

Sexual reproduction from the male (men) point of view

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Abstract: To counterbalance the views presented here by Suzana Moss de Oliveira, we explain here the truth: How men are oppressed by Mother Nature, who may have made an error inventing us, and by living women, who could get rid of most of us. Why do women live longer than us? Why is the Y chromosome for men so small? What are the dangers of marital fidelity? In an appendix we mention the demographic challenges of the future with many old and few young people.

1 Introduction

The French revolution was based on Freedom, Equality, Brotherhood and led to the first declarations of “Droits d’Homme”. Now, unfortunately, we are oppressed by Sisterhood, which claims human rights also for women. With this oppression of men by women and of the present authors by the author of the accompanying chapter in this sociophysics school, we ask why do we exist at all? Has Mother Nature made an error when inventing sexual reproduction in addition to the older and simpler cloning (most of the time) of asexual bacteria, bdelloid rotifers, or other asexual species? As the Gena Rowlands in the Kirk Douglas movie “Lonely are the Brave” pointed out, she would not have anything to do with any male if they were not needed to produce babies.

When we deal with sexual reproduction we have in mind species with a separation of male and female individuals where only the females give birth; and asexual reproduction refers to pure cloning, without any bacterial “parasex” (exchange of DNA). Thus sexual reproduction has a disadvantage by a factor of two over asexual cloning, since males do not get pregnant (with few exceptions like Arnold Schwarzenegger in “Junior”).

2 Extrinsic reasons

Our genetic properties are stored in the DNA of the genome, and are given on to our children. The same holds for bacteria and other forms of life. During the copying of the DNA some errors may occur, which are called mutations in biology and usually make life more difficult in the form of hereditary diseases.

If the whole genome is stored only once one calls it haploid; if it is stored twice, in two sets of chromosomes, it is called diploid.

Apart from the mutations, in asexual reproduction the offspring has the same genome as the parent; for example if one bacterium splits, each of the two new bacteria has the same genome. For sexual reproduction, the diploid offspring has a mixture of the genomes from father and mother, who had produce haploid gametes (sperm cells and ovum) which were fused into a diploid zygote, growing into an embryo. Thus each child is different from its parents and its siblings (identical twins excepted). Sexual reproduction thus produces more diversity than asexual one.

This diversity is a disadvantage if biological evolution optimises the genome for a fixed environment, through selection of the fittest. Once the optimum was found one should stay with it. But physicists [1] know from trying to find the ground state of a spin glass or other frustrated structure, one often ends up in one of the many local optima and not the single global optimum. Thus to find a better local optimum, or even the elusive global one, diversity can be good. In the simulated annealing method physicists have used for such optimization, the positive temperature produced this diversity away from the local optimum. In nature, the mutations as well as the greater diversity from sexual reproduction have caused the evolution of life from simple bacteria to its pinnacle, the Herr Professor.

Also, the environment is not fixed. Temperatures have changed in the past, and if the temperature drops as in the movie “The Day after Tomorrow”, then the Inuit (=Eskimo) will simply move from Northern Canada to Florida and hunt whales from the ice swimming off the coast of Miami Beach. The greater human diversity thus allows some humans to survive this catastrophe. Had mankind been transformed into a single race of Hitler’s Aryan Herrenmenschen they would not have been accustomed to the cold and all died [2].

More explicitly for sexual versus asexual reproduction, [3] used the Penna ageing model (see our Appendix 1 or our books [4]) to compare the response of a population to a sudden catastrophe, simulated by assuming that a previously good version of a gene suddenly threatens life (like adjustment to warm climate threatens life after a sudden drop in global temperature). The asexual version was superior to the sexual one before the catastrophe but died out after it. In contrast, the sexual version was able to recover.

Another advantage of sex are parasites. They have to adjust to their host, which is easier for the asexual case with little changes of the host with time, than for the sexual case where the host changes genetically from one generation to the next. Computer simulations [5], also for the Penna model [6], confirmed the advantage of sexual reproduction quantitatively.

Thus we have good external reasons to justify the existence of sexual reproduction in real nature, due to the increased diversity.

3 Intrinsic reasons

Far less clear is the justification of sex for purely intrinsic reasons, without catastrophes, parasites, ... A simulation by zoologist Redfield [7] triggered many physics articles like [8, 9]. If mutations happen as seldomly for the male as for the female, and if one distinguishes between the rare dominant and the more widespread recessive mutations, then asexual cloning is worse than sexual reproduction in spite of having twice as many births per individual. With a male mutation rate much higher than the asexual or female mutation rate, sex loses against cloning. (Mutations are dominant if they affect us even if stored in only one of the two sets of the diploid genome, while recessive mutations affect us only if present in both sets. For asexual individuals with haploid genomes each mutation is dominant.)

However, the Redfield model, similar to Weidlich's methods in sociophysics [11], is not a proper agent-based model and treats probability distributions instead of individuals. It also does not include the ageing of individuals, an effect well known to the present authors. Thus simulations of the Penna bit-string model [10] (see appendix) are more appropriate. Here each individual is simulated in many stages from birth to death.

