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A Bit-String Model for Biological Aging

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We present a simple model for biological aging. We study it through computer simulations and fint it to reflect some features of real populations.

KEY WORDS: Aging, Monte Carlo simulations.

The problem of aging has been studied recently[1] by computer simulations to understand how the survival rates for younger and older individuals evolve in time and affect the preservation of the species.[2-4] The lowering of survival rates as time goes by is called senescence. Mutations play an important role in senescence, modifying the survival rates, either increasing them (helpful mutations) or decreasing them (deleterious mutations). In this paper we introduce a simple model for aging using the so-called "bit string strategy." This approach has been applied to other biological systems.[5] Our model is particularly suited for implementation in computers, although analytical results may be obtained providing some approximations. Since it is based on Boolean variables, bit-handling techniques have been used.[6] A complete description of our code will be presented elsewhere.[7]

Let us consider a population of $N(t)$ individuals at the time t, each one characterized by a genome which contains the information on when a mutation will occur in the lifetime of a given individual. We consider the time as a discrete variable ($t = 1, 2, ..., B$) as suited for implementation on computers. Herrmann and Hötzel[8] simulated the largest number of ages simulated up to now (five ages). We denote each time step as one generation, since birhts can occur at each time step. Here, we will treat only hereditary mutations, although somatic mutations can be incorporated

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without great additional effort. Hence, a genome is a string of B bits define at birth and kept unchanged during the individual lifetime. The genome is built as follows: if an individual has the ith bit in genome set to one, it will suffer a deleterious mutation at age i. The parameter T represents a threshold, i.e., the maximum number of deleterious mutations that an individual can suffer and stay alive. In order to include the effect of food and space restrictions and to keep the population within the computer memory limits, we imposed the age-independent Verhulst factor. Hence, an individual who has passed the threshold test only stays alive with a probability $[1 - N(t)/N_{\max}]$. The next step is birth: an individual whose age is larger than the reproduction age R generates one baby. As far as we know, this is the first model for biological aging where the reproduction age appears as a parameter. Although sex is not a difficulty for us or for our model, we chose to work with asexual populations, for the sake of simplicity—sexual reproduction can be introduced, for example, by mixing the bit strings of two individuals (crossing over). Thus, the baby's genome will be the same as the parent, except by a fraction M of randomly changed bits (mutation rate, hereafter). As these new mutations are made randomly, a baby can suffer either a helpful mutation (flipping a bit set to one in the

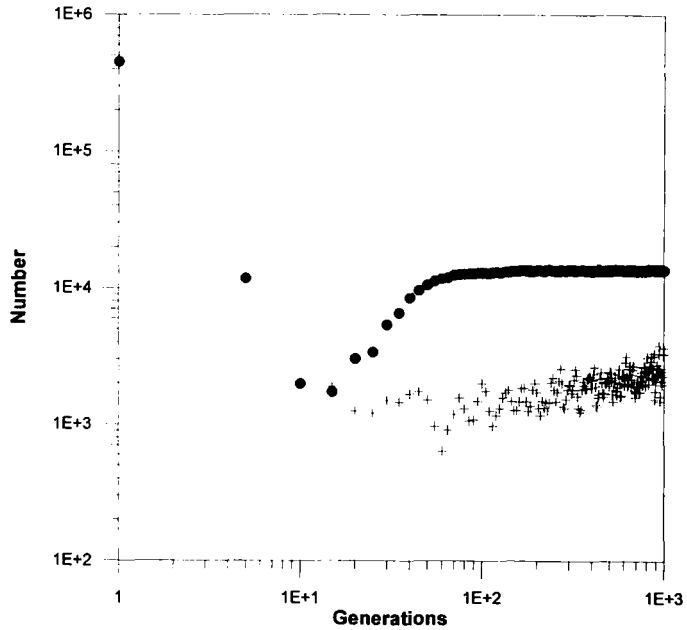

Fig. 1. Number of individuals versus generations for $M = 0$ (+) and $M = 2$ (●), starting from 450, 000 individuals. Mutations cause the equilibrium state to be reached faster.

parent's genome) or a deleterious one (flipping a bit set to zero in the parent's genome). In summary, we have the following parameters in this model: the genome size B, the threshold T, the maximum number of individuals in the population N_{max}, the mutation rte M, and the minimum age at reproduction R.

After the presentation of the model we are ready to show some computer simulation results. First of all, it is important to know whether the proposed dynamics leads to stationary states. Figure 1 shows results for the time evolution for $N_{max} = 10^5$, $T = 2$, $R = 8$, and $M = 0, 2$. The population first decreases rapidly and grows again, as a signature of the Darwinistic selection. As can be expected, the mutation rate controls the relaxation time: the larger the mutation rate, the lower the number of generations to reach an equilibrium state. Figure 2 shows the distribution by ages in the population for $T = 4$, $M = 1$, and $R = 2, 4, 6$. The aging can be noted since the frequency decreases stronger than exponentially with increasing age. This decrease is more noticeable for large ages than for small ones. In Fig. 2 we also can note that the larger the minimum age at reproduction, the larger the frequency of old ages. Old readers should not celebrate this comment before checking the results presented in Fig. 3. The average age

