Part II

Optimization Models

4

Optimization Models Based on Size

4.1 Size Matters

Hamilton did not prove that senescence is inevitable. Furthermore, it seems likely that the age-trajectory of mortality is largely shaped by optimization: only at advanced ages, when the bulk of total lifetime reproduction has been realized, might mutation accumulation play a role. So the question arises: could it be optimal for a species not to follow a senescent life-history strategy?

As Caswell argues, for many organisms "the age of an individual tells little or nothing about its demographic properties" [23, p. 39]. Often what is important is size or stage of development. He concludes that " [s]ize-dependent demography is probably the rule rather than the exception and is especially pronounced in species with a large range of adult body size as a result of indeterminate adult growth."

Trees, for example, continue growing over an extended period of their life, gaining strength, becoming more robust and thereby reducing their susceptibility to death. (If trees at sites exposed to wind are too tall, then their susceptibility to damage and death might increase: this, however, is a special case.) A larger size (tall, thick stem, more leaves, longer roots) lowers the risk of death and enables better access to resources (light, water, nutrients). Larger trees produce more seeds than smaller trees.

The same is true for some species of fish. For instance, in some species the young adult fish, still small, has only a few progeny and is the prey of bigger fish. Over time the fish grows large enough to become a predator itself, increasing its level of resources and lowering its own risk of death.

Small alligators are prey to a variety of predators including raccoons, otters, wading birds, and fish. But most dangerous to small alligators probably are predators of their own kind, the larger alligators. Large alligators also die of cannibalism and fight with each other (see http://myfwc.com/gators/facts.htm). An individual alligator's size and strength determines whether it receives or becomes an additional ration of food.

In this chapter, I hypothesize that candidate species for non-senescent life histories are species that continue to grow substantially after the onset of reproduction and for which size is strongly associated with continued survival and reproductive success. This appears to be the case for the plant *Plantago lanceolata* after seasonal effects are removed [167]. The study of *Plantago lanceolata* by Deborah Roach was the particular motivation for me to develop a general life-history model based on size rather than age to understand whether non-senescence is theoretically possible.

Evidence for size-dependent mortality is reported for herbaceous plants in general [44, 77, 173, 182], thistles in particular [162, 171], trees [88], corals [86] and fish [129, 154]. Sauer and Slade [174] also document the effect of body mass on reproduction and survival in vertebrates.

For some species mortality may not decrease as size increases: there may be no relation, or mortality may increase with size. In addition, it is important to note that, for some species, larger size may not *cause* lower mortality. Larger size may have co-evolved with lower mortality, both resulting from some other aspect of the species' life history. For some species, for instance species like *Drosophila*, which exhibits discrete developmental stages rather than continuous growth, size may not be a key determinant of mortality. So a size-based model can shed light on the life history of only some species. But these species are "conceivable organisms" and may show non-senescent life-history strategies.

Size is the central state variable in the models I will develop in this chapter. Size determines mortality and fertility. Age enters the models only insofar as it takes time to grow – age itself does not matter. Using size as the state variable in these kinds of models is a first step to understanding whether *any* life history could be non-senescent. Note that the state variable size can be understood not only as physiological size but more generally as "size and strength". In Chap. 5, I develop a new model that is based on "vitality" rather than size.

4.2 A Size-Based Life-History Model

An optimal life history maximizes lifetime reproductive success. Accordingly, the energy available to an organism, which is always limited, has to be distributed among the basic processes of life: reproduction, maintenance and growth. How evolution solves this allocation problem determines the optimal trajectory of growth and thereby the optimal trajectories of the main demographic schedules, mortality and fertility.

All forms of life have to deal with damage. Damage occurs all the time and is discarded or repaired continuously, sometimes fully, sometimes partially. Models that take into account the influence of damage on mortality and fertility can do so on the occurrence and/or on the disposal and repair side. Energy allocation problems imply that disposal and repair of damage decreases when more energy is allocated to reproduction and therefore less energy remains for processes of maintenance and growth. Models based on the concept of energy allocation do not necessarily account for where the damage comes from. Reproduction itself, for instance, can be a direct cause of damage. For simplicity, the model I am going to develop in this chapter will focus on the energy allocation trade-off between reproduction, on the one hand, and maintenance and growth on the other. That is, I treat growth and repair as elements of the same general process and I do not explicitly model damage resulting from reproductive activities. I assume that the occurrence of damage increases proportionally with size.

Models based on the concept of optimal energy allocation over the life cycle represent a fundamental approach in life history modeling. Early applications of this concept were developed more than three decades ago, for example by Cole [42], Gadgil and Bossert [62], Schaffer [175], Taylor et al. [192], and Leon [111]. More recent examples of the application of the concept of optimal energy allocation include Charlesworth [26], Perrin [152], Perrin and Sibly [153], Kozlowski [102], Chichon [37], Teriokhin [193], Charnov et al. [35], Mangel and Stamps[122], Kaplan and Robson [91], Chu and Lee [36] and Charlesworth [34].

Generally, such life-history models are driven by the trade-off between reproduction and growth. Depending on the particular research focus, growth is sometimes further differentiated into growth of acquisition structure, storage structure, defense structure, reproductive structure and/or cognitive functioning. The central quantity of interest is the fraction of energy allocated to reproduction, the reproductive effort of an individual.

Life history models based on the concept of reproductive effort have been studied intensively (for a review see Charlesworth [27, Section 5.3.4.]). Common to these models is the assumption of a direct, inverse relation between survival and reproduction, which is mediated by reproductive effort. One outcome of these models is that reproductive effort should increase with age [62, 175]. However, Fragen [61] produced some counter-examples and Charlesworth and Leon [31] derived conditions that would lead to a decreasing reproductive effort with age, i.e. to an increase in survival with age. These results illuminate the general pattern of how reproductive effort should change with age. But, as Charlesworth [27, p 214] put it: "The problem of solving for the optimal life history with this model is a formidable one."

My research aim is to study the variety of qualitative patterns of mortality and fertility over age. In particular, I wish to understand whether it can be optimal for mortality to be constant or to fall over an extended period of life after the onset of reproduction. Interestingly, optimal patterns of mortality and fertility were commonly found to be flat in numerical studies by Charlesworth [26]. In these studies, reproductive effort increased so slowly, that it appeared to be virtually constant.

The examples given in the previous section suggest that, for some species, mortality decreases with size and fertility increases with size. For species with continued growth that follow this pattern, constant or falling mortality after the onset of reproduction seems to be optimal, at least for some period of the lifespan. Consequently, the models developed in this chapter are designed to capture this simple pattern based on the state variable size.

In contrast to previous reproductive effort models, the link between survival and reproduction will be mediated by size. The important implication of this assumption is that an increase in reproductive effort does not necessarily lead to a decrease in survival, and a decrease in reproductive effort does not necessarily lead to an increase in survival. I will emphasize this point in Sect. 4.2.2.

