conditions tend to improve during the season, internal factors must contribute to the regulation of clutch size. These conclusions result from an extensive study of the energetics of the kestrel *Falco tinnunculus*



*lus* by Serge Daan and co-workers [171,458,474]. I see reproductive behaviour like this, for species that cease growth at an early moment in their life span, as variations on the general pattern that the DEB theory is aiming to grasp. Aspects of reproduction energetics for species that cease growth, are worked out under the heading 'imago' on {151}.

### 3.10.2 Division

If propagation is by division, the situation is comparable to the juvenile stage of species that propagate via eggs. The volume at division corresponds to the transition from juvenile to adult, so $V_d = V_p$. Donachie [175] pointed out that in fast growing bacteria the initiation of DNA duplication occurs at a certain volume $V_p$, but it requires a fixed and non-negligible amount of time $t_D$ for completion. This makes the volume at division, $V_d$, dependent on the growth rate, so indirectly on substrate density, because growth proceeds during this period. The mechanism (in eukaryotic somatic cells) of division at a certain size is via the accumulation of cdc25 and cdc13 mitotic inducers, which are produced coupled to cell growth. (The name for the genes 'cdc' stands for cell devision cycle.) If these inducers exceed a threshold level, $p34^{cdc2}$ protein kinase is activated and mitosis starts [493,498]. During mitosis, $p34^{cdc2}$ is deactivated and the concentration of inducers resets to zero. This mechanism indicates that for shorter inter-division periods, the cell starts a new DNA duplication cycle when its volume exceeds $2V_p$, $4V_p$, $8V_p$ etc. The inter-division time for *Escherichia coli* can be as short as 20 minutes under optimal conditions, while it takes an hour to duplicate the DNA. In a dynamic environment, where (3.36) and (3.37) are supposed to apply, the implementation of this trigger is not simple. At constant substrate densities, the scaled cell length at division, $l_d \equiv (V_d/V_m)^{1/3}$, and the division interval, $t(l_d) \equiv t_d$, can be obtained directly. When $i$ is an integer such that $2^{i-1} < V_d/V_p \leq 2^i$, $V_d$ can be solved from

$$t_D = it(V_d) - t(2^{i-1}V_p) \tag{3.52}$$

Figure 3.20 illustrates the derivation.

The volume at division $V_d$ can be found numerically when (3.15), (3.35) or (3.39) is substituted for $t(V)$ into (3.52), for isomorphs, filaments or rods, respectively.

## 3.11 Respiration

Respiration, i.e. the use of oxygen or the production of carbon dioxide, can be taken to represent the total metabolic rate in an organism. Initially, eggs hardly use oxygen, but oxygen consumption rapidly increases during development: see figure 3.21. In juveniles and adults, oxygen consumption is usually measured in individuals that have been starved for some time, to avoid interpretation problems related to digestion. (For micro-organisms this is not possible without a substantial decrease of reserves.) As mentioned in the introductory chapter, the conceptual relationship between respiration and use of energy has already undergone some changes in history. Von Bertalanffy identified it with anabolic processes, while the Scope For Growth concept, {43}, relates it to catabolic processes. In the DEB model, the most natural identification is with the total use of energy from the reserves,

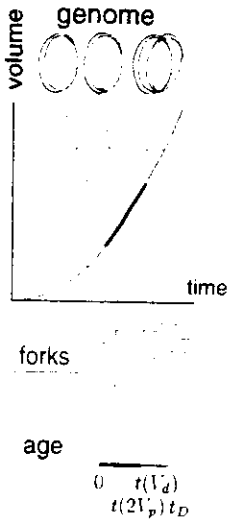



Figure 3.20: A schematic growth curve of a cell, where the fat part is used in steady state. This is the situation for $i = 2$, the number of forks switching between 1 and 3. If $V_d/V_p = 2^i$, equation (3.52) reduces to $t_D = it(2^iV_p) = it(V_d)$, with $t(2^{i-1}V_p) = 0$, which means that the time required to duplicate DNA is exactly $i$ times the division interval. So, during each cell cycle, a fraction $i^{-1}$ of the genome is duplicated, which implies that $2^i - 1$ DNA duplication forks must be visible during the cell cycle. At the moment that the number of forks jumps from $2^i - 1$ to $2^{i+1} - 1$, the cell divides and the number of forks resets to $2^i - 1$. This is obviously a somewhat simplified account, as cell division is not really instantaneous. If $V_d/V_p \neq 2^i$, the age of the cell at the appearance of the new set of duplication forks somewhere during the cell cycle is $t(2^{i-1}V_p)$, which thus has to be subtracted from $it(V_d)$ to arrive at the genome duplication time.