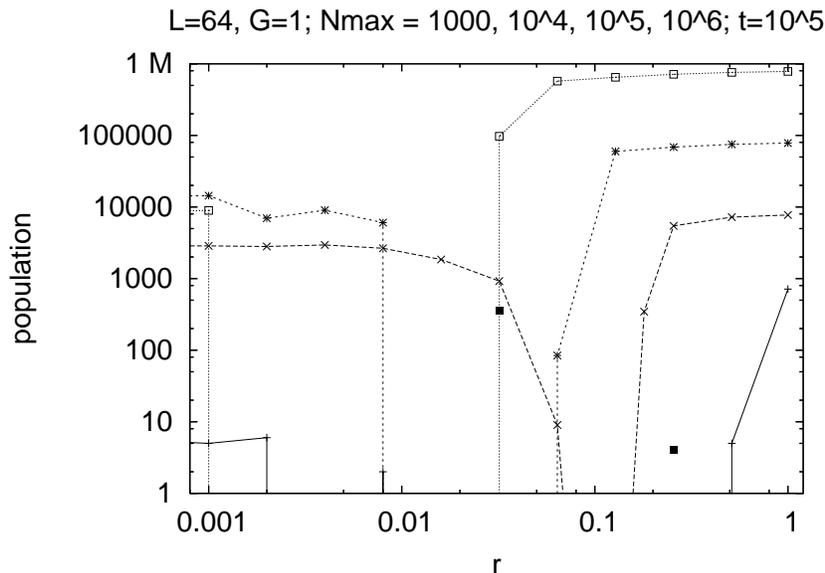


Figure 1: Sexual Penna model; populations versus recombination (= crossover) rate r for various values of the carrying capacity N_{max} . The gap at intermediate r shifts to smaller r for increasing N_{max} . To the left of the gap we have complementarity with many mutated bits; to the right we find Darwinian purification selection with much less bits mutated.

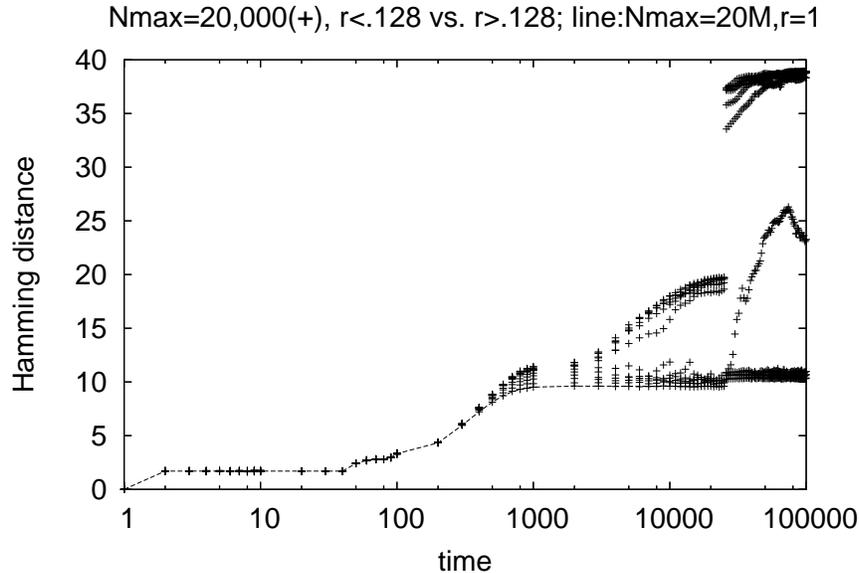


Figure 2: Illustration of complementarity and gamete recognition. For small recombination rates $0 \leq r \leq 0.016$, the average Hamming distances approach 20, and then move close to 40 after genome recognition is switched on at $t = 25,000$: Complementarity with about half of the 40 bits mutated. For $r = 0.032$ and 0.064 the population dies out, for $r = 0.128$ it does not know what it wants, and for larger r relatively few bits are mutated: purification independent of population size.

Again, also in the Penna model no clear advantage of sex was found [9] until Ref.[12] assumed that mutations (= inherited diseases) which finally kill us with certainty, already before reduce our health and increase our mortality. Moreover, F. Scharf (diploma thesis 2004, as presented on page 91 in the second of Ref.[4]) showed that preselection of sperm cells, before fusion with the ovum, may give sex an advantage since it is impossible for asexual cloning. For example, sperm cells with some genetic defect may swim slower than healthy ones and thus not reach the ovum in time.

Somewhat related is gamete recognition [19], where the ovum rejects those sperm cells for fusion into a diploid zygote whose haploid genome is too similar to the haploid genome of the ovum. This effect is beneficial if the population, due to a low recombination rate (see appendix), shows the recently discovered genome complementarity [13, 14, 15, 16]. In such a population nearly all individuals have the same bit-strings A and B in their diploid genome, thus producing haploid gametes (ovum and sperm cells) of type A only. An A sperm combined with an ovum of type A cannot survive with many mutations, since then even recessive

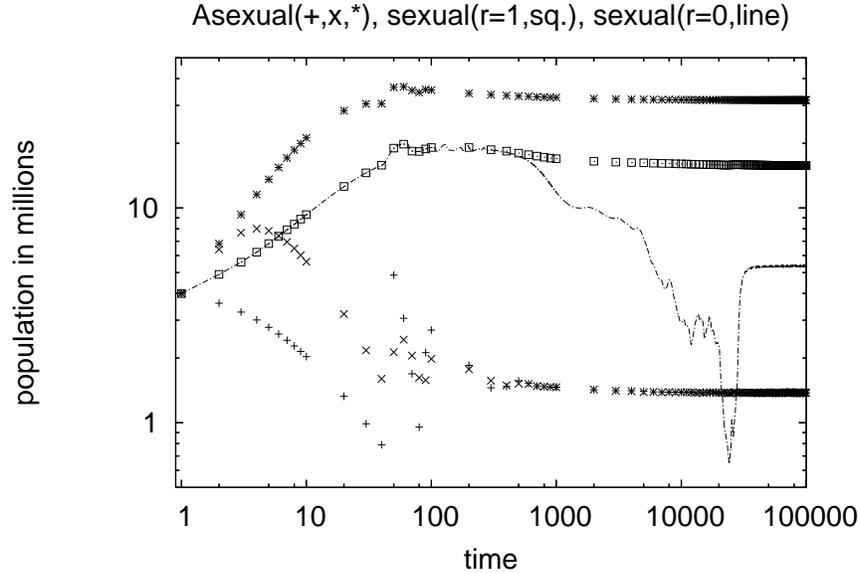


Figure 3: Comparison of sexual with asexual reproduction. The sexual case (line) is simulated with gamete recognition, for both $r = 0$ and $r = 1$, while for the asexual haploid case (other symbols) there is no crossover, no complementarity and no gamete recognition. Some asexual cases are worse but one is better than the sexual strategy.

