

# Evolution, Ageing and Speciation: Monte Carlo Simulations of Biological Systems

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We present a complete description of the Penna bit-string model for biological ageing and how it has been modified, along the last 10 years, to simulate and better understand many different evolutionary phenomena. Particularly, we show how a phenotype was included into the model in order to study speciation and correlated problems.

## 1 Introduction

According to Luca Peliti [1], models to explain the origins of life and its evolution can be divided in three groups: i) Models for microevolution - individuals belong to the same species or to closed ones. Interaction among individuals is generally introduced through some global competition mechanism; ii) Models for coevolution - two or more species interact strongly in such a way that the survival of one species depends on the survival of the other; iii) Models for macroevolution - also called large-scale models for evolution. They deal with all alive species at the same time, but with no particular interacting mechanism between them.

One of the pioneer models for microevolution was proposed by M. Eigen [2] as an attempt to explain the origins of life in Earth. It describes the dynamics of biological macromolecules (that can replicate) under the influence of selection and mutation mechanisms. The macromolecules can be represented by bit-strings of zeroes and ones, each one with a given replication rate. Its main results are: without competition there is segregation among the macromolecules and those that present a replication rate higher than its death rate increase exponentially, while the others disappear. If a global competition mechanism is introduced (the total number of macromolecules is forced to stay constant) then selection becomes active and only the macromolecule species with the maximum replication rate or fitness, called the master sequence, survives. When mutations are then included, different quasi-species coevolve in a fitness landscape where the initially best fitted master sequence occupies now one of the possible maxima of this landscape, surrounded by the other quasi-species. When the mutation rate surpasses a given threshold, however, selection disappears and all sequences become equally probable. The main conclusion obtained from this simple model is that without errors (mutations) or with too much errors, there is no evolution. In the first case there are no mutants and in the second case, there is no adaptation. We direct the readers interested in this model

to [3] and references therein.

Concerning models for macroevolution, may be the most popular one is the Bak-Sneppen model [4]. In this model each species occupies a site on a linear chain (ring) and is represented by its fitness, a number between zero and one. At every iteration the worst fitted species and its two neighbours disappear (or mutate) and are replaced by three new ones, randomly chosen. Observe that there is no specific interaction mechanism between the species: the most fitted one may disappear just because one of its neighbours happened to be a poorly-fitted species. In this context, the fitness landscape is continuously evolving, as well as the species, in order to stay always close to the peaks. This system converges to a situation where all species have fitness above a given value, except for those situated just on the border line and that may participate on occasional avalanches. Those particularly interested in this model and its properties can find a complete description and results in [5].

Instead of mentioning any model for coevolution, now we are going to describe the Penna model for biological ageing [6]. It is an extremely versatile model of microevolution, and coevolution will appear as a successful application of this model to explain the maintenance of sexual reproduction.

## 2 The asexual version of the Penna model

The reasons for ageing are controversial [7] (see also the whole special issues of *La Recherche*: July/August 1999 and *Nature*: November 9th, 2000). There may be exactly one gene for longevity, or senescence comes from wear and tear like for insect wings and athlete's limbs, from programmed cell death (apoptosis [8]), from metabolic oxygen radicals destroying the DNA [9], or from mutation accumulation [10]. The Penna model reviewed here use this last assumption, which does not exclude all the other reasons. For exam-

ple, the oxygen radicals may produce the mutations which then accumulate in the genome transmitted from one generation to the next. The concept behind the mutation accumulation theory is that a mutation endangering the life of an individual below the reproductive age reduces the number of offspring much more than a mutation affecting it only late in life, when it barely gets any descendents and has already accomplished its evolutionary mission of perpetuating the species. In this way, a very important ingredient of such a theory is the existence of a minimum reproduction age below which there is no breeding.

In the asexual version of the Penna model individuals are represented by a chronological genome that consists of a bit-string of 32 bits (zeroes and ones). Whenever a bit 1 appears at a given position (age) it means that the individual will start to suffer the effects of a genetic disease from that age until the end of its life. The age can be measured in years, days or any other time interval, depending on the species. Here we will arbitrarily call “year” our time unit, which means that each individual can live at most for 32 years. Each individual may accumulate  $T - 1$  diseases, where  $T$  is known as the threshold for bad mutations. Considering the bit-string 100101...11 as an example for a chronological genome, the individual carrying it would die at age 4 for  $T = 2$  and at age 6 for  $T = 3$  (reading the bit-string from the left to the right). There is also a dispute for food and space given by the logistic Verhulst factor  $V = N(t)/N_{max}$ , where  $N(t)$  is the current population size and  $N_{max}$ , known as the carrying capacity, is the maximum number of individuals that the environment can support. At every timestep, and for each individual, a random number between zero and 1 is generated and compared with  $V$ : if it is smaller than  $V$  the individual dies, independently of its age or genome. This is the global mechanism of competition, already mentioned in the previous section.

