

Programmed death theory revised with artificial evolution

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Abstract. In this paper we revise the Weismann’s idea of aging and death as adaptive traits. First, we analyse the adaptation process to changing environment of isolated populations (species) having different lifetime and show, that short lifetime may have adaptive value at a population level. Next, we use a Markov chain to model a competition between individuals in a finite population. We derive a condition for equilibrium in the population and explain why the long-lived individuals usually dominate the population. As far as the model reflects reality, our conclusion is that though not impossible, it would require an extraordinary environment for programmed death to be stable adaptive trait.

1 Introduction

Despite the ages of inquiry, the search for the ultimate answer to the fundamental question of “Why do we age?” [7] is still ongoing. One of the first explanation of aging on the ground of evolutionary theory was given by experimental biologist August Weismann in 1882 [2, 8]. Weismann’s original hypothesis, known also as ‘programmed death’ theory, assumes that biological organisms evolved a specific death mechanism in order to maintain swift generation turnover, resulting in better adaptability to changing environment. Programmed death presumably eliminates the old, “worn-out” members of a population, making room and sparing resources for younger individuals. So rather than individual’s adaptive trait, it would be an altruistic trait, beneficial for the whole population and allowing it to take advantage over some neighbor populations. So in general terms, Weismann suggested, that aging and death by senescence are adaptive traits, evolved by natural selection.

Later, Weismann himself discarded the programmed death hypothesis, long before a systematic research questioned the viability of any group selection mechanisms, and altruistic aging in particular. There are several arguments against the programmed death [2, 6, 8]. The major one is that organisms in the wild quite seldom die due to their age and more often causes are extrinsic, such as predation, infections or hunger. Thus, there is a weak selection pressure exerting the “one more reason to die”. From the programmed death it follows, that the age of the oldest organisms in the wild shall not significantly differ from the age of the old organisms kept in some protected environment. Gavrilov

and Gavrilova [2] give an example of the chaffinch (*Fringilla coelebs*), which can live for 29 years in captivity, but due to the extrinsic mortality, its mean lifespan in the wild is about 18 months and only 0.1% of chaffinches survive to age 11. So either the programmed death doesn't have much opportunity to exercise in the wild or the death in captivity isn't programmed at all.

Another argument results from the analysis of statistical data on the age of death. Programmed death predicts a sharp increase of mortality around some specific age, while studies over many species reveal, that the curve of rate of death is smooth and monotonic, without the expected peak. The last major argument comes from the evolutionary point of view itself, as it questions the adaptive value of the premature death. If an individual is poorly adapted, it will die in the one of many possible ways, but there is little point in putting it to death on schedule if it's well adapted. "The individual cost of programmed death is sufficiently great and the group benefit sufficiently diffuse that quantitative models that are based on multilevel selection do not support the evolution of ageing programmes through these mechanisms" [8].

Since Weismann's idea has been discarded, several other explanations of aging in evolutionary perspective were proposed. Namely 'mutation accumulation' theory, 'disposable soma' theory and the 'antagonistic pleiotropy' theory [7, 2, 8]. All the three theories are not mutually exclusive, but they interweave and there is evidence supporting each of them, though counterexamples also can be found. In general, they assert aging is a detrimental side effect and not adaptation.

Surprisingly, the Weismann's idea of aging as an adaptation occasionally returns and arguments in favour of the programmed death, or more generally 'programmed aging', are adduced [9, 8]. Apart from mentioned benefits in the form of increased adaptability at the population level, there is also possible role of aging in stabilizing population dynamics, i.e. preventing overcrowding and local extinction [10] and reducing the negative impact of epidemics [5]. The novel argument is that senescence contributes to "organized" generation turnover and helps to maintain genetic diversity in a population. Diversity, which is important for "genetic health" and which could be lost due to endless reproduction of some well-fit, non-aging and dominant individuals [3]. The likelihood of death as genetic adaptation was also suggested by Travis [11], reporting computational results from a model of spatially structured population with localized dispersal.

In the following sections we reconsider the Weismann's idea in a framework of artificial evolutionary models. In section 2 we refresh the famous Hinton and Nowlan's [4] experiment to show the existence of an optimal rate of generation turnover. Next, in section 3 we construct even further simplified model of evolution and show that short lifetime can be beneficial trait in isolated population, though it's rather detrimental in a mixed population of short- and long-lived "species"¹. Finally, we derive a condition for equilibrium between many co-existing species having different programmed lifetime and verify this condition with simulation experiments.