Every organism has to cope with the ubiquitous processes of deterioration. This means that some of the energy invested in " growth" is needed to repair damage. Only what is left after the requirements of maintenance have been met can be used to increase current size. Size changes according to the balance between repair and damage. Thus, size in this framework can increase, decrease or remain constant and, consequently, mortality can increase, decrease or remain constant. Whether mortality increases or decreases is an outcome of the model and not an

assumption. This is a crucial feature, which distinguishes this model from previous models.

The importance of size is generally recognized [23, p.39]. A state-based model that assumes an inverse relation between state and mortality has been developed before by Perrin [152]. However, Perrin implicitly assumes a non-senescent life history because mortality cannot increase in his model. Perrin's approach does not account for the occurrence of damage and its possible repair. A model that incorporates damage and repair was developed by Kozlowski [102], Cichon [37] and Cichon and Kozlowski [39]. In their framework mortality does not depend on state but on accumulated damage and can, at best, remain constant. Complete repair of current damage is realized only if all energy is invested in repair, i.e. at the cost of zero reproduction. Otherwise mortality rises at a pace determined by reproductive effort. An increase in mortality is inevitable.

An innovative feature of the approach I will be taking is that I combine the inverse relation of mortality and size with the possible accumulation of damage and its repair. My research builds on and further develops Vaupel, Baudisch et al. [200]. Mangel and colleagues [120, 121] have recently developed other models in which mortality is the consequence of growth and metabolism and associated damage.

4.2.1 The General Optimization Problem

The general optimization problem can be formalized as follows. Let $\xi(a)$ denote the size (and strength) of an individual at age a. Let $\pi(a)$ denote the fraction of energy allocated to growth at that age. Assume that the change in size over age depends on investment $\pi(a)$ and size $\xi(a)$ but not on age a itself, i.e. that the trajectory of $\xi(a)$ is determined by the autonomous first-order differential equation

$$\frac{d\,\xi}{d\,a} \;\equiv\; \dot{\xi} \;=\; g(\xi(a), \pi(a)) \;. \tag{4.1}$$

Note that the dot indicates a change over age. Initial size is given by $\xi(0)$. From that size onwards, the age-trajectory of $\pi(a)$ determines the age-trajectory of $\xi(a)$.

The optimal trajectory of $\pi(a)$ over the life course is assumed to be the strategy that maximizes Darwinian fitness, measured as lifetime reproductive success, a functional of the form

$$\max\, R \;=\; \int_0^\infty \; f(\xi(a), \pi(a))\; da \;, \tag{4.2}$$

where $f(\xi(a), \pi(a))$ depends on the age-trajectories of mortality and fertility and hence on the age trajectories of $\xi(a)$ and $\pi(a)$. The age horizon is potentially infinite, but non-zero mortality insures that every individual has a finite lifespan.

The general optimization problem is described by the objective as given in (4.2) and the autonomous first-order differential equation as given in (4.1), which determines the change in size over age.

4.2.2 The Specific Optimization Problem

The change in size is determined by the fraction of energy invested in growth, $\pi(a)$. Energy is allocated between growth and maintenance on the one hand, and reproduction on the other hand. The fraction of energy allocated to reproduction, the reproductive effort, is captured by $1 - \pi(a)$, since in this model maintenance and growth are assumed to be paid out of the same budget. In accordance with the literature, the change in size is assumed to be inversely related to reproductive effort.

Larger size implies higher complexity, which is more costly to maintain. The rate of occurrence of new damage will be assumed to increase proportionally with size [101, 210]. A simple way of modeling deterioration is to assume a linear relation with size, i.e.

$$\delta(\xi(a)) \;=\; \delta_0 \;+\; \delta_1\, \xi(a)\;, \tag{4.3}$$

where $\delta_0 > 0$ and $\delta_1 > 0$ are constant parameters.

Size is assumed to change proportionally to the level of current size $\xi(a)$. This implies the assumption that available resources are proportional to size, an assumption also made by Charlesworth and Leon [31], Gadgil and Bossert [62] and Leon [111]. Furthermore I assume that the change in size is proportional to the difference between investment $\pi(a)$ and deterioration $\delta(\xi(a))$. Growth only occurs if investment exceeds the current rate of deterioration. Therefore, the change in size can be specified as

$$\frac{d}{da}\,\xi(a) \;=\; k\,(\pi(a) \;-\; \delta(\xi(a)))\,\xi(a) \tag{4.4}$$

where $k > 0$ is a constant scaling parameter. Initial size can be normalized by setting $\xi(0) \;=\; 1$. Substituting (4.3) into (4.4) yields the following logistic differential equation

$$\frac{d\xi(a)}{da} \;=\; k\,(\pi(a) \;-\; \delta_0 \;-\; \delta_1\,\xi(a))\,\xi(a)\;. \tag{4.5}$$

This equation captures the change in size and specifies the general function $g(\cdot)$ of (4.1).

Life starts off with growth. Then at some age some energy is invested in reproduction. This age at onset of reproduction (reproductive maturity) α is determined by the age when $\pi(a) < 1$ for the first time. Figure 4.1 depicts the age-trajectory of size during development. The curve is given by the solution to (4.5), namely

$$\xi(a) = \left(\frac{\delta_1}{1 - \delta_0} + \left(1 - \frac{\delta_1}{1 - \delta_0}\right) e^{-k\,(1-\delta_0)\,a} \right)^{-1}, \qquad (4.6)$$

taking into account that investment is constant at $\pi(a) = 1$ over that period and $\xi(0) = 1$. This logistic function has an upper limit of $(1 - \delta_0) / \delta_1$, which reflects the size an organism would eventually approach if it continues to spend all available resources on maintenance and growth. In size-based approaches, growth functions that have an

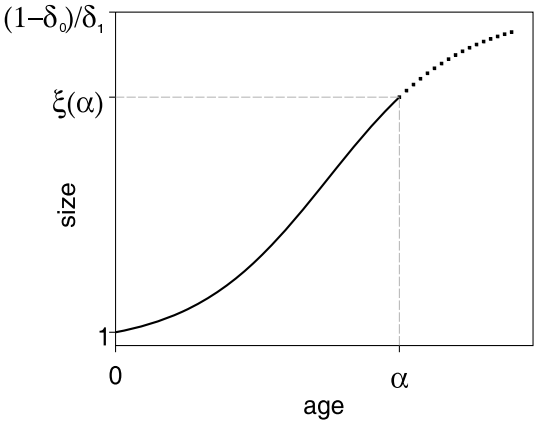

Fig. 4.1. Size $\xi(a)$ as a function of age a according to (4.5)

upper bound, such as the logistic function or the von Bertalanffy growth function, are frequently used, since size cannot increase indefinitely.

