Solutions to exercise sheet 3

Sex, Ageing and Foraging Theory

Exercise 1: Two-fold cost of sex

a. Assuming $f^{\mathrm{A}} = f^{\mathrm{S}} = f$, leads to

$$n^{\mathrm{A}}_{t+1} = \frac{1}{1+\gamma n^T_t} f n^{\mathrm{A}}_t \tag{1}$$

$$n^{\mathrm{S}}_{t+1} = (1-r)\frac{1}{1+\gamma n^{\mathrm{T}}_t} f n^{\mathrm{S}}_t \ . \tag{2}$$

Dividing Eq. 1 by Eq. 2, we have

$$\frac{n^{\mathrm{A}}_{t+1}}{n^{\mathrm{S}}_{t+1}} = \Big(\frac{1}{1-r}\Big)\frac{n^{\mathrm{A}}_t}{n^{\mathrm{S}}_t} \ , \tag{3}$$

and since $0 < r < 1$, the factor $1/(1-r) > 1$ is always greater than 1. Hence, the ratio of asexuals to sexuals, $n^{\mathrm{A}}_t/n^{\mathrm{S}}_t$, increases with time. The number of asexuals must therefore increase with time relative to sexuals. In short, when sexual and asexual females have the same fecunfity, asexuals always outcompete sexuals. This is the "demographic cost of sexuality".

b. Assuming $f^{\mathrm{A}} = f$ and $f^{\mathrm{S}} = 3f$, we get

$$\frac{n^{\mathrm{A}}_{t+1}}{n^{\mathrm{S}}_{t+1}} = \left(\frac{1}{3(1-r)}\right)\frac{n^{\mathrm{A}}_t}{n^{\mathrm{S}}_t} \ . \tag{4}$$

Two outcomes are thus possibles depending on the sex ratio at birth. (1) When the sex ratio is high $r > 2/3$ (i.e. sexual females produce many sons), the factor $1/[3(1-r)] > 1$ is greater than 1 so as in the previous scenario, asexuals outcompete sexuals. (2) By contrast when the sex ratio is sufficiently low $r < 2/3$, the factor $1/[3(1-r)] < 1$ is less than 1 so asexuals decrease relative to sexuals.

c. With $f^{\mathrm{S}}(r) = 3f\sqrt{r/(1-r)}$ and $f^{\mathrm{A}} = f$, we obtain

$$\frac{n^{\mathrm{A}}_{t+1}}{n^{\mathrm{S}}_{t+1}} = \left(\frac{1}{3\sqrt{r(1-r)}}\right)\frac{n^{\mathrm{A}}_t}{n^{\mathrm{S}}_t} \ . \tag{5}$$

This again suggests two possible outcomes depending on the sex ratio. (1) If the sex ratio is not too biased, i.e. if $(3-\sqrt{5})/6 < r < (3+\sqrt{5})/6$, or in other words, if sex ratio is not too far from $1/2$, sexuals outcompete asexuals (see calculation in Wolfram Alpha). (2) Otherwise, when the sex ratio is biased either towards females or males (i.e. if $r < (3-\sqrt{5})/6$ or $r > (3+\sqrt{5})/6$), then asexuals outcompete sexuals. This is because when sexual females produce many sons, asexuals can outcompete them by producing more daughters (as in question 1b). When sexual females produce few sons, it may be difficult for males to fertilise

sexual females lowering their average fecundity thus favouring asexual females.

Exercise 2: Consequences of asexuality

a. The function on line 22 models the addition of mutations across the L-locus genome. Mutations occur with probability u per locus. To model this, we sample from a Bernoulli distribution with probability of success u for each locus of each individual (i.e., sampling 0 or 1 with probability $1-u$ and u, respectively, and we do this L times). For each individual and locus $i = 1, \ldots, L$, we add the aforementioned sampled value for mutation ($M_i = 0$ or 1) to the allelic value before mutation ($x_i = 0$ or 1). If the locus before mutation was carrying the wild-type allele ($x_i = 0$) and no mutation occurred ($M_i = 0$), then the new allele is still wild type ($x_i + M_i = 0$). If a mutation occurred ($M_i = 1$), then the new allele is deleterious ($x_i + M_i = 1$). If the locus before mutation was carrying the deleterious allele ($x_i = 1$), then it remains deleterious whether or not a mutation has occurred (i.e. $1 + M_i = 1$ whether $M_i = 1$ or 0). To ensure this, we set values greater than 1 equal to 1, i.e., $x_i = 1$ if $x_i > 1$. For purposes of computational efficiency, we perform the operation described in this paragraph at all L loci at the same time with vectorized operations (try running each line in the console of RStudio if you want to see this).

