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## The case for negative senescence

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Received 18 July 2003

### Abstract

Negative senescence is characterized by a decline in mortality with age after reproductive maturity, generally accompanied by an increase in fecundity. Hamilton (1966) ruled out negative senescence: we adumbrate the deficiencies of his model. We review empirical studies of various plants and some kinds of animals that may experience negative senescence and conclude that negative senescence may be widespread, especially in indeterminate-growth species for which size and fertility increase with age. We develop optimization models of life-history strategies that demonstrate that negative senescence is theoretically possible. More generally, our models contribute to understanding of the evolutionary and demographic forces that mold the age-trajectories of mortality, fertility and growth.

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**Keywords:** Aging; Mortality; Longevity; Life history; Optimization models

### 1. Growing Younger

How long do individuals in different species live? How fecund are they? How big do they grow? Such questions about the age-trajectories of mortality, fertility and growth are of fundamental interest to biodemographers, life-history biologists and evolutionary theorists. There is a vast empirical literature about these age-trajectories and a large body of theoretical work. An important topic, however, has been neglected: negative senescence.

The now familiar concept of negative numbers horrified our ancestors. Similarly, the notion of negative senescence was received with howls of derision when one of us (Vaupel) uttered the phrase in 2002 at a research workshop on the biology of aging. “Hamilton (1966) proved senescence is universal!” Hamilton, however, proved no such thing. It is high time to explicitly confront and judiciously consider the possibility of negative senescence.

Three well-known gerontologists (Comfort, 1956; Stehler, 1977; Finch, 1990) emphasized that “certain

animals and plants do not manifest increases of mortality rate or other signs of senescence” (Finch, 1990, p. 221). In particular, Finch (1990, 1998), Finch and Austad (2001) and Ottinger et al. (2003) have prepared the way for studies of negative senescence by focusing research on species with “negligible senescence”, i.e., species for which death rates rise very slowly, if at all, with age.

Here we build on Finch’s insightful contributions to make a theoretical and empirical case that some and perhaps many species show negative senescence, with death rates falling with age for an extended period following the start of reproduction. Following Finch (1990, p. 5), we define senescence as “age-related changes in an organism that adversely affect its vitality and functions, but most important increase the mortality rate ...”. Hence, senescence is characterized by death rates that increase with age and negative senescence is characterized by death rates that decline with age. It may also typically be the case that fertility and functioning decline as mortality increases (in the case of senescence) and that fertility and functioning increase as mortality declines (in the case of negative senescence).

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## 2. Hamilton's narrow road

Hamilton's influential article on "The Moulding of Senescence by Natural Selection" (Hamilton, 1966; see also Hamilton, 1996) combines insights about the evolution of senescence (Medawar, 1952; Williams, 1957) with concepts and models of population dynamics (Lotka, 1924). Hamilton asserts that senescence "cannot be avoided by any conceivable organism" and that "senescence is an inevitable outcome of evolution". His argument is not based on the optimization of fitness in a life-history model. Instead he focuses on deriving a general measure of the force of natural selection to oppose age-specific deleterious mutations. These mutations are not adaptive: they either reduce fertility or increase mortality. He purports to show that the force of selection against such mutations declines with age after sexual maturity. He concludes that deleterious mutations will accumulate at older ages. His argument, however, has several major deficiencies.

Hamilton proposes two indicators of the force of selection:  $dr/d\ln p_a$  and  $dr/dm_a$ , where  $r$  is Lotka's intrinsic rate of population growth,  $p_a$  is the probability of surviving from exact age  $a$  to  $a + 1$  and  $m_a$  is fertility between  $a$  and  $a + 1$ . He proves that these two indicators decline with age. Other indicators are also reasonable, including  $dr/dp_a$ ,  $dr/d\ln m_a$ ,  $dr/dq_a$  and  $dr/d\ln q_a$  (where  $q_a$  is the probability of death between ages  $a$  and  $a + 1$ ). Each of these four indicators can decrease, remain level, or increase with age depending on the shapes of the age-trajectories of mortality and fertility (Baudisch, 2004a).

The build-up of deleterious mutations that act at some specific age depends not only on the rate at which such mutations are eliminated by selection but also on the rate at which the mutations occur. Hamilton implicitly assumes that deleterious age-specific mutations are not rare and that the rate at which they occur

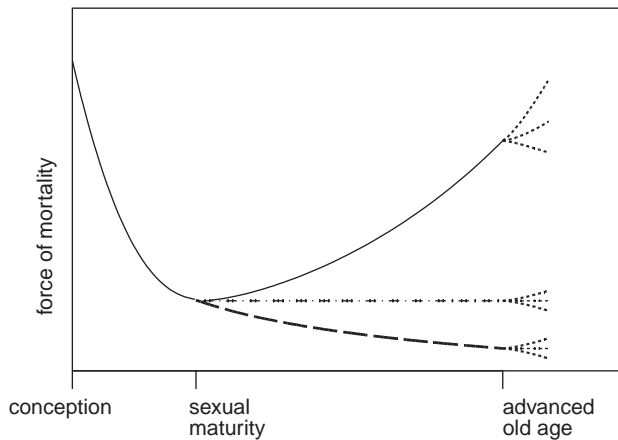


Fig. 1. Different mortality trajectories after onset of reproduction.

rise in mortality very late in life. There then could be a period of senescence following the extended period of negative senescence. This does not make the period of negative senescence uninteresting. Indeed, if almost all individuals are dead before the late period of senescence begins, then the extended period of negative senescence following development is clearly important.

Fig. 1 summarizes the various possibilities. During the first phase of life, development, mortality declines. During the second phase, mortality may increase (senescence), mortality may remain roughly constant (negligible senescence), or mortality may decline (negative senescence). Then late in life mortality may increase, level off or decline. As noted earlier, a levelling off or decline in mortality has been observed for several species (including humans, various insects and nematode worms) at advanced ages. This fact has not been used to cast doubt on the fact that these species show senescence over most of their adult lives. Gerontologists typically assume that death rates start rising when reproductive maturity is attained and there is considerable evidence for this for many mammals and birds (Finch et al., 1990). That is, gerontologists do not confine senescence to advanced old age. Hence, if it turns out that mortality increases late in life for some species whose adult lives are characterized by declining mortality, this fact should not be used to deprecate negative senescence. Special explanations may be required to understand rising or falling mortality at the outer end of life.

#### 4. Size-dependent mortality

As Caswell argues, for many organisms “the age of an individual tells little or nothing about its demographic properties” (Caswell, 2001, p. 39). This statement will not surprise ecologists, but it may astound some

gerontologists and demographers. Understanding of aging could be advanced by comparing species for which age is critical with species for which age is unimportant or only indirectly important.

Often what is important is size or stage of development. If mortality falls as size increases and if size increases with age, then mortality will fall with age. This appears to be the case for the plant *Plantago lanceolata* after seasonal effects are removed (Roach and Gampe, 2004). This tantalizing but tentative finding motivated us to take negative senescence seriously. Furthermore, it suggested to us that increases in size with age, in species for which size is strongly associated with continued survival, might be the most likely origin of negative senescence. Hence, we will emphasize size in this article. There may, however, be other factors contributing to negative senescence, as we discuss in the final section.

