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Kin Selection Aging Theory

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Synonyms

Selective advantage of aging in populations spatially structured and in conditions of K-selection

Definition

Kin selection aging theory is an evolutionary interpretation of aging explained as an adaptive phenomenon favored by supra-individual selection in populations spatially structured and in conditions of K-selection.

Overview

Kin selection aging theory belongs to the group of hypotheses that, in the search for the causes of aging, tries to take into account the mechanisms of evolution. It also belongs to the subset of these theories that consider aging as something favored by natural selection, because it involves advantages in terms of supra-individual selection, and not as a phenomenon caused by insufficient selection against harmful effects that accumulate with age.

This theory has some precursors. Alfred Russel Wallace proposed that aging was favored by natural selection because predeceasing individuals do not compete with their offspring (Wallace 1865–1870; Skulachev and Longo 2005). Some years later, Weissmann suggested, without a detailed exposition or scientific proofs, that natural selection favored aging because the death of older individuals frees space for the next generation and this accelerates the evolution of the species (Weismann 1889). However, later he repudiated this hypothesis (Weismann 1892; Kirkwood and Cremer 1982). In 1961, Aldo Carl Leopold, a botanist, again proposed that aging accelerates the generation turnover and therefore favors evolution: "... in plants senescence is a catalyst for evolutionary adaptability" (Leopold 1961, p. 1729).

However, the kin selection aging theory was proposed, without using this naming, first in a non-peer-reviewed book (Libertini 1983) and then in a regular scientific article (Libertini 1988) and afterward deepened in other works (Libertini 2006, 2008, 2009, 2013).

The logical thread of the original formulation of the theory is now briefly expounded.

Kin Selection as Explanation for Aging

Already in the 1980s and before, it was known that many species show an "increasing mortality

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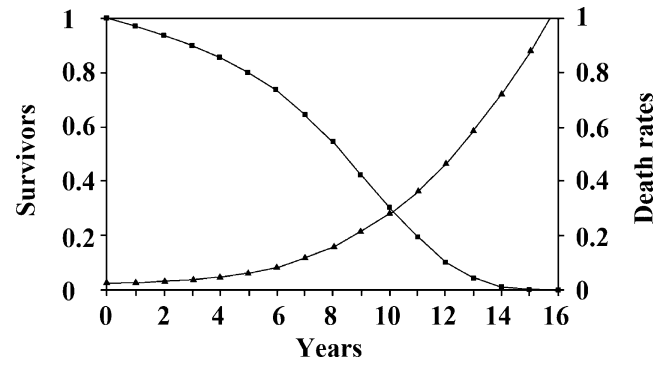


Kin Selection Aging Theory, Fig. 1 Life table and mortality of *Panthera leo* (lion) in the wild. (Data from (Ricklefs 1998))

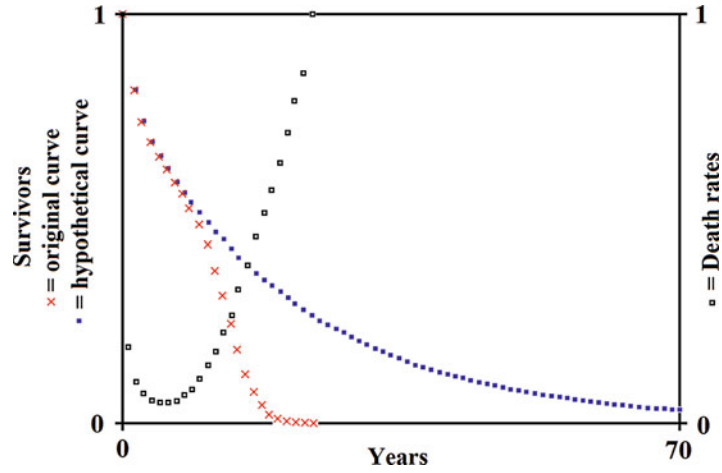
with increasing chronological age in the wild," or shortly IMICAW (Libertini 1988, p. 145), as documented in various studies (Deevey 1947; Laws 1966, 1968; Spinage 1970, 1972). Subsequently, the existence of this phenomenon, although denied or diminished in some works (Kirkwood and Austad 2000; Kirkwood 2005; Kirkwood and Melov 2011), has been authoritatively confirmed in other studies (Ricklefs 1998; Nussey et al. 2013). In particular, the review by Nussey et al. highlighted that the IMICAW phenomenon is reported for 175 animal species in 340 works (Nussey et al. 2013). An example of this phenomenon is shown in Fig. 1.