   The function in line 28 corresponds to the recombination between two given genomes, $\{x_i\}_i$ and $\{x_j\}_j$. First, both genomes are concatenated into one single vector (joint vector). Then, a new vector is returned by the function where each position i is either the value at i-th position or at the $(i + L)$-th position of this concatenated vector with equal probabilities. In this concatenated vector, i-th position carries the information of the first parent (x_i) and $(i+L)$-th position carries the information of the second parent (x_j) at the same locus.

b. To understand the evolution of an asexual population, we can keep track of three quantities at each generation (Fig. 1): (1) the population size; (2) the minimum number of mutations found in an individual's genome; and (3) the mean number of mutations per genome in the population. The final plot in Fig. 1 (bottom right) shows the distribution of the number of deleterious mutations per genome in the population at generation 1000.

   Fig. 1 shows that asexuals rapidly accumulate deleterious mutations (as the minimum and mean numbers of mutations per genome increase). This in turn causes a decrease in population size. After ~ 600 generations, the population size stabilizes with ~ 300 asexual individuals. We observe that by generation 1000, over two thirds of individuals carry 50 deleterious mutations (out of $L_{\text{loci}} = 50$ loci). Running the simulation for longer we would see that eventually, all individuals have deleterious mutations at all their 50 loci.

   As an illustration of Muller's ratchet, we observe in Fig. 1 (top right) that the minimum number of deleterious mutations in a single genome just keeps increasing over time (i.e. it never goes down). This is due to the inability of asexual genomes to eliminate deleterious mutations with genetic recombination.

c. Fig. 2 shows the evolution of a sexual population. Although deleterious mutations still increase (due to genetic drift and the fact that mutations only go from wild-type to deleterious), the minimum number of deleterious mutation per genome does not only increase, i.e. it can sometime decrease from one generation to the next. This reflects that mutation can be purged from one generation to the next by sexual recombination. As a result the accumulation of deleterious mutations takes much longer in a sexual than in an asexual population (compare both histograms of Fig.1 and 2 at time $t = 1000$).

d. Fig. 3 shows evolution in asexuals with stronger selection, $s = 0.02$. We see that the population accumulates deleterious mutations and as a result rapidly crashes and even goes extinct. By contrast, a sexual population is able to purge these mutations and thus avoid extinction (see Fig. 4)

