Numerical solution of the Penna model of biological aging with age-modified mutation rate

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In this paper we present results of numerical calculation of the Penna bit-string model of biological aging, modified for the case of a-dependent mutation rate $m(a)$, where a is the parent's age. The mutation rate $m(a)$ is the probability per bit of an extra bad mutation introduced in offspring inherited genome. We assume that $m(a)$ increases with age a. As compared with the reference case of the standard Penna model based on a *constant* mutation rate m, the dynamics of the population growth shows distinct changes in age distribution of the population. Here we concentrate on mortality $q(a)$, a fraction of items eliminated from the population when we go from age (a) to $(a+1)$ in simulated transition from time (t) to next time $(t+1)$. The experimentally observed $q(a)$ dependence essentially follows the Gompertz exponential law for a above the minimum reproduction age. Deviation from the Gompertz law is however observed for the very old items, close to the maximal age. This effect may also result from an increase in mutation rate m with age a discussed in this paper. The numerical calculations are based on analytical solution of the Penna model, presented in a series of papers by Coe *et al.* [J. B. Coe, Y. Mao, and M. E. Cates, Phys. Rev. Lett. **89**, 288103 (2002)]. Results of the numerical calculations are supported by the data obtained from computer simulation based on the solution by Coe *et al.*

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I. INTRODUCTION

Biological aging means that older individuals have higher mortality rate than the younger ones. In the simplest description, population dynamics may be described in terms of number of individuals $n(a,t)$ at age a and at time t. The sum over a yields the current population $n(t)$,

$$n(t) = \sum_a n(a,t). \tag{1}$$

Usually we are interested in the equilibrium state, $n(a) = n(a,t \to \infty)$. Mortality $q(a)$ is defined as the percentage of items eliminated when we go to the next time step: $t \to (t+1)$, $a \to (a+1)$. We have

$$q(a) = 1 - n(a+1)/n(a) = \delta n(a)/na(a), \tag{2}$$

where $\delta n(a)$ is the number of items eliminated at age a. In the discrete time model, the transition from time (t) to the next time step $(t+1)$ results from the balance between death rate p and birthrate b. If p and b were the only model parameters (constants), then only unacceptable trivial cases would turn up either $n=0$ for $p>b$ or else we get unlimited population growth $n \to \infty$. The Verhulst factor [1] restores the balance within a finite population n. The basic idea is to replace the death rate parameter p with a suitable function of population n, so that p becomes larger for overpopulated habitat. In the simplest logistic model, the death rate p (known as the Verhulst factor) is assumed to be proportional the current population $n(t)$,

$$p(t) = n(t)/N. \tag{3}$$

At equilibrium, logistic model yields population n and mortality $q(a)$ as follows:

$$n/N = q(a) = b/(1+b), \tag{4}$$

which results in an age-independent mortality $q(a)=n$. This disagrees distinctly with demographic data that are supposed to follow roughly the Gompertz law of exponential increase in $q(a)$ with age a,

$$q(a) \propto e^{\alpha a}, \tag{5}$$

for a above a minimum reproduction age R. Therefore, it is necessary to include in the model some other elimination mechanisms to be consistent with the Gompertz law. Vast literature on biological aging indicates many possible factors that may contribute to the process of aging. Oxygen radicals, which may damage the genome, programmed cell death after certain number of cell divisions where telomeres are partly lost during each division, or mutation accumulation is often named as a possible reason for aging. For review of models, theories, and selected data on population evolution, biological aging, population speciation, and other aspects of modern concepts the reader is invited to consult, for example, Stauffer *et al.* [2].