mutations affect our health. The same happens with ovum and sperm cell both of type B. But if one is of type A and one of type B, the A-B-zygote can survive even if half of the bits (alleles of the genome) are mutated, since there is always a one-bit combined with a zero-bit and thus for recessive mutations the health is not affected. Thus high numbers of mutations can be tolerated in this sexual version, while they lead to extinction in the asexual case.

Figure 1 (after [13]) shows the two regimes of low and high recombination rates. Each curve has a gap in the middle where the population dies out. For low r the population survives with the help of the above complementarity trick; for high r it survives through purification, i.e. the usual Darwinian selection of the fittest with a small number of bad mutations. These data are obtained without the above gamete selection. If this gamete selection is added to the model then the population size to the left of the gap (small r) is strongly enhanced while the populations to the right of the gap (r closer to unity) barely change.

Figure 2 illustrates through the Hamming distances this balance between complementarity at small r and purification at large r , separated by extinction at intermediate r . These Hamming distances are the number of bits which differ in a position-by-position comparison of the two bit-strings which form a zygote,

taking into account the first 40 of the 64 bits. For complementarity without gamete recognition, the whole diploid population has two bit-strings A and B, each of which with about 20 bits zero and 20 bits one. The zygotes thus are of type AA and BB with Hamming distances 0 and of type AB and BA with Hamming distances 40; the average Hamming distance therefore is 20, as shown in Figure 2 near $t = 10,000$. The AA and BB will die out in the next iteration, the AB und BA will survive. After 25,000 iterations, gamete recognition is switched on, neither AA nor BB is allowed to form a zygote, and the Hamming distances approach 40, as shown in the interval $26,000 \leq t \leq 100,000$. For large r and purification, the number of mutated bits and thus the Hamming distance is much smaller, and the latter shows only a small jump from 9.6 to 10.3 (independent of N_{\max}) when gamete recognition is switched on.

Population versus time is shown in Figure 3, with squares for $r = 1$ and the line for $r = 0$; in the latter case, the population nearly dies out but at time = 25,000 iterations the gamete selection is switched on and the population is saved.

How does this sexual reproduction fare compare with the asexual one? Figure 3 shows that victory depend on details. In the lowest curve (+) we start with only babies; thus at first the population goes down since nobody has reached maturity yet. The second curve (x) starts with a random age distribution and thus first increases. After 1000 iterations it has merged with the lowest curve and its population stays constant even to $t = 10^6$ beyond this plot. Both cases have an equilibrium population below the two sexual curves, i.e. we have justified the existence of men.

However, this comparison is unfair. In the sexual cases the Verhulst death probability (see appendix) was applied to the babies only, while for the asexual case it applied to all ages, with a stronger reduction of the population. If in the asexual case we apply the Verhulst factor to the babies only, as we did for the sexual case, then we get the highest curve (stars), and sex is worse than asexual cloning.

Thus since 13 years [7] the story is the same: whether sex is good or bad depends on details. Moreover, we do not yet understand why diploid hermaphroditism is not much better than both. There is much to do to justify our existence: “Men of all nations unite, you have nothing to lose but your computer time.”

4 The role of inbreeding

As has been shown in Fig. 1, reproduction success depends on the interplay between the intragenomic recombination rate (crossover frequency) and the size of population. Below a specific crossover rate populations prefer to complement haplotypes instead of to intensively eliminate defective alleles. In Fig.4. we have shown how this critical crossover rate depends on the population size (M. Zawierta, personal communication). In the range of two decades there is a power law relation. Nevertheless, the data shown in the plot were obtained

in simulations of panmictic populations. In such populations females look for and choose randomly a sexual partner from the whole population. In Nature the process of choosing the partner is usually nonrandom and, what is more important, it is spatially restricted. Individuals are looking for partners in their neighbourhood. Thus, the effect of the population size should be considered as an effect of the inbreeding, rather. Inbreeding (coefficient) is a measure of genetic relations between individuals. If the individuals live in small “inbreeding” groups, then the inbreeding coefficient is high and there is a high probability that they share some fragments of the same ancestral genome.

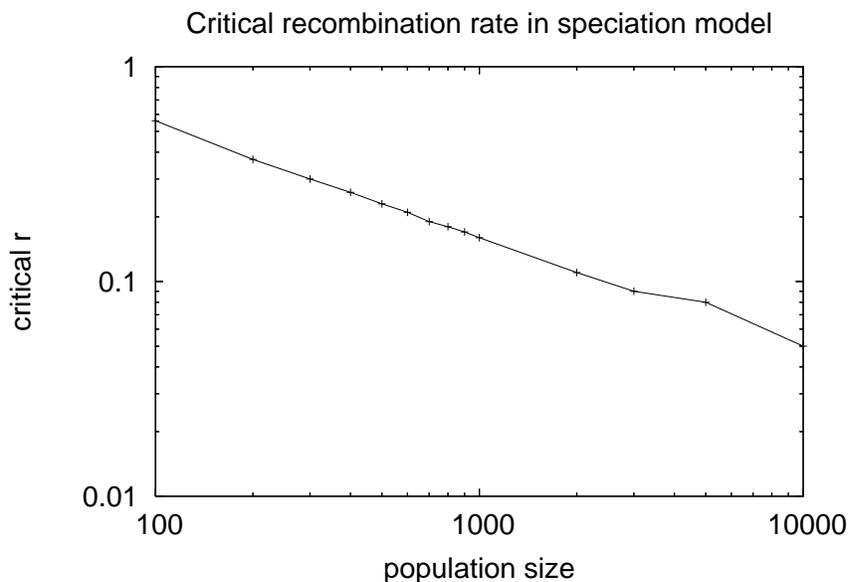


Figure 4: Log-log plot of critical recombination rate r versus population size. For r below this value, complementary haplotypes (bit-strings) are preferred.