Caswell (2001, p. 39) concludes that “[s]ize-dependent demography is probably the rule rather than the exception and is especially pronounced in species with a large range of adult body size as a result of indeterminate adult growth.” He discusses increases in fertility as well as decreases in mortality with size and provides numerous examples and references. Finch (1990), Finch and Austad (2001), Ottinger et al. (2003) also provide much useful information. Finch and his colleagues focus on species for which there is evidence that death rates increase very slowly if at all with age. Many of the species they review, however, are candidates for negative senescence.

The strongest evidence for negative senescence in animal species comes from studies of corals. Babcock (1991) shows in three coral species (*Goniastrea aspera*, *G. favulus*, and *Platygyra sinensis*) that mortality is inversely related to colony size and age. Furthermore, the total fecundity of the three species increases steeply with size and age, “due to a combination of increased polyp fecundity and increased surface area” (Babcock, 1991). Grigg (1977) presents comparable results for two other corals, *Muricea californica* and *Muricea fruticosa*. Like the massive reef-building corals, some plants develop into large clonal clusters (Finch, 1990, Table 4.2, p. 229). The quaking aspen (*Populus tremuloides*) grove studied by Kemperman and Barnes (1976) covered 81,000 square meters and was estimated to be at least 10,000 years old. It seems likely that the bigger such a clonal cluster is, the lower is its chance of death.

Other candidates for species with negative senescence include the wild leek *Allium tricocum* (Nault and Gagnon, 1993), brown algae *Ascophyllum nodosum* (Aberg, 1992), the forest tree *Garcinia lucida* (Guedje et al., 2003), the neotropical tree *Cecropia obtusifolia* (Alvarez-Buylla and Martinez-Ramos, 1992) and the cushion plant *Limonium delicatulum* (Hegazy, 1992).

The strongest evidence for negative senescence in non-modular animals can be found for some species of

molluscs. Fertility often increases by 10-fold or so as individuals grow following sexual maturity and mortality decreases sharply (e.g., for the marine gastropods *Umbonium costatum* (Noda, 1991; Noda et al., 1995) and *Littorina rudis* (Hughes and Roberts, 1981) and the bivalve *Yoldia notabilis* (Nakaoka, 1994, 1996)). There is also evidence of negative senescence for sea urchins (Ebert and Southon, 2003). Hydra (Martinez, 1998) may enjoy negative senescence at younger ages followed by negligible senescence at older age.

Some vertebrates may possibly enjoy negative senescence. Finch (1990) summarizes suggestive data on rockfish, hagfish and various other species. Although reliable mortality statistics are rare, many studies—reviewed by Finch (1990) and Caswell (2001)—demonstrate that fertility often increases with size (and hence age). For some reptiles death rates decline somewhat after age of reproductive maturity is reached, e.g., for *Sceloporus graciosus* (Tinkle et al., 1993), some populations of *Sceloporus undulatus* (Tinkle and Ballinger, 1972) and some populations of *Lacerta vivipara* (Heulin et al., 1997).

## 5. Models pertinent to the evolution of senescence

Evolutionary models of life-history characteristics fall into two types (Partridge and Barton, 1996). The usual kind of model is an optimization model. The forces of evolution are assumed to yield the best-possible design of a species' life history, the design that maximizes fitness. Hamilton's model of senescence provides an example of the second class of models, models in which evolutionary forces act in a non-adaptive way. Charlesworth (1994) provides further discussion of mutation-selection balance, i.e., of models of the opposing forces of (deleterious) mutation and subsequent Darwinian selection. Although we plan to develop models that include both optimization and deleterious mutations, the remainder of this article focuses on simple optimization models of senescence.

Williams (1957) proposed an optimization model of senescence, the so-called antagonistic-pleiotropy model. The basic idea is that some genes have a favorable or unfavorable effect on fertility or survival at younger ages but the opposite effect on mortality at older ages. A small positive (or negative) effect at younger ages may be more important than a large opposite effect at older ages if few individuals survive to these ages and if their reproduction is low. Williams' model is often formulated in terms of mutations that have a positive effect at some particular age (or age range) and a negative effect at some other particular age (Charlesworth, 1994). This formulation creates parallels with Hamilton's model. Williams' idea, however, is more general. It is simply an example of the kind of thinking about trade-offs that

underlies all optimization modeling. Williams clearly thought that his model implied senescence and he did not consider the logical possibility in such an optimization model of negative senescence. The “disposable soma” model (Kirkwood, 1987; Kirkwood and Holliday, 1986; Kirkwood and Rose, 1991) is a related example of this kind of thinking applied to senescence.

Optimization models of senescence can also be formulated in terms of specific parameters that affect the age-trajectories of fertility and mortality. This is the usual strategy in life-history analysis (Roff, 2002; Stearns, 1992). It has been applied to senescence by various researchers, including Gadgil and Bossert (1970), Charlesworth and Leon (1976) and Cichon and Kozłowski (2000). These researchers assume, as Cichon and Kozłowski (2000) put it, that “aging is a general feature of higher organisms.” They constrain their models such that the models imply senescence.

Fisher (1930) pioneered research on allocation over the life cycle. He focused on remaining age-specific reproductive value. This value is a natural measure of the potential of an organism to produce further offspring (Partridge and Barton, 1993) and it can be a useful quantity in backward optimization algorithms. As Hamilton (1966) correctly argued, however, it is an inappropriate measure of the force of selection against age-specific mutations. In many life-history optimization models, the optimal age-trajectories of fertility and mortality are the trajectories that maximize Lotka's intrinsic rate,  $r$ , of population growth. In other models, the population is assumed to be in optimal equilibrium, with no population growth or decline. In such a stationary population, optimal trajectories can be found by maximizing the net reproduction rate at age zero,  $R$ , which gives the expected number of offspring per individual and which can be calculated as the integral over the life course of the survival function,  $l(a)$ , and the fertility (or maternity) function,  $m(a)$ :

$$R = \int_0^{\infty} l(a)m(a) da. \quad (1)$$

Taylor et al. (1974) showed that maximizing reproductive value at age zero, for any arbitrary value of  $r$ , in particular  $r = 0$ , leads to an optimal life history. If  $r = 0$ , then reproductive value is given by  $R$ . We optimize  $R$  rather than  $r$  in this article.

In developing our models, we learned much from models of growth and mortality developed by Mangel, particularly Mangel and Abrahams (2001) and Mangel and Stamps (2001). The tradeoff we consider between reproduction and senescence is reminiscent of research on the evolution of iteroparity versus semelparity (e.g., Cole, 1954; Schaffer, 1974).

