Exact solution of an evolutionary model without aging

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We introduce an age-structured asexual population model containing all the relevant features of evolutionary aging theories. Beneficial as well as deleterious mutations, heredity, and arbitrary fecundity are present and managed by natural selection. An exact solution without aging is found. We show that fertility is associated with generalized forms of the Fibonacci sequence, while mutations and natural selection are merged into an integral equation which is solved by Fourier series. Average survival probabilities and Malthusian growth exponents are calculated and indicate that the system may exhibit mutational meltdown. The relevance of the model in the context of fissile reproduction groups like many protozoa and coelenterates is discussed. [S1063-651X(99)03109-8]

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I. INTRODUCTION

For all live organisms death is inexorable. If life is not abbreviated by disease, predation, or accidents then death is preceded by senescence [1,2]. Senescence seems to be related to reproductive strategies and it starts after an individual reaches the fertile age. It can be a slow process for species which reproduce many times (iteroparous) or an abrupt process for species reproducing only once (semelparous)—the catastrophic senescence of the Pacific salmon being a good example of the latter.

There are three kinds of aging theories: biochemical, evolutionary [3,4], and telomeric [5]. The biochemical invokes damage on DNA, cells, tissues, and organs. Defective proteins are synthesized, altering the normal course of metabolism. The presence of free radicals, i.e., of unpaired highly reactive electrons, can cause death of the cells or may even lead to cancer. As a consequence, such theories are today firmly connected to modern gerontology. Biochemical theories predict that species with higher metabolism would have shorter lifetime. A criticism against this result arises when the lifetimes of birds and mammals (of the same size and under optimal conditions in captivity) are compared. Usually, birds live longer. Even closely related organisms like bats and rodents with comparable sizes have very different lifetimes. Such differences should be explained by the evolutionary theories of aging. In the telomere hypothesis of senescence, replication of normal cells is accompanied by a telomeric shortening. This acts as a mitotic clock resulting in a permanent exit of the cell cycle.

Evolutionary theories of aging are hypothetico-deductive in character, not inductive. They fall into two classes: the optimality theory and the mutational theory. In the optimality theory [6,7], fitness is maximized by increasing the survival and reproduction rate early in life at the expense of late, i.e., it sees senescence as a necessary cost of processes beneficial to youth. On the other hand, the mutational theory [8] explains senescence as a result of late-acting deleterious mutations, i.e., that a greater mutation load on the last part of the life is less strongly selected.

In this paper, we obtain both analytical and numerical solutions for a simple age-structured population model. In this model, reproduction is asexual and the individuals are submitted to helpful or deleterious hereditary mutations. The number of offspring is fixed but arbitrary otherwise and the population dynamics is managed by natural selection. Amazingly, this system does not exhibit senescence even when deleterious mutations are predominant. From a mathematical point of view, the solution we found factorizes into a fertility and a mutational sector. It is shown that the fertility sector is completely described by generalized forms of Fibonacci sequences. On the other hand, the mutational sector, solved by Fourier series, incorporates the combined effects of mutation and natural selection. If harmful mutation is intense then a mutational meltdown [9] process can take place. All these results were corroborated by Monte Carlo simulations. From a biological point of view, our results make the model a good candidate to describe groups in which all reproduction occurs by fission, such as protozoa and a miscellany of coelenterates, since all of them appear to lack aging [3].

II. MODEL

Consider a population distributed by $L+1$ ages i ($i=0,1,\ldots,L$) with respective birth rates m_i. We call babies the individuals at age 0. They do not reproduce, i.e., $m_0=0$. Individuals with age $i=L$ die after reproduction. Let $N_i(J,t)$ be the number of individuals at age i with survival probability J between J and $J+dJ$ at time t (of course, $J\in[0,1]$). We choose, as initial condition, a uniform distribution of babies, i.e., $N_i(J,0)=N_0\ \delta_{i,0}$ with N_0 being a constant. Let us point out that senescence here means that the average survival probability drops with age i. At time t, each individual is submitted to mutation, which changes its survival probability from J to J' in the following way:

