5

An Optimization Model Based on Vitality

The models developed in Chap. 4 show that non-senescence can be optimal. Size constitutes the central state variable in this framework. Mortality falls with increasing size and reproductive potential rises. The case of determinate growth, however, poses a challenge to this framework. Determinate growers, such as humans, often reach their final size at about the age of maturity. While size remains constant after the onset of reproduction, mortality steadily rises. This is incompatible with the strict size-dependence of mortality. A new model can be developed to address the deficiencies of the size-based model. To capture changing mortality at a constant size, the quality of size will be considered. The approach is rationalized in the following way. Even if size remains unchanged, all cells progressively accumulate damage over time and deteriorate. *Vitality*, defined as an individual's size adjusted for the functioning of body cells, can decline and therefore mortality can increase despite a constant body size. This notion was introduced in Sect. 4.4, where vitality was defined as the product of two functions, size and functioning. Here, vitality captures the accumulated functioning of all body cells, i.e. if a cell has been damaged and only works at 80 % of the capacity of an undamaged cell, this cell will account for 0.8 units of total vitality.

Facing ubiquitous decay, life is sustained by processes of regeneration and rejuvenation. The continuous creation of new, undamaged cells counterbalances deterioration. This balance determines whether or not vitality declines. The level of rejuvenation and repair depends on the trade-offs between reproduction on the one hand and growth and maintenance on the other. The optimal schedule of resource allocation determines the optimal trajectory of vitality. Increasing vitality raises reproductive potential and lowers mortality. Reproduction results in

offspring but entails slower growth or even decline in vitality. The trajectory of vitality over age determines the age-trajectories of fertility, mortality and growth. The following evolutionary-demographic model sheds light on the fundamental questions of life-history theory based on the single state variable, vitality.

Anderson [8] developed a model based on the variable vitality. Anderson defines vitality as a randomly varying component of mortality which leads to death if vitality ever reaches zero. The use of the state variable vitality, as defined here, constitutes a new approach to life history modeling.

5.1 The Vitality Model

Survival is a function of mortality. In accordance with the size-based models it seems natural to model mortality as an inverse function of vitality, denoted by ψ. A simple function for the force of mortality, μ, is

$$\mu(\psi) = \frac{b}{\psi} + c\,, \tag{5.1}$$

where b and c are constant parameters. The intrinsic parameter b captures all causes of death an individual can escape from by increasing its vitality, while the extrinsic parameter c captures the always prevalent, non-zero risk of death. Note that "extrinsic" and "intrinsic" refer to vitality-dependent vs. vitality-independent mortality.

Reproduction and growth depend on the level of available energy. In the size-based models, energy was simply proportional to size. However, energy production is not equivalent to size but has been found to scale allometrically with it [107]. A sound theoretical basis for a particular relation between size and net energy available was given by West et al. [210], their Equation (3). This formula captures the difference between energy created by cell metabolism and energy required for it, based on an allometric relation between size and energy production.

The model developed in this chapter uses Equation (3) from West et al. [210] to determine the available resources of an individual at its current level of vitality. The formula of West and colleagues [210] is based on the variable size. The link between vitality and size is assumed to be tight enough to justify the substitution of vitality for size in this equation for this specific model. Net energy production, denoted

by $\epsilon(\psi)$, depends on the difference between build-up and break-down processes at current vitality,

$$\epsilon(\psi) = k\,\psi^{0.75} - \kappa\,\psi\,, \tag{5.2}$$

where k and κ are constant parameters. Anabolic, build-up processes are directly linked to metabolic output, which is assumed to be proportional to vitality to the power 0.75. Catabolic, break-down processes are assumed to be proportional to vitality to the power one.

The exact value 0.75 for anabolic processes was thought to be a so called life-history invariant [33]. The method of calculating these life-history invariants has recently been called into question [49, 137]. The particular value of 0.75 might therefore not be invariant across species. The qualitative results of my model, however, do not depend on the particular value 0.75 but only require the existence of such an allometric relation.

Energy production is maximal at vitality ψ_ϵ

$$\psi_\epsilon = \left(\frac{3}{4}\,\frac{k}{\kappa}\right)^4 . \tag{5.3}$$

As in the size-based model, growth and maintenance are paid out of the same budget. Part of the energy available must be used to offset the declining functioning of cells. The change in vitality is given by the difference between the fraction of resources allocated to growth (newly built cells) and the unavoidable deterioration of functioning of current cells at a constant rate δ. Damage is proportional to vitality and integrates naturally into the structure of West et al.'s equation. Consequently, vitality ψ changes over time according to

$$\dot{\psi} = \pi(\psi)^{\eta_g}\,\epsilon(\psi) - \delta\,\psi\,, \tag{5.4}$$

where $\pi(\psi)$ denotes the fraction of energy allocated to growth, as in the models in Chap. 4. In contrast to those models, $\pi(\psi)$ can now have a nonlinear effect on the change in state depending on the value of the constant parameter η_g (g for growth). In the extreme case of no energy allocation to growth and maintenance, vitality deteriorates exponentially and, as in the size-based model, mortality rises exponentially. The reasoning behind the incorporation of parameter η_g will be given below.

The level of initial vitality is $\psi(0) = 1$ and initial time zero corresponds to time at birth[1]. Vitality is treated as a dimensionless variable,

[1] Note that the model in its current form does not account for stage-specific life-histories.

assuming that vitality is normalized by dividing through with a reasonable base unit. If the state of an organism at birth corresponds to some $\psi_{real}(0)$, this implies that vitality $\psi(a)$ in this model is given by[2]

$$\psi(a) = \psi_{real}(a)/\psi_{real}(0) \tag{5.5}$$

and therefore

$$\psi(0) \;=\; \frac{\psi_{real}(0)}{\psi_{real}(0)} \;=\; 1\;. \tag{5.6}$$

The level of $\pi(\psi)$ that corresponds to maintenance of current vitality can be derived from (5.4). Denoting the level of $\pi(\psi)$ at $\dot{\psi} = 0$ by π_0 and inserting (5.2) yields

$$\pi_0 \;=\; \left(\frac{\delta}{k\,\psi^{-0.25} \;-\; \kappa}\right)^{\frac{1}{\eta_g}}\;. \tag{5.7}$$

Vitality cannot increase indefinitely. An upper limit to ψ, denoted by Ψ, is reached at maximum investment $\pi(\psi) = 1$ and $\dot{\psi} = 0$,

$$\Psi \;\equiv\; \left(\frac{k}{\kappa \;+\; \delta}\right)^4\;. \tag{5.8}$$

Available energy must be nonnegative. This implies that

$$\psi \;\leq\; \left(\frac{k}{\kappa}\right)^4 \tag{5.9}$$

must hold. This is always true since (5.9) implies that ψ cannot exceed maximum attainable vitality Ψ, as given by (5.8).

In the initial size-based model (Sect. 4.3) reproductive effort and reproductive output are related linearly. As explained in Sect. 4.3.2, it turns out that this assumption restricts optimal solutions to energy allocation exclusively to either growth or reproduction. To develop a model that permits a broad scope of possible investment strategies, a nonlinear influence of investment needs to be incorporated that still includes the possibility of exclusive allocation. This is the technical argument that motivates the introduction of parameter η_g in (5.4). The biological motivation for introducing nonlinear effects is the following.

[2] If functioning at birth is assumed to be perfect, then $\psi_{real}(0)$ is equal to the number of cells (corresponding to the minimum size) at birth. In order to establish the real vitality scale ψ_{real} from the algorithm, vitality has to be multiplied by $\psi_{real}(0)$.

Growing a human arm requires considerable effort and is so difficult that, if the arm is lost, no new arm can regrow. In contrast, growing a branch of a tree can be done readily to increase size or replace broken branches. The growth apparatus in humans and trees is inherently different. In the former case, it might be very costly and even impossible to keep or rebuild the machinery that would allow the regrowth of a lost arm. In the latter case, maintenance is cheap because existing machinery can be used to maintain the organism without much additional cost.

Parameter η_g captures the nature of the growth and maintenance apparatus of a species. When η_g exceeds one, the investment function π^{η_g} in (5.4) is convex. The marginal benefits in outcome become larger as π approaches one. Note that the convexity favors exclusive investment strategies. When η_g is below one, the investment function π^{η_g} is concave. The marginal benefits in output become smaller as investment approaches one. Note that concavity favors intermediate investment strategies. The parameter η_g in (5.4) captures the returns to scale in growth and maintenance investment. The parameter can also be interpreted as the efficiency of the growth system. Values of η_g below one correspond to efficient, i.e. cheap, growth, and values of η_g above one correspond to inefficient, i.e. costly, growth.

Figure 5.1 illustrates the influence of parameter η_g via investment π on the change in vitality. Note that the change in vitality is always larger for a given level of investment π when η_g is below one as opposed to being above one. Likewise, any particular level of change in vitality requires a smaller investment, given that η_g is below one rather than above one. Note further that values of η_g below one imply a concave shape, while values above one correspond to a convex shape of the change in vitality with increasing investment.

In the modified size-based model (Sect. 4.4) an arbitrary attempt was made to introduce nonlinearity with respect to reproductive effort. In the vitality model the maternity function is specified as

$$m(\psi) \;=\; \varphi\,(\,1 \;-\; \pi(\psi)\,)^{\eta_r}\,\epsilon(\psi)\;. \tag{5.10}$$

In accordance with the size-based models, fertility is proportional to available energy, in this model $\epsilon(\psi)$, and reproductive effort, $1 - \pi(\psi)$. In contrast to the size-based model, nonlinearity in reproductive effort is incorporated by parameter η_r (r for reproduction) which captures the efficiency of reproduction, analogous to η_g. As in the size-based models, the constant φ is a scaling parameter set to the value that ensures that optimal lifetime reproduction is equal to one and, hence, $r_{max} = 0$.

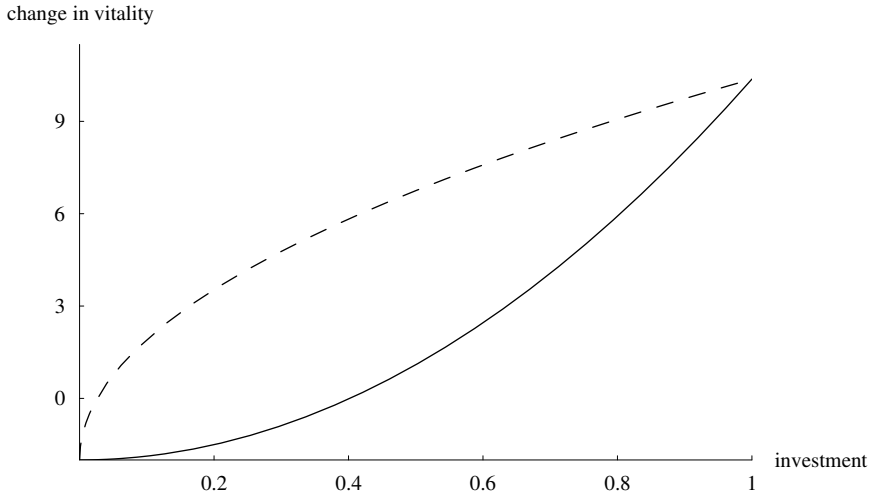

Fig. 5.1. The influence of η_g via investment π on the change in vitality as specified by (5.4) (*The dashed line exemplifies values of* η_g *below one, in particular* $\eta_g = 0.5$. *The solid line exemplifies values of* η_g *above one, in particular* $\eta_g = 2$. *In both cases* $\psi = 20$, $k = 3$, $\kappa = 0.8$ *and* $\delta = 0.1$.)

The manner in which nonlinearities enter the model is biologically and technically motivated. The approach makes use of the well-known concept used in economics of the Cobb Douglas production function. Each input factor to the production function is raised to a power reflecting how efficient each factor, in economics labor and capital, is in producing output. Two new parameters (that influence the optimal trajectory of investment) enter the model as exponents of investments. Power functions have previously been used to introduce nonlinearities into life-history models [26, 37, 39, 62, 175]; see Charlesworth [27, Sect. 5.3.4.] for review). In particular, the importance of the shape of the investment function for the optimal life history strategy has been recognized. In their reproductive effort models, Gadgil and Bossert [62] and Schaffer [175] found that concave investment functions favor iteroparous strategies (repeated breeding, i.e. intermediate reproductive effort) while convex investment functions favor semelparous strategies (a single breeding event, in which reproduction is fatal, i.e. exclusive investment).

George E. P. Box said: "All models are wrong, but some are useful." [13] Models are wrong because they simplify the complexity of life. But without this simplification, patterns can hardly be observed and understood. A useful model captures the most important aspects

of reality, reveals general patterns and provides a source for hypotheses that could explain basic processes of life. Such a model, although necessarily wrong, enhances our understanding of nature.

Adding efficiency to the size-based model increases complexity but it also considerably broadens the model's potential for predicting various life-history strategies. The non-linearities capture cases in nature when parallel investment in growth and reproduction is optimal. Therefore, these extensions to the model can be justified as a useful complication to a still simple model.

5.1.1 The Parameters

k, κ and δ

Parameter k captures the speed of growth of vitality (see (5.2) and (5.4)). Faster growth implies a rapid fall in mortality (5.1) and reduces the time of development. Furthermore, higher values of k decrease maintenance costs (5.7) and increase maximum vitality (5.8). Parameter κ is inversely related to maximum vitality. Elevating κ slows growth, increases maintenance costs (5.7) and decreases maximum vitality (5.8). Parameter δ determines the rate of decline in vitality (5.4). Higher δ increases maintenance costs (5.7) and decreases maximum vitality (5.8).

If all available energy is allocated to reproduction, then δ determines the constant rate of increase in mortality (5.1). A decline in vitality implies not only a reduction in survival but also in reproductive potential. Therefore, larger values of δ will tend to increase the investment of resources in growth in order to slow down the deterioration process.

Parameters k and κ determine the shape of the energy trajectory over vitality (5.2). If $\kappa < 3\delta$, then energy is an increasing function of vitality because the maximum attainable vitality is smaller than the level of vitality that maximizes energy, $\Psi < \psi_\epsilon$. Otherwise, if $\kappa > 3\delta$, then the trajectory of energy is hump-shaped with respect to vitality. The influence of the relation between κ and δ on the energy trajectory over vitality is visualized in Fig. 5.2. Note that an increase in vitality beyond the threshold given by (5.3), which corresponds to the peak of energy, can only be optimal if the corresponding reduction in mortality offsets the loss in available resources, i.e. in growth and reproductive potential.