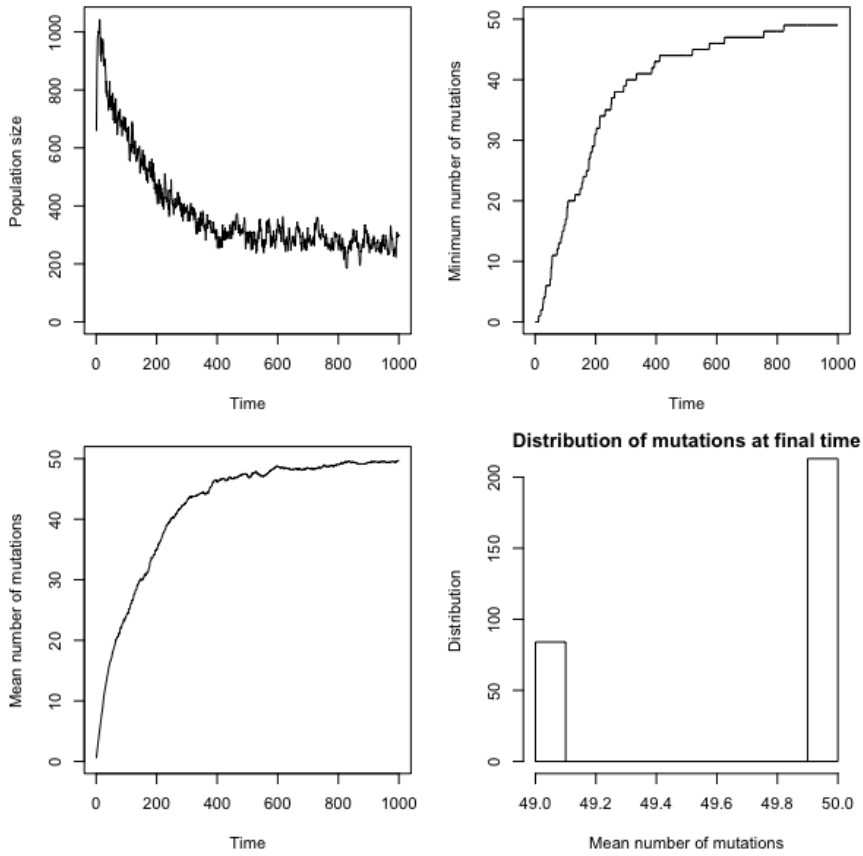

Figure 1: Evolution in asexuals with $s = 0.01$. Time is measured in units of life-cycle.

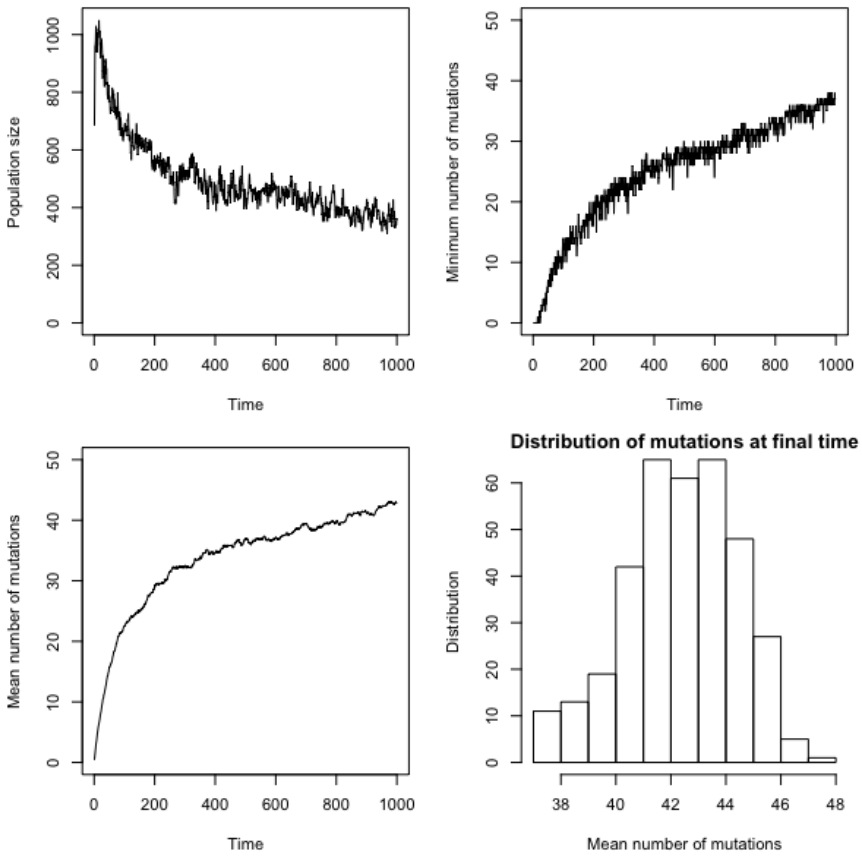

Figure 2: Evolution in sexuals with $s = 0.01$.

