Strategies for the evolution of sex

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We find that the hypothesis made by Jan, Stauffer, and Moseley [Theory Biosci. **119**, 166 (2000)] for the evolution of sex, namely, a strategy devised to escape extinction due to too many deleterious mutations, is sufficient but not necessary for the successful evolution of a steady state population of sexual individuals within a finite population. Simply allowing for a finite probability for conversion to sex in each generation also gives rise to a stable sexual population, in the presence of an upper limit on the number of deleterious mutations per individual. For large values of this probability, we find a phase transition to an intermittent, multistable regime. On the other hand, in the limit of extremely slow drive, another transition takes place to a different steady state distribution, with fewer deleterious mutations within the population.

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I. INTRODUCTION

Sexual reproduction is believed to have arisen already in unicellular organisms [1,2] and simple organisms who engage in sex, either habitually or facultatively, especially when the external conditions are unfavorable (e.g., certain yeasts, fungi or the green algae Chlamydomonas) [3–5] are extant today. Since for such simple organisms, each act of fusion typically reduces the population by one (one offspring is produced at the expense of two parents) how such a mode of reproduction could have established a foothold in the evolutionary game remains a puzzle [1,2].

It is well known that the simplest unicellular organisms (which include bacteria) multiply asexually by cloning (mitosis) resulting, modulo possible errors in replication, in two copies of the original cell. These organisms possess only one set of genes, and are known as haploids. By contrast, diploids, which comprise all the higher organisms, as well as many unicellular life forms, possess, at least in certain phases of their life cycle, two (not necessarily identical) sets of their complete genome. Although there is no firm evidence to support this fact, it is speculated that sexual reproduction may have arisen simultaneously with the emergence of diploidy, or at least soon thereafter [2].

Many authors have argued [1,2,6,7] that diploidy presents obvious advantages over haploidy, since such simple organisms face an even higher rate of random mutations than more complex beings. Having an unimpaired copy of the damaged alleles, and being able to make use of this copy in repairing the damage, presents a clearcut advantage. It is generally assumed, moreover, that deleterious mutations are recessive, so that in case an undamaged allele is present at some locus, this will be expressed in the phenotype rather than the damaged one, although it is not clear at what stage this mechanism of dominance has come into play as an inherited feature of genetic control.

The supposed advantages of fusion in giving rise to greater genetic variety, combined with these advantages of diploidy, have been studied via numerical models, by many groups [8–12,16]. Notably Redfield [8] has shown, within a constant population model, that unless there is a much greater mutation rate for males, sex increases the mean fitness in a population, if a step-function-like survival function in the number of deleterious mutations is assumed. The naturally occuring relative mutation rates for males, however, are apparently such that parthenogenesis would seem more favorable than sex. Modifying Redfield's model, Stauffer and co-workers [9,10], Bernardes [11], and Cui *et al.* [12] have been able to show within ''age structured'' populations [13], that sexual reproduction can indeed lead to better results. Others [14–16] have discussed and modeled the proposition that a parasitic infestation could afford sex with an advantage over both asexual reproduction and meiotic parthenogenesis in the greater genetic diversity it provides. Pekalski [17] has shown the superiority of sexual reproduction in adaptation to a periodically changing environment.

In this paper we would further investigate a very simple model for archaic sex, which does not have any of the more complex features discussed by the models cited above, such as sexual differentiation between males and females, or age structuring. We present computer simulations for several different strategies, and compare their outcomes.

In a previous paper [18] we have shown that a finite steady state sexual population may arise from a purely asexual one, if an excess of deleterious mutations causes haploid individuals to perform syngamy and become diploid, and diploid organisms to engage in sexual reproduction as a means of escaping death [19]. We assumed, as is generally done [8,10], that mutations that lead to departures from the ''wild type'' (the ideal type) are deleterious, and that deleterious mutations are recessive. Under various different assumptions regarding the subsequent mode of reproduction (e.g., whether sexual reproduction is hereditary or not) and of the number of offsprings, we found that the diploid population always persisted, and that it was consistently more successful in escaping the effects of deleterious mutations.

In the case where sexual reproduction was only practiced as a means of escaping death from too many deleterious mutations, but diploid cells were also allowed to multiply by mitosis, diploid individuals completely took over the popu-

lation. Thus, in one of our models (Model I) [18], we were able to demonstrate a possible scenario for the evolution of the analogue of a ''haploid-diploid cycle'' [1] where the organisms reproduce asexually as long as they are reasonably fit (or the conditions reasonably favorable) but engage in sexual reproduction when the going gets tough [3,4].

In the present paper we will test whether a threshold mechanism for switching to sexual reproduction is *necessary* for the successful establishment of a sexual population. We simulate two strategies for the evolution of sex within a fixed population N of simple organisms, who are all initially asexual (and haploid), and subject to a constant rate Γ of random mutations. Both haploid and diploid organisms die when the number of their expressed deleterious mutations exceeds a certain number. We have also adopted a more realistic set of rules than in Ref. [18] for the mechanism of dominance, that is, the expression of mutated alleles, than in our previous paper [18]. Here we allow a mutated gene to be expressed if the cell is homozygous for mutated alleles at this locus. Hence, the number of expressed deleterious mutations for diploid individuals is the number m of different loci at which the cell is homozygous for mutated alleles.

The first strategy (Model A) is the adoption, as in Ref. [18], of syngamy and consequent diploidy when the number of mutations exceeds a threshold, threatening extinction. The second strategy (Model B) involves a small but constant probability σ for the accidental conversion to diploidy, independently of the number of mutations (or, equivalently, the fitness) of the individual. In Model A, without habitual sex, the diploids then engage in sexual reproduction when they are similarly threatened by an excess of deleterious mutations. In Model B without habitual sex, they do so with a probability σ. The cloning of sexual individuals is not allowed in either Model A or B. We have also tested for the effect of habitual vs nonhabitual sex.

We find that *both* strategies A and B lead to a finite steady state sexual population, with typically a smaller average number of mutations (greater fitness) than the asexual population. Thus no threshold mechanism seems to be necessary for a successful sexual population to take hold. However, for habitual practice of sexual reproduction by diploid individuals (i.e., those that are not facing extinction in Model A) calls for unrealistically large mutation rates in order for a macroscopic sexual population to be established in the steady state.

To be able to distinguish the contribution of diploidy (just possessing two alleles of the same gene) from that of sexual reproduction (leading to greater variability via fusion), we have also performed simulations on a purely diploid population, with an initial population again consisting only of the wild type. We found that the steady state distributions of the sexual types had consistently more favorable m distributions.

The organization of the paper is as follows. In the next section we explain in detail the two models and we report the result of our simulations. In Sec. III, we display and examine the mean field evolution equations and discuss our findings in the light of these equations. In Sec. IV, we investigate the limits of strong and extremely weak driving of this system, for $\Gamma \to 1$ and $\Gamma \to 0$, as well as a transition to chaos via an intermittent route, found for large values of σ. A discussion of the results from similar models and directions for further research are provided in Sec. V.

II. MODELS FOR CONVERSION TO SEX

We represent the genetic code of each one-celled individual with a bit string of ''0's'' and ''1's.'' At each locus, we have taken the value ''0'' to correspond to the wild type and ''1'' to a deleterious mutation (which we will call ''mutation,'' for short, where this is not liable to lead to any confusion.) We use the bit defining the ''sign,'' to specify whether the individual is asexual $(+)$ or sexual $(-)$. For asexual, haploid, cells, we have one 15-bit string, whereas, for the sexual cells, we have two 15-bit strings that are allowed to be different, i.e., the individuals are now diploids. Since the genetic difference between individuals of the same species is typically less than 10% even for simple organisms [20], this rather short string for the genetic code may be considered as a coarse grained model for the complete genome of the individual, which we divide up into different zones, retaining a ''0'' where there are no mutations, and flipping the bit to ''1'' if there are one or more mutations in this zone.

A mutation consists of flipping a randomly chosen bit except the sign bit, and it is implemented by scanning all the individuals in the population, and, with probability Γ picking those to be mutated. Clearly there may be any number of mutated individuals at any one generation (time step), the number fluctuating around ΓN, where N denotes the total population.