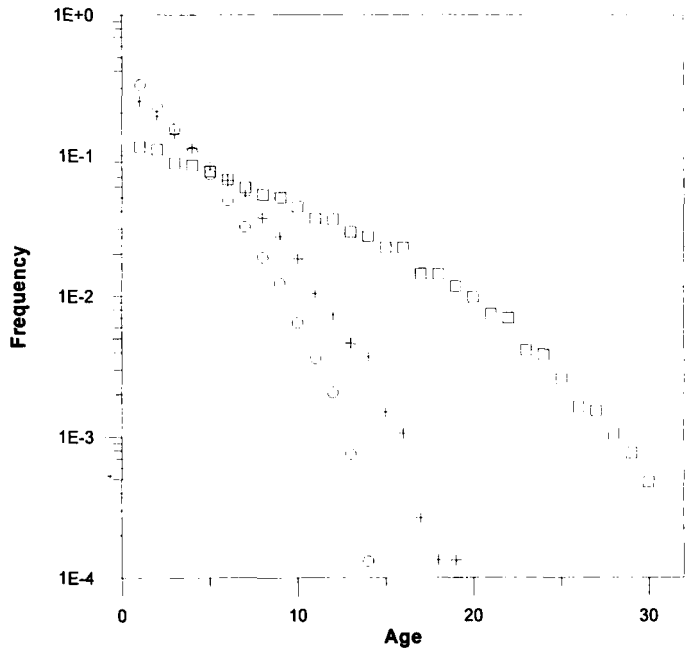

Fig. 2. Frequency of each age in a population for $R = 2$ (○), 4 (+), and 8 (□).

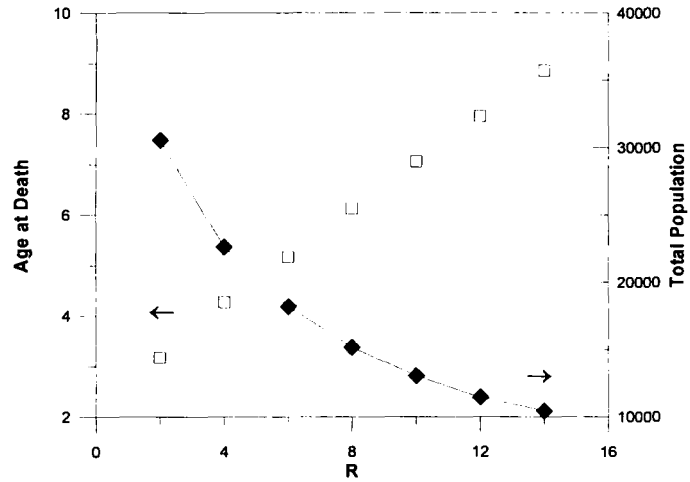

Fig. 3. Age at death (□) and total population (◇) as a function of the minimum age at reproduction R, for $M=2$ and $T=4$.

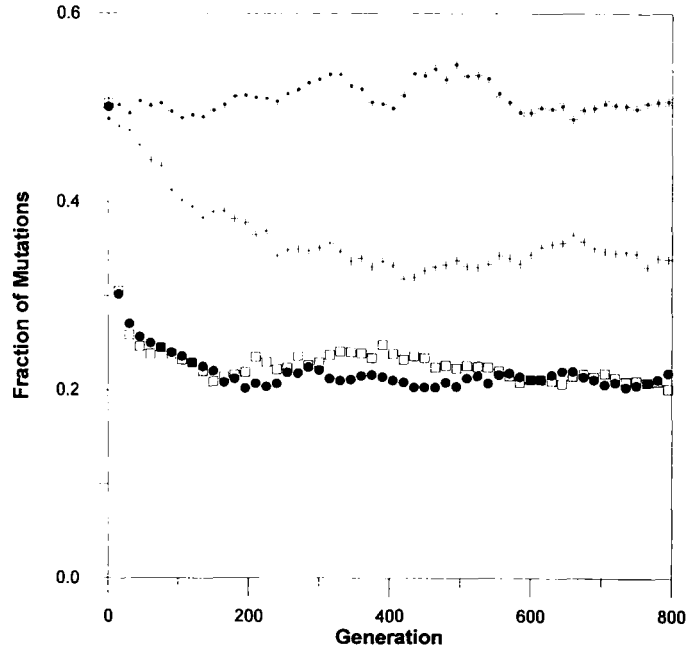

Fig. 4. Evolution in time of the fraction of individuals suffering mutations at ages 1 (●), 6 (□), 12 (+), and 30 (*). The data correspond to $R=6$, $M=2$, and $T=4$.

at death decreases as soon as the age at reproduction increases; consequently the total population decreases. This behavior has been found in *Physella virgata virgata* snail populations.[9] There, the age of reproduction is controlled by the presence of *Orconectes virilis* crayfish. In our model it is imposed as an additional parameter. It is worth stressing here it is the first model—to our knowledge—where the maturity age is introduced as a relevant parameter.

We can check the evolutive pressure in aging through the present model by measuring the frequency of deleterious mutation at each age. Starting from an uniform distribution, Fig. 4 shows the evolution of the frequency of bad mutations. The evolution pressure is also larger for smaller minimum age at reproduction. Note that the pressure is visible even at ages beyong the minimum age. Therefore, the weak evolutive pressure on aging is also reproducible in the present model.

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