$$J' = J\ e^{\epsilon}, \tag{1}$$

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where ϵ is a random number chosen in the interval $[-a,b]$ ($a>0$ and $b>0$), generated by the uniform probability density distribution $q(\epsilon)=[H(\epsilon+a)-H(\epsilon-b)]/(a+b)$ [$H(x)$ stands for the Heaviside function]. If $a>b$ then deleterious mutations are dominant. The transition probability $P(J',J)$ that an individual has its survival probability changed from J to J' is given by the integral $P(J',J)=\int_{-\infty}^{+\infty}\delta(J'-Je^{\epsilon})q(\epsilon)d\epsilon$. Observing that a helpful mutation is allowed as long as it does not increase the survival probability beyond unity, we get

$$J'(a+b)P(J',J)=\{[H(J'-Je^{-a})-H(J'-Je^{b})] \times[H(J)-H(J-e^{-b})]+[H(J'-Je^{-a}) -H(J'-1)][H(J-e^{-b})-H(J-1)]\}. \tag{2}$$

After mutation, the entire population passes through natural selection. The survivors then reproduce, generating babies with inherited characteristics, i.e.,

$$N_0(J,t)=\sum_{i=1}^{L} m_i \;\; N_i(J,t) \;\; \text{for} \;\; t\geq 1. \tag{3}$$

At time step $t+1$ individuals get older or die. Taking into account mutations and natural selection (in this order), their number is given by

$$N_{i+1}(J',t+1)=\int_0^1 J' \;\; P(J',J) \;\; N_i(J,t) \;\; dJ$$

$$\text{for} \;\; i=0,\ldots,L-1. \tag{4}$$

Clearly, $N_i(J,t<i)=0$ for $i\neq 0$. For $t\geq 1$, Eqs. (3) and (4) can be rewritten in the matricial form

$$\vec{N}(J',t+1)=M\int_0^1 f(J',J) \;\; \vec{N}(J,t) \;\; dJ, \tag{5}$$

where $f(J',J)=J' \;\; P(J',J)$ and $\vec{N}(J,t)$ and M are the following vector and matrix:

$$\vec{N}(J,t)=\begin{pmatrix} N_1(J,t) \\ N_2(J,t) \\ N_3(J,t) \\ \vdots \\ N_L(J,t) \end{pmatrix},$$

$$M=\begin{pmatrix} m_1 & m_2 & m_3 & \ldots & m_{L-1} & m_L \\ 1 & 0 & 0 & \ldots & 0 & 0 \\ 0 & 1 & 0 & \ldots & 0 & 0 \\ \vdots & \vdots & \vdots & & \vdots & \vdots \\ 0 & 0 & 0 & \ldots & 1 & 0 \end{pmatrix}.$$

Matrix M is a special form of the Leslie matrices [10]. Iterating Eq. (5) we obtain at time $t\geq 1$

$$\begin{pmatrix} N_1(J_t,t) \\ N_2(J_t,t) \\ N_3(J_t,t) \\ \vdots \\ N_L(J_t,t) \end{pmatrix} = N_0 \;\; F(J_t,t) \;\; M^{(t-1)} \begin{pmatrix} 1 \\ 0 \\ 0 \\ \vdots \\ 0 \end{pmatrix}, \tag{6}$$

where

$$F(J_t,t)=\int_0^1 \cdots \int_0^1 \prod_{j=1}^{t} f(J_j,J_{j-1}) \;\; dJ_{j-1} \tag{7}$$

and M^0 is the $L\times L$ identity matrix.

III. GENERALIZED FIBONACCI SEQUENCES

Equation (6) shows that the dynamics factorizes into two sectors: the fertility, exclusively contained in the matrix M, and the mutational, including both mutations and natural selection processes and represented by the function F. Clearly, at time t, we need only to know the first column elements of the $(t-1)$th power of the fertility matrix. As we shall see, these elements form a Fibonacci sequence. Let us call $A_L(t+1-i)$ the element of the ith line and first column. It is a simple exercise to verify that if $L=2$ and $m_1=m_2=1$ then $A_2(t)$ can be calculated, at any time t, through the relation $A_2(t)=A_2(t-1)+A_2(t-2)$, with $A_2(1)=A_2(2)=1$, which is exactly the Fibonacci sequence. The ratio of two successive numbers, $\lim_{t\to\infty}A_L(t+1)/A_L(t)=1.618\ldots$, gives the golden section or the divine proportion, as it was called by Kepler. It is astonishing to find this ubiquitous sequence in such disparate things as the cluster-cluster aggregates [11], the division of a line into extreme and mean ratio, the pentagram star worn by the Pythagoreans, the continued fractions, the aesthetic proportions of the Parthenon at Athens [12], and now, here, in population dynamics.

