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# PARASITIC INFESTATION AND CHOICE OF REPRODUCTIVE REGIMES

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The Penna model is used to simulate the competition between an asexual parthenogenetic and asexual population inhabiting the same environment represented by a square lattice. With a small probability, a newborn from the sexual population mutates into an asexual one and vice versa. Then, the asexual population rapidly dominates the sexual one, which all but disappears. However, when an infestation by mutating genetically coupled parasites, that mimic trematodes that feed on gonads, is introduced, the outcome may be one in which both populations coevolve or one in which one of the populations overcomes the other, depending on the density of parasites on the lattice.

*Keywords*: Evolution; simulation; order-disorder.

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## 1. Introduction

The prevalence of sexual reproduction among the great majority of species in nature, in spite of the inherent cost of having to produce males to assure reproduction, is still one of the great puzzles of biology.[1] Many theories have been put forth to try to explain it. A common feature of many of them is the idea that the advantage of sex relies on its ability to create greater genetic diversity, since the pool of alleles from which the newborn genome is extracted is enlarged by the male's genome and is different for each mating pair. A situation in which greater diversity would clearly enhance the survival probability of a population is if it falls under the action

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of genetically matching parasites, which would then become responsible for creating a rapidly changing environment. In this unstable ecology, only varieties that can mutate their genomic pool at least as fast as the adaptation of the parasites proceeds can survive.

The first well succeeded attempt to test this particular theory using the Penna model[2] was performed in Ref. 3. The biological motivation came from the observations of competing varieties of a freshwater snail, *Potamopyrgus antipodarum*, reported in Refs. 4–6. There it was shown that there is a strong correlation between the prevalence of one reproductive regime and the concentration in its habitat of the trematode *Microphallus*, a parasite that renders the snail sterile by eating its gonads. Namely, the asexual variety is predominant where the parasite appears in small concentrations, whereas higher concentrations of the trematode forces the species to prefer a sexual regime.

Here we present simulational results obtained through an equivalent approach to that of Ref. 3, but now distributing the "snails" and the parasites on a spatial lattice. Our main purpose has been to introduce more realistic dynamics, first by analyzing the success or failure of a given reproductive regime according to the parasites density, instead of considering a non-localized source of trematodes. Second, we can now avoid the usage of the Verhulst logistic factor, present in the original Penna model, which has already been criticized in the literature.[7]

In Sec. 2 we explain our model, Sec. 3 contains the results and Sec. 4, the conclusions.

## 2. The Model

### 2.1. *The Penna model for diploid populations*

Each individual is represented by a "chronological genome" consisting of *two bit-strings of* 32 *bits each*, that are read in parallel. Each position of the bit-strings is associated to a period of the individual's life, which cannot exceed 32 periods ("years"). Each step of the simulation corresponds to reading one new position of all individuals' genomes. Genetic defects are represented by bits 1, but may be active or not. If an individual has two bits 1 at the $i$th position of both bit-strings, it will start to suffer the effects of a genetic disease at its $i$th year of life. If the two bits at the $i$th position are equal, this position is said to be homozygous. If the individual is heterozygous in that position, meaning that the two bits have opposing configurations, it will become sick only if that locus is one for which the harmful allele is dominant. These positions are chosen randomly at the beginning of the simulation and remain fixed throughout the run. When the current number of accumulated diseases reaches a threshold $T = 3$, the individual dies.

In order to test the efficiency of each reproductive regime against parasites, two different populations are simulated to coevolve sharing the same habitat. Both are diploids, but one is asexual (parthenogenetic) while the other is sexual. In the first case *all individuals are females*; in the second one, some of them are males and

some are females. In both cases there is a minimum reproduction age $R = 8$; from this age on, and until its death, each female generates $b = 1$ offspring at every time step. The sexual or asexual nature of the *reproductive strategy of a newborn* is the same as that of its mother, but can mutate with a small probability $p_R$.