However, it is necessary to explain how it is possible that the existence of a phenomenon so widely documented in the wild is completely denied in authoritative articles. For example, "data on age-related mortality patterns in wild animal populations reveal that, in many species, individuals rarely survive to ages when senescent deterioration becomes apparent (Medawar 1952; Lack 1954; Finch 1990). For most natural populations, extrinsic mortality (due to accidents, predation, starvation, disease, cold, etc.) is such that death occurs well before 'old age'. This means that (a) there is no requirement for aging to weed out 'worn-out individuals'; b) there is no evidence that aging in fact serves as a significant mortality force in the wild; and (c) there can have been scant opportunity to evolve genes specifically for aging, even if they were beneficial, since natural selection would not normally 'see' them in action" (Kirkwood 2005, p. 438).

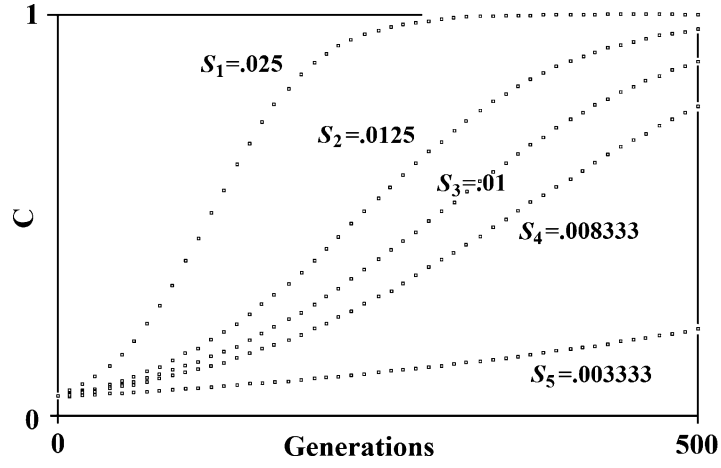
The negation arises from a misunderstanding, namely, the identification of the phenomenon aging with the "worn-out individuals" (Kirkwood 2005, p. 438), while aging is manifested by the progressive age-related increase in mortality, as well expressed already many years ago: "No one would consider a man in his thirties senile, yet, according to athletic records and life tables, senescence is rampant during this decade" (Williams 1957, p. 399). It is therefore correct to say that a centenary, or its equivalent depending on the species, does not exist in the wild, while it is completely contradicted by empirical data to deny the existence in the wild of an age-related increase in mortality for many species.

Simple calculations showed that a smaller rate of mortality increase, or even the absence of age-related increasing mortality, strongly influenced the "mean duration of life" (*ML*). An example is shown in Fig. 2, where it is noted that for a species under natural conditions, the hypothetical absence of age-related increasing mortality would cause approximately a doubling of ML. Other examples were shown in Fig. 5 and in Table 1 of the original work.

The existence of IMICAW phenomenon, i.e., what is commonly called aging, and its relevance for the shortening of the *ML* determined by it, obliged to explain why natural selection was not able to counteract the progressive increase in mortality. Only two alternatives were possible: (i) the phenomenon involved some advantages in terms of natural selection (adaptive hypothesis); (ii) the phenomenon was determined by degenerative effects that natural selection was unable to contrast (nonadaptive hypothesis).



Kin Selection Aging Theory, Fig. 2 Hypothetical survival curve of zebra compared with the real-life table. Abscissas from 0 to 70 years. Calculation for the hypothetical survival curve done from 0 to 350 years. Min. mortal.: at 6 years = 4.638216%; *ML* = 8.480926 years; hypothetical ML (*HML*) = 17.23965 years; ratio *HML/ML* = 2.032755 (Fig. 4 in (Libertini 1988), redrawn)



Kin Selection Aging Theory, Fig. 3 Spreading of a gene (C) according to the variation of *S*. The values of *S* (arbitrarily chosen) are indicated near each curve. Moreover, $C_o = 0.05$. The rate of generation turnover is considered equal in all the curves, i.e., the mean duration of life (*ML*) is assumed to be constant

In pursuing the first alternative, two theoretical arguments – which deepened the insights of the aforementioned precursors – were highlighted.