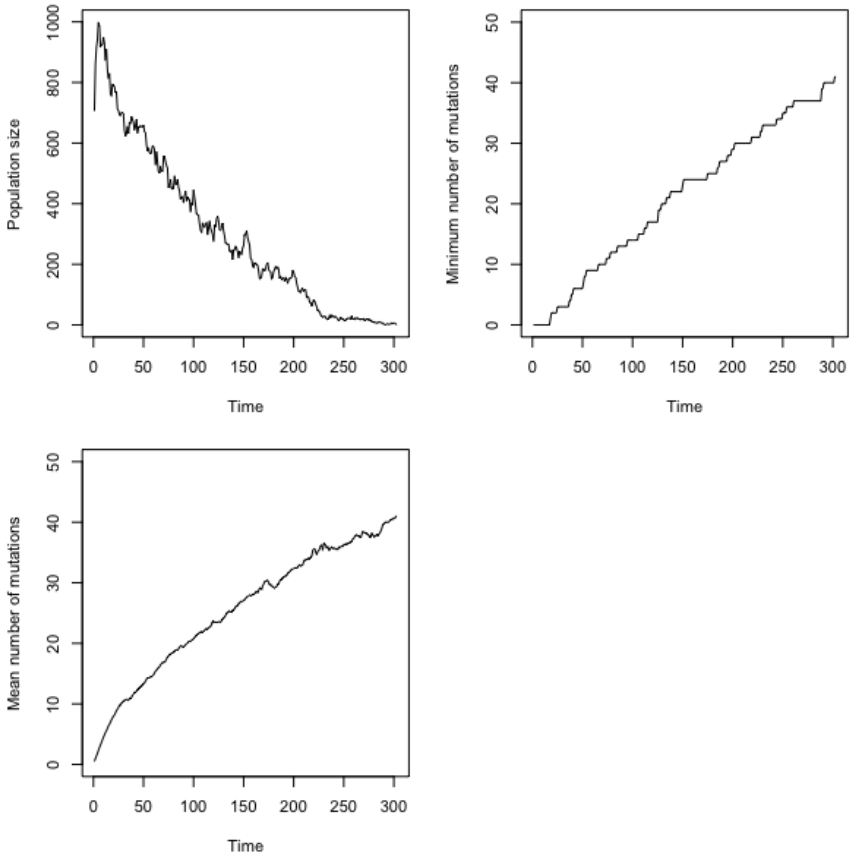

Figure 3: Evolution in asexuals under strong selection $s = 0.02$.

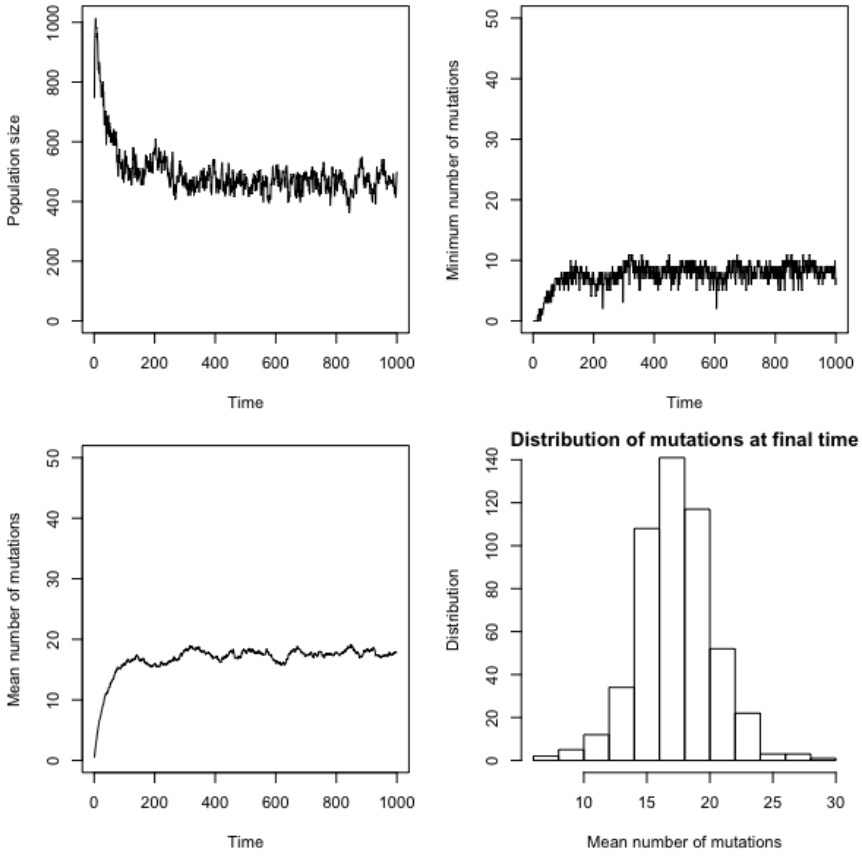

Figure 4: Evolution in sexuals under strong selection $s = 0.02$.