To ensure that the initial investment of $\pi_0 = 1$ actually leads to growth an additional restriction on the parameters in (4.3) is necessary. From (4.5) one gets

$$\left.\frac{d\xi(a)}{da}\right|_{a=0} = k\,(1 - \delta_0 - \delta_1) > 0$$

and hence

$$\delta_0 + \delta_1 < 1 \, . \tag{4.7}$$

This inequality concurrently guarantees that $\delta(\xi) < 1$.

The general function $f(\cdot)$ given in (4.2) can be specified by the product of the probability of surviving to age a, $l(a)$, and the amount of reproduction at that age, $m(a)$. The objective function is then specified by

$$\max R = \int_0^\infty l(a)\, m(\, \xi(a),\, \pi(a)\,)\; da \, . \tag{4.8}$$

The survival function $l(a)$ is determined by the trajectory of mortality up to age a via

$$l(a) = e^{-\int_0^a \mu(\xi(t))\, dt} . \tag{4.9}$$

The age-specific force of mortality, denoted by $\mu(a)$, is assumed to be inversely proportional to $\xi(a)$. As discussed in Sect. 4.1, I focus on species for which growth enhances future survival. A simple way to model mortality in this case is to let

$$\mu(a) = \frac{b}{\xi(a)} + c \, . \tag{4.10}$$

The constant parameter $b \geq 0$ captures the size-dependent, "intrinsic" component of death and the constant parameter $c > 0$ captures the size-independent, "extrinsic" component of death.

The model implies that, if no energy is allocated to growth, then size deteriorates exponentially and therefore mortality increases exponentially. However, whether it is optimal to invest all available energy in reproduction is an outcome of the model. An exponential increase in mortality is not a built-in property of the model. If mortality increases, it can do so at any pace, exponential being the extreme case. In the exponential case, the mortality function is the same as the Gompertz-Makeham function. Exponentially increasing mortality ("Gompertz Law") is frequently assumed in the literature, based on various empirical observations. The general structure of the mortality function is the same as that used by Perrin [152] (except for an exponent to size).

In accordance with the literature, I assume reproduction to be proportional to available resources (which are proportional to $\xi(a)$) and to the reproductive effort (in this model $(1-\pi(a))$). A simple way to specify reproduction is to assume a linear relation with reproductive effort; this approach was taken by Charlesworth [26], Perrin [152], Kozlowski

[102], Cichon [37] and Cichon and Kozlowski [39]. The maternity function, denoted by $m(a)$, is thus given by

$$m(a) \;=\; \varphi\,(1-\pi(a))\,\xi(a)\;. \tag{4.11}$$

Note that the constant, positive parameter φ can be adjusted to ensure that the optimal strategy yields a net reproduction rate $R = 1$. This implies that population density is assumed to affect lifetime reproductive success in a proportional manner. Note further that fertility and mortality are written as functions of age for purposes of brevity only. To be precise, $m(a) = m(\xi(a), \pi(a))$ and $\mu(a) = \mu(\xi(a))$.

The pleiotropic effects of size can be summarized as

$$\frac{d}{d\xi}\,\mu(\xi) \;<\; 0, \qquad \frac{\partial}{\partial\xi}\,m(\xi,\pi) \;>\; 0, \qquad \frac{d}{d\xi}\,\delta(\xi) \;>\; 0\;. \tag{4.12}$$

A larger size implies a lower risk of death, a higher reproductive potential but also a higher level of deterioration, which increases the costs of maintenance. Recall that the mediating effect of size between mortality, fertility and damage constitutes an important difference to previous models of reproductive effort, as emphasized at the beginning of this chapter. Equations (4.3), (4.4), (4.10) and (4.11) imply that an increase in reproductive effort $(1-\pi(a))$ does not necessarily lead to a reduction in survival. As long as the level of $\pi(a)$ does not fall below the level of damage $\delta(\xi(a))$, size does not shrink and therefore mortality does not increase. Conversely, a declining investment in reproduction does not lead to improved survival as long as the level of investment $\pi(a)$ is below the level of damage $\delta(\xi(a))$.

4.3 An Optimization Model that Leads to Non-senescence

The optimal solution is a trajectory over age. Therefore, this is a dynamic rather than a static optimization problem. Two main approaches can be distinguished: Bellman's dynamic programming approach [12] and Pontryagin's Maximum Principle [157]. Comprehensive treatments of dynamic programming methods applied to biological problems are given in Mangel and Clark [118] and Clark and Mangel [41] as well as Bulmer [16]. The Appendix to Mangel [119] shows how to connect dynamic state variable modeling with the ideas of classical demography and life history models.

4.3.1 The State Ratchet

Bellman's general way of thinking implies a feedback loop strategy. In any particular given state, make the best possible decision. This decision will steer the state to some subsequent level. Again, given this subsequent state, do the best you can do. An optimal trajectory of decisions can be found by beginning at the last possible state and working backwards. The most important precondition for this strategy is that decisions only depend on the current state and potential future gains and losses but not on the past.

In particular, at each size $\xi(a)$ the amount of energy invested in growth $\pi(\xi(a))$ at that size determines whether size increases, decreases or is maintained. Depending on this decision, size changes over age according to (4.2). The optimal trajectory of energy allocation to growth determines the optimal trajectory of size over age, which in turn determines the optimal age-trajectories of mortality and fertility.

Following Bellman's way of reasoning, the general nature of the optimal strategy can be understood intuitively. Assume each size is associated with a unique level of optimal investment and size changes continuously over age. Then each $\xi(a)$ is associated with a single $\pi^*(a)$ (the star indicating 'optimal') that determines whether size increases, decreases or is maintained.

Assume at a particular size $\xi(a)$ that the optimal investment results in an increase in size to $\xi(a^+) > \xi(a)$ at age $a^+ > a$. Assume further that, at the subsequent bigger size, it would be optimal to shrink. Then size would shrink to some lower value $\xi(a^{++}) < \xi(a^+)$ at age $a^{++} > a^+$. However, size is a continuous variable. In order to grow from $\xi(a)$ to $\xi(a^+)$ it must have been optimal to grow at each intermediate size between $\xi(a)$ and $\xi(a^+)$. Shrinking again from $\xi(a^+)$ to $\xi(a^{++}))$ would imply that this optimality is violated at each level of size between $\xi(a^+)$ and $\xi(a^{++})$. Each intermediate size would be associated with two optimal strategies instead of one, which is a contradiction.

This line of reasoning leads to an important result, which I will call "the state ratchet". If, for the optimization problem formulated above, an optimal solution exists and each state is associated with exactly one optimal strategy, then any continuous, optimal state trajectory must be a monotonic function over age. Consequently, if the state variable initially increases, it will never decrease and if the state variable initially decreases it will never increase. Since maintenance implies that state does not change, the optimal strategy, which is bound to state only, will not change over age. Therefore, if, for any finite interval, it is optimal to maintain the current state, it will be maintained forever.

