



Long-Run Economic Perspectives of an Ageing Society

## **The Mechanics of Ageing and Death: A Primer for Economists — Part IV: Evolutionary Theories of Ageing**

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# Evolutionary Theories of Ageing\*

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## Abstract

This chapter is devoted to evolutionary (life history) theories of ageing that aim for an explanation of the “genetic architecture” of the life history with a view to understanding why ageing and senescence occur. Life histories are defined as cycles of maturation, fertility and mortality. A key question is to understand why ageing, i.e. decreasing fertility and increasing mortality with age, has evolved, although it is clearly bad at the individual level. We begin by reviewing the ‘classical’ evolutionary theories of ageing: mutation accumulation, antagonistic pleiotropy and disposable soma theories. To some extent, these theories fail to explain, however, decreasing mortality at young ages and/or post reproductive survival, phenomena that occur in many species, in particular in birds and mammals, including humans. Newer theories seek to address these issues by incorporating transfer-giving to children and more detailed modeling of the physiological mechanisms underlying the process of ageing. We consider first an extension to mutation-selection theory including inter-generational transfers and then turn to discuss life history models that explicitly include optimization of a fitness criterion as an evolutionary objective function. We discuss how these models have been used to derive realistic mortality patterns and other features of ageing.

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# 1 Introduction

This chapter explores approaches from evolutionary biology to explain the phenomenon of ageing. Formalizations of the evolutionary advantage of ageing date back to Weismann (1891) who argued that ageing might help to keep population size in bounds and avoid overcrowding as well as to allow for generational replacement and hence adaptation to changing environments. However his theory was dismissed rather soon. The theory by Weismann only offers a purpose of ageing but no insight into the mechanisms of ageing.

More recently, Partridge and Barton (1993, p. 305) argue that ageing<sup>1</sup> may be seen as an evolutionary paradox: "If organisms can function well in youth, why can they not continue to do so in old age?" Since the genetic contribution to future generations is reduced by ageing, natural selection should therefore work against the force of ageing. Similarly Kirkwood (2002, p.737) states that "evolution theory argues against programmed ageing, suggesting instead that organisms are programmed for survival, not death."

While mechanistic explanations relate the process of ageing to damages of cells, tissues, organs, etc., evolutionary theories of ageing aim to explain the variation across species of avoiding or reducing these damages, i.e. why ageing may (or may not) occur.

Evolutionary theories help to explain the "genetic architecture" of the life history, i.e. when maturation, fertility and death occur. Against the backdrop of increasing age at childbirth and continuing improvements in old age survival among humans in industrialized countries, evolutionary theories of ageing may help to explain the underlying mechanisms of these changes. Moreover, by generating insights into these mechanisms, evolutionary theories of ageing may help to understand the constraints and opportunities of ageing in the future.

Following Partridge and Barton (1993) two explanations of the evolution of ageing can be distinguished. Both explanations are built on the assumption that the force of selection declines

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<sup>1</sup>While Partridge and Barton use the term ageing and senescence interchangeably, Baudisch 2008, 2009 (based on Medawar 1952) distinguish between those two concepts. While ageing can represent maintenance, deterioration or improvement with age, senescence denotes deterioration or decay, i.e. increasing mortality and/or decreasing fertility with age.

with age. The **mutation accumulation** theory of ageing (Medawar 1952, Hamilton 1966) argues that detrimental mutations that show up only late in life (i.e. after the age of reproduction) accumulate and are less likely selected out by nature. By contrast, deleterious mutations early in life (before the age of maturity) will be selected. For organisms that die young, the force of selection to oppose mutations within the genome that lead to deleterious effects in old age is therefore low. The second evolutionary explanation of ageing is based on an **optimality criterion**. In these models, forces of evolution are assumed to yield optimal life-histories in terms of an optimal trade-off of fertility and mortality, within the constraints of specific intrinsic physiological and extrinsic environmental resources.

Optimality theories of ageing include the **antagonistic pleiotropy model** by Williams (1957) and the **disposable soma theory** by Kirkwood (1977).