In this paper we concentrate on the Penna model [3,4] of population evolution which belongs to mutation accumulation theory, the most popular foundation of biological aging concept. This model of biological aging yields results which basically agree with the Gompertz law. In asexual version of the Penna model, the genome of a newly born, represented by a computer word with bit value "1" for the bad mutation and "0" for no mutation, is inherited from parent. The baby's age $a=0$ and its genome are not just a copy of parent's—it

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may acquire at the moment of birth some extra "bad" mutations—each bit may randomly be set to one with probability m. In the evolution rule leading from time (t) to $(t+1)$, we count all 1's in the genome of the individual of age a, from bit position 0 to bit a on entry to the new era, marking them as *active*. The actual number of activated mutations $\mu(a)$ will be checked against some threshold value T of maximum number of bad mutations at which genetic death occurs. The above recipe is executed in computer experiment in the following sequence:

(1) get current Verhulst factor $p=n(t)/N$;

(2) scan the population and eliminate each item with probability p;

(3) if the item survives, check for $\mu(a)=T$ to apply genetic death;

(4) if the item is still alive, consider the offspring production with rate b, providing $a \geq R$, the minimum reproduction age, and

(5) the child's inherited genome that gets more bad mutations with probability m per bit; already mutated bits stay 1; finally,

(6) increase the item's age a by 1, which completes the $t \rightarrow t+1$ transition rule.

The essence of the Penna model is the mutation accumulation mechanism that results in a constant deterioration of the baby genomes. This may lead to population extinction unless the genetic death toll is compensated by sufficiently high birthrate b, above a critical value. A stable $n>0$ solution corresponds to some dynamic balance. In the standard Penna model, we have a set of input parameters (m,T,b,R,N), where m is the number of bad mutations per genome's bit passed over at the moment of birth to the baby, T is the threshold value for activated bad mutations, b is the reproduction rate, R is the minimum reproduction age, and N is the environmental capacity.

The outcome of calculations is the normalized population distribution $n(a)/N$ from which the mortality distribution $q(a)$ [Eq. (2)] may be extracted. We also get the overall mortality $q=\delta n/n$, with δn being the number of members eliminated at all ages. Mortality may be split into several components due to Verhulst factor and genetic death. Obviously the $m=0$ case yields no genetic death contribution with increasing mutation rate m; the genetic death contribution increases at the cost of lower death rate due to Verhulst factor. Relative contributions of the two elimination mechanisms are shown in Fig. 1. For a given $m>0$, an increase in b acts in the opposite way and, for larger b, the Verhulst component prevails as seen in Fig. 2. This is so since the larger birthrate that helps to create a new population before mutations that will occur during the next generation would destroy the population.

The above algorithm makes a direct *computer simulation* of the population evolution. Probabilistic character of the algorithm is responsible for the fluctuations in $n(a)$, so to get more accurate numbers we need big enough population on order of 10^6. Also, the number of iterations must be sufficiently large to reach a stable solution, usually 10^4 iterations. (Yet, in some aspects of population structure it is necessary to execute a seriously greater number of iterations. This results from different characteristic time scales for different quantities—to get a total population $n(t)$ at equilibrium, we need only 10^3 iterations; for mutation distribution among older individuals or to see speciation effect we need 10^6 or so time steps.)

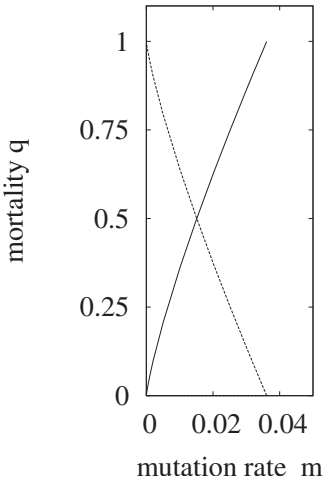

FIG. 1. Normalized mortalities q due to mutations (increasing line) and Verhulst factor (decreasing line) as functions of mutation rate m for $b=0.1$, $T=1$, and $R=0$.