The first is that the rate of diffusion of a favorable gene (C) within a species and against an inactive allele (C′) is a function of the advantage (*S*) deriving from the actions of the gene C. The diffusion of the gene is easily calculable by applying the iterative formula:

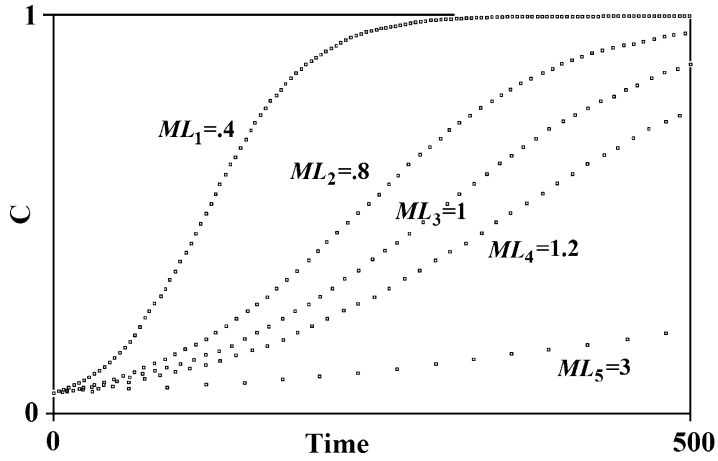
$$C_{n+1} = \frac{C_n \cdot (1+S)}{C_n \cdot (1+S) + C'_n} = \frac{C_n \cdot (1+S)}{1 + C_n \cdot S} \quad (1)$$

where C_n = frequency of C at the *n*th generation and the denominator has the function of keeping constant the sum of the frequencies: $C_n + C'_n = 1$.

An example of the application of this formula is given in Fig. 3.

The second is that a reduction of the *ML*, i.e., a quicker generation turnover, has the same effect as a proportional increase in the value of *S*. This is illustrated in Fig. 4, where the same formula of the previous figure is used and the value of *S* is constant for all five curves ($S = 0.01$), while *ML* values, indicated in the image, are given by the formula $ML_x = K/S_x$ (where the S_x values are the same of the previous figure).

The curves are morphologically identical to those of Fig. 3, and this shows that an increase in the value of *S* or a proportional identical reduction of *ML* has the same effect with respect to the diffusion of a favorable gene. The rigorous mathematical demonstration of this effect is expounded



Kin Selection Aging Theory, Fig. 4 Spreading of a gene according to the variation of *ML* (while $S = K$). The values of *ML* are indicated near each curve. The formula used is the same as shown in Fig. 3, but the abscissas indicate time and not the generations

in the appendix of the original work (Libertini 1988). Similarly, it is possible to demonstrate that the elimination of a harmful gene (i.e., with $S < 0$) is accelerated by a quicker generation turnover (i.e., by a shorter *ML*).

This showed that, in the comparison among species, the one with a lower *ML*, i.e., with a quicker generation turnover, had a greater rapidity of evolution. Examples of how the rate of generation turnover is extremely important for the speed of evolutionary adaptation of a species to new conditions are well known (bacteria, parasitic insects, weeds that become resistant in a few years to antibiotics, insecticides, and herbicides, respectively).

However, this did not prove at all in what way a hypothetical gene that had the ability to reduce the *ML* could be favored by natural selection against an inactive allele within a species.

As a solution to this problem, which seemed to have no positive response, the application of the concept of "inclusive fitness" (Hamilton 1964, 1970; Trivers 1971; Wilson 1975) was proposed. To calculate if a gene G is favored or opposed by natural selection, it is necessary to consider the consequences of the gene action both on the individual in which it is present (I_1) and on the individuals genetically related to it (I_2, I_3, … I_n) that have a probability of having the same G gene equal to the coefficient of kinship (r_x) between I_x and I_1:

$$\Delta_G \propto \sum_{x=1}^{n} (S_x \cdot P_x \cdot r_x) \qquad (2)$$

where Δ_G = difference of the frequency of the gene G between two subsequent generations; n = number of individuals for which the character has some effect; S_x = advantage/disadvantage for I_x; P_x = reproductive value of I_x; and r_x = coefficient of relationship between I_x and I_1.