To study the effect of inbreeding, the simulation of evolution was performed on lattices. The level of inbreeding was set by declaring the maximum distance where individuals can look for partners and where they can place their offspring [13]. The simulations were performed on a square lattice 1000×1000 . If the above distances were set to 5, the critical crossover rate was around 0.2. Populations evolving under lower recombination rate or shorter distances prefer the strategy of complementing the haplotypes while under higher recombination rate or longer distances they choose the strategy of purifying selection. Nevertheless, there are very important consequences of such a kind of choice. The complementarity evolves locally and remote subpopulations on the same lattice can have different distribution of defective alleles in their haplotypes. Using some tricks with coloring the individuals according to their genomes’ structure

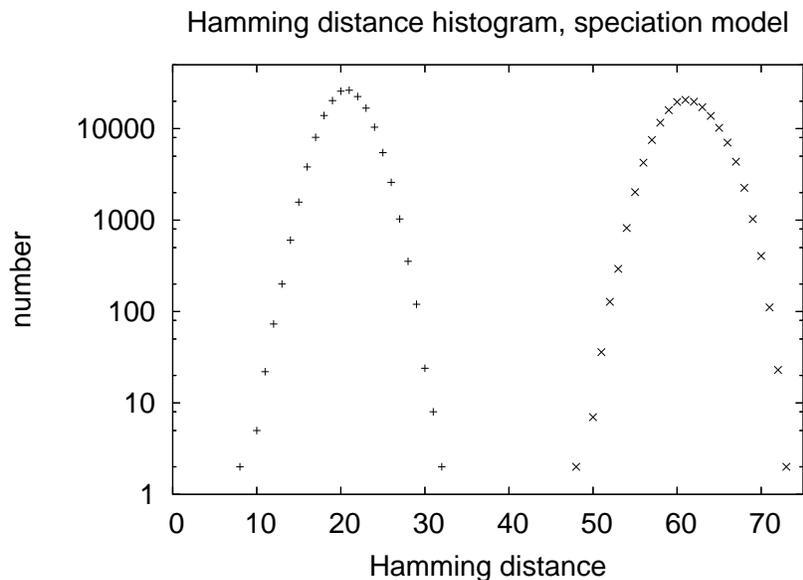


Figure 5: Histogram for Hamming distances within one species (left peak) and between different species (right peak). The bit-strings had 128 bits.

it has been shown that the lattice is occupied by individuals with different genotypes but they are clustered. Individuals with the same genotypes occupy the same territory (see <http://www.smorfland.uni.wroc.pl/sympatry/> for some examples of simulations under different conditions). In Fig.5 we have shown the Hamming distances between corresponding haplotypes (not complementary, in the description on page 4 they correspond to pairs of haplotypes AA and BB). The similarity of haplotypes inside a species is high while between species it is low. Further studies have shown that for the clustering only the central part of the genome is responsible. The lateral part of the genome is much more polymorphic and decides on biodiversity, rather than speciation. That is why the Hamming distances between homologous haplotypes inside species are noticeable. These simulations show that sympatric speciation is possible and there is no need for physical, geographical or even biological barriers for the new species to emerge inside the population of the older one.

5 Why do women live longer than us?

Of course, we die sooner because women oppress us. But since this truth cannot be published in detail (just because it is true), we have to find other reasons. The senile author is so thin and close to starvation because women eat his steaks and drink his beer. But since at least for rodents, caloric restriction prolongs

life (if one can call that life), this example may not be convincing.

One genetic reason could be the difference between the two X chromosomes for women, compared with one Y and one X chromosome for men. The Y chromosome, in comparison with the X chromosome, contains very little information. Thus if a mutation creates an error in the single male X chromosome, the correct information is lost. An error in one of the two female X chromosomes, on the other hand, can still be balanced by the correct information on the other X chromosome. Quantitative simulation [20] in a Penna model with many chromosomes gave good agreement with reality: Male mortality is about twice as high as female mortality, except that the two get relatively close at very old age.

Mammals share this chromosome difference between male XY and female XX, while for birds the situation is reversed: Same chromosomes for the males and different ones for the females. Thus the above argument would mean that male birds live longer than their female counterparts. The empirical observations are contradictory, as reviewed in [4, 21].

Male sleep is often disturbed, e.g. by a dean in faculty meetings, while their wives can calmly deal with many children and household chores at home. Does this stress kill us? Similarly, the stress of the industrial revolution was held responsible for the mortality difference [22]. But why do already in the first years of life the boys die more often than the girls? And why did Swedish men die sooner than Swedish women already 230 years ago? Why are there important differences between different industrialized countries in the male-female difference of life expectancies? More literature, but not more answers, are found in [21].

