

# Does sex induce a phase transition?

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**Abstract.** We discovered a dynamic phase transition induced by sexual reproduction. The dynamics is a pure Darwinian rule applied to diploid bit-strings with both fundamental ingredients to drive Darwin's evolution: (1) random mutations and crossings which act in the sense of increasing the entropy (or diversity); and (2) selection which acts in the opposite sense by limiting the entropy explosion. Selection wins this competition if mutations performed at birth are few enough, and thus the wild genotype dominates the steady-state population. By slowly increasing the average number  $m$  of mutations, however, the population suddenly undergoes a mutational degradation precisely at a transition point  $m_c$ . Above this point, the “bad” alleles (represented by 1-bits) spread over the genetic pool of the population, overcoming the selection pressure. Individuals become selectively alike, and evolution stops. Only below this point,  $m < m_c$ , evolutionary life is possible. The finite-size-scaling behaviour of this transition is exhibited for large enough “chromosome” lengths  $L$ , through lengthy computer simulations. One important and surprising observation is the  $L$ -independence of the transition curves, for large  $L$ . They are also independent on the population size. Another is that  $m_c$  is near unity, i.e. life cannot be stable with much more than one mutation per diploid genome, independent of the chromosome length, in agreement with reality. One possible consequence is that an eventual evolutionary jump towards larger  $L$  enabling the storage of more genetic information would demand an improved DNA copying machinery in order to keep the same total number of mutations per offspring.

**PACS.** 05.70.Fh Phase transitions: general studies

## 1 Introduction

In the Eigen quasispecies model, the genome of an individual is modelled by a string of  $L$  bits [1]. If all bits are set to zero (or to some arbitrary sequence defined as ideal), its fitness measured by the number of offspring is the largest; with some mutations its fitness is reduced. This model leads for large  $L$  to the possibility of a transition such that for small mutation rates an appreciable fraction of the population has the ideal genome, while for large mutation rates it leads to a mutation explosion (often called “error catastrophe”) where an appreciable fraction of all bits are mutated and similarity with the ideal bit-string is lost. Life cannot be stable under mutation explosion. We generalise this model here to an agent-based simulation with sexual reproduction and compare its results with the agent-based asexual model [2]. The main difference turns out to be that for the asexual case the crucial quantity is

the mutation rate per bit while for the sexual case it is the mutation rate per bit-string.

The theoretical question posed in this work concerns the length-scaling properties of chromosomes. Let's call  $L$  the chromosome length, an integer number measuring the number of coding units along the chain, which for simplicity we consider as a bit-string: 0-bits represent the wild alleles, whereas 1-bits correspond to harmful mutations, the “bad” alleles. The larger this length  $L$  is, the larger is the space to store more genetic information. Therefore, in principle, evolution should lead to species with larger and larger chromosomes, of course with the same value of  $L$  for all individuals belonging to the same species.

Consider first a simple case of haploid individuals which reproduce through cloning. The chromosome of each newborn is copied from an already alive individual, taken at random, plus an average fixed number  $m$  of point mutations. Being an average over all newborns, this number  $m$  is not necessarily an integer, it can be tuned

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continuously as explained later. One point mutation means a 0-bit in the parent's chromosome which is flipped into a 1-bit in the offspring's, or vice-versa. The position where this mutation is performed is random. The wild genotype corresponds to a bit-string where all bits are set to zero. A mutation in the sense  $0 \rightarrow 1$  makes the offspring farther from the wild genotype than its parent, another in the reverse sense makes it closer. A fixed birth rate  $b$  defines the probability of each individual to produce an offspring each new time step.

Let's ignore any kind of correlation along the chromosome, i.e. the fitness of individual  $i$  depends only on a single phenotype defined here as  $N_i$ , the total number of 1-bits in its genome. One individual with phenotype  $N+1$  is at a disadvantage, when compared to another individual with phenotype  $N$ . The disadvantage here corresponds to a smaller survival chance: the probability to survive a new time step is smaller for the former individual by a factor of  $x$ , when compared to the latter, where  $x$  is a number strictly smaller than 1. Therefore, the survival probability for different individuals decreases for increasing  $N$ . This number  $x$  measures the overall selection pressure, and can be tuned in order to keep the population size constant, i.e. to keep the death rate equal to the birth rate  $b$ . After evolving for many generations the distribution of phenotypes stabilizes. In order to keep the wild genotype (the only for which the phenotype is  $N=0$ ) inside this equilibrium distribution, the number of mutations  $m$  cannot be too high.

Let's now compare different chromosome lengths. One can follow a simple and intuitive reasoning:

- 1) the length  $L$  is increased;
- 2) the same ratio  $m/L$  is kept;
- 3) after many generations, the steady-state population presents the same distribution of phenotypes *versus*  $N/L$ , independent of the (large enough) chromosome length.

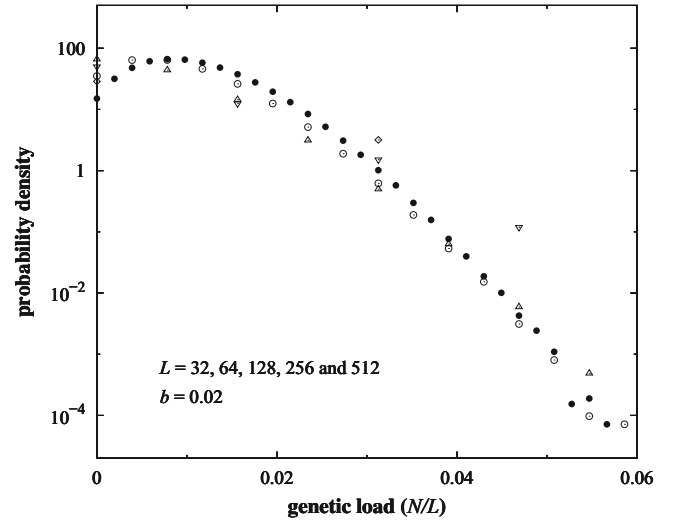
This expected behaviour is exactly what is obtained by simulating this simple haploid, asexual model on a computer [3,4]. Figure 1 shows an example of this behaviour [2].

The above-mentioned item 2) deserves an important remark: the genetic storing media (the bit-strings) are one-dimensional objects. Therefore, the average number  $m$  of mutations should be scaled proportionally to  $L$ . As a result, the whole genetic distribution curve and consequently both its average  $\langle N \rangle$  and its width  $\langle \Delta N \rangle$  also scale proportionally to  $L$  (note the collapsed distribution curves in Figure 1 plotted *versus*  $N/L$ , not  $N$ ).

The reasoning and the corresponding simulational results do not cause any surprise. The purpose of this work is to study a similar reasoning for sexual, diploid reproduction. Let's pose the first question.

Should the same ratio  $m/L$  be kept for increasing chromosome lengths?

