Penna bit-string model with constant population

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Abstract: We removed from the Penna model for biological ageing any random killing Verhulst factor. Deaths are due only to genetic diseases and the population size is fixed, instead of fluctuating around some constant value. We show that these modifications give qualitatively the same results obtained in an earlier paper, where the random killings (used to avoid an exponential increase of the population) were applied only to newborns.

Keywords: Biological ageing, Monte Carlo Simulations.

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1 Introduction

The most successful computational model for age-structured populations is by far the Penna model [1]. It’s biological support comes from the mutation accumulation theory of senescence [2, 3]. In essence, this theory relates the evolution of senescence to the action of age-specific deleterious mutant alleles and the maintenance of some of these genes by the combined effects of mutation and selection pressures [4].

One of the reasons for the Penna model’s success relies on a particularly well-suited computational representation of a genome by means of a sequence of bits, the bit-string. When grouped into computer words these strings can be efficiently operated on by very fast logical and bitwise CPU operations. It has also proved to be flexible enough to be of value in a number of different problems in population dynamics, running from the catastrophic senescence of salmon to the origins of menopause (for a review we address the reader to references [5, 6, 7]; the first one contains also a fortran program to implement the model).
In order to avoid an exponential increase of the population, the Penna model makes use of the so-called Verhulst factor. It is a logistic-type term that introduces a mean-field random death probability, independent of the quality of the genome. Its usual expression is \( V(Pop) = \frac{Pop}{Pop_{max}} \), where \( Pop \) is the total population at some time step and \( Pop_{max} \) is a parameter of the simulation, traditionally called the carrying capacity of the environment. Since really random deaths in nature can hardly play any significant role in population dynamics, this concept has already been criticized in the literature \cite{8, 9}. Here we modify the model in such a way that individuals now die only for genetic reasons. Moreover, the population size is fixed, without fluctuations, as the biologists usually prefer.

2 Model with constant population

We start the simulation with \( N \) individuals, each one represented by a chronological genome. Each of these genomes is reduced to the 32 bits of one computer word, where each bit represents a life-threatening inherited disease. The lifespan is divided into 32 intervals, each corresponding to one bit position. A zero bit means health; a bit set to one means that starting from that age interval until death, one additional disease is diminishing the individual’s health. \( T \) such diseases, i.e. \( T \) bits set to one in the bitstring from age zero to the current age, kill the individual. Those who survive up to age \( R \), the minimum reproductive age, may get offspring, from then on. Each offspring differs in \( m \) randomly selected bit positions from the parent. As usual, we consider only bad mutations which means that if a position is selected which has already its bit set to one, then it remains one in the offspring genome.

The reason why we underlined the word “may” in the above paragraph is because at that point we start to modify the standard model. Now as soon as an individual dies, either for reaching the age 32 or the limit \( T \) for allowed diseases, a random individual is chosen, among all those with age \( \geq R \), to generate an offspring. In this way we keep the population size constant, without using the Verhulst factor already mentioned. This strategy can be interpreted as if, at each iteration, we have been choosing a finite sample of an infinite population to observe. In the next section we present our results, which are qualitatively the same as those obtained earlier by Sá Martins and Cebrat \cite{9}. However, in their paper they still keep a Verhulst factor acting over the newborns. For this reason their population size fluctuates a lot at
each iteration.

3 Results

In the figures below we compare the results of the Penna model at constant population size with those obtained with the standard model. In order to have nearly the same population sizes, we have used for the standard model a carrying capacity $Pop_{\text{max}} = 3,000,000$ individuals, starting with $P_0 = 50,000$ individuals. With these parameters the population size of the standard model fluctuates around 360,000 individuals, after equilibration, while in the constant population model it is fixed to 300,000 since the beginning of the simulations. The rest of the parameters are the same for both: threshold for genetic diseases $T = 1$, minimum reproduction age $R = 8$, mutation rate of newborns $m = 1$, total number of 1,000,000 timesteps with averages taken over the last 10,000.

In figure 1 we show the average fraction of individuals per age. As obtained in [9], the overall concavity of the constant population curve is different from that of the standard one. Also, the maximum life span, the average age of an individual and the fraction of population with reproductive live (age above 8) are larger for the present model.

In figure 2 we present the fraction of defected genes (bits 1) for each locus in the genome (or age). As expected from the results of the previous figure, the fixation of deleterious mutations in the whole population genomes starts earlier with the standard model, causing a decrease of the maximum life span, when compared to the constant population model.

Figure 3 shows the mortality functions, $q(a)$, defined as:

$$q(a) = -\ln \left[1 - \frac{D_a}{N_a}\right].$$

$N_a$ is the number of individuals with age $a$ and $D_a$ is the number of genetic deaths at age $a$ (which means, discounting the deaths provoked by the Verhulst factor in the standard model). It can be seen that the mortality above the minimum reproduction age increases faster in the standard model than in the present one. However, the standard model seems to give a better
agreement with the Gompertz law [10], that predicts an exponential increase of the mortality above adult ages.

We have also measured the genetic diversity of the populations, computing the difference between each pair of genomes (Hamming distance). The diversity is higher for the constant population model, as also obtained in [9].

Real species, of course, can become extinct. Even within a constant population approach, by choosing an unrealistic set of parameters (for instance a high mutation rate), we observed mutational meltdown through a sudden explosion of the birth rate.

Figure 1: Fraction of individuals per age. Circles correspond to the constant population model and triangles to the standard one. Parameters are described in the text.
Figure 2: Fraction of deleterious alleles (bits set to one) as a function of age.

4 Conclusions

We removed from the Penna ageing model any random killing Verhulst factor and fixed the population size instead of allowing it to fluctuate around some constant value. We observe the same differences between this model and the standard one as those pointed out by Sá Martins and Cebrat: a longer life span, the fixation of deleterious alleles starting at older ages and a higher genetic diversity (not shown). However, we don’t have any fluctuation in the population size at each iteration, as they do, once we have a fixed number of individuals since the beginning of the simulations. The present model gives a mortality function that increases, for ages above the minimum reproduction age, in a slower way than that of the traditional Penna model. However, the
standard model seems to give a better agreement with the Gompertz law of mortality.

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