In the particular case in which G has no effect on other individuals besides I_1, formula (2) becomes:

$$\Delta_G \propto S_1 \cdot P_1 \qquad (3)$$

that is the case of selection at the individual level and in which the diffusion of a gene is influenced only by the advantage of the gene (and by the reproductive capacity of the individual at the moment when the action of the gene occurs).

In the case of a gene C that reduces lifespan, we have two conflicting actions:

- The first one, which is negative and contrasts the diffusion of the C gene, is due to the disadvantages (S') caused by a shorter lifespan.
- The second, determined by kin selection, which is positive and promotes the diffusion of the C gene, can be effective only if the species is divided into small groups and demographically stable (K-selection according to the definition of Pianka (1970), which is still valid (Engen and Saether 2017)). In such conditions an individual who dies prematurely by action of the gene C frees space for another individual that on average has a coefficient of

kinship equal to r with the predeceased individual. Therefore for an X gene that is in the process of diffusion within the species, the advantage S_x is multiplied for a factor equal to $r\,S_x\,(1/ML_C - 1)$, where ML_C is the average life span of individuals with the gene C, with $ML_C < 1$, while the ML of individuals with the C′ ($ML_{C'}$) allele is considered equal to 1.

Therefore, according to the theory, a gene C that determines a shorter *ML* is favored when:

$$\sum_{x=1}^{m} [r\,S_x\,(1/ML_C - 1)] > S' \qquad (4)$$

where S_x = advantage of an x allele that is spreading within the species; m = number of alleles that are spreading within the species; r = mean relationship coefficient between the individual that dies by action of C and the substituting individual; and S' = advantage of the greater *ML* of the individuals with the neutral allele C′.

If the m alleles that are spreading within the species have a mean advantage of S_m, the formula (4) becomes:

$$m\,r\,S_m\,(1/ML_C - 1) > S', \qquad (5)$$

which has been used for Fig. 5.

It is necessary to highlight that formulas (4) and (5) are equivalent to the formula used in the original work (Libertini 1988, formula 10):

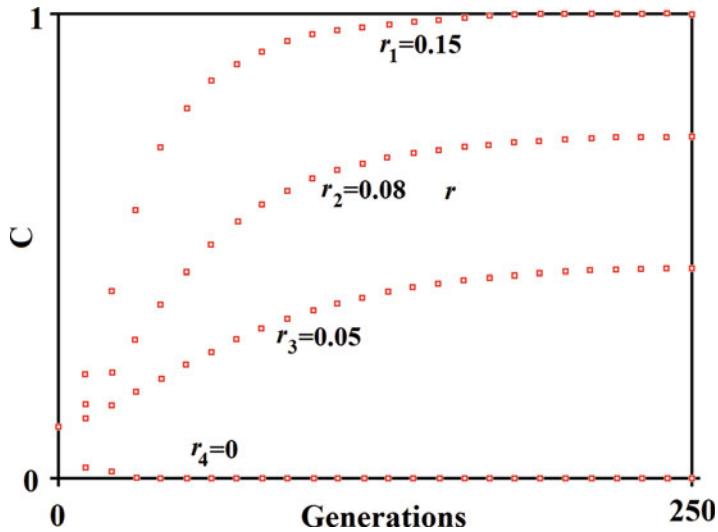
$$\mathrm{r}S(1/\mathrm{V_C} - 1) > S', \quad \text{that is, } rS(1/ML_C - 1) > S',$$

where it was "hypothesized $S \gg S'$, since S sums up the advantages of the m genes that are spreading within the species" (Libertini 1988, p. 155). In a work that disregarded this essential observation, the model was wrongly considered as unlikely (Kowald and Kirkwood 2016).