Antagonistic pleiotropy is similar to the mutation accumulation theory of Medawar but introduces life-history trade-offs. It assumes that some genes exhibit effects on fertility and mortality that turn from positive to negative (or vice versa) with progressing age. Such genetic trade-offs are seen as an important cause of ageing. For instance, if the evolutionary aim is to have a high number of offspring, large positive effects at younger ages might have a more important effect as compared to detrimental effects at later ages where survival and fertility is lower. Hence, natural selection declines with age.

The disposable soma theory is built on the assumption that longevity requires investment in somatic maintenance and repair and these investments compete with investments in growth and reproduction. Hence, genes that affect the maintenance and durability of the soma will also influence longevity. Kirkwood (1977) explicitly distinguishes between non reproducible (somatic) and reproductive tissues. While the former only serves a single generation it is the latter that determines the cell lineage (germ line) accounting for variability across generations. The soma therefore only helps to transport the genetic codes across generations. Since the costs of repair of the soma are too high, evolution trades off senescence of the soma with persistence of the germ line.

In life-history optimization models evolutionary success is typically measured by fitness or reproductive success of a genotype, i.e. the intrinsic rate of population increase  $r$  as implicitly defined by the Lotka equation

$$1 = \int_0^{\infty} e^{-ra} l(a) m(a) da, \quad (1)$$

where  $l(a)$  and  $m(a)$  denote the survival function and fertility rate, respectively, that apply to age  $a$  and the term  $e^{-ra}$  discounts later born offspring by the population growth rate. The unique  $r$  that solves this equation is termed Lotka's  $r$  or alternatively the intrinsic population growth rate  $r$  (i.e. intrinsic to the given survival and fertility schedule). In stationary populations where the optimal growth rate is zero, optimal trajectories of fertility and mortality can be derived by maximization of the net reproduction rate (the expected number of offspring per individual) instead as given by:

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

Partridge and Barton (1996) suggest to apply the concept of reproductive value (Fisher 1930) to measure senescence. Closely linked to fitness, the reproductive value at age  $a$  measures the remaining reproductive contribution of an individual at age  $a$  and is given by

$$v(a) = \frac{e^{ra}}{l(a)} \int_a^{\infty} e^{-rx} l(x) m(x) dx, \quad (3)$$

where  $l(x)$  and  $m(x)$  denote the survival function and fertility rate, respectively, that apply to age  $x$ . Future reproduction is discounted by the intrinsic population growth rate  $r$  through the term  $e^{-rx}$ . The constant  $\frac{e^{ra}}{l(a)}$  indicates the survival to age  $a$ .<sup>2</sup> As shown in Baudisch (2008, p. 6 ff), senescence occurs when the reproductive value declines with age. Since senescence involves the age specific dynamics of both mortality and fertility, it is the relation between those two processes that will determine whether senescence occurs. More specifically, if the relative change in mortality

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<sup>2</sup>Note that for  $r = 0$  the reproductive value at age  $a = 0$  equals the net reproduction rate  $R$ .

is greater than the relative change in fertility at a specific age, senescence will happen (Baudisch 2008, p. 8).<sup>3</sup>

Empirical tests of the evolutionary theories of ageing are numerous (see Partridge and Barton 1993 for a review) and commonly employ model organisms such as fruitflies. Organisms that are faced with low extrinsic risks of mortality or/and show up increasing fertility with age, are those that exhibit high maximum lifespan. These characteristics obviously reflect selection for high levels of maintenance and hence for long life spans. However, distinguishing between the specific theories is more difficult.

After this brief review of the 'classical theories' of evolutionary explanations of ageing, in the following sections we introduce more recent contributions that aim to explain decreasing mortality at young ages and/or post reproductive survival. These phenomena occur in many species, in particular in birds and mammals, including humans, and cannot be explained by the classical theories introduced so far. These newer theories seek to address these issues by incorporating transfer-giving to children and more detailed modeling of the physiological mechanisms underlying the process of ageing. We consider first an extension to mutation-selection theory including inter-generational transfers and then turn to discuss life history models that explicitly include optimization of the reproductive value as an evolutionary objective function. We discuss how these models have been used to derive the scope for negative senescence and realistic U-shaped mortality patterns.

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<sup>3</sup>Taylor et al. (1974) have shown that maximization of the net reproduction rate is mathematically equivalent to maximizing the ultimate rate of increase  $r$ , i.e. fitness.

