Strategies for the Evolution of Sex

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We find that the hypothesis made by Jan, Stauffer and Moseley [Theory in 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, multi-stable 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 asexual population.

Keywords: Dynamics of evolution, sexual reproduction, intermittency, Monte Carlo methods.

I. INTRODUCTION

In a previous paper\textsuperscript{1} we have shown that a finite steady state sexual population may arise from a purely asexual one, if an excess of deleterious mutations causes the individual to engage in sex as a means of escaping death\textsuperscript{2}. Since sexual reproduction led, in our model, to diploidy, it implied also the adoption of a mechanism for preferential expression of certain genes, and we assumed deleterious mutations to be recessive.\textsuperscript{3} Under various different assumptions regarding the subsequent mode of reproduction (i.e., whether sexual reproduction is hereditary or not) and of the number of offspring, 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 population. Thus, in one of our models (Model I)\textsuperscript{4}, we were able to demonstrate a possible scenario for the evolution of the analogue of a “haploid - diploid cycle”\textsuperscript{5} where the organisms produce by asexual reproduction as long as they are reasonably fit (or the conditions reasonably favorable) but engage in sexual reproduction when the going gets tough.\textsuperscript{6}\textsuperscript{7}

In this 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 $\Gamma$ of random mutations. Both haploid and diploid organisms die when the number of their expressed deleterious mutations exceed a certain number. (Henceforth we will omit “deleterious” where it is evident that we mean a change away from the wild type gene.)

The first strategy (Model A) is the adoption, with a certain probability, of sexual reproduction and consequent diploidy when the number of mutations exceeds a threshold, threatening extinction. The second strategy (Model B) involves a small but constant probability $\sigma$ for the accidental conversion to sex, independently of the number of mutations (or, equivalently, the fitness) of the individual. The cloning of sexual individuals is not allowed in either Model $A$ or $B$. On the other hand, we have tested for the effect of hereditary (habitual) v.s. non-hereditary (non-habitual) sex.

In the implementation of both models, we have adopted a more realistic set of rules for the mechanism of dominance, that is, the expression of mutated alleles, than in our previous paper\textsuperscript{1}. 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.

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.

The organisation 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 Section III, we display and examine the mean field evolution equations and discuss our findings in the light of these equations. In Section 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 intermit-
tent route, found for large values of $\sigma$. A discussion of
the results from similar models and directions for further
research are provided in section 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, after the Penna model [9]. 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 which are allowed to be different,
that is the individuals are now diploids.

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 $\Gamma$
picking those to be mutated. Clearly there may be
any number of mutated individuals at any one generation
(time step), the number fluctuating around $\Gamma N$, 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 alle-
les are set to “1.” This is how we model the mechanism
dominance of the wild type (or, equivalently, the re-
cessiveness of deleterious mutations.) We will use the
term “fitness” loosely, for $L - m$; thus increasing $m$ will
decrease the fitness of the individual.

In the steady state, the distribution of the asexual
and sexual populations over $m$, are independent of $\Gamma$, for $\Gamma > 1/N$. The cases where $\Gamma < 1/N$ and $\Gamma \approx 1$ have interesting
consequences, and are discussed in section IV.

The probability of survival as a function of $m$, is given
by a Fermi-like distribution [8], $P(m)$

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

(1)

For large $\beta$ (or “low temperatures,” in the language of
statistical mechanics), $P(m)$ behaves like a step function [8]. Individuals with $m > \mu$ die, those with $m < \mu$
survive, and those with $m = \mu$ survive with a probability
of 1/2. In the simulations we set $\beta = 10$ and $\mu = 4$.

We keep the total population constant, as in the Red-
field model [8], by making up for the deficit in the popu-
lation 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 one 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 proba-
ability 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, where $2L$ is the total number of genes
in a diploid individual, or, in other words, the number of
mutated genes in the population is greater than the
total number of genes of one individual. In all the sim-
ulations, 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. We will report on
situations where we encounter an intermittent route to
chaos in Section IV.

A. Asexual steady state

We start with a set of $N$ initially identical asexual
individuals, all identical to the wildtype, i.e., all
0’s. Under the conditions outlined above, without intro-
ducing sex, the population of asexuals settles down to
the steady state distribution given in $\Gamma$ for $\Gamma \geq 1/N$
namely, $n_{H}(m)/N_A = 0.012, 0.098, 0.356, 0.531, 0.001$ for
$m = 0, \ldots, 4$. In this region this steady state distribution
is independent of $\Gamma$, which only sets the scale of time.
That this should be so, is not self-evident, and only fol-

dows from the form of the solution to the set of evolution
equations, as shown in Section 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 ei-
ther case, the haploid organism first makes a copy of its
own set of genes, as if it were going to perform mito-
sis, but then forms two gametes instead. One of these
gametes will pair up with a gamete from another indi-
vidual who has been turned on to sex, and the other will
be discarded. One should note that sexual reproduction
may be implemented in different ways, resulting in dif-
ferent numbers of offsprings produced [10]. 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 offspring will in-
crease the advantage that the sexual population enjoys.
Indeed, judging from our previous results [10], the number
of offspring exceeding two would lead to the takeover of
the population by the diploid 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.