Exercise 3: Maintenance of sexuality

a. In line 133, we set the probability of sexual reproduction of a random individual in the population to 0. This operation happens in the 200th generation.

b. Figure 5 shows the survival probability of an individual as a function of its number of deleterious mutations under six different strengths of epistasis, ϵ. Four different qualitative cases can be observed: (i) When $\epsilon = 0$, the survival probability does not depend on the number of deleterious mutations, i.e., mutations are neutral; (ii) When $0 < \epsilon < 1$, the effect of each new mutation is reduced as the number of deleterious mutations increases. This is a case of negative epistasis, i.e. the combined effect of deleterious mutations is weaker than the sum of their individual effects. (iii) When $\epsilon = 1$, each mutation decreases the survival probability equally by a factor of $1 - s$. Finally, (iv) when $\epsilon > 1$, the effect of each extra mutation increases as the number of mutations increases. This is a case of positive epistasis. When $\epsilon >> 1$, there is a sharp transition in survival probability, which goes from 1 to 0 over a few mutations. This sharp transition occurs when individuals carry about K deleterious mutations, as seen in Fig. 6.

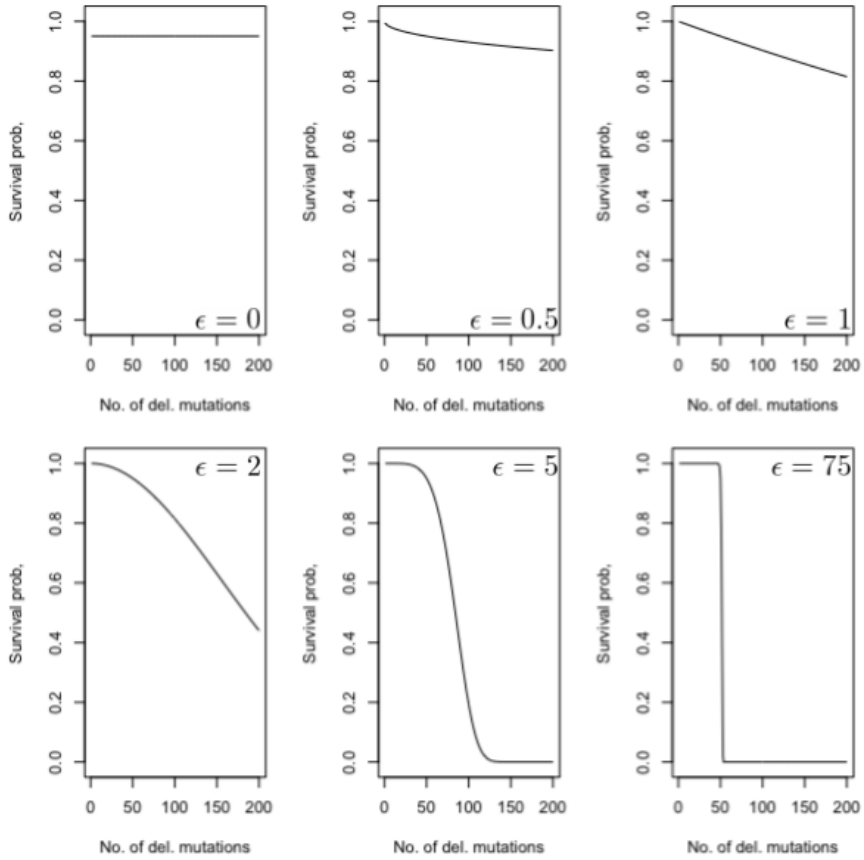

Figure 5: Survival probability as a function of the number of deleterious mutations for six different values of epistasis, ϵ. All plots have $K = 50$ and $s = 0.05$.

