The Age Patterns of Human Fertility and Mortality: A New, Evo-Demographic Model

Draft

Annette Baudisch and James W. Vaupel

July 14, 2005

Laboratory of Survival and Longevity
Max Planck Institute for Demographic Research
Rostock, Germany
Phone: +49 381 2081259, Fax: +49 381 2081559
e-mail: baudisch@demogr.mpg.de

Abstract

This paper will give the background to our presentation at the IUSSP 2005 in Tours. In particular, section 6 will help shed light on the following questions: Why does human mortality increase after reproductive maturity? Why do humans reach menarche only after more than a decade of intensive parental care? Why do humans not mature much earlier? Why do humans not stay healthy, young and productive until finally dying by some unlucky event? We will address these questions by developing a general life-history model of energy allocation between growth and reproduction. The human demographic schedules result as an optimal strategy for a specific set of parameters. The specific parameters distinguish the human life-course from that of other living creatures.

1 Introduction

The goal of evolutionary demography is to understand how evolution shapes the age-trajectories of fertility, mortality, growth and transfers. This goal is the central concern of life-history theory (Roff, 2002; Stearns, 1992). What is the optimal age and size at maturity? What is the optimal number and size of offspring? What is the optimal frequency of reproductive events? What is the optimal span of life? Typically, in each case, trade-offs determine the optimal strategy since resources are limited.

An optimal life-history strategy maximizes an individual's fitness. Fitness captures the reproductive success of a genotype and can be measured by Lotka's intrinsic rate of population increase, r, implicitly defined by the Lotka equation,

$$1 = \int_0^\infty e^{-r\,a}\, l(a)\, m(a)\, da. \tag{1}$$

Another frequently used measure of fitness is the net reproductive rate, R, given by

$$R = \int_0^\infty l(a)\, m(a)\, da. \tag{2}$$

The survival function $l(a)$ indicates the probability of survival from birth (or conception) to age a and the maternity function $m(a)$ indicates age-specific reproduction.

Life-history optimization attempts to find an "evolutionary stable" strategy that cannot be invaded by any other strategy Smith (1982). A mutant which diverges from the optimal schedule would have slower population growth, ultimately resulting in its extinction. Therefore, the evolutionary stable strategy is given by the functions $l(a)$ and $m(a)$ that maximize r. Because no population can continue growing, in equilibrium this r_{max} must equal zero. Taylor et al. (1974) proved that maximizing life-time reproduction, R, is equivalent to maximizing the intrinsic rate of population increase, r, such that $r_{max} = 0$. Hence, in this paper, the functions $l(a)$ and $m(a)$ that maximize R correspond to the optimal strategy.

Are there evolutionary stable life history strategies that prevent mortality from rising with age? If senescence is defined as an increase in mortality with age, then non-senescence corresponds to mortality trajectories that do not increase with age. Could non-senescent life-histories be optimal? Would a non-senescent strategy also involve increasing or constant fertility with age? What characteristics lead to senescent vs. non-senescent life-histories?

Our previously developed model shows that non-senescence can be optimal (Vaupel et al., 2004). 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, after the onset of reproduction, size remains constant, 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 by Vaupel et al. (2004), 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 the level of 80 % of an undamaged cell, this cell will account for 0.8 units of total vitality.

Facing ubiquitous decay, life is sustained by processes of 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.

This state variable is a new approach to life history modeling.

2 The Vitality Model

Survival depends on mortality. In accordance with the size-based model 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

$$\mu(\psi) = \frac{b}{\psi} + c, \tag{3}$$

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 model, energy was simply proportional to size. However, energy production is not equivalent to size

but has been found to scale allometrically with size (Lavigne, 1982). A sound theoretical basis for a particular relation between size and net energy available was given by West et al. (2001), their equation (3). This formula captures the difference between energy created by and energy required for cell metabolism.

The model developed in this paper uses equation (3) from West et al. (2001) to determine the available resources of an individual at current vitality. West et al. (2001)'s formula 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,

$$\epsilon(\psi) \;=\; k\,\psi^{0.75} \;-\; \kappa\,\psi, \tag{4}$$

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.

Energy is maximum at vitality ψ_ϵ

$$\psi_\epsilon \;=\; \left(\frac{3}{4}\,\frac{k}{\kappa}\right)^4. \tag{5}$$

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 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 relates proportional to vitality and integrates naturally into the structure of West et.al.'s equation. Consequently, vitality ψ changes over time according to

$$\dot{\psi} \;=\; \pi(\psi)^{\eta_g}\,\epsilon(\psi) \;-\; \delta\,\psi, \tag{6}$$

where $\pi(\psi)$ denotes the fraction of energy allocated to growth as in the initial model of Vaupel et al. (2004).In contrast to their model, $\pi(\psi)$ 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. Note that η_g has no effect if $\pi(\psi)$ equals either zero or one. The reasoning behind the incorporation of this parameter will be given below.

The level of $\pi(\psi)$ that corresponds to maintenance of current vitality is given by Eq. 6

when $\dot{\psi} = 0$,

$$\pi_0 = \left(\frac{\delta}{k\,\psi^{-0.25} - \kappa} \right)^{\frac{1}{\eta_g}}. \tag{7}$$

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

$$\Psi \equiv \left(\frac{k}{\kappa + \delta} \right)^4. \tag{8}$$

Available energy must be nonnegative. This implies that

$$\psi \leq \left(\frac{k}{\kappa} \right)^4 \tag{9}$$

must hold. This is always true since equation 9 implies that ψ cannot exceed maximum attainable vitality Ψ, as given by equation 8.

In the initial size-based model by Vaupel et al. (2004) reproductive effort and reproductive output are related linearly. This assumption restricts solutions to exclusive energy allocation to either growth or reproduction (Léonard and van Long, 1992). To develop a model that covers 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 equation 6. The biological motivation to introduce nonlinear effects is the following.

To grow an arm in humans requires considerable effort and is so expensive that, if the arm is lost, no new arm can regrow. To grow a branch in trees can be done readily to increase size or replace broken off branches. Clearly, the machinery for growth in humans and trees is inherently different and implies very different costs. The costs for some fish to produce an additional hundred thousand offspring might be negligible but the differences in costs for creating one vs. two vs. ten babies for human females are enormous. The marginal costs of offspring are small for some species and large for others. The machinery that is used for development vs. subsequent growth and maintenance might be the same for some species but different for others. In the former case, maintenance is cheap because an already build machinery can be used to maintain the organism without much additional costs. In the latter case, it might be very costly and even impossible to keep or rebuild the machinery that would allow , for instance in humans, to regrow a lost arm. Therefore, the parameter η_g in equation 6 captures the efficiency of the growth apparatus. 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.

In the modified size-based model by Vaupel et al. (2004) an arbitrary attempt was made

to introduce nonlinearity in reproductive effort. Here, nonlinearity in reproductive effort is captured by the parameter η_r (r for reproduction) that captures the efficiency of reproduction, analogous to η_g.

In the size-based model, offspring are born at size one. This initial offspring size determines the initial mortality rate, the amount of energy necessary to create one offspring and therefore the number of offspring produced. If offspring could be born at larger sizes they would be more robust and therefore would have higher survival chances from the very beginning of life. On the other hand, larger offspring require more energy per offspring. In this way, offspring size effects the optimal energy allocation strategy and is a variable expected to be optimized by the trade-off between quantity and quality of offspring.

In the vitality model the maternity function is specified as

$$m(\psi) \;=\; \varphi\,[\,1 \;-\; \pi(\psi)\,]^{\eta_r}\,\frac{\epsilon(\psi)}{\psi_0^{\eta_j}}. \tag{10}$$

In accordance with the size-based model, fertility is proportional to available energy, in this model $\epsilon(\psi)$, and reproductive effort, $1-\pi(\psi)$. Available energy is divided by the initial vitality of offspring, ψ_0. The constant parameter $\eta_j > 1$ (j for juvenile) accounts for energy that is needed to create one offspring but that does not accumulate in the vitality of an offspring. Contrary to the parameters η_r and η_g, parameter η_j captures the level of "wastage" rather then "efficiency". The wastage parameter η_j does not influence the optimal trajectory of investment over the life-course. As a constant, $\psi_0^{\eta_j}$ can be taken outside the integral in equation 2. Optimal offspring size can be determined once the optimal path of energy allocation over the life course is found. Perrin (1992) incorporated offspring size in a similar manner, without the exponent, but in Perrin's approach offspring size was a given constant, not a variable to be optimized. The constant φ is a scaling parameter set to the value that ensures that optimal lifetime reproduction is equal to one and, hence, $r_{max} = 0$.

The way that nonlinearities are incorporated in the present chapter is biologically and technically motivated. The approach leans on the well-known concept used in economics in 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. To my knowledge, this concept of the efficiencies of the reproduction and growth system has not previously been applied in life-history analysis.

George E. P. Box said: "All models are wrong, but some are useful." (Box, 1979) Models are wrong because they simplify the complexity of life. But without this simplification, pat-

terns 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, though necessarily wrong, enhances understanding of nature.

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

2.1 The Parameters

2.1.1 k, κ and δ

Parameter k captures the speed of growth of vitality (Eqs. 4 and 6). Faster growth implies a quick fall in mortality (Eq. 3) and reduces the time of development. Furthermore, higher values of k decrease maintenance costs (Eq. 7) and increase maximum vitality (Eq. 8). Parameter κ is inversely related to maximum vitality. Elevating κ slows growth, increases maintenance costs (Eq. 7) and decreases maximum vitality (Eq. 8). Parameter δ determines the speed of decline in vitality (Eq. 6). Higher δ increases maintenance costs (Eq. 7) and decreases maximum vitality (Eq. 8).

If all energy is allocated to reproduction, then δ determines the constant rate of increase in mortality (Eq. 3). A decline in vitality not only implies a reduction in survival but also a reduction 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 (Eq. 4). 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 Figure 1. Note that an increase in vitality beyond the threshold given by Eq. 5, which corresponds to the peek 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.

The parameters k, κ, and δ set the speed of growth and decay and therefore can be used to determine the time and size scale of the strategy.

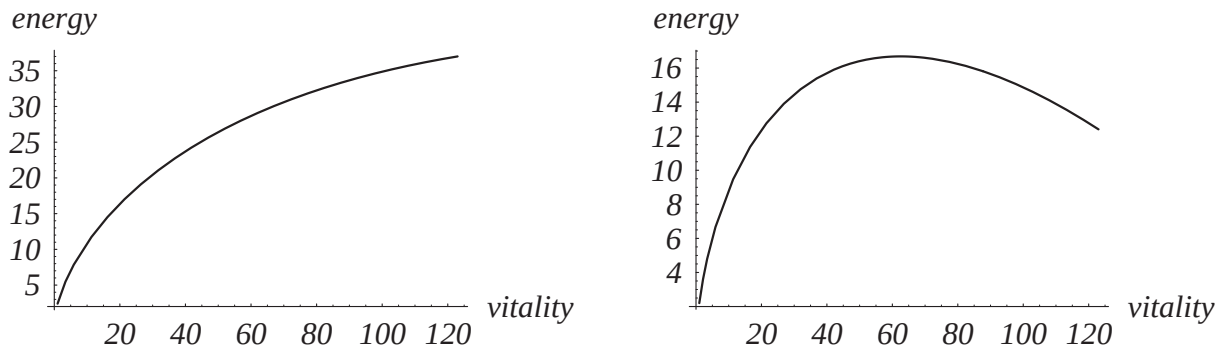

Figure 1: Comparison of trajectories of energy over vitality for two parameter combinations that lead to a maximum attainable vitality of $\Psi = 123$ but imply different shapes: left: $k = 3$, $\kappa = 0.6$, $\delta = 0.3$; right: $k = 3$, $\kappa = 0.8$, $\delta = 0.1$

2.1.2 b and c

Parameters b and c determine the overall level of mortality (Eq. 3). Parameter b captures the state-dependent, intrinsic component of mortality, i.e. b determines how important it is to attain and maintain a high vitality. High b determines the minimum level of state-dependent mortality, b/Ψ, which also depends on maximum vitality Ψ. Since b/ψ_0 determines infant mortality, the magnitude of b also influences the optimal vitality at birth. 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 $b/\Psi + c$.