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 \( \Xi \). 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 \( m \geq \mu \) mutations) survive, then they are turned sexual by deterministically and irreversibly switching their sign bits to one. Once their sex bit is turned on, these individuals will be labelled “sexually active” and mate 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.

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

![Figure 1](image1.png)

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

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.

![Figure 2](image2.png)

**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.

The steady state distributions of both asexuals and sexuals with respect to \( m \) are also independent of \( \Gamma \), (See Figure 2) for \( \Gamma \geq \frac{1}{N} \) and sufficiently smaller than 1. The peak of the distribution shifts towards lower \( m \) values for sexuals as a result of the salutary effect of dominance in diploidy, and the reshuffling effect of sexual reproduction.
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 previous generation. As in the case of non-hereditary 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 $\Gamma$ and reaches only $\sim 15\%$ (as compared to 70% for non-hereditary sex) as $\Gamma \approx 1$ (see Fig. 3). The $m$-distributions are shown in Figs. (4a,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.

Our second strategy for conversion to sex involves a constant probability $\sigma$ for the accidental conversion to sex, independently of the distance, as expressed by $m$, from the wildtype. For this model (Model B), once the asexual steady state is reached, at each generation we allow the sex gene to be “turned on” irresursively, with a small probability $\sigma$ for each individual. Like in 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 non-hereditary, after mating the sexual individual becomes sexually inactive. (Within some subsequent generation it can once more become sexually active with probability $\sigma$). 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 $\sigma/\Gamma$, as can be seen
from the figure, and grows with $\sigma/\Gamma$. In Fig. 6(a,b), we display the distribution of asexual and sexual individuals over the effective number of mutations $m$, for two small values of $\sigma$ and $\Gamma$. The characteristic sandpile like distribution of asexuals is accompanied by a distribution of sexuals which is again shifted towards smaller values of $m$. It is interesting to observe that raising $\sigma$ 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 $\sigma$ fixed and decreasing the overall mutation rate should decrease the number of asexuals. Clearly, raising $\Gamma$ 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 $\Gamma$ only benefits the asexuals who get cloned to make up the deficit population. For large values of $\sigma$, a novel phase transition takes place, which is the subject of Section 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 $\sigma$ alone. One sees that the growth is very close to linear with $\sigma$, however the collapse as a function of $\sigma/\Gamma$ 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 $\sigma$, the number of sexual individuals again fluctuates very strongly and is of $O(1)$. (see Section IV).

## III. MEAN FIELD EVOLUTION EQUATIONS

To try to understand analytically some of the features found from the simulations, we have examined the behaviour of the iterative equations that can be obtained for the different densities involved.

Let us define the mutation matrix for haploids, $T_{m,m+1}(\Gamma) = \Gamma (L - m)/L$ and $T_{m,m-1}(\Gamma) = \Gamma m/L$. Note that $\sum_{\delta = \pm 1} T_{m,m+\delta}(\Gamma) = \Gamma$. All the other elements of this matrix are zero.

For low temperatures and for $\mu$, the upper limit of the number of mutations tolerated by the haploid individual, being set to four, the survival probability is given by,

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

### 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
\[
n_H(m, t + 1) = (1 - \Gamma)n_H(m) + \sum_{\delta = \pm 1} T_{m + \delta, m} n_H(m + \delta, t) - [1 - P(m)]n_H(m, t) + \frac{1}{2}n_H(4, t) \frac{n_H(m, t)}{N_A} . \tag{3}
\]

The last term is the source term, arising from the replacement of the deceased individuals by randomly cloning the extant ones and \(N_A = \sum m n_H(m)\) is the total number of asexual individuals.