When the number of ages is 4 ($L=3$) and $m_1=m_2=m_3=1$ then $A_3(t)=A_3(t-1)+A_3(t-2)+A_3(t-3)$, with $A_3(1)=A_3(2)=1$ and $A_3(3)=2$ (this sequence generates the so called tribonacci numbers). For an arbitrary number of ages and fecundity we have

$$A_L(t)=\sum_{k=1}^{L} m_k \;\; A_L(t-k), \;\; \text{for} \;\; t\geq(L+1). \tag{8}$$

The first L numbers (necessary to initialize the sequence above) are evaluated through the identification $A_L(t)\equiv A_2(t)$, for $t=1,\ldots,L$. The numbers $A_2(t)$ are determined, on the other hand, by using $A_2(1)=1$ and $A_2(2)=m_1$ in the expression above. Recurrence formulas like Eq. (8) are *generalized forms* of the Fibonacci sequence. These results, together with Eq. (6), allow us to write down the number of individuals at time t with age $i\geq 1$ and survival probability J (we renamed $J_t\to J$),

$$N_i(J,t)=\begin{cases} N_0 \;\; A_L(t+1-i) \;\; F(J,t) & \text{if} \;\; t\geq i \\ 0 & \text{otherwise.} \end{cases} \tag{9}$$

The number of babies $N_0(J,t)$ may be determined using Eqs. (3) and (9). The mean survival probability at any time t and at arbitrary age i can be calculated as

$$\langle J\rangle_i(t)=\frac{\int_0^1 J\ \ N_i(J,t)\ \ dJ}{\int_0^1 N_i(J,t)\ \ dJ}=\frac{\int_0^1 J\ \ F(J,t)\ \ dJ}{\int_0^1 F(J,t)\ \ dJ}, \quad (10)$$

which is *independent* of i, that is, the model *does not show senescence*. It is important to note that, although aging is absent in our model (in the sense that the mean survival probability does not depend on the age i), an individual can show senile decay (that is, its survival probability J diminishes with time). This individual handicap is compensated by the natural selection of the fittest so, at a collective level, no senescence is observable. We also call attention to the fact that, in our model, the total number of individuals with age i diminishes with i. If we integrate $N_i(J,t)$ over J [Eq. (9)], the ratio between two successives ages i and $i+1$ is given by $A_L(t+1-i)/A_L(t+i)$ of the Fibonacci sequence. This ratio depends on age i only for small time t. Moreover, the logarithm of this ratio corresponds to the so-called mortality rate. For humans, the Gompertz law [13] suggests that mortality increases exponentially with age. In our model it is constant.

At a fixed age i and survival probability J, the population increases with time at a rate given by $N_i(J,t)/N_i(J,t-1)=[A_L(t+1-i)/A_L(t-i)][F(J,t)/F(J,t-1)]$. The first ratio of the right side can be easily calculated by the generalized Fibonacci sequences, but the second requires a numerical analysis.

IV. NUMERICAL ANALYSIS

The continous variable J can be divided into Q intervals such that $J\equiv j/Q$ for $j=1,\ldots,Q$ and infinitesimal increment $dJ\equiv 1/Q$. For Q big enough we expect to reobtain the continous limit. In the same way, the products of $f(J_j,J_{j-1})$ in Eq. (7) can be seen as simple matricial products. We wrote down a FORTRAN program with extended precision to determine $F(J,t)$ and $\langle J\rangle_i(t)$ at any elapsed time t. For $Q=400$, $L=10$, $a=0.04$, and $b=0.02$, Fig. 1 shows the dependence of $F(J,t)$ with J at different instants. After a time $t>50$ we verified that