## 2 The role of intergenerational transfers in mutation-selection equilibrium

Lee (2003) seeks to provide an explanation for both the U-shaped mortality schedules and post-reproductive survival that are found in many species, including humans.<sup>4</sup> In so doing he explicitly accounts for the relevance of inter-generational transfers, in particular of transfers from older individuals (parents but possibly also grand-parents) to the young. Such transfers, to be understood in a wide sense as the provision of care and food (potentially even within utero), improve the quality of the offspring in terms of their capacity to survive, reproduce and produce resources (e.g. by forageing).

In the presence of transfer-giving a quantity-quality trade-off arises with respect to children not dissimilar to the one studied by Becker and Lewis (1973): A larger number of children through higher fertility and/or lower child mortality implies that each surviving child receives a lower volume of transfers. But then fertility and mortality during childhood take on an ambiguous role for the maximization of fitness as in (1). Consider the death of a child during its early development. Here, the loss of the investments accumulated in that child is small and likely to be over-compensated by the resources freed up for improving the quality of the remaining offspring. Thus, *early child mortality* may well be a beneficial genotype that is *selected in*. In contrast, the death of an adolescent at the point of turning into an adult would constitute a considerable loss of reproductive and productive capacity. Furthermore, as such an individual is no longer prone to receive transfers, its death would not free up any additional resources. Consequently, *adolescent mortality* should be a trait that is *selected out*. Thus, the quality-quantity trade-off can, indeed, explain decreasing mortality over the period of childhood up to the point of adolescence. Likewise, by implying a quality disadvantage, too high a level of fertility may be a trait that is selected out within species for whom transfers towards their children are important.

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<sup>4</sup>The life-history models by Kaplan and Robson (2002, 2009) and Robson and Kaplan (2003, 2007) as well as those by Chu and Lee (2006) and Chu et al. (2008) also include intergenerational transfers. However, as these models follow the optimal life history approach we defer their discussion to section 3

At the other end of the life-line, the provision of transfers would imply a contribution to fitness even by post-reproductive individuals. Thus, in contrast to the earlier theories that cannot explain extended periods of post-reproductive survival, this phenomenon is consistent with the role of transfer giving. Indeed, *mortality* should be *selected out* for those older individuals that are still able to contribute towards transfers.

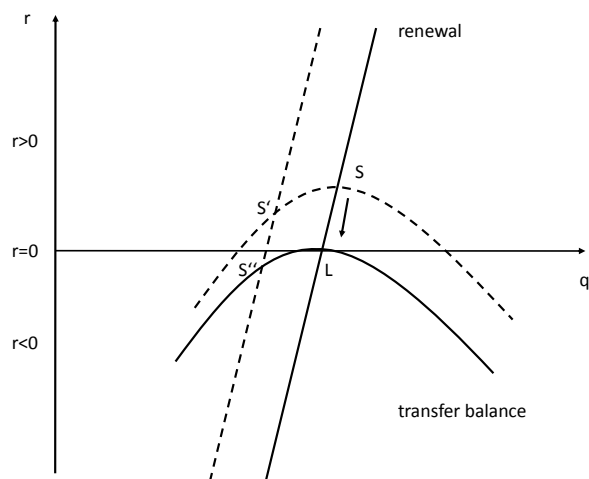
Building on this intuition, Lee (2003) studies population equilibrium in terms of net population growth,  $r$ , and the quality of individuals,  $q$ , when quality can be enhanced by intergenerational transfers (see Figure 1).<sup>5</sup> Equilibrium points in  $(q, r)$ -space (e.g. S, S', S'', L) are defined by the intersection of a renewal schedule and the locus at which transfers balance out across generations at each point in time. The positive slope of the renewal schedule follows as a higher average quality in the population allows higher survival  $l(a)$  and/or fertility  $m(a)$  in the fitness equation (1), thus allowing for a higher intrinsic rate of population growth,  $r$ . The quality-quantity trade-off implies a concave shape of the transfer balance. A higher rate of growth,  $r$ , brings with it a larger share of young individuals in the population. As young individuals receive rather than contribute transfers, fewer resources are available per capita and quality is lower. Hence for high levels of quality the transfer balance is downward sloped. Starting from low levels, however, improvements in  $q$  allow greater productivity and thus the maintenance of a balanced budget even at an increased rate of population growth.