If an individual succeeds in surviving until the minimum reproduction age  $R$ , it generates  $b$  offspring every year until death (unless a maximum reproduction age,  $R_{max}$ , smaller than 32, is included). The offspring genome is a copy of the parent’s one, except for  $m$  deleterious mutations introduced at birth. Although the model allows good and bad mutations, generally only the bad ones are considered. In this case, if a bit 1 is randomly tossed in the parent’s genome, it remains 1 in the offspring genome; however, if a bit zero is randomly tossed, it is set to 1 in the mutated offspring genome. In this way, for the asexual reproduction the offspring is always as good as or worse than the parent. Even so, a stable population is obtained, provided the birth rate  $b$  is greater than a minimum value, which was analytically obtained by Penna and Moss de Oliveira [11]. In fact, the population is sustained by those cases where no mutation occurs, when a bit already set to 1 in the parent genome is chosen. These cases are enough to avoid mutational meltdown, that is, population extinction due to the accumulation of deleterious mutations [13]. The reason why generally only harmful mutations are considered is that they are 100 times more frequent than the backward ones (reverse mutations deleting harmful ones [14]).

Resuming, the parameters of the model are:

$N(t = 0)$  - initial population;

$N_{max}$  - carrying capacity, generally taken as  $10 \times N(t = 0)$ ;

$T$  - threshold for bad mutations;

$R$  - minimum reproduction age;

$m$  - mutation rate from the parent’s to the offspring genome.

### 3 Catastrophic senescence and program for the asexual Penna model

The first big goal of the Penna model was the explanation of why some species like the salmon reproduce only once, always at the same age, and die a few days later [12]. The salmon, in particular, sometimes travels more than 1200 kilometers up river in order to reproduce, generally without eating after reaching sweet waters. A natural question is if it reproduces only once because it dies of starvation and exhaustion after such a travel, or if it dies because it reproduces only once. Modifying only one line in the Penna model program, substituting the instruction “if age  $a \geq R$ , reproduce” by “if age  $a = R$ , reproduce”, the answer was immediately obtained: it dies because it stops to reproduce. Such a result is a direct consequence of the mutation accumulation hypothesis in which the model is based: since mutations are unavoidable, after many generations they accumulate at the end part of the chronological genomes or, equivalently, at advanced ages. Selection pressure acts strongly before the reproduction period, trying to keep the genomes clean, to ensure that individuals will survive to generate offspring. When they loose this ability, they die, instead of remaining inside the population and competing for food with the youngsters. It is important to notice that such an instruction is not included in the model: It happens as a consequence of the mutation accumulation dynamics.

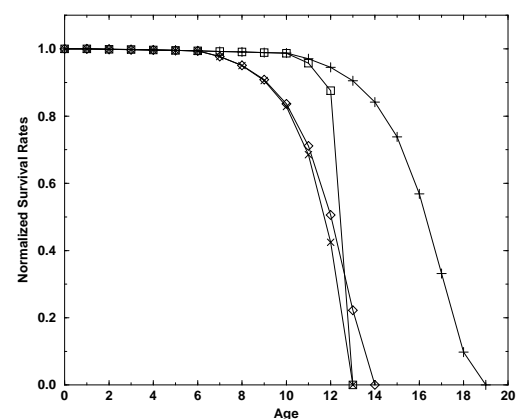


Figure 1. Normalized survival rates as a function of age. Each curve corresponds to a given period of reproduction: x from 6 to 12; squares from 10 to 12; diamonds from 6 to 32 and crosses from 10 to 32.

The survival rate at age  $a$  is defined, for an already stable population, by:

$$S(a) = \frac{N(a+1)}{N(a)},$$

where  $N(a)$  is the number of individuals with age  $a$ . It gives the probability that an individual with age  $a$  survives until age  $a+1$ . A stable population means that its number of individuals per age is already constant in time. Fig. 1 shows the survival rates as a function of age obtained for populations with different reproductive periods, where a maximum reproduction age  $R_{max}$  was also introduced. From this figure we see that the survival rates obtained with the Penna model starts to decay as soon as reproduction starts, as observed in real populations, and that there are no more individuals alive older than the maximum reproduction age.

These curves were obtained in a few hours on a Pentium, for several  $10^5$  individuals, and have the following common parameters:  $N_{max}/N(0) = 10$ ,  $T = 1$ ,  $b = 1$  and  $m = 1$ .