(3.42), with a fixed conversion factor from oxygen to energy use. This is consistent with the assumption of a constant chemical composition for the reserves. It is also consistent with the observation that respiration rate increases with reserve density {389}, while reserves themselves do not use oxygen. Moreover, it explains the reduction of respiration during starvation; see {128}.

At constant food density, the proportionality between respiration and use of energy from the reserves implies that the respiration rate can be written as a weighted sum of a surface area and a volume. Figure 2.9 shows that it is indistinguishable from the standard allometric relationship. Apart from avoiding dimension problems, the surface area related costs for heating in endotherms, which have given rise to Rubner's surface law, also fit much more naturally. As mentioned above, this point of view solves the long standing problem of why the volume-specific respiration of ectotherms decreases with increasing size, when organisms of the same species are compared. This problem has been identified as one of the central problems of biology {777}. Many theories have been proposed, see e.g. {590} for a discussion, but all use too specific arguments to be really satisfactory: heating (but many species are ectothermic), muscle power (but movement costs are relatively unimportant), gravity (but aquatic species escape gravity). Peters {542} even argued to cease looking for a general explanation. The DEB theory, however, does offer a general explanation: the overhead for growth. A comparison of different species will be covered in a later chapter, {217}, where it will be shown that interspecies comparisons work out a bit differently.

The proportionality of respiration with the utilization rate is not, however, completely free from conceptual problems. This mapping gives double counts of energy flows at two places: energy that is fixed in structural biomass during growth and energy reserves deposited in eggs (by females). It is essential to realize that the costs for synthesis $[G]$ include overheads. So $[G]$ is larger than, and I think much larger than, the energy content of the structural biomass. On the other hand, the energy content of organisms, which is



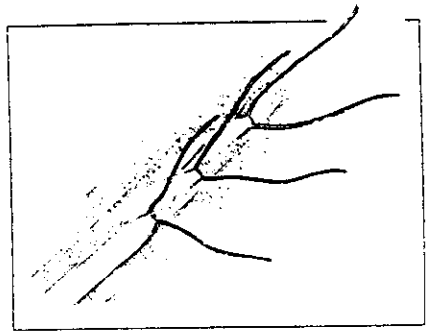


Figure 3.21: The water stick insect *Ranatra linearis* deposits its eggs in floating decaying plant material, where oxygen availability is usually poor. The eggs are easily spotted by the special respiratory organs that peek out of the plant. Just prior to hatching, eggs typically need a lot of oxygen, cf. figure 3.14.

frequently measured {147,527,540,512,572}, includes energy reserves. These two problems complicate the interpretation of such measurements in terms of energy parameters.

The use of respiration measurements to estimate the parameters of the DEB model is limited. Respiration is taken proportional to utilization, so that it follows from (3.42) that the respiration rate is proportional to $V + V^{2/3}V_m^{1/3}(l_h + g)$ at constant food density. Respiration is thus a weighted sum of volume and surface area. If respiration data are available for different body sizes of a particular species of ectotherm, so that $l_h = 0$, the ratio of the weight coefficient for volume$^{2/3}$ and that for volume stands thus for $gV_m^{1/3} = \frac{g}{m} = \frac{[A_m][G]}{[E_m][M]}$, which has the dimension of length. Four original parameters are combined into this compound parameter. The two coefficients of the ratio are negatively correlated in a statistical sense, which implies that respiration data give poor access to the value of the compound parameter.

The maintenance rate coefficient $\dot{m}$ can be estimated easily if growth data together with respiration data are collected at a constant food density. The $\kappa$-rule implies that the respiration rate of ectotherms is proportional to energy allocation to growth plus maintenance, so according to (3.11) the respiration rate is proportional to $\frac{d}{dt}V + \dot{m}V$. The observation that respiration is proportional to a weighted sum of volume and change in volume goes back to the study of Smith {670} in 1957 on eggs of salmon. At constant food density, the change in volume is of the von Bertalanffy type, which makes respiration proportional to $3\dot{r}(V_\infty^{1/3}V^{2/3} - V) + \dot{m}V$. This gives five parameters to be estimated from two data sets on respiration and growth: $V_0$, $V_\infty$, $\dot{r}$, a proportionality constant for respiration and the maintenance rate coefficient, $\dot{m}$. This gives 2.5 parameters per data set, which seems acceptable if the scatter is not too large.

