Monte Carlo Simulations of Sexual Reproduction
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Abstract: Modifying the Redfield model of sexual reproduction and the Penna model
of biological aging, we compare reproduction with and without recombination in age-
structured populations. In contrast to Redfield and in agreement with Bernardes we find
sexual reproduction to be preferred to asexual one. In particular, the presence of old but
still reproducing males helps the survival of younger females beyond their reproductive
age.

I. Introduction
According to Genesis, work, sex, ageing, and death started simultaneously. Most of
today’s higher species prefer sexual over asexual reproduction, and even asexual species
often employ some form of genetic recombination. There are many theories but no consen-
sus[1] why sex is preferred. For example, sex may spread advantageous mutations better
through the population, sex increases genetic variety which may help species to adjust after
a drastic change in the environment, or sex may protect against parasites. In a computer
model with only deleterious mutations, a constant environment, and no parasites, none
of these advantages hold. Indeed, in such a simulation Redfield[2] found only seldomly a
clear advantage of sex to justify its cost to feed the males (which were also stated [2] to
be unreliable in transmitting the correct genes). Then, what are males useful for ?

Charlesworth et al [3] have claimed that large sexual, as opposed to large asexual,
populations are protected against the mutational meltdown due to the accumulation of
deleterious inheritable mutations[4]. However, this claim was refuted [5] as being due to
insufficient observation times: In their model the decay time increases exponentially with
population size. Different models of Bernardes [5] with and without age structure gave
clear advantages for sex: The population can survive with sex better than without. More
recently, however, Bernardes [6] emphasized the advantages of asexual reproduction with
genetic recombination within the same individual (“meiotic parthenogenesis”) observed in
many asexual species.

The present paper ignores these intermediate forms and compares sexual with asexual
reproduction, assuming (as did Redfield[2]) genetic recombination only in the sexual case.
In contrast to Redfield we follow Bernardes[5,6] in assuming that only a fraction $h$ of
the genetic mutations are dominant, the others being recessive. We want to see if now sex
is more helpful in the Redfield model, using both her techniques (Chapter II) as well as
Monte Carlo simulations based on a bit-string model of the genome (Chapter III). Then we
use in Chapter IV the Penna bit-string model [7,5] for age-structured populations to find
out if the presence of males allows females to live beyond their maximum reproductive age; for the asexual case the accumulation of deleterious mutations kills all individuals beyond reproductive age [8] if parental care [9] is ignored. Our sexual Monte Carlo simulations are similar to those of ref.6 but include some features like male fidelity ignored by Bernardes.

II. Redfield Model

The Redfield model [2] is a very fast algorithm avoiding all random numbers and calculating from the probability distributions of the mutations at time $t$ the corresponding distributions at the next time step $t + 1$. It should be exact in the limit of very large populations and cannot give the finite-size effects studied in ref.6.

The algorithm assumes from the beginning that the population is constant; thus the birth rate must adjust to the temporal variation of the death rate. Thus the crucial effect of mutational meltdown due to the accumulation of only deleterious inherited mutations [4] cannot be studied with this assumption. In principle this assumption is biologically unrealistic [5] though widespread in the biological literature. It is known that many species have died out even without any human intervention. Thus we have not used this dangerous assumption in our Monte Carlo work of the following chapters. It seems, however, that in the context of the Redfield calculations [2] this assumption is allowed. Monte Carlo calculations [10] have confirmed the average survival probability observed by Redfield in the asexual case and following from traditional steady-state theory. Moreover, we now have applied the Redfield method also to the case where mutational meltdown occurs, by assuming exactly one mutation per genome at each time step (“generation”). Then the mutational meltdown was signalled clearly by an average survival probability decaying towards zero, whereas in ref.2 and in our corresponding simulations using a Poisson distribution of mutations these survival rates converged to a positive plateau value. Thus the Redfield algorithm can signal mutational meltdown if it occurs, and the assumption of a Poisson distribution can avoid this meltdown since now with a nonzero probability no new mutation happens and the accumulation of an unlimited number of mutations is avoided for some individuals.

In her algorithm, Redfield starts from some progeny distribution $P(m)$, $m = 0, 1, \ldots$, giving the probability that an individual has $m$ genetic diseases in the genome. Darwinistic selection of the fittest then transforms this $P(m)$ into a survivor distribution $L(m) \propto (1 - s)^m P(m)$ giving the probability that a survivor has $m$ deleterious mutations in the genome. Here the selection coefficient $s$ was taken as 0.1, and the various mutations (“genetic diseases”) are assumed to act independently (see below for the alternative of truncation selection [2]). Now $n$ new hereditary mutations happen according to a Poisson distribution $\mu^n \exp(-\mu)/n!$ so that the individual has $m + n$ mutations; here $\mu$ of order unity is the mutation rate per genome and generation. In the asexual case, the resulting distribution of mutations is already the new progeny distribution $P(m)$, and the above cycle of selection and mutation is repeated again and again.