2 The Hinton and Nowlan's experiment revisited

In their famous experiment, Hinton and Nowlan [4] investigate the Baldwin effect, i.e. the relationship between learning and evolution. Let's briefly describe their model and

¹We call them species, though the term 'population' shall refer to individuals of a single specimen.

see how it's related to the problem of aging.

Let there be some target binary string of length $l = 20$ (that is environment) and a population of $P = 1000$ individuals having a triallelic string genotype of matching length l . The genotype's alphabet is $\{0, 1, ?\}$, where 0's and 1's correspond to fixed alleles and the ?'s are learnable. Each individual is initialized with some random string from the uniform distribution of fixed and learnable alleles, so that initial frequencies of alleles are 0.25, 0.25 and 0.5, respectively. The individual lives for $T = 1000$ time steps and in every step it guesses a combination of learnable alleles. If the resulting bit string of fixed and learned alleles matches the target string, then the individual obtain fitness value $f = 1 + (l-1)n/T$ where n amounts to remaining lifetime. So individuals that didn't guess the target string get fitness 1 and those who guessed immediately get 20. Then follows a reproduction and individuals are selected proportionally to their fitness (roulette wheel) and recombined (one-point crossover). We introduce only a minor modification to the original model by also applying a mutation, with probability $p_m = 0.005$.

Hinton and Nowlan explain the model in more details and also consider its realistic plausibility. We are not concerned with the mechanisms of learning — whether it's performed by a neural network or it has a form of some biochemical adaptation, like perhaps in some microorganisms. The question we ask is if there is some optimal lifetime T , resulting in the fastest adaptation to the environment? Figure 1 presents the results of simulations averaged over 50 trials. Each curve along time (t axis) corresponds to a different specimen or isolated population of individuals having their lifetime T fixed. All the populations live for 2^{16} time steps, so that species having the lifetime shorter reproduce more frequently and *vice versa*. The number of generations a population lives through is then $2^{16}/T$.

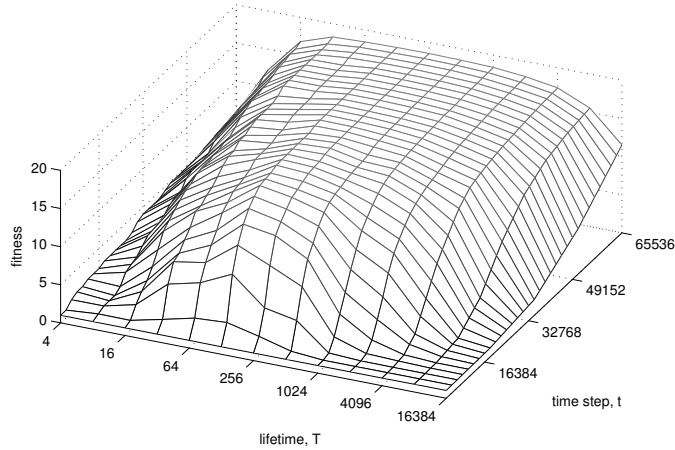


Figure 1. The course of adaptation of species having specific lifetime.

One could argue, that this comparison is biased toward short-lived species, as it doesn't take into account the possible discontinuity of learning due to reproduction and the period of development. In additional experiments we assumed a constant “penalty period” of 100 time steps for each generation. Such a penalty impacts mostly the short-lived populations, reducing their learning effectiveness and slowing their adaptation.

The fitness “fore-front” shifts toward the longer-lived populations. The negative impact, however, declines with increasing lifetime and the optimal lifetime appear not to shift very far — from around 128 to 256 time steps.

3 Another model of evolution

We used the Hinton and Nowlan’s model to show, that adaptability doesn’t necessarily increase monotonically with the lifetime and some optimum can be found. The model was devised to investigate the Baldwin effect and takes the learning into account. Let’s further simplify the model and reject learning, leaving the genetic adaptation only.

If we drop the learning and modify the fitness function slightly, what remains resemble the one-max problem. Let there be target binary string of length $l = 20$ and so long the genotypes of individuals. As before, extrinsic mortality is excluded and individuals are assured to live for T time steps. The fitness is calculated as

$$f = (m/l)^\phi, \quad (1)$$

where m denotes the number of alleles in a genotype matching the target and ϕ is some selection pressure factor (exponent). The reproduction cycle lacks recombination. Selection is proportionate and mutation acts at every locus with probability $p_m = 1/(2l)$.