The answer to this simple question is not so simple. Intuition can betray who thinks about it. Sex deals with half the genetic information inherited from each parent, a nonlinear behaviour which requires prudence to avoid false



**Fig. 1.** Collapsed distributions of the individual genetic loads  $N/L$ , for haploid, asexual reproducing populations with different chromosome lengths. The probability density plotted along the vertical axis is proportional to the number of individuals sharing the same  $N$ . The full circles correspond to the largest length  $L = 512$ . The mutation rate  $m/L = 1/320 \approx 0.003$  is the same for all lengths, as well as the population size  $P = 10\,000$ .

conclusions. Moreover, a crossing-over performed with homologous chromosomes within each parent's genome indicates that now the genetic information is no longer stored along strictly one-dimensional objects: we should not trust the linear reasoning leading to the fixed ratio  $m/L$ . Dominance and recessiveness are further sources of doubts. In order to answer this and many other related questions, we present in the next sections the results obtained from computer simulations of a population dynamics. Compared to reality, the model is simplified in order to retain only the fundamental features of sexual, diploid reproduction, a pure Darwinian evolutionary rule with two basic ingredients: random mutations and crossings-over which tend to increase the entropy (or diversity); and natural selection which acts on the opposite sense by removing from the population many of these mutations and, consequently, preventing entropy explosion.

Section 2 treats the implementation of the model on computers and its details. The following Section 3 deals with the most important, conceptual results. Further results are in Section 4.

## 2 The model

The population size is artificially kept constant with  $P$  individuals, by killing a fraction  $b$  of them per time step and restoring the same fraction with newborns which are offspring produced by the survivors. The set of  $P$  individuals is considered a random sample picked from a much larger population which can fluctuate in size, according to the selective dynamics. This is particularly important in case of extinction which occurs in the non-evolutionary phase

discussed later. We verify that  $P = 10^3$  or  $10^4$  is large enough to make the statistical fluctuations satisfactorily small for all quantities we have measured from our simulations. This  $P$  is also large enough to avoid inbreeding depression [5,6]. We adopted  $P = 10^4$  and  $b = 0.02$  (2%). The precise value of this fraction  $b$  is not important, provided it is small enough, because it is the artificial time step of the dynamics. The value  $b = 0.02$  simply means one needs to wait for 50 time steps in order to complete one generation replacement on average. We have also tested  $b = 0.01$  and  $0.03$  in some cases, with the same results.

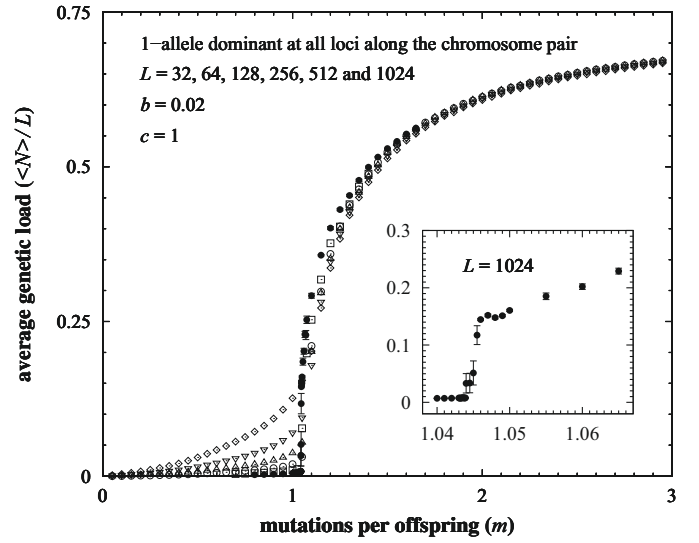
The genetic information of each individual is kept on the computer memory in two parallel bit-strings with  $L$  bits each. We tested  $L = 32, 64, 128 \dots 16384$ . We also keep on memory the histogram  $H(N)$  counting the current number of individuals sharing the same  $N$ , the number of homologous loci containing at least one copy of the “bad” allele, bit 1. This is the version where the 1-allele is dominant along the whole genome, exemplified in the next section. An alternative version, where the 1-allele is recessive, is treated in the last section. At each time step, the first process is the killing roulette, where each individual  $i$  survives according to a probability  $x^{N_i+1}$ , as assumed in numerous studies of population genetics. The number  $x$  measures the survival probability for individuals with the wild genotype,  $N = 0$ . For the others, the survival probabilities exponentially decay with  $N$ . This is the model’s selection ingredient. Note that  $x$  must be *strictly* smaller than 1, otherwise all individuals will survive forever.

We follow the decades-long tradition of Eigen model studies of keeping the population constant, at least on average. For this purpose the value of  $x$  is tuned in order to keep the population sample constant in size, and can be obtained by solving the polynomial equation

$$\sum_N H(N) x^{N+1} = P(1-b) \quad (1)$$

before killing anybody. (As a technical remark, we cannot use the solution of this equation if it surpasses some upper bound, say  $x_{\max} = 0.999$ : this limit simply imposes that every individual must die some day, no matter how good is its genetic patrimony. Extinction is the consequence of imposing this upper bound, as we will comment in the next section.) After the proper value for  $x$  is known, we scan the population,  $i = 1, 2, 3 \dots P$ . A random number  $r_i$  inside the interval  $(0, 1)$  is tossed for each individual  $i$ : if  $r_i < x^{N_i+1}$  it is kept in the population, otherwise it dies.

After deaths, the next task is to include  $(1-b)P$  newborns into the population. This is the diversity model’s ingredient. In order to construct a newborn diploid genome, first we toss two random parents among the survivors. (For simplicity, we do not consider different genders.) One parent’s chromosome pair is copied and the following procedure is performed on the copy. An average number  $m$  of random mutations are introduced. Each mutation acts at a random position along one of the two chromosomes, also taken at random. The corresponding bit is flipped from its current state, i.e. from 0 to 1 or from 1 to 0. The fixed number  $m$  is not necessarily an integer, as fol-



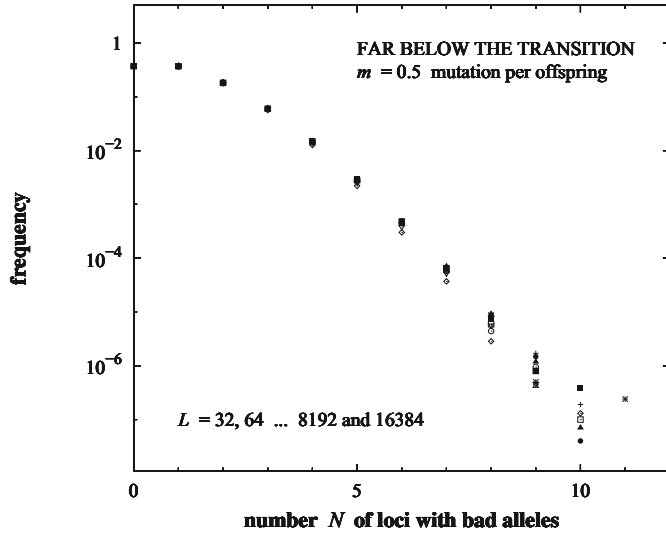
**Fig. 2.** Phase transition for a population of 10 000 individuals. For few enough mutations at birth, on the left side, life is possible. Beyond  $m_c = 1.0439$ , on the right side, the population displays a mutational explosion where all individuals carry a number of “bad” alleles proportional to the chromosome length  $L$ . For the largest length,  $L = 1024$ , the inset blows-up the transition region.