Women should not be trusted anyhow, as the biblical story of Adam, Eve and the snake tells us. This is particularly true for mortalities, defined as the negative age derivative of the logarithm of the number of survivors at the given age. Adult men obey nicely the Gompertz law of 1925 that the mortality μ increases exponentially with age, Fig.6. Women, in contrast, follow it only for middle ages; in old age their mortality increases stronger with increasing age, until for centenarians the ratio of male to female μ is close to one, Fig.6. These lawless women then misled some into the belief that there are strong downward deviations from the Gompertz law: “mortality deceleration”; just look at the \times symbols in Fig.6 for ages 70 to 100. The true Gompertz region for women is 30 to 70 years, and a straight line extrapolated from there to older ages gives upward, not downward, deviations from the Gompertz law. Only for ages beyond Fig.6 a mortality plateau may appear [23].

6 Genetic marriage counseling

1. One of the attributes of life is reproduction. Simulations of the age-structured populations using the Penna model show that if we set the upper age limit for reproduction, we simultaneously set the maximum lifespan, like in case of pacific salmon. Selection doesn't care about individuals which cannot reproduce any

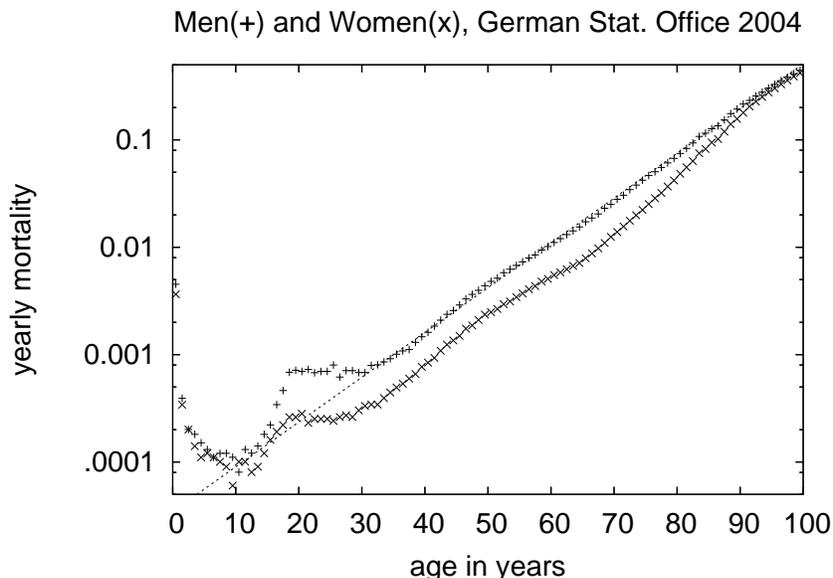


Figure 6: Comparison of male and female mortality function μ for modern Germany. The straight line indicates a Gompertz law [21]. No mortality deceleration is seen for adult men or women up to 100 years of age. Data from 2003-2005 published in 2006.

more, and the genes necessary for them to survive beyond the reproduction period accumulate mutations which kill the individuals.

2. The above example corresponds to female menopause and a male condition called sometimes andropause if both are set in the model at the same age. Usually it is not true for real life. Women exercise menopause earlier than men do andropause (if it is true that there is something like andropause). In fact, men's reproduction ability does not stop in the middle ages and men can reproduce late in their life. Menopause is a first-order phase transition while men's reproductive ability slowly decays.

It could be modeled just by setting the menopause (the upper age limit for reproduction) only for women. In such a case the life expectancy for both women and men stays the same as in the model version without menopause. Why? Because men at the higher age can reproduce and their genes expressed after the age of menopause of women are under selection, i.e. bad genes expressed at old age spread in the population less easily than good genes. These correct genes could be transferred to the daughter's genomes resulting in the higher life expectancy of women. One can obviously conclude that women live longer due to the cruel selection experienced by men. However, life is not so simple. Men and women use to live in pairs and usually they swear to be faithful not

only until the menopause of the woman but to the end of their life. Thus, when the wife reaches menopause the husband should simultaneously reach the unreproductive age, too. In such a case we are back to the point 1: both sexes have the same upper limit of reproduction period, given by female menopause, and they should die immediately after it.

There could be a few explanations of this inconsistency with reality:

- motherhood (child care) is necessary for higher chance of the offspring survival [17],
- even a grandmother is necessary to increase the reproduction potential of her children [18] (but see sec.3.5.3 in [4]b).
- older men are more attractive for young women when the latter ones are looking for partner (see e.g. the movie “The First-Wives Club”),
- men have to be not faithful to their wife in menopause to secure the longer lifespan of women (just read the newspapers).

One can notice, that there is no big difference between the last two items. If a husband betrays his wife, then he should do this with younger women to succeed in the prolongation of the human lifespan for everybody. The only problem not solved until now is what should be the fraction of men unfaithful to their wives after menopause to produce this effect.

Men live shorter than women, Section 5. There are a lot of hypotheses (even more than hypotheses) explaining this phenomenon [4]. One of the explanations is the role of men in cleaning out the genetic pool of the human population of defective genes. The good and well-described example is the role of X chromosome in this process. It is a rather large chromosome with high number of genes. There are two copies of this chromosome in the woman’s genome and only one copy in the man’s genome. Recessive defective alleles in one X chromosome in the female genomes can be compensated by the corresponding correct alleles while male genomes have no such a possibility. Each recessive defective X allele is seen for men like a dominant one and it is eliminated by the purifying selection. Men should not complain about their situation because Nature can be much more cruel. It produces male organisms which clean up not only one chromosome but the whole genome, like drones in the bees society. Drones are haploid and they have to be perfect to survive. Sometimes drones are considered as extremely selfish individuals, nothing is more wrong, they are exceptionally altruistic.