In such a model, where the adaptation is genetic only, the advantage of fast generation turnover is obvious. The expected rate of adaptation of randomly initialized population is inversely proportional to the lifetime of individuals, i.e. increase in mean fitness $\Delta f \propto 1/T$. However, this is only true in the very early phase of adaptation. As soon as the fitness rises above average, deleterious mutations come to dominate and the fitness saturates.

Adaptation to the static target (environment) could be reformulated as an adaptation to some permanently changing environment. Let there be some probability $p_r = 1/l$ of mutation of the target string (i.e. in every time step every locus is mutated with probability p_r). Then adaptability becomes adaptation and differences among rates of adaptation appear as differences in levels of adaptation. This is illustrated in Figure 2, presenting results of simulations averaged over 100 runs. Different rates for each of the species in the early phase of adaptation turn to stable fitness values.

Apparently, in this purely artificial and very simple model, populations of short-lived individuals demonstrate their advantage. But a significant assumption here is that populations are isolated. The picture changes quite radically if we assume the individuals of different lifetime species are mixed within a single, finite population. What are the conditions for equilibrium between competing species?

4 Employing a Markov chain

The model presented in the previous section can be described with a Markov chain (see e.g. [1]). Most evolutionary algorithms has a Markov property since the next generation usually depends only on the previous one, as in our case.

Consider some finite and fixed size population P consisting of n species, represented by S_i individuals each, i.e. $P = \{S_i\}_{i=1,\dots,n}$. Individuals of different species are identical, except of their expected (programmed) lifetime T_i and presumed fitness f_i . Let $p_i(t)$ denote the frequency (proportion) of individuals of specimen i in the population.

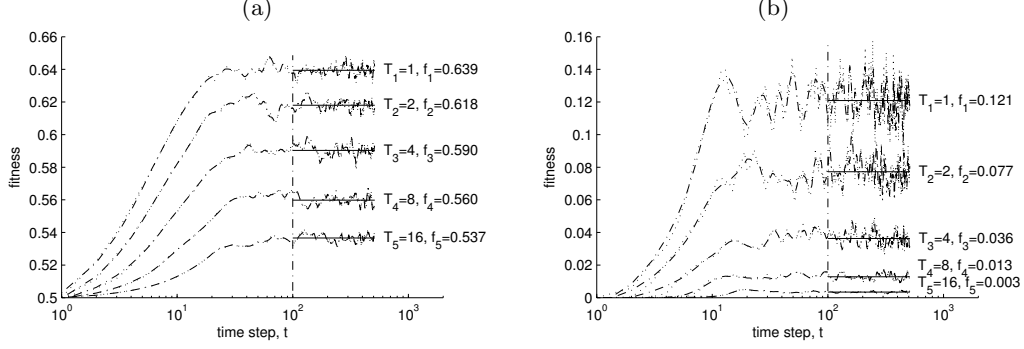


Figure 2. The course of adaptation to changing environment of isolated populations of individuals having specific lifetime T_i and for selection pressure $\phi = 1$ (a), and $\phi = 16$ (b).

The reproduction cycle doesn't differ much from that described in section 3. In every time step t individuals whose lifetime expired, get replaced by mutated copies of individuals selected from the population. Selection is proportional to the fitness of individuals, regardless of specimen, so that frequencies of species $p_i(t)$ may vary. At any time step, the probability that individual having lifetime T_i dies is equal to $\frac{1}{T_i}$ and the probability it's still alive is $\frac{T_i-1}{T_i}$. Probability that individual of specimen i will be replaced by individual of specimen j in the next step, given that individual i dies, is:

$$q_j(t) = \frac{p_j(t)f_j}{\sum_{i=1}^n p_i(t)f_i}.$$

Let consider the following Markov chain with n states, corresponding to the evolving population (Fig. 3). The state i of the chain denotes individuals of specimen i , i.e. whose lifetime is T_i . In every time step t , the state i can remain the same or switch to state j . Probabilities of transition from i to j are given by:

$$m_{ij}(t) = \begin{cases} \overbrace{\frac{T_i-1}{T_i}}^{i \text{ is still alive}} + \overbrace{\frac{1}{T_i} \cdot q_j(t)}^{i \text{ dies, but is reselected}} & \text{for } i = j \\ \underbrace{\frac{1}{T_i} \cdot q_j(t)}_{i \text{ dies and gets replaced by } j} & \text{for } i \neq j \end{cases}.$$