The number of deleterious mutations m is simply the number of ''1's'' for a haploid individual. For a diploid, the number of ''expressed'' deleterious mutations is taken to be the number of loci at which both homologous alleles are set to ''1.'' This is how we model the mechanism of dominance of the wild type (or, equivalently, the recessiveness of deleterious mutations.) We will use the term ''fitness,'' loosely, for $L-m$; thus increasing m will decrease the fitness of the individual. The usual assumption made by biologists is that each deleterious mutation decreases the fitness by a fixed factor, so that the fitness would decay exponentially with $L-m$. Other functions have also been used [7], which assume a positive or negative correlation between the effect of successive deleterious mutations. The fitness, or survival, function we have chosen is of the ''truncation type'' [8], i.e., essentially a step function, to describe the threshold behavior in switching to diploidy and sex under an excess mutation load [18,19,21]. The probability of survival as a function of m is given by a Fermi-like distribution [21], $P(m)$,

$$P(m) = \frac{1}{\exp[\beta(m-\mu)]+1}. \tag{1}$$

For large β (or low ''temperatures'' T, in the language of statistical mechanics, with $\beta \propto 1/T$), $P(m)$ behaves similar to a step function [18]. Individuals with $m > \mu$ die, those with $m < \mu$ survive, and those with $m = \mu$ survive with a probability of 1/2. In the present paper we have confined ourselves to the ''zero temperature'' limit, taking $\beta = 10$, which is large

enough for practical purposes. The threshold μ was chosen as 4, which allows for just sufficient variability of the typical fitness of the steady state population without becoming totally unrealistic as to the percentage of mutated domains, for $L=15$. *A posteriori*, it can be seen [22] that this choice for the survival function favors sex most strongly, as was also found by Redfield [8], since it is tolerant to genetic diversity for small deviations from the wild type, while strictly punishing for $m>\mu$. For finite temperatures, this distribution interpolates between the step function and the exponential decay (i.e., the Boltzmann distribution). Reducing β would correspond to increasing the noise in the system, and allowing individuals with a large m to survive with a finite probability, while at the same time sacrificing some individuals with small m.

We keep the total population constant, as in the Redfield model [8], by making up for the deficit in the population after all the bacteria have been either found fit for survival or killed off according to the survival probability in Eq. (1). Asexual individuals multiply by simply making another copy of themselves, namely by mitosis, while a pair of sexual organisms each contribute 1-bit string to their offspring and die in the process.

We performed the simulations on a fixed population of $N=1000$, for 16-bit strings. The total number of time steps in each simulation is taken to be much larger than the time necessary for the transients to die off and the system to settle down to a steady state. Since the probability to mutate a single gene in a diploid individual is $\Gamma/(2L)$, on the average the steady state is reached after $2L/\Gamma$ time steps, or, in other words, when the number of mutated genes in the population is greater than the total number of genes of one individual. In all the simulations, the reported results are averages over 10 runs. The fluctuations depend on the model chosen, however, the relative error estimate based on one standard deviation is typically less than about 6%, as long as there is only one fixed point for the dynamics. In the steady state, the distribution of the asexual and sexual populations over m are independent of Γ, for $\Gamma>1/N$. The cases where $\Gamma<1/N$ and $\Gamma\approx 1$ have interesting consequences, and are discussed in Sec. IV.

A. Asexual steady state

We start with a set of N initially identical asexual individuals, all identical to the *wild type*, i.e., all 0's. Under the conditions outlined above, without introducing sex, and for $\Gamma\geqslant 1/N$, the population of asexuals settles down to the steady state given in [18], where the number $n_H(m)$ of asexual (haploid) individuals with m mutations is $n_H/N_A=0.012$, 0.098, 0.356, 0.531, 0.001 for $m=0,\ldots,4$, where N_A is the total number of asexuals (equal, at this stage to the total population, N). In this region this steady state distribution is independent of Γ, which only sets the scale of time. That this should be so, is not self-evident, and only follows from the form of the solution to the set of evolution equations, as shown in Sec. III.

B. Triggering sex

The alteration of the sex gene can be accomplished in two different ways. One can choose to trigger sex with a threshold mechanism or define a constant probability for each individual to become sexual. These mechanisms are further discussed in the following subsections. In either case, the haploid organism first makes a copy of its own set of genes, as if it were going to perform mitosis, but then forms two gametes instead. One of these gametes will pair up with a gamete from another individual who has been turned on to sex, and the other will be discarded. This conversion from haploidy to diploidy can be termed syngamy [7], or fusion. It should be noted that endomitosis (simple doubling of the genetic material without subsequent cell devision) as a means of making the transition to diploidy would not help the organism in escaping the effects of an excess of deleterious mutations, since the two copies of the genome would be identical.

One should note that sexual reproduction may be implemented in different ways, resulting in different numbers of offsprings produced [18]. In this paper, we will define sexual reproduction in such a way that when two sexual individuals mate they always give rise to one sexual offspring; thus, the population is reduced by one, each time an act of sexual reproduction takes place. Clearly, increasing the number of offsprings will increase the advantage that the sexual population enjoys. Indeed, judging from our previous results [18], the number of offsprings exceeding two would lead to the takeover of the population by the dipoid sexual types.

When two diploid cells engage in sexual reproduction, they each contribute one gamete towards a single diploid sexual offspring. Let us denote the two gametes as $\{Aa\}$ in one parent and $\{Bb\}$ in the other parent. Then the genome of the offspring may be $\{AB\}$, $\{Ab\}$, $\{aB\}$, or $\{ab\}$. We do not allow for crossover between the gametes during sexual reproduction. We are aware that recombination with crossover leads to enhanced variability in the genetic code. This presents sexual reproduction with yet a further advantage, and would only strengthen our results. However, we have refrained from introducing it at this stage to be able to see the contribution of diploidy more clearly. Moreover, it is not known [23], whether recombination (i.e., with crossover) is a feature that existed in very early forms of sexual reproduction. With a reproduction rate of once every 12 min, mutation in unicellular organisms is a much more effective means of adaptation.

1. Sex at the threshold of extinction: Model A

In Model *A*, alteration of the sex gene takes place only under special conditions, namely, the threat of death due to too many mutations [19]. Once the asexual steady state is reached, we allow the sex gene to be ''turned on'' for the least fit members of the population. In any pass through the population, if those individuals that are in the tail of the distribution (i.e., those with $m\geqslant\mu$ mutations) survive, then they are turned sexual by deterministically and irreversibly switching their sign bits to 1. Once their sex bit is turned on, these individuals will be labeled ''sexually active'' and mate

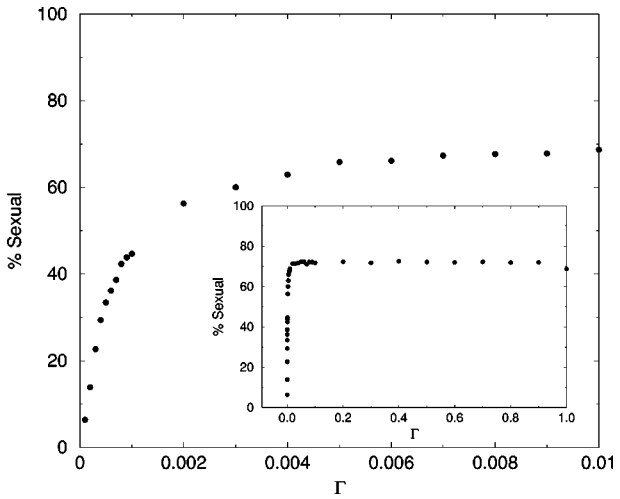

FIG. 1. The percentage of sexual population vs Γ is plotted for Model *A* where hereditary sex is not allowed, for a population of 1000 individuals. The inset shows a larger range of Γ where the step-function-like jump is more apparent. Both curves represent averages over 10 runs.

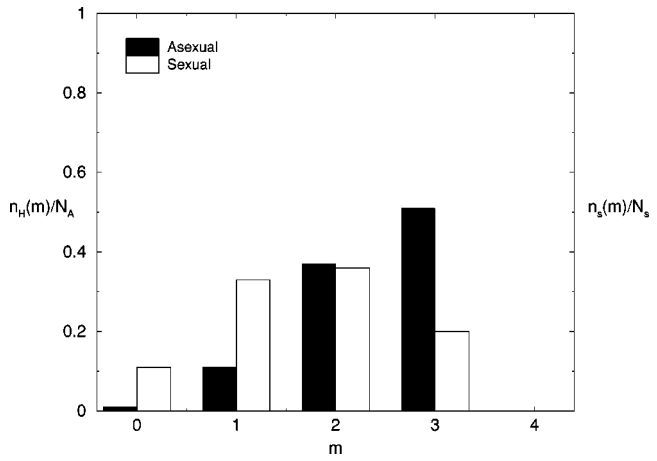

FIG. 2. The distribution of both sexuals and asexuals over the number of expressed deleterious mutations *m*, for Model *A*. $\Gamma = 10^{-3}$. Hereditary sexuality is not allowed and the distributions are normalized to unity over each population separately.

with other sexually active individuals. If there is only one active sexual at a certain time step then it must wait subsequent generations until it finds a partner. After mating, the sexual individual becomes sexually inactive and the only way for it to become sexually active again is to face extinction once more. The deficit in the population due to deaths and to sexual reproduction is then made up by copying randomly selected asexual individuals.