For large \(\beta\), one effectively has,
\[
n_H(m, t + 1) = (1 - \Gamma)n_H(m, t) + \sum_{\delta = \pm 1} T_{m + \delta, m} n_H(m + \delta, t) - \frac{1}{2}n_H(4, t) . \tag{4}
\]

for \(m < 4\). The source term \([1 - P(4)]n_H(4)n_H(m)/N_A\) has been replaced by its value \(\frac{1}{2}n_H(4)/N_A\), and it is assumed that \(n_H(m > 4) = 0\). 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,
\[
n_H(4, t + 1) = (1 - \Gamma)n_H(4, t) + \sum_{\delta = \pm 1} T_{4 + \delta, 4} n_H(4 + \delta, t) - \frac{1}{2}n_H(4, t) + \Gamma(1 - 3/L)n_H(3) \tag{5}
\]

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 homogeneous in \(\Gamma\) in the steady state, yielding, therefore, a steady state distribution of the population between sexual v.s. asexual individuals which are independent of \(\Gamma\) at least for \(\Gamma \geq 1/N\). (see Fig. 1) Iterating these equations leads to a steady state with an \(m\)-distribution that is in agreement with the simulation results [6].

**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
\[
p(m; m_1, m_2) = \frac{m_1 m_2 [(L - m_1)!(L - m_2)!]}{L![(m_1 - 1)!(m_2 - m)! ((L - m_1 - m_2 + m)!)}, \tag{6}
\]

for \(L - m_1 - m_2 + m > 0\) and 0 otherwise. This expression is symmetrical in \(m_1\) and \(m_2\), both of which must be \(\geq m\). The number of diploid organisms with \(m\) expressed mutations is then,
\[
n_s(m) = \frac{1}{2} \sum_{m_1 = m}^{L} \sum_{m_2 = m}^{L} p(m; m_1, m_2) \times n_D(m_1) n_D(m_2)/(2N_S), \tag{7}
\]

where \(N_S\) is the number of diploid organisms, \(\sum_{m=0}^{L} n_s(m)\), and \(L^* = \min[L, L + m - m_1]\). The factor of \(1/2\) 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 \(n_D(m_1)/2N_S\) in the sum is the probability of encountering a gamete with \(m_1\) 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,
\[
D_D = \frac{1}{2} \sum_{m=0}^{L} \sum_{m_1 = m}^{L} \sum_{m_2 = m}^{L} [1 - P(m)]p(m; m_1, m_2) \times n_D(m_1) n_D(m_2)/(2N_S), \tag{8}
\]

where \(L^*\) as defined as above.

The number of gametes with \(m\) mutations, which get removed because they happen to be members of diploid organisms which die, is
\[
d_m = \sum_{m''=0}^{L} \sum_{m'=0}^{L} [1 - P(m'')]p(m', m'') \times n_D(m, t)n_D(m'', t)/(2N_S). \tag{9}
\]

We must also define the number of gametes with \(m\) bits set to “1,” that can take part in sexual reproduction, which is
\[
\hat{d}_m = \sum_{m'=0}^{L} p(4; m, m') n_D(m, t)n_D(m', t)/(2N_S), \tag{10}
\]

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

Here we have only considered the scenarios without habitual sex.

**Model A**

We now have, from (6), for sufficiently large \(\beta\)
\[
n_H(m, t + 1) = (1 - \Gamma)n_H(m, t)
\]
Dynamics of the number of strands \( n_D(m) \) that make up diploid organisms is,

\[
n_D(m, t + 1) = (1 - \frac{1}{2} \Gamma)n_D(m, t) + \frac{1}{4} \sum_{\delta = \pm 1} T_{m+\delta, m} n_H(m + \delta, t) - d_m(t) - \frac{1}{4} \delta m A \sigma n_H(m + 4, t) n_H(m, t)/N_A .
\]

(11)

The terms proportional to \( \Gamma \) are due to random mutation. The coefficient of the Kronecker delta \( \delta_{m, A} \) 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 which get cloned to keep the population constant; the expression in the square brackets is the number of individuals which 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: \((1/2)\) of the haploids with 4 mutations die; the other half is converted to sex, and mate, their number being once more halved when they 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.

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 \( \beta \)) is nonzero only for \( m \geq 4 \). 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 situation where a) there is only one active sexual individual is present, so that no mating with concomitant discarding of a gamete, can take place; or b) a conversion from haploid to diploid is impeded because there is only one haploid strand with 4 mutations. It can be checked explicitly that Eqs. (11,12) conserve the total population.

Iterating these equations leads to a steady state distribution that is roughly comparable 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 \( \Gamma \) 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.

### 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 |

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.