The stronger selection on genes located on X chromosomes in men’s genomes leads to a significantly lower level of defective genes in the X chromosome when compared with autosomes (all chromosomes except X and Y) [29]. That is why the probability of disorders caused by defective alleles located on X chromosomes in women is much lower than in men. As mentioned above, this purifying selection results in higher mortality of men what is observed in human populations as well as in simulations [20], especially in the middle age [30]. In the

standard Penna model a relatively low fraction of genes is expressed before the reproduction age. In Nature, the fraction of genes expressed during development up to the puberty is probably much larger. Moreover, a large proportion of these genes are expressed before the birth, what is usually not considered in the modeling. To reach the level of spontaneous abortion as in the natural human population (about 60 %) [31], [32] a substantial fraction of genes have to be switched on before the birth. When such genes were introduced into the model, the other effect emerged; among these genes some genes located on X chromosome were also expressed causing higher mortality of male embryos. Since the sex ration in human populations at birth is close to 1, one should assume that for compensation the higher mortality of male embryos during the pregnancy, the sperm cells containing Y chromosome should have about 50 % higher probability of fertilizing the egg than sperm cells containing X chromosome [30]. This is a plausible hypothesis because Y chromosome is smaller and the cells containing it could be faster.

On the other hand men should not complain about their role because they themselves decided about that, putting the genes responsible for sex differentiation into one copy of the sex chromosome. As [24] has shown, this chromosome (known as Y chromosome) has to shrink during the process of the genome evolution. [24] proposes a very plausible explanation of this process of shrinking noting the role of selection which acts on different number of sex chromosomes; 3 X chromosomes for each one Y chromosome in the genetic pool. Additionally X chromosomes can recombine in the female genomes while a Y chromosome never has a partner for recombination. Nevertheless, there could be another explanation; men's genomes are under much weaker selection pressure than female genomes because the reproduction potential of the population depends on the fraction of female individuals. Women are promiscuous and they can seduce men which already have children with other women. Thus, to give birth the woman is indispensable but a large fraction of men is dispensable (see the end of the Kubrick movie "Dr. Strangelove"). And in fact, these fractions of men which really have no children are not under the selection. All chromosomes exercise alternatively the selection in the male/female genomes with one exception — Y chromosomes evolving in the mostly dispensable men bodies, Fig.7.

To prove that the women's promiscuity is responsible for shortening the men's life, some additional changes have been introduced into the Penna model [33]. It has been assumed that man is indispensable for baby survival and he has to stay only with one woman during the period of her pregnancy. It means that the reproduction potential of population depends equally on both, the fraction of females and males in the population, though they need not swear to be faithful for life. In such a case the shrinking effect of Y chromosome disappears, Fig.8.

Geneticists call the males a heterogametic sex because they produce two different gametes (with X and Y). In all mammals males are heterogametic but in birds females are heterogametic. To underline the difference in sex determination geneticists call for birds the sex chromosome of males ZZ and of females ZW. In the species with such a type of sex determination the shrinking of W chromosome is not observed in the nature neither in the model. Moreover, it

seems that in such species the faithful males are losers.

Since not all men are necessary to keep the reproduction potential of the population at high level, some of them, especially younger men with their genomes not tested yet by life, can be altruistic and sacrifice their lives [25] for other members of the society. That is why we have armies and it is not wise to enroll women into the army because such a procedure diminishes the reproduction potential of the population.

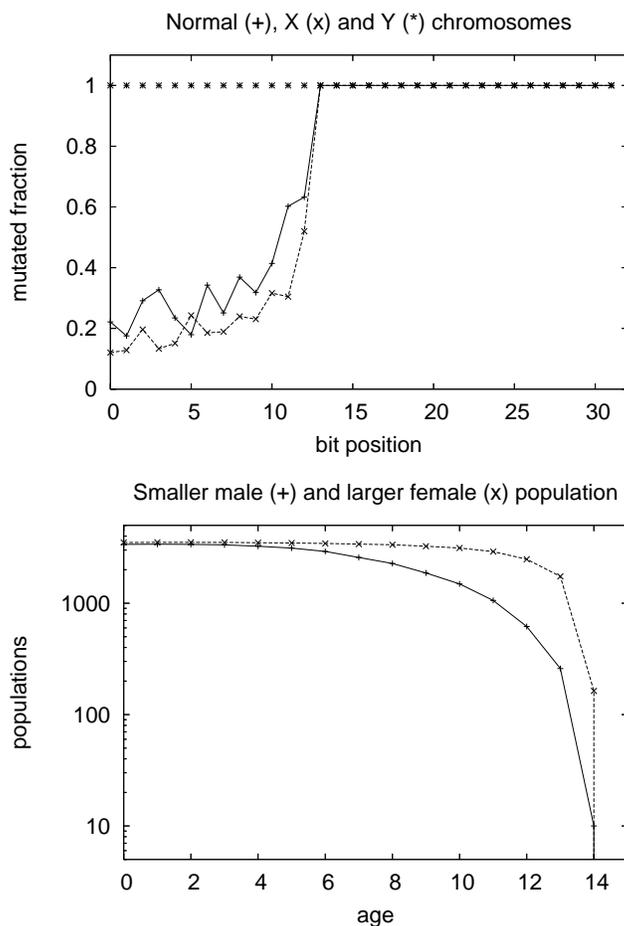


Figure 7: Top part: Fraction of defective genes in the autosomes and the two sex chromosomes if woman can seduce a man who already have been chosen by other woman as a sexual partner. “Autosomes” are the “normal” chromosomes which are not the X or Y sex chromosome. Bottom part: Age structure of females and males in the above population. From [33].