In the sexual case, according to Redfield [2] the mutation rate for males can be larger by a factor $\alpha$ than that for females. Thus after selection transformed the progeny $P(m)$ distribution into the survivor distribution $L(m)$, mutations of a rate $\mu$ produce the female distribution $F(m)$ and those of a rate $\alpha \mu$ give the male distribution $M(m)$. Then male gametes (sperm cells) are produced containing half of the male genome; thus their mutation
number $m_m$ is roughly half of the number of mutations in the father’s genome. Analogously, the number $m_f$ of mutations in the female gametes (egg cells) is roughly half of the mother’s number of mutations. The fusion of two gametes adds these two numbers,

$$m = m_m + m_f,$$

(1)

to produce the mutations in the progeny distribution $P(m)$. Now the cycle of selection, mutation, gamete production, and fusion is repeated again and again until the changes with each iteration become negligible.

(More precisely, the mutations in each gamete are not exactly half of those of the parent but follow a binomial distribution, which simulates the random selection of the transmitted half of the genome via the processes of meiosis, crossover, and mitosis [5].)

For efficiency purposes, the number of mutations was assumed to be below $10^2$. Then this whole procedure gives within a few seconds on a workstation after less than 100 iterations an average survival probability which barely changes with the number of iterations.

In this model the fitness is the average survival probability

$$\sum_m P(m) (1 - s)^m / \sum_m P(m);$$

its steady state value is $[2] \exp(-\mu)$ in the asexual and $\exp(-\mu(1 + \alpha)/2)$ in the sexual case, as confirmed by computer simulations. In particular, for $\alpha = 1$ sex does not change at all the average fitness and thus fails to justify its additional cost and complication. However, it is obvious from eq(1) that this Redfield model does not distinguish between dominant and recessive mutations. Usually, only a rather small fraction $h$ of the genetic diseases are dominant and reduce the survival probability even if only one of the two parents (for sexual reproduction) carries the dangerous mutation; the remaining mutations are recessive and affect the health only if present in both parents. Thus instead of eq(1) we could assume

$$m = (m_m + m_f)h$$

due to dominant mutations. The recessive mutations are negligible if we have an infinite size of the genome where the finite number of mutations can be distributed; otherwise the recessive mutations produce $rm_m m_f$ dangerous diseases, where $r$ varies as the reciprocal size of the genome. Thus we now replace eq(1) by

$$m = (m_m + m_f)h + rm_m m_f \quad (h \ll 1, r \ll 1).$$

(2)

The original Redfield model has $h = 1$, $r = 0$ and from this point of view corresponds to the limit of extreme inbreeding, which is known to be bad. Nature usually has weeded out most of the dominant mutations, and therefore most of the actually observed mutations are recessive.

With eq(1) replaced by eq(2) in the algorithm, the advantages of sexual compared with asexual reproduction become obvious even at the high value $\alpha = 10$ of male unreliability: For the Redfield value $\mu = 0.3$, asexual reproduction gives a fitness of 0.74, whereas sexual reproduction gives a fitness of 0.95 to 0.97 using eq(2) with $h = 0.2$ and $0 \leq r \leq 0.2$, to be compared with 0.19 using eq(1). Thus the cost of sex is more than justified by the drastic increase of the survival probability, in contrast to its drastic decrease found by Redfield
Fig. 1 gives an overview of the resulting fitness as a function of parameters; cases where more than 1000 mutations became relevant are ignored.

These computer simulations thus justify sex already from the fact that it suppresses the effects of recessive mutations.

Redfield[2] suggested instead to justify sex with the assumption that females select only the youngest males for mating. Since there is no age-structure in this model, we instead follow Bernardes[5,6] by assuming that females select only the healthiest males as mates. Then indeed similar drastic advantages of sex are found even if we use eq(1) without distinction between recessive and dominant mutations. Thus only males with a number of mutations below a sexual truncation threshold $sextruc$ mutations mate with females (independent of the female number of mutations). For $sextruc=1$ and 2 the fitness increased from 0.74 to 0.97 and 0.90, respectively. Fig. 2 shows some examples of the time evolution. (Following ref.2 we show here versus time first the simulation without sex, and then starting from the asexual equilibrium we show how for different values of the sexual truncation parameter we get different results with fitness higher or lower than before.) For consistency we also replaced the survival probability by a step function with a threshold similar to $sextruc$: Instead of $(1 - s)^m$ the probability is 1 for up to 6 mutations, and 0 for more mutations, a truncation method already used by Redfield[2].