In the short-run, population equilibrium may imply positive, negative or zero population growth as long as the transfers are balanced (i.e. any of the equilibria S, S', S'' and L are feasible). Here, a population carrying a certain mutation would gradually replace the original population if and only if it exhibits stronger growth. In the long-run, only zero growth,  $r = 0$ , is feasible, however. Any shrinking population ( $r < 0$ ) would eventually vanish, whereas sustained growth at  $r > 0$  is unfeasible as it would at some point hit the carrying capacity of the ecosystem. At that point the transfer balance shifts downwards, where for any level of quality the reduction in available resources stifles growth (e.g. the shift from S to L). Lee (2003) examines the effects on

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<sup>5</sup>Lee (2003) himself refers to the 'level of consumption' ( $\gamma$ ) rather than 'quality'.

Figure 1: Tradeoff between population growth and quality of individuals.



Source: adapted from Lee (2003)

short-run equilibrium growth and quality of a variety of mutations in fertility and mortality, which in some cases run counter to the 'classic' selection theory. For instance, starting from S and for a given transfer balance, a mutation leading to greater fertility would in the new equilibrium S' result in both lower quality and growth.<sup>6</sup> More prominently, however, Lee (2003) finds that in a long-run equilibrium with  $r = 0$ , selection can only work through the transfer balance and not through the 'classical' channels of renewal. Selection should ensure that the long-run equilibrium is characterized by an optimal transfer-structure.<sup>7</sup> This implies that any long-run equilibrium point must lie at the peak of the balanced budget line (as e.g. L). But then any further mutation is inconsequential unless it affects the budget balance.<sup>8</sup> This leads Lee (2003) to the conclusion that transfers, not births, shape the process of ageing in social, i.e. transfer-giving, species.

<sup>6</sup>In fact, an increase in fertility would also shift downwards the transfer budget, implying that  $q$  and  $r$  will lie at even lower levels, as e.g. in S''.

<sup>7</sup>This reasoning begs an interesting analogy: Optimality theories of ageing are equivalent to social planner solutions in economics; whereas theories of mutation-selection balance are more about the adaptive processes that may (or may not) lead to an optimal long-run equilibrium (as e.g. in Lee 2003). Thus, to a degree they resemble the 'decentralized' solution in economics.

<sup>8</sup>Consider, e.g. an upward shift of the renewal line (e.g. due to an increase in fertility) starting from L. The new equilibrium compatible with the transfer balance is S'', where  $r < 0$ . The same obviously holds for a downward shift in renewal.

Lee (2008) simulates the evolutionary process for a single-sex hunter-gatherer society in the presence of transfers. The underlying model is somewhat more general than the one in Lee (2003) and allows for non-stable age-structures and, importantly, for different structures of kinship. As it turns out, the results of Lee (2003) are largely confirmed, namely that the presence of transfers explains declining child-mortality and post-reproductive survival. Kinship, however, plays a crucial role. More specifically, the impact of transfer giving on the mortality pattern is the more pronounced the closer is kinship as measured by the degree to which resources are shared within the kingroup only. If resources are shared at population level, then the 'classical' result reemerges: mortality is flat up to the age of sexual maturity and there is no post-reproductive survival.

Despite their prominent role in qualifying the classical evolutionary theory of ageing, Lee's (2003, 2008) contributions have a number of drawbacks arising from the fact that by taking a macro-perspective they make a number of ad-hoc assumptions about the physiological underpinnings (e.g. the relationships between mortality, fertility and production and between all of these and 'quality'). Thus, the trade-offs involved with the resource allocation within the individual remain obscure and the optimal allocation of resources is not characterized. A precise statement of the age-schedules of fertility, mortality and transfers that would maximize fitness in (1) is also ruled out.

A more detailed characterization of the resource allocation and its age-structure can be gained by the use of optimal life-history models. These models focus at the optimal long-run equilibrium (as e.g. point L in Figure 1) straight away without asking, however, as to whether or not such a state can be attained. The optimal age-schedules of fertility, mortality and, where relevant, transfer-giving are then derived as the solution to a problem where fitness in (1) is maximized subject to physiological and transfer constraints.