However, the observed deviations in demographic data of calculated mortality $q(a)$ from the reference exponential Gompertz law are both important and interesting. A better fit is obtained with Makeham modification that adds a small constant to exponential increase. This improves the agreement for the younger items. In the opposite region of the oldest members, an anomalous plateau in late-life mortality can be often observed (see [5–7]). The standard Penna model is very flexible and it may easily be modified to include new ingredients for a better match with demographic data. Some inherited mutations may have positive effect in the youth and bad influence for old individuals—the antagonistic pleiotropy [8,9], which also may be incorporated into the Penna model. Threshold T parameter is sometimes modified to replace a sharp genetic death at certain age with a less deterministic scenario. In [10], fluctuations in T come from mutations already activated at birth time [7,11]; use a smooth probability trial function of genetic death. Sexual against asexual reproduction [4], effect of population migration [12],

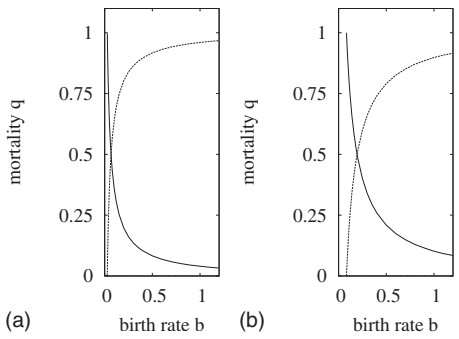

FIG. 2. Normalized mortality q due to mutations (decreasing lines) and Verhulst factor (increasing lines) as functions of birthrate b. The left figure is for mutation rate $m=0.01$; the right one is for $m=0.03$.

and tracing back to numerous species in the past and its severe reduction in the evolution process [13] are few examples of many implementations of the Penna model.

In this paper we intend to explore the model by assuming a specific $m(a)$ dependence in order to account for the effect that older individuals may produce less healthy children. The paper is organized as follows. Section II describes the Penna model with the modification proposed, numerical results are given in Sec. III, and Sec. IV contains the summary and conclusions.

II. MODEL

In the Introduction we described a direct computer simulation in the Penna model. Simpler asexual version that may be solved analytically is proposed by Coe *et al.* in a series of papers [11,14–16]. Population structure is given by a function $n(a,l,t)$—the number of individuals at age a of genome length l at time t. The genome length l is defined as the bit position with critical 1 for the deadly number T of bad mutations.

The main outline of their calculations, leading to iterative procedure for $n(a,l,t)$ with a self-consistency condition for stationary solution, is described below. The *analytical solution* of Coe *et al.* comes from the fact that we can reduce some sums (geometrical series) to elementary functions, provided that all model parameters (m,T,b,R,N) are constant. The same algorithm may also be applied if some of the parameters are not fixed, for example, when we modify bad mutation rate m as a function of parent's age a; in such case we estimate the values of relevant sums by means of computer summation. We refer to these results as *numerical solution*. In this paper we get full consistency of analytical and numerical solutions in trial runs when we reduce the proposed $m(a)=m(0)+sa$ to a constant m by putting slope $s=0$. As additional tests, also direct computer simulations were carried out.

In analytical solution, we consider contributions to the new population structure at time $t+1$, coming from the assumed population distribution $n(a,l,t)$ at time t. This is given by the following map:

(1) New

$$n(a+1,l',t+1)=n(a,l,t)\sigma, \quad l'=l, \tag{6}$$

where

$$\sigma=1-n(t)/N \tag{7}$$

is the survival rate as dictated by the Verhulst factor of a system with environmental capacity N and $n(t)$ is the total population on entry to the next evolution time step. This makes (a) Verhulst elimination to appear as the first action in each evolution step and (b) no mutations take place for grown-up items since we assumed $l'=l$ only. Age is being increased by 1. [At this stage we may expand the concept of environmental capacity N to $N(a,l)$ dependence, which we will not consider here.]