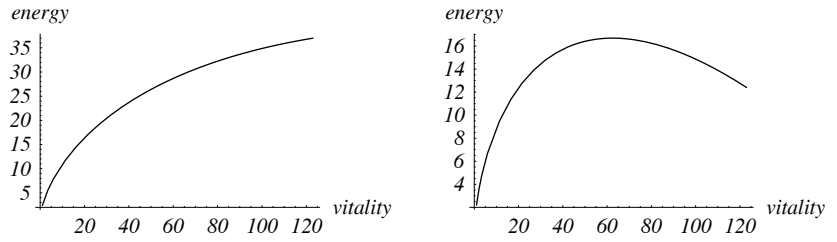

Fig. 5.2. Comparison of trajectories of energy over vitality for two parameter combinations that lead to a maximum attainable vitality of $\Psi = 123$ but imply different shapes (*left:* $k = 3$, $\kappa = 0.6$, $\delta = 0.3$*; right:* $k = 3$, $\kappa = 0.8$, $\delta = 0.1$)

The parameters k, κ, and δ set the speed of growth and decay and can therefore be used to determine the time and size scale of the strategy. Getting a handle on measurable quantities like time and size in this model is one future project that naturally follows from my work (see Chap. 6).

b and c

Parameters b and c determine the overall level of mortality (5.1). Parameter b captures the state-dependent, intrinsic component of mortality, i.e. b determines how important it is to attain and maintain a high level of vitality. Reasonable magnitudes of parameter b are given by the fact that $b/\psi(0)$ determines infant mortality. Furthermore, the minimum level of state-dependent mortality depends on parameter b and on maximum vitality Ψ and is given by b/Ψ. Parameter c captures the state-independent, extrinsic mortality component. The overall level of infant mortality is given by $b/\psi(0) + c$ and the minimum mortality that can be attained is given by $b/\Psi + c$.

The influence of extrinsic and intrinsic mortality in this model is investigated below (see Sect. 5.5.3).

η_r and η_g

Parameter η_r captures the intrinsic costs of reproduction (5.10). It determines the propensity to share resources between reproduction and growth. Clearly, if an organism follows an exclusive strategy, i.e. either reproduction or growth and repair, then π equals one or zero and an exponent will have no influence. However, if energy is shared between processes, then larger values of η_r reduce the reproductive output that

could have been achieved with the same level of investment at lower values of η_r. Values below one favor parallel investment in growth and reproduction.

Parameter η_g captures the intrinsic costs of growth and determines the maintenance costs of a certain level of vitality (5.7). A large value of η_g implies higher maintenance costs at each level of vitality. Therefore, low values of η_g favor non-senescence strategies. During periods of parallel growth and reproduction, higher η_g implies a reduced speed of growth.

Both parameters η_r and η_g capture the efficiency of energy use and determine how advantageous it is to specialize in growth and reproduction, i.e. how costly it is to run a growth and reproduction system in parallel. The costs of reproduction and maintenance are expected to crucially determine the optimal energy allocation between reproduction and growth. In this chapter I will investigate whether or not this expectation is fulfilled.

5.2 The Vitality Model as a Control Problem

The model developed in the previous section is an autonomous control problem with an infinite time horizon. In the following sections the problem is formulated and subsequently a solution is approached including a discussion of the range of possible optimal solutions.

5.2.1 Problem Formulation

The objective function to be maximized is given by

$$\max_{\pi} \int_0^{\infty} e^{-\phi} (1 - \pi)^{\eta_r} \epsilon(\psi) \, da \tag{5.11}$$

where

$$\epsilon(\psi) = k \psi^{0.75} - \kappa \psi \tag{5.12}$$

is associated with the level of energy available, as defined earlier. The cumulative hazard of death, ϕ, is defined as the logarithm of survival $l(a)$ at age a

$$\phi(a) = -\ln (l(a)) \, . \tag{5.13}$$

The only control variable in this problem is the proportion of investment $\pi(a) \, \epsilon \, [0, 1]$ towards growth and survival versus reproduction. The state variables of this problem are vitality $\psi(a) \, \epsilon \, \mathbf{R}^+$ and the cumulative hazard of death $\phi(a) \, \epsilon \, \mathbf{R}^+$.

The change in vitality with age is given by

$$\dot{\psi} \;=\; f(\psi,\pi) \;\equiv\; \pi^{\eta_g}\,\epsilon(\psi) \;-\; \delta\,\psi\;, \tag{5.14}$$

and the change in the cumulative hazard of death is given by

$$\dot{\phi} \;=\; \mu(\psi)\;, \tag{5.15}$$

obeying the initial conditions[3]

$$\psi(0) \;=\; 1 \tag{5.16}$$

and

$$\phi(0) \;=\; 0\;. \tag{5.17}$$

The Hamiltonian function[4] associated with this problem is given by

$$H \;=\; e^{-\phi}\,(1-\pi)^{\eta_r}\,\epsilon(\psi) \;+\; \lambda_\psi\,\dot{\psi} \;+\; \lambda_\phi\,\dot{\phi}\;, \tag{5.18}$$

i.e.

$$H \;=\; e^{-\phi}\,(1-\pi)^{\eta_r}\,\epsilon(\psi) \;+\; \lambda_\psi\,(\pi^{\eta_g}\,\epsilon(\psi) \;-\; \delta\,\psi) \;+\; \lambda_\phi\,\mu(\psi)\;, \tag{5.19}$$

with the transversality conditions for the two co-state variables[5]

$$\lambda_\psi(\infty) \;=\; \lambda_\phi(\infty) \;=\; 0\;. \tag{5.20}$$

Note that the Hamiltonian function, denoted simply as H, is a function of the control, state and costate variables but does not explicitly depend on time t, i.e. $H = H(\pi(t), \psi(t), \phi(t), \lambda_\psi(t), \lambda_\phi(t))$.

[3] Note that the model does not account for optimization of size at birth. This is an interesting topic that could be explored with an extended version of this model. Including variable size at birth which can be reasonably interpreted as vitality at birth implies several issues that will be discussed in a future joint paper by Kenneth Wachter and me.

[4] see Sec. 4.3.2

[5] Note that the subscripts 'ψ' and 'ϕ' to λ should not be confused with denoting partial derivatives.

5.2.2 Solution

The Maximum Principle requires that an optimal control path has to maximize the Hamiltonian function H. If H is differentiable w.r.t. π the optimal π^* (star indicates "optimal") can be found by

$$\frac{dH}{d\pi} \;=\; -\eta_r\, e^{-\phi}\,(1\,-\,\pi)^{\eta_r-1}\,\epsilon(\psi) \;+\; \eta_g\,\lambda_\psi\,\pi^{\eta_g-1}\,\epsilon(\psi) \;=\; 0\,. \tag{5.21}$$

Rearranging this expression yields

$$\frac{(1\,-\,\pi)^{\eta_r-1}}{\pi^{\eta_g-1}} \;=\; \frac{\eta_g}{\eta_r}\,e^{\phi}\,\lambda_\psi\,. \tag{5.22}$$

A maximum further requires the second derivative of H to be negative, and therefore condition

$$\begin{aligned} H_{\pi\pi} \;&= \eta_r\,(\eta_r-1)e^{-\phi}\,(1\,-\,\pi)^{\eta_r-2}\,\epsilon(\psi) \\ &\quad +\; \eta_g\,(\eta_g-1)\lambda_\psi\,\pi^{\eta_g-2}\,\epsilon(\psi) \;<\; 0 \end{aligned} \tag{5.23}$$

must hold, which after rearranging becomes[6]

$$(\eta_r-1)\,\frac{(1\,-\,\pi)^{\eta_r-2}}{\pi^{\eta_g-2}} \;<\; -(\eta_g-1)\,\frac{\eta_g}{\eta_r}\,e^{\phi}\,\lambda_\psi\,. \tag{5.24}$$

If both conditions (5.22) and (5.24) are true, expression (5.22) can be substituted for in (5.24), yielding

$$(\eta_r-1)\,\frac{(1\,-\,\pi)^{\eta_r-2}}{\pi^{\eta_g-2}} \;<\; -(\eta_g-1)\,\frac{(1\,-\,\pi)^{\eta_r-1}}{\pi^{\eta_g-1}}\,, \tag{5.25}$$

and after rearranging

$$(\eta_r-1)\,\frac{\pi}{1\,-\,\pi} \;<\; -(\eta_g-1)\,. \tag{5.26}$$

It should be emphasized that conditions (5.22) and (5.24) are not necessary for an optimal solution to exist. But if they are satisfied, then the sufficiency condition derived by Mangasarian [117] is satisfied. The Hamiltonian function is concave in π and an interior solution is optimal.

A Hamiltonian function that is linear (as in Sect. 4.3.2) or convex in π implies that a potential maximum can only be achieved at the boundaries of the feasible set of π. Thus this maximum cannot be

[6] I am not dividing by $\eta-1$ terms, since they could become negative depending on the parameter values in which case the inequality would turn around.

found by differentiating the Hamiltonian function as done in 5.21. The case of a convex Hamiltonian will be discussed later in this chapter.[7]

Condition (5.26) helps determine the range of parameters η_r and η_g for which it is safe to say that problem (5.11) has an optimal solution:

- It is immediately apparent that condition (5.26) is true for values of $\eta_r < 1$ & $\eta_g < 1$, $\eta_r = 1$ & $\eta_g < 1$, and $\eta_r < 1$ & $\eta_g = 1$. This range of the parameter space corresponds to a concave Hamiltonian function.
- If $\eta_r = 1$ & $\eta_g = 1$, the condition is violated. Instead, the Hamiltonian is linear in π and, as discussed in Sect. 4.3.2, the optimal solution is singular.
- For values $\eta_r > 1$ & $\eta_g < 1$ and $\eta_r < 1$ & $\eta_g > 1$ the Hamiltonian can be concave as well as convex, depending on the value of π and on the relative magnitude of η_r and η_g. I will further investigate this very interesting case later in this chapter.
- If both $\eta_r > 1$ & $\eta_g > 1$ the concavity condition is violated, the Hamiltonian is convex. Do optimal solution exists for problem (5.11)? And if not, is there a way to modify the model in order to get optimal solutions for the case of $\eta_r > 1$ & $\eta_g > 1$? As for the previous case I will tackle those questions later in this chapter.

Maximizing the Hamiltonian function is not the only condition the Maximum Principle requires to be fulfilled. As discussed in Sec. 4.3.2 in Chap. 4, the co-state variables have to meet the following conditions:

$$\dot{\lambda}_\psi = -\frac{dH}{d\psi} \tag{5.27}$$

and

$$\dot{\lambda}_\phi = -\frac{dH}{d\phi}\,. \tag{5.28}$$

Solving the differential equations (see App. A.1) yields the following results:

The shadow price of vitality at age a is given by the associated cumulated changes in fertility and mortality over all remaining ages discounted by the corresponding cumulative changes in growth.

$$\lambda_\psi(a) = \int_a^\infty \left(e^{-\phi}\,(1-\pi)^{\eta_r}\,\epsilon_\psi + \lambda_\phi\,\mu_\psi \right) \times e^{\int_a^x \pi^{\eta_g}\,\epsilon_\psi - \delta\; ds}\; dx\,. \tag{5.29}$$

[7] The existence of optimal solutions for my problem will be discussed in more detail in a forthcoming paper by Kenneth Wachter and me.

The shadow price of the cumulative hazard of death at age a is the negative value of remaining reproduction at age a, i.e. the penalty for having one unit higher cumulative hazard:

$$\lambda_\phi(a) \;=\; -\int_a^\infty \; e^{-\phi}\,(1\,-\,\pi)^{\eta_r}\,\epsilon(\psi)\;dx\;. \tag{5.30}$$

Equation (5.30) can be substituted in (5.29) to yield the final expression for $\lambda_\psi(a)$, being

$$\begin{aligned}\lambda_\psi(a) \;=\; &\textstyle\int_a^\infty \; e^{\int_a^x \; \pi^{\eta g}\,\epsilon_\psi\,-\,\delta\;ds} \\ &\times\left(e^{-\phi}\,(1-\pi)^{\eta_r}\,\epsilon_\psi \;+\; \tfrac{b}{\psi^2}\;\textstyle\int_x^\infty \; e^{-\phi}\,(1-\pi)^{\eta_r}\,\epsilon(\psi)\;d\tau\right)dx\;.\end{aligned} \tag{5.31}$$

The shadow price of vitality is given by the benefits of increasing reproduction due to higher vitality as well as the gains in remaining reproduction due to lower mortality, both weighted by the change in growth. As long as an increase in vitality leads to faster growth, this weight is above one (revaluating), if the increase in vitality leads to slower growth, then the weight is below one (devaluating).

5.2.3 The Role of the Second State Variable

A condition for optimal investment is given in (5.22). This condition requires that $\lambda_\psi(a)$, given in (5.31), is multiplied by $\exp(\phi(a))$. It becomes apparent that an optimal solution depends only on the current value[8] $\lambda_\psi^c \equiv \exp(\phi(a))\;\lambda_\psi(a)$ which is not discounted by death up to age a, in other words the cumulative hazard of death from birth to age a is erased. Consequently, expression (5.22) and thus an optimal investment path after age a is independent of the state variable $\phi(a)$; the hazard of death accumulated between age zero and age a has no effect.

Why is that so? Why does the second state variable not influence the optimal solution? The answer can be found reformulating the control problem. Maximizing the objective function in (5.11) from age zero to infinity requires maximizing the objective from any time point T onwards. Thus, the objective can be written as

$$\max_\pi\;\left(\;\int_0^T\;e^{-\phi(x)}\,m(x)\;dx\;+\;\max_\pi\;\int_T^\infty\;e^{-\phi(x)}\,m(x)\;dx\right)\;, \tag{5.32}$$

[8] The concept of current values, i.e. values at time t rather than their equivalent at time zero is well described in [90, pp. 164–174]

where $m(x)$ denotes the maternity function at age x. Maximization before age T is conditional on the maximization after T. But the maximization after T is conditional only on the value of $\psi(T)$. In particular the cumulative hazard of death between age zero and T does not influence the optimal strategy after age T. Instead, the factor $\exp -\phi(T)$ can be drawn outside the integral, i.e.

$$\max_{\pi} \int_T^{\infty} e^{-\phi}\, m(x)\, dx \;=\; e^{-\phi(T)} \max_{\pi} \int_T^{\infty} e^{-\phi(x-T)}\, m(x)\, dx\;, \tag{5.33}$$

revealing its nature as a mere scaling factor. Effectively, the cumulative hazard starts off at $\phi = 0$ for any time point the control problem is supposed to be solved from. It has no effect on the first state variable since the change in vitality is entirely driven by investment π and by vitality itself. The fact that the change in cumulative hazard ϕ depends on mortality which in turn depends on vitality further emphasizes the point that vitality is the only actual state variable that matters.