In this model, therefore, there is no hereditary sexuality. There is, however, a hereditary transition to diploidy. This gives an unfair advantage to the sexuals in that they both enjoy the benefits of diploidy and escape the disadvantage of $2 \rightarrow 1$ reproduction.

We see that for $\Gamma \sim 10/N$ the proportion of the sexuals in the population saturates to $\sim 70\%$ as shown in Fig. 1, and remains at this value independently of the value of Γ. In order to obtain points near $\Gamma \simeq 0$ one has to do very long runs to get accurate results, and these are discussed in Sec. IV, as well as the chaotic behavior displayed when Γ becomes too close to 1.

The steady state distributions of both asexuals and sexuals with respect to *m* are also independent of Γ (see Fig. 2) for $\Gamma \geqslant 1/N$ and sufficiently smaller than 1. The peak of the distribution shifts towards lower *m* values for sexuals [18].

Model A with hereditary sex. We have also tested the case of hereditary, or habitual, sex, in which sexually active individuals can mate randomly either with sexually active individuals who have been converted to sex in that generation, or with individuals who have already been converted in some preceding generation. As in the case of nonhereditary sex, the population is allowed to grow back to its fixed value by cloning randomly selected asexual units.

This small difference results in a noticeable increase in the number of matings at each time step, and therefore leads to a decrease in the number of sexual individuals in the steady state. We have found that the steady state comprises a macroscopic sexual population only for $\Gamma > 1/N$. For $\Gamma < 1/N$, the average number of sexual individuals drops to about 1%, or around 10 individuals in a population of $N = 1000$. The sexual population increases linearly with Γ and reaches only $\sim 15\%$ (as compared to 70% for nonhereditary sex) as $\Gamma \simeq 1$ (see Fig. 3). The *m* distributions are shown in Figs. 4(a) and 4(b) for the asexual and sexual populations. The peak of the sexual population has shifted to 1 as a result of the greater number of mating events. Thus we may conclude that hereditary and habitual sex in this model is punished more severely. The relative improvement in the mean value of *m* does not compensate sufficiently for the loss of the parents.

2. Mutating the sex gene with constant probability: Model B

Our second strategy for conversion to sex involves a constant probability σ for the accidental conversion to sex, in-

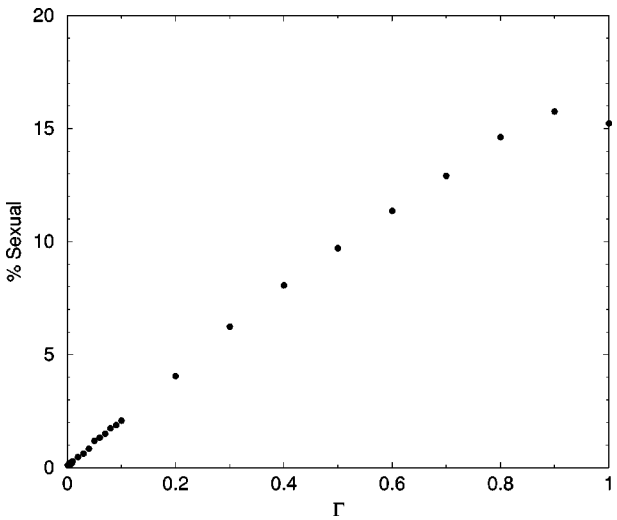

FIG. 3. The percentage of the sexual population vs Γ for Model *A* with hereditary sexuality introduced. The total population is 1000 individuals and the results are averaged over 10 runs.

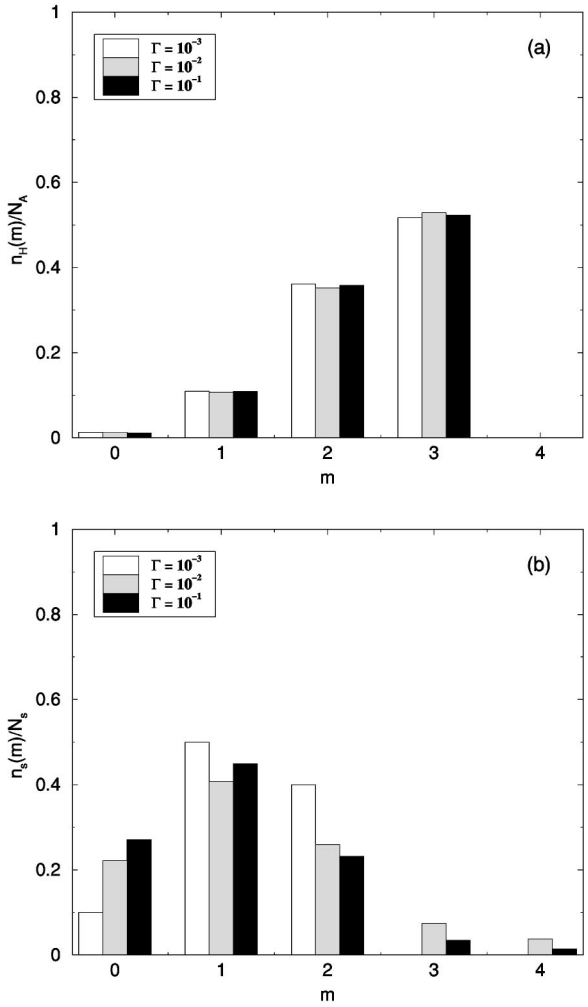

FIG. 4. The distribution of the (a) asexual and (b) sexual population with respect to m, for different values of Γ for Model *A* with hereditary sexuality. The histograms are normalized to unity.

dependently of the distance, as expressed by m, from the wild type. For this model (Model *B*), once the asexual steady state is reached, at each generation we allow the sex gene to be ''turned on'' irreversibly, with a small probability σ for each individual. Similar to Model *A*, these individuals will be ''sexually active'' and mate with other sexually active individuals of that generation. (If there is only one active sexual at a certain time step then it has to wait till it finds a partner at a subsequent generation.) If we take sexual reproduction to be nonhereditary, after mating the sexual individual becomes sexually inactive. (Within some subsequent generation it can once more become sexually active with probability σ.) The deficit in the population is made up by copying randomly selected asexual individuals.

We find (see Fig. 5) that this scenario again gives rise to a steady state macroscopic population of sexuals, but it is smaller than the one in Model *A*. The total percentage of sexuals is a function of σ/Γ, as can be seen from the figure, and grows with σ/Γ. In Figs. 6(a) and 6(b), we display the distribution of asexual and sexual individuals over the effective number of mutations m, for two small values of σ and Γ.

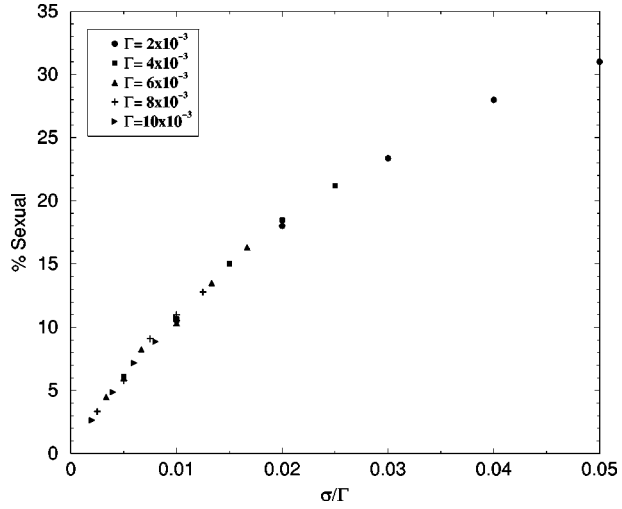

FIG. 5. Percentage of the sexual population vs σ/Γ plots for various Γ values for Model *B*. Hereditary sexuality is not allowed. All the points collapse onto a single curve in the interval shown.