If there are as many males as females, then this method of selecting only the healthiest males for mating means, that males mate with more than one female. If instead we use monogamy in our probability distributions, then this selection of males may diminish the average survival rate since many females now do not find a suitably healthy mate.

III. Redfield-type Bitstring Model

While all mutations in the Redfield model (as well as those in this paper) are hereditary, the method of probability distributions does not allow to identify offspring with particular parents who share the same genetic diseases (mutations). Thus an entirely different Monte Carlo simulation was made which should reproduce qualitatively though not quantitatively the effects of the Redfield model in our previous chapter. For asexual reproduction, the genome is then represented by one computer word containing 32 bits. Each bit is either set (=1) or off (=0) and represents one serious inheritable disease. Thus perfect health corresponds to the whole computer word consisting of zeroes. If a word contains $m$ set bits, it represents $m$ dangerous mutations reducing, as above, the survival probability to $(1 - s)^m$ with $s = 0.1$.

At each time step, each surviving individual gives birth to $b$ offspring, and each offspring differs by $\mu$ mutations from the parent. Such a mutation consists in selecting randomly one of the 32 bits, and setting this bit. If the selected bit is already set it stays that way and nothing happens. In this way, as with a Poisson distribution of mutations, with a nonzero probability no new mutation is accumulated, and mutational meltdown[4] can be avoided. Therefore no complicated Poisson distribution was simulated, in contrast to ref.6; a value of $\mu < 1$ means that one such mutation was attempted with probability $\mu$. In this version the simulation would give a population $N(t)$ which asymptotically would decrease or increase exponentially with time $t$, depending on the birth rate. To avoid the increase towards infinity we assume an additional death rate due to environmental restrictions of food and space. Then the survival probability is reduced by a Verhulst factor
1 − \( N(t)/N_{\text{max}} \) where the carrying capacity \( N_{\text{max}} \) was taken as 200,000.

For the sexual case, each individual has two such computer words as the genome. Biological recombination of genes is now a genetic algorithm: a crossing point between 0 and 32 is selected randomly to mix the genes of father and mother. The first computer word of the child has the first bits up to this crossing point from the father, and the remaining bits from the mother. (We select randomly for father and mother separately whether their first or their second computer word is used.) The second word for the child takes the other bits of the used computer words of father and mother. (Nature and refs.2,5,6 do these steps partially in different order; we found no important changes when we followed this computationally less efficient order.) The random selection of computer words and crossing points is repeated independently for each of the \( b \) children which are born simultaneously. A mutation is active, reducing the survival probability by a factor \( 1 − s \), if it is dominant or if it is carried by both computer words; otherwise the recessive mutation remains stored in the genome but does not affect the health.

For the sexual selection process we go through all the females and let each of them select randomly a male for mating. However, generalizing the methods of Bernardes [5,6], we jump over those females who gave birth recently within an interval of \( p \) time steps; \( p = 2 \) means that they do not mate again in the current time step or in the following one. This takes into account maternal care for the offspring[9]. Also, males are assumed to show some degree of marital fidelity and are not selected to mate if they have already mated before during the same time step. If a female cannot find a suitable male after many attempts, she gives up for this iteration and tries again at the next time step; otherwise she produces \( b \) offspring, each of which is randomly either male or female. Since the males have little to say in this selection process, we have not punished them further. Thus in contrast to chapter II and to Bernardes [5,6] we did no restrict mating to only the healthiest of them.

Fig.3 shows that for sexual reproduction with a small fraction \( 5/32 \) of dominant mutations there are many mutations but only few of them really count by diminishing the survival probability by 10 percent each; no such advantage is seen for the corresponding asexual case. The average fitness is 0.87 in the sexual and only 0.81 in the asexual case; it sinks to 0.79 if all mutations are dominant in the sexual case. Thus this bit-string Monte Carlo simulation confirms roughly the conclusions from the simpler probabilistic approach of chapter II.

IV. Ageing and Sex

In numerous species, sexual reproduction sets in only after some minimum reproductive age has been reached; and e.g. for women it stops after some maximum reproductive age (menopause). This effect can be described only by a model incorporating age as a variable. In particular we want to explain life after menopause.

We thus interpret the bit-string of the previous chapter as representing not only 32 diseases but also 32 intervals (“years”) in the life of an individual. Thus each year we feel the effect of at most one more hereditary disease. In contrast to the previous section, at a certain age we see only the bits (diseases) corresponding to this and earlier ages; we do not yet know and feel the diseases starting to affect us only later in life but hidden already in our genome.
The asexual version then becomes the well-studied Penna aging model[7] (see ref.5 for a review), if we replace the survival probability \((1 - s)^m\) by truncation selection: An individual survives up to the age when \(T\) inherited diseases become active; then it dies. We also use again a Verhulst factor. The sexual reproduction follows the lines of chapter III, but again with truncation selection and the interpretation of the bit position as the age.