Thus, the control problem formulated in this chapter is essentially a single state, single control, autonomous, infinite horizon optimal control problem. As discussed earlier in Chap. 4 this implies that any optimal state path has to be monotone. Since life starts by growth, only initially increasing vitality trajectories are sensible. This means mortality cannot increase; senescence is impossible. Only growth followed by either a period of parallel growth and reproduction and then maintenance or development followed by maintenance directly are the possible strategies. Given the idea of "inevitable senescence" it is remarkable how challenging it is to actually develop a model that can lead to senescence as an optimal life history strategy.

In the size chapter I was able to modify my model by adding a second state variable to come up with solutions that yielded senescence. How could I change my vitality model to broaden the scope of possible solutions, including the pattern of human senescence? As mentioned above there are several parameter combinations for η_r and η_g for which the Hamiltonian function is convex. Maybe the "weird" cases of $\eta_r > 1$ & $\eta_g < 1$, $\eta_r < 1$ & $\eta_g > 1$ and $\eta_r > 1$ & $\eta_g > 1$ provide a rich ground for exploring exotic strategies? Before I make the leap to modifying my model let me step back for a moment and have another look at the results.

5.2.4 Hamilton and Reproductive Value - Revisited

First note that the shadow price of the cumulative hazard of death at age a given in (5.30) corresponds to the negative of remaining repro-

duction at that age, which is equivalent to the numerator in Hamilton's indicator for the force of selection on age-specific mortality. As discussed at length in Chap. 2, this quantity inevitably declines with age. But in my control problem it does not automatically mean that mortality becomes less important, and that mortality would ultimately increase with age. My results just showed that when the Hamiltonian function is concave, then vitality has to increase and thus mortality has to decline, despite an inevitable falling shadow price of the cumulative hazard of death.

Hamilton emphasized how important it was to use 'remaining reproduction' and not 'reproductive value' for quantifying selection pressure. Interestingly, reproductive value can be recovered if I modify my model formulation, though leaving it essentially unchanged: as just shown the cumulative hazard of death at age a has no influence on the optimal solution at age a. Looking closer at the problem formulation, the definition of the cumulative hazard $\phi(a)$ as the second state variable seems somewhat arbitrary. Indeed, I could have equally well let survival $l(a)$ serve as the second state variable. This would have given me a equation of motion, different to the one in (5.15), namely $\dot{l}(a) = -\mu(a)\, l(a)$, but again the condition for optimal π would have been independent of the second state. Doing the corresponding modifications and calculations leads to a shadow price of survival that is equal to

$$\lambda_l(a) = \frac{1}{l(a)} \int_a^\infty l(x)\,(1-\pi)^{\eta_r}\,\epsilon(\psi)\,dx\,. \tag{5.34}$$

This expression corresponds to the reproductive value at age a. Contrary to the shadow price of the cumulative hazard of death which inevitably declines with age, the shadow price of survival can also be constant or increase with age.

Both the quantities – remaining reproduction and reproductive value – that were of central importance to Hamilton's discussion of the evolution of senescence emerge as part of my life history optimal control problem. Furthermore, the quantities have been associated with opposite answers to the essential question of whether senescence is inevitable or not. Now it turns out that both quantities have their interpretation as shadow prices for either survival or cumulative hazard. They are weights in the Hamiltonian function. But neither of them actually influences the optimal control path (see (5.22)). The optimal control path only depends on the current value of the shadow price of vitality and the magnitude of the η parameters.

This is an important finding because it is tempting to predict the shape of mortality and fertility from the fitness sensitivities directly.

My result underlines the fact that one should not do so. Explaining the change in a trait (and ultimately the evolution of age-trajectories of mortality and fertility) requires multiplying the sensitivity in fitness by the variance-covariance matrix [105], as discussed in Chap. 3. Lande's so called G-matrix essentially contains all the trade offs among the fitness relevant traits.

People studying the evolution of senescence try to understand whether optimization approaches that rest on trade-offs or approaches based on fitness sensitivities (which is what Hamilton's indicators are and reproductive value is the fitness sensitivity with respect to survival) are a better way of explaining the evolution of senescence (see Chap. 3). I find that in my optimization framework fitness sensitivities appear as weights in the optimization formulas. Thus, both approaches are intertwined. The justification and importance for fitness sensitivities in shaping age-trajectories of mortality and fertility is in giving appropriate weights to the trade-offs that are balanced by evolution. Giving weights to trade-offs is exactly the same function that sensitivities take on in determining short term evolutionary change, when fitness sensitivities are multiplied by the appropriate G-matrix. For both approaches the central role of trade offs is conspicuous. Nailing down the trade offs - however - is one of the hardest nuts to crack for life history biologists, independently of which approach is taken.

5.3 The Constrained Vitality Model

Let me now get to the 'weird' ranges of parameters η_r and η_g. For values of η_r and η_g that lead to a convex Hamiltonian function no simple answer can be given as to whether an optimal solution exists or not. Mangasarian's theorem [117] is sufficient for an optimal solution but not necessary. An example will help to understand why convexity is a problem.

Let me focus on the case of $\eta_r > 1 \,\&\, \eta_g > 1$. If both η_r and η_g exceed one, then the Hamiltonian is convex in π. Therefore the highest value of H is found at the boundaries of the feasible set for values of π. Thus the only two possible values of π that could maximize H are either $\pi = 1$ or $\pi = 0$.

If $\pi = 1$ no reproduction is realized at all, so the Hamiltonian equals zero. If $\pi = 0$, then all resources are spend on reproduction, and vitality declines. For $\pi = 0$ the Hamiltonian function takes on some positive value, hence the Hamiltonian is larger for $\pi = 0$ than for $\pi = 1$, i.e. $H(\pi = 0) > H(\pi = 1)$. As $\pi = 1$ implies growth in vitality whereas

$\pi = 0$ implies shrinkage in vitality, π would have to remain at the level of zero forever because an optimal trajectory for this problem has to be monotone. It can be proven, however, that $H(0) > H(1)$ will be violated at some point, at least for the special case of constant mortality, $\mu = c$ (see App. A.2). This case illustrates the fact that the model has no optimal solution if both η's are larger than one[9].

An interesting aspect of the proof, however, is the insight that at some point the Hamiltonian will lead to higher values for $\pi = 1$ than for $\pi = 0$. Thus the strategy switches from full reproduction to full growth. One can show that the convex Hamiltonian produces zig-zagging strategies. And it is this zig-zagging that holds the key to the explanation for the non-existence of a solution[10]. To prevent this behavior one could constrain the vitality model in a way that zig-zagging is not an option. Let me explore this avenue further.

The mode of change of an organism can be constrained such that it can be changed only once. Hence, periods of growth and shrinkage cannot occur repeatedly but can only alternate once. Since life starts with growth, initially the mode of change for any organism is to increase in vitality. The organism is free to grow and increase in vitality until eventually maintenance level is reached or the organism could switch to shrinkage. Once the organism is on a decreasing vitality path, it will eventually reach maintenance but cannot get back on an increasing path. For the example discussed above this would imply that initially π equals one, at the onset of reproduction π switches to zero and at the age when λ_ψ rises above one, π rises up to its maximum permissable level, i.e. maintenance $\pi = \pi_0$. In this way, the problem encountered without the constraint disappears. There is an optimal solution inside the range of feasible trajectories of π. It is clear that this strategy could be beaten in the unconstrained model, but for the modified, constrained version π_0 is the best feasible strategy.[11]

Let me formalize the constrained model. I will introduce a second control variable defined as the time point $T \epsilon [0, \infty]$ of switch between growth and shrinkage. Since life starts with growth, all ages younger than T are associated with increasing vitality. Note that theoretically, optimal strategies can imply pure shrinkage ($T = 0$; initially being in shrinkage mode) as well as pure growth strategies ($T = \infty$; never switch

[9] As discussed in more detail in a forthcoming paper by Kenneth Wachter and me.
[10] Ditto.
[11] It may well be that for some species it is physiologically impossible to switch from a senescent back to a non-senescent trajectory, i.e. from increasing back to decreasing mortality, and thus my constraint might not be unrealistic.

to shrinkage). Thus, the change in vitality is given by

$$\dot{\psi} \;=\; max[f(\psi,\pi),\,0] \;\text{ for }\; a \;\text{ in }\; [0,T)\;, \tag{5.35}$$

where f is the same as in the unconstrained model given in (5.14). Equation (5.35) implies that any feasible strategy π associated with negative changes in vitality in the unconstrained model is now mapped on the zero-line for all ages before T. After T the change in vitality is given by

$$\dot{\psi} \;=\; min[f(\psi,\pi),\,0] \;\text{ for }\; a \;\text{ in }\; [T,\infty]\;. \tag{5.36}$$

Now the objective function can be written as

$$\max_{\pi,T}\;\left(\int_0^T l(x)\,m(x)\;dx \right. \tag{5.37}$$
$$\left. +\; e^{-\phi(T)}\;\int_T^\infty e^{-(\phi(x)-\phi(T))}\,m(x)\;dx\right)\;,$$

using general notation for survival $l(x)$ and reproduction $m(x)$. Applying (5.33) this expression is equivalent to

$$\max_{\pi,T}\;\left(\int_0^T l(x)\,m(x)\;dx \right. \tag{5.38}$$
$$\left. +\; e^{-\phi(T)}\;\max_{\pi}\int_T^\infty e^{-(\phi(x)-\phi(T))}\,m(x)\;dx\right)\;.$$

It turns out that the constrained model consists of two optimization problems that are linked. The inner maximization problem in (5.38) is given by

$$\max_{\pi}\;\int_T^\infty e^{-(\phi(x)-\phi(T))}\,m(x)\;dx\;. \tag{5.39}$$

Note that the mode of change in vitality for the inner problem is shrinkage. The initial conditions for the inner problem are

$$\psi^S(T) \;=\; \psi^G(T) \tag{5.40}$$

and

$$\phi^S(T) \;=\; \phi^G(T)\;. \tag{5.41}$$

The upper case letters S and G indicate the different modes of change for the inner and the outer problem. The outer problem (where vitality can not decrease) sets initial conditions for the inner problem (where vitality can not increase) but the inner problem can otherwise be solved independently of the outer one.

The change in vitality for the inner problem is given by (5.36) and the transversality conditions are as before

$$\lambda_{\psi}^{S}(\infty) \;=\; \lambda_{\phi}^{S}(\infty) \;=\; 0\,. \tag{5.42}$$

The Hamiltonian function is essentially the same as in the unconstrained model, and the solutions for λ_{ψ}, λ_{ϕ} and ψ remain the same, just with new initial vitality level $\psi(T)$. An optimal solution for this problem again has to be monotone, therefore vitality can only decline until zero or some level that is maintained.

The outer maximization problem depends on the maximum value of the objective of the inner problem. The initial conditions of the outer problem are

$$\psi^{G}(0) \;=\; 1 \tag{5.43}$$

and

$$\phi^{G}(0) \;=\; 0 \tag{5.44}$$

while now the transversality conditions are unusual in the way that they connect the outer problem to the inner problem,

$$\lambda_{\psi}^{G}(T) \;=\; e^{-\phi(T)}\lambda_{\psi}^{S}(T) \tag{5.45}$$

and

$$\lambda_{\phi}^{G}(T) \;=\; e^{-\phi(T)}\lambda_{\phi}^{S}(T)\,. \tag{5.46}$$

These conditions ensure that the problems are properly related.

The Hamiltonian function for the outer problem is again the same as for the unconstrained model, taking into account that the change in vitality is now given by (5.35). Effectively, the constraint on the change in vitality is a constraint on the range of possible trajectories of π to the subset $\pi\epsilon[\pi_0, 1]$. Note that the second control variable T marks start and end conditions for the two connected problems but does not enter the associated Hamiltonian functions. Further note that if one applies conditions (5.43), (5.45) and (5.46) in the solutions for ψ, λ_{ψ} and λ_{ϕ} it turns out that the solutions remain the same for both problems.

The Hamiltonian for the constrained model is

$$H \;=\; H^{G} \;+\; e^{-\phi(T)}\, H^{S} \tag{5.47}$$

where H^{G} is different from zero in $[0, T)$ and H^{S} is different from zero in $[T, \infty]$. Both H^{G} and H^{S} are essentially the same except for $min/max[f, 0]$ (see (5.35) and (5.36)) and the different initial values for vitality. To find the optimal value for T it is not necessary, however, to go through the procedure of maximizing H w.r.t. T. There is a simpler

way: Basically, one has two optimization problems that can be solved separately and be spliced together for any given value of $\psi(T)$. Solving the two problems for any $\psi(T)$ the optimal $\psi(T)$ can subsequently be found by simple maximization.

The big advantage of introducing the second control variable T is that for the modified model an optimal solution for vitality has to be monotone not for one whole but for two distinct time intervals corresponding to two distinct yet related optimization problems. Between age zero and age T vitality can increase and between age T and infinity vitality may go down but must not increase. Obviously, all solutions to the modified model are subsets to solutions of the initial, unconstrained model. As such, optimal solutions for the initial model are optimal solutions for the modified model. But for ranges of parameters that have no optimal solution in the unconstrained model there might well be an optimal solution in the constrained model.

One runs into the paradoxical situation that adding restrictions opens up opportunities. Though the modified model is a constrained version of the initial model it extends the range of the parameter space for which optimal solutions exist, and in this way it also extends the range of possible qualitative trajectories. In particular, the constrained model has a solution for the case where both η parameter exceed one, namely full growth at $\pi = 1$ followed by full reproduction at $\pi = 0$ accompanied by a decline in vitality and thus increasing mortality, eventually followed by maintenance at $\pi = \pi_0$, when mortality plateaus. Thus, the constrained model can explain senescent as well as non-senescent life history strategies while the unconstrained model can only explain non-senescence.

5.3.1 Expected Solutions

In the following I list the expected solutions for all subsets of the parameter space for combinations of η_r and η_g, depending on whether those parameters are smaller, equal or larger than one:

- $\eta_r < 1$ & $\eta_g < 1$**:** If both η's are smaller than one the Hamiltonian is concave in π. An optimal solution exists and intermediate investment is expected. Parallel growth and reproduction followed by maintenance should be optimal. The optimal strategy for the second control should be to not throw the switch ($T = \infty$). I will call this strategy *Enhancement* (one may also call it *Negative Senescence* as in Vaupel et al. 2004).