Below we present a C version of the asexual population program; The Fortran program is listed in [15]. Observe that the program contains, for each individual, an auxiliary word called "data", where its characteristics are stored. Particularly, it is not necessary to count, at every time step, the current number of accumulated diseases of each individual. The total number of mutations is counted only once, when the individual is born, and is compared to the limit  $T$ ; the genetic death age of that individual, according to  $T$ , is stored in its data as "dage", and defines when the individual will die if the Verhulst factor does not kill it before.

```
#include <stdio.h>
#include <math.h>

/* file for results */           #define resfile "res1.dat"
/* initial random seed */       #define R0      899665
/* maximum pop for Verlhust factor */ #define Popmax  1000000
/* initial population */       #define Inipop  100000
/* array dimension for population */ #define Popdim  1000000
/* number of steps (years) */   #define Maxstep 60000
/* final averaged steps */     #define Medstep 10000
/* minimum reproduction age */  #define minage  8
/* maximum reproduction age */  #define maxage  32
/* threshold of bad mutations */ #define lim      3
/* mutation rate */            #define mut       1
/* birth rate (per individuum*year) */ #define birth   1
/* only bad mut (0) or also good (1) */ #define good    0

#define MAXUINT    4294967295U
#define rmaxint    4294967296.0
#define N6         63

unsigned          age, nmut, dage, error, R, T, Verhu, Mstep,
                 number[33], Bit[32], Gen[Popdim], Data[Popdim];

long             Pop, Lpop, Spop, Tl, Ts;

double          x, ant[33], ymed[33], xmed[33];

void            Init(), Evolve(), Result();

void Init() {
    unsigned          i;
    unsigned long     I, P;

    error = 0; R = R0 | 1; Mstep = Maxstep - Medstep - 1;
    Pop = Inipop;
    Spop = Lpop = Pop;
    Tl = Ts = 0;
    Bit[0] = 1; for(i=1; i<32; i++) Bit[i] = Bit[i-1]<<1;
    for(i=0; i<=32; i++) {
        ant[i] = xmed[i] = ymed[i] = 0.0;
        number[i] = 0;
    }
}
```

```

for(i=0; i<1000; i++) R += (R<<1) + (R<<16);

number[0] = Pop;          /* clean genes at begining */
for(I=0; I<Pop; I++) {Gen[I] = 0; Data[I] = 32<<6;}

/*  Data[I] stores data concerning individuum I:
        age          at bits 0...5
        programmed death age    at bits 6...11
*/

}

void Evolve() {
  unsigned long    I,P,Gene,Pa;
  unsigned         n,i,agei,r;
  double          Poprint;

  Poprint = Pop*255.0;
  for(T=0; T<Maxstep; T++) {
    x = Pop; Verhu = (x/Popmax)*MAXUINT;
    Poprint += Pop;
    if(Pop<Spop) {Spop = Pop; Ts = T;}
    if (Pop>Lpop) {Lpop = Pop; Tl = T;}
    if((T&255L)==0) {
      printf("  %10lu   %10.11f\n",T,Poprint/256.0);
      Poprint = 0.0;
      fflush(stdout);
    }

    I = 0;
    Pa = Pop;
    while(I<Pa) {
      age = Data[I]&N6;
      dage = (Data[I]>>6)&N6;
      number[age]--; age++;
      R += (R<<1) + (R<<16);
      if((R<Verhu) || (age==dage)) {          /* death */
        Pop--;
        if(Pop<=0) {error = 1; Result(); exit(1);}
        Gen[I] = Gen[Pop]; Data[I] = Data[Pop];
        if(Pop>=Pa) I++; else Pa--;
      }
      else {          /* alive */
        number[age]++;
        Data[I] = age|(dage<<6);
        r = (age>=minage)&&(age<=maxage);
        if(r) {          /* breed */
          for(n=0; n<birth; n++) {          /* birth */
            Gene = Gen[I];
            for(i=0; i<mut; i++) {          /* mutations */
              R += (R<<1) + (R<<16); P = Bit[R>>27];
            }
          }
        }
      }
      #if good
        Gene ^= P;
      #else
        Gene |= P;
      #endif
      I++;
    }
  }
}

```

```

    number[0]++; Gen[Pop] = Gene;
    nmut = 0; for(i=1; i<32; i++) {
        nmut += Gene&1;
        if(nmut>=lim) break;
        Gene >>= 1;
    }
    Data[Pop] = i<<6;
    Pop++; if(Pop>=Popdim) {error = 2; Result(); exit(1);}
}
}
I++;
}
}
if(T>Mstep) {
    /* averages */
    for(i=1; i<33; i++) xmed[i] += number[i]/(0.00001+ant[i-1]);
    for(i=0; i<33; i++) {ymed[i] += number[i]; ant[i] = number[i];}
}
else if(T==Mstep) for(i=0; i<33; i++) ant[i] = number[i];
}
}

void Result() {
    FILE          *file;
    unsigned      i;
    double        f,r,pop;

    file = fopen(resfile,"w");
    fprintf(file,"Age   Pop   SR ");
    fprintf(file,"\n      ASEXUAL BIT STRING MODEL   (file  ");
    fprintf(file,resfile); fprintf(file,")\n");
    fprintf(file,"      R0 = %lu\n",R0);
    fprintf(file,"      Popmax = %lu\n",Popmax);
    fprintf(file,"      Inipop = %lu\n",Inipop);
    fprintf(file,"      Popdim = %lu\n",Popdim);
    fprintf(file,"      Maxstep = %lu\n",Maxstep);
    fprintf(file,"      Medstep = %lu\n",Medstep);
    fprintf(file,"      minage = %u\n",minage);
    fprintf(file,"      maxage = %u\n",maxage);
    fprintf(file,"      lim = %u\n",lim);
    fprintf(file,"      mut = %u\n",mut);
    fprintf(file,"      birth = %u\n\n",birth);
    if(good) fprintf(file,"      bad and good mutations");
    else fprintf(file,"      only bad mutations");
    fprintf(file,"\n\n      population maximum = %8ld at time %8ld\n"
        ,Lpop,Tl);
    fprintf(file,"      minimum = %8ld at time %8ld\n\n"
        ,Spop,Ts);
    if(error==0) {
        pop = 0.0;
        for(i=0; i<33; i++) pop += ymed[i]; pop /= Medstep;
        fprintf(file,"      population = %10.11f",pop);
        r = xmed[1]/Medstep; if(minage==maxage) r *= minage;
        fprintf(file,"      sr from age 0 to 1 = %8.4lf\n\n",r);
        fprintf(file,"      age      averaged population      survival rate\n");
        f = 1.0/ymed[0]; r = 1.0/xmed[1];
        fprintf(file,"      0          1          1");
        for(i=1; i<33; i++) {