Higher levels of mortality discount future reproduction. Williams (1957) hypothesizes that low levels of mortality should be associated with non-senescent strategies and high levels of mortality should be associated with senescent strategies. "Williams hypothesis" is a testable implication of the evolutionary theories of senescence. The empirical evidence is not unambiguous (Finch (1990) and Resznick et al. (2004)). Abrams (1993) shows that the influence of extrinsic mortality hinges on specific population dynamics. The influence of extrinsic and intrinsic mortality in this model is investigated below.

2.1.3 η_r and η_g

Parameter η_r captures the intrinsic costs of reproduction (Eq. 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 rejuvenation, 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 vitality (Eq. 7). Large 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 efficiencies 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. Whether this expectation is correct will be investigated in this paper.

3 Solution

The solution to this life-history problem consists of two distinct optimization procedures. First, the optimal path of investment is determined in a dynamic optimization procedure. The term $\varphi/\psi_0^{\eta_j}$ in equation 10 is a constant and does not influence the optimal trajectory of investment. It is taken into account in a the subsequent, second part of the solution, being a simple maximization.

The optimal state-trajectory has to be monotonous in this single state model because the state ratchet applies (Vaupel et al., 2004). To overcome the state ratchet, a binary indicator that contains information about the mode of change in vitality ("up" for growth and "down" for shrinkage) will serve as a second state variable, which itself has no direct influence on the optimal strategy. Since life necessarily starts off by growth, the switch is initially in up mode and optionally can change into down mode. In this framework, I assume that the switch can only occur once.

To solve the first optimization problem I applied a dynamic programming approach by developing an algorithm, following a backward procedure and assuming stepwise constant vitality (Bellman, 1965). Crucial to Bellman's approach is that the optimal decision does not depend on the past, but is solely based on the current state. The state determines possible current and future payoffs. An absolute essential requirement for this backward optimization to work is the knowledge of an ultimate state with known payoffs, the ultimate future expectation. The procedure starts at this ultimate state and then works backwards along the state-trajectory.

The state-trajectory is assumed to be stepwise constant. The time it takes to change from

vitality ψ to vitality $\psi \pm \Delta$ ($\Delta > 0$, step-size) is given by the step-time

$$\tau(\psi, \pi) \;=\; \frac{\Delta}{\dot{\psi}}, \tag{11}$$

where $\dot{\psi}$ is defined in equation 6. Note that if vitality falls, then $\tau(\psi, \pi) \;=\; -\Delta/\dot{\psi}$ and if vitality is maintained then $\tau(\psi, \pi) \;=\; \infty$.

At each level of vitality the algorithm maximizes remaining reproduction, given by

$$R(\psi) \;=\; \int_0^{\tau} \; e^{-\mu(\psi)\, a} \; m(\psi, \pi) \; da \;+\; e^{-\mu(\psi)\, \tau(\psi, \pi)} \; R(\psi_{next}). \tag{12}$$

Since vitality is constant over the time interval τ the integral in equation 12 can be solved, yielding

$$R(\psi) \;=\; \frac{m(\psi, \pi)}{\mu(\psi)} \left[1 \;-\; e^{-\mu(\psi) \tau(\psi, \pi)}\right] \;+\; e^{-\mu(\psi)\, \tau(\psi, \pi)} \; R(\psi_{next}). \tag{13}$$

Remaining reproduction is given by current reproduction weighted by the chance of dying in that interval and remaining reproduction at the subsequent level of vitality weighted by the probability of surviving the time interval.

The algorithm to determine the optimal investment trajectory $\pi^*(\psi)$ (the star indicates "optimal") has two parts, one for each mode. For this application the ultimate state corresponds to a vitality of $\psi = 0$ and therefore to a mortality that is infinite and a remaining reproduction of zero. Consequently, the first part of the algorithm begins in down mode at the end of possible state-trajectories, i.e. at the last level of vitality $\psi > 0$ when the switch is in down mode. Choosing $\psi = 1$ is convenient. Then, the initial step is to find $\pi_d^*(1)$ and the corresponding $R_d^*(1)$ (the d indicates "down mode") using equation 13:

$$\begin{aligned}
\pi_d^*(1)) \;&=\; \max_{\pi \,\epsilon\, [0,\, \pi_0]} \; R_d(1) && (14)\\
&=\; \max_{\pi \,\epsilon\, [0,\, \pi_0]} \; \frac{m(1, \pi)}{\mu(1)} \left[1 \;-\; e^{-\mu(1) \tau(1, \pi)}\right] \;+\; 0 && (15)\\
&=\; \max_{\pi \,\epsilon\, [0,\, \pi_0]} \; \frac{(1 \;-\; \pi)^{\eta_r} (k - \kappa)}{b + c} \left[1 \;-\; e^{-(b+c)\, \Delta \,/\, (\pi^{\eta_g}\, (k - \kappa) - \delta)}\right]. && (16)
\end{aligned}$$

The procedure is repeated working backwards for all levels of vitality up to the maximum attainable vitality $\psi = \Psi$, determined by equation 8. For each level of vitality the optimal investment is found by

$$\pi_d^*(\psi) \;=\; \max_{\pi \epsilon [0,\, \pi_0]} \; \frac{m(\psi, \pi)}{\mu(\psi)} \left[1 \;-\; e^{-\mu(\psi) \tau(\psi, \pi)}\right] \;+\; e^{-\mu(\psi)\, \tau(\psi, \pi)} \; R_d^*(\psi - \Delta). \tag{17}$$

This part of the algorithm gives an optimal decision for each level of vitality in down mode. [1]

Maximum attainable vitality Ψ gives the ultimate state if the switch is in up mode because, due to the state ratchet, the only possible investment at $\psi = \Psi$ is $\pi(\Psi) = \pi_0(\Psi)$. Therefore, the second part of the algorithm starts in up mode at $\psi = \Psi$. The procedure is initialized by the decision, whether to stay in up mode and maintain maximum vitality or whether to switch into down mode:

$$\pi_u^*(\Psi) = \begin{cases} \pi_0(\Psi) & \text{if} \quad \frac{m(\Psi,\pi_0)}{\mu(\Psi)} > R_d^*(\Psi) \\ \\ \pi_d^*(\Psi) & \text{otherwise.} \end{cases} \tag{18}$$

Note that if mortality μ and fertility m are constant, then remaining reproduction is given by m/μ.

Then vitality is followed backwards, down to the smallest vitality $\psi = 1$. [2] At each level of vitality the optimal investment is found by

$$\pi_u^*(\psi) = \max_{\pi \epsilon [\pi_0, 1]} R_u(\psi) = \max_{\pi \epsilon [\pi_0, 1]} \frac{m(\psi,\pi)}{\mu(\psi)} (1 - e^{-\mu(\psi)\tau(\psi,\pi)}) + e^{-\mu(\psi)\,\tau(\psi,\pi)}\, R_u^*(\psi + \Delta) \tag{19}$$

if $R_u^*(\psi) > R_d^*(\psi)$ and otherwise $\pi_u^*(\psi) = \pi_d^*(\psi)$. The second part of the algorithm gives an optimal strategy for each level of vitality in up mode.

The optimal strategy over the life course can be found connecting the results from part one and two of the algorithm in the following way: Results are saved in form of a vector

$$\begin{pmatrix} \textit{remaining reproduction} \\ \textit{direction of change} \\ \textit{vitality} \\ \textit{investment} \\ \textit{time} \end{pmatrix} = \begin{pmatrix} R^*(\psi) \\ G,\ S \textit{ or } M \\ \psi \\ \pi^*(\psi) \\ \tau^*(\psi) \end{pmatrix} \tag{20}$$

Note that the variable "direction of change" , takes on the value G for growth if vitality increases, S for shrinkage if vitality decreases and M for maintenance if vitality remains

[1] Note that I imposed one restriction. I assume symmetric division of cells. This implies that if a cell has been damaged, then all its copies will inherit this damage. If $\pi = 0$ is optimal at any time, then no copies are made from currently undamaged cells. Each cell suffers some damage and no undamaged copies are left to make new cells from. Therefore π has to remain zero. This assumption restricts the scope of possible life-histories. Relaxing this assumption can easily be done.

[2] Vitality in the model is treated as a dimensionless variable, assuming that vitality is normalized by dividing through with a reasonable base unit ($\psi_0 = \psi_{base}/\psi_{base} = 1$). For simplicity functioning at birth is assumed to be perfect. Therefore ψ_{base} is equal to the number of cells (corresponding to the minimum size) at birth. In order to establish the real vitality scale from the algorithm vitality has to be multiplied by ψ_{base}.

constant. For each level of vitality the optimal vector is saved in a list. The optimal solution can be found from this list connecting the vectors in the right order. The only logic succession of vectors regarding the direction of change are $(G, \ldots, G, M)$, $(G, \ldots, G, S, \ldots, S, M)$ and $(G, \ldots, G, S, \ldots, S)$. Trivially, vectors need be be nested according to subsequent levels of vitality. Note that if *direction of change* $= M$, then $\tau = \infty$.

The second optimization procedure attempts to find the optimal vitality at birth. Note that the vitality an individual is endowed with at birth can be seen as a transfer from parent to offspring before birth. In order to solve this problem knowledge about the expected lifetime reproduction at each level of vitality is necessary. The remaining reproduction at each level of vitality, $R^*(\psi)$, is given by the succession of first entries in each vector of the list, the result of the first optimization procedure. Taking into account equation 10, lifetime reproduction is given by

$$R^* = \varphi \frac{R^*(\psi_0)}{(\psi_0)^{\eta_j}}. \tag{21}$$

Consequently, optimal vitality at birth can be calculated solving

$$\psi_0^* = \max_{\psi \epsilon [1, \Psi]} \frac{R^*(\psi)}{(\psi)^{\eta_j}}, \tag{22}$$

Finally, the constant parameter φ can be used to adjust R^* to be equal to one.

4 The Eight Varieties of Life Histories

Eight different types of optimal strategies can be found to result from this model. Strikingly, the variety of strategies is broad and includes senescent as well as non-senescent life-histories. In this section, I will describe these eight strategies with the help of illustrative examples. Then, in the following section. I will analyze under what conditions each strategy can be optimal.

Strategies are classified with respect to the specific trajectory of π. From birth to maturity $\pi(\psi) = 1$ and vitality increases. The age of and vitality at maturity are defined as the age and vitality when $\pi(\psi)$ drops below one for the first time. After maturity, vitality might be maintained, increase or decrease. Once maintenance of vitality is optimal, it will be optimal at all subsequent ages in this one state model.

Each description of a strategy begins at maturity. Note that the function $\pi(\psi)$ captures the trajectory of actual investment whereas the function $\pi_0(\psi)$ determines the level of investment that would be necessary to maintain the current level of vitality ψ.

4.1 Strategies with Senescence

Senescence, in this framework, is defined as an increase in mortality with age. Exponentially increasing mortality is often assumed and exponential curves are often fit to empirical data on mortality. Whether mortality increases exponentially or at a different pace, however, is dependent on the particular life-history. The following two strategies capture cases where mortality increases exponentially from a certain age onwards. Interestingly, the two subsequent strategies capture cases where a slower than exponential increase in mortality is optimal.