- $\eta_r < 1$ & $\eta_g = 1$ **or** $\eta_r = 1$ & $\eta_g < 1$ **:** If one of the η's is equal to one and the other is smaller than one, then the Hamiltonian is concave in π. Therefore Enhancement should be optimal.
- $\eta_r = 1$ & $\eta_g = 1$**:** If both η's are equal to one the Hamiltonian is linear in π. As discussed in Chap. 4 the optimal solution involves a period of full growth corresponding to $\pi = 1$ followed by maintenance at π_0. I will call this strategy *Sustenance*.
- $\eta_r > 1$ & $\eta_g > 1$**:** If both η's are larger than one the Hamiltonian is convex in π. As discussed above, my modified model yields optimal solutions corresponding to full growth ($\pi = 1$) followed by full reproduction ($\pi = 0$) followed by maintenance ($\pi = \pi_0$). I call this kind of strategy *Senescence*.
- $\eta_r > 1$ & $\eta_g = 1$ **or** $\eta_r = 1$ & $\eta_g > 1$**:** If one of the η's is equal to one but the other is larger than one the Hamiltonian is convex in π and Senescence should be optimal.
- $\eta_r > 1$ & $\eta_g < 1$ **or** $\eta_r < 1$ & $\eta_g > 1$**:** If one η is larger than one and the other is smaller than one, the balance between η_r and η_g determines whether the Hamiltonian is concave or not. These exotic cases need further investigation.

Exotic Strategies

In the beginning of this section I derived the condition that determines whether the Hamiltonian function is concave or convex (see inequality (5.26)). Strikingly, the Hamiltonian function H can be concave or convex, if one η is larger and the other is smaller than one, depending on the value of π. To see this more clearly one can equate both sides of inequality (5.26) to find the point π_c that separates the two regions of π that are associated with concave vs convex H:

$$\pi_c = \frac{1 - \eta_g}{\eta_r - \eta_g} , \tag{5.48}$$

lower case c indicating "cut between convex and concave". With a minute of thought one can establish that $0 < \pi_c < 1$, i.e. the cut point truly lies within the interval $[0, 1]$.

Interestingly, depending on whether η_r or η_g is the parameter that exceeds one, it is the interval to the left or to the right side of π_c that corresponds to concave H. This should have important implications for the associated optimal strategies. It should matter whether it is growth or reproduction that faces increasing vs. decreasing returns to scale.

From inequality (5.26) one can show that the concave zone of H lies to the left of π_c when η_r exceeds one. An optimal π^*-strategy that does not run into my constraint has to obey

$$\pi^* < \pi_c \text{ if } \eta_r > 1 . \tag{5.49}$$

Conversely, the concave zone of H lies to the right of π_c if η_g exceeds one. An optimal π^*-strategy that does not run into my constraint has to obey

$$\pi^* > \pi_c \text{ if } \eta_g > 1 . \tag{5.50}$$

A helpful insight can be gained from (5.48): As η_r approaches one the cut point π_c moves towards one,

$$\lim_{\eta_r \to 1} \pi_c = 1 . \tag{5.51}$$

Equations (5.49) and (5.51) together imply that – as η_r approaches the limit of 1 from above – the whole interval will become concave. The strategy converges to Enhancement. This is intuitively right since one is back to the well behaved case of $\eta_r = 1 \,\&\, \eta_g < 1$. For values of $\eta_r < 1$, (5.50) and (5.51) imply convergence to the case of $\eta_r = 1 \,\&\, \eta_g > 1$, where the whole interval corresponds to convex H, so the strategy converges to Senescence.

Analogously, it holds that

$$\lim_{\eta_g \to 1} \pi_c = 0 . \tag{5.52}$$

If η_g approaches 1 from below, then the strategy converges to senescence, since $\eta_g < 1$ means $\pi < \pi_c$, so the region where H is concave disappears as $\pi_c \to 0$. If η_g approaches 1 from above, then the strategy converges to Enhancement, since $\eta_g > 1$ means $\pi > \pi_c$, so the region where H is concave covers the whole interval.

These dynamics can also be seen from

$$\frac{\partial \pi_c}{\partial \eta_r} = -\frac{1 - \eta_g}{(\eta_r - \eta_g)^2} \quad \begin{cases} < 0 \text{ if } \eta_g < 1 \\ > 0 \text{ if } \eta_g > 1 \end{cases} \tag{5.53}$$

and

$$\frac{\partial \pi_c}{\partial \eta_g} = \frac{1 - \eta_g}{(\eta_r - \eta_g)^2} - \frac{1}{\eta_r - \eta_g} \quad \begin{cases} < 0 \text{ if } \eta_g < 1 \\ > 0 \text{ if } \eta_g > 1 . \end{cases} \tag{5.54}$$

What can be deduced about the shape of possible strategies? It is hard to predict the expected optimal solutions. Presumably, some part of a strategy should correspond to extreme cases of $\pi = 1$, or $\pi = 0$ followed by maintenance $\pi = \pi_0$ and the other half of the strategy could be intermediate investment, i.e. either parallel growth and reproduction or parallel shrinkage and reproduction. In the latter case, slower than exponential senescence would be optimal.

For $\eta_g > 1$ the well-behaved area is right of π_c (i.e. above π_c), thus larger values of π up to one correspond to concave H. Therefore, a smooth transition from $\pi = 1$ to lower values is expected. This suggests parallel growth and reproduction. Hence, the "good" part of the strategy would be in the beginning of life. Therefore, the second part of life might correspond to the extreme case where the constraint of my constrained model comes into action.

For $\eta_r > 1$ the well-behaved area is left of π_c (i.e. below π_c), thus smaller values of π down to zero correspond to concave H. Therefore, a distinct jump of π from $\pi = 1$ to some lower value is expected. Since the case of $\eta_r > 1$ mirrors the one of $\eta_g > 1$, I would deduce that now the second part of the strategy is the "good" one. Thus, full growth followed by slower than exponential senescence could be an optimal strategy.

The discussion above suggests that there are several stages of life. For my models, life always starts with a period of development in which an organism grows and mortality falls but there is no fertility. Then there are four possibilities: First, the organism could maintain itself at a constant level of vitality, mortality and fertility – I call this "sustenance". Second, the organism could start to reproduce but continue to grow, with declining mortality – I call this "enhancement". Third, the organism could reproduce as much as possible, with mortality rising exponentially – this I call "senescence". Fourth, the organism could reduce the increase in mortality and decline in vitality by diverting some resources from fertility to repair – I call this "subsenescence". In my vitality model, the first stage is always development and the last stage is always maintenance. Thus, possible life history stages resulting from my model are development, enhancement, senescence, subsenescence and maintenance.[12]

[12] Note that maintenance and Sustenance both pertain to cases of constant mortality and fertility. I use Sustenance with capital letter to describe a life history *strategy*, namely that of development followed by maintenance, whereas maintenance with lower case letter simply refers to one *phase* of a life history. Analogously, I use Enhancement and Senescence with capital letters to describe life history strategies while the same terms in lower case letters refer to phases of a life history.

5.4 Numerical Results

While it is possible to write down integral equations that can explicitly be solved for the cases where $\eta_r > 1 \,\&\, \eta_g > 1$, finding solutions for the remaining η parameter space is difficult to do analytically. To solve for the age-trajectories of optimal investment, vitality, mortality and fertility, I implemented an algorithm based on Bellman's approach of dynamic programming (see App. A.3).

Applying this algorithm[13] I gain particular solutions for each part of the η parameter space and can check, whether the general shape of my numerical results fits the expected solutions based on my theoretical considerations in Sect. 5.3.1.

5.4.1 The Five Varieties of Life History Strategies

Five different types of optimal strategies can be found to result from this model. They are:

- *Sustenance* – development followed by maintenance,
- *Enhancement* – development followed by enhancement followed by maintenance
- *Senescence* – development followed by senescence followed by maintenance
- *Subsenescence* – development followed by subsenescence followed by maintenance
- *Ensenescence* – development followed by enhancement followed by senescence followed by maintenance

Note that this variety of strategies includes senescent as well as non-senescent life histories as predicted by my theoretical considerations above. The following figures exemplify particular optimal life history strategies for particular parameter values of η_r and η_g to show the scope of possible solutions and to emphasize the major importance of those two parameters.

Strategies Without Senescence

As expected, parameter values that are below one lead to Enhancement (see Figs. 5.3, 5.4 and 5.5). Note that for $\eta_r = 1 \,\&\, \eta_g = 0.5$ the onset of

[13] Due to numerical approximation errors caused by step length in vitality and investment, the algorithm sometimes leads to solutions that are pseudo maintenance, i.e. very slow senescence, where π is almost π_0. This pseudo maintenance converges to true maintenance when step length for vitality and investment are more and more reduced.

reproduction is abrupt, investment "falls down a cliff" whereas for $\eta_r = 0.5$ & $\eta_g = 0.5$ as well as $\eta_r = 0.5$ & $\eta_g = 1$ the onset of reproduction comes smoothly. Thus, if reproduction faces constant returns to scale in investment, reproduction starts abruptly while decreasing returns to scale imply a smooth transition (in agreement with my theoretical considerations in Sect 5.3.1).

If both parameters equal one, then - as predicted - Sustenance is optimal, as can be seen in Fig. 5.6.[14]

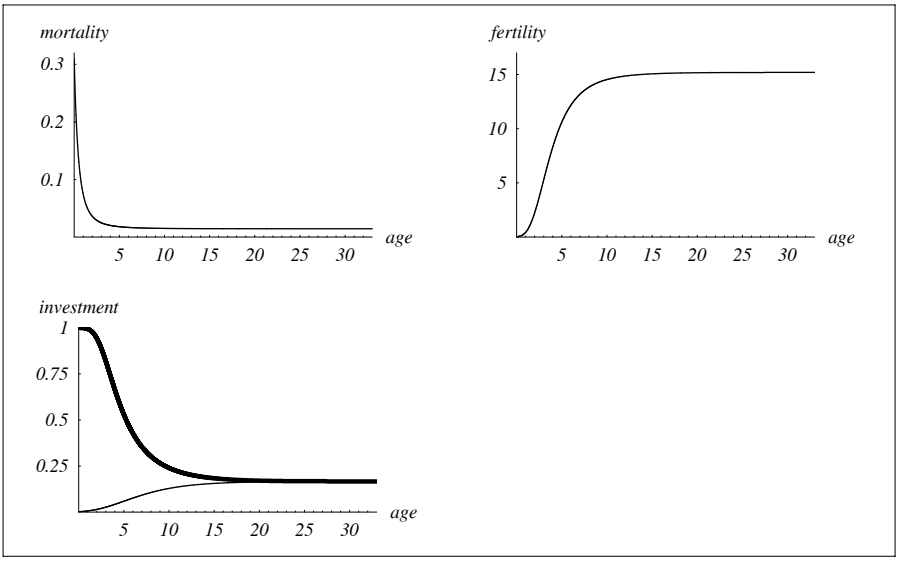

Fig. 5.3. Example Enhancement: $\eta_r = 0.5$ and $\eta_g = 0.5$

Strategies with Senescence

Strategies with senescence emerge as the η parameters start to exceed one as exemplified in the following figures. Note the difference between Fig. 5.7 and Fig. 5.8: a higher value of η_r leads to lower vita-

[14] For this and all the following figures in this section, the thick line in the lower left graph depicts the optimal investment strategy across age and the thin line depicts the corresponding level of investment π_0 required for maintenance.