The difference between the two reproductive regimes lies on the way an offspring inherits genetic material from its parent(s). The way to mimic the recombination process, in both cases,[8] is to select a random position out of the 32 loci of the parents' genome and cut the two strings at this position. Two new strings are generated by crossing the resulting four pieces: the left side coming from one of the strings is attached to the right side coming from the other. Of the two new strings, one is chosen randomly to be the parents' gamete, and constitutes the genetic material to be inherited by the newborn. For asexual reproduction (meiotic parthenogenesis), this single string is cloned: since the two bit strings are identical before mutations, all loci are homozygous and the new genome is totally homozygote. For sexual species, a male individual is selected randomly and his genetic material also undergoes crossing and recombination. The two strains, each one coming from one of the parents, form the newborn genome. In both reproductive regimes, one harmful mutation is introduced in a random position of each newborn string. In the sexual case the newborn gender is chosen randomly, with equal probabilities.

### 2.2. *Spatial distribution of individuals and parasites*

The population lives on sites of a square lattice; at every time step, each individual moves from its present position $(i, j)$ to a site on its Moore neighborhood (nearest and next-nearest neighbors), provided the occupation of this site is not larger than some limit (taken to be 2048 in our simulations) and chosen differently if the individual is old enough to mate or not. If it is too young — age smaller than the minimum reproduction age — the site chosen is the one that presents the smallest occupation, if this occupation is also smaller than or equal to that of its current site. If it is in the reproductive stage, it chooses the neighboring site with the largest number of potential mates. We start the simulations by randomly distributing one *sexual* individual per site on a (diluted) square lattice. Each female old enough to reproduce (age $\geq R$) selects randomly, in the site she occupies and in its Moore neighborhood, a male able to breed and generates $b$ offspring. Then, she chooses among its eight neighboring sites (maternal care), the one with the smaller occupation to place each baby. If there is no site with space available, *the baby dies*. This is the ingredient that replaces the random killing Verhulst factor, present in the traditional versions of the Penna model to prevent unlimited population growth. It was first introduced in the Penna model in Ref. 9 and subsequently used in Refs. 10 and 11 where the issue of speciation was addressed.

We allow the initial population to evolve for some period of time and then we randomly distribute on the lattice a fixed number $N$ of parasites, which have a dynamically changing memory. Every time step, each of them moves to a randomly

chosen site of its Moore neighborhood. Whenever a parasite contacts a female, it *memorizes* that genomic pattern. If the parasite contacts the same pattern *twice in a row*, it *learns* that pattern and becomes active against it. If any female carrying a genome already *learned* by the parasite is contacted, it is turned sterile. This strategy means that the parasite acts against a genomic pattern only if this same pattern is contacted three or more times in a row.

## 3. Results

The initial population consists of 200 sexual individuals randomly distributed on a $100 \times 100$ square lattice. This population is left to evolve for 20 000 time steps, without mutations in its reproductive regime; then the mutation probability $p_R = 0.01$ is switched on. The evolution proceeds until, at time step 40 000, the parasitic infestation starts.

Figure 1 shows the population evolution before the parasites strike. When the mutations in the reproductive regime start, the sexual character almost disappears and the asexual one dominates, due to the advantage of the asexual population over the sexual one in producing twice the number of offspring. However, when the trematodes appear, there is a competition between two factors: the genomic diversity (which is higher for the sexual population) and the effective reproductive rate. Figure 2 shows that, depending on the parasite density, one or the other may win this competition. From Fig. 3 we can see that for some intermediate