```

```

    pop = ymed[i]*f;
    fprintf(file, "\n %2u          %8.4lf          %8.4lf"
           , i, pop, xmed[i]*r);
  }
}
else {
  if(error==1) fprintf(file, "\n\n meltdown T = %lu\n", T);
  if(error==2) fprintf(file, "\n\n overflow T = %lu\n", T);
}
fclose(file);
}

main() {

  Init();          /* initializes data */
  Evolve();       /* evolves the population maxsteps years */
  Result();

}

```

The catastrophic senescence effect rises the question of why women live even longer than men if they stop to reproduce before, due to menopause. In order to understand this phenomenon, it was necessary to introduce sex into the model.

#### 4 Sexual version of the Penna model

The sexual version of the Penna model was first introduced by Bernardes [16, 17], followed by Stauffer et al. [18] who adopted a slightly different strategy. We are going to describe and use the second one. Now individuals are diploids, with their genomes represented by two bit-strings that are read in parallel. One of the bit-strings contains the genetic information inherited from the mother, and the other from the father. In order to count the accumulated number of mutations and compare it with the threshold  $T$ , it is necessary to distinguish between recessive and dominant mutations. A mutation is counted if two bits set to 1 appear at the same position in both bit-strings (inherited from both parents) or if it appears in only one of the bit-strings but at a dominant position (locus). The dominant positions are randomly chosen at the beginning of the simulation and are the same for all individuals.

The population is now divided into males and females. After reaching the minimum reproduction age  $R$ , a female randomly chooses a male with age also equal to or greater than  $R$  to breed. To construct one offspring genome first the two bit-strings of the mother are cut in a random position (crossing), producing four bit-string pieces. Two complementary pieces are chosen to form the female gamete (recombination). Finally,  $m_f$  deleterious mutations are randomly introduced. The same process occurs with the male's genome, producing the male gamete with  $m_m$  deleterious mutations. These two resulting bit-strings form the offspring

genome. The sex of the baby is randomly chosen, with a probability of 50% for each one. This whole strategy is repeated  $b$  times to produce the  $b$  offspring. The Verhulst killing factor already mentioned works in the same way as in the asexual reproduction case. Fig. 2a shows how one gamete is formed in a diploid sexual population. The program for sexual populations is too long to be presented here, but it can be requested by e-mail to the author.

Figure 3 compares some different survival rates as a function of age. The common parameters are:  $R = 10$ ,  $T = 4$ ,  $N(0) = 100,000$  (half for each sex in case of sexual reproduction) and  $N_{max} = 10 \times N(0)$ ; birth rate  $b = 2$  in asexual case and  $b = 4$  for females in sexual case (giving  $b = 2$  per individual as in the asexual one). In the sexual cases 6 randomly chosen positions were considered as the dominant ones.

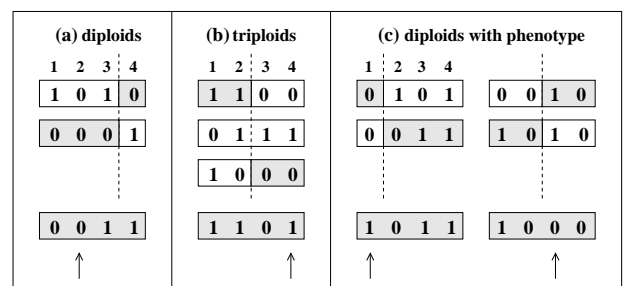


Figure 2. Schematic representation of gamete formation for a) a diploid sexual population; b) for a triploid population; c) for a diploid sexual population with a non-age structured phenotype. Arrows indicate where mutations occurred. For diploids a second gamete is generated using this same strategy; for triploids the process is performed three times, generating one gamete per parent.

If we compare the two sexual cases presented in the figure (diamonds and crosses), we notice that life expectancy

is shorter if menopause is considered, but there is no catastrophic senescence: menopause sets in at age 12 and the total population survives until age 19. A crucial aspect of the model as well as in Nature is that *sex is not transmitted genetically*; independent of the genome we take each child as male with probability 1/2, and as female otherwise. So if death is hidden in the offspring genes, then either both males and females die soon, or both males and females die late. It is important to note that with this version of the model males and females present exactly the same survival rates, even if the male mutation rate is larger than the female one, and so neither Nature nor the model allows females to die sooner from accumulated genetic mutations than the males. However, when both males and females reproduce from 10 to 32 (diamonds in Fig. 3), the whole population presents a larger life expectancy. In this case, we return to the same question, now slightly modified: Why does menopause exist?