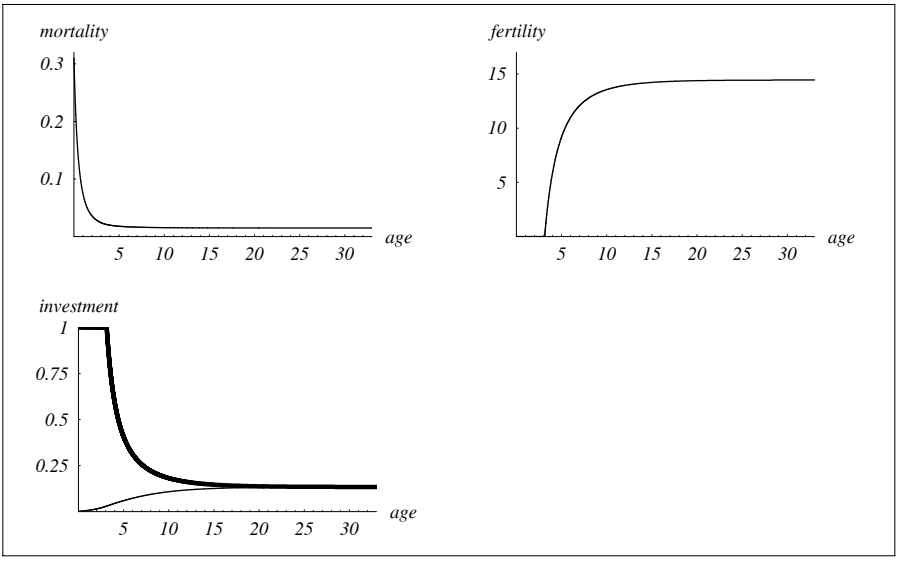

Fig. 5.4. Example Enhancement: $\eta_r = 1$ and $\eta_g = 0.5$

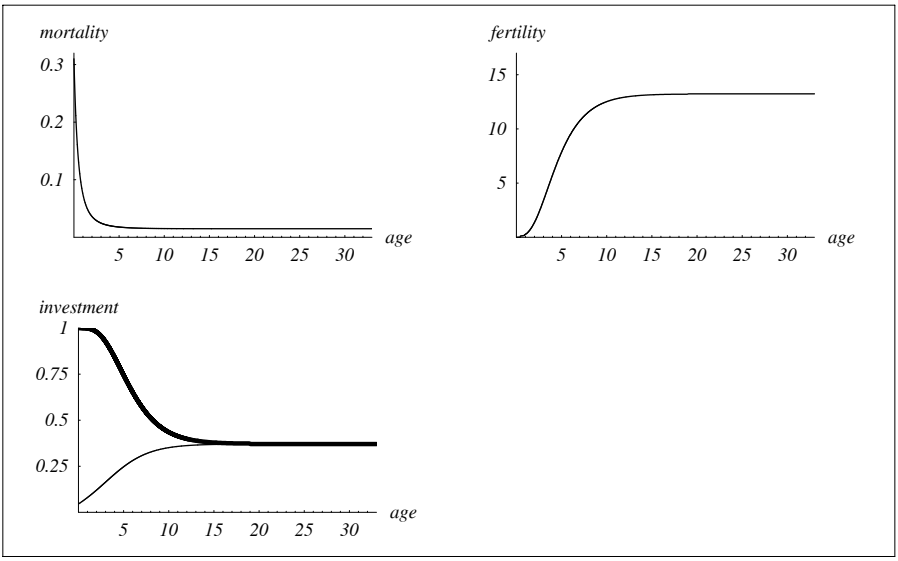

Fig. 5.5. Example Enhancement: $\eta_r = 0.5$ and $\eta_g = 1$

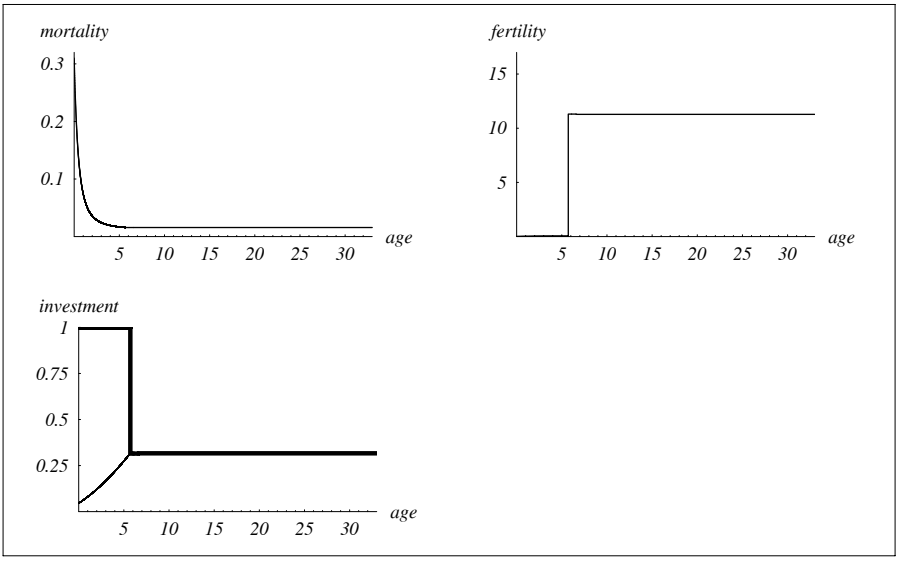

Fig. 5.6. Example Sustenance: $\eta_r = 1$ and $\eta_g = 1$

lity at maintenance and thus higher mortality. Interestingly, mortality increases only a little bit (relative to initial mortality[15] of 0.3) before reaching a plateau. For both η's equal to 3, Fig. 5.9 depicts the associated strategy (note that the time axis is double as long as in the previous figures). If the time units corresponded to years this setting of parameters captures a life history strategy of with humanesque features. Mortality falls until the age of maturity at about 13. Thereafter, mortality rises exponentially at a constant rate $\delta = 0.1$. Reproduction follows a hump-shaped curve. At the age of 60 reproduction drops to close to zero, corresponding to its level at the plateau. Note that virtually all lifetime reproduction is realized before this age and further contributions of later age classes are negligible. My model does not lead to true menopause – reproduction continues albeit at a very low level, and mortality remains constant. In a human life history reproduction, at least for females, should cease and mortality should keep on rising exponentially. Clearly, a model as simple as mine that is solely based on vitality can only capture a rough, humanesque pattern.

[15] Note that in Figs. 5.7, 5.8, 5.10, 5.11 and 5.13 the increase in mortality is barely visible because mortality is shown on a scale set by initial mortality, which is at the magnitude of 0.3. It should be emphasized, however, that the force of mortality is increasing substantially in all these figures relative to its level at reproductive maturity.

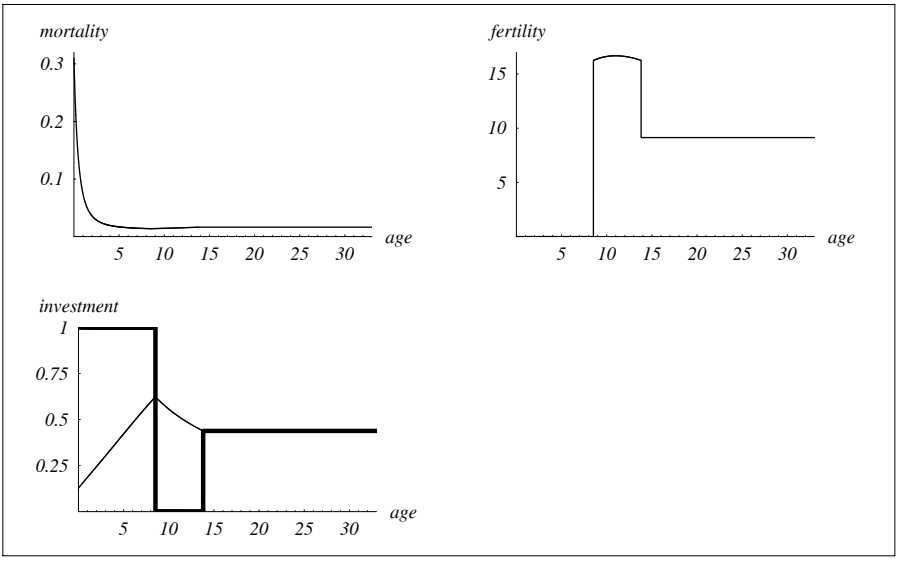

Fig. 5.7. Example Senescence: $\eta_r = 1$ $\eta_g = 1.5$

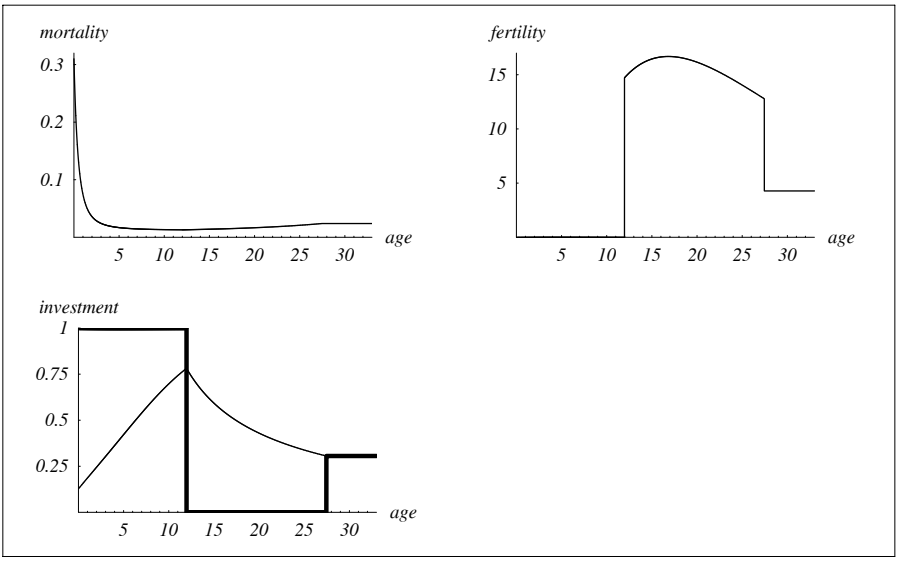

Fig. 5.8. Example Senescence: $\eta_r = 3$ $\eta_g = 1.5$

Ensenescence

Figures 5.10, 5.11, 5.12 and 5.13 show results that exemplify parameter combinations corresponding to areas where exotic strategies were

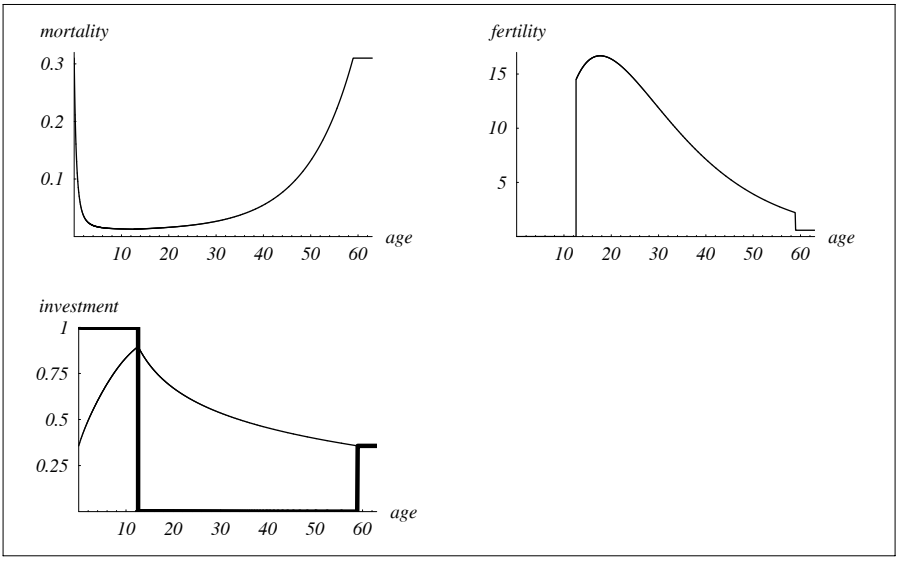

Fig. 5.9. Example Senescence (humanesque case): $\eta_r = 3$ $\eta_g = 3$

expected. And indeed, the strategies are really interesting: The combination of $\eta_r = 0.5$ & $\eta_g = 1.5$ and $\eta_r = 0.5$ & $\eta_g = 3$ (Figs. 5.10 and 5.11) depict strategies that include parallel reproduction and growth. An initial period of development ($\pi = 1$) is followed by a period of parallel growth and reproduction ($\pi > \pi_0$), leading into a period of exponential senescence due to a drop in the strategy to $\pi = 0$ and eventually reaching a level of vitality that will be maintained ever after ($\pi = \pi_0$). I call this strategy *Ensenescence* because it includes a period of enhancement and a period of senescence. Figure 5.11 shows the effect of increasing η_g, which is to widen the period of senescence and flatten the dip.

Note that the dip in the strategy depicted in Figs. 5.10 and 5.11 is not an artefact. I calculated many different η combinations for the case of $\eta_r < 1$ and $\eta_g > 1$ and always found this peculiar strategy.

Senescence and Non-senescence Close Together

The range of parameters $\eta_r > 1$ and $\eta_g < 1$ reveals a surprise: For $\eta_r = 1.5$ & $\eta_g = 0.5$ (Fig. 5.12) the optimal strategy is Enhancement. Investment shows a cliff at the onset of reproduction as predicted from $\eta_r > 1$ and the discussion of π_c. This kind of enhancement differs from the very early and gradual start of reproduction shown in Figs. 5.3–5.5

Fascinatingly, there is yet another strategy to be found in this parameter region. Increasing η_r to 3 as shown in Fig. 5.13 moves the non-senescent life history towards a senescent one. Parallel reproduction and growth is not favored anymore. Instead the optimal strategy is development ($\pi = 1$) followed by slower than exponential senescence[16] ($0 < \pi < \pi_0$), followed by maintenance ($\pi = \pi_0$). I call this strategy *Subsenescence*.

If the optimal strategy is Enhancement the vitality trajectory is monotone increasing (and my model constraint is not in action). If, however, Subsenescence is optimal the vitality trajectory first increases and then decreases (my model constraint applies)[17]. Thus, for increasing returns to scale in reproduction but decreasing returns to growth and maintenance, both senescent and non-senescent strategies can be optimal.

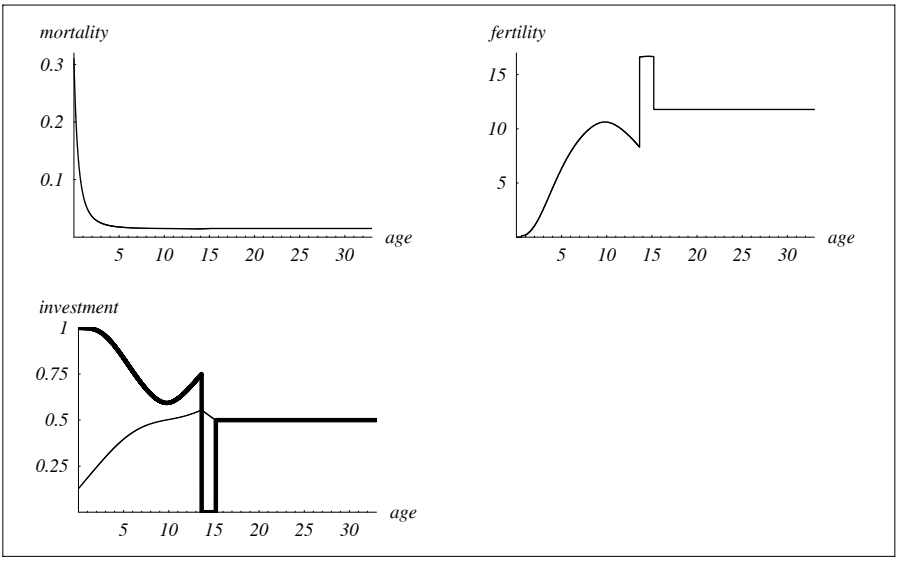

Fig. 5.10. Example Ensenescence: $\eta_r = 0.5$ $\eta_g = 1.5$

[16] This is a true strategy and not an artifact, the strategy is robust with decreasing step length.

[17] The cliff when π drops from one to below π_0 shows that the investment trajectory jumps over the convex area of π's which lies above π_c for this range of η parameters.