### 3 Optimal life-history theories of ageing

Optimal life-history theories are based on the trade-off between reproduction and survival. From an evolutionary point of view, the strategy that yields the highest fitness (in terms of maximizing lifetime reproductive success) will be selected. The specific assumption on the trade-off will determine the age pattern of mortality and fertility that is chosen by evolution. An excellent review on how different assumptions on the trade-off between survival and reproduction shape the patterns of ageing (allowing for senescence, sustenance, or inverse senescence) is given in Baudisch (2009). "Crucial points in the model assumptions are linearity vs. non-linearity in the trade-offs, inclusion or exclusion of mediating variables that determine either or both mortality and fertility, endpoint conditions of the problem's time horizon, future returns to current investment reflected in the potential for indeterminate growth, and constraints on the qualitative shape of mortality and fertility patterns." (Baudisch 2009, p. 9).

#### 3.1 The case for negative senescence

In the following we review a model by Vaupel et al. (2004) that introduces an intermediate variable (the physiological state size) that determines fertility and mortality. Size itself evolves dynamically over the life-history and is determined by optimal investment into the maintenance of size and reproduction. Depending on the specific dependence of fertility and mortality on size, various patterns of ageing are possible.

Based on optimization models of life-history strategies Vaupel et al. (2004) present an evolutionary explanation of age specific mortality and fertility. In particular, they question Hamilton's theory (Hamilton 1966) that senescence (defined as age-related deterioration of the organism) cannot be avoided. Observations from human, animal and plant populations provide evidence against Hamilton's theory since mortality declines from age of conception to age of sexual maturity and may thereafter exhibit increasing, decreasing or stable age trajectories (see Figure 1 in Vaupel et al. 2004). Hence, mortality may decline and fertility may increase after the age of reproductive

maturity. Such a pattern would constitute negative senescence.

To conciliate negative senescence with an evolutionary optimal strategy the authors refer to a model of size-dependent mortality. Under conditions of increasing size with age and negative dependence of mortality on size, negative senescence (i.e. decreasing mortality with age) could be the result. The evolutionary model chosen involves maximization of the net reproduction rate at age zero,  $R$ , as given by equation (2).

The authors assume that an individual's size (encompassing its vitality and strength) determines its resources that are split between growth and maintenance on the one hand and reproduction on the other hand. Starting from the *maintenance mode*, i.e. a state where size and hence mortality and fertility are constant over age, they derive conditions under which it would be optimal to temporarily invest more in growth and sacrifice reproduction for the sake of larger size and hence higher reproduction and lower mortality later on. The temporary phase of higher investment in growth coincides with the period of negative senescence. Formally, the condition for negative senescence is an increase in the reproductive value of the latter strategy (investing temporarily in growth) as opposed to the maintenance strategy.

The authors next introduce a more general framework assuming that size is a function of age and its change over time is determined by size specific deterioration (higher size implies more complexity and consequently higher depreciation) together with age specific investments (as measured by a specific fraction  $0 \leq \pi(a) \leq 1$  of the age specific size). Hence, damage and repair determine the stage of the size variable. The remaining investments  $1 - \pi(a)$  determine reproduction. Mortality is assumed to be inversely related to the size of the organism. Unless the authors assume a non-linear fertility function (i.e. the control  $\pi(a)$  enters the fertility function in a non-linear way), negative senescence does not occur. The only possible outcome is a strategy of first putting all investment into growth and then switching to the maintenance strategy at the onset of reproduction. Though negative senescence is not the standard outcome it is definitely not positive senescence that can be observed in the theoretical model. This is because the maintenance mode implies constant size and hence unchanging mortality. To obtain positive senescence the

authors introduce a model with determinate growth as opposed to the models of indeterminate growth considered so far. For this purpose they introduce a second state variable in addition to size, describing the functionality of the body. The product of the size and functionality variable denotes the vitality of the individual.

In summary, the theoretical models outlined in Vaupel et al. (2004) imply that (i) senescence is likely for species that obtain their maximum size around the age of maturity and for which fertility declines with age. (ii) If fertility does not decline with age and if maximum size is attained near but not at the age of maturity, senescence is not likely, and finally (iii) negative senescence may be characteristic for species where fertility increases with age and maximum size of the species is obtained far above the age of maturity.