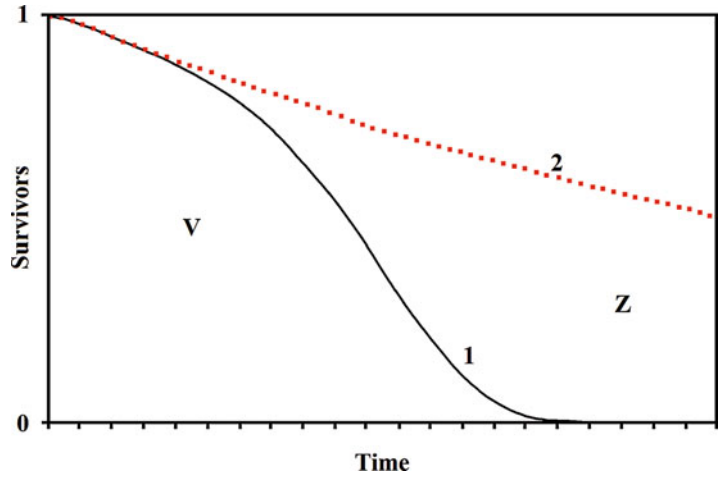
An analogous idea of the advantage of aging in spatially structured populations was later proposed using complex models (Travis 2004; Martins 2011; Mitteldorf and Martins 2014). However, the first model is much simpler and easier to understand.

A common prediction of both these models and the 1988 work (Libertini 1988) is that, in the comparison between species and other factors being equal, extrinsic or environmental mortality must be inversely related to the proportion of deaths due to intrinsic mortality (P_s, "proportion of senescent deaths" (Ricklefs 1998, p. 33); the concept of P_s is illustrated in the Fig. 6).

This means that species in conditions of high environmental mortality should show a reduction of life span due to aging to a lesser extent than



Kin Selection Aging Theory, Fig. 5 The spread or elimination of gene C that determines a reduced *ML* ($ML_C = 0.7$). The value of C at generation 0 is equal to 0.1; $S_m = 0.01$; $m = 600$; $S' = 0.2$. The values of r are indicated near each curve

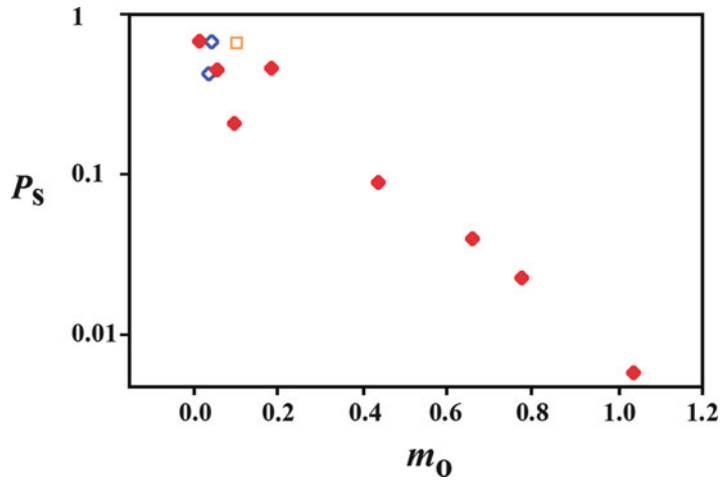


Kin Selection Aging Theory, Fig. 6 Curve 1 shows the life table of a species with age-related mortality increase. The overall mortality is given by the sum of extrinsic mortality (m_0) and intrinsic mortality (m_i) due to aging. Curve 2 shows a hypothetical life table with the same m_0 and with m_i equal to zero (no age-related increment of mortality). Curve 1 delimits the area V, the area Z is the space between the curves 1 and 2, and the curve 2 delimits the sum of the areas V and Z. The term P_s, proportion of deaths due to aging, indicates the ratio Z/(V + Z)

species with low environmental mortality. This phenomenon, defined in its first proposition with the name of the "Methuselah effect" (Libertini 1988, p. 156), is the exact opposite of what predicted by the nonadaptive theories of aging that explain aging not as an adaptive phenomenon but as the consequence of insufficient selection against harmful agents. In fact, in the case of higher environmental mortality, natural selection would become even weaker and ineffective and as a result aging should be earlier. The first prediction, namely, the inverse correlation between environmental mortality and P_s, was shown in fieldwork while trying to prove the crucial opposite prediction of nonadaptive theories (Ricklefs 1998) (s. Fig. 7). The contrast between these empirical data and the prediction of nonadaptive theories has never been explained by the supporters of such theories (Libertini 2015).