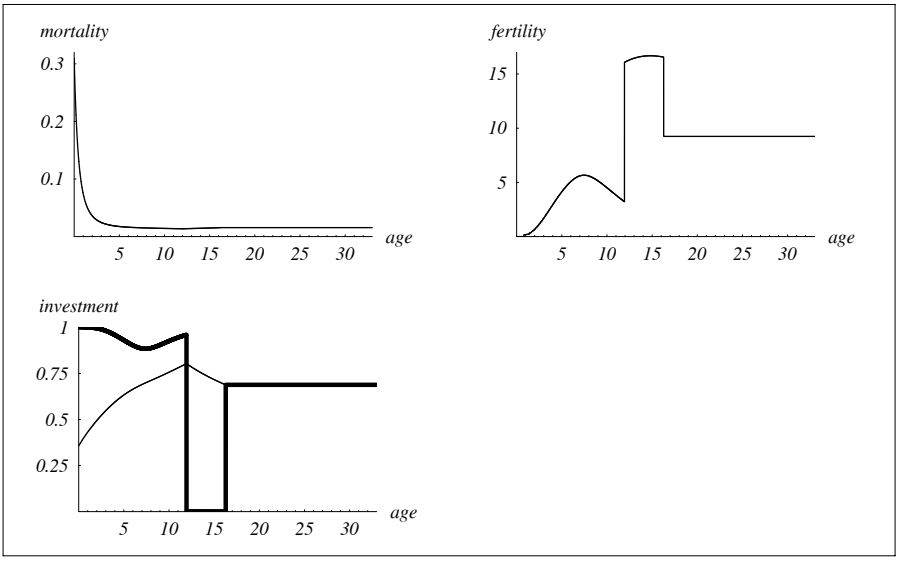

Fig. 5.11. Example Ensenescence: $\eta_r = 0.5$ $\eta_g = 3$

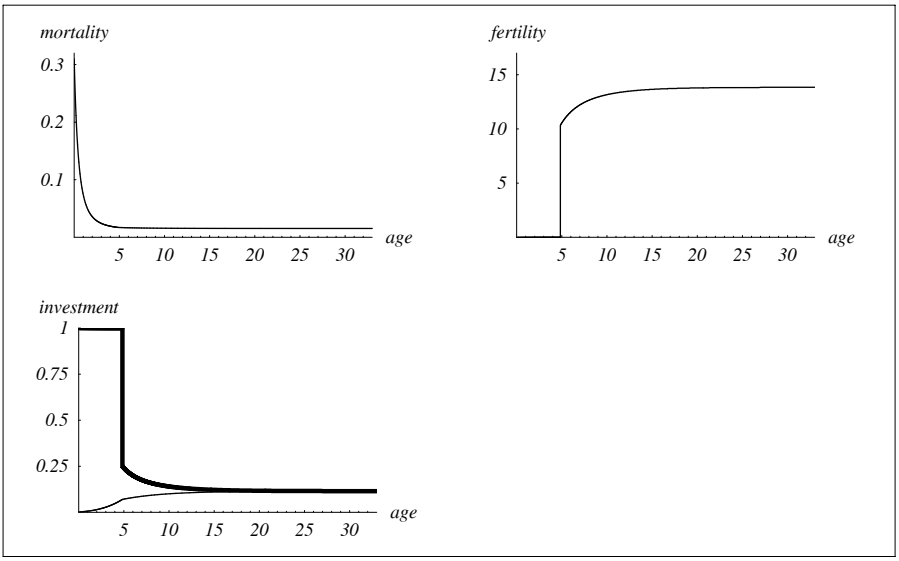

Fig. 5.12. Example Enhancement: $\eta_r = 1.5$ $\eta_g = 0.5$

Strategies Across η Parameters

The matrix of strategies in Fig. 5.14 shows how strategies change across the range of η_r and η_g. Each graph displays the optimal investment

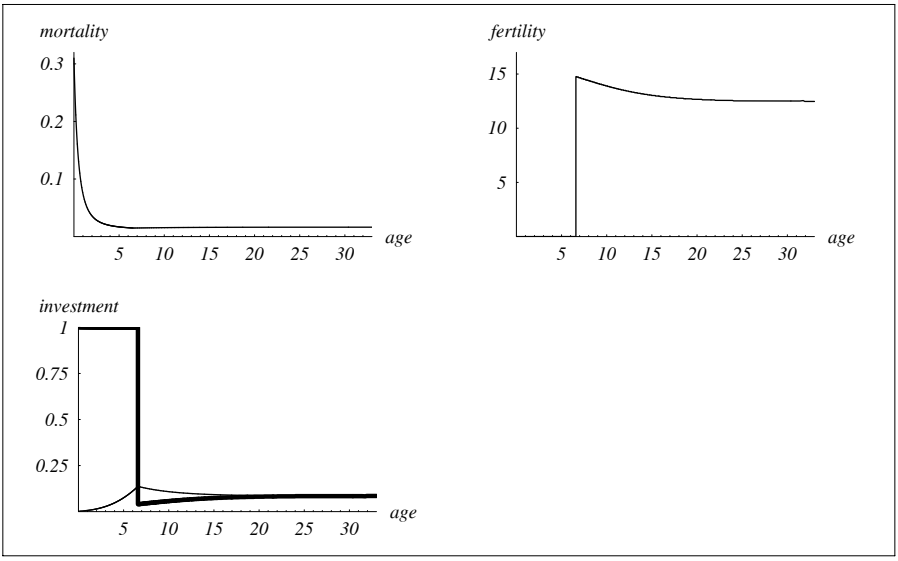

Fig. 5.13. Example Subsenescence: $\eta_r = 3$ $\eta_g = 0.5$

strategy π^* across age. In each graph, initially π^* equals one and drops below one at the age of maturity[18].

Consider for instance the first column, with η_g fixed at 0.5. As η_r increases from 0.5 to 1.5, the onset of reproduction becomes more abrupt. When η_r is 3.0 the optimal strategy is Subsenescence rather than Enhancement: there is no longer a period of parallel reproduction and growth.

Another interesting example concerns the upper right corner of Fig. 5.14. The strategy in the corner is Ensenescence. The name "Ensenescence" combines two strategies – Enhancement and Senescence, and it is those two strategies that Ensenescence converges to as either of the η parameters changes. My computational results (not presented here) show how a reduction in η_g from 1.5 to 1 drives the strategy towards Enhancement. As η_g decreases the valley of the dip becomes more and more pronounced, eventually flattening out and the period of senescence becomes shorter and eventually disappears. When instead η_r increases from 0.5 to 1, Ensenescence converges to Senescence. The

[18] Note that the thin line that depicts the required investment for maintenance originating from the lower left corner of each graph starts off at higher and higher values as η_g increases along each row. The reason are higher costs of maintenance due to larger values of η_g (compare (5.7)).

dip becomes more and more shallow, eventually disappearing and the period of senescence lengthens[19].

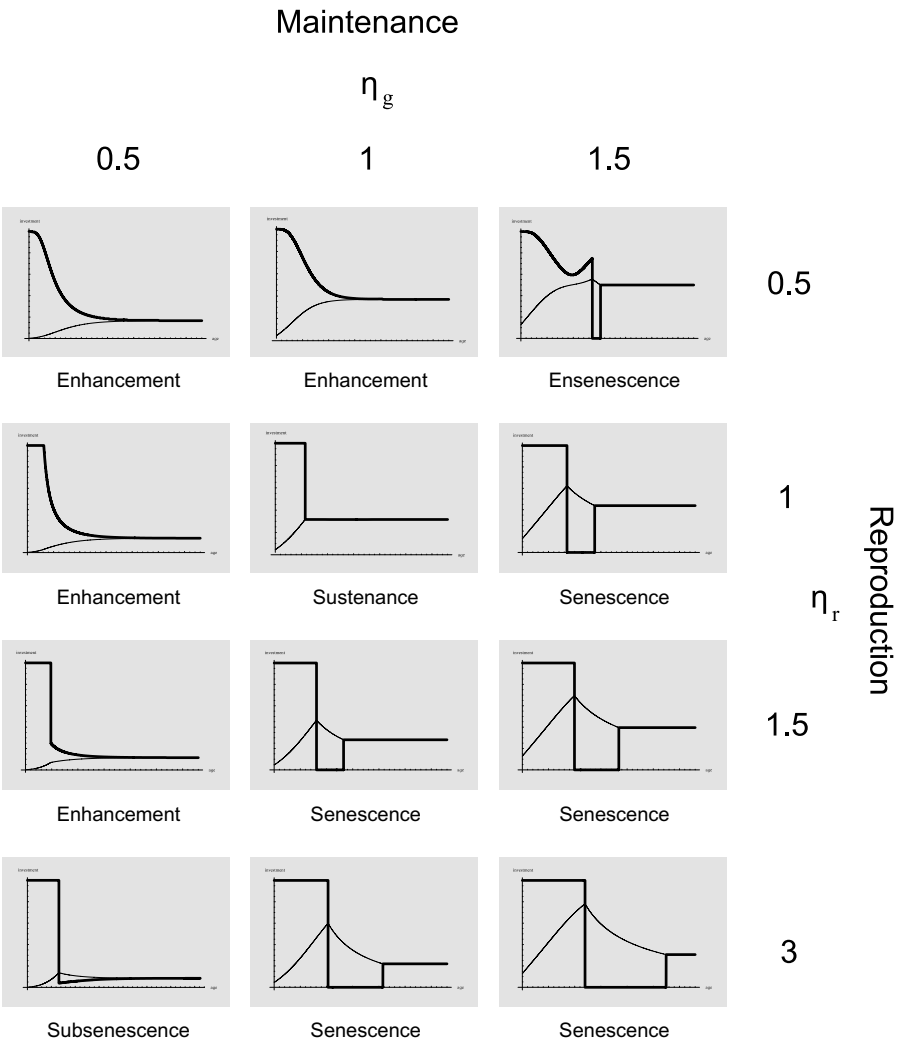

Fig. 5.14. Overview of optimal investment strategies across η parameters

[19] I believe that the increase in π just before the onset of senescence is due to the fact that the organism invests more in growth and survival to ensure survival to the beneficial phase of full reproduction.

5.4.2 When Senescence Is Optimal and when It Is Not

The previous section concentrated on qualitative age-patterns of mortality. Several of the mortality and fertility trajectories increase over some part of reproductive life but decrease or remain constant over other parts of reproductive life. How should one decide whether those life histories are senescent or non-senescent? How could one describe the 'degree of senescence' of a life history strategy? This section offers a measure of the degree of senescence and subsequently sheds light on the characteristics that determine whether senescent or non-senescent life histories are optimal.

The Degree of Senescence

My suggestion for a definition of 'senescence' in Chap. 1 applies to particular age-groups. What criteria should be used to label a complete life-history strategy senescent or non-senescent? In the following, one way of approaching such a classification is suggested.

Whether a particular life history is classified as senescent or non-senescent can be determined by the proportion of lifetime reproduction that is realized at ages when mortality rises, i.e. when $\pi < \pi_0$. This senescence indicator, S, measures the degree of senescence for a particular life history strategy. S is given by

$$S = \frac{\sum_{x=0}^{\infty} J_x \, l_x \, m_x}{\sum_{x=0}^{\infty} l_x \, m_x} \, , \tag{5.55}$$

where $J_x = 1$ if investment in growth is below maintenance level ($\pi(\psi(x)) < \pi_0(\psi(x))$) and $J_x = 0$ if investment is greater than or equal to the amount required for maintenance of vitality. If $S = 1$, the strategy is fully senescent and if $S = 0$, the strategy is fully non-senescent. All values in between describe mixed strategies.

The Crucial Parameters

The crucial parameters that are responsible for the qualitative shape of an optimal life history strategy are the η parameters and the mortality parameters.

Figure 5.15 shows how different levels of mortality can influence the degree of senescence in a particular life history strategy depending on the range of η parameters. Each combination of η's corresponds to one graph. Each graph displays the degree of senescence, i.e. values of S

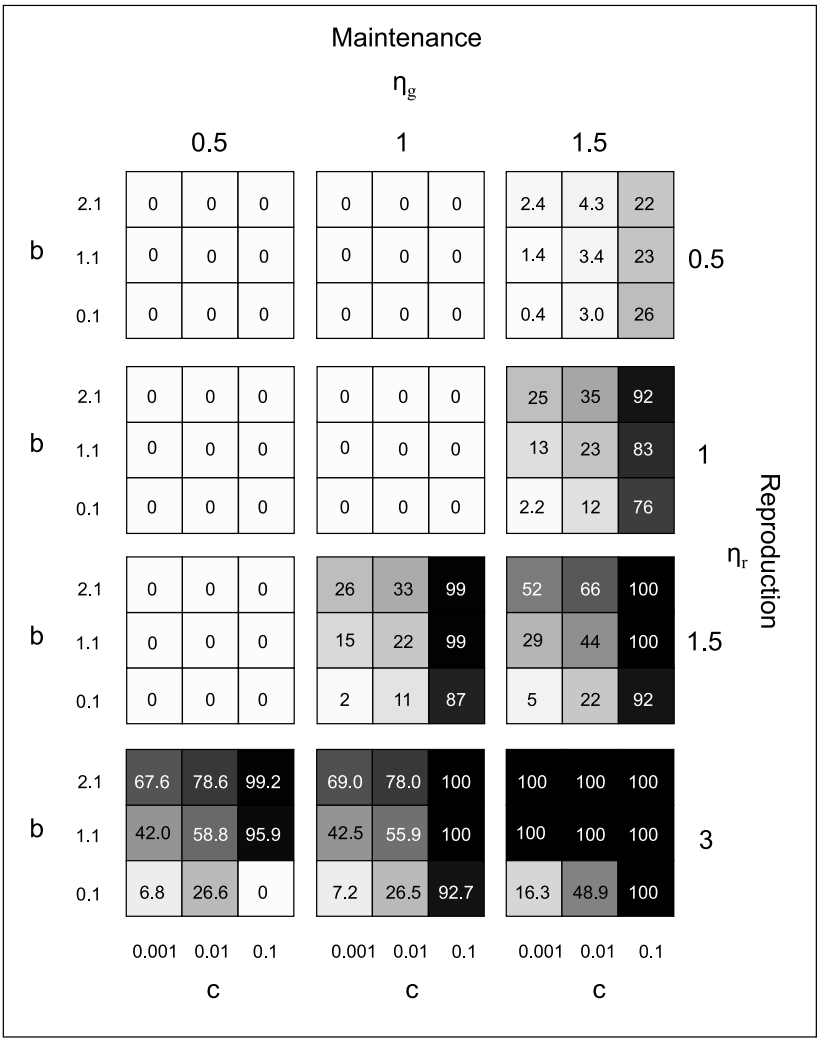

Fig. 5.15. Degree of senescence S, in%, across η and mortality parameters; *Color code: darker = more senescent; numbers refer to percent life time reproduction realized at ages when mortality increases*

across extrinsic[20] mortality c along the x-axis and intrinsic mortality b along the y-axis. Two general results can be derived from this figure:

- The upper left triangle of Fig. 5.15 is white; those ranges of parameters are associated with pure non-senescence, i.e. $S = 0$. Towards the lower right corner the areas become more and more shady and dark. Thus, higher levels of the η parameters generally correspond to higher degrees of senescence.[21]
- Inside each of the 12 graphs, lighter areas can be found towards the left and further down. Hence, higher levels of mortality – be it extrinsic (on the x-axis) or intrinsic (on the y-axis) – go hand in hand with higher degrees of senescence, in other words a larger fraction of lifetime reproductive success is realized during ages when mortality increases.