### 6. A simple optimization model on the frontier

Our purpose in this article is to make a persuasive case that negative senescence is theoretically possible and may be widespread among many plant species and some animal species. The literature suggests to us that negative senescence may be particularly common among species for which mortality depends on size. More specifically, we hypothesize that negative senescence may be frequent among such species when growth is indeterminate and when some adults reach sizes that are much larger than size at reproductive maturity. Hence, we have developed a simple model that highlights the role of size. The model is illustrative and is intended to help make the case that negative senescence is theoretically possible.

We consider a species in optimal equilibrium. That is, the species has perfected its age-trajectories of fertility, mortality, and growth to maximize fitness. All individuals in the species follow identical trajectories. The environment is unchanging. The population size is constant. Such a steady-state best-possible world is, of course, highly unrealistic, but the drastic simplification permits insight. Focusing on the optimal equilibrium has proven to be a useful strategy in life–history analysis, as exemplified by Lee (2003). We plunge *in medias res* and consider the species in the middle of an individual’s life at some specific age after reproduction has started. We assume that the reproductive capacity of an individual at this age as well as the individual’s ability to gather resources and to avoid death can all be captured by some measure that we denote by  $\xi$ . We refer to  $\xi$  as “size”, but please bear in mind that  $\xi$  is a complicated measure that is associated not only with physical size but also with strength and vitality. We assume that the resources available to the individual depend on the individual’s  $\xi$ . Some fraction,  $\pi$ , of these resources are devoted to growth and maintenance; the remaining  $(1 - \pi)$  of the resources are devoted to reproduction. The reproductive output (e.g., number of progeny) of the individual is given by  $(1 - \pi)\xi$ . Hence, in this simple and quite general model,  $\xi$  provides a direct measure of reproductive capacity.

We assume that the individual’s  $\xi$  can be maintained if but only if  $\pi$  is equal to  $\delta$ . If  $\pi$  is greater than  $\delta$ , then  $\xi$  increases. If  $\pi$  is less than  $\delta$ , then  $\xi$  decreases. Hence,  $\delta$  is the parameter that determines the proportion of resources that have to be devoted to maintenance to assure steady-state  $\xi$ . Like  $\xi$  and other variables in this model,  $\delta$  can be a function of age and size. We simply focus on the situation at a particular age and size.

If an individual is of size  $\xi$ , then the individual suffers a hazard of death  $\mu$ . We assume that  $\mu$  decreases as  $\xi$  increases, so  $\xi$  is a pleiotropic variable. The bigger  $\xi$  is, the more resources the individual garners each time period, the greater is its reproductive capacity, and the

lower is its mortality. The formula for reproductive capacity is very simple:  $\xi$  is measured in such a way that it equals reproductive capacity. The formulas for resource acquisition and mortality can be complicated. Furthermore  $\xi$  can be an intricate function of ordinary measures of size, such as weight, length, number of cells, or number of modular units.

In “maintenance mode”, with  $\pi = \delta$ ,  $\xi$  stays the same and  $\mu$  therefore also remains constant. Hence, the remaining reproduction of the individual, i.e., the expected number of progeny the individual will produce over the rest of its life, is given by

$$R^o = \int_0^\infty (1 - \delta)\xi e^{-\mu a} da = \frac{(1 - \delta)\xi}{\mu} = (1 - \delta)\xi e^o, \quad (2)$$

where  $e^o = 1/\mu$  denotes life expectancy. Note that the starting point 0 in the integral denotes the individual’s current age.

Suppose the individual invests a small fraction  $\gamma$  more than  $\pi$  in growth and maintenance for a short period of time  $\varepsilon$  and then goes into maintenance mode. The individual’s remaining reproduction will be given by

$$\begin{aligned} R^* &= \int_0^\varepsilon (1 - \delta - \gamma)\xi e^{-\mu a} da \\ &\quad + e^{-\mu\varepsilon} \int_\varepsilon^\infty (1 - \delta)\xi^* e^{-\mu^*(a-\varepsilon)} da \\ &= \frac{(1 - \delta - \gamma)\xi}{\mu} [1 - e^{-\mu\varepsilon}] + e^{-\mu\varepsilon} \frac{(1 - \delta)\xi^*}{\mu^*}. \end{aligned} \quad (3)$$

This formula can be understood as follows. If the individual invests  $\gamma$  extra in growth, then the individual will grow a little. Its future reproductive capacity will increase a bit to  $\xi^*$ . Because survival is assumed to rise with size, its mortality will be somewhat reduced to  $\mu^*$ . However, its reproductive output over the period from now to  $\varepsilon$  will be slightly lower. Note that we assume that during the growth interval of length  $\varepsilon$ , size is constant at the level  $\xi$  and jumps to its new value  $\xi^*$  only at the end of the interval.

Let  $\sigma$  denote the proportion of reproduction  $R^o$  that is sacrificed during this interval. Note that, by approximating  $1 - e^{-\mu\varepsilon} \approx \mu\varepsilon$  for small  $\varepsilon$ ,

$$\sigma = \frac{\gamma\xi\varepsilon}{R^o} = \frac{\gamma\varepsilon}{(1 - \delta)e^o}. \quad (4)$$

If  $R^* > R^o$ , then the individual will experience negative senescence, over the period from now to  $\varepsilon$ . On the other hand, if  $R^* < R^o$  then it would be optimal for the individual to invest a bit less in maintenance, leading to positive senescence from now to  $\varepsilon$ . When  $R^* = R^o$ , the individual is on the frontier between negative and positive senescence. The time to  $\varepsilon$  is very short, but if negative or positive senescence is optimal for this period, a similar strategy might be optimal for some period longer than  $\varepsilon$ . Hence, the frontier where  $R^* = R^o$  is of considerable interest.

This frontier can be described in two ways. Equating (2) and (3) and rearranging terms leads to

$$\frac{\xi^*}{\xi} \frac{\mu}{\mu^*} = 1 + \frac{\gamma}{1-\delta} [e^{\mu\varepsilon} - 1]$$

or, again by using  $e^{\mu\varepsilon} \approx 1 + \mu\varepsilon$  and substituting life expectancies for  $1/\mu$  and  $1/\mu^*$ , respectively,

$$\frac{\xi^* e^* - \xi e^0}{\xi e^0} = \frac{\gamma\varepsilon}{(1-\delta)e^0} = \sigma. \tag{5}$$

Dividing by  $\sigma$  leads to

$$\frac{\xi^* e^* - \xi e^0}{\xi e^0 \sigma} = 1. \tag{5a}$$

Eq. (5) simply states that if the relative gain in remaining reproduction after  $\varepsilon$  balances the fraction of sacrificed reproduction during the growth interval, then the individual is on the frontier between negative and positive senescence. If the relative gain in remaining reproductive value is larger than the loss  $\sigma$ , then negative senescence is favored.