lows. We toss a random number  $M$  inside the interval  $(0, 2m)$ . Then, we perform just  $\text{int}(M)$  mutations, where  $\text{int}(\dots)$  means the integer part of the argument. After that, with probability  $\text{frac}(M)$  we perform a last mutation, where  $\text{frac}(\dots)$  means the fractional part of the argument,  $M = \text{int}(M) + \text{frac}(M)$ . Also a total of  $c$  crossings-over are performed on the diploid genome, where  $c$  is not necessarily an integer as well:  $\text{int}(c)$  crossings are performed first, and a last one with probability  $\text{frac}(c)$ . The crossing position along the diploid genome is also tossed at random. After the whole process of mutations and crossings, we have two possible gametes. We choose one of them, also at random, to be passed on to the newborn. The same process is performed on the chromosome copies of the other parent, leading to the second newborn gamete. Then, it is included into the population.

In short, our model incorporates natural selection in the step of deaths, and creates genetic diversity in the step of births, completing in this way the Darwinian selection-diversity paradigm.

### 3 Conceptual results

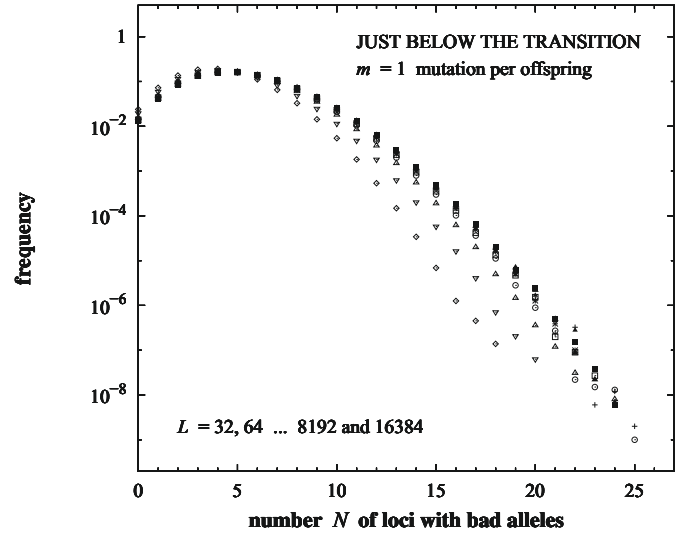
As we shall see later, the eventual result of this dynamic process can be divided into two opposed possibilities, depending on the fixed number  $m$  of mutations performed at birth. Small values of  $m$  define the evolutionary phase: the population continues its evolution forever. Large values of  $m$  correspond to the non-evolutionary, extinction phase. Figure 2 displays an example of such a phase transition, the evolutionary phase on the left side, extinction on the right.



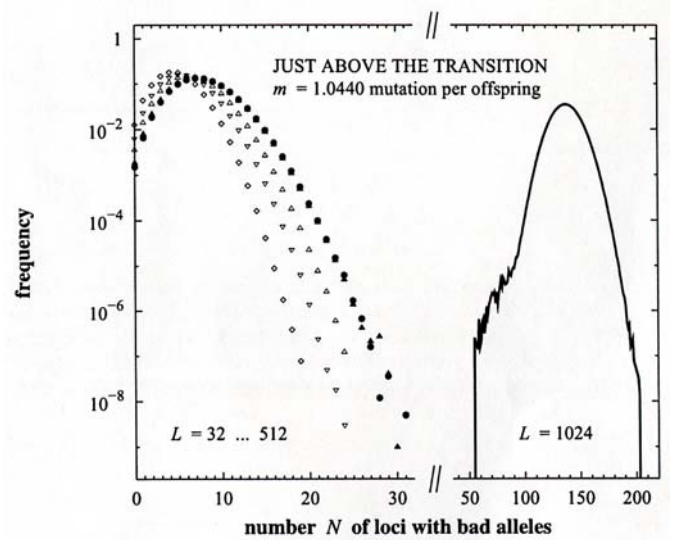
**Fig. 3.** Collapsed distributions of “bad” alleles among the population, for different chromosome lengths. Note that  $N$ , displayed along the horizontal axis, is *not* divided by  $L$ , contrary to the asexual case, Figure 1. In all cases, the number  $m$  of mutations per offspring performed at birth is fixed far below the transition value observed in Figure 2, i.e.  $m = 0.5$ , again not divided by  $L$ . Would we fix the same mutation rate  $m/L$ , instead of  $m$ , the plots would no longer collapse onto each other. Moreover, in this case, for large  $L$  the curves would undergo a run-away to the right as soon as the value of  $m$  surpasses the critical point  $m_c = 1.0439$  of Figure 2, as explained soon. The parameter controlling the phase transition is the number of mutations  $m$  per offspring, not the mutation rate  $m/L$  per locus and per offspring.

One time step is complete after the two processes of deaths and births are performed, scanning the whole population twice. We start the simulation at time step  $t = 0$ , with all bit-strings filled with zeroes (except for the hysteresis case shown later). The initial genetic distribution corresponds to  $H(N = 0) = P$  and  $H(N \neq 0) = 0$ . As time goes by, 1-bits spread more or less over the population, and  $H(N)/P$  eventually stabilises in some steady state distribution as shown in Figures 3–6. The relaxation time required for stabilisation increases for increasing  $L$ , and also depends on the number  $m$  of mutations performed at birth. Near the transition points like  $m = 1.0439$  in Figure 2 the relaxation time is very large. The majority of our results were taken with  $L = 1024$ , for which we observed that  $10^7$  (ten million) time steps are enough, and decided to adopt this number as default. In some cases, specially for larger chromosome lengths, we have done the simulations beyond this. Due to this extremely slow convergence rate, each point of a plot like Figure 2 corresponds to approximately two entire processing days on our fastest computer processor (AMD Opteron 250). Recessiveness requires even more computer power.