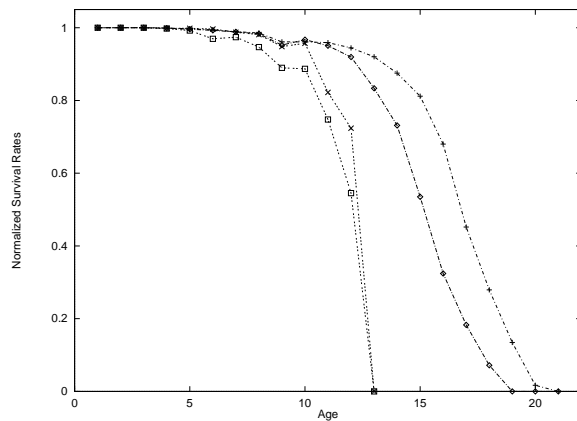


Figure 3. Normalized survival rates for sexual and asexual reproduction. Squares ( $m = 2$ ) and  $x$  ( $m = 1$ ) correspond to asexual reproduction from age 10 to 12. Diamonds and  $+$  correspond to sexual reproduction,  $m_f = m_m = 1$  (one mutation from each parent) and dominance =  $6/32$ ; diamonds for female reproduction from age 10 to 12 (males from 10 to 32) and stars for reproduction from age 10 to 32.

## 5 Self-organization of menopause

A possible explanation for menopause was already pointed out in 1957 by Williams [19]. He suggested that due to a reproduction risk that increases with advancing ages and a long period of child dependence, it is more advantageous to the females to cease reproduction in order to take care of the already born young offspring. This idea was simulated [20] by introducing into the Penna model the following restrictions: a) there is a risk due to reproduction which is proportional to the number of current accumulated mutations (which means proportional to age, since in the model bad mutations accumulate at advancing ages); b) there is a period of parental care: offspring whose mothers die within this period, are killed; c) the menopause age is no longer

imposed, but transmitted to the female offspring with mutations. That is, all the females start with a maximum reproduction age (menopause age) equal to 32. When a daughter is born, it inherits the mother's menopause age with probability 25%, or the mother's menopause age  $\pm 1$  with probability 75%. The minimum reproduction age is still imposed and the same for both sexes, and the male maximum reproduction age is fixed at 32. With this strategy a self-organized distribution of menopause ages was obtained, as well as a period of post menopause survival. The distribution of menopause ages is shown in Fig. 4. It is important to note in this figure that despite of the risk for later reproduction, there is no self-organization of the menopause ages if there is no parental care. Both ingredients are necessary to obtain such an effect.

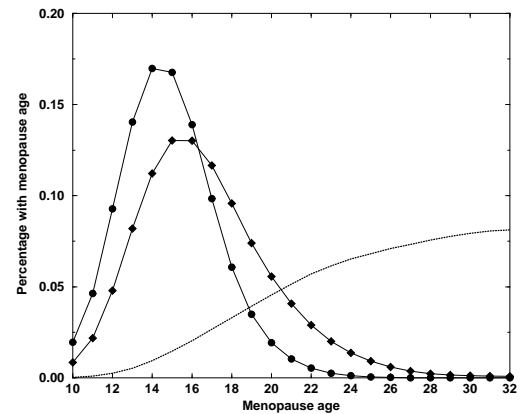


Figure 4. Percentage of females with a given menopause age as a function of the menopause age. Circles: parental care for 5 years; Diamonds: parental care for 4 years; Dotted line: no parental care.

## 6 Coevolution and the Red-Queen hypothesis

Comparing again the curves of Fig. 3, we see that sexual reproduction provides a higher longevity than the haploid asexual one. The reason is that sexual reproduction contains the fundamental ingredients of recombination and recessive mutations. These two ingredients allow a generation of offspring with better genomes than the parents, even if only detrimental mutations are considered. However, the asexual reproduction provides a number of offspring twice larger than the sexual reproduction, where only females give birth. In this way, if an asexual and a sexual population co-evolve in the same environment, disputing for the same resources (Verhulst factor), the asexual population dominates and the sexual one disappears. To make the dispute between the advantages of each kind of reproductive regime more complicated, there is a kind of asexual reproduction, called meiotic parthenogenesis, where individuals are diploids. In

the Penna model language, each individual carries two bit-strings that are read in parallel, as in the sexual case, and there are also recessive and dominant positions. During reproduction, the bit-strings are cut in a random position, and two complementary pieces are jointed, forming the equivalent of a gamete in sexual reproduction (Fig. 2a). Then this same “gamete” is copied, generating the second bit-string of the baby, and random mutations are then introduced. It has been shown [21] that this kind of reproduction produces survival rates that are completely equivalent to those of the sexual case, besides being much faster and requiring much less effort.

A number of theories have been put forth to try to explain the evolution and maintenance of sexual reproduction. In the center of this debate is the so-called “Red Queen” hypothesis, that relies heavily on the concept of diversity. In essence, it holds the action of genetically matching parasites as responsible for creating a rapidly changing environment. In this unstable ecology only varieties that can mutate their genomic pool, at least as fast as the adaptation of the parasites proceed, can survive. The theory derives its name from this endless race, quoting from the Red Queen of Lewis Carol’s *Alice in Wonderland*: “It takes all the running you can do, to keep in the same place.” In fact, observations of competing varieties of a freshwater snail, *Potamopyrgus antipodarum*, have shown that there is a strong correlation between the prevalence of one reproduction regime and the concentration in its habitat of the trematode *Microphallus*, a parasite that renders the snail sterile by eating its gonads [22, 23, 24]. Namely, the asexual variety is predominant where the parasite appears in small concentrations, whereas higher concentrations of the trematode forces the species to prefer a sexual regime.