If  $d\xi = \xi^* - \xi$  and  $de^0 = e^* - e^0$ , then (5a), dropping the very small  $d\xi de^0$  term, leads to

$$\frac{d\xi}{\xi} + \frac{de^0}{e^0} = 1 \tag{6}$$

which—by extracting a common factor—can also be written as

$$\frac{d\xi}{\xi} \left( 1 + \frac{de^0}{d\xi} \right) = 1. \tag{6a}$$

Let  $\dot{\xi}_\xi$  denote the elasticity of  $e^0$  with respect to  $\xi$ , that is

$$\dot{\xi}_\xi = \frac{de^0}{d\xi} \frac{\xi}{e^0}$$

This elasticity quantifies the percentage increase in  $e^0$  given a 1% increase in size, and thus denotes the responsiveness of life expectancy to changes in size. The leading factor in (6a),  $d\xi/\xi/\sigma$ , similarly is a “quasi-elasticity” in that it relates the relative change in  $\xi$  to another relative change, namely the change in the reproductive output over the small interval from 0 to  $\varepsilon$  relative to the total reproductive output  $R^0$ . If we use the same notation for this quasi-elasticity with respect to  $\sigma$  we can describe the maintenance frontier in (6) as

$$\dot{\xi}_\sigma + \dot{\xi}_\xi = 1$$

or from Eq. (6a) as

$$\dot{\xi}_\sigma \cdot (1 + \dot{\xi}_\xi) = 1. \tag{7}$$

Negative senescence prevails if the product exceeds one. This implies that negative senescence will tend to be favored if a small sacrifice of reproduction leads to a large increase in size. This effect will be reinforced to the extent that an increase in size leads to an increase in remaining life expectancy. Fig. 2 shows this relationship.

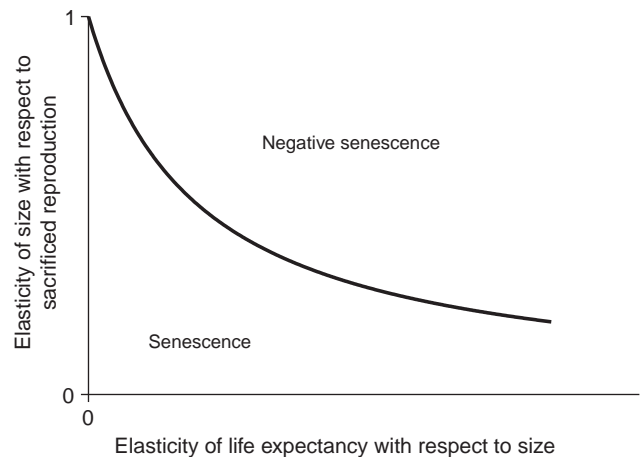


Fig. 2. Frontier between negative senescence and senescence as given in Eq. (7).

### 7. An optimization model that leads to negligible or negative senescence

#### 7.1. When senescence is impossible

The frontier model discussed above pertains to some unspecified age  $a$ . Consider now a more general life-course model. There is a single state,  $\xi(a)$ , which describes the size and vitality of an individual at age  $a$ . At each age, some proportion  $\pi(a)$  of the individual’s available resources is invested in growth and maintenance. Remaining resources are invested in fertility. Growth occurs if but only if  $\pi(a)$  exceeds some index of deterioration  $\delta(a)$ . If  $\pi(a) > \delta(a)$ , then  $\xi$  increases. If  $\pi(a) = \delta(a)$ , then  $\xi$  remains unchanged. And if  $\pi(a) < \delta(a)$ , then  $\xi$  decreases. An individual starts life by growing as rapidly as possible, so  $\pi(0) = 1$ . The optimal strategy of allocations  $\pi(a)$  over the life-course is the strategy that maximizes lifetime reproduction as given by Eq. (1). By using the following theorem (Baudisch, 2004b), it is possible to determine the general nature of the optimal strategy.

**The size ratchet.** Consider an optimization problem that is solely determined by a single state that changes continuously over age (or time or some other monotonically and continuously changing variable). If an optimal solution exists and each state is associated with exactly one optimal strategy, then the state trajectory must be a monotonic function over age. Once the organism chooses to maintain a state for any finite interval, it will maintain this state forever.

**Proof.** Let  $\pi^*(a)$  denote the optimal strategy at age  $a$  associated with state  $\xi(a)$ . Assume this strategy implies an increase in  $\xi(a)$  to  $\xi(a^+) = \xi(a) + \varepsilon$ ,  $\varepsilon > 0$ . If at the higher age  $a^+$  the optimal strategy  $\pi^*(a^+)$  would lead to

a decrease in  $\zeta$ , then  $\xi$  would shrink continuously. Clearly, when it reaches its former state  $\zeta(a^{++}) = \zeta(a)$  at some higher age  $a^{++}$ ,  $\pi^*(a^{++})$  is known to be such that  $\zeta$  increases again since  $\pi$  is solely determined by state and not age. But the continuity in state is even stronger. To reach the higher state  $\zeta(a) + \varepsilon$  it must have been optimal to grow at all intermediate states between  $\zeta(a)$  and  $\zeta(a) + \varepsilon$  so shrinkage would violate the optimal strategy at  $\zeta(a) + \varepsilon - \iota$  for any  $0 < \iota < \varepsilon$  and  $\iota \rightarrow 0$  and for any  $\varepsilon > 0$  and  $\varepsilon \rightarrow 0$ . Consequently, if the optimal strategy at starting age zero implies that  $d\zeta(a)/da > 0$  at  $a = 0$ , then  $d\zeta(a)/da \geq 0$  for any  $a > 0$ . Similarly, if the optimal strategy at starting age zero implies that  $d\zeta(a)/da < 0$  at  $a = 0$ , then  $d\zeta(a)/da \leq 0$  for any  $a > 0$ . Finally, if the optimal strategy at starting age zero implies that  $d\zeta(a)/da = 0$  at  $a = 0$ , then  $d\zeta(a)/da = 0$  for any  $a > 0$ . More generally, if  $d\zeta(a)/da = 0$ , at any age  $\hat{a}$  then  $d\zeta(a)/da = 0$  for all  $a > \hat{a}$ .  $\square$

The size ratchet is a very general result that may, perhaps, have already been published in some other context. When it is applied to the life-course model described above, it implies that an organism that starts life by growing must grow monotonically all its life. That is,  $\pi(a)$  must be greater or equal to  $\delta(a)$  at all ages. Furthermore, if  $\pi(a) = \delta(a)$  at some age, then this equality must be maintained at all subsequent ages: size remains constant. Suppose that  $dm(a)/d\zeta(a) > 0$  and  $d\mu(a)/d\zeta(a) \leq 0$ , so that senescence at some age occurs if and only if  $\zeta(a)$  declines at this age. Then the size ratchet implies that senescence is impossible.

## 7.2. A more specific model

To illustrate our finding that senescence is impossible in a single-state model, we develop in the following paragraphs a more specific model. Age-specific size, which stands for the general notion of strength and vitality, is denoted by  $\zeta(a)$  and size at age zero is set equal to one. The pace of growth of an organism is determined by the level of deterioration it is subject to and by the effort it spends on maintenance and growth. At any age  $a$  this effort is defined as the fraction,  $\pi(a)$ ,  $0 \leq \pi(a) \leq 1$ , of available resources the organism devotes to growth and maintenance, whereas the remaining fraction  $(1 - \pi(a))$  is invested in reproduction. All individuals start off with a period of development during which all available resources are invested in growth, i.e.  $\pi(a) = 1$  for all  $a \in [0, \alpha)$  where  $\alpha$  is the age of first reproduction. Age  $\alpha$  marks the point at which the investment strategy  $\pi(a)$  falls below 1 for the first time.