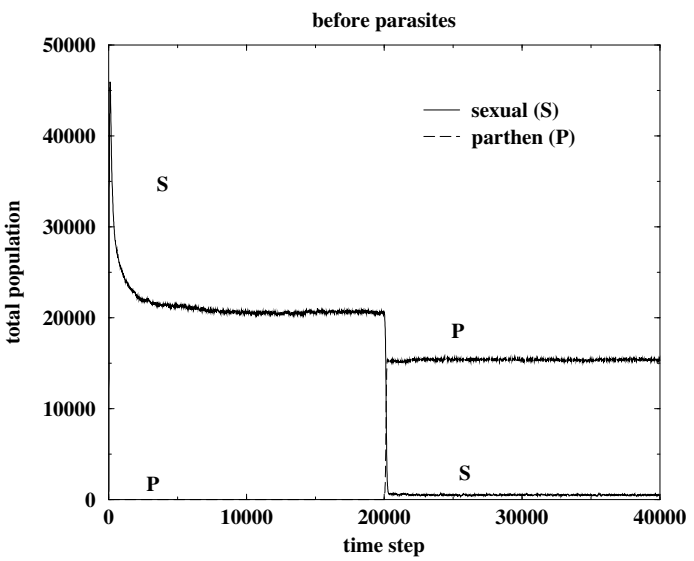
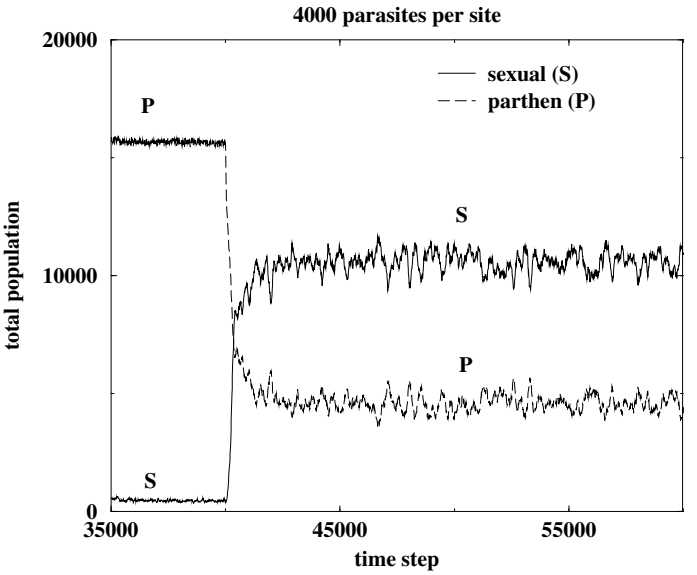
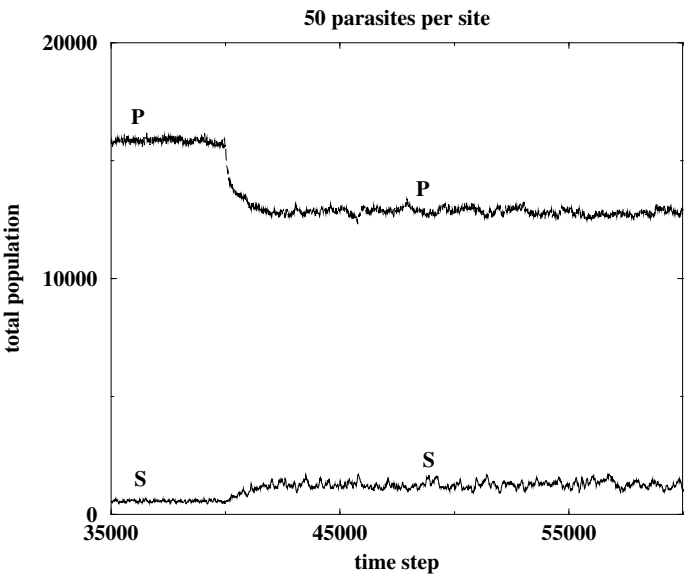



Fig. 1. Population evolution before the parasitic infestation. The initial population consists of only sexual individuals, half males and half females. When the mutations in the reproductive regime are switched on (time step 20 000), the asexual parthenogenetic character dominates and the sexual population almost disappears.




(a)




(b)

Fig. 2. Population evolution just before and after the parasitic infestation. For a large density of parasites (upper plot) the small fraction of sexual individuals increases and becomes greater than the parthenogenetic one. For a small density of trematodes (bottom plot) the asexual reproductive regime remains more advantageous for the population.