The accumulation of deleterious mutations is more detrimental in the case of strong epistasis. Since asexual are more prone to accumulate deleterious mutations, strong epistasis should disfavor asexuals and thus help maintain sexual reproduction.

c. In the absence of epistasis ($\epsilon = 1$), an asexual mutant introduced in the population at the 200th generation

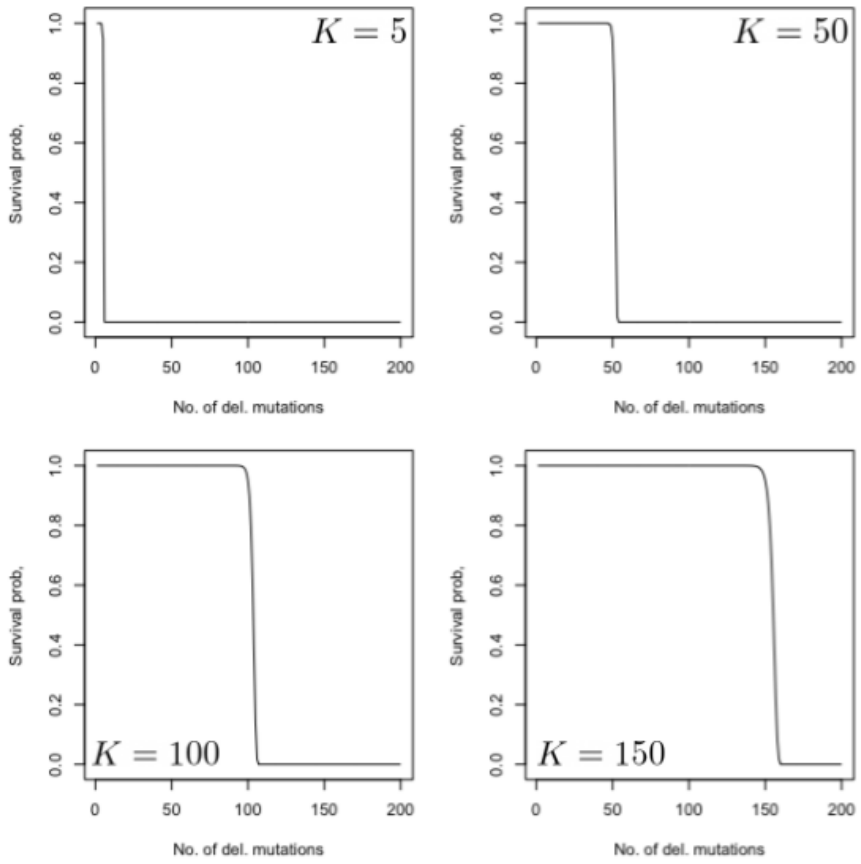

Figure 6: Survival probability as a function of the number of deleterious mutations for different values of K. All plots have $\epsilon = 75$ and $s = 0.05$.

is able to invade and its lineage quickly replaces sexuals (Fig. 7). Under strong epistasis ($\epsilon = 75$), asexuals are able to initially increase in frequency owing to their two-fold demographic advantage (Fig. 8). Due to epistasis however, asexuals quickly suffer a steep fitness reduction due to accumulating more mutations than sexuals. As a result, asexuals eventually go extinct.

d. When there are fewer loci, asexuals are able to invade and replace the sexual population even when epistasis is strong. This is because when there are fewer loci, the total rate of deleterious mutation per genome is lower so that deleterious mutations accumulate more slowly. In other words, larger genomes favor the sexual reproduction over asexuality.