In the section on mass-energy coupling, {192}, it will be shown how the respiration rates for micro-organisms can be tied to energy fluxes in a more rigorous way. I expect that the overheads for growth are much smaller for them, compared to animals, but I cannot substantiate this.

## 3.12 Aging

Since age is not a state variable, the steady shift in properties due to the poorly understood process of aging is only of secondary relevance to the DEB model. In a number of situations,

however, one should consider life span which has well recognized roots in energetics. The frequently observed correlation between life span and the inverse volume-specific metabolic rate for different species (see e.g. [638]) has guided a lot of research. The impressive work of Finch [219] gives well over 3000 references. Animals tend to live longer at low food levels than at high ones. The experimental evidence, however, is rather conflicting on this point. For example, Ingle [349] found such a negative relationship, while McCauley [463] found a positive one for daphnids. This is doubtlessly due to the fundamental problem that death can occur for many reasons, such as food related poisoning, that are not directly related to aging. Some species such as salmon, octopus, *Oikopleura* die after (first) reproduction, cf. {149}. This cause, like many other causes of death, does not relate to aging. On approaching the end of the life span, the organism usually becomes very vulnerable, which complicates the interpretation of the life span of a particular individual in terms of aging. Experiments usually last a long time, which makes it hard to keep food densities at a fixed level and to prevent disturbances.

In a first naïve attempt to model the process of aging, it might seem attractive to conceive the senile state, followed by death as the next step in the sequence embryo, juvenile, adult, and then tie it to energy investment into development just as has been done for the transitions to the juvenile and adult stages. This is not an option for the DEB model, since at sufficiently low food densities, the adult state is never entered, even if the animal survives for nutritional reasons. This means that it would live for ever, as far as aging is concerned. Although species exist with very long life spans (excluding external causes of death [219]), this does not seem acceptable. Attempts to relate hazard rates directly to the accumulation of hazardous compounds formed as a spin off of respiration, such as oxidized lipids, have failed to produce realistic age-specific mortality curves: the hazard rate increases too rapidly for a given mean life span. See [388,518] for reviews on the role of secondary products from metabolism in aging. The same holds for the hazard tied to damage to membranes, if this damage accumulates at a rate proportional to volume-specific respiration. Accurate descriptions of survival data where aging can be assumed to be the major cause of death seem to call for an extra integration step, which points to DNA.

It has been suggested that free radicals, formed as a spin off of respiration, cause irreparable damage to the DNA in organisms [288,287,719]. The specific activity of antioxidants correlates with life span within the mammals [219]. The structure of the antioxidant enzyme manganese superoxide dismutase has recently been solved [621]. Although too unspecific to be of much help for molecular research, for energetics purposes the free radical hypothesis specifies just enough to relate the age-specific survival probability, and so life span, to energetics. The idea is that the hazard rate is proportional to damage density, which accumulates at a rate proportional to the concentration of changed DNA, while DNA changes at a rate proportional to utilization rate. Although it is not yet possible to draw firm conclusions on this point, this mechanism does provide the extra integration step that is required for an accurate description of data. It is further assumed that the cells with changed DNA do not grow and divide, while the density of affected cells is reduced owing to the propagation of the unchanged cells. This assumption is supported by the recent identification of gene chk1 [748], whose products are involved in the detection of DNA damage:



damaged DNA prevents entry into mitosis by controlling the activity of the protein that is produced by cdc2, cf. {103}. Because of the uncertainty in the coupling with molecular processes, I prefer to talk about damage and damage inducing compounds, rather than wrong proteins (or their products) and DNA. This idea can be worked out quantitatively as follows.