This correlation could be shown to exist in simulations of a conveniently modified Penna model [25]. The parasites are represented by a dynamically changing memory bank of genomes of some fixed number of entries. Each entry is modified if it comes into contact with the same genome twice in a row; in this case, it memorizes this pattern and stores it in the memory bank. At each time step, before the reproduction cycle, each female of the population is probed by a fixed number  $E$  of randomly chosen entries of the parasite bank. If one of these entries is a perfect match for the female’s genome, she is rendered sterile and can no longer reproduce. The number of parasite exposures  $E$  is an indirect measure of the parasite concentration in the habitat. For the host population, the reproductive regime of the females is no longer a fixed character, but can mutate with some small probability. That is, the offspring generated by a meiotic parthenogenetic female has a small probability to mutate to a sexual reproduction regime, and vice-versa. The simulations begin in the absence of the parasite infestation, and the initial population is set to have a sexual reproductive regime. As soon as the meiotic population appears, due to the mutations in the reproductive regime, it overrides the sexual variety: sex barely subsists due to infrequent back-mutations from the asexual variety. At some time step, the parasite infestation is turned on. The resulting predominant variety is going to depend solely on the intensity of this infestation,

as measured by the exposure parameter  $E$ . For small values of  $E$ , the asexual variety has the upper hand. As  $E$  is increased, a first-order transition is seen to a configuration dominated by the sexual population. Fig. 5 shows the fraction of females in the population that reproduces sexually, as a function of the exposure parameter  $E$ . The sudden jump in this fraction signals the order of the transition.

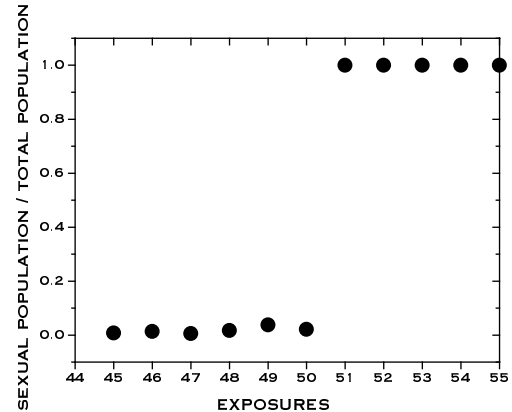


Figure 5. The fraction of females that reproduce sexually in the population is plotted against the value of the exposure parameter  $E$ . The correlation between the dominant pattern of reproduction and the intensity of the infestation, as measured by this last parameter, is clearly seen.

It is easy to understand that the sexual reproduction generates a larger genetic diversity than the meiotic parthenogenetic one, since the former involves two different individuals instead of only one. In fact, it was already pointed by Stearns [26] that the meiotic parthenogenesis produces, in general, individuals that are homozygous in all positions. Because of this disadvantage, it is easier for the parasites to contact twice the same genomic pattern among the asexual individuals than among the sexual ones. Of course such a conclusion gives rise to another question: Why are we diploids instead of triploids?

## 7 Diploids X Triploids

Since a diploid sexual population has the upper hand when competing against an asexual one due to the diversity generated by the use of genetic material coming from two different parents, why does not Nature enhance this effect by allowing the genome of the offspring to benefit from three different templates? Is the fact that for triploids mutations need to appear at the same position in the three homologous chromosomes (bit-strings) to be counted (except for dominant positions) enough to overcome the burden of using three individuals to generate one offspring? Simulations of a triploidal Penna population [27] have gathered arguments against this possibility.

For this comparison to be made, the rules for survival of the Penna model were changed according to the findings of Ref. [28], in which a modified survival probability was adopted, generating sexual populations with sizes compatible with those of the asexual ones, even in the absence of



parasites or any other external agent. This modification consists in assuming that harmful mutation reduces the survival probability. At each iteration, or “year,” each individual survives with probability  $\exp(-m\epsilon)$  if it has a total of  $m$  harmful mutations (taking into account dominant positions) in its whole genome (it is killed if a random number is tossed that is smaller than the survival probability).  $\epsilon$  is a parameter of the simulation, fixed from the start. To summarize, an individual may now die for any one of three reasons: i) randomly, due to the Verhulst logistic factor; ii) if its actual number of accumulated diseases reaches the limit  $T$ ; iii) if its survival probability becomes too small.

In the triploid population, individuals have genomic material in three different bit-strings that are read in parallel. It is assumed that mating involves three individuals (two males and one female or vice-versa). Homozygous positions are those with three equal bits at homologous loci. Harmful mutations are active only if there are three bits 1 at that same position, or at a heterozygous locus at which harmful mutations are dominant. Only females generate offspring. Crossing and recombination are performed by a random choice of a locus at which the three strings are cut, generating six pieces. Two complementary pieces of those are randomly chosen to form one gamete. This process is performed for each one of the three parents. Deleterious mutations are randomly introduced in each gamete (see Fig. 2b). The baby is a male or a female, with equal probability.