Figure 7 is an example of the slow convergence rate. Since the initial population has no 1-bits at all, the starting average genetic load  $\langle N \rangle$  is zero. The curves show the evolution of  $\langle N \rangle/L$  for 4 different values of  $m$ . The



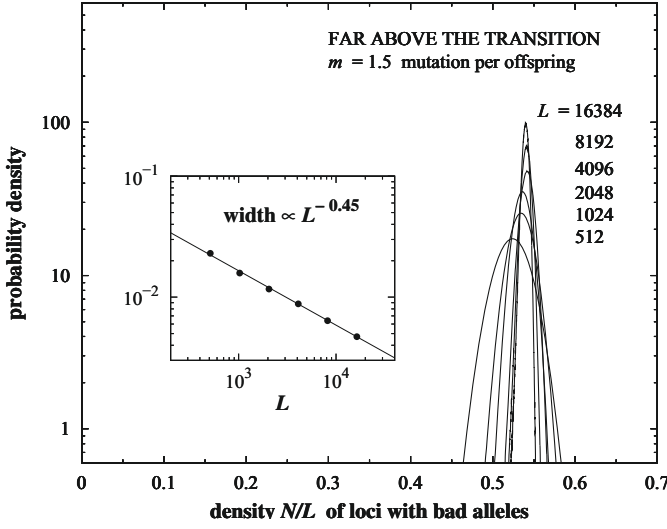
**Fig. 4.** Distribution of “bad” alleles for different chromosome lengths, as in Figure 3. Now, the number  $m$  of mutations per offspring performed at birth is fixed just below the transition displayed in Figure 2,  $m = 1$ . For large enough  $L$  the curves also collapse onto each other, full symbols.



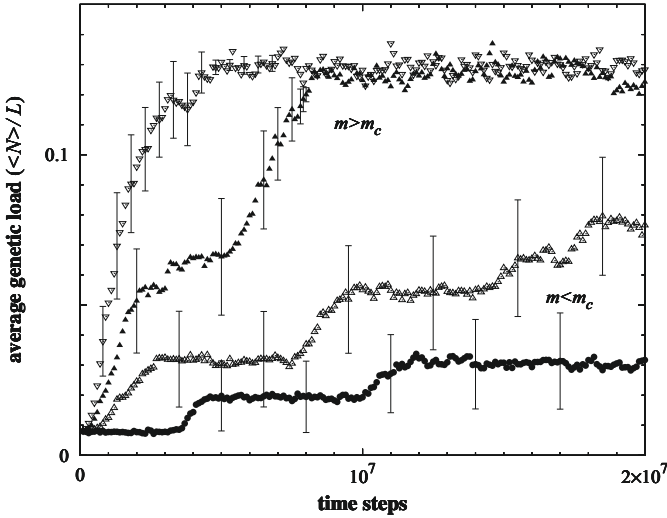
**Fig. 5.** The same as the previous two figures, now with  $m = 1.0440$ , just above the transition displayed in Figure 2. For large enough chromosome lengths, the distribution runs away from the wild genotype represented here by  $N = 0$ . The rightmost continuous line obtained for  $L = 1024$  shows this behaviour, the whole distribution being confined between the two vertical walls.

two lower curves correspond to  $m$  below but very near the transition point  $m_c$ , therefore still at the evolutionary phase. In this case, the fluctuations are large, denoted by the error bars included only at some points for clarity. The two upper curves correspond to  $m$  above but also very near the transition point  $m_c$ , therefore already at the non-evolutionary phase. Now, one gets a not-so-slow convergence, after which the fluctuations become smaller with





**Fig. 6.** Distributions for large enough chromosome lengths, above the transition. Now, they are plotted against the density  $N/L$ , as in the asexual case of Figure 1. However, contrary to the asexual case, the number  $m$  of mutations performed at birth is kept fixed for different  $L$ , instead of the mutation rate  $m/L$  kept fixed in Figure 1. The inset shows the corresponding  $L$ -dependence of the widths.



**Fig. 7.** Time evolution for 4 different values of  $m$ , all of them very near  $m_c$ . Just below the transition (two lower curves) the convergence is very slow. In this example,  $c = 1.5$  and  $L = 1024$ . By increasing the chromosome length, the convergence of the two lower curves becomes still slower. However, for large  $L$ , the average genetic load  $\langle N \rangle / L$  goes to zero. Contrary to that, inside the disordered phase one observes the two upper curves already stuck to their final *finite* plateaux, independently of the  $L$  value.

error bars of the same size of the symbols. Fluctuations also become very small if one takes  $m$  below but not near the transition (not shown).

We have identified the non-evolutionary phase on the right side of Figure 2 as the *extinction* phase, although the population sample is kept with constant size. In this

case, the genetic distribution among the population corresponds to the sharp peaks displayed in Figure 6, centred on  $\langle N \rangle \propto L$ , with a narrow relative width vanishing for large enough values of  $L$ . Therefore, the last equation (1) could be replaced by

$$x^{\langle N \rangle + 1} = 1 - b$$

for which the solution approaches  $x = 1$  if we consider large  $L$  and consequently large  $\langle N \rangle$ . However, we have already seen that  $x$  should be *strictly* smaller than 1, limited by some upper bound, say  $x_{\max} = 0.999$ , otherwise nobody dies. This upper bound is surpassed when solving equation (1) just when the run-away shown in Figures 5 or 6 occurs. Replacing its solution  $x$  by a lower value  $x_{\max}$  leads to extinction. In this way, as soon as the number  $m$  of mutations performed at birth surpasses the transition point  $m_c$ , not only Darwin evolution stops because all individuals become selectively alike, but also extinction is the next step. Therefore, within the model, we don't need to observe a real extinction of the population sample in order to identify the extinction already in course for the whole population, the genetic run-away or “error catastrophe” suffices, and we can forget the limit  $x_{\max}$ . That is why we adopt the interpretation of a fixed-population sample representing a much larger fluctuating population, by tuning the value of  $x$ . This interpretation is valid as far as  $x$  does not approach unity, i.e. before the run-away characterising the non-evolutionary phase above  $m_c$ . This approach of tuning  $x$  in order to keep constant the population sample goes back to [7]. For smaller values of  $L$  ( $\leq 64$ ), simulations [8] with fixed  $x$  and varying  $P$  give different behaviour.

Figure 2 shows a computer-simulated result of this model. It considers a sexually reproducing population, with individual genomes subjected to random mutations as well as crossing-over during reproduction, under a selective environment. Homologous chromosomes are represented by double, diploid bit-strings consisting of 0-bits (the wild type) and 1-bits (the “bad” allele). The quantity we consider for selection is the total number  $N$  of loci containing at least one “bad” allele; the larger  $N$  is, the smaller is the individual's fitness. Data in Figure 2 correspond to the average over 10 independent stable populations (after many enough generations). All other data presented in this paper also correspond to the average over 10 independent populations. Error bars were determined from fluctuations between these 10 samples.

Figure 2 shows plots for different chromosome lengths, and one verifies at the left side the corresponding curves approaching the horizontal axis for larger and larger  $L$ . The average genetic load  $\langle N \rangle / L$ , where the symbol  $\langle \dots \rangle$  means population average, vanishes for large enough chromosome lengths along *all* this phase,  $m < m_c$ .