Notice the transition matrix $M_t = [m_{ij}(t)]$ depends on time, what means that Markov chain is non-stationary. Having transition matrix M_t and initial distribution p_j^0 we can produce frequencies $p_j(t)$ of individuals belonging to different species at any time step t :

$$\begin{aligned} p_j(1) &= p_j^0, \quad j = 1, \dots, n \\ p_j(t+1) &= \sum_{i=1}^n p_i(t)m_{ij}(t), \quad j = 1, \dots, n, \quad t = 1, 2, \dots \end{aligned} \quad (2)$$

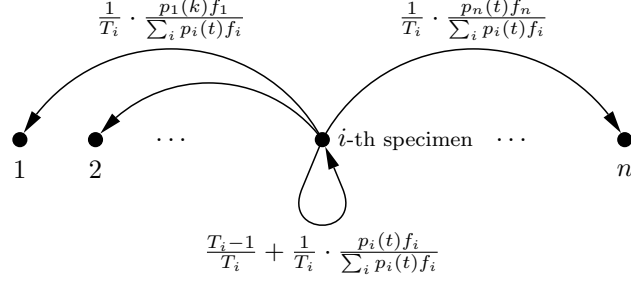


Figure 3. Markov chain corresponding to the evolving population, i -th specimen considered.

Let's consider the simplest case, when we have only two species — the short-lived $S_s(f_s, T_s)$ and the long-lived $S_l(f_l, T_l)$. We want to find the conditions on the fitness function to keep the population in the equilibrium.

From the stationary distribution condition:

$$[p_s, p_l] \cdot \begin{bmatrix} \frac{T_s-1}{T_s} + \frac{1}{T_s} \cdot \frac{p_s f_s}{p_s f_s + p_l f_l} & \frac{1}{T_s} \cdot \frac{p_l f_l}{p_s f_s + p_l f_l} \\ \frac{1}{T_l} \cdot \frac{p_s f_s}{p_s f_s + p_l f_l} & \frac{T_l-1}{T_l} + \frac{1}{T_l} \cdot \frac{p_l f_l}{p_s f_s + p_l f_l} \end{bmatrix} = [p_s, p_l],$$

the equilibrium condition appears to be $T_l f_l = T_s f_s$. This result clearly shows, that short-lived individuals must be as many times more fit as they life is shorter if they are to have equal chance to survive.

Similar condition can be derived in case of a mixture of n species, i.e. when $P = \{S_i(f_i, T_i)\}_{i=1, \dots, n}$. Suppose that $[p_1, p_2, \dots, p_n]$, $p_j > 0$ is the stationary distribution:

$$\sum_{i=1}^n p_i m_{ij}(t) = p_j, \quad j = 1, \dots, n,$$

what expands to:

$$p_1 \frac{1}{T_1} \cdot \frac{p_j f_j}{\sum_i p_i f_i} + \dots + p_j \left(\frac{T_j-1}{T_j} + \frac{1}{T_j} \cdot \frac{p_j f_j}{\sum_i p_i f_i} \right) + \dots + p_n \frac{1}{T_n} \cdot \frac{p_j f_j}{\sum_i p_i f_i} = p_j,$$

and after some algebraic transformations we obtain equilibrium condition:

$$f_j = \frac{1}{T_j} \frac{\sum_i p_i f_i}{\sum_i p_i / T_i}, \quad i = 1, \dots, n.$$

This condition is met, when

$$f_i T_i = \text{const}, \quad i = 1, \dots, n. \quad (3)$$

Then probabilities of survival among species are all equal and the proportions in the population shall remain constant during the evolution.

When the condition (3) is not satisfied, some species, namely those maximizing $f_i T_i$, take selection advantage over the other and the proportions evolve. Example simulation results are shown in Figure 4. In case all the species have equal fitness (or the selection

pressure $\phi = 0$ in eq. 1), the longest-lived specimen quickly dominates the population, regardless of the initial distribution (still assuming $p_i > 0$). The domination of species maximizing $f_i T_i$ value can be observed in Figure 4b.

Using a Markov chain to model the evolution of population consisting of many competing species one can explain, why the long-lived individuals usually dominate the population. Despite they are less fit, the product of their fitness and lifetime is expected to be still higher than those of short-lived individuals. Simulation results are shown in Figure 5. Assuming the linear fitness function ($\phi = 1$) and starting with a population dominated by the short-lived individuals, the longest-lived individuals quickly dominate the population, even though their fitness is lower. This may not be the case with a nonlinear fitness function ($\phi > 1$), where small fitness advantage resulting from swift generation turnover translates into large selection advantage.