In terms of the trade-off between reproduction and maintenance the findings are that inverse senescence is related to concave trade-offs, convex trade-offs lead to senescence and linear trade-offs imply sustenance as characterized by constant mortality and fertility (see Baudisch 2009b, page 9).

### **3.2 Explaining U-shaped mortality and post-reproductive survival: Models including intergenerational transfers**

In the following, we focus on a number of life-history models drawing on intergenerational transfers as a key factor in explaining U-shaped mortality profiles and post-reproductive survival. As it turns out, these models yield important lessons for ageing.<sup>9</sup>

Kaplan and Robson (2002) and Robson and Kaplan (2003) seek to explain the coevolution of brain size and longevity as it has occurred in primates, including humans. The brain is interpreted

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<sup>9</sup>U-shaped mortality is also explained by uncertainty with respect to mortality (Sozou 1998, Sozou and Seymour 2003) and size effects (Tuljapurkar and Boe 1993, Tuljapurkar 1997). In the former case, the gradual resolution of initial uncertainty with respect to (e.g. extrinsic) mortality leads to increasing investments into survival and thus declining mortality during childhood. When size enhances fertility or mortality, then the fitness returns from an individual increase in its size, implying again declining mortality during the growth phase. Tuljapurkar and Boe (1993) and Tuljapurkar (1997) also show that stochastic fertility may lead to multiple equilibria in the sense of several different phenotypes generating the same (maximal) level of fitness. In this case, mortality may exhibit a relatively flat profile late in life.

as a capital stock that requires a costly investment in life's early stages and yields returns later on, flowing e.g. from an improved capability in foraging. Mortality renders uncertain the returns to investments in brain size, similar to investments in financial or human capital within economic life-cycle models with uncertain survival (e.g. Ehrlich 2000, Becker 2007). In analogy to these models, the desire to self-insure against the mortality risk then generates an incentive to invest in survival, where improved survival boosts, in turn, investments into 'brain' capital. Brain size and survival turn out to be complements, again very much in analogy to the complementarities between health and education (Becker 2007). Due to this complementarity one would, indeed, expect brain size and longevity to coevolve. In particular, as the productivity of the brain increases with the complexity of the environment, it follows that species living in more complex environments develop both larger brain sizes and lower mortality. Indeed, these patterns are confirmed by empirical evidence.<sup>10</sup>

Kaplan and Robson (2002) and Robson and Kaplan (2003) start out from a typical life-history model with a fitness objective as in (1) but then go on to show that the problem is equivalent to an economic problem in which an individual's life-time energy surplus is maximized subject to a per period production and survival function. The period production of energy increases in brain size and is used to 'finance' investments in survival. The remaining (net) energy can be transferred over time and is converted into fertility. The brain is treated as a capital asset, serving to increase per period productivity. In Robson and Kaplan (2003) investments into the brain are subject to increasing costs and are, therefore, stretched out over several periods (of childhood). The model is solved for the optimal steady-state level of capital, implying an investment path during childhood, and for the optimal path of investment into survival. They show that the solution implies, indeed, a U-shaped mortality schedule. The mortality decline during childhood is driven by an increase in the value of life (or survival) over the period in which there are ongoing investments in brain size. Here, the value of life is to be understood as the discounted expected

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<sup>10</sup>Kaplan and Robson (2002) give an illustrating example: The human brain, for instance, exceeds the brain size of our closest relatives, chimpanzees and gorillas by a factor of four, while our lifespan is longer by a factor of about two.

net energy surplus over the remaining life course, which increases in the capital stock. Mortality reductions carry on for a certain period after maturity of the brain as long as learning by-doing, say, leads to further increases in productivity. When productivity declines from some age onwards, this implies a decrease in the value of life and, thus, declining investments into survival.<sup>11</sup> Thus, while not inevitable, the onset of ageing is determined by reductions in productivity, as triggered by reductions in the quality of brain-capital.