The state ratchet has important consequences for any optimal life history in this framework. Since life begins with growth it can never be optimal to shrink. Size can only increase and then be maintained at some point. Since mortality is assumed to be inversely related to size, mortality can never increase. Senescence is impossible. Intriguingly, this simple approach challenges Hamilton's postulate of inevitable senescence. It is possible to overcome the state ratchet, as I will discuss in a later section of this chapter, but only by making the model more complicated. Let's first consider the basic model.

4.3.2 The Maximum Principle

Pontryagin's way of thinking involves planning the whole future at time zero, in contrast to Bellman's backward step-by-step approach. Optimizing all future decisions at time zero requires knowledge about how decisions, the "control variable(s)", influence the change in the state variable(s) over time. The change in state(s) over time is determined by the so called "equation(s) of motion", i.e. first order differential equations that capture the change in any state variable over age. For my particular problem the control variable is the investment in growth, $\pi(a)$. One state variable is size, $\xi(a)$. Equation (4.5) determines the corresponding equation of motion, the change in size over age.

As in Bellman's approach, there is an important precondition. The optimal decision at any age a should only depend on the current state and potential future gains and losses but not on previous ages. However, survival to age a, as given in (4.9), depends on the trajectory of mortality between age zero and age a. Therefore, survival must be treated as an additional state variable. Note that survival changes over time according to

$$\frac{d}{da}\, l(a) \;=\; -l(a)\,\mu(a) \tag{4.13}$$

with initial condition $l(0) = 1$. Equation (4.13) depicts the equation of motion for the second state variable, survival.

Pontryagin's Maximum Principle [157] associates a specific function with the optimal control problem stated above, the "Hamiltonian"

$$\begin{aligned} H(\,\xi, l, \pi, \lambda_1, \lambda_2\,) \;=\; & l(a)\, m(\xi, \pi) \\ & + \lambda_1(a)\,[\,k\,(\,\pi(a) \;-\; \delta_0 \;-\; \delta_1\,\xi(a)\,)\,\xi(a)\,] \\ & - \lambda_2(a)\, l(a)\,\mu(\xi)\;. \end{aligned} \tag{4.14}$$

The first term is the contribution of the objective function (given in (4.8) at age a: This term captures the current gains from a decision

$\pi(a)$ at states $\xi(a)$ and $l(a)$. The remaining terms are the weighted sum of the change in the state variables. The factors $\lambda_1(a)$ and $\lambda_2(a)$ are costate variables. Costate variables capture the values of a hypothetical additional unit of $\xi(a)$ and $l(a)$ respectively at age a, the "shadow price" of size and survival.

Conditions for an Optimum

The Maximum Principle requires that an optimal solution necessarily fulfills the following criteria:

- The Hamiltonian function is maximized with respect to the investment strategy. In general, if $H(\cdot)$ is differentiable, then

$$\frac{d}{d\pi} H(\cdot) \;=\; 0\;. \tag{4.15}$$

  In particular

$$H_\pi(\cdot) \;=\; l(a)\, m_\pi(\xi,\pi) \;+\; \lambda_1(a)\, k\, \xi(a) \;=0\;, \tag{4.16}$$

  the subscript π indicating the partial derivative. Clearly, if the Hamiltonian is linear in the control variable, then the maximum is attained at the boundaries of the feasible set for the control. Note that the last term dropped out. The shadow price of survival does not influence the maximum of the Hamiltonian.
- Furthermore the "adjoint equations"

$$\frac{d}{d\xi} H(\cdot) \;=\; -\frac{d}{da}\,\lambda_1(a), \quad \text{and} \quad \frac{d}{dl} H(\cdot) \;=\; -\frac{d}{da}\,\lambda_2(a) \tag{4.17}$$

  must hold. The change in the shadow price of a state variable must equal the negative change in the Hamiltonian with respect to that state. More specifically, the adjoint equations associated with size and survival, respectively, are given by

$$\begin{aligned}\dot\lambda_1 &= -H_\xi(\cdot)\\ &= -\,l\, m_\xi(\xi,\pi)\\ &\qquad -\,\lambda_1\, k\,(\,\pi \;-\; \delta_0 \;-\; 2\,\delta_1\,\xi\,)\\ &\qquad\quad +\,\lambda_2\, l\, \mu_\xi(\xi)\end{aligned} \tag{4.18}$$

  and

$$\dot\lambda_2 \;=\; -H_l(\cdot) \;=\; -\,m(\xi,\pi) \;+\; \lambda_2\,\mu(\xi)\;. \tag{4.19}$$

- As age approaches infinity the values of an additional unit of size and survival, as captured by λ_1 and λ_2, respectively, have to approach zero. This is reflected in the transversality conditions, given by

$$\lim_{a\to\infty} \lambda_1(a) \;=\; \lim_{a\to\infty} \lambda_2(a) \;=\; 0\,. \tag{4.20}$$

Note that the state, control and costate variables are all functions of age. However, for brevity they are written as ξ, π, λ_1 and λ_2 wherever no confusion arises.

Solution

Taking into account that

$$k\,(\pi \;-\; \delta_0 \;-\; 2\,\delta_1\,\xi) \;=\; \frac{\dot{\xi}}{\xi} \;-\; k\,\delta_1\,\xi \tag{4.21}$$

the solution to the differential Equation in (4.18) gives the shadow price of an additional unit size at age a,

$$\begin{aligned}\lambda_1(a) = -\frac{1}{\xi(a)} \int_a^\infty\; & e^{-k\,\delta_1\,\int_a^t\,\xi(\tau)\,d\tau}\,\xi(t) \\ & \times l(t)\;(\lambda_2(t)\,\mu_\xi(t) \;-\; m_\xi(t))\;\,dt\,. \end{aligned} \tag{4.22}$$

Equation (4.19) can be solved as

$$\lambda_2(a) \;=\; \frac{1}{l(a)} \int_a^\infty\; l(t)\,m(t)\,dt\,. \tag{4.23}$$

The shadow price of survival at age a is equivalent to the reproductive value at that age. Inserting (4.23) into (4.18) leads to

$$\begin{aligned}\lambda_1(a) \;=\; & \tfrac{1}{\xi(a)} \textstyle\int_a^\infty\; e^{-k\,\delta_1\,\int_a^t\,\xi(\tau)\,d\tau}\,\xi(t) \\ & \times \left(\; l(t)\,m_\xi(t) \;-\; \mu_\xi(t)\,\textstyle\int_t^\infty\; l(\tau)\,m(\tau)\,d\tau \;\right)\;dt\,. \end{aligned} \tag{4.24}$$

To find an explicit expression for size, (4.5) can be solved, resulting in

$$\xi(a) \;=\; \frac{\exp\left\{\int_0^a\; k\,(\pi(t) \;-\; \delta_0)\,dt\right\}}{\frac{1}{\xi(0)} \;+\; \int_0^a\; k\,\delta_1\,\exp\left\{\int_0^t\; k\,(\pi(\tau) \;-\; \delta_0)\,d\tau\right\}\,dt}\,. \tag{4.25}$$

It can be seen that the state variable size increases in a logistic manner.

