No senescence despite declining selection pressure: Hamilton's result in broader perspective ☆

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HIGHLIGHTS

- Falling selection pressure alone does not explain why senescence evolves.
- We propose accounting for life history trade-offs via perturbation functions.
- We show that trade-offs do not inevitably favor senescence to evolve.
- Alternative indicators of selection pressure are mathematically related.
- Biologically justified perturbation functions are key to explain senescence.

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ABSTRACT

Theory predicts that senescence should inevitably evolve because selection pressure declines with age. Yet, data show that senescence is not a universal phenomenon. How can these observations peacefully coexist? Evolution of any trait hinges on its impact on fitness. A complete mathematical description of change in fitness, the total fitness differential, involves selection pressure along with a perturbation function that describes how the vital rates, mortality and fecundity, are affected across ages. We propose that the perturbation function can be used to model trade-offs when vital rates are perturbed in different directions and magnitude at different ages. We find that for every trade-off we can identify parameter values for which senescence does evolve and others for which it does not. We argue that this reconciles the apparent contradiction between data and theory. The total fitness differential is also instrumental in deriving mathematical relationships between alternative indicators of selection pressure. We show examples and highlight that any indicator combined with the right perturbation function can be used to parameterize a specific biological change. Biological considerations should motivate what perturbation functions are used. We interpret the relevance of Hamilton's finding that selection pressure declines for the evolution of senescence: declining selection pressure is a necessary but not a sufficient condition.

1. Introduction

Higher ages are of less evolutionary importance than younger ages. As organisms go through their life course, more and more offspring are born, so more and more of the organism's contributions to the gene pool come to lie in the past. Since earlier contributions cannot be affected by later events, death of older individuals incurs less of a penalty to evolutionary fitness than death of younger individuals. In a nutshell, this declining selection pressure is the basis of evolutionary explanations of senescence, the deterioration of organism's vital rates due to changes in its state as the organism gets chronologically older (Medawar, 1952; Williams, 1957; Hamilton, 1966). Selection pressure declines for any pattern of fecundity and survival (Hamilton, 1966), even for organisms that initially exhibit 'sustenance', unchanging rates of reproduction and survival with age (sensu Baudisch, 2008), or organisms that show 'negative senescence', defined by rising rates of reproduction declining rates of mortality with age (sensu Vaupel et al., 2004).

If declining selection pressure were a sufficient condition for the evolution of senescence, then evolution should mold any life

course, even those that initially exhibit no or negative senescence, to the senescent phenotype after sufficient evolutionary time. Yet, patterns of sustenance and negative senescence can be observed in nature (Vaupel et al., 2004; Baudisch and Vaupel, 2012). Therefore declining selection pressure alone cannot be the decisive argument, and something else must be at play (Baudisch and Vaupel, 2012).

Selection pressure expresses the sensitivity of fitness to some standard unit of change in a vital rate, mortality and fecundity, at a specific age. To know how fitness changes as a result of some real biological perturbation, it is necessary to know which vital rate (s) are affected, at which ages, and how strongly. These changes can be captured in a perturbation function, which describes the effects on mortality and fecundity as a function of age. The perturbation function completes the total fitness differential, which is the full and general analytical description of how fitness changes if mortality and/or fecundity change(s) (Arthur, 1984; Caswell, 2010). Any effect on fitness can only be known if the total fitness differential is considered.

To find an appropriate perturbation function, one has to consider the underlying biology: if mortality is perturbed at one age, what would happen biologically at other ages, and what does that mean for the perturbation function? The complex causal pathways leading to changed gene expression, the accumulation of damage, loss of physiological control, but also growth and learning (all of which affect mortality and fecundity patterns), are likely to be tied in some more or less continuous trajectory of change. These cannot be reduced to independent age-specific changes (Wensink, 2013; Kirkwood and Shanley, 2010). Here, the perturbation function is helpful, since it describes such age-patterns.

The combination of selection pressure and perturbation is commonly studied in age-structured models (Charlesworth, 1994, 2001), matrix population models (Caswell, 1982; Caswell, 2001, Section 9.1.6), and quantitative genetics (Falconer and Mackay, 1996). Yet, studies of senescence typically invoke standard-unit changes at particular ages (or age-ranges), drawing conclusions from verbal comparison of 'early' (low ages) versus 'late' (high ages) (e.g. Medawar, 1952; Williams, 1957; Hamilton, 1966; Kirkwood and Rose, 1991; Partridge and Barton, 1993; Abrams, 1993). In the same vein, conclusions about the evolution of senescence are frequently drawn directly from patterns of selection pressure (e.g. Partridge and Gems, 2006; Martin, 2007; Metcalf and Pavard, 2007; Monaghan et al., 2008; Kirkwood and Melov, 2011; Shahrestani et al., 2012). We exemplify biologically realistic perturbation functions and use those in combination with the associated selection pressure, thus completing the evolutionary analysis. This leads to results that are not evident from models based on selection pressure alone. Mathematical relationships between alternative indicators of selection pressure are clarified using the perturbation function. We conclude with showing that Hamilton's finding is a necessary but not a sufficient cause for the evolution of senescence.