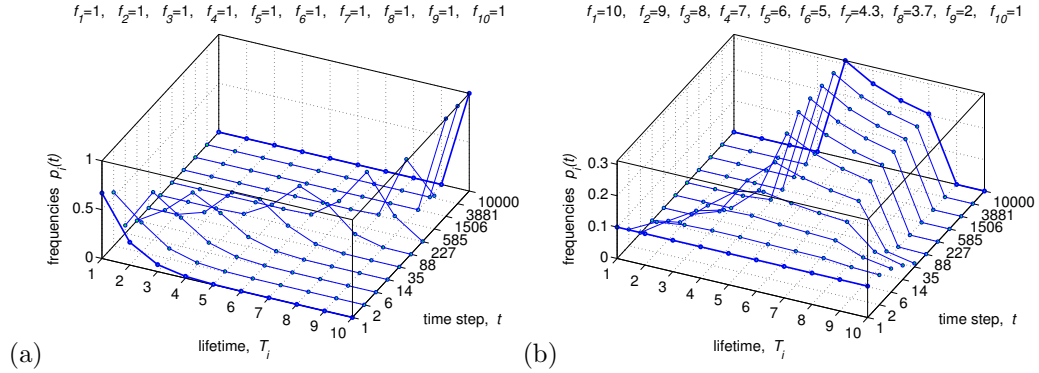


Figure 4. Evolution of species proportions in the population with equal fitness ($f_i = 1$, a) and with the arbitrarily modified fitness values (b).

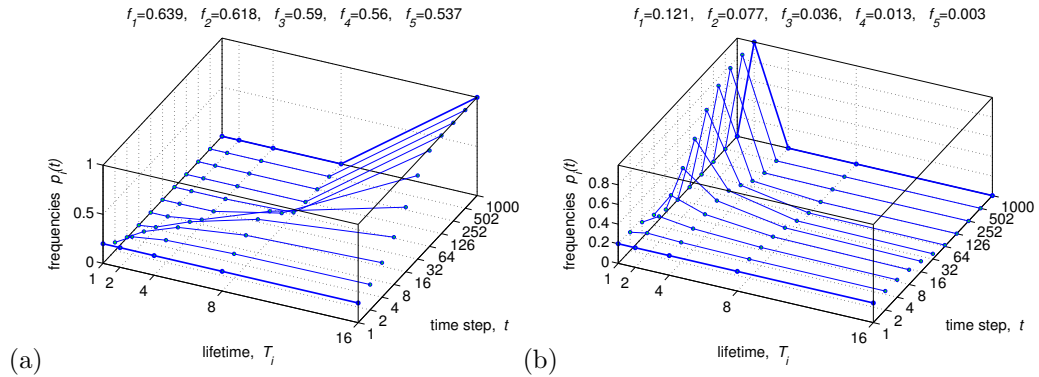


Figure 5. Evolution of species proportions with fitness values f_i identified experimentally (Fig. 2) for $\phi = 1$ (a) and $\phi = 16$ (b).

5 Conclusions

Swift generation turnover induced by the short lifetime may result in better genetic adaptation of a population or specimen. But for the short lifetime to be stable adaptive trait

some conditions must be satisfied. Either the short lifetime must be altruistically shared across the whole population and the population protected from a long-lived mutants or invaders, or the fitness benefit resulting from the rapid generation turnover must be sufficiently high. It might be quite difficult to give an example of the environment, where organisms adapt so quickly, that increase in their fitness from generation to generation is proportional to the inverse of their lifetime. This is reflected by the high nonlinearity ($\phi = 16$ in eq. 1) in the fitness function, required to promote the short lifetime. So the trade-off between individual costs and the group benefits of the short lifetime is usually determined by the former.

The model described in sections 3 and 4 is drastically simple. Among many other simplifications we excluded the cost of reproduction, possible fitness benefits from learning or the mutability of lifetime. As far as the model is realistically plausible, we conclude that aging and the programmed death can not be precluded as adaptive traits. Theoretical conditions we derived in section 4 to support this claim seem quite unrealistic, but not impossible to satisfy. Perhaps there are environmental niches, where selection conditions are sufficiently “nonlinear”, resembling the winner-takes-all rule, to foster adaptive lifetime.

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