Instead, on the right side of Figure 2, the curves go *up*. By increasing the chromosome length, they converge to the universal curve displayed by full-circles ( $L = 1024$ ). The average genetic load is no longer null, because  $\langle N \rangle$  becomes proportional to  $L$ : life through Darwinian selection becomes impossible, as explained below.

Figure 3 shows the distributions of homologous loci containing “bad” alleles. For larger and larger chromosome lengths, all curves collapse into a single one. The data are collected below the transition, deeply inside the ordered phase on the left side of Figure 2,  $m = 0.5$ . The typical number  $\langle N \rangle$  of loci containing the “bad” allele ( $\langle N \rangle < 5$  in Fig. 3) remains the same in spite of the increasing chromosome lengths. That is why the curves go down on the left side of Figure 2 as

$$\frac{\langle N \rangle}{L} \propto L^{-1}$$

where the symbol  $\propto$  represents proportionality. The exponent  $-1$  governs the finite-size-scaling of the genetic load  $\langle N \rangle/L$ , and is our first important result.

Figure 4 shows again the  $N$ -distribution for the same transition displayed in Figure 2. Now, the average number of mutations performed at birth is  $m = 1$  for all chromosome lengths, very near but still below the transition point  $m_c = 1.0439$  of Figure 2. The typical number  $\langle N \rangle \approx 5$  of loci containing the “bad” allele is larger now, when compared with Figure 3. However, it also remains the same for increasing chromosome lengths. Note also that the “optimum” configuration  $N = 0$  is still present, although with a small frequency.

At each new time step, a fraction  $b$  of new individuals are included into the population. We adopted  $b = 2\%$ . Their genomes are taken from random parents, with mutations. Since the number of 0-bits among the population is much larger than that of 1-bits, these mutations occur more likely in the sense  $0 \rightarrow 1$  (“bad” mutations) than in the reverse one, as in Nature. This asymmetry tends to shift the curves like Figure 4 to the right, increasing its rightmost parts. Selection, which eliminates the same fraction  $b$  of individuals from the population, within the same time step, tends to shift the curves back to the left, in a compensatory movement. The figures show the steady-state situation, where the distribution remains the same after both movements were performed, i.e. after each computer time step with deaths and births. These two opposed movements, however, come from different ingredients of the Darwinian paradigm: the first (shifting the curve to the right) from random mutations which we control through the parameter  $m$ ; the second (back to the left) from the selection pressure which is always the same, since we keep the death rate  $b$  constant. Therefore, by further increasing  $m$ , this balance which keeps the wild genotype alive will become impossible.

Figure 5 corresponds to  $m = 1.0440$ , just beyond the transition. Indeed, the curves falsely seem to obey the same kind of convergence displayed in Figure 3 or 4, up to  $L = 512$ . Suddenly, however, for  $L = 1024$  the distribution escapes towards a finite-density  $\langle N \rangle/L$  of loci containing the “bad” allele, shown by the rightmost curve where  $\langle N \rangle \approx 140$  (note the cut on the horizontal axis). The distribution curve runs away from the wild genomic form  $N = 0$ , which becomes extinct. This is the same phenomenon which occurs in Eigen-type models [1], sometimes called the “error catastrophe”. Now, the typical

number  $\langle N \rangle$  of loci containing “bad” alleles grows proportionally to  $L$ : would we plot the distribution for  $L = 2048$ , the corresponding bell-shaped curve would be positioned around  $\langle N \rangle \approx 280$ , far to the right, not visible in Figure 5; for  $L = 4096$  it would fall around  $\langle N \rangle \approx 560$ , far yet to the right, and so on. We cannot show all these curves on the same plot: even for  $L = 1024$  we were forced to perform the artificial cut on the horizontal axis.

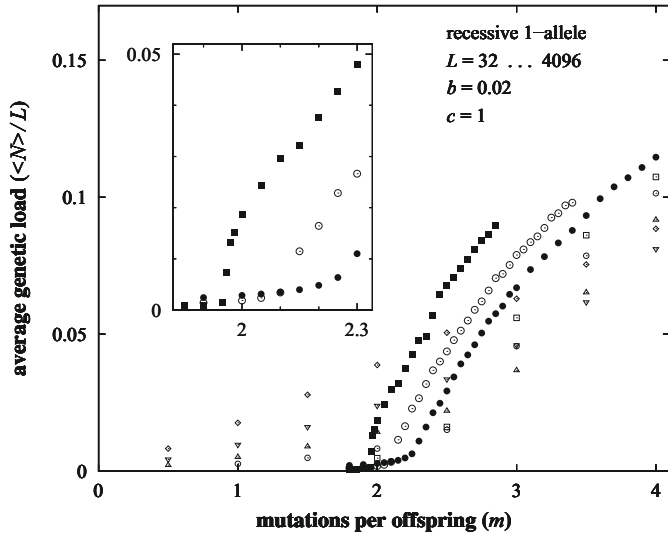
In order to see the distribution curves for different chromosome lengths above the transition, we therefore replace (on the horizontal axis) the number  $N$  of loci containing the “bad” allele by its *density*  $N/L$  along the genome. Figure 6 shows the result for  $m = 1.5$ .

The widths  $\Delta N/L$  of these distributions shrink for larger and larger chromosome lengths. Therefore, for large enough values of  $L$ , all individuals share the same genetic load  $\langle N \rangle/L$ , within negligible fluctuations. Individuals no longer show different selectivities when compared to each other, all individuals become alike in what concerns the selection pressure. Darwinian evolution cannot proceed for  $m > m_c$ . This side of the transition is the non-evolutionary phase, and corresponds to population extinction already discussed in the previous section.

Figure 7 shows the slow evolution of the average genetic load  $\langle N \rangle/L$  near the transition.

In short, the transition point  $m_c$  separates two phases, for large  $L$ . Region  $m < m_c$  represents the evolutionary phase where the typical number  $\langle N \rangle$  of “bad” alleles remains the same for increasing chromosome lengths: the average genetic load  $\langle N \rangle/L$  vanishes. The other phase,  $m > m_c$ , is non-evolutionary and behaves differently:  $\langle N \rangle$  increases proportionally to  $L$ , the genetic load no longer vanishes, and the genetic pool no longer includes the wild genotype  $N = 0$ . Note again that the transition occurring at  $m_c$  is controlled by the number of mutations  $m$  (per genome), not by the mutation rate  $m/L$  (per genome unit), and consequently the transition point  $m_c$  remains the same, independently of how large is  $L$ .

A dynamic phase transition corresponds to the competition of different possible attractors to which the steady-state population converges after many generations. In our case, one attractor is characterised by the presence of the wild genotype  $N = 0$ , which is preserved only in the ordered phase  $m < m_c$ . We may call this evolutionary phase “ordered” by analogy with Physics where order-disorder transitions of this kind are ubiquitous, and also because the whole steady-state population remains “orderly” similar to the wild genotype, everybody with a vanishingly small fraction of “bad” alleles. On the other phase  $m > m_c$ , the population genetic pool melts into a disordered situation characterised by the absence of the wild genotype  $N = 0$ , everybody presenting a non-vanishing fraction of “bad” alleles. The selection mechanism is no longer able to contain the entropy explosion driven by too many random mutations at birth. Therefore, here we will call  $\langle N \rangle/L$  the *dis-order* parameter which characterises the transition, being non-null only at the disordered phase.