(2) Next we allow for the offsprings birth. In case of no mutations we have $n(0,l,t+1)=n(a,l,t)\sigma b$ for birthrate b. This means that the baby's genome has the same length as parent's since babies get genomes inherited from parents. However, for mutation rate $m>0$, we introduce $g(l')$ function which modifies $n(0,l,t+1)=n(a,l,t)\sigma b$ to

$$n(0,l',t+1)=n(a,l,t)\sigma b g(l') \tag{8}$$

to account for possible mutations imposed onto child's genome on top of the inherited one. Mutation-free case is recovered for $g(l')$ in the form of Kronecker delta, $g(l')=\delta_{l,l'}$. The approach of Coe *et al.* is equivalent to the proposed form of $g(l')$ as

(i) $g(l')=(1-m)^{l'}m$, for $l'<l$, which reflects the probability that the first l' bits, from bit 0 to bit $l'-1$, would not catch bad mutations and bit l' receives the bad 1;

(ii) $g(l')=(1-m)^{l'}\cdot 1$, for $l'=l$, which states that all bits from bit 0 to bit $l'=l$ get no mutations; and

(iii) $g(l')=0$, for $l'>l$, since child cannot have longer genome than parent's one.

[Note that m may be seen as a-dependent $m(a)$, as it is proposed in this paper; we also might complicate b as $b(a,l,N,T,\text{etc.})$, which, however, we did not try to do.]

The above procedure makes the map of transformation of the population structure $\{n(a,l,t)\}$ at time t to $\{n(a,l,t+1)\}$. Numerical solution is obtained as the above computer iterative procedure, leading to a stable solution after sufficiently big number of iterations. Analytical solution of Coe *et al.* is obtained as the fixed point of the above map for the case of constant parameters (m,T,b,R,N), for which sums of geometrical series are expressed explicitly.

To get analytical solution for $n(a,l)$, we start with an auxiliary $u(l)=n(0,l)$ distribution function of just-born babies of the assumed steady population, from which we may recover $n(a,l)$,

$$n(a,l)=u(l)\sigma^a, \tag{9}$$

where

$$\sigma=1-n/N \tag{10}$$

and

$$n=\sum_{a,l} n(a,l). \tag{11}$$

The recursive procedure [Eq. (12)] yields the $u(l)$ series with an arbitrary multiplication factor. This factor must be adjusted, so that when $u(l)$ is substituted to Eqs. (9) and (11), Eq. (10) is fulfilled,

$$u(l+1)/u(l)=\frac{\binom{l+1}{T-1}}{\binom{l}{T-1}}\frac{1-\sigma^{l+1}}{1-\sigma^{l}} \times\frac{e^{\beta(l+1-T)}-b\chi(\sigma,R,l)}{e^{\beta(l+2-T)}-b\chi(\sigma,R,l+1)(1-T+Te^{-\beta})}, \tag{12}$$

$$\chi(\sigma,R,l)=\frac{\sigma^R-\sigma^l}{1-\sigma}, \tag{13}$$

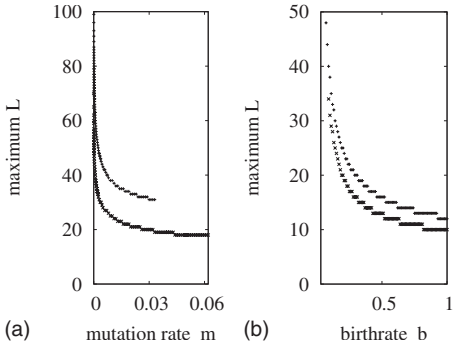

FIG. 3. Maximum genome length L as a function of mutation rate m. Figure on the left is for birth rates $b=0.1$ (upper line) and $b=0.2$ (lower line). The right figure shows L as a function of birth rate b for mutation rates $m=0.01$ (upper line) and $m=0.03$ (lower line). The plots are for $T=1$ and $R=2$.