Figure 6 presents the time evolution of a diploid sexual population and of two different triploid ones, showing that the diploid sexual population is larger than any of the other two.

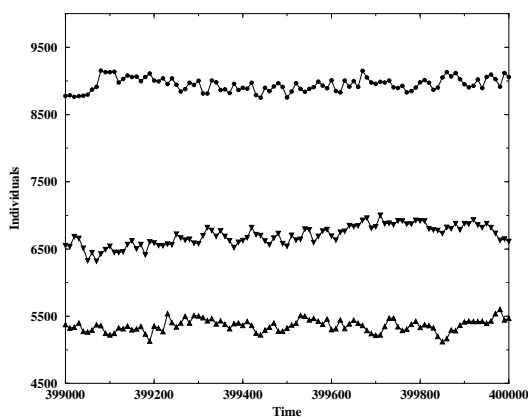


Figure 6. Time evolution of a diploid population (upper curve) and two triploid populations: in the central curve, reproduction involves one male and two females, while in the lower one it involves one female and two males.

The genetic diversity is obtained computing the Hamming distance, in this case defined by the number of different loci (bits) between the genomes, for all pairs of individuals. The probability distribution of these distances is obtained by making a histogram of the fraction of pairs, out of all possible pairs in the population, that present a given Hamming distance, normalized by its maximum possible

value (64 for diploids and 96 for triploids). Fig. 7 shows the resulting distributions for the diploid and triploid populations. It is clear that the diploid population presents both a larger mean distance between pairs, indicated roughly by the position of the peak of the distribution, and a larger variance, measured by the width at half the maximum height of the curves. The results are essentially the same if a double crossing of the triploid genome is performed during reproduction, and there is no benefit for the triploids to ensure that the offspring have their genetic material gathered from all three parents.

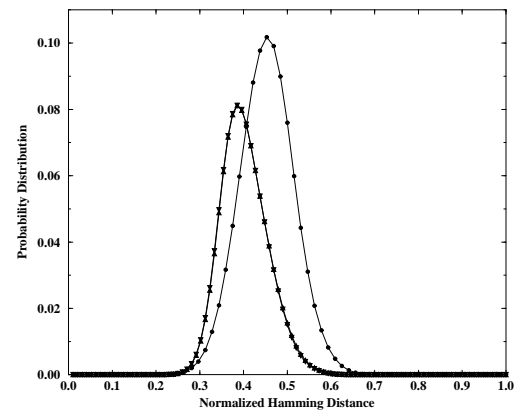


Figure 7. Genetic diversity of a diploid population (full circles) and of the two triploid populations mentioned in the captions of the previous figure, which are, for this particular measure, indistinguishable.

The diploid population also presented a slightly better survival rate, with comparable longevities [27]. These results show that genetical reproduction has to recombine material in the correct amount, in order to balance the extra cost of reproduction involved when multiple parents are needed - more is not necessarily better!

## 8 Sympatric speciation with phenotypic selection

Speciation involves the division of a species on an adaptive peak, so that each part moves onto a new adaptive peak without either one going against the upward force of natural selection. This process is readily envisioned if a species becomes subdivided by a physical barrier, whereby each part experiences different mutations, population fluctuations and selective forces, in what is called the allopatric model of speciation. In contrast, conceiving the division of a single population and radiation onto separate peaks without geographical isolation, in what is called sympatric speciation, is intuitively more difficult. Through which mechanism can a single population of interbreeding organisms be converted into two reproductively isolated segments in the absence of spatial barriers or hindrances to gene exchange? In this section we describe the features that were added to the standard

Penna model to represent phenotypic selection and speciation.

The version with a single phenotypic trait [29] was motivated by field observations. The intention was to mimic the seasonal effect of rainfall on the availability of seeds of different sizes in the Galapagos islands and its impact on the morphology of beak sizes in the population of ground finches that feed on these seeds [30, 31, 32]. It has been observed that depending on the amount of rain, the distribution of seed sizes changes from a broad distribution centered at middle sized seeds to a double-peaked distribution, of only small or large seeds. The beak sizes of the ground finches follow this same dynamics in a very impressive and fast (few generations) process of adaptation.

The beak is represented by a single pair of non age-structured bit-strings, added to the chronological genome of each individual. The dynamics of reproduction and mutations are the same for both the age-structured and the new strings - for the latter, a mutation that changes a bit from 1 to 0 is also allowed (see fig.4c). The beak size is determined by counting, in this non-structured pair of bit-strings, the number of recessive bit-positions (chosen as 16) where both bits are set to 1, plus the number of dominant positions with at least one of the two bits set. It will be a number  $k$  between 0, meaning a very small beak, and 32, for a very large one. Its selective value is given by a fitness function  $F(k)$ , that indicates how much the individual is fitted to the environment. For a given value of the beak size  $k$ ,  $F(k)$  quantifies the availability of seeds for individuals with that particular morphology.