To model the necessity of the male altruism, Bońkowska [34] introduced into

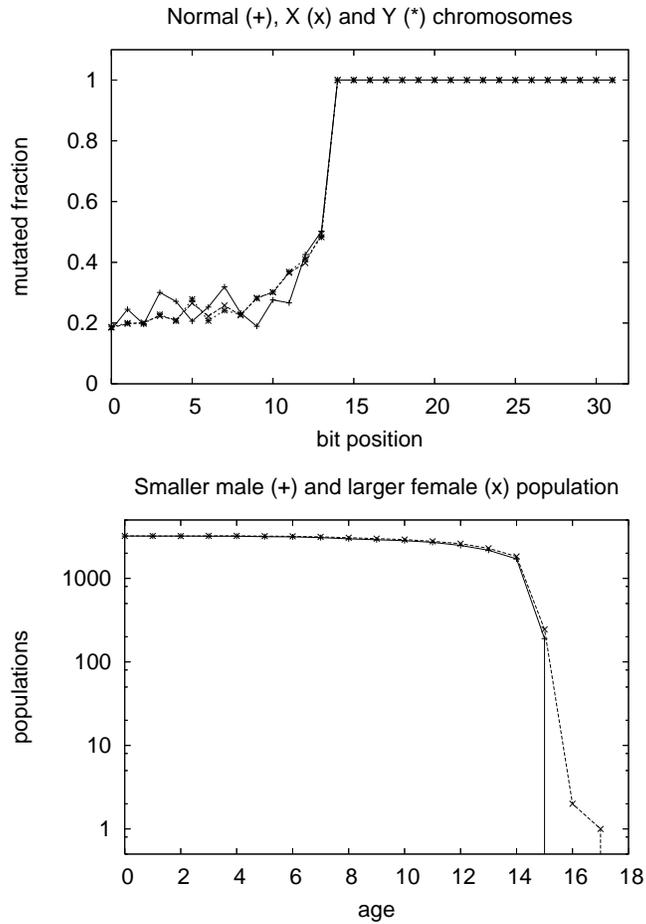


Figure 8: As preceding figure but for faithful men. The Y chromosome now behaves like the other chromosomes. From [33].

the Penna model the random death for specific sex at specific age (random in this case means that individuals at a declared age and sex could be killed with a declared probability independently of their genetic status). These individuals, in some instances could be saved by other individuals but then, the savior has to die. She described the parameters of four populations:

- females at age 10 are randomly killed with probability 0.2 and nobody tries to save them,
- females at age 10 are killed randomly with probability 0.2 and males at age 90 try to save them with probability 0.5. If a man succeeds in saving the oppressed young woman - he dies,

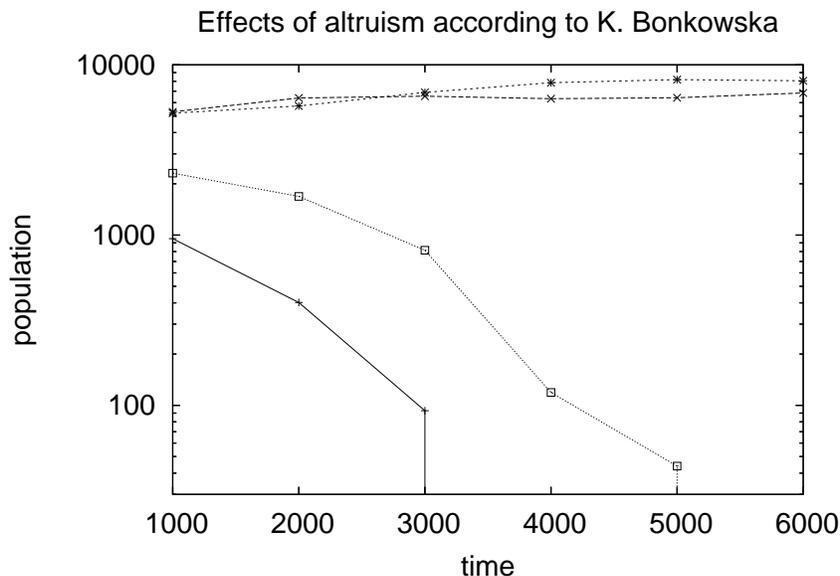


Figure 9: Growth and decay for the four altruism choices: Women sacrificed (+), women saved by men (x), men sacrificed (stars), men saved by women (squares).

- males at age 10 are randomly killed with probability 0.2 and nobody tries to save them,
- males at age 10 are killed randomly with probability 0.2 and females at age 90 try to save them with probability 0.5. If a female succeeds in saving the stupid man - she dies.

Other parameters of simulations are the same for all four populations: the length of the bitstring is $L = 128$, minimum reproduction age 80, menopause 110, birth rate 0.2. Simulations were started with the same size of each population. All four populations were placed in one environment where they have to compete. The results are shown in Fig.9. The results are so pronounced that they need a special attention. The worst situation is when women die randomly without any help. This decreases the population reproduction potential and the population loses in competition. The other losing strategy is when women try to save men. It is much better if the oppressed women may count on the male altruism; then the population is almost as good as when a part of men are eliminated randomly. The evolution of altruism is much more complicated because it depends on the age at which the men sacrifice their life (not shown here). It is better if they are younger, that is why one can hardly find an old general in the front line.

All these examples show how crucial is the role of male genomes in the human genetic pool evolution. But there is one puzzle. It is assumed that the mutation rate per one cell generation (per genome replication) is constant [26]. It has been estimated that the number of cell divisions from zygote to the sexual maturity of woman (production of the egg in the oogenesis in the female germ line) is 24 according to [27] and it is roughly the same for all eggs produced by a woman during her whole reproduction period. The corresponding process of sperm production (spermatogenesis) is quite different. The number of cell divisions during the spermatogenesis for 20 years old men is estimated as 150 and increases by 23 each year [28]. The resulting higher mutation numbers in male gametes was one reason to warn against the too high cost of sex for females [7].