The model helps to explain the rectangularization of mortality in humans relative to primates, where for a given level of mortality the human mortality profile exhibits a steeper decline (increase) during youth (old age). The reason lies in the value of life, the change of which over time is driven by the current net-surplus lost with the progress of time. As for humans both the costs and returns from investments into brain size are larger, the net surplus during youth is prone to be smaller (or more negative) than that for chimpanzees, say, implying a lower depreciation of the value of life. In contrast, for older individuals the loss of net-surplus per life-year (in terms of productive capacity) is positive and larger for humans than for chimpanzees. This implies an overall steeper mortality profile for humans (albeit at a lower level for all ages). More generally, this insight has the interesting implication that the speed of ageing, as measured by increasing mortality, is importantly governed by the functionality of the body.

However, this leaves unanswered the question as to what precisely is governing the functionality of the body. The assumption by Kaplan and Robson (2002) and Robson and Kaplan (2003) that the productivity of the human brain declines from some age onwards is intuitive and evidently confirmed. However, as the authors themselves point out, this is not sufficient from the perspective of life-history modelling. A priori it is not clear why evolution should arrange the body in a way that functionality (of the brain or other parts) declines with age. Indeed, Vaupel et al. (2004) show that it may as well be optimal to maintain functionality at a steady level for all times and,

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<sup>11</sup>The notion of value of life has been used extensively in the modelling of mortality risks over the life-cycle (see e.g. Shepard and Zeckhauser, 1984; Murphy and Topel, 2006). In these models, the economic value of life corresponds to the willingness to pay for a decline in mortality. Notably, it is subject to a similar pattern as the evolutionary value of life in Kaplan and Robson (2003): Increases (declines) in earnings capacity during the early (late) career boost (lower) the value of life.

thereby, forestall senescence. Thus, declining functionality would somehow have to be explained as an optimal strategy. This problem is addressed in Robson and Kaplan (2007) and Kaplan and Robson (2009), where a life-history model similar to Robson and Kaplan (2003) is amended to include not only the size of the body (or brain) but also its quality. Drawing on the disposable-soma theory (e.g. Kirkwood 1977, 1997), they assume that 'somatic' (i.e. body) cells are subject to damage, reducing their functionality/quality, but may be repaired (or even improved) at a cost (per cell). Thus, while the quality of the body may be maintained at full functionality, the cost of doing so increases with body size. This triggers a quantity-quality trade-off, where large bodies are more productive in generating energy but are also more costly in repair. In contrast, 'germ' cells, serving as the vehicle to pass on the genetic code to offspring, can be maintained at full functionality at negligible cost. The optimal evolutionary investment strategy is then to build up a certain body size and keep it constant for the remaining life. During the initial phase of growth, the marginal benefit but also the marginal cost from quality improvements increase jointly, triggering an ambiguous path of quality investments. At higher ages where body size is fixed, rising mortality depresses the marginal benefit from quality investments (related to the value of life). For a given marginal cost of quality this leads to a decline in maintenance effort and, thus, to a decline in cell quality. This, in turn, depresses the productivity of the somatic capital, triggering a reduction in investments into survival and, thus, an increase in mortality.

Senescence is therefore induced for two reasons: In the presence of a germ line, the quality of which can be maintained at virtually no cost, the only reason to maintain the quality of somatic capital lies in its role for providing energy. However, as it is efficient to grow to a (relatively) large body size, the associated maintenance cost renders it efficient to run down the quality of somatic capital. While being plausible these results hinge on the assumptions about the depreciation of quality and the cost of maintenance. Indeed, for certain specifications of the cost function, it may well be the case that permanent maintenance of quality is efficient. Thus, a deeper understanding yet of the physiological properties is called for.

As Kaplan and Robson (2009) point out, two assumptions are to some extent critical. First, the

irreversibility of growth implies that somatic capital cannot be shrunk in order to lower the cost of maintenance. Second, inter-generational transfers towards offspring are crucial in an environment in which resources cannot be stored and in which initial productivity is low. Similar to Lee (2003) these transfers provide the only plausible explanation for post-reproductive survival.<sup>12</sup>