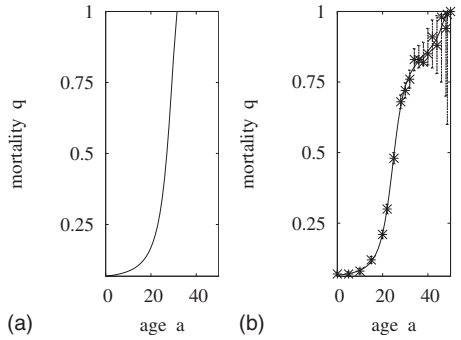

FIG. 4. Example of influence of age-modified mutation rate, $m(a)=m(0)+sa$, on mortality $q(a)$ for effective mutation rate $m_{eff}=0.01$ and birthrate $b=0.1$. The left figure is for standard Penna model $s=0$; the right figure is for $s=0.001$, where points with error bars are obtained from direct simulation.

$$e^{\beta} = 1/(1-m). \tag{14}$$

The sum in Eq. (11) runs over a from $a=0$ to $a=L$ and the maximum genome length L is given from recursive formula (12), so that numerator is still positive. The minimum genome length l must be T, so the sum over l runs from $l=T$ to $l=L$, with the maximum value depending on parameters m, T, b, and R (see Fig. 3). It is obvious that mutations reduce genome length in consecutive generations. This leads to shorter maximal genome length L for larger mutation rate m, and $m=0$ limit brings unlimited genome length. For a given mutation rate m per bit for a born item, a higher birthrate b results in a lower number of iteration steps necessary to produce a given number of babies and, therefore, lower number of mutations introduced to population. It may also be noticed that for $m>0$, L as a function of b starts from small, yet nonzero critical value of b necessary to compensate genetic deaths (see [17] for details).

The above analytical result of Coe *et al.* may be checked against numerical calculations described above if the model parameters are kept constant. In this paper, most of the calculations were done for single $T=1$ mutation threshold and minimum reproduction age $R=0$. We modified the mutation rate m per bit per time step as parent's age a-dependent value, $m(a)$, which increases with age a. This may be seen as an attempt to account for the known fact that older individuals give birth to less fit babies. In the model we may associate the bad mutation rate m with a. The simplest linear dependence of the form

$$m(a) = m(0) + sa \tag{15}$$

was adopted and different slopes $s>0$ were tried to examine the deviations of $q(a)$ from the case $s=0$. Thus we replace single parameter m in the standard approach $s=0$ with a set of two $(m(0),s)$. However, for each choice of s we adjust the mutation rate $m(0)$ of the new born items, so that we recover the same total population n. (It seems reasonable to make choice of any alternative set of model parameters in a way that leads to the same population if we intend to compare results of two different sets of model parameters.) In other words, we may use an effective mutation rate m_{eff} that replaces m in the reference $s=0$, yet producing the same population n. So, the two sets of parameters $(m(0),s)$ and $(m_{eff},0)$ give the same n. Assumption of linear dependence of $m(a)$ has already been applied in [18] where the mutation rate was simplified to $m(0)=0$, $m(a)=sa$. This form of $m(a)$ dependence, however, does not keep n fixed and their conclusions may be different quantitatively from results of this paper. Berntsen's calculations [18] were based on computer simulations.

In our calculations we replace the set of the Penna model parameters (m,T,b,R,N) with $(m(0),s,T,b,R,N)$. We restrict our attention to values of $T=1$ and $R=0$. We scan $m_{eff}=0,0.01,0.03$ and $s=0,0.001,0.002$. The limiting case $[m(0)=0,\ s=0]$ stands for the reference logistic model. Two birthrate values $b=0.1$ and $b=0.2$ were used.

As far as analysis of the results of calculations is concerned, we concentrate on normalized population n/N, maximum genome length L, and mortality distribution function $q(a)$. We also split the death rate into its components due to genetic death resulting from the bad mutations and from the Verhulst factor responsible for the limited environmental capacity.