4.1.1 Gompertzian Senescence

Gompertzian Senescence corresponds to a strategy of $\pi = 0$ from the moment of maturity and thereafter. Vitality decreases exponentially at a rate δ. Senescence captures the familiar case of Gompertzian mortality, with mortality and fertility patterns similar to those of many mammals, birds, and other species. Reproduction is initiated and mortality rises exponentially when investment switches from one to zero. An example is illustrated in Fig. 2 with the parameter combination

$$\eta_r = 2,\ \eta_g = 2,\ b = 0.3,\ c = 0.01,\ k = 3,\ \kappa = 0.8,\ \delta = 0.1. \tag{23}$$

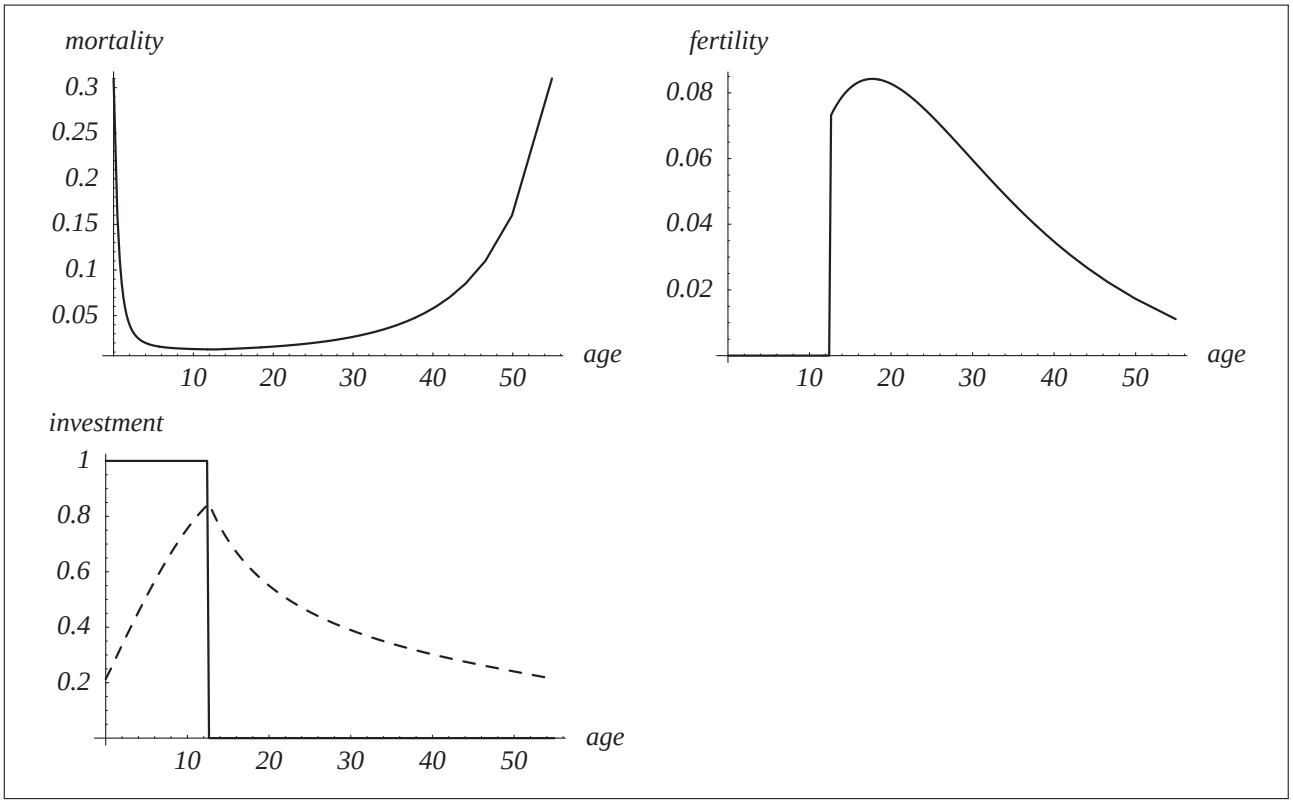

Figure 2: Example of Gompertzian Senescence. (Dashed line: π_0, level of investment required for maintenance)

4.1.2 Delayed senescence

Delayed senescence corresponds to a strategy of $\pi > \pi_0$ followed by $\pi = 0$. Vitality first increases and then decreases exponentially at a rate δ. After an early age of maturity a larger reproductive potential is striven for and established during a period of parallel growth and reproduction. Fertility increases while mortality decreases. Then, in a second reproductive peak the reproductive potential is harvested at the cost of deterioration of the individual. Mortality increases exponentially. An example is illustrated in Fig. 3 with the parameter combination

$$\eta_r = 0.5,\ \eta_g = 2,\ b = 0.2,\ c = 0.1,\ k = 3,\ \kappa = 0.8,\ \delta = 0.1. \tag{24}$$

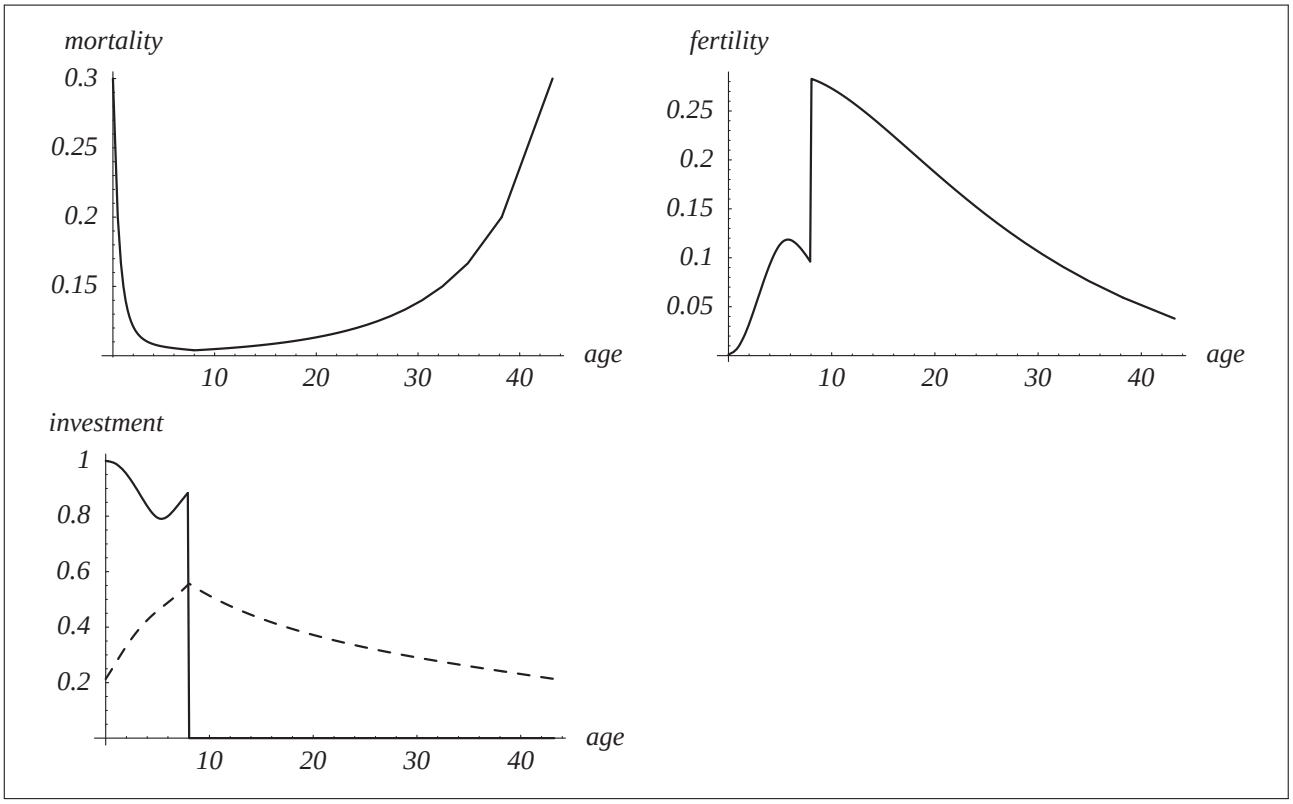

Figure 3: Example of Delayed Senescence. (Dashed line: π_0, level of investment required for maintenance)

4.1.3 Subsustenance

Subsustenance corresponds to a strategy of $0 < \pi < \pi_0$, where $\pi \approx \pi_0$. Vitality slowly decreases. The missing fraction of energy that would be necessary to truly maintain vitality is used to increase reproductive output. An example is illustrated in Fig. 4. Life-expectancy at birth is only 13, and, at reproductive maturity $\alpha = 8$ it is about 31. Investment after maturity falls just slightly below maintenance level, as indicated by the dashed line. Note that the increase in mortality is retarded in such a way that the strategy is almost equivalent to real maintenance. The corresponding vitality trajectory is shown in Fig. 5 The example pertains to the parameter combination

$$\eta_r = 2,\ \eta_g = 0.5,\ b = 1,\ c = 0.001,\ k = 3,\ \kappa = 0.8,\ \delta = 0.1. \tag{25}$$

4.1.4 Delayed subsustenance

Delayed subsustenance corresponds to a strategy of $\pi > \pi_0$ followed by $0 < \pi < \pi_0$, where $\pi \approx \pi_0$. Vitality first increases and then decreases at a very slow pace. An example is

illustrated in Figs. 6 and 7 with the parameter combination

$$\eta_r = 1,\ \eta_g = 0.5,\ b = 0.1,\ c = 0.02,\ k = 3,\ \kappa = 0.7,\ \delta = 0.2. \tag{26}$$

In contrast to the case of subsustenance where investment suddenly drops (Fig. 4), investment smoothly falls from one down to just below maintenance level in Fig. 6. The slow deterioration in vitality in case of delayed subsustenance (Fig. 7) is preceded by a period of parallel growth and reproduction. In case of subsustenance (Fig. 5) this period is missing: vitality falls markedly after the onset of reproduction before a further decrease in vitality is retarded more strongly. To understand the difference between subsustenance and delayed subsustenance, note in particular the difference between the lower left hand graphs in Fig. 4 vs. Fig. 6.

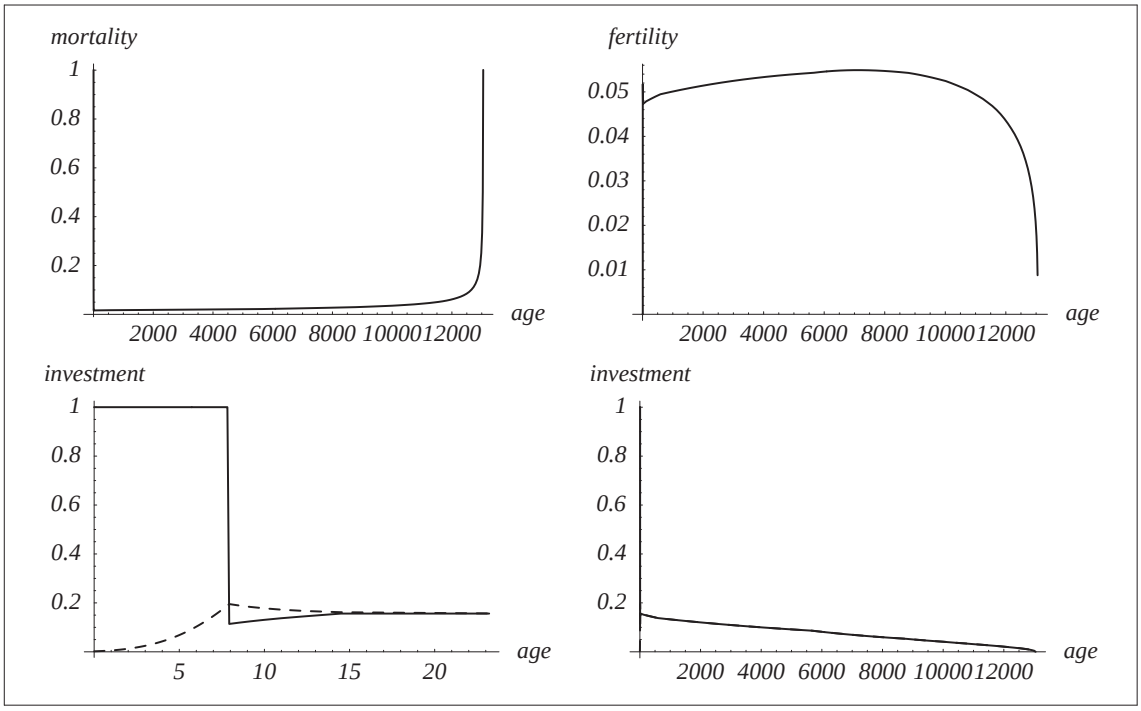

Figure 4: Example of Subsustenance. (Dashed line: π_0, level of investment required for maintenance) Note that in the lower right graph the trajectories of π and π_0 overlap, because π falls just slightly below π_0.