$$F(J,t+1)=c\ \ F(J,t), \quad (11)$$

where c depends on a and b but not on J. This means that after enough time, the system reaches an asymptotic limit where $F(J,t)\equiv c^t\overline{F}(J)$, i.e., there is a separation of variables and $\overline{F}(J)$ is a stationary solution. Fixing $b=0.02$ and varying $a=0.02$, 0.04, and 0.08, we determine numerically that $c=0.92$, 0.76, and 0.48, respectively. If, for example, we choose $L=10$ and $m_i=1$ for any i then $\lim_{t\to\infty}[A_L(t+1)/A_L(t)]=1.9990\ldots$, and a Malthusian exponential growth of the population e^{rt} with $r=0.61$, 0.42, and -0.04, respectively, is obtained. The last value shows that the system exhibits mutational meltdown [9] (extinction).

The discretized form of the variable J can also be used in order to calculate the mean survival probability $\langle J(t)\rangle$ [Eq. (10), dropping the now unnecessary lower index i] as a function of time. Table I shows our results for $L=10$. For fixed matrix dimension, the time convergence is very fast.

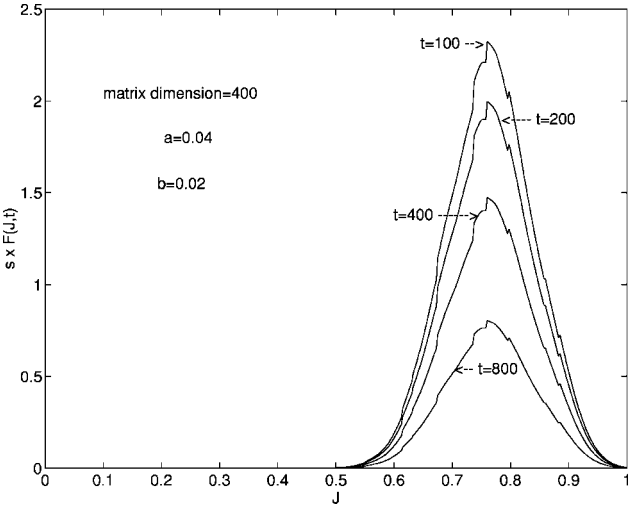

FIG. 1. Plots of the function $F(J,t)$ for $t=100$, 200, 400, and 800 against the survival probability J. In order to bring them to the same scale, they were multiplied by a factor $s=10^{11}$, 10^{23}, 10^{47}, and 10^{95}, respectively.

V. MONTE CARLO SIMULATION AND EXACT SOLUTION

To check out all these features, we also made some simulations of the model. Starting with a uniform distribution of the babies, we submit all of them to mutations as described in Eq. (1). For each baby, a random number ϵ is generated in the computer and its new survival probability J' is calculated. Then, playing the role of natural selection, a random number r is generated and compared with J'. If $r<J'$ then the baby becomes an individual of age 1 and produces m_1 offspring with inherited characteristics (that is, with the same survival probability J'). As the process continues, care should be taken in order to avoid an explosion of the computer's memory. To this end, we limited the number of individuals by a random decimation. Usually, in population dynamics simulations, this decimation is interpreted as a result of food restrictions [14]. Figure 2(a) shows that for $L=10$, $a=0.04$, and $b=0.02$, the final mean survival probability $\langle J\rangle=\lim_{t\to\infty}\langle J\rangle(t)$ approaches 0.78 in very good agreement with our numerical results. For $a=0.02$ and $b=0.02$ we find $\langle J\rangle\sim 0.96$. The case $a=0.08$ [Fig. 2(c)] leads to extinction, as was predicted.

As another important check, let us look at the Euler-Lotka

TABLE I. The mean survival probability $\langle J\rangle$, obtained by using our matricial formalism, as a function of the matrix dimension Q and time t for $L=10$, $a=0.04$, and $b=0.02$.