Let $[Q] \equiv Q/V$ denote the concentration of damage inducing compounds (changed DNA), which accumulate from value 0 in an embryo of age 0. Its dynamics can be obtained via the chain rule for differentiation: $\frac{d}{dt}[Q] = V^{-1}\frac{d}{dt}Q - [Q]\frac{d}{dt}\ln V$ and amounts to

$$\frac{d}{dt}[Q] = d_Q\frac{\dot{C}}{V} - [Q]\frac{d}{dt}\ln V \tag{3.53}$$

where $d_Q$ is the contribution of the volume-specific utilization rate to the compounds per unit of energy. The second term stands for the dilution through growth, where cells with changed DNA become mixed with cells with unchanged DNA.

Substitution of (3.11) gives for ectotherms

$$\frac{d}{dt}[Q] = \frac{d_Q}{\kappa}[G]\frac{d}{dt}\ln V + \frac{d_Q}{\kappa}[M] - [Q]\frac{d}{dt}\ln V \tag{3.54}$$

The concentration of damage inducing compounds as a function of time for ectotherms thus equals

$$[Q](t) = \frac{d_Q}{\kappa}[G]\left(1 - \frac{V(0)}{V(t)}\right) + \frac{d_Q}{\kappa}\frac{[M]}{V(t)}\int_0^t V(t_1)\,dt_1 \tag{3.55}$$

As explained in the section on embryonic growth, {83}, the initial volume, $V(0)$, is infinitesimally small. The accumulated damage during the embryonic stage, however, is usually negligibly small. The high generation rate of damage inducing compounds is balanced by the high dilution rate through growth. The fact that the embryonic period is usually a very small fraction of the total life span ensures that one does not lose much information by starting from the moment of hatching.

Damage (wrong protein) accumulates at a rate proportional to the concentration of damage inducing compounds, so the damage density is proportional to $\int_0^t [Q](t_1)\,dt_1$. The hazard rate, $h(t)$, is finally taken to be proportional to the damage density, which leads to:

$$h(t) = \tilde{p}_a\int_0^t\left(1 - \frac{V(0)}{V(t_2)} + \frac{\dot{m}}{V(t_2)}\int_0^{t_2} V(t_1)\,dt_1\right)dt_2 \tag{3.56}$$

The proportionality constant $\tilde{p}_a$, here called the aging acceleration, absorbs both proportionality constants leading to this formulation of the age dependent hazard rate and is proportional to $d_Q[G]/\kappa$. This most useful property means that only a single parameter is necessary to describe the aging process.

The hazard rate relates to the survival probability according to the differential equation $\frac{d}{dt}\text{Prob}\{\underline{a}_t > t\} = -\text{Prob}\{\underline{a}_t > t\}h(t)$ or $h(t) = -\frac{d}{dt}\ln\text{Prob}\{\underline{a}_t > t\}$. The survivor probability is thus

$$\text{Prob}\{\underline{a}_t > t\} = \exp\{-\int_0^t h(t_1)\,dt_1\} \tag{3.57}$$

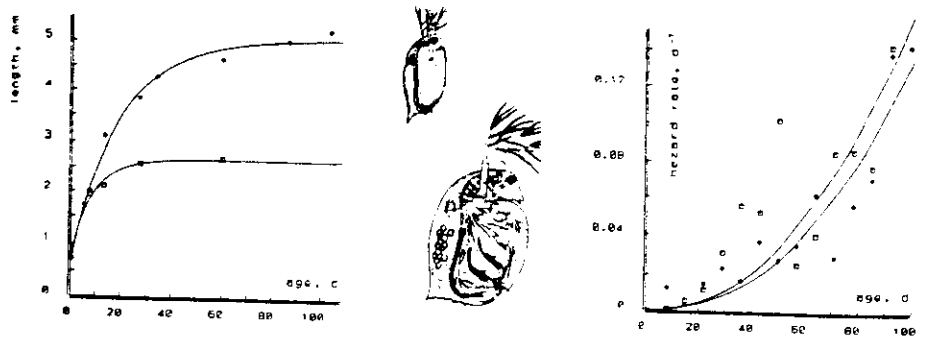


Figure 3.22: The growth curves of female (◇) and male daphnid (□)*Daphnia magna* at 18 °C and the observed hazard rates. Data from MacArthur and Baillie [445]. The growth curves are of the von Bertalanffy type with common length at birth. The hazard rates are fitted on the basis of the damage genesis discussed in the text, with a common aging acceleration of $2.587 \times 10^{-5}$ $d^{-2}$. The difference in the hazard rates is due to the difference in ultimate lengths.