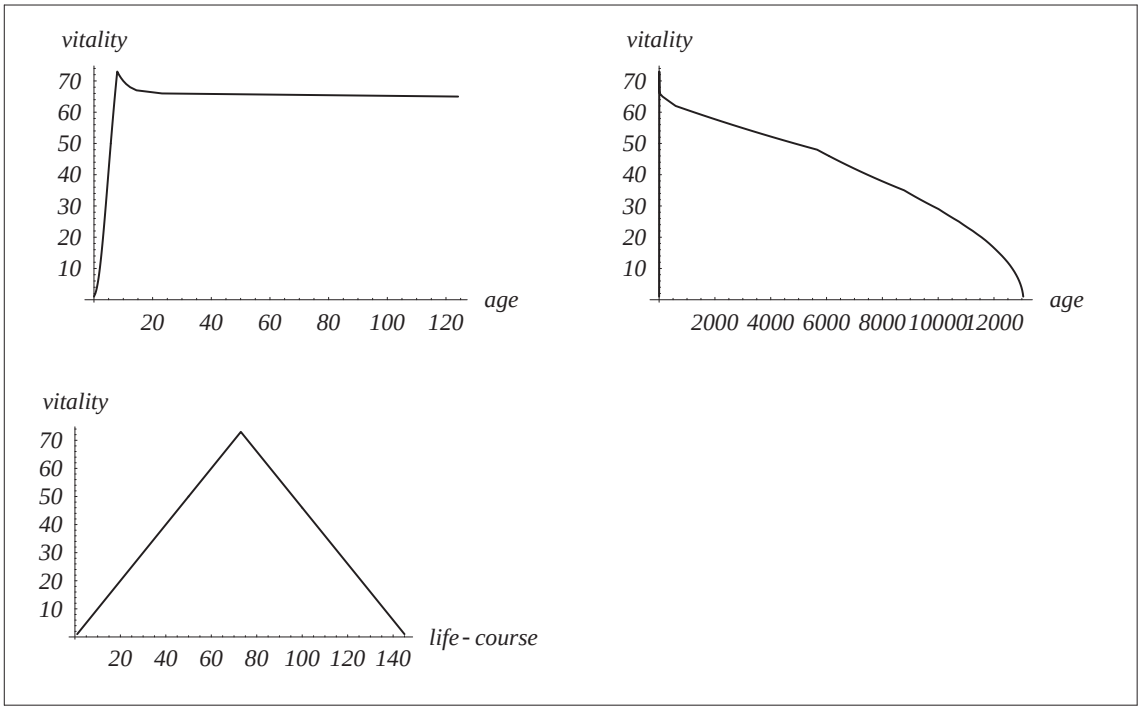

Figure 5: (Life-course: measures the number of one unit changes in vitality). Vitality trajectory

4.2 Strategies with Sustenance

The following two strategies capture cases, where senescence is never optimal, i.e. mortality never increases. The parameter combinations are not peculiar or extremely different from the previous cases. Non-senescence and senescence, in this model, can be found to coexist in contiguous neighborhoods of the parameter space.

4.2.1 Sustenance

Sustenance corresponds to a strategy of $\pi = \pi_0$ immediately after the period of development. Vitality is maintained. The case of sustenance is illustrated in Fig. 8. At the age of maturity investment drops down to maintenance level. Reproduction starts and both mortality and fertility remain at non-zero, constant levels. This example pertains to the parameter combination

$$\eta_r = 2,\ \eta_g = 2,\ b = 0.2,\ c = 0.001,\ k = 3,\ \kappa = 0.8,\ \delta = 0.1. \tag{27}$$

Note that this parameter combination differs from the example in Fig. 2 only by the level of mortality, where senescence is optimal. Lowering mortality at the boundary can shift a strategy from senescence to non-senescence. This remarkable, surprising result merits further theoretical attention and empirical investigation.

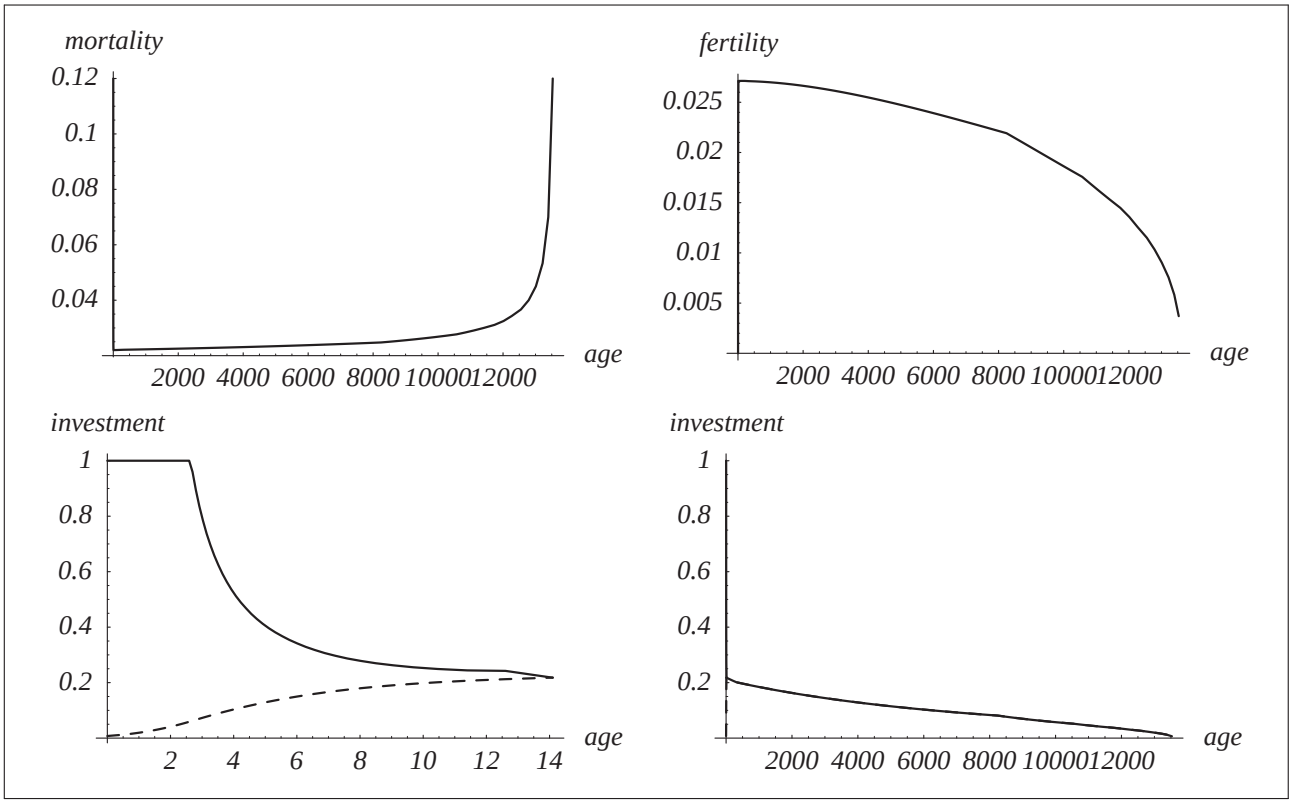

Figure 6: Example of Delayed Subsustenance. (Dashed line: π_0, level of investment required for maintenance) Note that in the lower right graph the trajectories of π and π_0 overlap, because π falls just slightly below π_0.

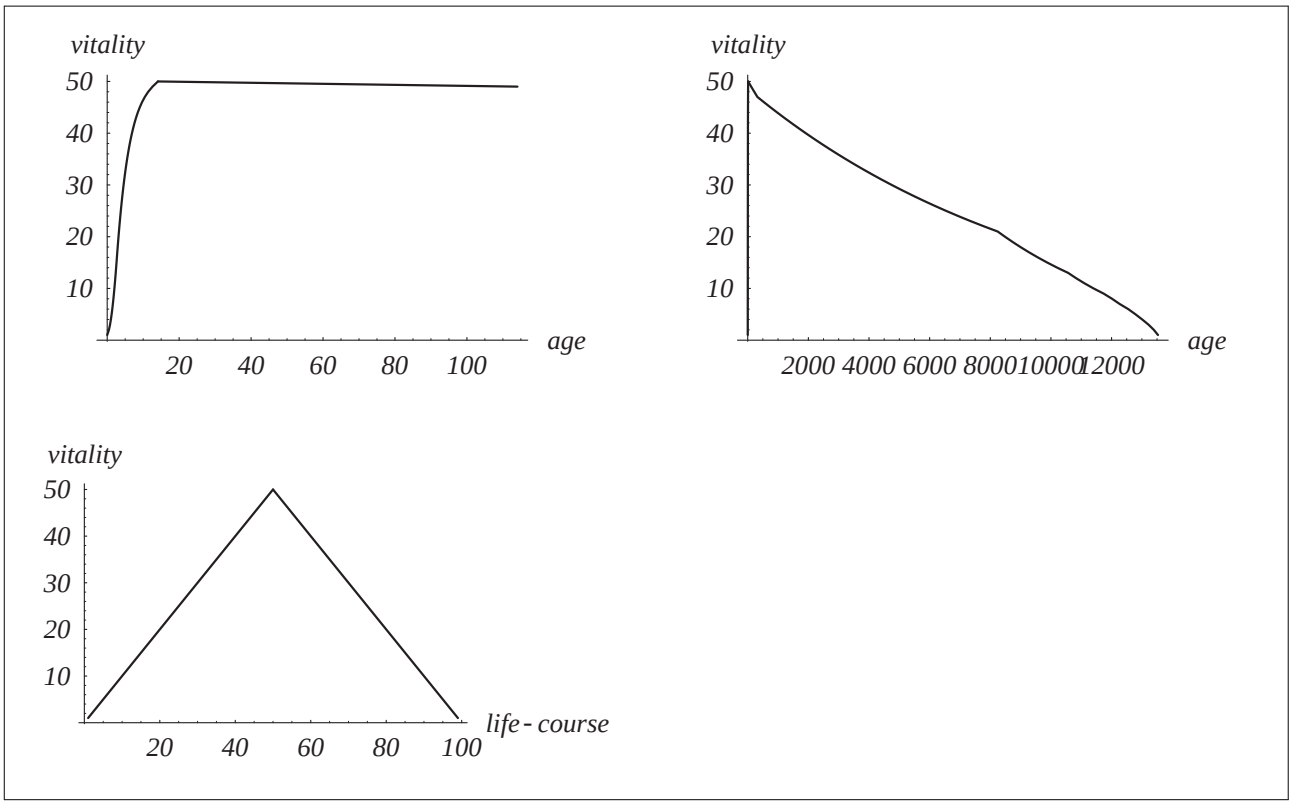

Figure 7: Example of Delayed Subsustenance (Life-course: measures the number of one unit changes in vitality). Vitality trajectory