Some points must be highlighted:

1. Individuals with a greater mean duration of life (*ML*) have certainly some remarkable advantages, that is, a greater value of S' in formulas (4) and (5). For example: (i) the growth, which is the more vulnerable life period and is related to body mass, is a smaller part of *ML*, and (ii) a greater *ML* which allows a better exploitation of learning abilities. These advantages, both in the case of an adaptive and of a nonadaptive meaning of aging, should determine a positive relationship between *ML* and body mass, as well as between *ML* and learning abilities (Sacher 1959).
2. In a deme, the first copy of a C gene causing an age-related increasing mortality cannot be advantaged by the proposed mechanism of kin selection since there is no copy of C in other individuals that might be benefited by its action. The answer is probably the same as that proposed to explain the evolution of social cooperation by unselfish genes, i.e., nonselective mechanisms are important up to a critical frequency (Boorman and Levitt 1973).
3. Kin selection aging theory proposes that in particular ecological conditions previously highlighted (species divided into demes with stable populations, i.e., K-selection) a gene C that reduces the lifespan may be favored by natural selection. On the contrary, for a species where such ecological conditions are absent, the gene C is eliminated by the natural selection due to the individual disadvantages it entails and therefore individuals of this species



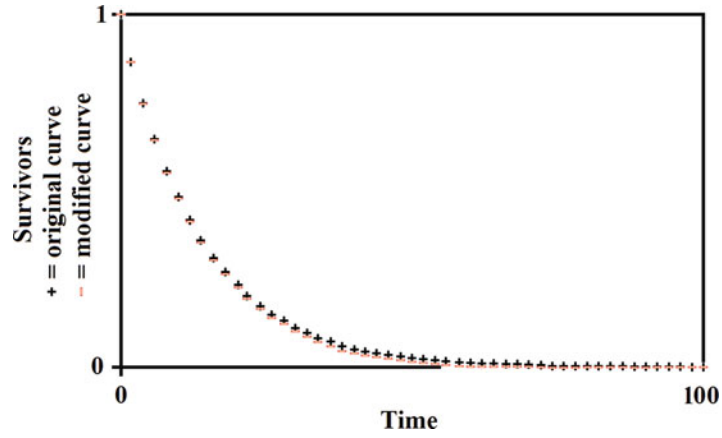
Kin Selection Aging Theory, Fig. 7 Inverse relation between extrinsic mortality (m_0) and the proportion of deaths (P_s) due to intrinsic mortality (m_i), i.e., the deaths due to the age-related increasing mortality. Open rhombs = mammal species; solid rhombs = bird species; open square = Ache people in wild conditions. Data are from (Ricklefs 1998), and Table 2 (p. 30), for Ache people, from (Hill and Hurtado 1996). Ricklefs' Fig. 7 (p. 34) has been redrawn, and the datum from Ache people has been added. Ordinates are in logarithmic scale (figure from (Libertini 2013), redrawn)

should not exhibit any increase in age-related mortality in the wild, i.e., at ages present in natural conditions. This occurs for many species that under natural conditions do not show any age-related increase in mortality (Comfort 1979; Finch 1990; Dahlgren et al. 2016; Jones and Vaupel 2017).

These same species, under protected artificial conditions in captivity, can reach ages inexistent in natural conditions and, starting from these ages, may present an age-related decline in survival capacities. In common language and also in scientific publications, this phenomenon is described by saying that also individuals of these species grow old. However, from an evolutionary point of view, this phenomenon is completely different from the phenomenon described as "increasing mortality with increasing chronological age in the wild" (IMICAW) (Libertini 1988, p. 145) and has been also defined (clearly for species that do not show the IMICAW phenomenon) as "increasing mortality with increasing chronological age in captivity"(IMICAC) (Libertini 1988, p. 146). It may seem a subtle and minor distinction, but it also signals a substantial difference.