The mean life span equals $\mathcal{E}\underline{a}_t = \int_0^\infty \text{Prob}\{\underline{a}_t > t\}\, dt = \int_0^\infty \exp\{-\int_0^t \dot{h}(t_1)\, dt_1\}\, dt$. This hazard rate thus ties aging to energetics, which explains for instance why dormancy prolongs life span, cf. {131}.

Figure 3.22 shows that the fit with experimental data for male and female daphnids is quite acceptable, in view of the fact that the combined hazard curves have only one free parameter $\ddot{p}_a$ (so half a parameter per curve). The differences in survival probability of male and female daphnids can be traced back to difference in ultimate size (i.e. in the surface area-specific maximum assimilation rate $\{\dot{A}_m\}$).

It is instructive to compare this model with that of Weibull where $\text{Prob}\{\underline{a}_t > t\} \equiv \exp\{-\int_0^t \dot{h}(t_1)\, dt_1\} = \exp\{-(\dot{p}_G t)^3\}$. The model was first proposed by Fisher and Tippitt [220] in 1928 as a limiting distribution of extreme values, and Weibull [757] has used it to model the failure of a mechanical device composed of several parts of varying strength, according to Elandt-Johnson and Johnson [198]. The (cumulative) hazard increases allometrically with time. Like many other allometrically based models for physiological quantities, it is attractively simple, but fails to explain, for instance, why the sexes of *Daphnia* have different shape coefficients $\beta$ [415]. As long as both parameters of the Weibull model can be chosen freely, i.e. if only one data set is considered, it will be hard to distinguish it from the DEB-based model. See figure 3.23. The maintenance rate coefficient in the fit is here considered as a free parameter, so both curves then have two free parameters. This is done because the available estimate for the maintenance rate coefficient on the basis of egg development as reported in table 3.1 is rather far out of range. The resulting estimate of $\dot{m} = 0.073$ $d^{-1}$ at 20 °C is much more realistic, which in itself lends strong support to my interpretation. It can be shown that the Weibull model with shape parameter 3 results if the growth period is short relative to the mean life span, {154}.

The Gompertz model for survival $\text{Prob}\{\underline{a}_t > t\} = \exp\{\beta(1 - \exp\{\dot{p}_G t\})\}$ is also frequently used as a model for aging; see e.g. [779]. It can be mechanistically underpinned by a constant and independent failure rate for a fixed number of hypothetical critical ele-



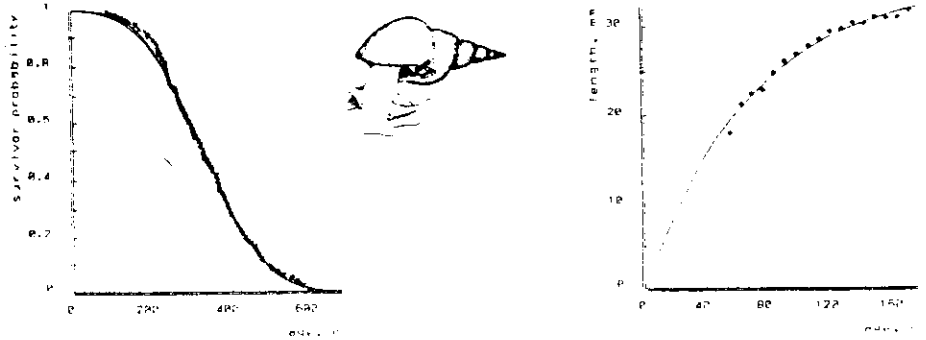


Figure 3.23: The survival probability and the growth curve of the pond snail *Lymnaea stagnalis* at 20 °C. Data from Slob and Janse [664] and Bohlken and Joosse [76,788]. The fitted growth curve is the von Bertalanffy one, giving an ultimate length of 35 mm and a von Bertalanffy growth rate of $\dot{\gamma} = 0.015$ $d^{-1}$. The survival curve was used to estimate both the maintenance rate constant, $\dot{m} = 0.073$ $d^{-1}$ and the aging acceleration $\ddot{p}_a = 2.563 \times 10^{-6}$ $d^{-2}$. The Weibull curve with shape parameter 3.1 is plotted on top of the DEB model to show that both curves are hard to distinguish in practice.

ments. Death strikes if all critical elements cease functioning. The curvature of the survival probability then relates to the number of critical elements, which Witten [779] found to be somewhere between 5 and 15. Their nature still remains unknown. A property of this model is that the hazard rate does not approach zero for neonates (or embryos), which does not seem to be consistent with data [664]. Finch [219] favours the empirical description of aging rates given by the Gompertz model because its property of a constant mortality rate doubling time, $\dot{p}_G^{-1} \ln 2$, provides a simple basis for comparison of taxa.