One can expect that the mutation rate per male gamete should grow very fast linearly with age. The experimental data are inconsistent; for some genes the sex bias in the mutation rate is observed while for some other genes is not. This is also contradictory with the results shown in the above sections which clearly indicate that the role of the older men is very important in keeping the longer lifespan of humans of both sexes. That is why it could be biologically legitimate to assume that there is the above-mentioned gamete preselection during or before the fertilization.

It would be completely wrong to assert that we are against women. We are very grateful for them to have reverted the 2002 final of the male-football world cup. The senile author thanks the senior editor for movie education. We thank COST P10 for supporting the visit of DS.

7 Appendix 1: Penna ageing model

The Penna model of mutation accumulation describes well the biological ageing, with a mortality increasing exponentially with adult age, and has been reviewed in detail in [4]; the present description is adapted from [13]. The sexual population is composed of a changing number N of individuals, each represented by its diploid “genome” corresponding to two bit-strings (haplotypes) $L = 64$ bits long. At an age x , only the first x bits are active. Bits set to 0 or 1 correspond to the correct (wild) or defective genes, respectively. Only if both genes are set for 1 at the same position on the bit-strings, the effect of the „locus” (bit position) on the individual (having reached at least the age corresponding to the bit position) is bad and the individual dies because of the genetic death. Thus all defects are recessive and one single active mutation kills, and the older the individual gets, the more bit positions are checked for possible defects.

Females try to give $B = 2$ births if they have reached at least the minimum reproduction age $R = 40$ and found a male partner, randomly selected from the whole population, who is also old enough. Each attempt succeeds with probability $N(t)/N_{\max}$, which is the Verhulst factor due to limited food and space. To give a birth, the female genome is replicated, during the replication one new

mutation is introduced for each copy of bit-string into a randomly chosen locus. If the bit at the locus is 0 it is replaced by 1, if it is 1 it stays 1 (there are no reversions). The two copies of bit-strings recombine with probability r at a randomly chosen point just by exchanging the corresponding arms (crossover). After these processes, each of the two new bit-strings corresponds to a gamete. A randomly chosen female gamete is joined with another gamete produced in the same way by the male partner. The pair of gametes corresponds to the newborn's diploid genome. The newborn's sex is established with equal probability to be male or female. (Some parameters were selected differently in [14, 34, 33].)

Sex is complicated; the asexual version has only one bit-string, and no recombination. Fortran programs are published by Moss de Oliveira et al [4].

8 Appendix 2: Future demographic problems

As the reader may have noticed, these authors became old. Over most of the world, life expectancies have increased and birth rates have fallen. Who will feed us when we become even older?

Some people claim that demographic predictions of the future are just propaganda to help the government to reduce old-age pensions. Similarly one could claim that warnings of man-made global warming is just propaganda for nuclear power plants; and if there is really more sunshine, we just buy more sunglasses. Of course, any prediction of future ratios of old to young people is just theory and could be wrong. Similarly, the assertion that all people die at some time, or that the sun will rise tomorrow, is merely theory. It becomes invalid if an immortality gene is found and activated, or if Bruce Willis fails to prevent the impact of a huge meteorite on Earth.

Future age distributions have been simulated with the Penna model in [35]. Such simulations take into account heredity and slow changes in the human genome. If we restrict ourselves to the 21st century, the hereditary correlations between mother and daughter do not have enough time to change, and instead of an agent-based simulation [35] of individuals one may evaluate the changes in probability distributions as in [7], which is much simpler [36, 37, 38, 39, 40, 41].

(The difference between the proper way [35] and the approximation by distributions [36, 6, 39, 38, 40, 41] can be explained by a simple example. Let the present population consist of two groups such that on average, women have 1.9 children in one and 2.1 children in the other group, and let us assume that this difference is propagated culturally or via mitochondrial DNA from mother to daughter, without any change. Then, after many generations most of the population will belong to the group with the higher number of births, but that effect is ignored if only one age distribution is simulated. However, until the year 2050 we do not have that many generations to get this domination of one group, and a simulation with an average number of 2.0 children per women remains sufficient.)

Also, babies who die in their first years neither require schooling nor pensions and may be neglected in a simulation of the number of people in retirement age

to the number of people in working age. The birth rates and the mortalities put into such a simulation are thus those for adults: How many people reach the working age (20 years, for example), and how many reach retirement age. Finally, the number of people older than 110 years, when mortality might reach a plateau, is still very small (one in a million for West-Central Europe) [23]. Thus we can use the Gompertz law, Fig.6, that mortality increases exponentially with age. Either the medical progress since two centuries leads to a rectangularisation of survival probabilities, as a result of which asymptotically we will run Marathon races in two hours at the age of 102, and die of old age within the following year. Or, alternatively, the survival curves since about 1970 shift to older age without changing anymore their shape [42]. Simple Fortran programs using this alternative are published in [40] and [4]b.

The results are roughly the same in the various types of simulated countries [43]: If the number of children per women (fecundity; often misleadingly called the total fertility rate) sinks far below the replacement level near 2.1, problems appear decades later when the ratio of retired to working people becomes very high. When that will happen depends on the time development of this number of births, which is near 1.3 in Germany [36, 38] since a third of a century (similarly in Poland [41] since a shorter time), increased from 1.7 to 1.9 in France from 1995 to 2005, is close to the replacement level in Algeria [39] and is still much higher in the Palestinian territories [40].

Possible remedies are immigration [36], increases in retirement ages [6], more work by women [40] or an increased birth rate [41]: Romania nearly doubled the number of births per women, for a short time around 1968. (These last choices allow to make women responsible for our problems.) Only people were simulated, not money, since the buying power of pensions may be changed by law or by inflation.

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