This quantification was done through the Verhulst factor, which now becomes dependent on genetic material (the beak size). It gives the probability of death by intra-specific competition at each time step:

$$V(t) = \frac{N(t, k)}{(N_{\max} * F(k))},$$

where  $N(t, k)$  is the number of individuals of beak size  $k$  at time step  $t$ .

The simulations were done with two different functional forms for the function  $F(k)$ . At the beginning of the simulations,  $F(k)$  is a single-peaked function with a maximum at  $k = 16$ , representing large availability of medium-sized seeds:

$$\begin{aligned} F(k) &= 1 - \frac{16 - k}{A}, \quad k < 16 \\ &= 1 - \frac{k - 16}{A}, \quad k \geq 16 \end{aligned} \quad (1)$$

where  $A$  is a constant that controls the intensity of the environment pressure.

After 20 000 time steps, there is a sudden change in the pattern of seed availability (simulating the variation in the rainfall regime). The fitness function that expresses this new pattern is, for instance,

$$\begin{aligned} F'(k) &= 1 - \frac{A - 16 + k}{A}, \quad k < 16 \\ &= 1 - \frac{A - k + 16}{A}, \quad k \geq 16 \end{aligned} \quad (2)$$

This change force evolution to give rise to a polymorphism, shown in Fig. 8 (diamonds) as a resulting 2-peaked equilibrium distribution of beak sizes. This polymorphism is reversible: if, in a subsequent time step, the pattern of availability of edible seeds reverts to its original configuration, so does also the distribution of beak sizes.

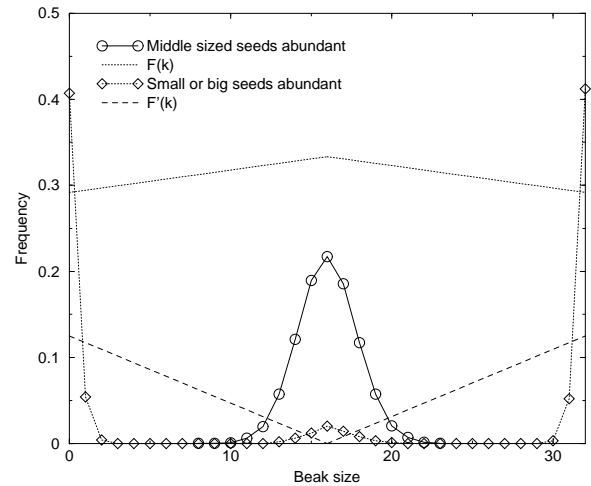


Figure 8. The distribution of phenotypes in the population is shown for two different regimes of seed availability. The circles correspond to the equilibrium population at time step 12 000, in a situation in which seeds are available for a broad distribution of sizes, peaked at beak size  $k = 16$ ; the corresponding fitness function  $F(k)$ , adequately rescaled to fit in the graph, is shown for a comparison. The two-peaked phenotype distribution corresponding to the diamonds, is a snap-shot of the population at time step 50 000, that is, after  $F(k)$  has been double-peaked for 30 000 time steps. The population has already split into a (reversible) polymorphism, with two different beak sizes. Since there is no reproductive isolation, mating between birds feeding on different niches generate offspring with medium-sized beaks, represented by the small bump at  $k = 16$ ).

In order to really have speciation, which implies in a non reversible polymorphism, it was necessary to introduce sexual selection into the reproductive strategy, to avoid mating between small and large beaks. A single locus was introduced into the genome that codes for this selectiveness, also obeying the general rules of the Penna model for genetic heritage and mutation. If it is set to 0, the individual will not be selective in mating (random mating), and it will be selective (assortative mating) if this locus is set to 1. The mutation probability for this locus was set to 0.001 in all simulations. Individuals that are selective will choose mating partners with its same morphological characteristics, that is, if an individual has  $k < (>)16$  and is selective, it will only mate with a partner that also has  $k < (>)16$ .

Assortative mating is essentially equivalent to speciation in this context, and one of the purposes of these simulations was to follow the rising of the fraction of the population that becomes sexually selective. Starting with a non selective

population, it was observed that the fraction of selective individuals increased to at most 0.003 while  $F(k)$  was single-peaked, and jumped to nearly 1.0 **after** the establishment of a double-peaked distribution of seeds. Two distinct populations, each of which does not mate with a partner from the other, is the final result: evolutionary dynamics made it advantageous to develop assortative mating in this bi-modal ecology, and as a consequence of reproductive isolation, one single species has split into two.

Both a simpler and a more elaborated model (with two phenotypic traits) using the Penna bit-string strategy to simulate sympatric speciation can be found in [33].

## 9 Conclusions

We have presented the Penna model for biological ageing and some of its most important results. Ageing is an unavoidable process (experimentally confirmed by the author) and has been extensively studied by many different scientists, since a very long time. Although the evolutionary theories for senescence have appeared around 1950, Monte Carlo Simulations on this subject started only after the publication of the Partridge-Barton analytical mathematical model [34] in 1993. The Penna model is now the most widespread Monte Carlo technique to simulate and study the different aspects of population dynamics, including ageing. In this review we have focused attention on results concerning the differences between reproductive regimes and the advantages of sexual reproduction, as well as on the modifications introduced into the Penna model in order to study sympatric speciation.

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