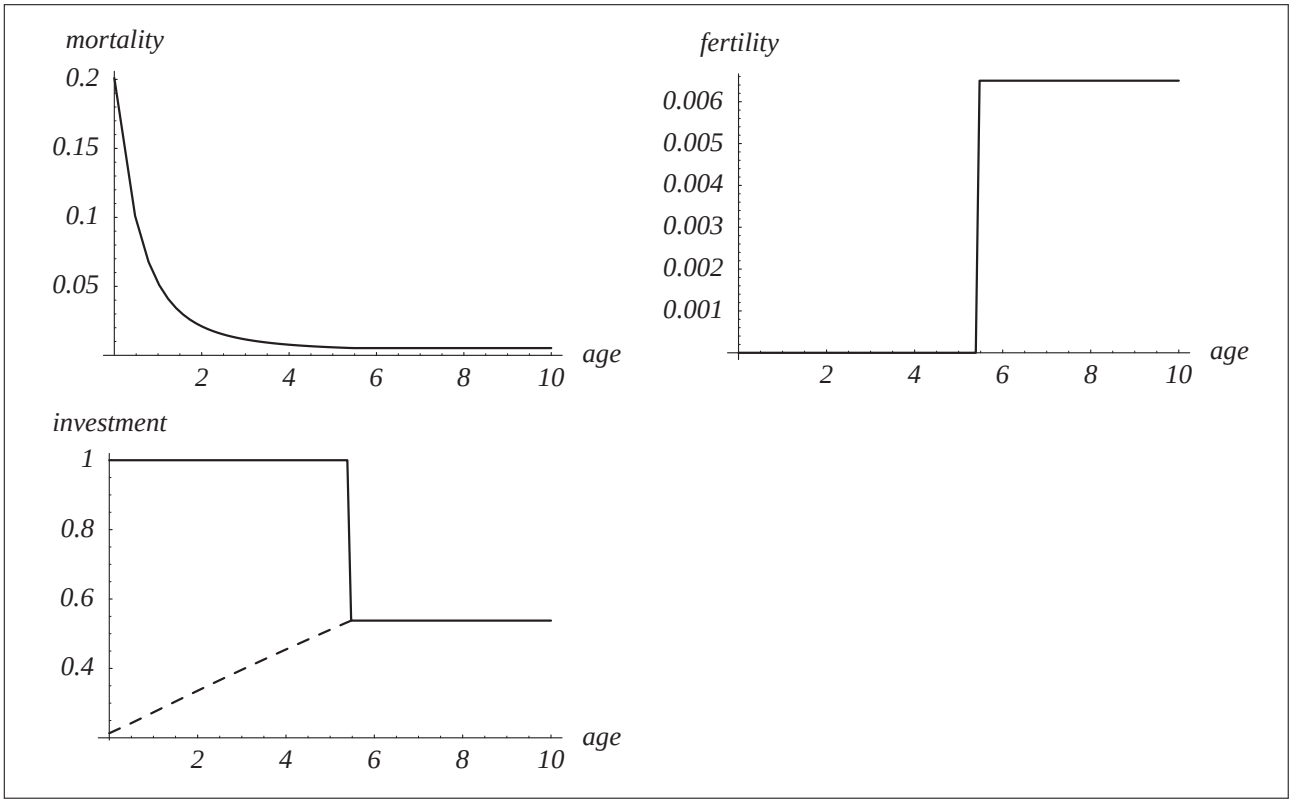

Figure 8: Example of Sustenance. (Dashed line: π_0, level of investment required for maintenance)

4.2.2 Supersustenance

Supersustenance corresponds to a strategy of $\pi > \pi_0$ followed by $\pi = \pi_0$. Vitality first increases and then is maintained. The case of Supersustenance is illustrated in Fig. 9. Investment falls smoothly from one down to maintenance level. Mortality decreases while fertility increases until the trajectories reach a constant level. This example pertains to the parameter combination

$$\eta_r = 0.5,\ \eta_g = 0.5,\ b = 0.2,\ c = 0.004,\ k = 3,\ \kappa = 0.8,\ \delta = 0.1. \tag{28}$$

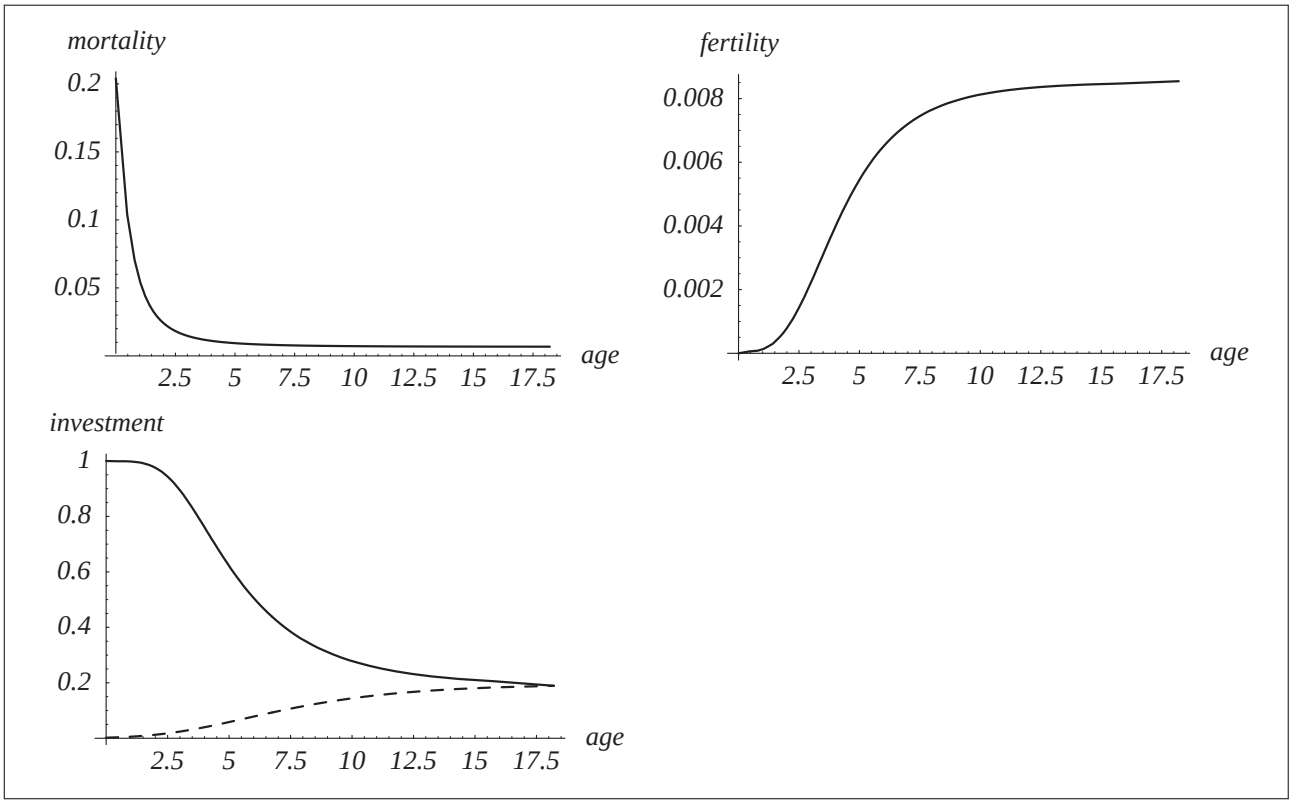

Figure 9: Example of Supersustenance. (Dashed line: π_0, level of investment required for maintenance)

4.3 Strategies with Both Senescence and Sustenance

Life does not have to be "either/or" but can be "both/and". The subsequent two strategies illustrate life-histories where senescence and non-senescence co-exist.

4.3.1 Partial senescence

Partial senescence corresponds to a strategy of $\pi < \pi_0$ followed by $\pi = \pi_0$. Vitality decreases and then is maintained. The case of partial senescence is very interesting. A high reproductive potential is build up during development and then harvested at the cost of falling vitality until a level of vitality is reached that is sufficient to keep mortality at a low level. An example is illustrated in Fig. 10 with the parameter combination

$$\eta_r = 2,\ \eta_g = 0.5,\ b = 0.2,\ c = 0.004,\ k = 3,\ \kappa = 0.7,\ \delta = 0.2. \tag{29}$$

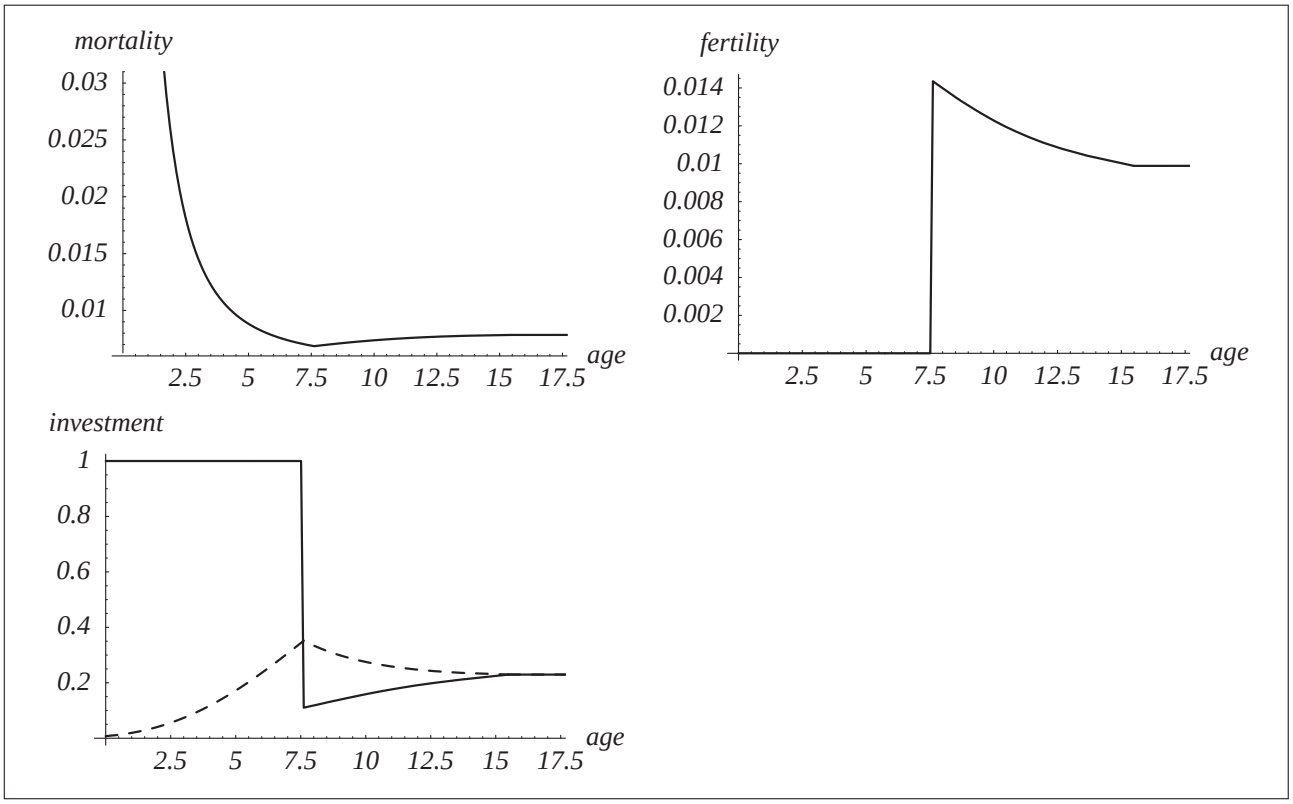

Figure 10: Example of Partial Senescence. (Dashed line: π_0, level of investment required for maintenance)

4.3.2 Delayed partial senescence

Delayed partial senescence corresponds to a strategy of $\pi > \pi_0$, followed by $0 < \pi < \pi_0$, followed by $\pi = \pi_0$. Vitality first increases, then decreases and then is maintained. A period of parallel growth and reproduction precedes a long period of life when the organism deteriorates at a slow pace before a lower level of vitality is eventually maintained. An example is presented

in Figs. 11 and 12. Note that investment is not only plotted over age but also over the life-course to clarify the strategy. Each step of the life-course corresponds to a one unit change in vitality. Vitality increases after the age of maturity $\alpha = 4$ until it reaches a peak of about 60 at age 14. Then vitality starts to fall. The period of decline in vitality is slowed down to an extent that the corresponding changes in mortality and fertility are negligible over the main part of life ($e^0(\alpha) = 22$). The example pertains to the parameter combination

$$\eta_r = 1,\ \eta_g = 0.5,\ b = 0.3,\ c = 0.01,\ k = 3,\ \kappa = 0.7,\ \delta = 0.2. \tag{30}$$