III. RESULTS

As we mentioned earlier, calculations were done for $T=1$, $R=0$, $m_{eff}=0,0.01,0.03$, $s=0,0.001,0.002$, and $b=0.1,0.2$. The computer simulation part was carried out for environmental capacity $N=10^7$ $(10\,000+1121)$ iterations for which population distribution reaches equilibrium, apart from statistical fluctuations. The effect of fluctuations is reduced by taking averages over last 1121 steps.

For the assumed age dependence of the mutation rate, $m(a)=m(0)+sa$, one gets the normalized steady-state population $n/N<1$ as a function of $(m(0),b)$ which, by definition, may be represented by an effective m_{eff} with the same n as for the set $(m_{eff},0)$. Checking whether mortality $q(a)=1-n(a+1)/n(a)$ follows the exponential Gompertz law dependence for $R<a<L$ is usually considered to be a good evaluation of the validity of the model for population dynamics. As it was mentioned earlier, important conclusion from

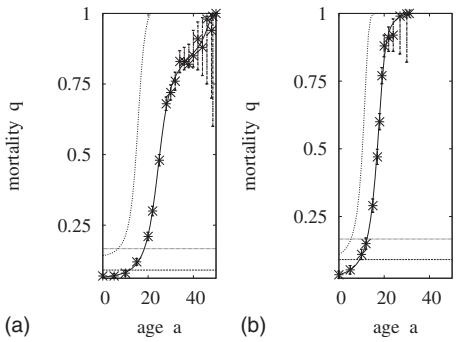

FIG. 5. Example of changes in mortality $q(a)$ for different birthrates: $b=0.1$ of bigger maximum genome length L and $b=0.2$ of smaller L. Horizontal lines refer to logistic cases. The left figure is for effective mutation rate $m_{eff}=0.01$ and $s=0.001$; the right one is for $m_{eff}=0.03$ and $s=0.002$. Horizontal lines refer to logistic cases $m_{eff}=0$ with $q=b/(1+b)$ and $L\to\infty$. Points with error bars represent direct simulation results.

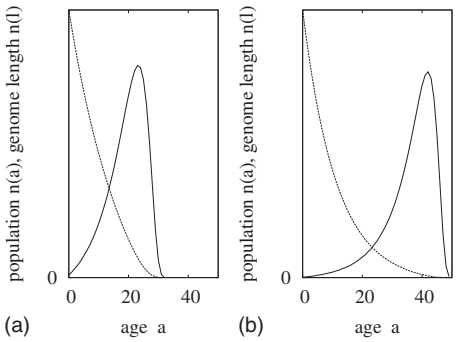

FIG. 6. Influence of age-modified mutation rate $s>0$ on population distribution $n(a)$, declining lines, and genome length (lines with maximum) $n(l)$ for effective mutation rate $m_{eff}=0.01$ and birthrate $b=0.1$. The left figure is for standard Penna model $s=0$; the right figure is for $s=0.001$.

the standard Penna model is that essentially it meets this expectation. The observed mortality $q(a)$, however, shows some deviations from the Gompertz law for the oldest members.

In Fig. 4 we compare mortality $q(a)$ for $s=0$ and $s>0$ for which mutation rate is larger for older items. It can be seen that serious changes in mortality distribution $q(a)$ take place for $s>0$. Solid lines are obtained from numerical calculations based on the exact analytical solution of Coe *et al.* executed here as an iterative self-consistent solution of the map [Eqs. (6)–(8)]. Points with error bars on the right plot come from direct simulations. As the simulation is nondeterministic, the numbers fluctuate. The errors for population $n(a)$ at age a were estimated as $1/\sqrt{n(a)}$ and confirmed by magnitude of fluctuations of results for mortality $q(a)$ in several runs. Less precise agreement between crosses and the solid line for larger a is due to poorer statistics as population at old ages $n(a)$ is greatly reduced, leading to larger fluctuations in $q(a)$.