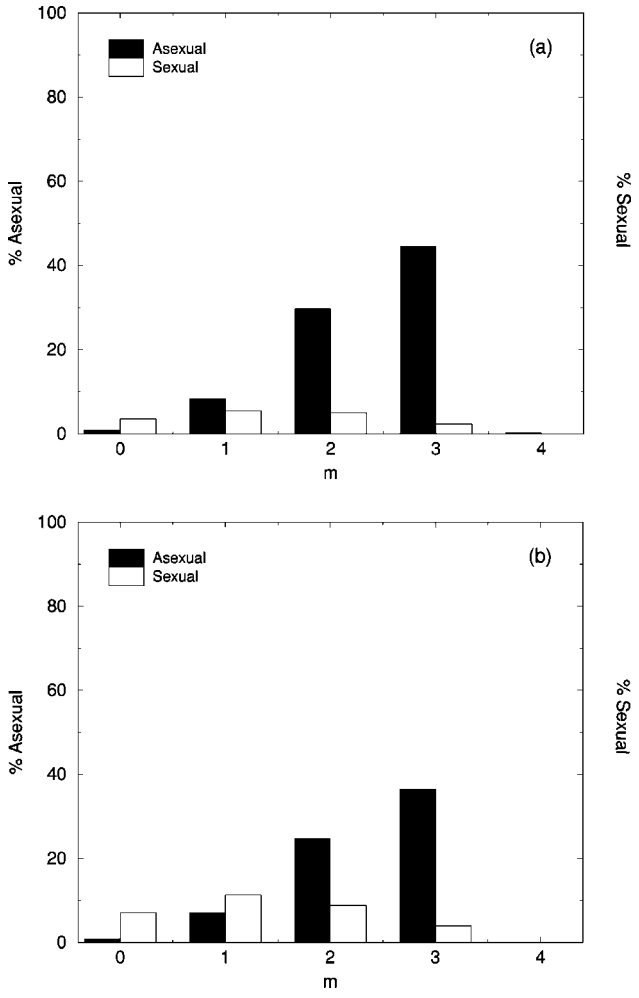

FIG. 6. Distribution of the asexual and sexual populations with respect to m, for different mutation rates in Model *B*, without hereditary sex. (a) $\Gamma=6\times10^{-3}$, (b) $\Gamma=2\times10^{-3}$. The histograms represent averages over 10 runs for a population of 1000, and $\sigma=10^{-4}$.

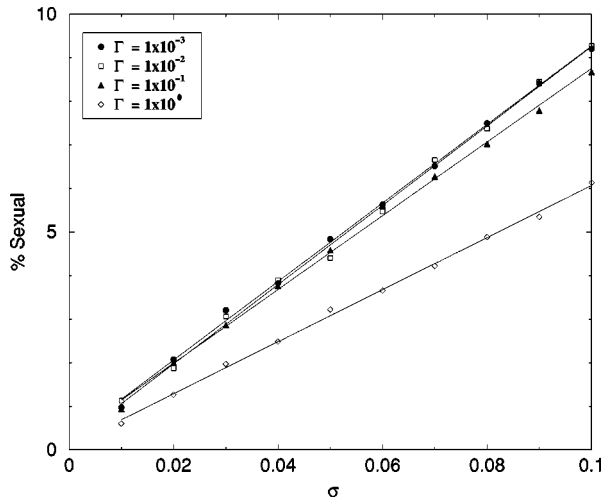

FIG. 7. The percentage of the sexual population vs σ for various Γ values for Model *B* with hereditary sex. The growth with σ is linear for the different Γ values.

The characteristic sandpilelike [24] distribution of asexuals is accompanied by a distribution of sexuals that is again shifted towards smaller values of *m*. It is interesting to observe that raising σ increases the total number of sexuals, and therefore depresses the number of asexuals, as is to be expected. However, it is not immediately obvious why keeping σ fixed and decreasing the overall mutation rate should decrease the number of asexuals. Clearly, raising Γ increases the death rate of both types of organisms, but since the conversion to sex is not coupled to the increase in the number of mutations, an increased Γ benefits the asexuals who get cloned to make up the deficit population. For large values of σ, a novel phase transition takes place, which is the subject of Sec. IV.

Model B with hereditary sex. If the conversion to sexual reproduction is hereditary, then at any given time step all the sexual individuals mate, except for the odd guy out. In Fig. 7 we show the total percentage of the sexual population as a function of σ alone. One sees that the growth is very close to linear with σ, however, the collapse as a function of σ/Γ does not occur here. The curves extrapolate to zero at $\sigma=0$. As long as $\sigma>1/N$ one may have a small but nonvanishing sexual population. For smaller values of σ, the number of sexual individuals again fluctuates very strongly and is of $O(1)$ (see Sec. IV).

We have performed simulations on a purely diploid population with no conversion to sex, to be able to compare the advantages afforded by pure diploidy with those coming from sexual reproduction, under the same mutation rate. For $\Gamma=1/N$, the steady state distribution for the purely diploid population is 7, 27, 40, 26, and 0%, ($\pm 1\%$), for $m=0,\ldots,4$, respectively. The steady state distributions of all the models involving a conversion to sex yield *m* distributions for sexuals that are shifted to smaller values of *m* than those found for pure diploidy. Furthermore, the advantage is greatly increased in those cases where sex is hereditary, i.e., practiced more often.

III. MEAN FIELD EVOLUTION EQUATIONS

To try to understand analytically some of the features found from the simulations, we have examined the behavior of the iterative equations that can be obtained for the different densities involved. These equations follow directly from the definitions of the various rates and densities involved. The only assumptions needed are that (i) the hydrodynamic limit obtains, i.e., that the number of events per generation are correctly given by the product of the relevant rates and the densities, and (ii) each individual is able to sample the total population when it picks a mate. It is because of the latter assumption that we refer to the equations as ''mean field'' if all the individuals can pair with each other at any instant, it means that the interactions are of ''infinitely long range.'' On the other hand, in the limit of very slow driving, the first assumption breaks down and the simulation results depart from the iterative solution of the hydrodynamic equations, due to the eventually discrete nature of the phenomenon. This is discussed in Sec. IV B. Moreover, as we show in Sec. IV A, the iterative equations tend to smooth out the intermittent route to chaos that we observe in the numerical simulations, in the limit of strong driving (very large mutation rates).

Given that the mutation rate per individual is Γ, and that any of the *L* bits have equal probabilities of being hit, we see that in each generation, a haploid makes a transition from a state with *m* mutations to one with $m+1$ mutations with the probability $T_{m,m+1}(\Gamma)=\Gamma(L-m)/L$. If a mutation hits the same bit twice, its value will be reset to ''0,'' so that $T_{m,m-1}(\Gamma)=\Gamma m/L$ gives the probability per generation that a haploid with *m* mutated bits makes a transition to a state with one less. All other elements of this transition matrix *T* are zero, since each individual is tested only once to see if it will undergo a mutation (with probability Γ) and if yes, only 1 bit is mutated at random.

For low temperatures and for $\mu=4$, the survival probability is given by

$$P(m)=\begin{cases} 1, & m=0,\quad 1,\ldots,3 \\ \dfrac{1}{2}, & m=4 \\ 0, & m>4. \end{cases} \tag{2}$$

A. Asexual steady state

The time-evolution equations for the asexual population, with $n_H(m)$ being the number of individuals with *m* mutated genes, are, with the above definition of the mutation matrix,

$$\begin{aligned} n_H(m,t+1)=&(1-\Gamma)n_H(m)+\sum_{\delta=\pm 1} T_{m+\delta,m}n_H(m+\delta,t) \\ &-[1-P(m)]n_H(m,t) \\ &+\sum_{m'}[1-P(m')]n_H(m',t)n_H(m,t)/N_A. \end{aligned} \tag{3}$$

The first two terms describe the building up of the mutation load, i.e., "Muller's ratchet" [6], the third term subtracts off the number of individuals with m mutations that die off with probability $1-P(m)$. The last term is the source term, arising from the replacement of the deceased individuals by randomly cloning the extant ones and $N_A=\Sigma_m n_H(m)$ is the total number of asexual individuals.

For large β (say $\beta=10$), one effectively has

$$n_H(m,t+1)=(1-\Gamma)n_H(m,t)+\sum_{\delta=\pm 1} T_{m+\delta,m}n_H(m+\delta,t) + \frac{1}{2}n_H(4,t)n_H(m,t)/N_A \tag{4}$$

for $m<4$. The source term $[1-P(4)]n_H(4)n_H(m)/N_A$ has been replaced by its value $\frac{1}{2}n_H(4)n_H(m)/N_A$, and it is assumed that $n_H(m>4)\equiv 0$. This assumption is supported by numerical data in the steady state.

Note that for $\Gamma N\sim O(1)$, $n_H(4)$ will be small, i.e., of the order of unity. For $m=4$, this enables us to put the source term in the last equation equal to zero, since it will be of $O(1/N)$ while the other terms are of $O(1)$, and we get

$$\begin{aligned} n_H(4,t+1)&=(1-\Gamma)n_H(4,t)+\sum_{\delta=\pm 1} T_{4+\delta,4}n_H(4+\delta,t) - \frac{1}{2}n_H(4,t) \\ &=(1-\Gamma)n_H(4,t)+\Gamma(1-3/L)n_H(3) - \frac{1}{2}n_H(4,t). \end{aligned} \tag{5}$$

Then we see that in the steady state, one may replace $n_H(4)/2$ appearing in the source terms by $\Gamma[(1-3/L)n_H(3)-n_H(4)]$. This leads to equations that are homogenous in Γ in the steady state, yielding, therefore, a steady state distribution of the population between sexual vs asexual individuals that are independent of Γ at least for $\Gamma \geqslant 1/N$ (see Fig. 1). Iterating these equations leads to a steady state with an m distribution that is in agreement with the simulation results [18].