The influence of the η and mortality parameters on age and vitality at maturity can be seen in Figure 5.16, which follows the same logic as the previous figure. Clear gradients in shading from light to dark become apparent: from left to right, top to bottom and – inside each graph – right to left. Three general observations can be made:

- Higher levels of overall mortality are generally associated with smaller vitality at maturity, higher extrinsic mortality c in particular has this effect. Note, however, that higher intrinsic mortality b affects vitality at maturity in a nonlinear fashion: intermediate b leads to higher vitality at maturity, i.e. continued growth pays off via better survival at maturity. Too high a level of b, however, counterbalances this advantage because of increasing overall mortality – earlier maturation at a smaller level of vitality is favored.
- Higher levels of the η parameters are generally associated with larger vitality and thus a later age at maturity.
- For non-senescent strategies (white areas), vitality at maturity barely exceeding 50 % of the maximum vitality possible, i.e. development does not proceed beyond the point where the increase in vitality starts to slow down (The age-trajectory of vitality is s-shaped for $\pi = 1$). For senescent strategies vitality at maturity is generally higher than for non-senescent strategies, reaching about 75% and more percent of maximum vitality. Thus there is a distinct shift in vitality at maturity relative to maximum attainable vitality

[20] In this monograph I use the term "extrinsic" meaning "state-independent" and "intrinsic" meaning "state-dependent".

[21] A forth column for $\eta_g = 3$ would reveal the same pattern, mainly being dark; it is not displayed here.

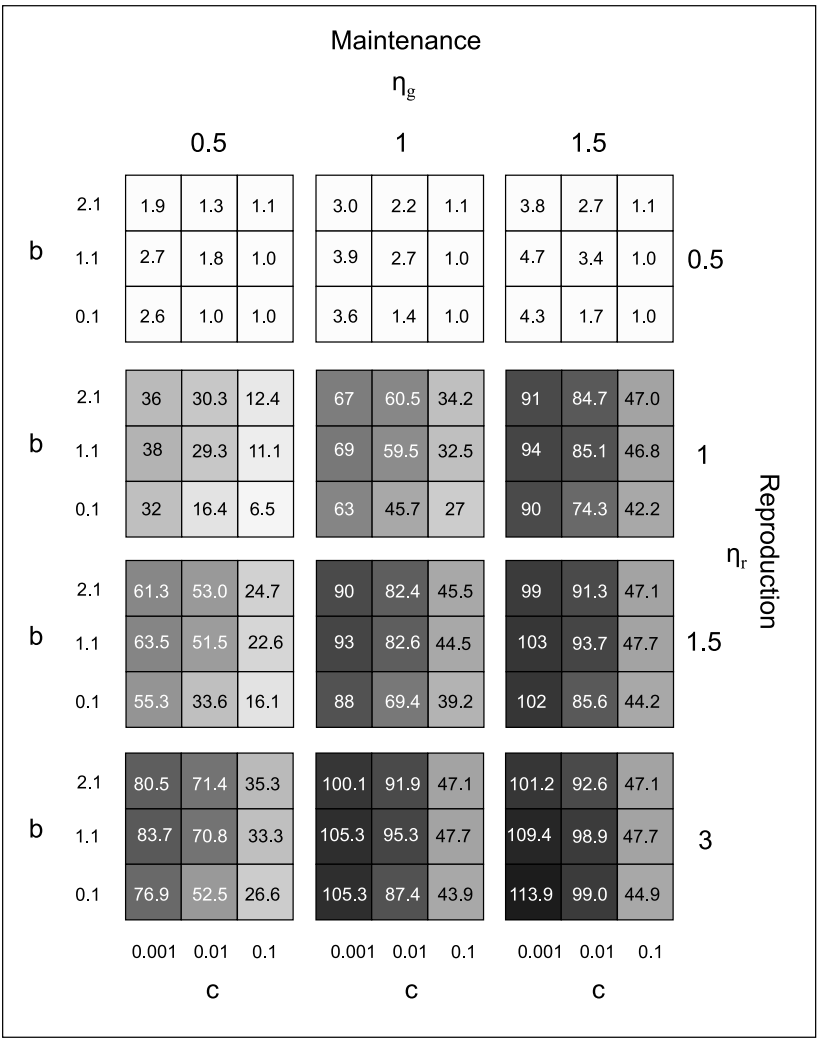

Fig. 5.16. Vitality at maturity across η and mortality parameters; *Color code: darker = larger vitality at maturity*

Ψ between senescent and non-senescent strategies(as can be seen from the distinct shift between light and dark areas).

In short, higher levels of the η parameters correspond to a higher degree of senescence as well as greater vitality at maturity. Higher levels of mortality lead to higher degrees of senescence but reduce vitality at maturity. Non-senescent strategies are associated with relatively[22] low vitality and thus early age at maturity, while senescent strategies are associated with relatively large vitality and thus late age at maturity.

A Peculiar Little White Square

Figure 5.15 reveals a peculiarity: the lower right little square of the lowest left graph is white, there is no senescence. Given the patterns elsewhere in the figure one would expect a value of S above 80. While all strategies for $\eta_r = 3$ and $\eta_g = 0.5$ are Subsenescence, the highest value of $c = 0.1$ at the lowest value of $b = 0.1$ corresponds to Enhancement. Why?

First note that all except one (namely Ensenescence) of my strategies exhibit monotone state trajectories after reproductive maturity. Thus, for those strategies the relative level of vitality at maturity to vitality at maintenance can predict whether vitality increases or decreases across adult ages. If vitality at maturity is larger than vitality at maintenance, the organism must have been shrinking after maturity to reach that lower level, thus there must have been senescence. If vitality at maturity is smaller than vitality at maintenance, the organism must have been growing after maturity to reach that higher level, thus there must have been non-senescence.

If one secondly recalls that non-senescence implies small and senescence implies large vitality at maturity, then it seems that vitality at maturity and vitality at maintenance hold the key to explain the peculiar little white square.

Influence of η Parameters

The influence of the η parameters on vitality at maturity and at maintenance is shown in more detail in Figs. 5.17 and 5.18 [23]:

[22] "Relative" here means relative to maximum vitality.

[23] Both figures are calculated keeping all other parameters unchanged at $b = 0.3$, $c = 0.01$, $k = 3$, $\kappa = 0.8$, $\delta = 0.1$ which correspond to a maximum attainable vitality of 123. Remember that vitality is a dimensionless variable so the specific value of 123 only means that maximum vitality is 123 times greater than vitality at age zero.

- Optimal vitality at maturity increases as either η_r or η_g increases, i.e. the steeper the returns to scale in either reproduction or growth and maintenance (stronger convexity), the later the age at maturity and the higher the acquired reproductive potential. Figure 5.17 visualizes this finding, both by reading the figure across the four lines from thinnest to thickest and by looking at each line from left to right.
- The ultimate level of vitality that will be maintained decreases as either η increases, i.e. the more convex the returns to scale in either reproduction or growth and maintenance, the later the age at maintenance and the lower the reproductive potential ultimately maintained. Figure 5.18 visualizes this finding, again both by reading the figure across the four lines from thinnest to thickest and by looking at each line from left to right.

Thus, for low values of the η's maturity happens early at a low level of vitality while a much higher level of vitality eventually is maintained. For high values of the η's, maturity is postponed, hence vitality at maturity is large, but the level that is eventually maintained is small. Consequently, there is a region of η parameters where vitality at maturity is smaller than vitality at maintenance, and there is a region of η parameters where vitality at maturity is larger than vitality at maintenance. This implies that there is a cut point where vitality at maturity equals vitality at maintenance. Figure 5.19 makes this verbal argument clearer by displaying the results from Fig. 5.17 and 5.18 together. The cut points for lines of equal thickness correspond to the values of η_r and η_g that lead to strategies where vitality at maturity equals vitality at maintenance[24].

The influence of the mortality parameter c on vitality at maturity and vitality at maintenance is shown Figure 5.20. Clearly, both vitality at maturity and vitality at maintenance decline as extrinsic mortality c increases, but since vitality at maturity declines faster than vitality at maintenance, there is a level of c where maturity and maintenance cross. At this cut point, Sustenance is the optimal strategy. For values of c smaller than the cut point, vitality at maturity exceeds vitality at maintenance, which implies a falling trajectory of vitality. Subsenescence is optimal. For values of c larger than the cut point, vitality at maturity falls below vitality at maintenance, which implies an increasing trajectory of vitality. Enhancement is optimal. Note that the curves

[24] Analogous figures not displayed here can be calculated for the case when η_g runs across the x-axis while different levels of η_r are given by lines of different thickness.

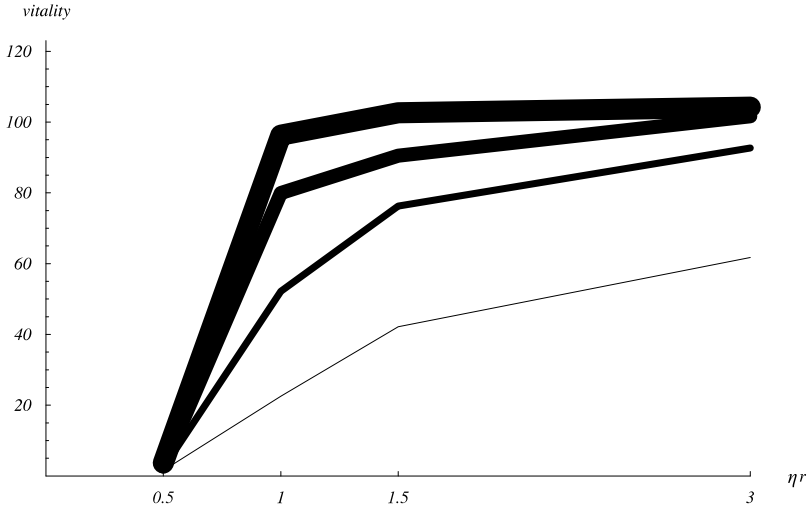

Fig. 5.17. Vitality at maturity across four distinct values of η_r plotted for the same four values of η_g; *line thickness being proportional to the value of* η_g

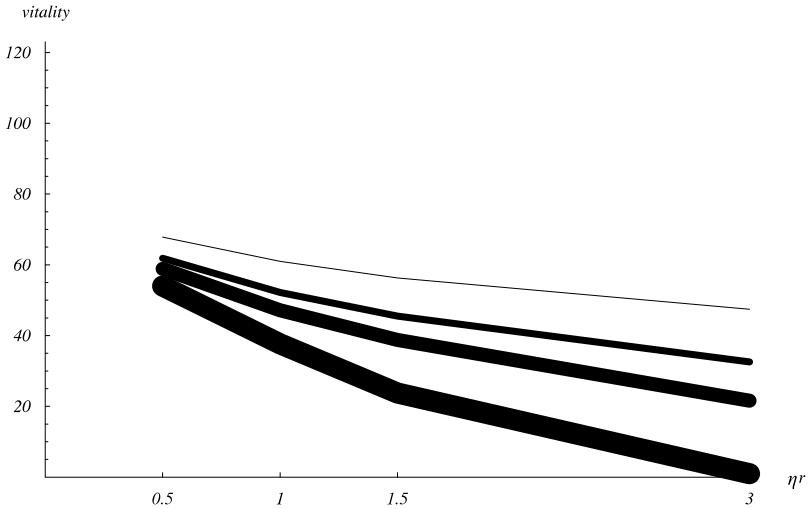

Fig. 5.18. Vitality at maintenance across four distinct values of η_r plotted for the same four values of η_g; *line thickness being proportional to the value of* η_g

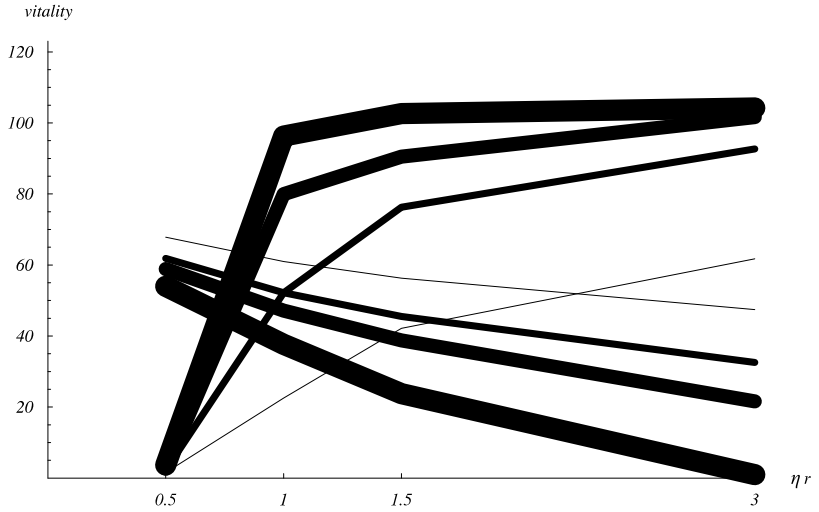

Fig. 5.19. Vitality at maturity and vitality at maintenance across four distinct values of η_r plotted for the same four values of η_g; *line thickness being proportional to the value of* η_g

for different values of b converge for large values of c, as the level of c dominates total mortality, i.e. when b is small compared to c [25].

In sum, the peculiar white little square is not so peculiar anymore. The explanation is straightforward: c can shift a strategy from Subsenescence to Enhancement by shifting vitality at maturity from above to below vitality at maintenance. At the cut point, where vitality at maturity is also the level of vitality that is maintained, Sustenance is optimal. Thus, a change in the extrinsic mortality parameter c can change a strategy from senescent to non-senescent, at least for the part of the η parameter space where $\eta_r > 1$ & $\eta_g < 1$.[26] The transition at the boundary between senescence and non-senescence is smooth, when caused by a change in c, contrary to the transition caused by changes in the η parameters. At the boundary between senescence and non-senescence that is set by the η parameters, the shift in vitality at maturity is distinct (compare Fig. 5.16).

[25] For values of c larger than those depicted in Fig. 5.15, non-senescence would have emerged in the graphs for values of $\eta_r > 1$ & $\eta_g < 1$. It is only because the range of c is not wide enough that this pattern did not become more apparent in this picture.