Another example is given in Fig. 5. The deviation of mortality distribution $q(a)$ from the standard Penna model due to $s>0$ is rather small for higher birthrate b and more visible for higher mutation rate m_{eff}. Also population distribution characteristics $n(a)$ and $n(l)$ in Fig. 6 are strongly influenced by $s>0$. For example, shift of maximum of $n(l)$ for larger values comes from the fact that $s>0$ brings smaller $m(a)$ for younger members of the population, which are in majority. Therefore smaller m must produce longer genomes. For the oldest members, say close to maximum of $n(l)$ around $a=40$, we get $sa=0.04$ which gives about four times higher mutation rate in offsprings produced by parents in this age group of population members—and so in this group we observe the most serious changes in distribution functions.

In Tables I and II we summarize results reflecting some population characteristics: normalized population n [and corresponding $n(sim)$ population from simulation], maximum genome length L, or percentage of genetic death rate *mut* due to mutation against the Verhulst death rate *vrh* from limited environmental capacity. Population estimates from numerical calculation and simulation agree. Obviously, the population is larger when b grows, and it decreases with increasing mutation rate. The change in slope from $s=0$ to $s>0$ pushes maximum genome length L to larger values, yet the ratio of genetic to Verhulst death rate remains intact. This ratio, however, shows the same tendency as in the case of the standard Penna model $s=0$ (Fig. 2 in the Introduction) if we change b or mutation rate m_{eff}.

As it may be expected, with increasing mutation rate, the genetic death becomes dominant and it decreases for bigger b. This results from the fact that the increase in birthrates, when population grows, leads to higher death rate from limited environmental capacity.

IV. SUMMARY AND CONCLUSIONS

The aim of this paper was to see how the possible increase in the bad mutation rate of babies born by older par-

TABLE I. Population n, genome length L, and mortality ($mut+vrh$) for birthrate $b=0.1$.

b	$m(0)$	s		n	$n(sim)$	L	$mut+vrh$	Remarks
0.1	0.0000	0.000	→	0.0909	0.0917	∞	0.000+1.000	Logistic case
0.1	0.0100	0.000	→	0.0582	0.0594	32	0.359+0.641	Standard
0.1	0.0010	0.001	→	0.0582	0.0597	49	0.359+0.641	$p>0$
0.1	0.0000	0.002	→	0.0000	0.0000			No solution
0.1	0.0300	0.000	→	0.0125	0.0147	27	0.863+0.137	Standard
0.1	0.0114	0.002	→	0.0125	0.0118	32	0.863+0.137	$p>0$

TABLE II. Population n, genome length L, and mortality ($mut+vrh$) for birthrate $b=0.2$.

b	$m(0)$	s		n	$n(sim)$	L	$mut+vrh$	Remarks
0.2	0.0000	0.000	→	0.1667	0.1681	∞	0.000+1.000	Logistic case
0.2	0.0100	0.000	→	0.1338	0.1355	19	0.197+0.803	Standard
0.2	0.0009	0.002	→	0.1338	0.1350	30	0.197+0.803	$p>0$
0.2	0.0300	0.000	→	0.0855	0.0876	15	0.487+0.513	Standard
0.2	0.0202	0.002	→	0.0855	0.0862	17	0.487+0.513	$p>0$

ents may account for the reported changes in mortality distribution $q(a)$ of the oldest individuals. The proposed increase in mutation rate $m(a)$ with parent's age a offers possible explanation. The deviation of calculated $q(a)$ from the Gompertz law shows the right tendency, and the degree of the deviation from negligible to quite noticeable one depends on the model parameters. The slope $s>0$ also changes the proportion of genetic deaths in the population. We conclude that the proposed modification, perhaps well grounded from the point of view of the known fact that old parents may have less healthy children, seems to be a good direction for future studies of the anomalies in the Gompertz law. The Penna $s=0$ model does not reflect observed deviations of $q(a)$ from the Gompertz law for the oldest population members; the $s>0$ modification brings possible explanation of the changes in $q(a)$, especially for higher ages a.

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