B. Coexisting asexual and sexual populations

We now define a new quantity $n_D(m)$ as the number of m-mutation strings that *belong to a diploid organism*. The expected number of diploid organisms with m expressed deleterious mutations can be obtained, once the $n_D(m)$ are known.

The probability for two strings with m_1 and m_2 mutations (i.e., bit set to "1") to give rise to m loci at which both bits are "1" can easily be calculated. It is given by

$$p(m;m_1,m_2) = \frac{m_1!m_2!(L-m_1)!(L-m_2)!}{L!m!(m_1-m)!(m_2-m)!(L-m_1-m_2+m)!}, \tag{6}$$

for $L-m_1-m_2+m>0$ and 0 otherwise. This expression is symmterical in m_1 and m_2, both of which must be greater or equal to m. The number of diploid organisms with m expressed mutations is then

$$n_s(m)=\frac{1}{2}\sum_{m_1=m}^{L}\sum_{m_2=m}^{L^*} p(m;m_1,m_2)n_D(m_1)n_D(m_2)/(2N_S), \tag{7}$$

where $N_S=\Sigma_{m=0}^{L} n_s(m)$ is the number of diploid organisms, and $L^*=\min[L,L+m-m_1]$. The factor of $\frac{1}{2}$ out front comes from converting from the number of gametes that are members of diploid organisms with m expressed mutations, to the number of such diploid organisms. The factor $n_D(m_2)/(2N_S)$ in the sum is the probability of encountering a gamete with m_2 mutations as the other member of the pair making up the diploid organism.

A similar computation leads to the number of diploid individuals who die as a result of too many mutations,

$$D_D=\frac{1}{2}\sum_{m=0}^{L}\sum_{m_1=m}^{L}\sum_{m_2=m}^{L^*}[1-P(m)] \times p(m;m_1,m_2)n_D(m_1)n_D(m_2)/(2N_S), \tag{8}$$

where L^* is defined as above.

The number of gametes with m mutations, which get removed because they happen to be members of diploid organisms that die, is

$$d_m=\sum_{m''=0}^{L}\sum_{m'=0}^{\min[m,m'']}[1-P(m')] \times p(m';m,m'')n_D(m,t)n_D(m'',t)/(2N_S). \tag{9}$$

We must also define the number of gametes with m bits set to "1," that can take part in sexual reproduction, which is

$$\tilde{d}_m=\sum_{m'}^{\tilde{L}} p(4;m,m')n_D(m,t)n_D(m',t)/(2N_S), \tag{10}$$

where $\tilde{L}=\min[L,L+4-m]$. Since $\tilde{d}_m$ is only defined for $m \geqslant 4$, $\tilde{L}=L+4-m$. Note that $\Sigma_{m=4}\tilde{d}_m=2n_s(4)$.

Here we have only considered the scenarios without habitual sex.

1. Model A

We now have, from Eqs. (4) and (5), for sufficiently large β

$$n_H(m,t+1)=(1-\Gamma)n_H(m,t)+\sum_{\delta=\pm 1} T_{m+\delta,m}n_H(m+\delta,t) - \delta_{m,4}n_H(4,t)+\left[\frac{3}{4}n_H(4,t)+D_D(t) + \frac{1}{4}n_s(4,t)\right]n_H(m,t)/N_A. \tag{11}$$

The terms proportional to Γ are due to random mutation. The coefficient of the Kronecker delta $\delta_{m,4}$ is $n_H(4)$ since all of the asexuals with $m=4$ are removed either due to death or conversion to sexuals. The final term represents the number of m-mutation haploids that get cloned to keep the population constant. The expression in the square brackets is the number of individuals that get removed from the population and determines the strength of this source term. The 3/4 factor multiplying $n_H(4)$ comes from two parts: one-half of the haploids with 4 mutations die; the other half is converted to sex, and mate, their number being once more halved as a result, contributing $(1/4)n_H(4)$ to the "removals." D_D [which is $=(1/2)n_s(4)$ for large β] is the number of diploids that die, and $(1/4)n_s(4)$ comes from half of the $m=4$ diploid population being converted to sex, their number being once more halved when they mate.

The dynamics of the number of strings $n_D(m)$ that make up diploid organisms is given by

$$n_D(m,t+1)=\left(1-\frac{1}{2}\Gamma\right)n_D(m,t) + \sum_{\delta=\pm 1} T_{m+\delta,m}\left(\frac{1}{2}\Gamma\right)n_D(m+\delta,t)-d_m(t) - \frac{1}{4}\tilde{d}_m+\delta_{m,4}P(4)n_H(4,t). \tag{12}$$

For diploids, the probability of a mutation hitting any one gene is halved, because there are twice as many of them. The d_m term is the number of m gametes that are removed as a result of death, and in practice (for large β) is nonzero only for $m\geqslant 4$. The next term gives the reduction in the number of m gametes as a result of sexual reproduction. A factor of 1/2 comes from the probability to engage in sex, and another from the fraction of gametes that are discarded as a result. Finally, there is a contribution from the conversion of haploids to diploids. We have neglected the situations where (a) there is only one active sexual individual present, so that no mating can take place and a gamete is discarded, or (b) there is only one haploid strand with 4 mutations, and therefore no pairing of two such haploids can take place to give rise to a diploid. It can be checked explicitly that Eqs. (11) and (12) conserve the total population.

Iterating these equations leads to a steady state distribution that is roughly compareble but not identical to the simulation results (see Table I). For $\Gamma=10^{-3}$ the percentage of the sexual population is 24% of the total, and saturates to 36% as Γ is increased, as compared to 70% from the simulations. This discrepancy seems to come from the fact that the dynamics is really driven by the strongly fluctuating small population at $m=4$, and mean field theory is simply not able to capture this.

The distribution over m is also modified. One sees that the distribution of the asexuals is quite similar to the simulation results, while the peak of the sexual distribution has shifted to $m=1$ from $m=2$. This indicates that the mean field theory overestimates the effect of remixing, as is to be expected, since the gametes, instead of being paired in a definite way at any given moment, are perpetually part of a single gene pool.

TABLE I. The distribution of the population with respect to the number of expressed mutations, obtained from an iteration of the mean field equations for Model *A*.

	$\Gamma=10^{-3}$			$\Gamma=10^{-2}$	
m	Asexual%	Sexual%	m	Asexual%	Sexual%
0	0.9	8.5	0	0.8	9.4
1	7.8	11.0	1	6.5	16.2
2	26.7	4.4	2	22.3	8.8
3	40.1	0.6	3	33.8	1.9
4	0.0	0.0	4	0.1	0.2
Total	75.5	24.5	Total	63.5	36.5

2. Model B

In this case we have a uniform probability for conversion to sex. The equations become

$$n_H(m,t+1)=(1-\Gamma)n_H(m)+\sum_{\delta=\pm 1} T_{m+\delta,m}n_H(m+\delta) -[1-P(m)]n_H(m,t)-\sigma n_H(m) +\left\{\sum_{m'}[1-P(m')]n_H(m')+\frac{1}{2}\sigma N_A +D_D(t)+\frac{1}{2}\sigma N_S(t)\right\}n_H(m)/N_A. \tag{13}$$

Here, haploids are converted to diploids and removed at the rate of σ, and the reduction in the population due to mating of recent converts gives the $\frac{1}{2}\sigma N_A$ term in the source. The sexuals moreover mate among each other with probability σ, which leads to a further sink with strength $\frac{1}{2}\sigma N_S$. Apart from these, the terms are identical to Eq. (11). The dynamics of the m gametes are

TABLE II. The distribution of the population with respect to number of expressed mutations, obtained from an iteration of the mean field equations for Model *B*.

	$\sigma/\Gamma=0.01$			$\sigma/\Gamma=1.00$	
m	Asexual%	Sexual%	m	Asexual%	Sexual%
0	2.9	9.3	0	1.7	32.2
1	14.3	3.0	1	8.3	14.7
2	32.4	0.4	2	18.8	2.3
3	37.7	0.0	3	21.9	0.1
4	0.1	0.0	4	0.0	0.0
Total	87.3	12.7	Total	50.7	49.3

$$n_D(m,t+1)=\left(1-\frac{1}{2}\Gamma\right)n_D(m,t)$$
$$+\sum_{\delta=\pm 1} T_{m+\delta,m}\left(\frac{1}{2}\Gamma\right)n_H(m+\delta,t)-d_m$$
$$-\frac{1}{2}\sigma n_D(m,t)+\sigma n_H(m). \quad (14)$$

In this case, the iterations of mean field equations yield results (see Table II) that are much closer to those found from the simulations.