Result

With the state ratchet I showed that size must follow a monotonic path. The same result can be proved applying optimal control theory. For an infinite horizon autonomous optimal control problem with a single state variable, the optimal state path must be monotone (Kamien and Schwartz [90, p. 179] and Léonard and Van Long [114, p. 294]). Recall from (4.11) that fertility is linear in π. Therefore, the Hamiltonian function is linear in π, which results in solutions at the boundaries of the feasible set of investment strategies π, i.e. either one or zero.

Initially, $\pi_0 = 1$ and π remains at one until maturity. At maturity, a boundary solution implies that $\pi = 0$. If this were so, size would decrease, contradicting the state ratchet. Therefore, one expects what is called a "singular solution" in control theory. A singular solution requires that

$$\dot{H}_\pi \;=\; 0 \;=\; \dot{l}\, m^*_\pi \;+\; \dot{\lambda}_1\, k\, \xi^* \tag{4.26}$$

has to be satisfied. It would be natural if $\pi = \delta(a)$ were the singular solution required. Since size is constant in maintenance mode, the optimal solution would stay on the singular path forever. It turns out that $\pi = \delta(a)$ is the singular solution, as discussed below.

Since a logistic increase in size implies an upper limit to growth, there must be an age a^* at which size is finally maintained,

$$\pi \;=\; \delta(\xi), \qquad \forall\, a \;\geq\; a^* \,. \tag{4.27}$$

Consequently $\xi(a^*) = \xi^*$, $m(a^*) = m^*$ and $\mu(a^*) = \mu^*$ will be constant. If size is constant the reproductive value is simply given by the quotient of m^* and μ^*. Since the reproductive value of an individual at age a is captured by the costate variable $\lambda_2(a)$, this costate will be constant as well.

Assume $\pi = \delta(a)$ from age a^* onwards. Taking into account that

$$l(a) \;=\; l(a^*) e^{-\mu^*\,(a\,-\,a^*)}, \tag{4.28}$$

it follows from (4.24) for all $a \geq a^*$ that

$$\lambda_1(a) \;=\; \frac{l(a)\, m^*}{(k\,\delta_1\,\xi^* \;+\; \mu^*)} \left(\frac{m^*_\xi}{m^*} \;-\; \frac{\mu^*_\xi}{\mu^*} \right) . \tag{4.29}$$

This expression combined with condition (4.26) leads to an equation that determines the size at which the optimal investment should switch to maintenance mode,

$$\frac{m_\pi^*}{m^*} = \frac{k\,\xi^*}{k\,\delta_1\,\xi^* + \mu^*}\left(\frac{\mu_\xi^*}{\mu^*} - \frac{m_\xi^*}{m^*}\right) . \tag{4.30}$$

The relative change in reproduction with respect to the investment in growth must equal the weighted difference between the relative changes in mortality and reproduction with respect to size. Note that this condition does not depend on age: (4.26) will be zero for all ages $a > a^*$ once maintenance mode is reached.

In this model fertility is given by (4.11). From (4.30) it follows that a singular solution is determined by

$$\frac{\mu(\xi_a^*)}{k} = (1 - \delta_0 - 2\,\delta_1\,\xi_a^*) + \frac{(1 - \delta_0 - \delta_1\,\xi_a^*)\,b}{\mu(\xi_a^*)\,\xi_a^*} . \tag{4.31}$$

The individual will grow at full speed until its size satisfies (4.31)[1].

Substituting $\mu(\xi) = b\,/\,\xi + c$ yields a cubic polynomial with three roots. Generally, these roots can be real and complex. Viable strategies correspond to real, nonnegative roots. The optimal size at maturity corresponds to the root that maximizes life-time reproduction. Strategies can be determined numerically; I used MATHEMATICA™ to calculate the solution.

4.3.3 An Alternative Derivation

The state ratchet implies that if there is a single state variable, then the optimal investment strategy of an organism has to be growth, possibly followed by maintenance, i.e. the feasible set of $\pi(a)$ is

$$\pi(a) \in [\,\delta(a),\,1\,] . \tag{4.32}$$

A valuable hint follows from Pontryagin's Maximum Principle. Since the Hamiltonian is linear in $\pi(a)$ the optimal investment maximizes the Hamiltonian function at the boundaries of the feasible set (4.32). The upper limit $\pi(a) = 1$ is associated with full growth and no reproduction. The lower limit $\pi(a) = \delta(a)$ switches the organism to maintenance mode with constant, nonzero fertility and mortality.

[1] I thank Anatoli Michalski for his explanations regarding optimal control theory.

In this case the integral in (4.8) can be solved explicitly. The switching age, when $\pi(a)$ drops to $\delta(a)$, marks the onset of reproduction, age α. It follows that

$$R \,=\, l(\alpha) m(\alpha) \int_{\alpha}^{\infty} \exp\left\{ -\int_{\alpha}^{a} \mu(t)\, dt \right\} da \,=\, l(\alpha)\, \frac{m(\alpha)}{\mu(\alpha)}\,, \quad (4.33)$$

where $m(\alpha)$ and $\mu(\alpha)$ are the constant levels of fertility and mortality in maintenance mode after α.

The age α at which reproduction starts is determined by the value ξ_α that maximizes R in (4.33). Using the fact that from age zero to α there is a one-to-one correspondence between age a and size ξ, one can express (4.33) as a function of ξ_α. Inverting the logistic growth function $\xi = L(a)$ given in (4.6) leads to

$$a = L^{-1}(\xi) = \frac{1}{k\,(1\,-\,\delta_0)}\, \ln\left(\frac{1\,-\,\frac{\delta_1}{1-\delta_0}}{\frac{1}{\xi}\,-\,\frac{\delta_1}{1-\delta_0}} \right)\,. \quad (4.34)$$

Thus, by substituting $\alpha = L^{-1}(\xi_\alpha)$ in (4.33) one can express $R = R(\xi_\alpha)$ as a function of size at reproductive maturity ξ_α. The optimization problem now can be solved by setting the derivative of $R(\xi_\alpha)$ with respect to ξ_α equal to zero, i.e.,

$$l_{\xi_\alpha}\, \frac{m}{\mu} \,+\, m_{\xi_\alpha}\, \frac{l}{\mu} \,-\, \mu_{\xi_\alpha}\, \frac{l\, m}{\mu^2} \,=\, 0\,. \quad (4.35)$$