$Q\backslash t$	50	100	200	400	800
50	0.539 056	0.508 864	0.503 227	0.501 411	0.500 663
100	0.692 779	0.607 288	0.577 722	0.575 042	0.575 013
200	0.751 139	0.736 371	0.734 567	0.734 534	0.734 534
400	0.771 775	0.765 403	0.765 341	0.765 341	0.765 341

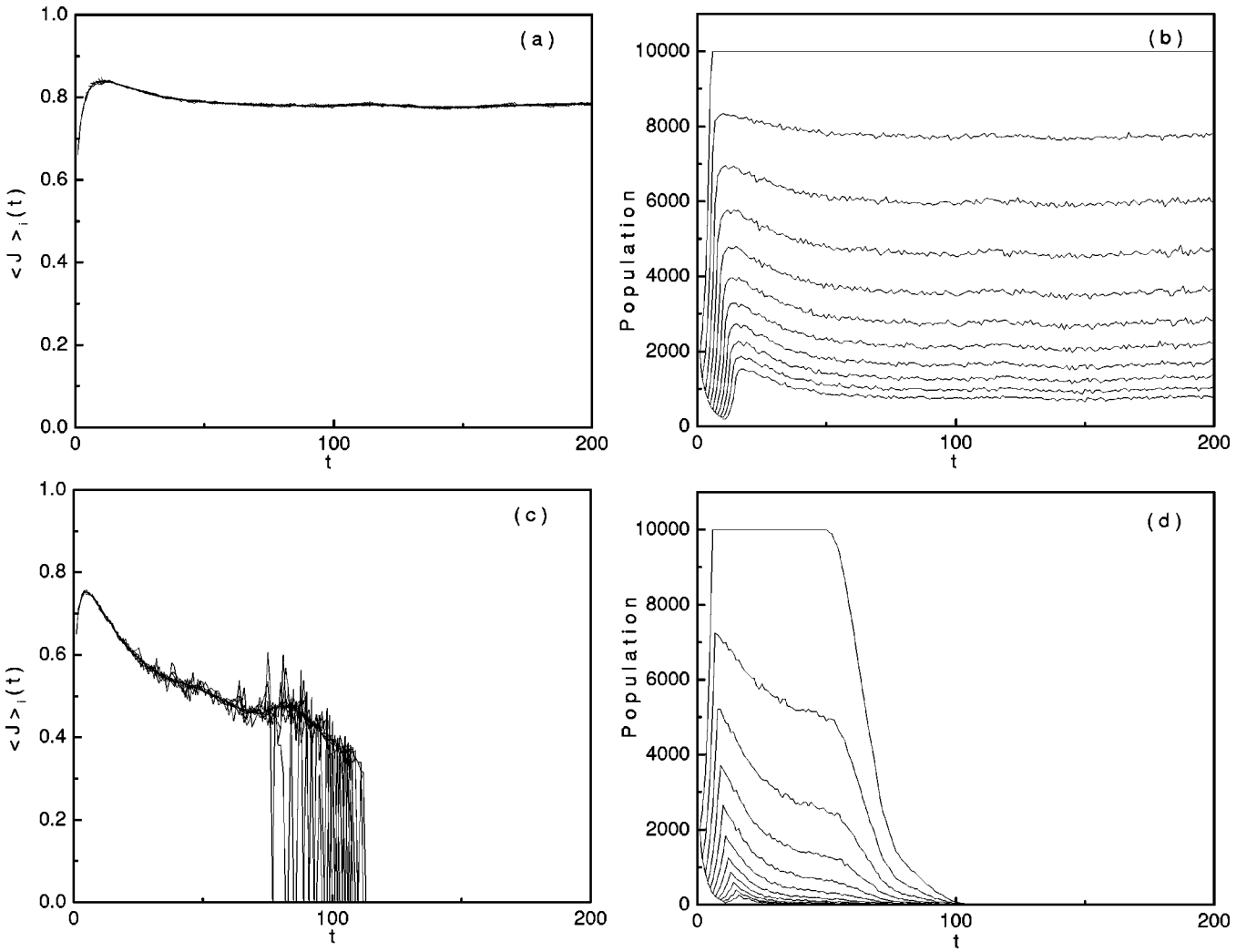

FIG. 2. (a) Time evolution of the simulated values of $\langle J\rangle(t)$ for $L=10$, $a=0.04$, and $b=0.02$. The 11 age curves coincide and are indistinguishable; (b) the corresponding population plotted against the time. The stationary behavior is an artifact of the decimation process; (c) the same for $L=10$, $a=0.08$, and $b=0.02$; (d) the corresponding population comes to extinction.