The evolution equations, which we have written as difference equations, are of course nonlinear. In the simplest case of asexual reproduction [Eqs. (4), (5)] this second order nonlinearity comes purely from the condition of a fixed finite population, and appears in the source term for the restoration of the population to its fixed value by randomly sampling the asexual population and cloning it. With the introduction of sex, the source term in the equations for the asexual organisms (11) and (13) acquires a contribution from the number of sexual individuals that are removed either through death or through sexual reproduction. Such terms contain nonlinearities up to third order. We expect to find nontrivial behavior in the limit of large nonlinearities in these equations, and this turns out to be the case, as we explore in the next section.

IV. LIMITS OF STRONG AND EXTREMELY WEAK DRIVING, CHAOTIC BEHAVIOR

A. The limit of strong driving

An inspection of the hydrodynamic equations describing the system, presented in the last section, leads us to suspect that the nonlinearities in the problem could give rise to chaotic behavior when their amplitude is sufficiently large. In this section we present results obtained in the strong driving limit from numerical simulations of the stochastic models and from iterations of the hydrodynamic equations.

We have performed simulations in the limit of $\Gamma=1$ and found that for Model *A* with hereditary sex, the system becomes unstable. The total asexual population and sexual population display oscillations with a period of 2 time steps. The *m* distributions also oscillate for both populations, with the same period, the amplitude of the oscillations being much larger for the asexuals. For such large values of Γ, at each time step a large number of asexuals are driven to large *m* values and are converted to sexuals, they mate, and reduce their expressed mutations. This leads to a macroscopic fluctuation in the number of sexuals, with the halving of the mating population, which then causes a very large number of asexuals to be cloned in turn. The time average of the sexual population is depressed slightly below the saturation value as a result, as can be seen in Fig. 3. These oscillations are not observed in the iteration of the mean field equations.

A much more striking behavior is found in Model *B* for large values of σ. As we increase the value of σ, the probability of random conversion to sex, beyond about 0.05, a spectacular transition takes place to a strange attractor for the

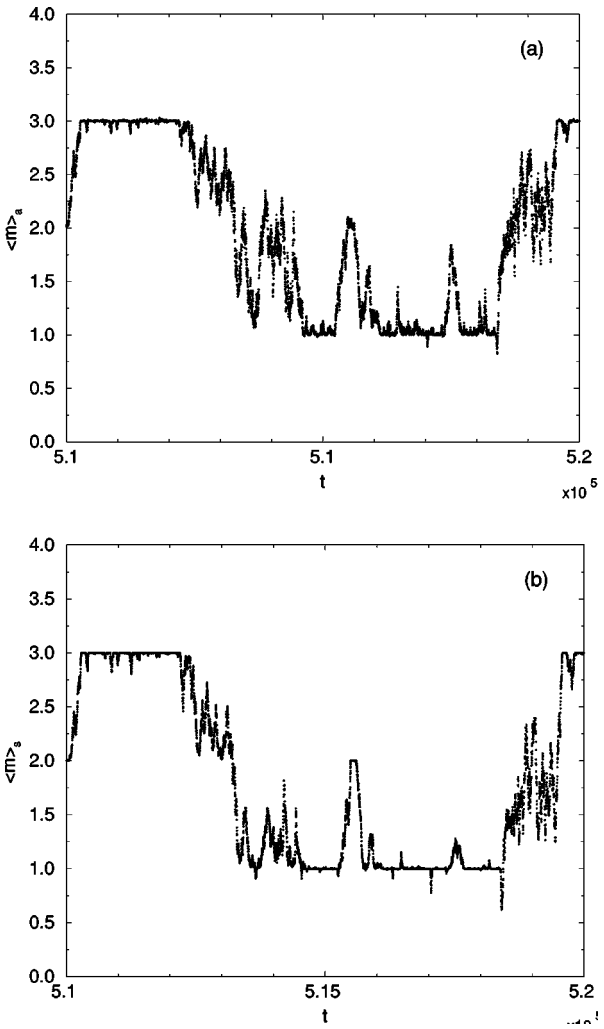

FIG. 8. The intermittent variation with time of simulations results for (a) $\langle m\rangle_a$, the average number of mutations for the asexual population, and (b) $\langle m\rangle_s$, the average number of expressed mutations for the sexual population, in Model *B*, for $\sigma=0.5$. The averages are taken over the population at time *t*. $\Gamma=10^{-3}$. It is clearly seen that there are two metastable states. The figures show a window of 10^4 time steps after the transients are dropped.

dynamics of both the asexual and sexual populations. In place of the well converged *m* distributions for both asexual and sexual populations, shown in Figs. 6 one observes that both distributions are intermittently switching between several metadistributions. The average value of *m* computed over the asexual and the sexual populations is shown in Fig. 8, and displays this striking intermittent behavior, where the distribution of the two populations becomes much more closely coupled than in the lower σ values. They now move more or less *in phase*, and their excursions take them all the way down to the wild type. Now it is only possible to talk about a distribution of distributions. To display this graphically, we have plotted the distribution of the average number of expressed mutations in the two populations, $\langle m\rangle_a$ and $\langle m\rangle_s$, as a function of σ. In Fig. 9, we show three dimensional plots for these distributions, compiled over 10^4 time steps for each value of σ. In Fig. 10, a contour plot of the

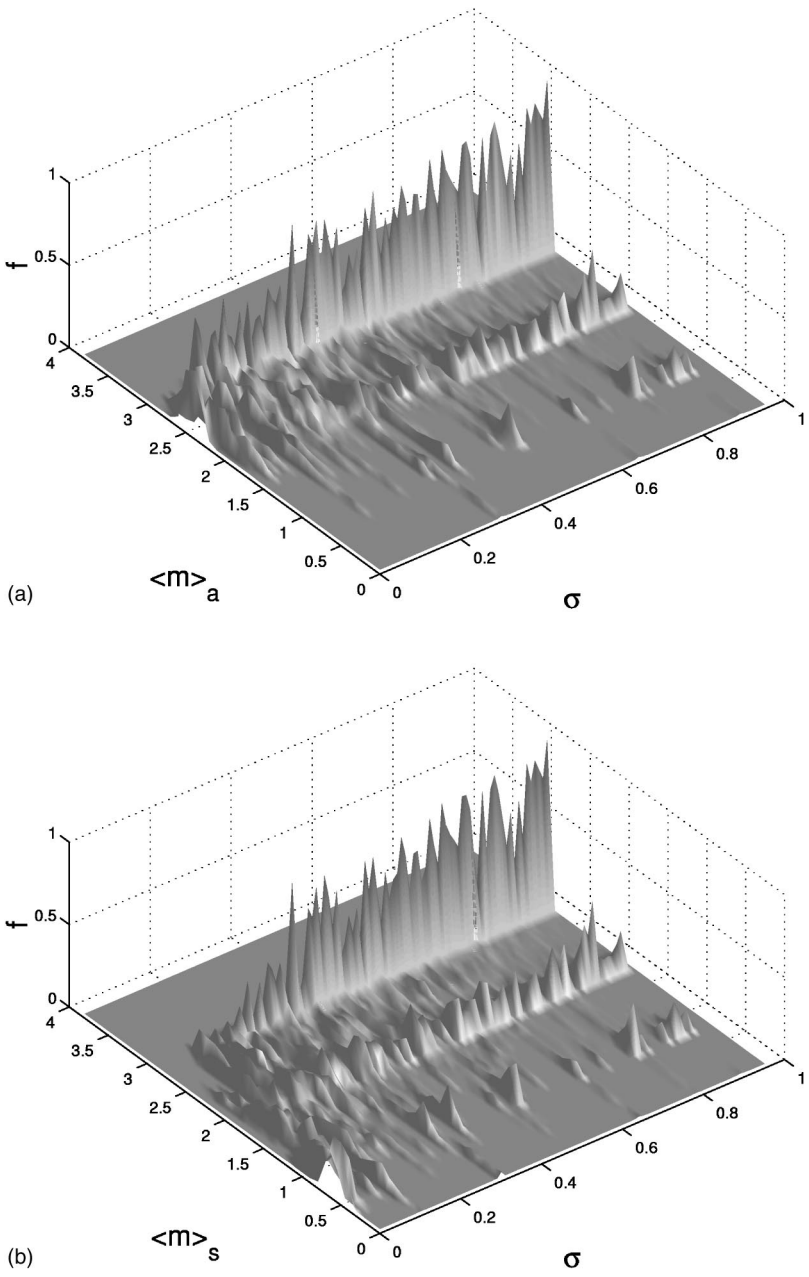

FIG. 9. A three-dimensional plot showing the branching distributions of (a) $\langle m \rangle_a$ (b) $\langle m \rangle_s$ with respect to σ. After a threshold at $\sigma \sim 0.05$, the distribution displays more than one peak. The z axis indicates the relative weights of these peaks. The total population is 1000 and the figure represents single runs of 10^4 steps after transients, for each σ value.

same distribution as in Fig. 9 are shown. It is possible to read off from the contour plots that the transition is taking place around $\sigma_c \simeq 0.05$.