We assume that the level of deterioration  $\delta(a)$  at age  $a$  depends on current size because larger size implies higher complexity, which is more costly to maintain. To indicate that  $\delta$  depends on age only indirectly via  $\zeta(a)$  we write  $\delta(\zeta)$  unless we want to stress the age-trajectory

of  $\delta$  explicitly. One simple case is to assume that the level of deterioration depends linearly on size,

$$\delta(\zeta) = \delta_0 + \delta_1 \zeta(a), \quad (8)$$

where  $\delta_0 > 0$  and  $\delta_1 > 0$ .

and hence

$$\delta_0 + \delta_1 < 1. \tag{12}$$

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

We assume that fertility and mortality depend on age only indirectly via the age-specific vitality function  $\xi(a)$ . Fecundity  $m(a)$  is assumed to be directly proportional to  $\xi$  and to the reproductive effort  $(1 - \pi(a))$ :

$$m(a) = \varphi(1 - \pi(a))\xi(a).$$

Because

$$R = \int_0^\infty l(a)m(a)da = \varphi \int_0^\infty l(a)(1 - \pi(a))\xi(a) da, \tag{13}$$

the constant, positive parameter  $\varphi$  can be chosen to ensure that the optimal strategy yields  $R = 1$ .

Let the age-specific force of mortality be given by

$$\mu(a) = \frac{b}{\xi(a)} + c, \tag{14}$$

where  $b \geq 0$  and  $c > 0$ . Note that  $c$  is the constant size-independent component of external mortality. The mortality function determines the probability of survival to age  $a$ , given by the survival function

$$l(a) = e^{-\int_0^a \mu(t) dt}. \tag{15}$$

### 7.3. Results

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

$$\pi(a) \in [\delta(a), 1]. \tag{16}$$

The size-trajectory  $\xi(a)$  is the result of the optimal investment strategy  $\pi(a)$  over age that maximizes lifetime reproduction  $R$  defined in (1) subject to the logistic growth equation (9). This maximization problem can be tackled by optimal control theory using Pontryagin’s Maximum Principle (Léonard and van Long, 1992; Pontryagin, 1962). The part of the associated Hamiltonian that contains the control variable  $\pi(a)$  is

$$\begin{aligned} &\lambda_0 \cdot l(a)m(a) + \lambda_1(a) \cdot d\xi/da \\ &= \lambda_0 \cdot \{l(a)\varphi[1 - \pi(a)]\xi(a)\} \\ &\quad + \lambda_1(a) \cdot \{k[\pi(a) - \delta_0 - \delta_1\xi(a)]\xi(a)\}. \end{aligned} \tag{17}$$

Note that (17) is linear in  $\pi(a)$ . The optimal investment  $\pi(a)$  has to maximize the Hamiltonian. For linear functions this is only possible at the boundaries of the feasible set (16), leading to a so called bang–bang solution. The upper limit  $\pi(a) = 1$  is associated with full growth and no reproduction. The lower limit  $\pi(a) = \delta(a)$  switches the organism to maintenance mode with constant, nonzero fertility and mortality.

In this bang–bang case the integral in (1) can be solved explicitly. The switching age, when  $\pi(a)$  drops to  $\delta(a)$ , is the age,  $\alpha$ , of onset of reproduction. It follows from (13) that

$$\begin{aligned} R &= l(\alpha)m(\alpha) \int_\alpha^\infty \exp\left\{-\int_\alpha^a \mu(t) dt\right\} da \\ &= l(\alpha) \frac{m(\alpha)}{\mu(\alpha)}, \end{aligned} \tag{18}$$

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

The age  $\alpha$  at which reproduction starts is determined by the value  $\xi_\alpha$  that maximizes  $R$  in (18). Using the fact that from age zero to  $\alpha$  there is a one-to-one correspondence between age  $a$  and size  $\xi$ , we can express (18) as a function of  $\xi_\alpha$ . Inverting the logistic growth function  $\xi = L(a)$  given in (11) leads to

$$a = L^{-1}(\xi) = \frac{1}{k(1 - \delta_0)} \ln\left(\frac{1 - \frac{\delta_1}{1 - \delta_0}}{\frac{1}{\xi} - \frac{\delta_1}{1 - \delta_0}}\right). \tag{19}$$

Thus, by substituting  $\alpha = L^{-1}(\xi_\alpha)$  in (18) we can express  $R = R(\xi_\alpha)$  as a function of size at reproductive maturity  $\xi_\alpha$ . The optimization problem now can be solved by setting the first derivative of  $R(\xi_\alpha)$  with respect to  $\xi_\alpha$  equal to zero, i.e.,

$$l_{\xi_\alpha} \frac{m}{\mu} + m_{\xi_\alpha} \frac{l}{\mu} - \mu_{\xi_\alpha} \frac{lm}{\mu^2} = 0. \tag{20}$$

Because

$$\begin{aligned} l_{\xi_\alpha} &= \frac{d}{d\xi_\alpha} l(\xi_\alpha) \\ &= \frac{d}{d\xi_\alpha} \exp\left\{-\int_1^{\xi_\alpha} \mu(\xi)[k(1 - \delta_0 - \delta_1\xi)\xi]^{-1} d\xi\right\} \\ &= -l(\xi_\alpha)\mu(\xi_\alpha)[k(1 - \delta_0 - \delta_1\xi_\alpha)\xi_\alpha]^{-1}, \end{aligned}$$

optimal size at maturity is given by

$$\frac{\mu(\xi_\alpha)}{k} = (1 - \delta_0 - 2\delta_1\xi_\alpha) + \frac{(1 - \delta_0 - \delta_1\xi_\alpha)b}{\mu(\xi_\alpha)\xi_\alpha}. \tag{21}$$

Substituting  $\mu(\xi) = b/\xi + c$ , yields a cubic polynomial with three roots, one of which is real and the other two complex. For viable strategies, however, the imaginary parts vanish. They can be determined numerically; we used MATHEMATICA™ to calculate the solution.

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

$$\xi_\alpha = \frac{(1 - \frac{c}{k} - \delta_0)}{2\delta_1}. \tag{22}$$

Results for three illustrative parameter combinations are shown in Fig. 4. Eq. (22) implies

$$\frac{d\xi_\alpha}{dc} < 0, \quad \frac{d\xi_\alpha}{d\delta_0} < 0, \quad \frac{d\xi_\alpha}{d\delta_1} < 0 \quad \text{and} \quad \frac{d\xi_\alpha}{dk} > 0. \tag{23}$$

Furthermore, (22) and (19) imply

$$\frac{d\alpha}{dc} < 0 \quad \text{and} \quad \frac{d\alpha}{d\delta_1} < 0. \quad (24)$$

Changes in  $\alpha$  with respect to  $k$  and  $\delta_0$  depend on the parameter combination in a rather complicated way. For very small maximum attainable sizes and very slow speed of growth,  $\alpha$  can increase with increasing  $k$  and decrease with increasing  $\delta_0$ .