Because

$$l_{\xi_\alpha} \,=\, \frac{d}{d\xi_\alpha}\, l(\xi_\alpha) \,=\, \frac{d}{d\xi_\alpha} \exp\left\{ -\int_{1}^{\xi_\alpha} \mu(\xi)\, \left[k\,(1\,-\,\delta_0\,-\,\delta_1\,\xi)\,\xi\right]^{-1}\, d\xi \right\}$$

$$= \,-l(\xi_\alpha)\, \mu(\xi_\alpha)\, \left[k\,(1\,-\,\delta_0\,-\,\delta_1\,\xi_\alpha)\,\xi_\alpha\right]^{-1}\,,$$

optimal size at maturity is given by

$$\frac{\mu(\xi_\alpha)}{k} \,=\, (\,1\,-\,\delta_0\,-\,2\,\delta_1\,\xi_\alpha\,) \,+\, \frac{(\,1\,-\,\delta_0\,-\,\delta_1\,\xi_\alpha\,)\, b}{\mu(\xi_\alpha)\,\xi_\alpha}\,. \quad (4.36)$$

This equation is equivalent to (4.31). Using calculus and static optimization and applying Bellman's way of thinking with a hint from Pontryagin leads to the same result as using dynamic optimization applying Pontryagin's Maximum Principle.

4.3.4 The Simplest Model Leads to Sustenance

In the simplest case of size-independent mortality, i.e. $b = 0$, an explicit solution for the optimal size at maturity can be derived:

$$\xi_\alpha = \frac{(1 - \frac{c}{k} - \delta_0)}{2\,\delta_1} . \tag{4.37}$$

Results for three illustrative parameter combinations are shown in Fig. 4.2. Equation (4.37) implies

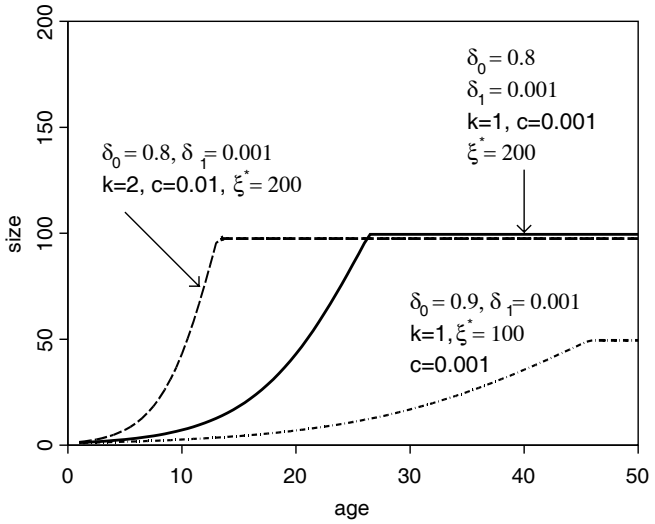

Fig. 4.2. Size $\xi(a)$ for three selected parameter combinations (Note that ξ^* denotes maximum possible size.)

$$\frac{d\xi_\alpha}{dc} < 0\,,\ \frac{d\xi_\alpha}{d\delta_0} < 0\,,\ \frac{d\xi_\alpha}{d\delta_1} < 0 \text{ and } \frac{d\xi_\alpha}{dk} > 0\,. \tag{4.38}$$

Furthermore, (4.37) and (4.34) imply

$$\frac{d\alpha}{dc} < 0 \text{ and } \frac{d\alpha}{d\delta_1} < 0\,. \tag{4.39}$$

Increasing extrinsic mortality reduces age and size at maturity. Changes in α with respect to k and δ_0 depend on the parameter combination

Table 4.1. Optimal size ξ_α and age α at the start of reproduction for size-dependent mortality ($b > 0$) according to (4.31)

ξ_α	α	ξ_{max}	$l(\alpha)$	b	c	k	δ_0	δ_1
62.26	50.96	100	0.005	0.5	0.001	1	0.9	0.001
53.46	47.34	100	$1.1 \cdot 10^{-9}$	2	0.001	1	0.9	0.001
60.02	50.02	100	0.00003	1	0.001	1	0.9	0.001
25.68	17.66	100	0.0012	1	0.1	2	0.9	0.001
56.86	24.36	100	0.0045	1	0.01	2	0.9	0.001
64.06	25.87	100	0.0056	1	0.000001	2	0.9	0.001
127.66	29.31	200	0.006	1	0.001	1	0.8	0.001
129.18	14.74	200	0.08	1	0.001	2	0.8	0.001

in a rather complicated way. For very small maximum attainable sizes and very slow speed of growth, α can increase with increasing k and decrease with increasing δ_0. Usually, however, an increase in k will lead to a decline in α while an increase in δ_0 will lead to a decrease in α.

If $b > 0$ in (4.10), then mortality declines as size increases. Hence for positive but small b

$$\xi_\alpha \,|_{b>0} > \xi_\alpha \,|_{b=0} \,. \tag{4.40}$$

If, however, b is large then the increased risk of death may make it optimal to start reproducing at a smaller size. Some illustrative results are shown in Table 4.1. If b gets too large then the resulting solutions are nonviable strategies: the species cannot survive because mortality is too high. Such nonviable strategies correspond to roots of (4.31) that are complex or negative.

In sum, the simplest model in which a single state variable determines the optimal strategy and reproductive effort affects fertility in a linear way can only lead to sustenance, i.e. a period of development followed by maintenance. Senescence is impossible and all there is to

be optimized is the age at maturity. From this age onwards the individual maintains its state forever. Complications have to be added to the simple model to get optimal strategies that are more flexible than this basic strategy. Note that flat mortality and fertility profiles were found to be very common in numerical studies by Charlesworth [26].

4.3.5 Introducing Nonlinearity Can Lead to Enhancement

Enhancement[2] – a sustenance strategy that includes a period of parallel growth and reproduction after the initial period of development and before the terminal period of maintenance – is precluded by the linearity in $\pi(a)$ of Pontryagin's Hamiltonian. To allow enhancement a model specification has to be found which results in a Hamiltonian that is nonlinear in $\pi(a)$.

To solve such an optimization problem the Bellman principle of dynamic programming can be used. Because the size ratchet precludes an organism from returning to previous states, the optimal trajectory of the allocation strategy can be found by a backward algorithm starting at the maximum attainable size at which maintenance is the only possible strategy. I developed such an algorithm, which produced results that were consistent with the analytic solution in the case of fertility being linear in $\pi(a)$. This algorithm can be readily applied to the following nonlinear fertility function:

$$m(a) = \varphi\, \pi(a)\, (1 - \pi(a))\, \xi(a) = \varphi\, (\pi(a) - \pi^2(a))\, \xi(a)\,. \quad (4.41)$$

The second term in the product, $\pi(a)$, can be interpreted as the efficiency of converting size $\xi(a)$ into reproduction $m(a)$. As $\pi(a)$ approaches zero, i.e. as resources are largely directed to fertility rather than growth and maintenance, this efficiency declines.