Besides being intermittent, this transition has a dramatic effect on the m distribution of the sexual population, in that it shifts it to much higher values. It can be seen in Fig. 10(b) that for $\sigma < \sigma_c$, the mean m for the sexual population is $\langle m \rangle_s \sim 0.75$, while for large σ it is comparable to the corresponding value for the asexual population, closer to 3. The reason seems to be that with the great depletion of the population when too many individuals are being switched on to sex and engaging in sexual reproduction, the asexuals are cloning too many identical copies to make up for the deficit. When these are subsequently turned sexual and mate among each other, ''inbreeding'' takes place. There is not sufficient genetic diversity for sex to lead to sufficient mixing and therefore an amelioration of the effective fitness.

We have iterated the mean field equations (13) and (14) for Model *B* and found that this intermittent behavior is suppressed. The sexuals simply evolve along the lower branch that in the simulations has the smaller weight, while the asexuals evolve along the higher (large m) branch, which has the greater weight in the simulations, and the evolution is completely stable. For $\sigma = 0.9$ and $\Gamma = 0.1$, $\langle m \rangle_a = 2.43$ and $\langle m \rangle_s = 0.47$.

B. The limit of infinitely slow driving ($\Gamma \to 0$)

We find that for very slow driving, below a threshold at $\Gamma \sim 1/N$, there is an abrupt transition to qualitatively different m distributions in both the asexual and sexual populations.

In the purely asexual population without any conversion to sex, for $\Gamma < 1/N$, we find a qualitatively different asexual steady state, where the m distribution has shifted to lower m

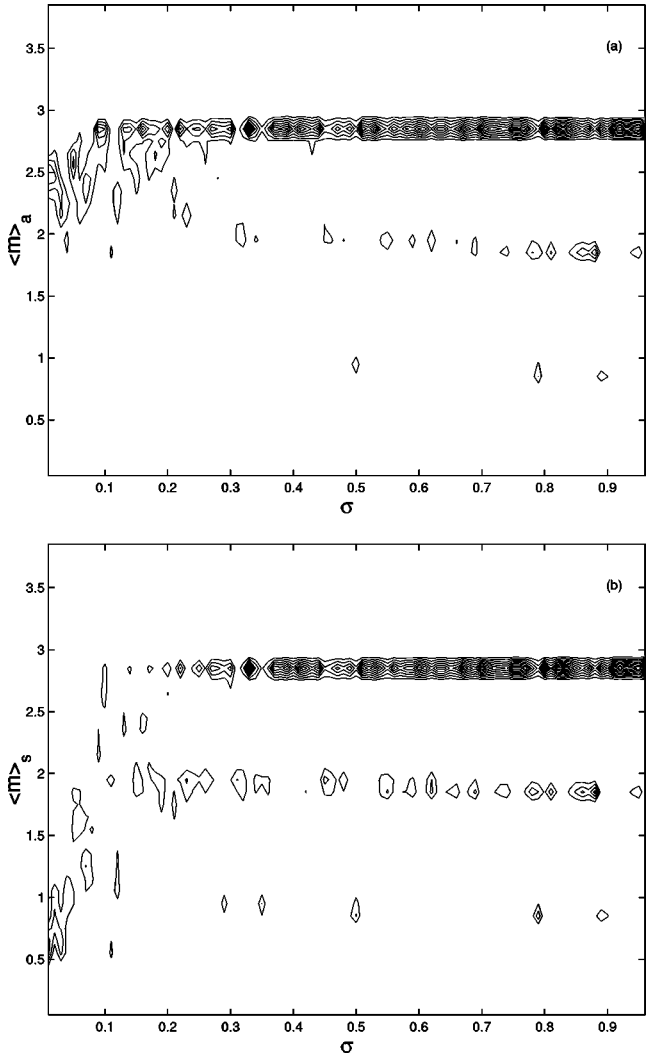

FIG. 10. Contour plot corresponding to Fig. 9, showing the branching of (a) $\langle m\rangle_a$ (b) $\langle m\rangle_s$, as σ increases, for a population of 1000, computed over 10^4 time steps.

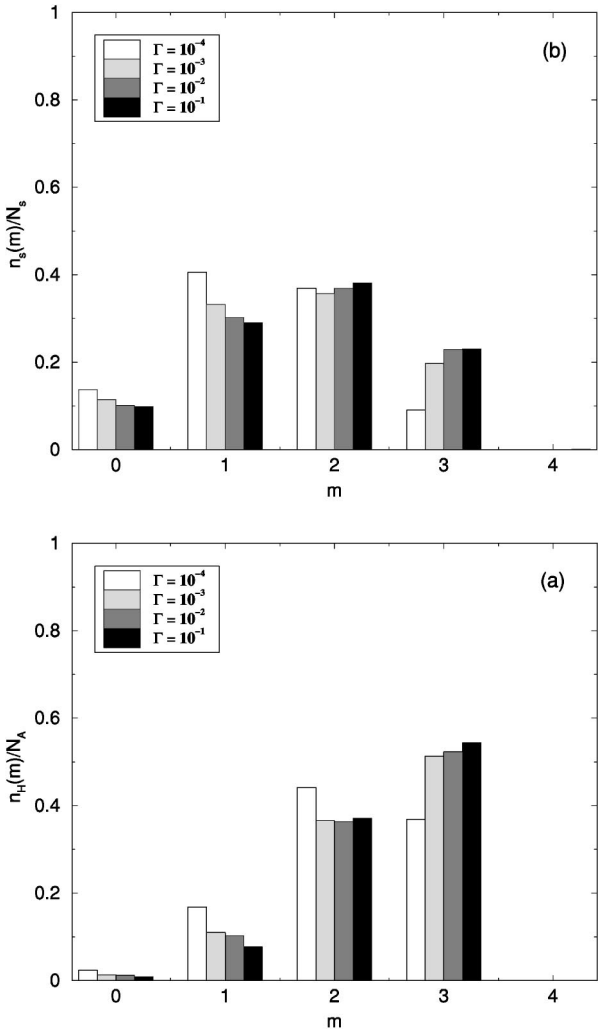

FIG. 11. The distribution over m for (a) asexual (b) sexual populations, for different values of Γ for Model *A*, without hereditary sex. The steady state distribution changes and the peak of the distribution shifts to a smaller m value as one lowers the Γ value below the threshold $1/N=10^{-3}$.

values (compare with Fig. 2 of [18]) and no longer has the characteristic minimally stable sandpilelike [24] distribution. For $\Gamma=10^{-4}=(10N)^{-1}$, over a run of 10^6 steps, we find $n_H(m)/N\simeq 0.03,\ 0.14,\ 0.44,\ 0.39$ for $m=0,\ldots,3$ respectively, where the peak has moved to $m=2$ from $m=3$, and broadened towards the left.

Once sex is turned on in Model *A*, we similarly observe that the peaks in the distribution of the asexual and sexual populations have shifted to lower m values ($m=2$ and $m=1$ respectively) for $\Gamma<1/N$, as shown in Fig. 11. Although the total sexual population is relatively small here, we have checked that the fluctuations in the histogram over ten different realizations stay small.

Iteration of the dynamical equations, on the other hand, reveal no transition at $\Gamma\sim 1/N$, and, for the asexual steady state, converge to the same steady state distributions as found for $\Gamma>1/N$. In Fig. 12, we show the time series for $n_H(m)$ ($N=100$) for the asexual population without conversion to sex. At time $t=0$, the largest density is of course at $m=0$, and then the maximum shifts successively to $m=1,2$ and finally to $m=3$ where it stabilizes. This is a graphic manifestation of the breakdown of the hydrodynamic approximation in the weak driving limit.

The mechanism for the transition can be understood as follows. In the simulations one has to wait around until, with a very low probability, a discrete individual is pushed over the $m=4$ barrier, dies, and a live organism is cloned at random to replace it. The separation of the time scales for mutation (and eventual death) and the immediate replenishment from the distribution at that instant, is what gives rise to the transition. On the other hand, the mean field equations describe a situation with a weak but steady seepage due to the nonvanishing mutation rate, and this prevents the transition from taking place.