In the asexual case we know already that life stops after the maximum reproductive age[8] in this model, as seen most drastically for Pacific salmon. After many generations, mutations have accumulated in all the bits beyond the maximum age of reproduction and then cause sudden death: The survival probability as a function of age jumps to zero. For the sexual case, however, this no longer is true. If the females reproduce only between the ages of 10 and 12, and the males for all ages starting from 10 (up to certain death at age 33), then both males and females live until about an age of 16, roughly the same life expectancy as for the asexual case; see fig.4a. Fig.4b, produced on 136 processors of the Intel Paragon at KFA Jülich (Germany), shows that as for the asexual Penna model the Gompertz law[11] is reasonably confirmed: The death rate after sexual maturity increases roughly exponentially with age.

Why can the females in this sexual model survive after their reproductive age is passed when they cannot in the asexual model[9]? A crucial aspect of our model as well as 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’s genes, then either both males and females die soon, or both males and females die late. Neither nature nor our model allows all females to die sooner from accumulated mutations than the males. Thus, men might be useful for something (in this model).

Omitting the requirement for male fidelity does not change much, whereas omission of the female waiting period by setting \(p = 0\) causes slightly faster aging. (Whereas in the asexual case eventually all survivors have one common ancestor, this is not the case for the sexual case.)

Separately [12] we discuss why women live longer than men, and offer somatic (not inheritable) mutations as an explanation, in contrast to the hereditary mutations discussed here and in most of the biological ageing literature.

V Summary

The Redfield algorithm is computationally fast and was easily modified to distinguish between dominant and recessive mutations. Its exact treatment of infinitely large populations is somewhat similar to that of Dasgupta for aging [13].

Various computer models have shown in this work that sexual recombination is advantageous compared to asexual reproduction without genetic recombination. Sex can hide recessive mutations and ensure the survival of females past reproductive age as long as males of the same age are still able to reproduce. Moreover, also the sexual version of the Penna model agreed with the naturally observed Gompertz law. We do not claim that our models are the only ones giving this desired agreement with nature, though we are not aware of other “microscopic” models explaining the Gompertz law. “Virtually all of many efforts to find a unifying theory of aging have foundered when rigorous questions were asked” [14].
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Fig.1: Average survival probabilities for Redfield algorithm with dominant fraction $h = 0$ (diamonds), 0.2 (+), 0.4 (squares), 0.6 (x), 0.8 (triangles), and 1.0 (stars), for various recessive parameters $r$. Ref.2 corresponds to stars at the $r = 0$ axis. Part a gives our results for $\alpha = 1$, part b for $\alpha = 10$.

Fig.2: Average survival probability for Redfield model, $\alpha = 10$, if only males with less than sextru mutations (as given in figure) are selected as mates. Part a assumes independent selection according to $0.9^m$, part b assumes truncation selection where more than 6 mutations kill.

Fig.3: Distribution of active genetic diseases (mutations) for the asexual and the sexual case, $\mu = 0.3$, if 5/32 of the mutations are dominant, for the bit-string model. Recessive mutations refer to the sexual case but do not reduce the survival rate.

Fig.4 Part a: Survival rate as a function of age ($T = 4$); initially 100,000 individuals, minimum reproduction age = 10, birth rate = 2 in asexual case and = 4 and 0 for females and males in sexual case ($p = 1$, $h = 6/32$). Squares: sexual reproduction, mutation rate = 1 per individual, maximum reproduction age = 32 for both sexes; triangles: same except maximum female reproduction age = 12; circles: asexual reproduction, mutation rate = 1 per individual, maximum preproduction age = 12; stars: same as circles except twice as high mutation rate. Part b shows the death rate (in two definitions) logarithmically versus age indicating the exponential increase of the Gompertz law for high ages (6.8 million individuals of each sex initially, about 10 million between 5000 and 10000 time steps; $b = 8$, $h = 5/32$, $p = 2$, $T = 4$).
Redfield algorithm versus $r$, for $h=0, 0.2, ..., 1.0$ (from top); $a=1$
Redfield algorithm versus $r$, for $h=0, 0.2, ..., 1.0$ (from top); $a=10$
Independent Selection, $a=10$

![Graph showing generation and mean population fitness for different sextruc settings.](image-url)
Truncation Selection for threshold = 6, a=10

Mean Population Fitness

Generation

sextruc=1
sextruc=3
sextruc=5
sextruc=6
sextruc=7
sextruc=8
sextruc=9
mutations

probability

sexual

asexual

recessive

mutations
\ln\left(\frac{N(\text{age}-1)}{N(\text{age})}\right) \text{ (diamonds)} \text{ and } \frac{N(\text{age}) - N(\text{age}-1)}{N(\text{age}-1)} \text{ (plusses)}