Figure 4.3 shows an illustrative result. For the parameters used in this model, reproduction starts when the organism grows to about 25% of its potential maximum size. Then, until maintenance mode is eventually reached at age 250, there is an extended period of enhancement.

This still simple model leads to optimal strategies of development followed by a period of parallel growth and reproduction followed by

[2] Maren Rebke and James W. Vaupel suggested this term to describe a period of life with increasing fertility and declining mortality. This enhancement is due to some kind of growth, but perhaps in strength or capability and not in size. I also use it to describe a life history strategy that starts with development, switches to parallel growth and reproduction and then ends with maintenance.

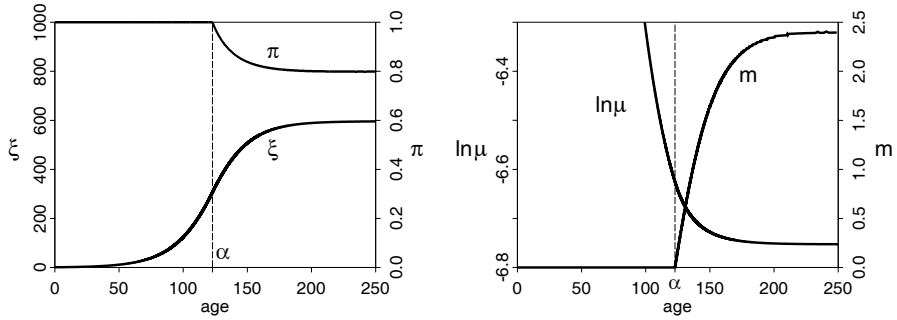

Fig. 4.3. Enhancement for model variant (4.41) (Parameter values were $k = 0.1$, $\delta_0 = 0.5$, $\delta_1 = 0.0005$, $b = 0.1$, $c = 0.001$, $\varphi = 0.02$. The force of mortality before age 100 is very high and rapidly falling.)

maintenance. In addition to the age at maturity, the age at maintenance as well as the path of investment between maturity and maintenance need to be optimized. However, senescence is still not an option. Any decline in size (i.e. an increase in mortality) is precluded by the state ratchet. To arrive ultimately at a framework where senescence is a possible optimal outcome the basic model has to be complicated even further.

4.4 An Optimization Model that Leads to Senescence

The state ratchet implies that any single-state life-history model along the general lines described above will always yield growth, declining mortality and increasing fertility followed by maintenance mode. Even if an exogenous event reduces ξ to some lower level ξ^-, then the individual would simply resume growth with the π-strategy previously followed at ξ^-.

In this kind of model, the single variable size ξ determines the capability of an individual to gather resources, to produce progeny and to avoid death. This spectrum might be too broad to be captured by size alone. Size can be measured by weight, length, number of cells, number of modular units or some similar index. While body size is determined by the number of cells and may remain constant, the functioning of cells may decline due to insufficient investment in maintenance because each cell is subject to continuous wear and tear. Therefore, it seems reasonable to distinguish between quantity and quality of cells. Functioning can be captured by a second state variable denoted by the Greek let-

ter υ, which can take values between one and zero. The "vitality" of an individual can then be modeled as the product of ξ times υ, size weighted by functioning. Adding a second state variable to the model is a way to escape the state ratchet.

The model can be reformulated as follows. Fertility is given by

$$m(a) = \varphi\,(\pi(a) - \pi^2(a))\,\xi(a)\,\upsilon(a)\,, \tag{4.42}$$

and mortality is given by

$$\mu(a) = \frac{b}{\xi(a)\,\upsilon(a)} + c\,. \tag{4.43}$$

Note that both fertility and mortality now depend on the product of size and functioning, $\xi(a)\,\upsilon(a)$, which captures vitality. The particular nonlinearity in fertility was retained.

This model can lead to determinate growth. Let a^* be the age at which growth is completed. Then $d\xi/da = 0$ for all $a > a^*$, where $\xi(a^*) = \xi^*$ denotes the size attained at the end of the determinate growth period. For $a < a^*$, functioning does not change, i.e. $\upsilon(a) = 1$. If investment falls below maintenance level, i.e. $\pi(a^*) < \delta_0 + \delta_1\,\xi(a^*)$ at a^*, functioning starts to deteriorate exponentially at the rate $\acute{\upsilon} = \kappa\,(\pi(a) - \delta_0 - \delta_1\,\xi^*)$ with initial condition $\upsilon(a^*) = 1$. If $\pi(a^*)$ is chosen to equal the deterioration at that age, the individual maintains its current functioning: this corresponds to the case of determinate growers with sufficient repair or replacement of tissues to escape senescence. The age a^* is not necessarily identical to age at reproductive maturity α, although for many determinate growers the two approximately coincide. The parameter combinations I used in the algorithm led to strategies for which $a^* = \alpha$.

Growth in ξ is positive until determinate size is attained and zero afterwards:

$$\frac{\frac{d\xi(a)}{da}}{\xi(a)} = \begin{cases} k\,(\pi(a) - \delta_0 - \delta_1\,\xi(a)) & \text{if } \pi(a) > \delta_0 + \delta_1\,\xi(a) \\ \\ 0 & \text{otherwise,} \end{cases} \tag{4.44}$$

where $\xi(0) = 1$. Functioning is constant at one until determinate size is reached and then declines:

$$\frac{\frac{dv(a)}{da}}{v(a)} = \begin{cases} 0 & \text{if } a < a^* \\ \kappa\,(\pi(a) - \delta_0 - \delta_1\,\xi^*) & \text{if } a \geq a^* \end{cases} \tag{4.45}$$

where $v(0) = 1$. Note that $\pi(a) - \delta_0 - \delta_1\,\xi^* < 1$. The parameters k and κ determine the speed of increase in size and the speed of decline in functioning, respectively.

Figure 4.4 exemplifies the optimal trajectories of $\pi(a)$, $\xi(a) \cdot v(a)$, $\mu(a)$ and $m(a)$ for determinate growth for this model. The results were obtained numerically. The maximum attainable size is $\xi = 25$; this size is almost reached at age of reproductive maturity α.