Biologists denote the threshold mutation rate, below which the population is peaked at $m=0$, i.e., around the wild type, as the ''error threshold'' [25–27]. Above this threshold, it is generally observed [26,27] that there is a sudden delocalization of this peak to a finite value of $m\neq 0$. Strictly

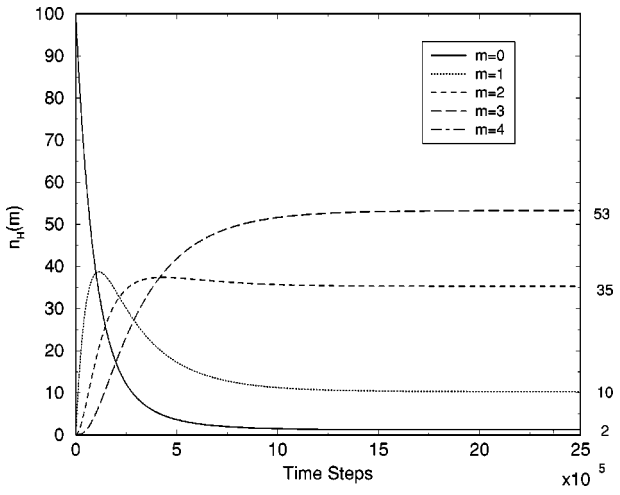

FIG. 12. The iterated solutions of the equations for the purely asexual population, without the introduction of sex, as a function of time for different values of m. $\Gamma = 10^{-4}$.

speaking, this is a nonequilibrium phase transition [26] only in the thermodynamic limit, where L, the size of the genome, goes to infinity. Nevertheless, what we have here is a finite system manifestation of the same phenomenon. The transition that we observe at $\Gamma \sim 1/N$ suggests that at even smaller values of Γ (and for larger L) there could be yet other transitions where the the peak in the distribution would shift down to $m=1$ and eventually $m=0$.

V. DISCUSSION AND CONCLUSIONS

The mechanism of random conversion to sex, in the presence of a constant rate of mutation, investigated in this paper as a scenario for the maintanence of a macroscopic sexual population, is in fact very closely related to ''coevolution of cell senescence and diploid sexual reproduction in unicellular organisms,'' studied by Cui *et al.* [12]. In this paper a ''senescence clock'' ticks off a finite lifetime for each bit string. Sexual reproduction (conjugation) resets the senescence clock; unless this happens after a number of generations of cloning, the offspring stop dividing and die.

Our Model *B* can be seen as a simpler version of the model proposed by Cui *et al.*, with an intrinsic mechanism, provided by Muller's ratchet [6], for cell senescence. The constant mutation rate sets the time scale for the survival of any given individual, unless it succeeds engaging in sex, with a given probability (our σ). A survival function [Eq. (1)] leads to the elimination of genomes carried by haploid individuals multiplying by asexual reproduction, once they have accumulated too many mutations as a result of prolonged exposure to the constant mutation rate [1,6].

Our Model *A* goes one step further, in that it makes the number of mutations (the cell clock), provide the triggering mechanism for the transition to diploidy and sex. It is gratifying to find that this is a more successful strategy for establishing a sexual population than a constant rate of conversion to sex.

Chopard *et al.* [28] have pointed out that care must be taken in the investigation of finite populations, amplifiying and stabilizing small fluctuations that in the thermodynamic limit would be attenuated to zero. They emphasize the importance of spatial variations that cannot be captured by mean field theories. In this paper we have demonstrated the relevance for finite populations of discrete stochastic events, whose effect in the very weak driving limit cannot be captured by the ''mean field'' equations. In the very weak driving limit the system is below the hydrodynamic regime, and exhibits a qualitatively different phase than that described by the continuum approximations.

In a recent paper Pekalski [17] has studied a model that is in many ways similar to ours. There the success of sexual reproduction, meiotic parthenogenesis, and asexual reproduction, in maintaining a finite population in the face of periodically changing environmental conditions and a constant mutation rate, is studied in terms of the relative sizes of the populations. Age is included in the model as a parameter that reduces the fitness. The populations do not interact. The findings are that meiotic parthenogenesis and sexual reproduction are more favorable than mitotic reproduction, with slight differences between them depending on the precise conditions.

We may conclude that the advantage of sexual reproduction over pure diploidy, in leading to greater fitness and therefore to a reduced mortality rate, comes more strongly into play with a sufficiently large frequency of mating, as found for the hereditary and habitual practice of sex in both Models *A* and *B*. This frequency is driven by the mutation rate Γ in Model *A*, and by the probability σ in Model *B*, namely, the same mechanism as the conversion from haploidy to diploidy. On the other hand, greater frequency of mating, with the fusion of two gametes, one from each parent, means a ''$2 \rightarrow 1$'' reduction in numbers, and this effect competes with the advantage gained from increased fitness, leading to a saturation of the sexual population at increased rates, to $\sim 15\%$ as $\Gamma \rightarrow 1$ for Model *A*, and $\sim 10\%$ as $\sigma \rightarrow 1$ for Model *B*.

It is important to note that in both models the steady rate of conversion from haploidy creates a small but vital source of diploid and (for hereditary sex) sexual organisms. Results on the autonomous viability of the sexual population, after the steady conversion from the haploid population has been switched off (but mitosis allowed for the diploids), will be reported in a future publication.

Further work is in progress, to investigate the effect of finite temperature, and of including the possibility of genetic crossover and meiotic parthenogenesis, in our models.

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[1] J.M. Smith and E. Szathmáry, *The Major Transitions in Evolution* (Oxford University Press, Oxford, 1995).
[2] J. Maynard Smith, *Evolutionary Genetics* (Oxford University Press, Oxford, 1999), p. 230.
[3] L.M. Prescott, J.P. Harley, and D.A. Klein, *Microbiology* (McGraw-Hill, New York, 1999), p. 533.
[4] E.P. Solomon, L.R. Berg, and D. Martin, *Biology* (Saunders, Philadelphia, 1999), pp. 31–33.
[5] S.S. Mader, *Biology* (McGraw-Hill, New York, 1996), p. 414.
[6] H.L. Muller, Mutat Res. **1**, 2 (1964).
[7] A. Kondrashov, Nature (London) **336**, 435 (1988).
[8] R.J. Redfield, Nature (London) **369**, 145 (1994).
[9] D. Stauffer, P.M.C. de Oliveira, S. Moss de Oliveira, and R.M. Zorzenon dos Santos, Physica A **231**, 505 (1996).
[10] D. Stauffer, Physica A **273**, 132 (1999).
[11] A. Bernardes, J. Stat. Phys. **86**, 432 (1997).
[12] Y. Cui, R.S. Chen, and W.H. Wong, Proc. Natl. Acad. Sci. U.S.A. **97**, 3330 (2000).
[13] T.J. Penna, J. Stat. Phys. **78**, 1629 (1995).
[14] W.D. Hamilton, R. Axelrod, and R. Tanese, Proc. Natl. Acad. Sci. U.S.A. **87**, 3566 (1990).
[15] E.J. Lyons, Nature (London) **390**, 19 (1997).
[16] J.S. SaMartins, Phys. Rev. E **61**, 2212 (2000).
[17] A. Pekalski, Eur. Phys. J. B **13**, 791 (2000).
[18] B. Örcal, E. Tüzel, V. Sevim, N. Jan, and A. Erzan, Int. J. Mod. Phys. C **11**, 973 (2000), and references therein.
[19] N. Jan, D. Stauffer, and L. Moseley, Theory Biosci. **119**, 166 (2000).
[20] J. Maynard Smith, *Evolutionary Genetics* (Oxford University Press, Oxford, 1999), p. 52.
[21] J. Thoms, P. Donahue, and N. Jan, J. Phys. I **5**, 935 (1995).
[22] V. Sevim and A. Erzan (unpublished).
[23] J. Maynard Smith, *Evolutionary Genetics* (Oxford University Press, Oxford, 1999), p. 228.
[24] C. Chisholm, N. Jan, P. Gibbs, and A. Erzan, Int. J. Mod. Phys. C **11**, 1257 (2000).
[25] M. Eigen, *Steps Towards Life, a Perspective on Evolution* (Oxford University Press, Oxford, 1992).
[26] B. Drossel, Adv. Phys. **50**, 209 (2001).
[27] L. Peliti, e-print cond-mat/0105379.
[28] B. Chopard, M. Droz, and S. Galam, Eur. Phys. J. B **16**, 575 (2000).