The present formulation allows for a separation of the aging and energy based parameters. The estimation of the 'pure' aging parameter in different situations and for different species will hopefully reveal patterns that can guide the search for more detailed molecular mechanisms, however, many factors may be involved, cf. {154}. It has been suggested in the literature that the neural system may be involved in setting the aging rate. The fact that brain weight in mammals correlates very well with respiration rate [330], makes it difficult to identify factors that determine life span in more detail. The mechanism may be again via the neutralization of free radicals.

An indication for this pathway can be found in the age-specific survival probability for humans, see figure 3.24, which can be described well by a Weibull distribution with shape parameter 6.8. Compared with the data on ectotherms, we have here an extremely low hazard rate for the young ages, which increases rapidly after the age of 50 years. This pattern suggests that the system that is involved in the neutralization of free radicals is itself subjected to aging, while for ectotherms it is not necessary to build in this complication. As explained in the next chapter, {151}, a constant neutralization probability, combined with low mortality during growth, leads to survival curves which are close to the Weibull

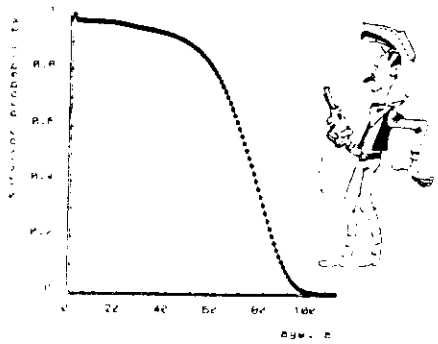


Figure 3.24: The survival curve for humans: white males in the USA in the period 1969-1971. Data from Elandt and Johnson [198]. The fitted empirical survival curve is $q\exp\{-\dot{p}t-(\dot{p}_W t)^\beta\}$, with $q = 0.988$, $\dot{p} = 0.0013$ a$^{-1}$, $\dot{p}_W = 0.01275$ a$^{-1}$ and $\beta = 6.8$. The parameter $q$ relates to neonate survival and $\dot{p}$ to death by accident.

curve with shape parameter 3. Aging as a result of free radicals is partially supported by the observation that the life spans of both ectotherms and endotherms correlate well with the specific activity of antioxidants [219]. It should be noted that if we compare an endotherm with a body temperature of 40 °C with that of an otherwise similar ectotherm at 20 °C, we should expect a 10 times shorter life span, on the basis of an Arrhenius temperature of 10000 K. Endotherms, therefore, have a problem to solve, which possibly involves additional mechanisms to remove free radicals.

One of the many questions that remain to be answered is how aging proceeds in animals that propagate by division rather than by eggs. Unlike eggs, they have to face the problem of initial damage. It might be that such animals have (relatively few) undifferentiated cells that can divide and replace the damaged (differentiated) ones. A consequence of this point of view is that the option to propagate by division is only open to organisms where the differentiation of specialized cells is not pushed to the extreme. If aging affects all cells at the same rate, it becomes hard to explain the existence of dividing organisms. This is perhaps the best support for the damage interpretation of the aging process. Theories that relate aging, for instance, to the accumulation of compounds as an intrinsic property of cellular metabolism, should address this problem. The same applies to unicellulars. If accumulated damage carries over to the daughter cells, it becomes hard to explain the existence of this life style. The assumption of the existence of cells with and without damage seems unavoidable. Organisms that live in anaerobic environments cannot escape aging, because other radicals will occur that have the same effect as oxygen. Note that if one follows the fate of each of the daughter cells, this theory predicts a limited number of divisions until death occurs, so that this event itself gives no support for aging theories built on cellular programming. Only the variation in this number can to some extent be used to choose between both approaches. The present theory can be worked out quantitatively for unicellulars as follows.