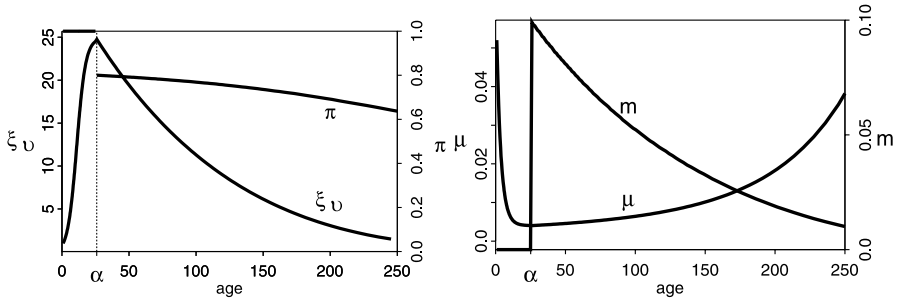

Fig. 4.4. $\xi(a) \cdot v(a)$, force of mortality μ and fertility m resulting from optimal strategy $\pi(a)$ as a function of age a, for model with parameters $k = 3$, $\delta_0 = 0.9$, $\delta_1 = 0.004$, $\kappa = 0.05$, $b = 0.05$, $c = 0.002$, $\varphi = 0.02$

In this model, the state variable that effectively determines the strategy switches from size to functioning at age a^*. Before age a^* size is the only effective state variable, since functioning is constant. After age a^* functioning is the only effective state variable, since size is constant. Therefore, the state ratchet applies and functioning cannot increase again once it has fallen below one. The switch between size and functioning is assumed to occur only once. Growth cannot be resumed.

Another possibility for overcoming the state ratchet, but keeping a model that is essentially based on a single state, is to introduce a switch variable, which is a binary indicator that determines whether the organism is in up or down mode. The switch itself does not affect survival or reproduction. To jump the maintenance barrier, the switch needs to change from up into down mode. In this case the optimality of the strategy is not violated, as the smaller state is now associated with a different value of the switch. Depending on whether the switch is

triggered once or several times, internally or externally, different state trajectories can emerge. Any repeated trajectories of increase and decrease have to be identical. This line of reasoning will be taken further in the next chapter.

4.5 Discussion

The first, simplest, model developed above led to sustenance as the only possible life-history strategy. The function describing reproduction had to be made nonlinear to get divergence from this prototype life history. The slightly more complex model led to a variety of possible life-history strategies between sustenance and enhancement. But senescence could still never be optimal.

To arrive at senescent strategies the state of the individual had to become more complicated, now being, effectively, a product of two variables, size and functioning. The product of size and functioning can be interpreted as reflecting the vitality of the individual. Vitality and not size determines mortality and fertility. Consequently it is possible that individuals might maintain about the same body weight, length or cell number over an extended period of life but suffer a decline in vitality due to wear and tear and lack of repair.

Although the eventuality was not considered here, size could increase over an extended period of life with this growth counterbalancing forces of deterioration and functional decline. In such species the ability to escape mortality, as captured by ξ times υ, may remain roughly constant—resulting in non-senescence.

Note the distinction between senescence, on the one hand, and deterioration and functional decline, on the other. The term senescence is used only with regard to entire organisms, not parts of organisms. In this model deterioration is captured by $\delta(a)$ and decline in functioning by a decrease in $\upsilon(a)$. A tendency for existing body parts to deteriorate and to require repair or replacement to maintain functioning may possibly be a "fundamental, universal, and intrinsic" property of living organisms [9]; senescence, as defined here, is not.

The theoretical results of this chapter and the empirical evidence presented in Sect. 3.3.3, suggest the following hypotheses:

- Senescence characterizes individuals in species that attain a size at reproductive maturity that is close to maximum size. Such determinate- growth species include mammals, birds, insects and some other species including the nematode worm *C. elegans*. The

main model species studied by gerontologists are mammals (including humans, rats and mice), insects (especially *Drosophila* but also Medflies and some other insect species), *C. elegans*, and yeast. All of these species fall into this determinate- growth category. Many determinate- growth species also have fixed oocyte stocks or are otherwise limited with regard to reproductive capacity. Species that experience declines in fertility with age or that have limited fertility seem likely to suffer senescence.

- Non-senescence characterizes individuals in species that attain a size at reproductive maturity that is less than maximum size and that gain reproductive capacity as they grow. Such species with indeterminate growth include most trees, many other perennial plants, many modular animals such as corals and perhaps sponges, some kinds of algae, many fish, reptiles and amphibians, and probably various nonmodular invertebrates such as some mollusks and some echinoderms.

Species falling into the second category are not typically model organisms in gerontological research. This might be one reason why the universality of senescence was accepted as gerontological dogma.

Many biologists would agree that, for many species, stage is what determines mortality and fertility rather than age. If age itself matters at all, this line of thinking leads to the conjecture that biological age may be better captured by the "average age" of an individual — i.e., by some appropriate measure of the average age of the organs, body parts or cells of an individual — than by the chronological age of the individual. In indeterminate- growth species, continuing increases in size keep average age below chronological age. Furthermore, organisms that can repair, replace or rejuvenate body parts may show, over chronological time, slow increases or even decreases in average age. For instance, trees that replace their leaves annually, that develop new roots and new branches to replace damaged or lost ones, and that continue to grow may be of an average individual age that remains roughly constant and may even decline with chronological age. For some species of plants and animals, there can be a complete turnover of body parts over a time interval: for these species, average individual age can be much lower than chronological age and can decline over time if the individual grows and its component parts continue to renew themselves with time.

A remarkable example is Hydra [123]. Most species as small as hydra have a short life expectancy. Hamilton's reasoning would imply that hydra should senescence quickly after having lived past its typical lifespan in the wild. Contrary to this prediction, mortality is constant

and has been effectively zero for hydras kept in the laboratory of Daniel Martinez for four years. Because there is rapid turnover of a hydra's cells, this example directs attention to considering not only size, i.e. quantity of cells, but also quality of cells. The first two models developed in this chapter consider size only, while the third model is a first attempt to incorporate not only quantity but also quality of cells. The model I develop in the following chapter accounts for both quantity and quality of cells.

This chapter has shown that non-senescence is a life-history strategy that is theoretically possible. Senescence can be avoided by "conceivable" organisms, namely by species with size-dependent vital rates. This finding together with the empirical evidence presented in Sect. 3.3 leads me to the hypothesis that non-senescence may indeed be a life history followed by some and maybe many plant and animal species. In the following chapter I develop a more general model to further study the evolution of senescence vs. non-senescence.

4.6 Next Steps

A critical examination of the model developed above indicates several directions to explore.

- The nonlinearity in fertility was introduced by means of efficiency of reproduction. Is there a more elegant way to incorporate efficiency?
- Reproduction and growth relate directly to size. This implicitly assumes that available resources are proportional to size. Is there a more realistic way to model resources?
- The vitality of an organism was modeled as a product of the two states size and functioning, in order to develop a model that can lead to non-senescent as well as senescent life-history strategies. The resulting model specifications seem rather complicated. Furthermore this model is not able to capture a simultaneous increase in size with a decrease in functioning. Size and deterioration were assumed to remain constant once functioning starts to decline. An idea for getting around this complication was suggested in Sect. 4.4.

The following chapter will take these points into account.