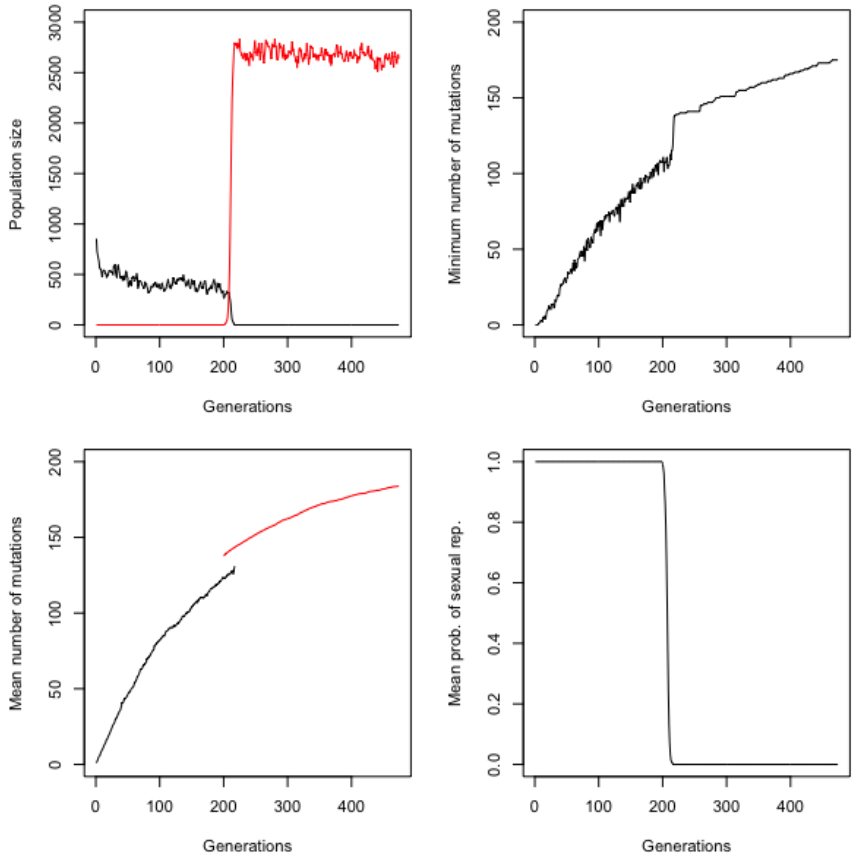

Figure 7: Invasion and substitution of asexuals in a sexual population in the absence of epistasis, $\epsilon = 1$. In each graph, the black curves represent quantities measured in sexuals, while the red curves represent the same quantities measured in asexuals.

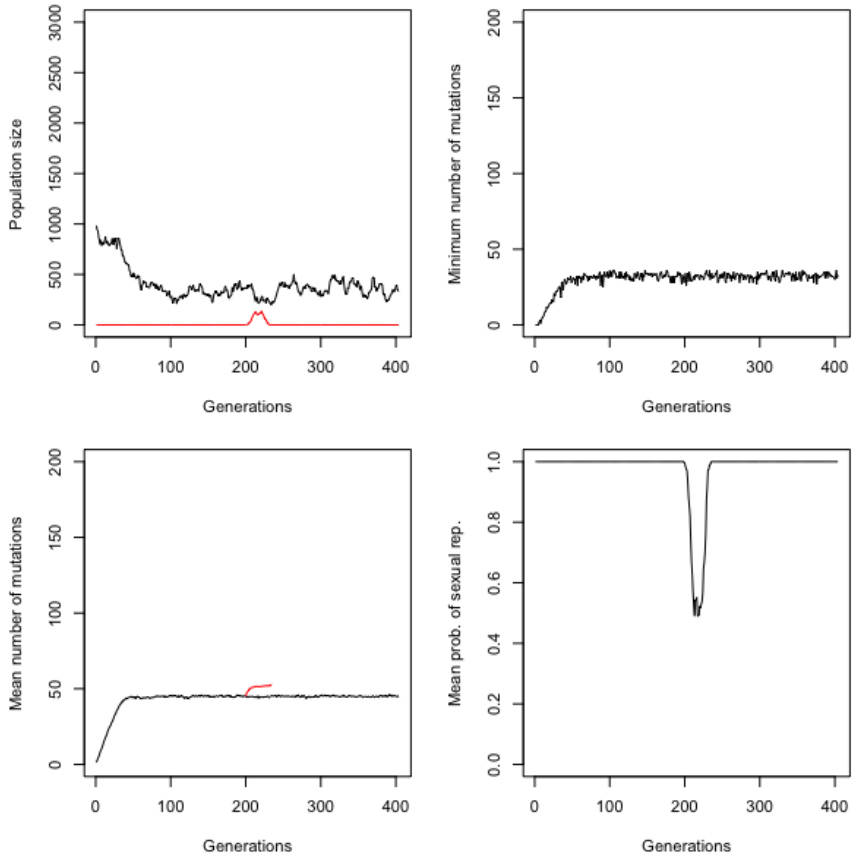

Figure 8: Asexuals fail to invade a sexual population under strong epistasis, $\epsilon = 75$.