[26] From the analytic section in the beginning of this chapter it is clear that this range is the only one where mortality could shift the strategy from senescent to non-senescent

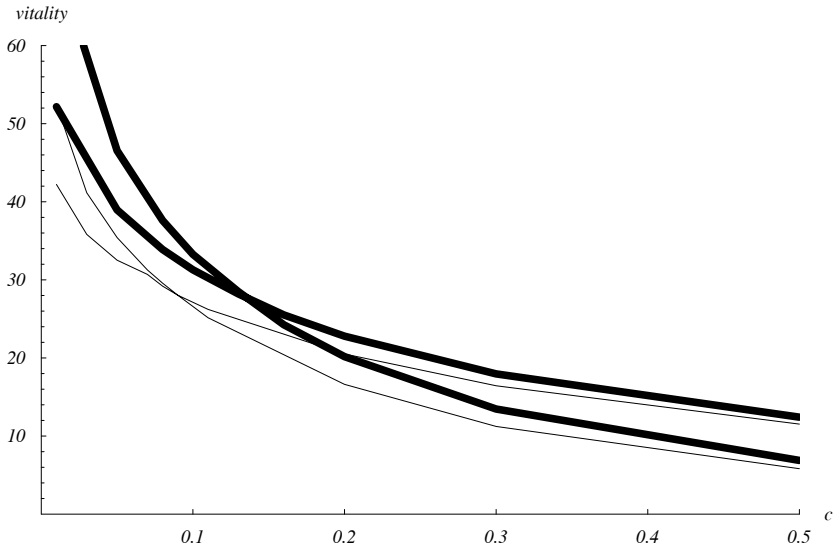

Fig. 5.20. Vitality at maturity and vitality at maintenance are plotted across extrinsic mortality c for values of parameter $b = 0.1$ and $b = 1.1$. Since the curves for $b = 2.1$ turned out to be very similar to those for $b = 1.1$, the case $b = 2.1$ is not shown. The pair of thick lines correspond to $b = 1.1$, the pair of thin lines correspond to $b = 0.1$. The initially upper curve of each pair depicts vitality at maturity, and the initially lower curve of each pair depicts vitality at maintenance.

5.5 Discussion

Whether a strategy follows a senescent or a non-senescent path is crucially dependent on the η parameters.

5.5.1 Senescence vs. Non-senescence

Non-senescent strategies are favored when the η parameters are below or equal to one. Values below one imply that returns to investment are decreasing, i.e. concave. An alternative verbal interpretation of decreasing returns to scale would be "cheap" or "efficient" investment, since a substantial amount of output can be realized with a small fraction of input. Thus, organisms with growth, maintenance and reproductive systems that can be used efficiently with only a fraction of total resources should follow non-senescent life history strategies.

Senescent strategies are favored when none of the two η parameters is below one and at least one of the two η parameters is above

one. Values above one imply that returns to investment are increasing, i.e. convex. An alternative verbal interpretation of increasing returns to investment would be "costly" or "inefficient" investment, since a significant amount of output can only be realized if all resources are concentrated on one process exclusively. Thus, organisms with growth, maintenance and reproductive systems that work best if used successively should tend to adopt senescent life history strategies.

Mixed strategies that include aspects of both senescent and non-senescent life histories can be found if one η- parameter is above and the other below one, i.e. when returns to investment are decreasing (concave, cheap, efficient) in one and increasing (convex, costly, inefficient) in the other process - either reproduction or growth and maintenance.

If growth and maintenance are efficient, then strategies can be either non-senescent (mortality never increases) or partially senescent (mortality increases slower than exponentially for some time after reproductive maturity) depending on the extrinsic hazard of death.

If reproduction is efficient, then it is optimal to grow and reproduce simultaneously for some time after reproductive maturity. Following this non-senescent phase of life, it is favorable to concentrate all resources on reproduction and let mortality increase exponentially (eventually levelling).

5.5.2 The η Parameters in Nature

How could one identify a species' η- parameter range? Species that show concave returns to scale in both reproduction and growth are species that can easily share resources between those processes and that do not gain much by specializing in either one of them. Organisms that are capable of vegetative propagation, where growth can be considered an investment in reproduction are candidates for this category. It is important to note, however, that species with the ability for this asexual mode of reproduction also have the ability to reproduce sexually. I believe that the values of η_r, i.e. the returns to investment in reproduction, are significantly different from each other for asexual vs. sexual reproductive mode. If asexual reproduction is associated with concave investment and sexual reproduction with convex investment, then the former case is associated with non-senescence and the latter case with senescence. An example is *Hydra oligactis* that sustains its state unless it starts to reproduce sexually [220]. Understanding the different returns to scale for asexual and sexual reproduction and its implications for senescence vs. non-senescence of a species is one interesting avenue for future research.

Non-senescence is part of a species's life history if at least one of the η parameters is below one. Life histories with at least one η parameter below one should involve a period of simultaneous growth and reproduction. All indeterminately growing species, as for example most trees and many fish fall into this category. Whether those species show exponential senescence later in life or not depends on whether growth and maintenance or reproduction is efficient.

Convexity or concavity in reproduction can be distinguished as follows: if organisms face convexity in reproduction, i.e. $\eta_r > 1$, then the onset of reproduction should be sudden and pronounced. If on the other hand $\eta_r < 1$, then the transition into adult ages happens smoothly. An analogous prediction for growth and maintenance can not be derived from my model.

An indication for concave (cheap and efficient) reproduction might also be given by number and size of offspring. Many small offspring (each single offspring only contributing an iota to life time reproduction) could indicate concave reproduction and few large offspring (each offspring requiring 'heavy' investment and contributing substantially to life time reproduction) could indicate convex reproduction.

Convexity with respect to reproduction could also mean that it is initially costly to build the required machinery that is necessary to reproduce at all. Lavish reproductive structures like huge fancy flowers, long stalks in bamboo, or feeding structures inside and outside the mothers body in animals could be an indication.

Convexity in growth and maintenance could mean that the machinery for growth and maintenance can only keep going efficiently if all energy is used for that purpose.

An efficient, cheap way of maintenance is "throw away and grow new". Disposing of damaged tissue does not require a lot of energy, indeed it should not cost much at all. If lost tissue can easily be replaced at reasonable costs without disturbing an organism's functioning and integrity it may be a cheaper strategy than repairing the damage that occurred. Repair of existing structure is costly because it requires error recognition, knowledge of the undamaged state that is about to be restored and the appropriate machinery to do so. Loosing body parts or tissue (like leaves in a plant) might not need much information or energy at all but happen more or less automatically. Replacement of parts and tissue (in case of the leaves) might only need the template for making that part or tissue but can do without any knowledge about the damaged one. The signal for producing a new leaf, for instance,

might solely require the signal of too little overall energy production by the remaining leaves.

Therefore organisms that are made of simple, repeated structure that can easily be discarded and regenerated when damaged, are candidates for concave returns to investment in maintenance and growth.[27] A thorough understanding of the $\eta-$ parameters in nature is an important avenue for future research.

5.5.3 The Mortality Paradox

In this section I further consider the case when reproduction is costly and growth and maintenance are cheap ($\eta_r > 1$ and $\eta_g < 1$). I found that an increase in parameter c, which captures state-independent mortality, can shift a strategy from Subsenescence to Enhancement. It appears that non-senescence is favored more strongly, the greater the extrinsic hazard. This is striking. Exactly the opposite has generally been stated – that a high risk of extrinsic death should favor senescence [212]. But my model predicts that this hypothesis is not true for species with low costs of maintenance and high costs of reproduction. What could explain this unexpected and seemingly paradoxical result? I addressed this above in terms of vitality at maturity vs. at maintenance. Here I take a different perspective.

It is a well-supported [169, 186] and intuitively appealing fact that a high extrinsic hazard of death favors early reproductive maturity. A short juvenile period reduces the time available for development and hence the time to attain a certain vitality. Vitality, however, determines the level of energy available and therefore the potential to reproduce. If individuals have to mature early because of a very risky

[27] Another example for repeated, easily replaced parts are red blood cells. Each cell wears out over its average lifetime of 120 days in humans, but new undamaged red blood cells are constantly provided by the bone marrow. Thus, there is senescence at the level of the red blood cells, but there could be sustenance at the next higher level of organization – the blood – if the age- and damage-structure in the population of red blood cells is constant, at least over a long period of a human's life. But at the whole organism level, the human being senesces. This further leads to the idea of looking at senescence vs. non-senescence at different levels of organization of an organism. Different levels may have different η parameters. The individual red blood cells might face increasing returns to scale, but the blood itself may have decreasing returns to scale while the whole body is subject to convex investment. Also, the value of the η parameters might change with age, being concave early in live and transitioning to convex later in live. For now this remains speculation and is far beyond the scope of this chapter. But it points to exciting research questions for future research.

environment, their reproductive potential might be small. The short life to be expected gives only few opportunities to reproduce. Thus, every additional reproductive event increases total lifetime reproduction by a large relative share compared to what has been realized before. Therefore, depending on the costs of reproduction, a small potential should be maintained (Sustenance) and, if possible, further increased (Enhancement) when maintenance costs are low ($\eta_g < 1$).

If, on the other hand, life is safe the individual can afford to spend a long time building up a high level of vitality, i.e. a large reproductive potential. Instead of paying the price of maintaining a high level of vitality, it may be evolutionarily advantageous to harvest this potential at the cost of a loss in functioning. Subsenescence is the strategy that particularly suits this circumstances. It is Subsenescence and not Senescence that is the optimal strategy at low levels of extrinsic mortality when maintenance is cheap but reproduction is expensive. And this is why: The propensity to share resources between reproduction and growth is small due to costly reproduction. Therefore exclusive investment is desirable. Low maintenance costs, on the other hand, favor the preservation of vitality rather than decay, which implies sharing of resources. As long as mortality is low the individual can afford to mature late, attaining a high reproductive potential. However, maintaining this level of vitality would be strongly penalized in terms of reduced reproduction. Instead, the individual harvests the large potential and mortality increases after reproductive maturity. But when vitality has fallen to a level that can be preserved without too much penalty, any further deterioration is suboptimal. The individual maintains its state and mortality is constant.

Williams [212] conjectures that low levels of extrinsic mortality should be associated with slow-senescent strategies and high levels of extrinsic mortality should be associated with fast-senescent strategies. His hypothesis is in accordance with the results from previous reproductive effort models (for a review see Charlesworth [27, Section 5.3.4.]). Higher extrinsic risk tends to increase reproductive effort, which implies higher levels of mortality. I have shown that my results predict under some circumstances the opposite effect of an increase in parameter c. Moreover, my results imply that non-senescent strategies can be optimal. A theory based on optimization of trade-offs can account for constant or declining age-patterns of mortality while a theory based on mutation accumulation cannot explain these patterns. In Sects. 6.3.1, 6.4.1, and 6.4.3, I will discuss the concept of extrinsic mortality and return to Williams's hypothesis.

5.5.4 Plateaus

All my life history strategies show maintenance from a certain vitality onwards, i.e. plateaus in mortality and fertility are part of all life history strategies. Plateaus have been observed in large populations of Medflies, *Drosophila*, nematode worms, beetles, and humans ([21], [47], [189], [201]), and understanding the cause of this pattern has caught the attention of many researchers (for example [32], [151], [201], [208], [209]).

In my model plateaus naturally arise as part of non-senescent life history strategies, since vitality cannot increase indefinitely and thus at some level will have to be maintained. The plateau that follows a period of exponentially increasing mortality results from the constraint I impose that vitality can not follow a zig-zag path. Therefore, even though my model can lead to mortality plateaus one has to be careful in interpreting this technical result. From a biological perspective there may be organisms that cannot halt further deterioration after they have deteriorated substantially. In such species, individuals may continue to suffer senescence with age; observed plateaus maybe due to population heterogeneity [201, 202, 204, 206, 207]. On the other hand, it may indeed be the case that mortality plateaus for some species are due to a strategy of sustenance at older ages. Research is needed.

5.6 Summary

The simple model developed in this chapter captures the main features of life: mortality, reproduction, development, growth and maintenance. The results show that the range of optimal life histories is wide. Senescent as well as non-senescent life history strategies can be optimal.

Whether an optimal life history follows a non-senescent strategy or a senescent strategy is crucially determined by the returns to scale to growth and maintenance as well as to reproduction. Efficient maintenance and growth systems favor maintenance strategies after growth is completed while efficient reproductive systems favor strategies of parallel growth and reproduction.

Senescent and non-senescent strategies show distinct differences in the level of vitality and age at maturity: Non-senescence is associated with early maturity at relatively low vitality and senescent strategies are associated with late maturity at a level of vitality that is close to its maximum attainable level. Since vitality at maturity can reasonably be interpreted as size at maturity, my vitality-based model leads to a

hypothesis that also followed from my size-based model: species that mature early at a relatively small size with the potential of growth and increase in reproductive potential afterwards are likely to follow a non-senescent strategy, whereas species who mature more closely to their maximum attainable size are more likely to show senescence.

An exception is the special case of costly reproduction and cheap maintenance and growth. This is the only case where the level of extrinsic mortality can shift a strategy from a senescent to a non-senescent one. Here, the difference in vitality at maturity between two otherwise similar organism, one of which exhibits non-senescence and the other senescence, can be small.

In general, however, it can be stated that the the qualitative shape of a life history is determined by the returns to scale in growth and maintenance and reproduction. Senescence, i.e. exponentially increasing mortality during adult ages, is the prevalent optimal strategy only if both reproduction and maintenance are costly. If maintenance is cheap, then exponentially increasing mortality is not favored. In this case, Senescence is never optimal but instead Subsenescence can be optimal. If maintenance is costly but reproduction is cheap then exponentially increasing mortality is part of an optimal life history strategy. Ensenescence is optimal.

The degree of senescence and the age of and vitality at maturity are determined by the η parameters and the mortality parameters: Higher values of either of the η parameters (i.e. investment becomes increasingly costly and less efficient) are associated with a higher degree of senescence and larger vitality at maturity. Generally, low overall mortality favors low degrees of senescence. Higher values of extrinsic mortality are associated with a higher degree of senescence and smaller vitality at maturity. Higher values of intrinsic mortality, on the other hand can favor larger vitality at maturity as long as overall mortality does not rise to a level where again early maturation, i.e. small vitality is more favorable.

In sum, the crucial determinants of the degree of senescence, i.e. of the fraction of lifetime reproduction realized at ages when mortality increases, are the η parameters as well as the mortality parameters. The crucial determinants of whether a species follows a senescent or a non-senescent life history strategy are the returns to investment. Last but not least this chapter contributes to the discussion about the importance of fitness sensitivities to the evolution of age-patterns of mortality and fertility – these sensitivities are weights on the trade-offs that are balanced by evolution.