Table 1  
Optimal size  $\xi_x$  and age  $\alpha$  at start of reproduction for size-dependent mortality ( $b > 0$ ) according to Eq. (21)

$\xi_x$	$\alpha$	$\xi_{max}$	$l(\alpha)$	$b$	$c$	$k$	$\delta_0$	$\delta_1$
62.26	50.96	100	0.005	0.5	0.001	1	0.9	0.001
53.46	47.34	100	$1.1 \times 10^{-9}$	2	0.001	1	0.9	0.001
60.02	50.02	100	0.00003	1	0.001	1	0.9	0.001
25.68	17.66	100	0.0012	1	0.1	2	0.9	0.001
56.86	24.36	100	0.0045	1	0.01	2	0.9	0.001
64.06	25.87	100	0.0056	1	0.000001	2	0.9	0.001
127.66	29.31	200	0.006	1	0.001	1	0.8	0.001
129.18	14.74	200	0.08	1	0.001	2	0.8	0.001

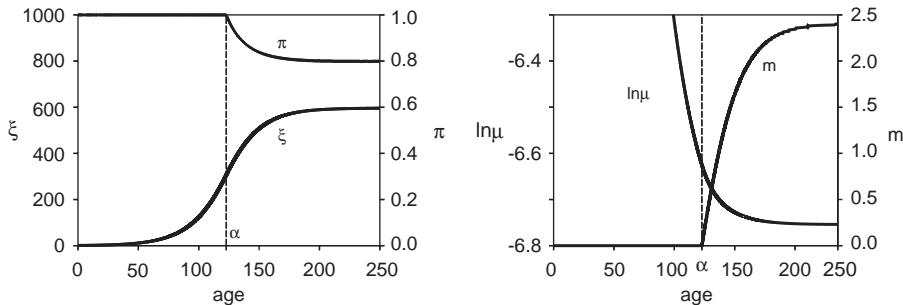


Fig. 5. Negative senescence for model variant (26). Parameters values were  $k = 0.1$ ,  $\delta_0 = 0.5$ ,  $\delta_1 = 0.0005$ ,  $b = 0.1$ ,  $c = 0.001$ ,  $\varphi = 0.02$ . The force of mortality before age 100 is very high and rapidly falling.

identical to age at reproductive maturity  $\alpha$ , although for many determinate growers the two coincide.

Our model can then be reformulated as follows. Fertility is given by

$$m(a) = \varphi(\pi(a) - \pi^2(a))\xi(a)\psi(a), \tag{27}$$

and mortality is given by

$$\mu(a) = \frac{b}{\xi(a)\psi(a)} + c. \tag{28}$$

Note that both now depend on the product of size and functioning,  $\xi(a)\psi(a)$ . We call this product “vitality”. Growth in  $\xi$  is positive until determinate size is attained and zero afterwards:

$$\frac{d\xi(a)}{da} = \begin{cases} k(\pi(a) - \delta_0 - \delta_1\xi(a)) & \text{if } \pi(a) > \delta_0 + \delta_1\xi(a), \\ 0 & \text{otherwise,} \end{cases} \tag{29}$$

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

$$\frac{d\psi(a)}{da} = \begin{cases} 0 & \text{if } a < a^*, \\ \kappa(\pi(a) - \delta_0 - \delta_1\xi(a^*)) & \text{if } a \geq a^*, \end{cases} \tag{30}$$

where  $\psi(0) = 1$ . The parameters  $k$  and  $\kappa$  determine the speed of increase in size and the speed of decline in vitality, respectively.

Fig. 6 shows the optimal trajectories of  $\pi(a)$ ,  $\xi(a) \cdot \psi(a)$ ,  $\mu(a)$  and  $m(a)$  for this model. The results were

obtained numerically. Maximum attainable size is  $\xi = 25$ ; this size is almost reached at age of maturity  $\alpha$ .

### 9. Discussion

Based on the theoretical and empirical evidence presented above, we hypothesize:

- Senescence characterizes individuals in species that attain a size at reproductive maturity that is close to maximum size. Such determinate-growth species include mammals, birds, insects and some other species including yeast and the nematode worm *C. elegans*. The main model species studied by gerontologists are mammals (including humans, rats and mice), insects (especially *Drosophila* but also *Medflies* and some other insect species), *C. elegans*, and yeast. All of these species fall into this determinate-growth category. Many determinate-growth species also have fixed oocyte stocks or are otherwise limited with regard to reproductive capacity. Species that experience declines in fertility with age or that have limited fertility seem likely to suffer senescence.
- Negligible senescence characterizes individuals in species that attain a size at reproductive maturity that is somewhat less—but not greatly less—than maximum size and that have undiminished

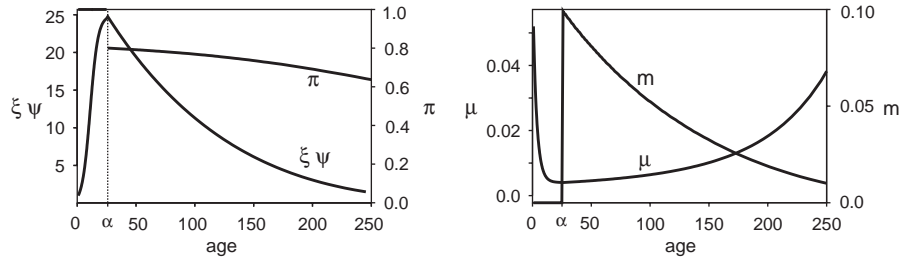


Fig. 6.  $\xi(a) \cdot \psi(a)$ , force of mortality  $\mu$  and fertility  $m$  resulting from optimal strategy  $\pi(a)$  as a function of age  $a$ , for model with parameters  $k = 3$ ,  $\delta_0 = 0.9$ ,  $\delta_1 = 0.004$ ,  $\kappa = 0.05$ ,  $b = 0.05$ ,  $c = 0.002$ ,  $\varphi = 0.02$ .

reproductive capacity. Species with modestly indeterminate growth and continuing oogenesis include many fish, reptiles and amphibians.

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

Negligible senescence is an intermediate and rather arbitrary category. We explicitly mention it because the important research of Finch and colleagues (Finch, 1990; Finch and Austad, 2001; Ottinger et al., 2003) suggests that a variety of species may be characterized by death rates that increase very slowly, if at all, with age.

We hypothesize that indeterminate growth may be the underlying cause of negative and negligible senescence. In our model of indeterminate growth, the state variable  $\xi$  plays a central role. We emphasize that size, as measured by weight, length, number of cells, number of modular units or some similar index, is only roughly related to  $\xi$ , which captures strength and vitality as well as size and which determines the capability of an individual to gather resources, to produce progeny and to avoid death.