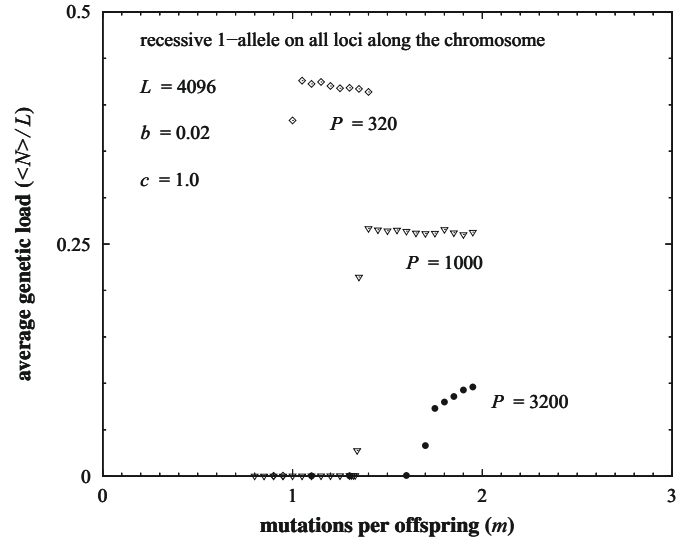
**Fig. 8.** Similar to Figure 2, for the recessive case, with  $L$  increasing from bottom to top on the right. Now,  $N$  is the number of homozygous loci with both bad alleles. The inset blows-up the transition region for  $L = 1024$  (full circles), 2048 (open circles) and 4096 (squares). In spite of the large chromosome lengths, the collapse of all curves onto a single one is not yet obtained. It should appear beyond  $L = 4096$ , defining the transition point (note the negative curvature which already appears for this length, when the full squares jump from zero to higher values, near  $m = 2$ ).

## 4 Further results

### 4.1 Recessiveness

Alternatively to the 1-bit dominance, the phenotype  $N$  of each individual can be counted as the number of loci where *both* homologous alleles are 1-bits. This is the recessive version, much more interesting from the biological point of view. It allows a much larger degree of genetic diversity among the population, since heterozygous loci do not represent any handicap for the individual survival. Figure 8 is the would-be equivalent of Figure 2 in this case.

Larger chromosome lengths are necessary in order to observe the collapse of all curves onto a single one, which starts beyond  $L = 4096$ . These plots correspond to  $2 \times 10^7$  time steps and  $P = 10\,000$ , which were enough to obtain convergence in the case of dominant 1-bit allele, Figures 2 to 7. Now, it is clear that these time and population size may be no longer enough. For instance, from these plots, Figure 8, one could wrongly infer a transition point near  $m_c \approx 2$ . As a test, we have run much longer times for smaller populations  $P = 320$ , 1000 and 3200, and verified the appearance of the sudden run-away already for smaller values  $1 < m_c < 2$ , Figure 9. Some kind of staircase seems to appear within this interval, which is an indication that some of the 10 independent populations considered in the averaging process have already undergone the run-away while others did not at the same time step. This behaviour also indicates the presence of hysteresis, shown in detail in



**Fig. 9.** Test runs with much larger times than Figure 8 (with smaller populations), showing the run-away (extinction phase) already appearing below  $m = 2$ .

next subsection. In this case, Figure 9, each point of the plot ( $P = 3200$ ) consumes more than a month of computer time, and even so we cannot be sure that all 10 independent populations were already genetically stabilized.

When, in contrast with the 1-bit allele dominant case (Fig. 2), recessiveness is turned on (Fig. 8) the current state of our simulations cannot precisely define the point where the phase transition occurs. However, this does not mean our simulations are useless for the recessive case. The phase transition certainly occurs in some point  $1 < m_c \approx 2$ , and we can use the populations leading to Figure 8, for instance, in order to compare the features and differences between the survival and extinction phases. This is done in the two next subsections.

### 4.2 Crossing-over

Figures 2 and 8 correspond to just one crossing-over performed during the gamete formation, i.e.  $c = 1$ . We have also tested other values.

Figure 10 corresponds to  $c = 0$ , i.e. no crossing at all, for the case where the 1-bit allele is dominant, to be compared with Figure 2.

Now, the transition is clearly a first order one, with a big gap on the dis-order parameter at  $m_c$ . However, also Figure 2 seems to display a first order transition, but weaker as displayed in its inset there.

Also without crossing, more interesting is the case where 1-bits are recessive, Figure 11. For the lower branch, filled circles, we start the whole process with  $m = 1$  and all initial chromosomes filled with 0-bits. The first point on the left side corresponds to the resulting populations after  $10^7$  time steps. Then, *starting from these populations*, we tune  $m = 1.05$  and run other further  $10^7$  time steps, getting the second point on the left side, and so on, increasing  $m$  in steps of 0.05, following the lower branch.

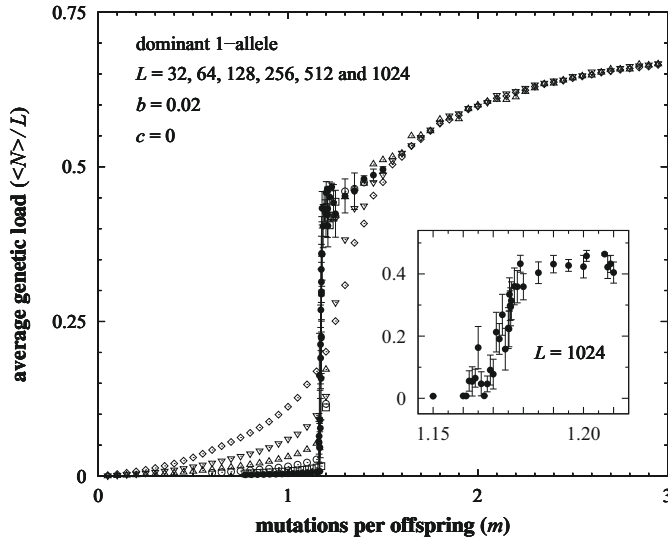


Fig. 10. First order phase transition without crossing ( $c = 0$ ), for dominant 1-bit alleles, to be compared with Figure 2.