Eq. [3] which, for our model, reads

$$\sum_{i=1}^{L} m_i \ (\langle J\rangle \ e^{-r})^i, \tag{12}$$

with r being the Malthusian growth exponent.

Substituting the simulated values $\langle J\rangle=0.78$ and $\langle J\rangle=0.96$ into the Euler-Lotka equation, we obtain the Malthusian growth exponents $r=0.44$ and $r=0.65$, respectively, which are in fairly good agreement with those of our numerical prediction.

Incidentally, Eqs. (11) and (7) can be consistently used to guide us to an analytical solution of the stationary $\overline{F(J)}$. One can write the integral equation

$$c \ \overline{F}(J')=\int_0^1 f(J',J) \ \overline{F}(J) \ dJ. \tag{13}$$

Integrating the right side and expanding the result in a Fourier series, Eq. (13) turns out to be a set of linear equations for the Fourier coefficients. In order to have nontrivial solutions, this set must have null determinant. The condition of zero determinant allows us to obtain the constant c. For example, if $a=0.04$ and $b=0.02$ we find $c=0.75$, in good accordance with the numerical results. The Fourier coefficients have complicated expressions so we will not give them.

VI. DISCUSSION

Let us discuss the relevance of our model by comparing it with other evolutionary models. The Penna model is certainly the most intensively investigated [15]. It exhibits aging and sometimes catastrophic senescence. Contrary to what happens in our case, in the Penna model only babies are affected by mutations. Moreover, mutation plays the role of a programmed death—individuals which have accumulated a number of mutations (i.e., number of 1's in the bit string) larger than a threshold T die. This fate can be anticipated only if the individual dies by food restrictions (Verhulst factor, which acts irrespective of individual fitness). In our model there is not such a threshold and individuals may live longer. Besides that, natural selection here operates in a very hard (and explicit) way to eliminate those individuals who have suffered bad mutations. Also exact solutions have been

found for the Penna model [16]. The evolution equations directly involve the Verhulst factor. We have similar equations [Eqs. (3) and (4)], but with the survival probability J instead. Amazingly, the Leslie matrices were also found in the Penna model but in a different context. There, the elements of these matrices are connected to the mutation rate while in our model they are associated to the birth rate.

As we said in the beginning, there are two kinds of evolutionary theories: optimal and mutational. Our model belongs to the latter. The optimality theory is based on the fact that some genes have antagonistic effects, that is, they can be very beneficial early in life but deleterious late in life. For example, genes enhancing early survival by promoting a bone hardening might reduce later survival by promoting arterial hardening. These ideas were completely embodied by the Partridge-Barton model [7]. Further studies on this model have incorporated somatic as well as hereditary mutations [17,18], leaving the model with two mechanisms of senescence: antagonistic pleiotropy and accumulation of bad mutations. But aging (due to mutations) emerges in these works as a result of turning more intense with age (in some artificial or arbitrary way) the mutational strength. Their procedures would be equivalent to assuming, in our model, that the ϵ [the mutational control parameter of Eq. (1)] is a function of the age i. Clearly, this would also trigger an aging process in our model.

More interesting, however, is a different version of the Partridge-Barton model which is called the Vollmar-Dasgupta [19] model. It was generalized by Heumann and Hötzel [20] to support many age intervals. In this model, each individual carries, like its genome, a whole set of *independent* survival probabilities $\{J_0,\ldots,J_L\}$ where J_i $(i=0,\ldots,L)$ is the *actual* survival probability at age i. Deleterious mutations can now affect any of them but only those coincident with the actual age i will pass through natural selection. This means that most of the damage will only manifest later on. This accumulation of harmful mutations leads to senescence. Our model differs from Heumann and Hötzel only in the point that our individuals carry just one survival probability—that of its actual age. This is sufficient to radically change the results.

In summary, although containing all the relevant features of evolutionary systems like age structure, advantageous or deleterious mutations, reproduction with inherited characteristics, and natural selection, our model does not show senescence. In this way, it is a good candidate to appropriately describe some coelenterate and prokaryote groups, since all of them appear to lack senescence. On the other hand, the analytical solution that we find and the techniques involved encourage us to look forward to new and more sophisticated models.

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