Nevertheless, the analysis of the flow of transfers and their role within evolution remains somewhat implicit in Kaplan and Robson (2002) and Robson and Kaplan (2003, 2007). A more detailed take on the role of intergenerational transfers is provided in the models of Chu and Lee (2006) and Chu et al. (2008). Chu and Lee (2006) focus on the coevolution of intergenerational transfers and longevity. They consider a discrete-time life history model where a fitness term similar to (1) is maximized by choice of an age-dependent strategy of fertility, maintenance and growth subject to a linear (energy) budget constraint. Total available energy depends on body-size. From the solution to the dynamic programming problem they conclude that transfer-giving is the more likely to evolve the greater survival and the greater the increase with age in the efficiency of energy production (e.g. by way of foraging). Conversely, if transfer-giving evolves this enhances improvements in survival up to the age at which transfers are made. Chu and Lee (2006) do not endogenize the age-structure of transfer giving, they rather focus on the preconditions for a transfer to occur at some given age and on its effects on mortality. Thus, to some extent the model remains open ended.

Chu et al. (2008) consider a continuous-time version of the model in Chu and Lee (2006) with a more general (possibly non-linear) energy budget constraint. They also endogenize intergenerational transfers in the sense that they can be chosen freely as long as they balance out across generations at each point in time. They formally identify the reason for declining juvenile mortality that was more intuitively proposed in Lee (2003): namely as the early death of a juvenile sets free a larger set of resources to be used for surviving offspring than a late death, this lowers

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<sup>12</sup>For humans, who can at least to some extent make conscious decision about reproduction and the allocation of resources, the question emerges as to how evolution 'implements' an optimal strategy. Here the literature on the evolutionary foundation of preferences provides some answers (Robson and Samuelson, forthcoming). In particular, it is asked which age profile for the rate of time preference is consistent with the evolutionary optimum (Hansson and Stuart, 1990; Rogers, 1994; Sozou, 1998, 2009; Sozou and Seymour, 2003; Robson and Samuelson 2007, 2009; Robson and Szentes 2008; and Kageyama 2009).

the value of 'early' relative to 'late' juvenile survival.<sup>13</sup> Chu et al. (2008) identify a further effect driving towards declining mortality during young ages: to the extent that young individuals are already self-supporting (at least partially so) it pays to sacrifice early-life survival for early-life growth, as the latter serves to relax the energy budget constraint.

In contrast to Robson and Kaplan (2003), the models by Chu et al. (2008) and Kaplan and Robson (2009) explicitly optimize over fertility, which allows them to provide a more precise account of the determinants of adult mortality. Again, the benefit from survival can be summarized in the value of life. As it turns out, given an optimal fertility schedule, the value of adult life now falls into two distinct age-specific components: the remaining reproductive value and the value (in fitness terms) of transfers still to be made to juveniles.<sup>14</sup> The former corresponds to Hamilton's (1966) value of survival for the purpose of reproduction, whereas the latter provides an explicit measure of the value of post-reproductive survival.

We can summarize the most important lessons for an understanding of ageing as follows. (i) The speed of ageing, as measured by the rate of mortality (and its change), depends on three driving factors: First, the remaining reproductive value. Second, the degree to which the adult individual still contributes towards investments into descendants. Third, the productivity, in terms of generating energy surplus, of the somatic capital (body, brain size and quality) and the pace at which it depreciates. Bodies with greater productivity tend to age later but at a greater pace. (ii) From a life-history perspective, the process of ageing cannot be understood fully without an equal understanding of the process of fertility and investments during childhood. This is obvious for the first two determinants. But even the third determinant cannot be understood without an early life perspective, as both the size and the quality of the somatic capital is determined during

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<sup>13</sup>To some extent this is the flipside of the effect identified in Robson and Kaplan (2003), where an increased stock of capital - as accumulated by way of transfers - raises the value of (juvenile) life.

<sup>14</sup>Thus, the value of life falls into a reproductive part and a productive (economic) part. Again, there is an interesting analogy to economic life-cycle models. There, the value of life has conventionally been determined under the assumption of egoistic preferences, excluding any value assigned to descendants. This value of 'current' life corresponds to the 'economic' part in the evolutionary value of life. However, once dynastic preferences a la Becker and Barro (1988) are considered, the value of life is amended by a reproductive part, measuring the individual's value of progeny (Birchenall and Soares, 2009; Kuhn et al., 2010).

childhood. Thus, an analysis of the late stages of life must remain incomplete unless it draws at least to some extent on the processes during early life.

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