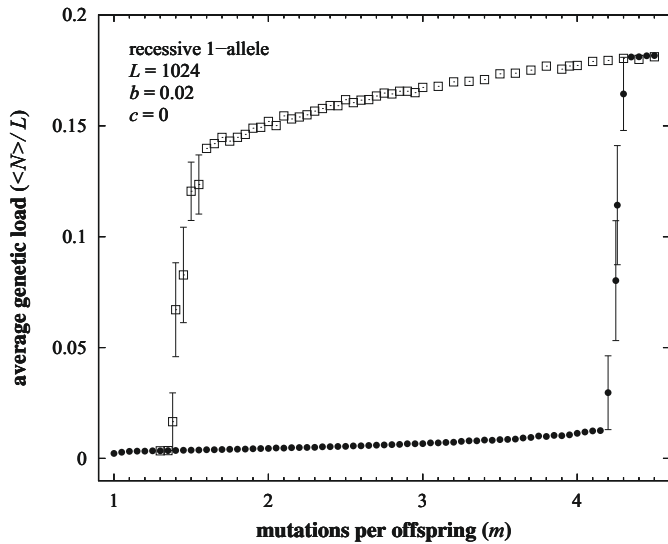


Fig. 11. Hysteresis without crossing ( $c = 0$ ), for recessive 1-bit alleles.

Only when we reach  $m = 4.2$  the gap on the right side appears, and the fluctuations among the 10 independent populations become large, as denoted by the error bars. Soon the fluctuations shrink again at  $m = 4.35$ , and we reach the non-evolutionary phase displayed by the last four filled circles up to  $m = 4.5$ .

Now, we do the reverse path, upper branch, starting from  $m = 4.5$  again, with all initial chromosomes filled with 0-bits. After  $10^7$  time steps we have already reached the non-evolutionary phase, rightmost open square (at the same position of the rightmost filled circle obtained before, which thus does not depend on the starting populations). Then, *starting from these already stabilized populations*, we tune  $m = 4.4$  and run other further  $10^7$  time steps, getting the second rightmost open square (also coincident with the filled circle branch), and so on, decreasing  $m$

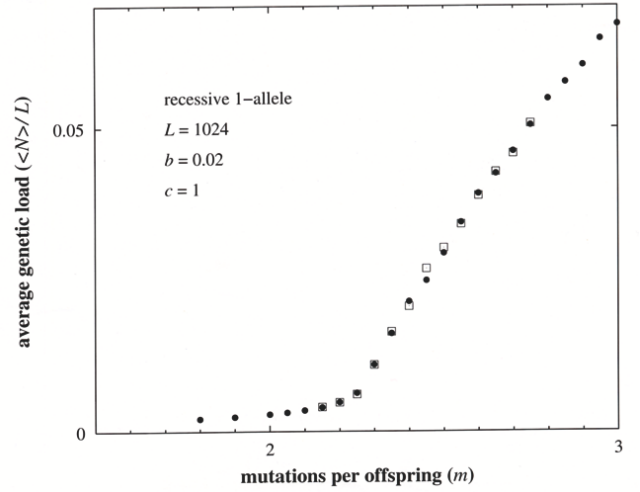


Fig. 12. Crossing destroys the hysteresis.

and running more  $10^7$  time steps for each new value. The result is the upper branch displayed by the open squares. Only when we reach  $m = 1.55$  this branch goes down following the gap at the left side, where the fluctuations (error bars) become visible again. At the end of this gap downwards, the upper branch meets again the lower one. In between  $m_1 \approx 1.4$  and  $m_2 \approx 4.2$  the system displays a clear bi-stability, the equilibrium population depending on the initial one. For any fixed value of  $m$  within this interval, the population goes to the evolutionary phase *if* the genetic load of the initial population is small enough. Otherwise, it goes to the non-evolutionary phase, for the same fixed  $m$ .

The presence of crossing-over destroys this behaviour, as shown in Figure 12 for  $c = 1$ . Now, both branches are indistinguishable, there is no hysteresis.

Finally, a larger number of crossings seems to have no effect, Figure 13; that means we swapped between the two bit-strings not only the parts on the two sides of one single crossing point, but allowed for several such crossing points with corresponding swaps of parts of the bit-strings.

### 4.3 Heterozygosity

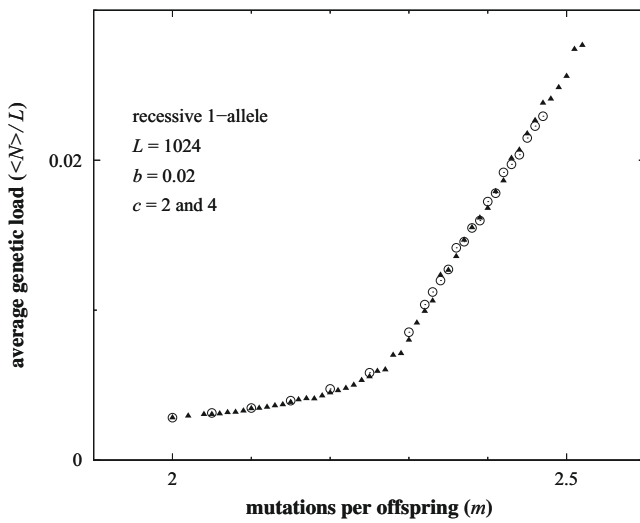
With crossings and recessive 1-bit allele, Figure 14 shows the heterozygosity, i.e. the fraction of heterozygous loci averaged over all individuals of all 10 independent populations after  $10^7$  time steps, as well as the corresponding fractions of both homozygous loci 11 and 00 (homologous loci filled with the same allele 1 or 0).

Figure 15 shows again the recessive case, now without crossing and near the upwards jump on the right side of Figure 11. At the extinction phase, the heterozygosity is simply random.

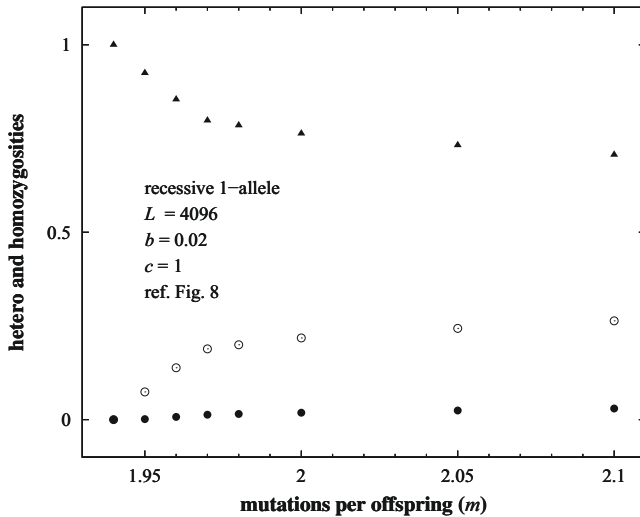
Figure 16 also refers to the case of Figure 11, now near the downwards jump on the left side. Again, the heterozygosity is random at the extinction phase.

Comparing Figure 14 where crossing is present to Figure 15 or 16 where  $c = 0$ , one concludes that crossing





**Fig. 13.** As previous figure but now obtained for more than one crossing,  $c = 2$  (triangles) and  $c = 4$  (open circles).