Since unicellulars cannot dilute changed DNA with unchanged DNA and cannot compensate for its effect, the hazard rate for unicellulars must equal $\dot{h}(t) = d_Q\dot{C}/V$, where $d_Q$ is the net hit frequency per unit of energy density. (Note that the range of the cell volume is $(V_d/2, V_d)$, so that the volume-specific respiration rate is restricted, while for embryos,



lution by growth solves this problem for embryos.) From (3.45) it follows that the hazard rate for filaments is

$$\dot{h}(E) = d_Q(\dot{\nu}+\dot{m})\frac{g[E]}{g+[E]/[E_m]} \quad \text{, thus} \tag{3.58}$$

$$\dot{h}(e) = \dot{p}_a e\frac{1+g}{e+g} \tag{3.59}$$

where $\dot{p}_a \equiv d_Q[E_m]g\frac{\dot{\nu}+\dot{m}}{1+g}$ represents the maximum aging rate and $e \equiv [E]/[E_m]$ the reserve density as a fraction of the maximum capacity. At constant substrate densities, the scaled energy reserve density, $e$, equals the scaled functional response, $f$, so the hazard rate is constant and independent of the age of the filament. The hazard rate for rods is likewise found from (3.44):

$$\dot{h}(e,l) = \dot{p}_a e\frac{1+g}{e+g}\frac{((l_d/l)^3-1)\delta/3+1+l_d/g}{1+l_d/g} \tag{3.60}$$

For the hazard rate of unicellular isomorphs we obtain from (3.42)

$$\dot{h}(e,l) = \dot{p}_a e\frac{1+g}{e+g}\frac{1+g/l}{1+g/l_d} \tag{3.61}$$

In contrast to filaments, rods and isomorphs experience a reduction of the hazard rate during the cell cycle.

If DNA is changed, the cell will cease functioning. This gives a lower boundary for the (population) growth rate because the population will become extinct if the division interval becomes too long. To prevent extinction (in the long run) the survival probability to the next division should be at least 0.5, so the lower boundary for substrate density can be found from $\text{Prob}\{a_j > t_d\} = \exp\{-\int_0^{t_d}\dot{h}(t)\,dt\} = 0.5$. Substitution of (3.60) and (3.39) leads to the lower boundary for the substrate density for rods, which must be found numerically. It is tempting to relate this aging mechanism, which becomes apparent at low substrate densities only, to the occurrence of stringent responses in bacteria, as described by, for example, Cashel and Rudd [122]. This will be discussed further when populations are considered. {165}.

It is intriguing to realize that the present mechanism for aging implies that organisms use free radicals to change their DNA. Although most changes are lethal or adverse, some can be beneficial to the organism. Using a selection process, the species can exploit free radicals for adaptation to changing environments. By increasing the specific activity of antioxidants, a species can prolong the life span of individuals in non hostile environments, but it reduces its adaptation potential as a species if the environment changes. This trait defines an optimal specific activity for antioxidants that depends on the life history of the organism and the environment. Large body size, which goes with a long juvenile period, as will be discussed on {234}, requires efficient antioxidants to ensure survival to the adult state. It implies that large bodied species have little adaptation potential, which is further reduced by the long generation time; this makes them vulnerable from an evolutionary perspective. It is possibly one aspect of the extinction of the dinosaurs,

although not all of them were large and they may have been endothermic. Endotherms appear to combine a high survival probability of the juvenile period with a high aging rate, thus having substantial adaptation potential during the reproductive phase; they reach this by a reduction of the efficiency of antioxydants during puberty.

The present formulation assumes that growth ceases as soon as DNA is changed. The background is that many genes are involved in the synthesis of one or more compounds that are essential to structural body mass and so to growth. A few genes are involved in suppressing unregulated growth of cells in multicellular organisms. If such genes are affected, tumors can develop. This theory can, therefore, also be used to work out the age-dependent occurrence rate of tumors as well as the growth rate of tumors, cf. {252}.

The energy parameters can be tied to the accumulated damage to account for the well known phenomenon that older individuals eat less and reproduce less than younger ones with the same body volume. Senescence can be modelled this way. This role of age in energetics is not worked out here to keep the model as simple as possible.