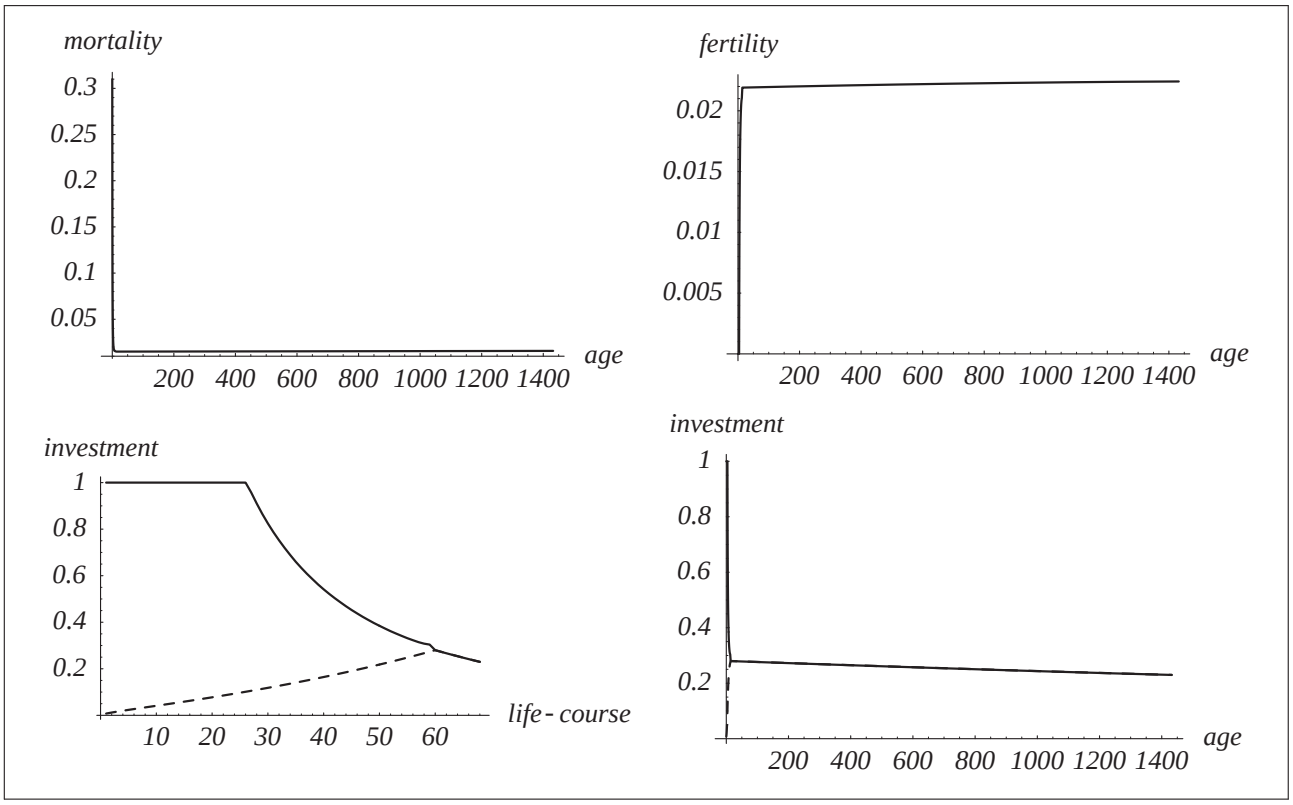

Figure 11: Example of Delayed Partial Senescence. (Dashed line: π_0, level of investment required for maintenance. Life-course: measures the number of one unit changes in vitality) Note that in the lower right graph the trajectories of π and π_0 overlap, because π falls just slightly below π_0.

5 When Senescence Is Optimal and When It Is Not

The model sheds light on the characteristics that determine whether senescent vs. non-senescent life-histories are optimal. Senescence, in this framework, is defined as an increase of mortality with age. Several strategies, however, show mortality patterns that increase over

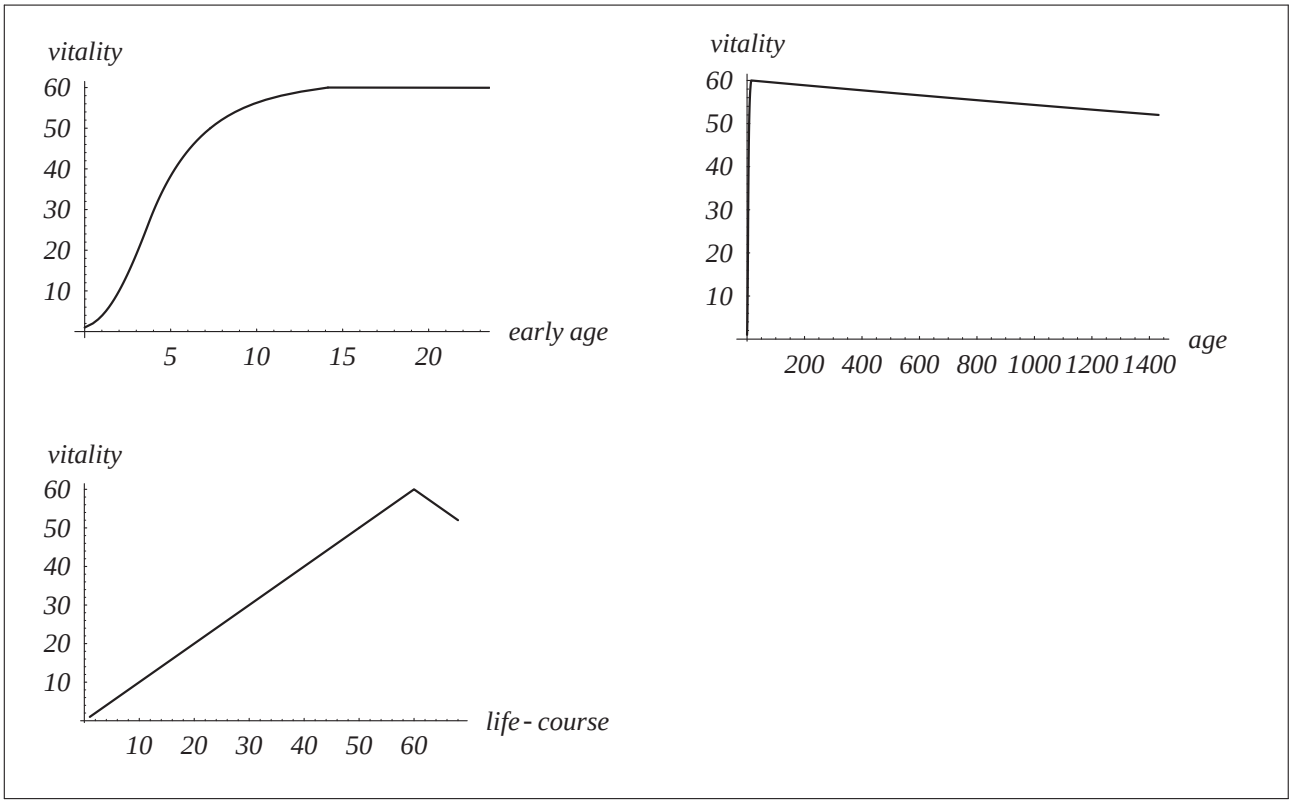

Figure 12: Example of Delayed Partial Senescence (Life-course: measures the number of one unit changes in vitality). Vitality Trajectory.

some part of reproductive life but decrease or remain constant over other parts of reproductive life. Obviously, it requires more than only the pattern of mortality to label a 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 indicator of senescence, S, is given by

$$S = \frac{\sum_{x=0}^{\infty} J_x \, l_x \, m_x}{\sum_{x=0}^{\infty} l_x \, m_x}, \tag{31}$$

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 larger or equal to the amount required for maintenance of vitality. If $S = 1$ the strategy is fully senescent and if $S = 0$ then the strategy is fully non-senescent. All values in between describe mixed strategies. For the eight strategies discussed above, the "Gompertzian senescence" and "subsustenance" strategies are fully senescent, the "sustenance" and "supersustenance" strategies are fully non-senescent, and the other strategies are mixed.

Table 1: Strategies for cheap versus costly reproduction and maintenance for low and high intrinsic and extrinsic hazard of death.

	$\eta_g = 0.5$				$\eta_g = 2$			
$\eta_r = 0.5$	b 4.2	*del sub*	*del sub*	*super*	b 4.2	*super*	*del sen*	*del sen*
	2.2	*del sub*	*super*	*super*	2.2	*super*	*super*	*del sen*
	0.2	*super*	*super*	*super*	0.2	*super*	*super*	*super*
	c	0.001	0.041	0.081	c	0.001	0.041	0.081
$\eta_r = 2$	b 2.2	*sub*	*sub*	*sub*	b 2.2	*sen*	*sen*	*sen*
	1.2	*sub*	*sub*	*sub*	1.2	*sen*	*sen*	*sen*
	0.2	*part*	*sub*	*del sub*	0.2	*sust*	*sust*	*sen*
	0.1	*part*	*del part*	*sust*				
	c	0.001	0.005	0.009	c	0.001	0.005	0.009

sen: Gompertzian senescence, del sen: delayed senescence, sub: subsustenance, del sub: delayed subsustenance, sust: sustenance, super: supersustenance, part: partial senescence, del part: delayed partial senescence

After running the algorithm for many different parameter combinations I found that most influential for the qualitative differences in mortality patterns are the efficiencies η_r and η_g and the mortality parameters b and c. Therefore, I studied the interplay between high and low levels of efficiencies and mortality parameters in more detail. Figure 13 illustrates the degrees of senescence indicated by S for combinations of η_r and η_g given the specific parameter combination $k = 3, \kappa = 0.8$ and $\delta = 0.1$. The surfaces span over different mortality conditions determined by b and c. The words "falling" and "constant" pertain to cases where mortality is decreasing or does not change over reproductive ages. The words "slow" and "exponential" pertain to cases where, if mortality rises, it does so either very slowly or at an exponential pace. The values of the indicator of senescence S are determined by the underlying life-history strategies. Table 1 contains the detailed strategies corresponding to the colors in Figure 13.

Together, Figure 13 and Table 1 summarize the results of the vitality model. Several main features are noteworthy:

5.0.3 The Implications of Costly Reproduction

Low costs of reproduction ($\eta_r < 1$, row one in Figure 13 and Table 1) correspond to non-senescent strategies over a broad range of intrinsic and extrinsic mortality. When reproduction is cheap the dominant color is blue in Figure 13, meaning the indicator of senescence is equal