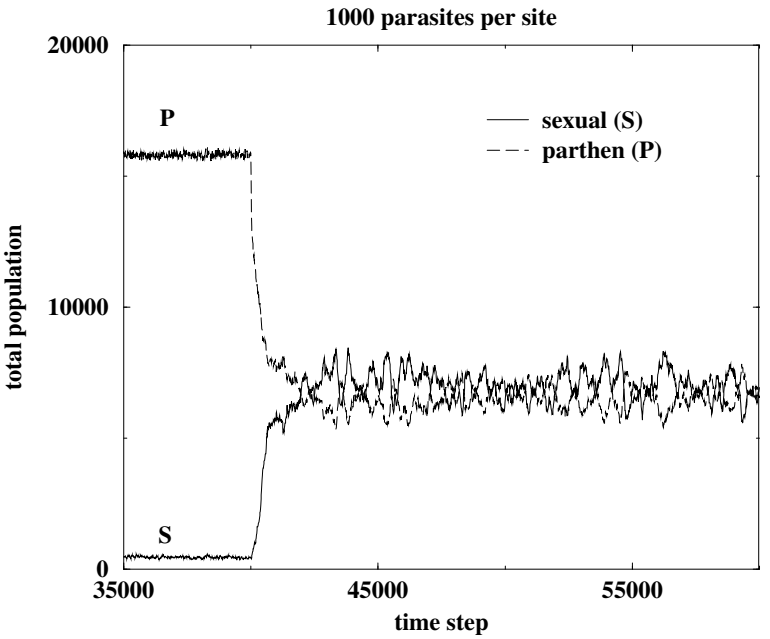



Fig. 3. Population evolution before and after the parasitic infestation for an intermediate density of parasites. In this case both reproductive strategies survive in roughly equal proportions.

densities both populations coevolve in roughly equal proportions, a result that was not observed in the absence of a spatial lattice.[3] In that case, the frequency of female contacts with parasites, i.e. the number of exposures per cycle $E$, is a parameter that plays the role of the parasites density. It is the same for all females and drives a sharp transition between reproductive regimes. By allowing spatial variations of parasite density, this feature is lost, showing that it was in fact an artifact of a mean-field model.

## 4. Conclusions

We simulate two populations, one asexual and the other sexual, sharing the same habitat represented by a square lattice. A newborn of either population can mutate and become a part of the other with some small probability $p_R$. Individuals move on this lattice and a newborn survives only if there is an empty site for it in the neighborhood of the mother's site. The initial population consists of only sexual individuals, half males and half females. When the mutation probability $p_R$ is switched on, the sexual population almost disappears due to the advantage of the asexual population over the sexual one in producing twice the number of offspring. However, when the environment is infested by a high concentration of rapidly mutating parasites, the sexual population increases and surpasses the asexual one due to its higher genetic diversity. In fact, the effectiveness of this simulated parasitic infestation is an indirect measure of the lack of genetic variability

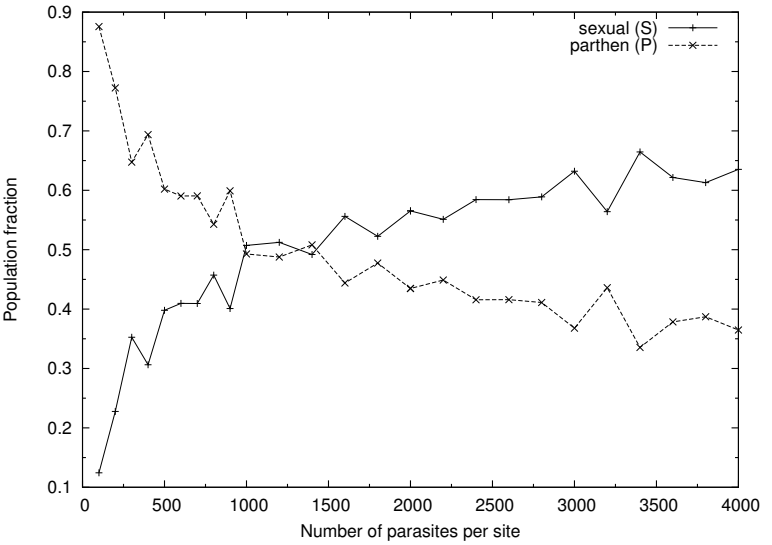



Fig. 4. Average sexual and parthenogenetic polulations as a function of the parasite densities. Both populations survive and coevolve, even for high parasites concentrations, contrary to what was obtained in Ref. 3, where a phase transition was observed.

within the female population of each variety, since whenever a parasite contacts the same female genomic pattern for the third consecutive time, it renders the female sterile.

For small parasites concentrations, we obtain that the majority of the individuals keeps the asexual reproductive strategy. For intermediate concentrations of trematodes, the two populations coevolve with roughly equal sizes, a result that previous simulations, also using the Penna model but without a spatial distribution, failed to obtain.[3] In that case, a sharp transition from an asexual-dominated regime to a sexual-dominated one was observed, as a function of the intensity — or density — of the parasitic infestation, measured by the number of parasites with which each female got into contact at each time step. The failure to obtain the same feature here, as shown in Fig. 4, may be attributed to the mean-field character that results when the model, such as the one used in the work cited, ignores spatial fluctuations. Indeed, it is well known that mean-field approaches may show spurious phase transitions, for instance when applied to one-dimensional systems.

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