## 3.13 Genetics and parameter variation

The parameter values undoubtedly have a genetically determined component, which can to some extent be modulated phenotypically. As has hopefully been made clear, the processes of feeding, digestion, maintenance, growth, reproduction and aging are intimately related. They involve the complete cellular machinery. Although mechanisms for growth which involve just one gene, have been proposed {176}, the DEB theory makes it likely that thousands are involved. This restricts the possibilities of population genetic theories to deal with auxiliary characters that do not have a direct link to energetics. (This is not meant to imply that such theories cannot be useful for other purposes.) In the context of quantitative genetics, some instructive points should be mentioned here. For this purpose a particular property of the DEB model, which I call the invariance property (just to have a name to refer to), should be discussed first. This property is at the basis of body size scaling relationships to be discussed later. These relationships express how species-specific characters depend on body size.

The invariance property of the DEB model is that two species with parameter sets that differ in a very special way behave identically with respect to energetics as long as food density is strictly constant. So they will have exactly the same energy dynamics, volume and reproduction ontogenies, and so on, for all life stages. The derivation of the relationship between both parameter sets is simple when two individuals are compared with the same body volume and with a maximum surface area-specific ingestion rate that differ by a factor $z$, so $\{\dot{I}_m\}_2 = z\{\dot{I}_m\}_1$. To behave identically, the ingestion rates must be equal: $\dot{I}_2 = \dot{I}_1$. Since their volumes are equal, $V_2 = V_1$, (3.2) implies that $f_2 = f_1/z$ or $K_2 = zK_1 + (z-1)X$. Since the assimilation rates must be the same, $\dot{A}_2 = \dot{A}_1$, it follows that $\{\dot{A}_m\}_2 = z\{\dot{A}_m\}_1$. They must have the same storage dynamics, so (3.7) implies $[E_m]_2 = z[E_m]_1$. Identical growth defined by (3.12) implies that the other parameters should be the same, so $V_{b,2} = V_{b,1}$, $V_{p,2} = V_{p,1}$, $V_{h,2} = V_{h,1}$, $\kappa_2 = \kappa_1$, $[\dot{M}]_2 = [\dot{M}]_1$, $[G]_2 = [G]_1$ and $\ddot{p}_{a,2} = \ddot{p}_{a,1}$.



If food density is not strictly constant, but fluctuates a little, both species behave in a different manner as far as energy is concerned. This is due to the non-linear relationship between the scaled functional response $f$ and food density $X$. The change of $f$ with respect to $X$ is $\frac{d}{dX}f = K(K+X)^{-2} = (1-f)^2/K$. So if $f$ approaches 1, the change in the ingestion rate, and so in the energy reserve density, becomes negligibly small. This overall homeostasis is probably selectively advantageous, because it implies that regulation systems have a much easier job to coordinate the various processes of energy allocation, which allows for optimization. The mechanism is not unlike the restriction of the tolerance range for temperature of enzymes of homeotherms relative to heterotherms. The invariance property has an interesting consequence with regard to selection processes. At a constant food density, the (constant) surface area-specific ingestion rate, surface area-specific assimilated energy, and reserve energy density can be regarded as achieved physiological characters. Small fluctuations in food density drive selection to a (genetic) fixation of these characters as the maximum possible ones: $\{\dot{I}_m\} \rightarrow \{\dot{I}\}$, $\{\dot{A}_m\} \rightarrow \{\dot{A}\}$ and $[E_m] \rightarrow [E]$. This phenomenon is known as 'dwarfing'.

The parameter values for different individuals are likely to differ somewhat. Differences in ultimate volume at constant food density testify to this basic fact. To what extent this has a genetic basis is not clear, but the heredity of size in different races of dogs and transgenic mice and turkeys reveals the genetic basis of growth and size. Since only a tiny fraction of available DNA in eukaryotic cells is in active use, one can easily imagine that changes in the pieces that are used, or in the intensity with which the active parts are used, can result in changes in energy parameters. These regulation processes can be subjected to phenotypic influence and to factors located in the cytoplasm, and so to maternal effects. An important statistical consequence of this point of view is that parameter estimates can in principle no longer be based on means: the mean of von Bertalanffy curves with different parameters is not a von Bertalanffy curve. This problem obviously grows worse with increasing scatter. The modelling of parameter variation can easily introduce a considerable number of new parameters. To select just one or two parameters to solve this problem seems arbitrary. An attractive choice might be to conceive the factor $z$, just introduced, to be a stochastic variable, which couples four energy parameters. This introduces stochasticity only at fluctuating food densities.