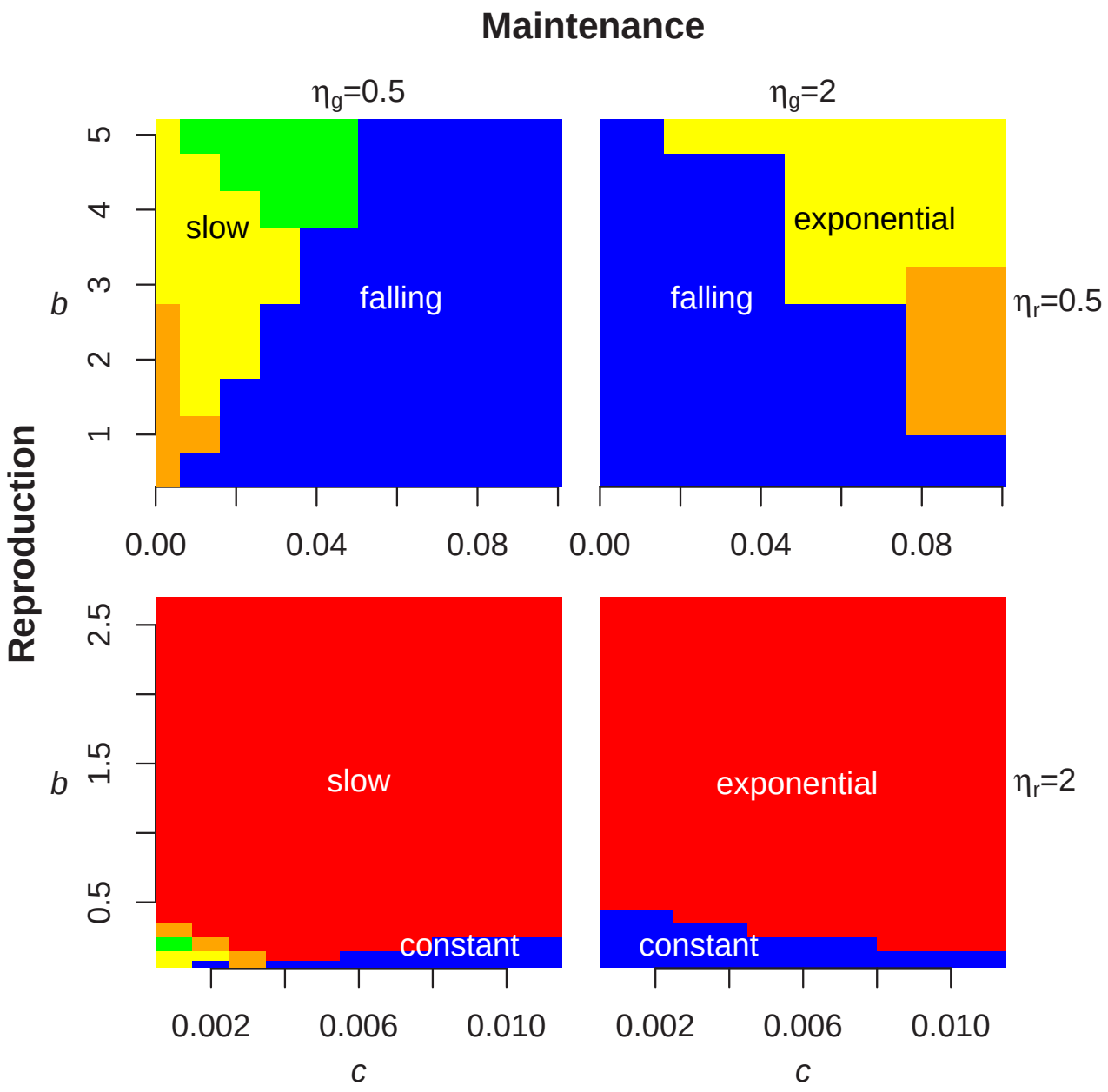

Figure 13: Senescence surfaces: *Red* = Senescence (S=1), *Blue* = Non-senescence (S = 0), *Green* = Mixed ($0 < S < 0.35$), *Yellow* = Mixed ($0.35 < S < 0.65$), *Orange* = Mixed ($0.65 < S < 1$). Rows: $\eta_r = 0.5, \eta_r = 2$, Columns: $\eta_g = 0.5, \eta_g = 2$. In all cases $k = 3, \kappa = 0.8, \delta = 0.1$. The words "falling" and "constant" pertain to cases where mortality is decreasing or does not change over reproductive ages. The words "slow" and "exponential" pertain to cases where, if mortality rises, it does so either very slowly or at an exponential pace.

to zero. Efficient reproduction implies a high propensity for parallel investment in reproduction and growth. Consequently, a period of parallel reproduction and growth is optimal and therefore the prevalent strategy is supersustenance. All species that exhibit an efficient way of reproduction should have at least some period of reproductive life when mortality falls after the onset of reproduction. After that period the level of mortality determines whether it is optimal to withstand deterioration (supersustenance) or not (delayed subsustenance or delayed senescence). Fully senescent strategies (S=1, color=red) are suboptimal if reproduction is cheap.

High costs of reproduction ($\eta_r > 1$, row two in Figure 13 and Table 1) mainly result in senescent life-histories. When reproduction is expensive the dominant color is red in Figure 13, meaning the indicator of senescence is equal to one. Deviations from exclusive investment in reproduction ($\pi = 0$) are penalized the stronger, the more η_r exceeds one, i.e. in order to reproduce successfully the individual can not afford to reproduce and maintain its state simultaneously unless repair is sufficiently cheap. Therefore, over a broad range of mortality conditions, strategies where mortality increases over reproductive life are optimal.

5.1 The Mortality Paradox

To fully understand the optimal patterns of mortality, the influence of the costs of maintenance, captured by the columns in Figure 13 and Table 1, has to be taken into account. Interestingly, the left and right column appear to be roughly mirrored which demands attention. Clearly, low and high costs of maintenance imply opposite effects of changes along the horizontal axis, i.e. of changes in the extrinsic hazard of death.

In case of low costs of maintenances ($\eta_g < 1$, first column in Figure 13 and Table 1), non-senescence is favored the more, the stronger the extrinsic hazard. This is striking. Exactly the opposite has generally been stated – that a high risk of death should favor senescence (Williams, 1957). But my model predicts that this hypothesis is not true for low costs of maintenance. What could explain this unexpected and seemingly paradoxical result?

A well supported (Stearns (1992) and Roff (2002)) and intuitively appealing fact is 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 environment, their reproductive potential might be small. Therefore, depending on the costs of reproduction, a small potential should be maintained (sustenance) and, if possible, further increased (supersustenance) since maintenance costs are low.

If, on the other hand, life is safe the individual can afford to spend a long time to build up a high vitality, i.e. a large reproductive potential. Instead of paying the price to maintain a large vitality, it may be evolutionarily advantageous to harvest this potential at the cost of loss in functioning. The crucial point is that the age at which this loss in functioning truly becomes apparent can be postponed to an extent that the decline in vitality is almost equivalent to real maintenance, facilitated by the efficient repair system. This strategy (subsustenance) has the benefits that more energy can be allocated to reproduction, which is particularly important if reproduction is expensive.

An interesting strategy (partial senescence) is optimal at low levels of mortality, when maintenance is cheap but reproduction is expensive. 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 preservation of vitality rather than decay, which implies sharing of resources. Since mortality is low the individual can afford to mature late, attaining a high reproductive potential. However maintaining this level of vitality would strongly be 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.

In sum, for low costs of maintenance, an increase in the extrinsic hazard of death shifts the strategy from from virtual maintenance to real maintenance of vitality. Note that it might be hard to distinguish subsustenance from sustenance in reality as sample sizes in empirical data might be too small to detect an increase in mortality.

5.2 Further Results

High costs of maintenance ($\eta_g = 2$, second column in Figure 13 and Table 1) imply that a strong extrinsic hazard of death favors senescence and a weak extrinsic hazard favors non-senescence, in line with the general way of thinking. Any attempt to retard deterioration is expensive. The levels of vitality that correspond to viable reproductive potentials cannot be preserved due to the high costs of maintenance. Instead, reproductive potential is build up and subsequently harvested using all energy available and no energy is allocated to maintenance. In this case, any decay is exponential.

If both reproduction and growth are costly, $\eta_r > 1$ and $\eta_g > 1$, then exponentially increasing mortality (Gompertzian senescence) is optimal unless mortality is sufficiently low in which case sustenance is the optimal strategy.

Generally, low levels of intrinsic mortality favor non-senescence. If reproduction is cheap,

then the non-senescent strategy is supersustenance, If reproduction is costly, then the non-senescent strategy is sustenance.

As long as maintenance and/or reproduction are cheap it can be optimal to precede any period of decay by a period of parallel investment in growth and reproduction (delayed sub-sustenance, delayed partial senescence, delayed senescence). Note that parallel growth and reproduction simultaneously allows for an early age at maturity (at still small vitality) to ensure at least some reproduction in high risk environments but also for a further build up of reproductive potential.

6 The Humanesque Case

The example in Figure 2 corresponds to a senescent strategy ($S = 1$) and captures the general features of human life history. 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. Note that the simple model does not capture menopause. Life-expectancy at birth as well as life-expectancy at maturity equal 25. If the time units correspond to years this setting of parameters captures the main features of ancient human life-history. However, vitality in humans is only partly determined by the functioning of body cells. What makes humans a special case is the large brain with the capacity to learn and to acquire human capital (Kaplan and Robson, 2002). Still, the "humanesque" case can be used to understand which parameter crucially affects the boundary between senescence and non-senescence. Results are shown in Tables 2 and 3.

6.1 Changes In Efficiencies

The effects of deviations of the efficiency parameters from the humanesque case are shown in Table 2. (The humanesque case is given in the first row and again in the sixth row).

Decreasing η_g at constant η_r shifts the strategy between five (!) different categories, ranging from senescence to supersustenance. This is a striking finding. The alternating pattern between senescent and non-senescent strategies is remarkable. As discussed in section 5.1 this phenomenon can be explained by shifts between virtual and real maintenance and corresponding age and vitality at maturity. The important point, emphasized by the results in Table 2, is that the costs of maintenance crucially affect a species' characteristic age-pattern of mortality.

Decreasing η_r at constant η_g shifts the strategy between three different categories, ranging from senescence to supersustenance. Changes in the costs of reproduction have a strong influence on the age-patterns of mortality.

Table 2: Changes in Efficiencies

Cost of Reproduction η_r	Cost of Maintenance η_g	S	Strategy
2.0	2.0	1	Senescence
2.0	1.0	0	Sustenance
2.0	0.6	1	Subsustenance
2.0	0.45	0.87	Delayed subsustenance
2.0	0.4	0	Supersustenance
2.0	2.0	1	Senescence
1.0	2.0	0	Sustenance
0.4	2.0	0	Supersustenance

$k = 3$, $\kappa = 0.8$, $\delta = 0.1$, $b = 0.3$, and $c = 0.01$

In sum, the costs of maintenance, η_g, are a crucial determinant of a species' characteristic age-pattern of mortality. The costs of reproduction, η_r, are of almost equal importance to the optimal age-patterns of mortality. The humanesque life-history is at one end of two key life-history dimensions determined by the costs of maintenance and reproduction.

6.2 Changes in Mortality

Reduction in the mortality parameters, either b (from 0.3 to 0.1) or c (from 0.01 to 0.004), can change the strategy from senescent (Gompertzian senescence) to non-senescent (sustenance), as shown in Table 2.

The humanesque optimal life-history can be shifted from exponential increasing mortality to constant, non-humanesque mortality patterns by a reduction in mortality. Extrinsic causes of death have been reduced considerably over human history. Does this result suggest that the human life-history could possibly evolve towards non-senescence? Probably not, because I will show in the following that the impact of mortality changes is constrained by the magnitude of η_g and η_r.

Note that the strategy of Gompertzian senescence corresponds to a strategy of $\pi = 1$ followed by $\pi = 0$ at maturity. If this strategy is optimal for any level of η_g or η_r the strategy will not change for larger values of these parameters. The true values for the costs of maintenance and reproduction are unknown. In this specific example I chose values of 2. But maybe values of 3 or 4 or 10 or even 100 are more appropriate for the humanesque case? It turns out that even an efficiency value of 3 can prevent the shift from senescence to

Table 3: Interaction Between Efficiencies and Mortality

Cost of Reproduction η_r	Cost of Maintenance η_g	Intrinsic Mortality b	Extrinsic Mortality c	Age of Maturity α	Vitality at Maturity ψ_α	Strategy
2	2	0.3	0.01	13	104	senescent
2	2	0.3	0.004	6	51	non-senescent
2	2	0.1	0.01	3	21	non-senescent
3	2	0.3	0	20	120	senescent
3	2	0	0.01	13	106	senescent
2	3	0.3	0	19	119	senescent
2	3	0	0.01	13	106	senescent

$k = 3$, $\kappa = 0.8$, and $\delta = 0.1$

non-senescence. Table 3 illustrates, that the ultimate reduction of either intrinsic or extrinsic mortality to zero still implies that senescence is the optimal life history in the humanesque case. The efficiencies of the growth and reproductive systems restrict life-histories in their adaption to changing mortality conditions. If humanesque efficiency parameters were at the level of 2, then the model suggests that evolutionary forces could promote non-senescence in humans. If humanesque efficiency parameters are larger than 2, then senescence is inevitable in humans, at least in my model.

6.3 Influence of changes in mortality on age and vitality at maturity

For $\eta_r = \eta_g = 2$, the effects of changes in mortality on age and vitality at maturity, life-expectancy and the indicator of senescence can be seen in Figures 14 and 15. Generally in this model, higher extrinsic mortality c reduces age and vitality at maturity. This is also true for intrinsic mortality b, as long as the strategy is senescent. Higher b for non-senescent strategies increases age and vitality at maturity. Remarkably, there is a pronounced bifurcation between Gompertzian Senescence and Sustenance. For Gompertzian Senescence strategies it is important to initially build up a large reproductive potential that subsequently can be harvested at the costs of loss in functioning. Sustenance relies on a small but persistent potential that is harvested from an early age onwards.

Figures 14 and 15 exemplify the narrow line between senescence and non-senescence. Close to the bifurcation the characteristic differences in age and vitality at maturity for $\eta_r = \eta_g = 2$ become apparent. Non-senescent life-histories correspond to an early age at maturity and a

low but constant reproductive potential, whereas senescent life-histories correspond to a late age at maturity and a high but decreasing reproductive potential. This can also be seen in Table 3.