The IMICAW phenomenon exists under natural conditions and must in some way have an evolutionary explanation. On the contrary, as the IMICAC phenomenon by definition is not present in the wild, it does not require evolutionary explanations but only mechanisms that do not require an evolutionary advantage. In fact, a hypothetical gene harmful at age t but without effects in the preceding ages may be removed by natural selection if individuals of age greater than t are present in natural conditions but cannot be removed if individuals of that age, or older, are inexistent in the wild. Similarly, harmful physiological mechanisms starting from a certain age t can be modified by natural selection if individuals of age t are present in natural conditions, but this cannot happen if individuals of this age or older are inexistent in the wild. In short, genes or mechanisms of the aforementioned type may very well explain the IMICAC phenomenon, but this does not necessarily explain the IMICAW phenomenon (Libertini 1988).

4. The mutation accumulation theory (Medawar 1952; Mueller 1987) proposes that the age-related reduction of surviving individuals



Kin Selection Aging Theory, Fig. 8 Effects of many t-genes on survival curve in the wild

weakens the elimination capacity of mutated harmful genes and this causes an age-related decline of fitness, namely, aging. However, already in the work that proposed the kin selection aging theory (Libertini 1988), a simple theoretical demonstration showed that this hypothesis was unlikely.

A gene which caused a disadvantage only and exclusively at the time t was defined as "t-gene," and the possibility that the IMICAW phenomenon could be the caused by many t-genes was discussed.

In a population with a constant death (λ), the survivors at time t (Y_t) are given by the equation:

$$Y_t = Y_0 \cdot (1 - \lambda)^t \tag{6}$$

where Y_0 = starting population.

If C is a t-gene, S the damage caused by C at time t, C′ a neutral allele of C, and V the mutation rate C′ → C, while for simplicity we suppose the mutation rate C → C′ = 0, the frequency of C at the (n + l)th generation will be:

$$C_{n+1} = \frac{C_n \cdot (1 - S \cdot Y_t - V) + V}{1 - C_n \cdot S \cdot Y_t} \tag{7}$$

The equilibrium frequency of C (C_e), i.e., when $C_{n+1} = C_n$, is given by:

$$C_e = V/(S \cdot Y_t) \tag{8}$$

Now, if in the population there are m different t-genes expressing themselves at time 1, each with a disadvantage S, the same number of t-genes with the same disadvantage at times 2, 3, . . ., the survivors at the time t + 1, will be:

$$Y_{t+1} = Y_t \cdot (1 - C_e \cdot S \cdot m) = Y_t \cdot \left(1 - \frac{V}{Y_t} \cdot m\right) \tag{9}$$

As V is small, the decrement of Y_{t+1} will be notable only with small values of Y_t. Moreover, in the equation S is absent and so its value is unimportant.

In Fig. 8, the original curve is given by formula (7) and the modified curve by Eq. (9). The values are $\lambda = 0.07$; $m = 100$; and $V = 0.00001$.

The modified curve shows that a very strong load of t-genes shifts only a little down the original curve and does not make it similar to that of an IMICAW population.

Conclusion

Kin selection aging theory offers an adaptive explanation for aging in populations spatially structured and in conditions of K-selection. This theory is analogous to others that, through complex models, suggest an advantage for aging in populations in such conditions (Travis 2004; Martins 2011; Mitteldorf and Martins 2014). All these hypotheses, and in general any model proposing an adaptive meaning of aging, predict an inverse correlation between extrinsic mortality and

proportions of deaths due to aging. This inverse correlation, which is confirmed by data from natural populations (Ricklefs 1998; Libertini 2013), is not shared by nonadaptive aging theories that actually predict a direct correlation (Kirkwood and Austad 2000). A detailed discussion of the evidence and arguments against or in support of the two opposite interpretations of aging is presented elsewhere and appears clearly in favor of the adaptive interpretation (Libertini 2015).

Cross-References

- ▶ Animal Models of Aging
- ▶ Non-evolutionary and Evolutionary Aging Theories
- ▶ Programmed (Adaptive) Aging Theories
- ▶ Timeline of Aging Research

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