**Fig. 14.** Heterozygosity (open circles), homozygosity 11 (filled circles), and homozygosity 00 (triangles), with one crossing as in Figure 8.

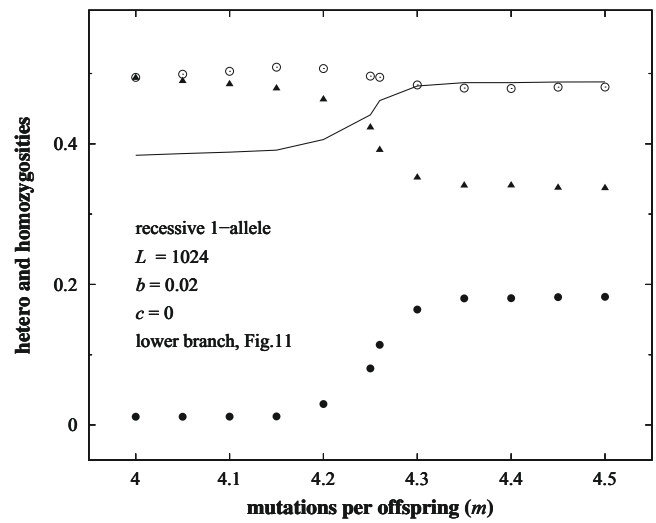
has a fundamental role: homozygosity 00 dominates the population within the evolutionary phase, at the very left side of Figure 14. On the other hand, without crossing, heterozygous individuals occupy a large fraction of the surviving population at the evolutionary phase, left side of Figure 15 or 16. However, the presence of heterozygosity on the evolutionary phase does not mean a larger genetic diversity. On the contrary, without crossings, diploid individuals tend to get two complementary homologous chromosomes, an example of which is

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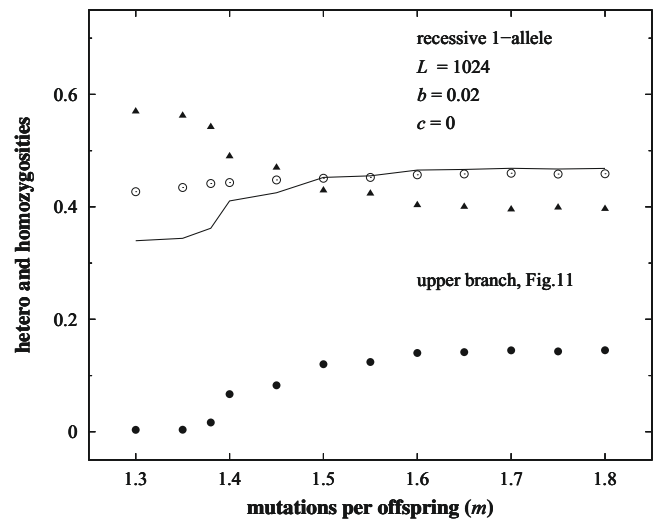
A    0 1 1 1 0 1 0 1 0 0 0 1 0 0 1 0 1 0 0 1
B    1 0 0 0 1 0 1 0 1 1 1 0 1 1 0 1 0 1 1 0

```

where homozygous 00 and 11 loci are not shown for clarity. (Both do not matter for our following argument: 11



**Fig. 15.** Heterozygosity (open circles), homozygosity 11 (filled circles), and homozygosity 00 (triangles), without crossings. If the bad alleles were randomly distributed the heterozygosity would be given by the full line.



**Fig. 16.** Heterozygosity (open circles), homozygosity 11 (filled circles), and homozygosity 00 (triangles), without crossings.

because it is anyway virtually absent from the evolutionary phase, according to Figure 15 or 16; and 00 because it does not mean any handicap.) The same kind of complementarity was also found in [9,10].

In spite of its many “bad” genes, the above-exemplified individual has no handicap at all! Forget mutations for a while, and consider that all individuals become like this AB example. (This is not impossible, inbreeding helps.) Without crossings, their offspring are two-fold: those exactly like the parents (which survive), or those suffering from a strong handicap (which die). Surviving newborns are clonings from their parents, all of them genetically identical to each other. Evolution stops.

Crossing-over, on the other hand, avoids the population to reach this genetic trap. Homozygosity 00 can be

restored from heterozygous individuals. The consequence, as shown in Figure 14, is that all individuals remain genetically near (or at) the “optimum” state. Nevertheless, the population as a whole keeps the necessary genetic diversity to evolve: individuals with (nearly) complementary homologous chromosomes are exceptions, not the rule.

## 5 Conclusions

We have considered the evolution of sexual reproducing populations under a strict Darwinian-Mendelian prescription. Our model is one of the sexual generalisations [11,12] of the asexual Eigen quasispecies model. Gene transfer within haploids was also recently studied [13]. As in the original model [1] we work with a single-peak fitness and consider only a constant-size sample picked from the total population [14,15]. Each individual carries a pair of diploid chromosomes with length  $L$ . Random mutations at birth are performed as well as crossings-over. The selection pressure removes more likely from the population individuals with higher numbers of harmful mutations. We have investigated the  $L$ -scaling properties and discovered a phase transition occurring at a sharply defined number  $m_c \approx 1$  of mutations performed at birth, the same value independent of the (large enough) chromosome length  $L$ . If the average number  $m$  of mutations per offspring remains below  $m_c$ , then the whole population survives. Above  $m_c$  the population undergoes a genetic meltdown, the number of harmful mutations explodes for all individuals (Eigen catastrophe [1]), and finally the whole population is extinct. This behaviour comes from the dynamics of Darwin’s evolution itself, under Mendel’s genetic heritage rules, nothing more. Thus we believe it is completely general.

The interesting point is that the average number of mutations  $m$  performed at birth is the important parameter controlling the phase transition, not the mutation rate per bit  $m/L$ . In reality, the DNA-copying chemical machinery is the same for all living beings, and works as a zipper scanning the whole chromosome length  $L$ . Therefore, apart from further error-correction mechanisms, the number of “errors” (mutations) should be proportional to  $L$ . This behaviour imposes a limit on  $L$ , in order to keep the number of mutations below the extinction transition point. Thus, according to our model it is not possible to evolve by simply increasing the chromosome length in order to store more and more genetic information, which will require an improvement on replication fidelity. Perhaps this feature explains why big animals have their genetic information stored in more-than-one chromosome pair (23

for humans). Moreover, considering only the coding parts of our genetic information, the real number of mutations per genome [16–19] is indeed below or near  $m_c \approx 1$ , in agreement with our results.

Also interesting is the absence of such a transition for haploid, asexual reproducing populations [2]. In this case, the same genetic meltdown also occurs, but it can be circumvented by artificially increasing the population proportionally to  $L^\alpha$ , with  $\alpha \approx 2.3$  [2]. For the present case of sexual reproducing populations, the transition remains no matter how large are the populations we have tested.

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