To model the life-course of species with determinate growth, we had to introduce a second state variable,  $\psi$ . This variable can capture a decline in functioning of an individual whose size remains constant. We modelled the vitality of an individual as the product of  $\xi$  times  $\psi$ . Because of wear and tear and failure of repair, individuals may maintain about the same body weight, length or cell number over an extended period of life but suffer a decline in vitality. Furthermore, individuals in some species may grow in terms of ordinary measures of

size, with this growth sufficiently counterbalancing forces of deterioration and functional decline. In such species the ability to escape mortality, as captured by  $\xi$  times  $\psi$ , may remain roughly constant—resulting in negligible senescence. We did not develop this kind of model, but it is not difficult to do so.

Note the distinction between senescence, on the one hand, and deterioration and functional decline, on the other. We use senescence only with regard to entire organisms, not parts of organisms, and we stipulate that senescence is characterized by an increase in age-specific death rates. In our model we capture deterioration by  $\delta(a)$  and decline in functioning by a decrease in  $\psi(a)$ . A tendency for existing body parts to deteriorate and to require repair or replacement to maintain functioning may possibly be a “fundamental, universal, and intrinsic” property of living organisms (Arking et al., 1991); senescence, as we define it, is not.

In any case, this general (and speculative) line of thinking leads us to conjecture that biological age may be better captured by the “average age” of an individual—i.e., by some appropriate measure of the average age of the organs, body parts or cells of an individual—than by the chronological age of the individual. In indeterminate-growth species, continuing increases in size keep average age well below chronological age. Furthermore, organisms that can repair, replace or rejuvenate body parts may show, over chronological time, slow increases or even decreases in average age. For instance, trees that replace their leaves annually, that develop new roots and new branches to replace damaged or lost ones, and that continue to grow may be of an average individual age that remains roughly constant and may even decline with chronological age. For some species of plants and animals, there can be a complete turnover of body parts over a time interval: for these species, average individual age can be much lower than chronological age and can decline over time if the individual grows and its component parts continue to turnover with time.

Negative senescence may thus be especially characteristic of species for which (1) the average age of an

individual is steady or decreasing, (2) mortality declines with increasing size, and (3) fertility increases with increasing size. Negative senescence may be favored in species with strong repair or rejuvenative capabilities as exemplified by the following kind of traits: continuing oogenesis, an abundance of stem cells, no distinction between germ and soma cells, the ability to reproduce clonally from a severed part or from a root or offshoot. This last ability is common among plants and is often termed vegetative reproduction, but it is also found among animals, including platyhelminthes and annelida (Finch, 1990, Table 4.3, p. 231).

Finch (1990, pp. 206–247) discusses these and other relevant factors in a thoughtful review of species that may experience negligible senescence. He does not explicitly consider negative senescence but much of his review is pertinent to declining mortality with age and many of his candidate species for negligible senescence may enjoy negative senescence.

Among many other topics, Finch considers modularity in multicellular organisms. He argues that “it is useful to distinguish between organisms that possess internal repeated structure, which can continuously regenerate themselves internally as well as vegetatively by fragmentation (call these modular), and organisms that have a nonrepeating internal structure, which typically do not reproduce vegetatively but which also typically show senescence (call these unitary).” He notes that “older modules may degenerate” but that “such degeneration should not be considered an organismic senescence”. This is in keeping with his (and our) definition of senescence. To the extent that the organisms with a larger number of modules face a lower chance of death and to the extent that surviving modules, at any chronological age of the organism, are relatively young, then such species may be prime candidates for exhibiting negative senescence.

This article has made a case for negative senescence by presenting evidence that negative senescence is theoretically possible and may be widespread among plants and some kinds of animals. We hope we have made a good enough case to stimulate thinking and to justify further theoretical and empirical research on positive vs. negative senescence. Understanding why death rates increase with age for some species but may decrease with age for other species could lead to deeper comprehension of the evolutionary and demographic forces that mold the age–trajectory of mortality and the age-trajectories of fertility and growth as well.

## Acknowledgments

We thank Steven Orzack and Shripad Tuljapurkar for helpful comments. Our research was supported by the

Max Planck Society and US National Institute on Aging Grant P01-08761.

## References

- Aberg, P., 1992. A demographic study of two populations of the seaweed *Ascophyllum nodosum*. *Ecology* 73 (4), 1473–1487.
- Alvarez-Buylla, E., Martinez-Ramos, M., 1992. Demography and allometry of *Cecropia obtusifolia*, a neotropical pioneer tree—an evaluation of the climax pioneer paradigm for tropical rain forests. *J. Ecol.* 80 (2), 275–290.
- Arking, R., Buck, S., Berrios, A., Dwyer, S., Baker, G.T.I., 1991. Elevated paraquat resistance can be used as a bioassay for longevity in a genetically based long-lived strain of *Drosophila*. *Develop. Genet.* 12, 362–370.
- Babcock, R., 1991. Comparative demography of three species of scleractinian corals using age- and size-dependent classifications. *Ecol. Monograph* 61 (3), 225–244.
- Baudisch, A., 2004a. Hamilton’s indicators of the force of selection. Available at [www.demogr.mpg.de/papers/working/wp-2004-###.pdf](http://www.demogr.mpg.de/papers/working/wp-2004-###.pdf).
- Baudisch, A., 2004b. Monotonic state trajectories from single-state dynamic optimization models. Available at [www.demogr.mpg.de/papers/working/wp-2004-###.pdf](http://www.demogr.mpg.de/papers/working/wp-2004-###.pdf).
- Caswell, H., 2001. *Matrix Population Models: Construction, Analysis, and Interpretation*, 2nd Edition. Sinauer, Sunderland, MA.
- Charlesworth, B., 1994. *Evolution in Age-Structured Population*, 2nd Edition. Cambridge University Press, Cambridge.
- Charlesworth, B., Leon, J., 1976. The relation of reproductive effort to age. *Am. Nat.* 110, 449–459.
- Charlesworth, B., Partridge, L., 1997. Ageing: levelling of the grim reaper. *Curr. Biol.* 7, R440–R442.
- Cichon, M., Kozłowski, J., 2000. Ageing and typical survivorship curves result from optimal resource allocation. *Evol. Ecol. Res.* 2, 857–870.
- Cole, L.C., 1954. The population consequences of life history phenomena. *Q. Rev. Biol.* 29 (2), 103–137.
- Comfort, A., 1956. *The Biology of Senescence*. Routledge & Kegan Paul, London.
- Ebert, T.A., Southon, J.R., 2003. Red sea urchins (*Strongylocentrotus franciscanus*) can live over 100 years: confirmation with A-bomb <sup>14</sup>carbon. *Fish. Bull.* 101 (4), 915–922.
- Finch, C., 1990. *Longevity, Senescence, and the Genome*. University of Chicago Press, Chicago.
- Finch, C., 1998. Variations in senescence and longevity include the possibility of negligible senescence. *J. Gerontol. Biol. Sci.* 53A (4), B235–B239.
- Finch, C.E., Austad, S.N., 2001. History and prospects: symposium on organisms with slow aging. *Exp. Gerontol.* 36, 593–597.
- Finch, C.E., Pike, M., Witten, M., 1990. Slow increases of the Gompertz mortality rate during aging in certain animals approximate that of human. *Science* 249, 902–905.
- Fisher, R.A., 1930. *The Genetical Theory of Natural Selection*. Clarendon Press, Oxford.
- Gadgil, M., Bossert, W., 1970. Life historical consequences of natural selection. *Am. Nat.* 104, 1–24.
- Grigg, R., 1977. Population dynamics of two gorgonian corals. *Ecology* 58 (2), 278–290.
- Guedje, N., Lejoly, J., Nkongmeneck, B.A., Jonkers, W., 2003. Population dynamics of *Garcinia lucida* (clusiaceae) in cameroonian atlantic forests. *Forest Ecol. Manage.* 177 (1–3), 231–241.
- Hamilton, W., 1966. The moulding of senescence by natural selection. *J. Theor. Biol.* 12, 12–45.