2. Fitness consequences of changes in vital rates

Hamilton (1966) used the intrinsic rate of increase 'r' as a measure of fitness, defined as the unique real root of the Euler–Lotka equation, within the framework of stable population theory (Lotka, 1924; Charlesworth, 1994; Caswell, 2001):

$$\int_0^\infty e^{-rx} l(x) m(x)\, dx = 1 \tag{1}$$

In this equation $l(x)$ denotes survival up to age x and $m(x)$ denotes age-specific fecundity. Survival is related to the instantaneous

mortality rate $\mu(x)$:

$$l(x) = e^{-\int_0^x \mu(t)\, dt} \tag{2}$$

By implicit differentiation of r with respect to an additive perturbation of mortality and fecundity respectively, Hamilton (1966) derived indicators of selection pressure on age-specific additive perturbations of mortality and fecundity. These indicators are as follows:

$$\frac{dr}{dF_a} = \frac{e^{-ra} l(a)}{T} \tag{3}$$

$$\frac{dr}{d\Delta_a} = -\frac{\int_a^\infty e^{-rx} l(x) m(x)\, dx}{T} \tag{4}$$

where

$$T = \int_0^\infty x e^{-rx} l(x) m(x)\, dx \tag{5}$$

which is the average age at reproduction in a population, i.e. generation time (Charlesworth, 1994). Furthermore, $d\Delta_a = d\mu(a)\, da$, an infinitesimal additive change in mortality multiplied by an infinitesimal neighborhood of the age at which this change takes place, and $dF_a = dm(a)\, da$, an infinitesimal additive change in fecundity multiplied by an infinitesimal neighborhood of the age at which this change takes place.

Using functional calculus, Arthur (1984) derived a general analytical expression for the sensitivity of r to changes in the patterns (rather than age-specific values) of fecundity and survival, writing r in its differential form:

$$dr = \frac{1}{T}\left[\int_0^\infty e^{-ra} dl(a) m(a)\, da + \int_0^\infty e^{-ra} l(a)\, dm(a)\, da\right] da \tag{6}$$

If the perturbation of survival is considered at the mortality level, the two being related through Eq. (2), applying the product rule to $dl(a)$ and integrating by parts, this expression can be rewritten as follows:

$$\frac{dr}{d\varepsilon} = \int_0^\infty \left[\frac{e^{-ra} l(a) dm}{T}(a, \cdot) - \frac{\int_a^\infty e^{-rx} l(x) m(x)\, dx d\mu}{T}\frac{d\mu}{d\varepsilon}(a, \cdot)\right] da \tag{7}$$

Perturbation parameter ε captures small perturbations in fecundity ($dm/d\varepsilon(a, \cdot)$) and mortality ($d\mu/d\varepsilon(a, \cdot)$). These perturbations can be functions of age, and possibly other parameters, indicated by the dot. The two other elements can be recognized as Hamilton's indicators of selection pressure, Eqs. (3) and (4). Writing H^* and $H^\dagger$ for Hamilton's indicators of selection pressure on additive changes in fecundity and mortality rate respectively, the general equation for change in r is

$$\frac{dr}{d\varepsilon} = \int_0^\infty \left[H^*(a)\frac{dm}{d\varepsilon}(a, \cdot) + H^\dagger(a)\frac{d\mu}{d\varepsilon}(a, \cdot)\right] da \tag{8}$$

At every age, the effect of change in mortality and fertility on fitness is given by the product of fitness sensitivity (H^* or $H^\dagger$) and the perturbation in the vital rate ($dm/d\varepsilon$ and $d\mu/d\varepsilon$). Integration over all ages then yields the full fitness consequences.

As an example of a perturbation function, mortality μ could equal some constant c in the baseline scenario, while perturbed mortality could be given by

$$\mu(a, \varepsilon) = c + \varepsilon(a - p)s \tag{9}$$

where age p is the one age at which the perturbed mortality function crosses the baseline (constant) mortality, $\varepsilon \geq 0$ is a perturbation parameter, while parameter $s > 0$ models the strength of the trade-off. Both s and ε are given in units of time^{-1}. Except for its dimensionality, parameter s is redundant in this case, but not in other perturbations (see below), and is included here for consistency. The perturbation function expresses how strongly mortality gets to