**Model B**

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

\[
n_H(m, t + 1) = (1 - \Gamma)n_H(m) + \sum_{\delta = \pm 1} T_{m+\delta, m} n_H(m + \delta, t) - \sigma n_H(m) + \frac{1}{2} \sigma N_A + D_D(t) + \frac{1}{2} \sigma N_S(t) n_H(m)/N_A .
\]

(13)

Here, haploids are converted to diploids and removed at the rate of \( \sigma \), 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 \( \sigma \), 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,

\[
n_D(m, t + 1) = (1 - \frac{1}{2} \Gamma)n_D(m, t) + \frac{1}{4} \sum_{\delta = \pm 1} T_{m+\delta, m} \frac{1}{2} n_H(m + \delta, t) - d_m - \frac{1}{2} \sigma n_D(m, t) + \sigma n_H(m) .
\]

(14)

In this case, the iteration 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. (11,12)) 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
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.

| $\sigma/\Gamma$ | Asexual% | Sexual% | $\sigma/\Gamma$ | Asexual% | Sexual% |
|----------------|----------|---------|----------------|----------|---------|
| 0.01           | 2.9      | 9.3     | 1.00           | 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    |

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

A. The limit of strong driving

After the discussion of the last section, it is natural to expect that the nonlinearities present in the problem should drive it to chaotic behaviour when their amplitude is sufficiently large.

We have tested 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 $\Gamma$, 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 behaviour is found in Model B for large values of $\sigma$. As we increase the value of $\sigma$, the probability of random conversion to sex, beyond about 0.05, a spectacular transition takes place to a strange attractor for the 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 meta-distributions.

The average value of $m$ computed over the asexual and the sexual populations is shown in Fig.8, and displays this striking intermittent behaviour, where the distribution of the two populations becomes much more closely coupled than in the lower $\sigma$ 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 $\sigma$. In Fig.9, we show three dimensional plots for these distributions, compiled over $10^4$ time steps for each value of $\sigma$. In Fig.10, a contour plot of the 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 $\sigma_c \approx 0.05$.

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 $\langle m \rangle_s \approx 0.75$, while for large $\sigma$ 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 for Model B and found that this intermittent behaviour is suppressed. The sexuals simply evolve along the lower branch which 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 ($\Gamma \to 0$ or $\sigma \to 0$)

In the limit of infinitely slow driving, i.e., $\Gamma \to 0$ or $\sigma \to 0$, we observe a transition to a different phase.

For $\Gamma < 1/N$, we find a qualitatively different asexual steady state, where the $m$ distribution has shifted to lower $m$ values (compare with Fig. 2 of [1]) and no longer has the characteristic minimally stable sand-pile like distribution. For $\Gamma = 10^{-4} = (10N)^{-1}$, over a run of $10^6$ steps, we find $n_H(m)/N \approx 0.03, 0.14, 0.44, 0.39$ for $m = 0, \ldots, 3$ respectively, where the peak has moved to $m = 2$ from $m = 3$, or broadened towards the left. This does not seem simply to be due to a slowing down of the dynamics. Rather, once the mutation rate drops below $1/N$, the flow over the $m = 4$ threshold which stabilizes the skewed distribution slows down to a dribble. This gives the $m$ distribution time to get stabilized at $m = 2$.
FIG. 11: The distribution over $m$ for a) asexual b) sexual populations, for different values of $\Gamma$ for Model $A$, without hereditary sex. The steady state distribution changes and the peak on the distribution shifts to a smaller $m$ value as one lowers the $\Gamma$ value below the threshold $1/N = 10^{-3}$.

rather than being pushed to the $m = 3$ limit. The mechanism for the stabilization is provided by the dead bacteria being replenished from among the most prevalent extant ones.

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 ($m = 1$ and $m = 2$ respectively), as shown in Fig.11. Although the total sexual population is relatively small here, we have checked that the fluctuations in the histogram over 10 different realizations stay small.

Iteration of the dynamical equations, on the other hand, reveal no such phase transition 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. Comparison with the simulation results seem to indicate that the simulations get stuck at an intermediate “metastable” state, while the peak is around $m = 2$. The fact that in the simulation one has to wait around until, with a very low probability, a discrete individual is pushed over the $m = 4$ barrier, dies, and is cloned from among the live bacteria, while in the mean-field equations, there is a weak but steady seepage, which prevents this phase transition from taking place.

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 $m$. $\Gamma = 10^{-4}$.

V. DISCUSSION

The mechanism of random conversion to sex, in the presence of a constant rate of mutations, as investigated in this paper as scenario for the maintenance of a macroscopic sexual production, is in fact very closely related to “coevolution of cell senescence and diploid sexual reproduction in unicellular organisms,” studied by Cui et al. [11]. 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 [12], for cell senescence. The constant mutation rate sets the time scale for the survival of any given bitstring, unless it succeeds engaging in sex, with a given probability (our $\sigma$). 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 [4, 12].
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. [13] have pointed out that care must be taken in the investigation of finite populations, amplifying and stabilizing small fluctuations which in the thermodynamic limit would be attenuated to zero. They emphasize the importance of spatial variations which 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 which is described by the continuum approximations.

In a recent article Pekalski [14] has studied a model which 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 which 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.

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. 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.

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