- Hamilton, W.D., 1996. Narrow Roads of Gene Land: The Collected Papers of W.D. Hamilton, Vol. 1: Evolution of Social Behaviour. W.H. Freeman Spektrum, Oxford, New York, Heidelberg.
- Hegazy, A.K., 1992. Age-specific survival, mortality and reproduction, and prospects for conservation of *Limonium delicatulum*. J. Appl. Ecol. 29 (3), 549–557.
- Heulin, B., Osenege-Leconte, K., Michel, D., 1997. Demography of a bimodal reproductive species of lizard (*Lacerta vivipara*): survival and density characteristics of oviparous populations. Herpetologica 53 (4), 432–444.
- Hughes, R., Roberts, D., 1981. Comparative demography of *Littorina rudis*, *L. nigrolineata* and *L. neritoides* on three contrasted shores in north wales. J. Anim. Ecol. 50 (1), 251–268.
- Kemperman, J., Barnes, B., 1976. Clone size in American aspens. Cana. J. Bot. 54, 2603–2607.
- Kirkwood, T., 1987. Immortality of the germ-line versus disposability of the soma. In: Woodhead, A., Thompson, K. (Eds.), Evolution of Longevity in Animals. Plenum, New York.
- Kirkwood, T.B.L., Holliday, R., (Eds.) 1986. Selection for Optimal Accuracy and the Evolution of Ageing: Its Control and Relevance to Living Chapman & Hall, London, pp. 363–379 (Chapter 12).
- Kirkwood, T.B.L., Rose, M.R., 1991. Evolution of senescence: late survival sacrificed for reproduction. Philos. Trans. R. Soc. London 332, 15–24.
- Lee, R., 2003. Rethinking the evolutionary theory of aging: transfers, not births, shape senescence in social species. Proc. Nat. Acad. Sci. USA 100 (16), 9637–9642.
- Léonard, D., van Long, N., 1992. Optimal Control Theory and Static Optimization in Economics. Cambridge University Press, Cambridge.
- Lotka, A.J., 1924. Elements of Mathematical Biology. Dover Publications, Inc., New York (reprinted 1956).
- Mangel, M., Abrahams, M.V., 2001. Age and longevity in fish, with consideration of the ferox trout. Exp. Gerontol. 36 (4–6), 765–793.
- Mangel, M., Stamps, J., 2001. Trade-offs between growth and mortality and the maintenance of individual variation in growth. Evol. Ecol. Res. 3, 583–593.
- Martinez, D.E., 1998. Mortality patterns suggest lack of senescence in hydra. Exp. Gerontol. 33 (3), 217–225.
- Medawar, P.B., 1952. Uniqueness of the Individual. An unsolved problem of biology. H.K. Lewis, London, pp. 44–70.
- Nakaoka, M., 1994. Size-dependent reproductive traits of *Yoldia notabilis* (bivalvia, protobranchia). Mar. Ecol. Progr. Ser. 114 (1–2), 129–137.
- Nakaoka, M., 1996. Size-dependent survivorship of the bivalve *Yoldia notabilis* (Yokoyama, 1920): the effect of crab predation. J. Shellfish Res. 15 (2), 355–362.
- Nault, A., Gagnon, D., 1993. Ramet demography of *Allium tricoccum*, a spring ephemeral, perennial forest herb. J. Ecol. 81 (1), 101–119.
- Noda, T., 1991. Shell growth of the sand snail *Umbonium costatum* (Kiener) in hakodate bay. Bull. Fac. Fish. Hokkaido Univ. 42 (4), 115–125.
- Noda, T., Nakao, S., Goshima, S., 1995. Life history of the temperate subtidal gastropod *Umbonium Costatum*. Mar. Biol. 122 (1), 73–78.
- Ottinger, M.A., Ricklefs, R.E., Finch, C.E., (Eds.), 2003. Second Symposium on Organisms with Slow Aging (SOSA-2); of Exp. Gerontol. (Special Issue) 38(7).
- Partridge, L., 1997. Evolutionary biology and age-related mortality. In: Wachter, K.W., Finch, C.E. (Eds.), Between Zeus and the Salmon. National Academy Press, Washington, DC, pp. 78–95.
- Partridge, L., Barton, N.H., 1993. Optimality, mutation and the evolution of ageing. Nature 362, 305–311.
- Partridge, L., Barton, N.H., 1996. On measuring the rate of ageing. Proc. R. Soc. London B 263, 1365–1371.
- Pontryagin, L.S., 1962. The Mathematical Theory of Optimal Processes. Wiley Interscience, New York.
- Roach, D.A., Gampe, J., 2004. Age-specific demography in *Plantago*: uncovering age-dependent mortality in a natural population. Naturalist (in press).
- Roff, D.A., 2002. Life History Evolution. Sinauer Associates, Sunderland, MA.
- Schaffer, W.M., 1974. Selection for optimal life histories: the effects of age structure. Ecology 55 (2), 291–303.
- Stearns, S.C., 1992. The Evolution of Life Histories. Oxford University Press, Oxford.
- Strehler, B.L., 1977. Time, Cells and Aging. Academic Press, New York.
- Taylor, H.M., Gourley, R.S., Lawrence, C.E., Kaplan, R.S., 1974. Natural selection of life history attributes: an analytical approach. Theor. Popul. Biol. 5, 104–122.
- Tinkle, D., Ballinger, R.E., 1972. *Sceloporus undulatus*: study of the intraspecific comparative demography of a lizard. Ecology 53 (4), 570–584.
- Tinkle, D., Dunham, A., Congdon, J., 1993. Life history and demographic variation in the lizard *Sceloporus graciosus*: a long-term study. Ecology 74 (8), 2413–2429.
- Tuljapurkar, S., 1997. The evolution of senescence. In: Wachter, K.W., Finch, C.E. (Eds.), Between Zeus and the Salmon. National Academy Press, Washington, DC, pp. 65–77.
- Williams, G.C., 1957. Pleiotropy, natural selection, and the evolution of senescence. Evolution 11 (4), 398–411.