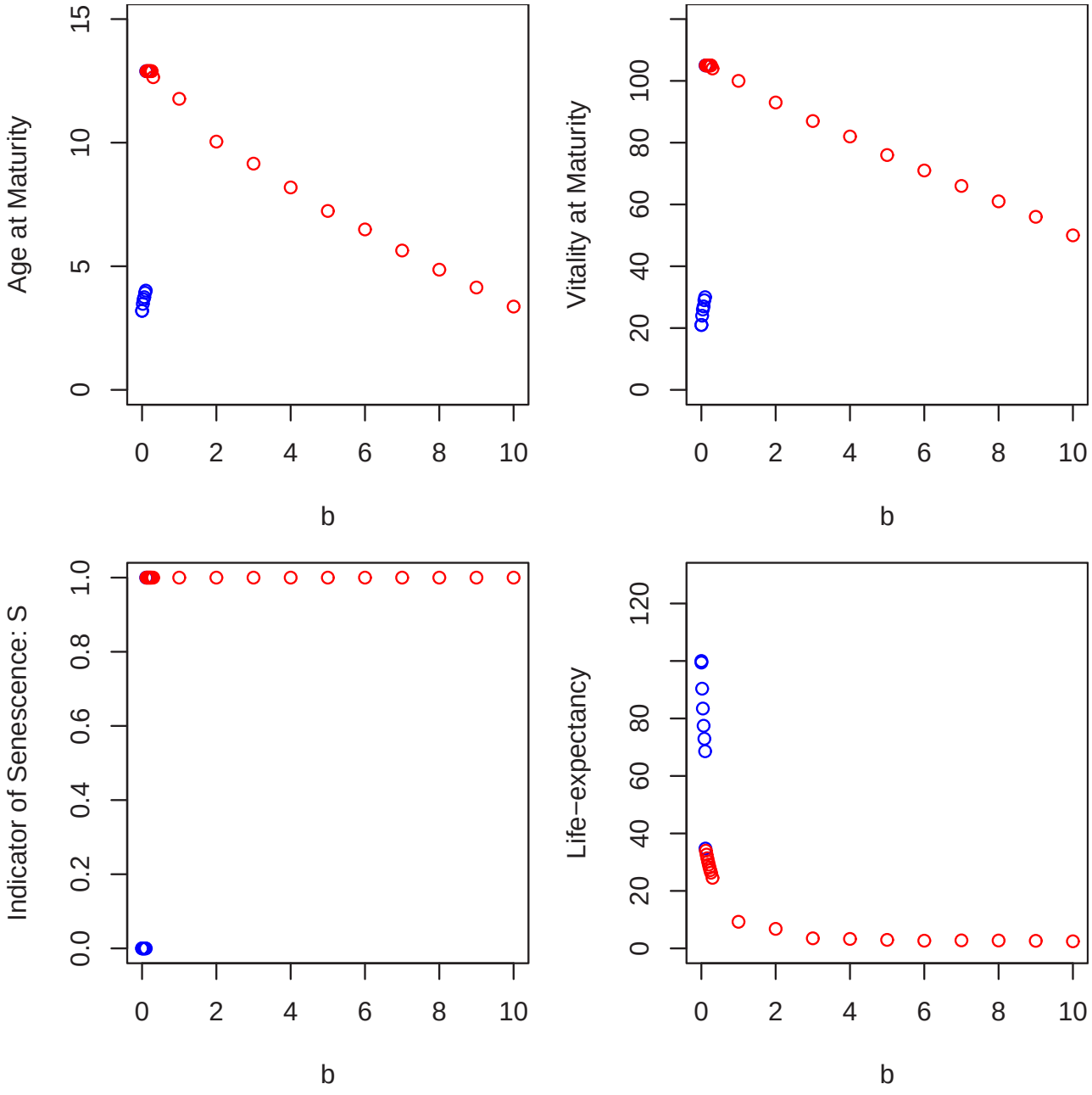

Figure 14: Impact of deviations of intrinsic mortality (b) from the humanesque case ($b = 0.3$). (red = senescence, blue = sustenance)

The values of k, κ, and δ are of no direct importance to the boundary between non-senescence and senescence. Their influence on the strategy by changing the level of maximum vitality Ψ can be offset by changes in intrinsic mortality b.

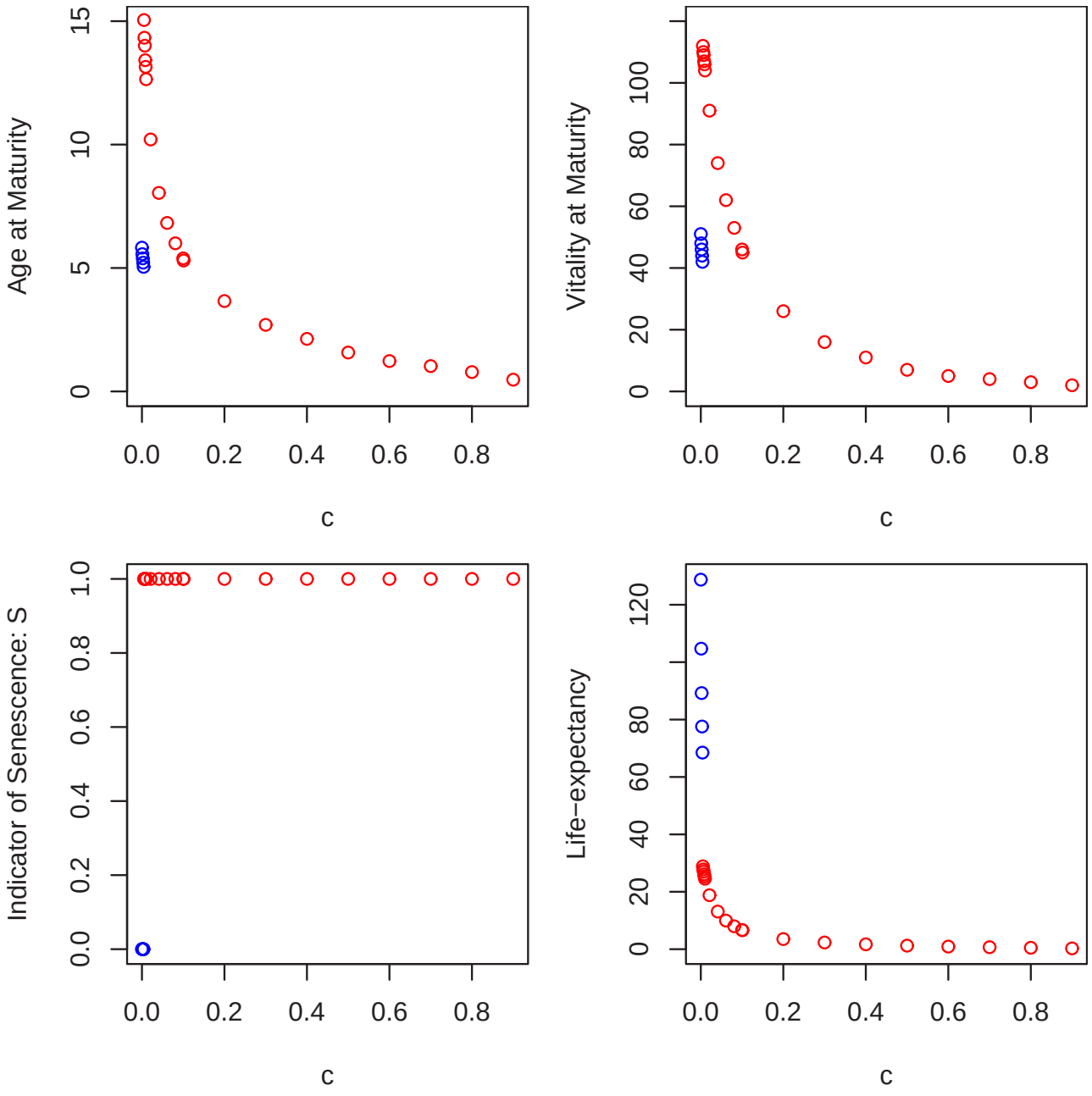

Figure 15: Deviations of extrinsic mortality (c) from the humanesque case ($c = 0.01$). (red = senescence, blue =sustenance)

7 Summary

The simple model developed in this paper captures the main features of life - mortality, reproduction, growth and maintenance. The results show that the range of optimal life histories is wide.

The costs of growth and maintenance fundamentally determine whether an optimal life-history follows a non-senescent strategy or a senescent strategy. Of almost equal importance are the costs of reproduction. The influence of mortality conditions on the boundary between non-senescence and senescence can be strong. This influence, however, is constrained by the costs of maintenance and reproduction. If the costs are too high, even reduction of either intrinsic or extrinsic mortality to zero cannot shift a senescent strategy to a non-senescent one.

An increase in the extrinsic hazard of death promotes non-senescent strategies if costs of maintenance are low,and senescent strategies if the costs of maintenance are high. Clearly, the influence of changes in mortality on a species' characteristic pattern of aging can only be understood in the light of efficiencies of reproduction and maintenance.

Generally, a low level of intrinsic mortality favors non-senescent strategies. Both intrinsic and extrinsic mortality influence details of a life-history, i.e. optimal age and vitality at maturity as well as life-expectancy but are less crucial in determining senescence versus non-senescence. The line between senescence and non-senescence is narrow and implies a sharp shift in the optimal age and vitality at maturity.

Gompertzian senescence, i.e. exponentially increasing mortality, is the prevalent optimal strategy only if both reproduction and maintenance are costly. If maintenance is cheap, then Gompertzian senescence is never optimal. If maintenance is costly but reproduction is cheap then Gompertzian senescence is only optimal at high levels of mortality.

Efficient maintenance and growth systems favor maintenance strategies while efficient reproductive systems favor strategies of parallel growth and reproduction.

Research is needed to identify the kind of species that follow different kinds of life-histories. The model suggests that life history categories may be largely determined by the efficiencies of growth and reproductive systems. Modularity, vegetative reproduction, cheap reproduction and growth as well as protected environments are characteristics that may lead to non-senescent strategies.

References

Abrams, P. A. (1993). Does increased mortality favor the evolution of more rapid scenescence? *Evolution 47*(3), 877–887.

Bellman, R. E. (1965). *Dynamic Programming*. New Jersey : Princeton University Press.

Box, G. E. P. (1979). Robustness in scientific model building. In R. L. Launer and G. N. E. Wilkinson (Eds.), *Robustness in statistics*, pp. 201–236. New York: Academic Press.

Finch, C. E. (1990). *Longevity, Senescence, and the Genome*. Chicago: University of Chicago Press.

Kaplan, H. S. and A. J. Robson (2002). The emergence of humans: The coevolution of intelligence and longevity with intergenerational transfers. *Proceedings of National Academy of Science, USA 99*(15), 10221–10226.

Lavigne, D. M. (1982). Similarity in energy budgets of animal populations. *Journal of Animal Ecology 51*, 195–206.

Léonard, D. and N. van Long (1992). *Optimal Control Theory and Static Optimization in Economics*. Cambridge: Cambridge University Press.

Perrin, N. (1992). Optimal resource allocation and the marginal value of organs. *American Naturalist 139*(6), 1344–1369.

Resznick, D. N., M. J. Bryant, D. Roff, C. K. Ghalambor, and D. E. Ghalambor (2004). Effect of extrinsic mortality on the evolution of senescence in guppies. *Nature 431*, 1095–1099.

Ricklefs, R. E. (1998). Evolutionary theories of aging: Confirmation of a fundamental prediction, with implications for the genetic basis and evolution of life span. *American Naturalist 152*, 24–44.

Roff, D. A. (2002). *Life History Evolution*. Sunderland, Massachusetts: Sinauer Associates.

Smith, J. M. (1982). *Evolution and the Theory of Games*. Cambridge University Press.

Stearns, S. C. (1992). *The Evolution of Life Histories*. Oxford: Oxford University Press.

Taylor, H. M., R. S. Gourley, C. E. Lawrence, and R. S. Kaplan (1974). Natural selection of life history attributes: An analytical approach. *Theoretical Population Biology 5*, 104–122.

Vaupel, J. W., A. Baudisch, M. Dölling, D. A. Roach, and J. Gampe (2004). The case for negative senescence. *Journal of Theoretical Population Biology 65*, 339–351.

West, G. B., J. H. Brown, and B. Enquist (2001). A general model for ontogenetic growth. *Nature 413*, 628–631.

Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution 11*(4), 398–411.