Solutions to exercise sheet 5

Sex, Ageing and Foraging Theory

1 Maintenance of sexuality

a. In line 133, we set the probability of sexual reproduction of a random individual in the population to 0. This command is only run in the 200th generation. Biologically, we are mutating an offspring from a sexual behavior to an asexual behavior.

b. Fig. 1 shows the profiles of survival probability of an individual as a function of its number of deleterious mutations for six different strengths of epistatic interactions, ϵ.

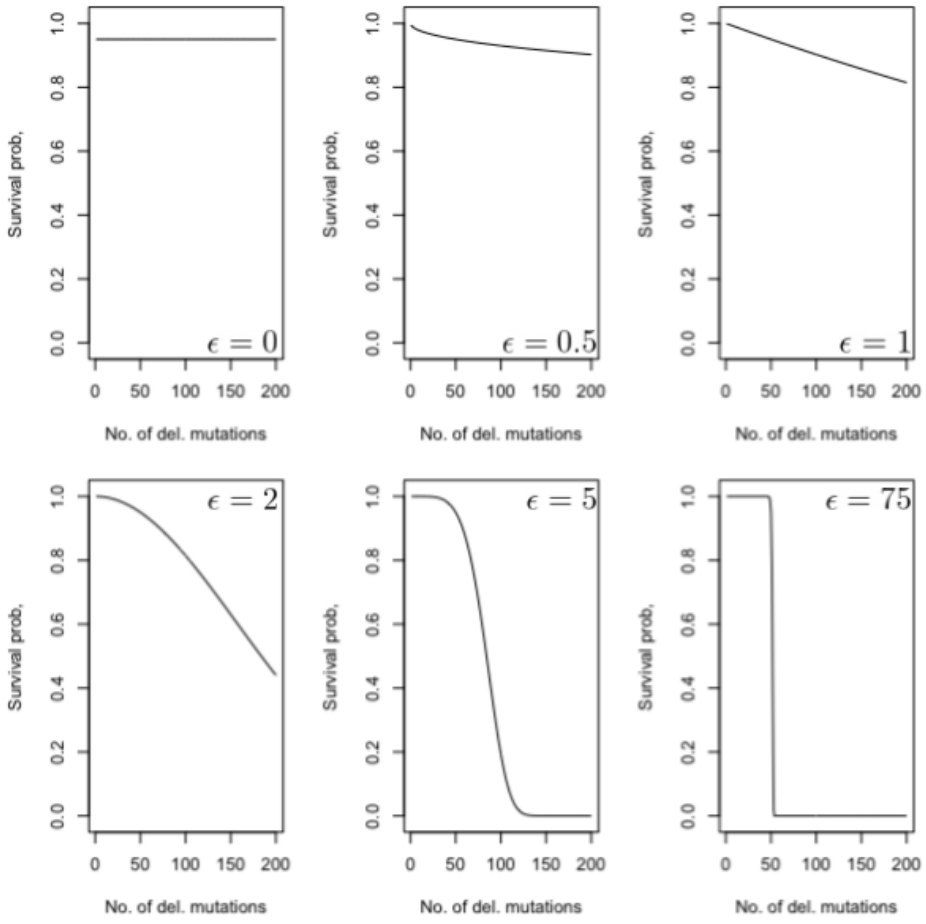

Figure 1: Survival probability as a function of the number of deleterious mutations for six different values of epistatic effects, ϵ. All plots have $K = 50$ and $s = 0.05$.

We see four different qualitative cases in those profiles: (i) When $\epsilon = 0$, the survival probability does not depend on the number of deleterious mutations, i.e., mutations become neutral instead of deleterious. (ii) When $0 < \epsilon < 1$, a few deleterious mutations quickly decrease the survival probability. As the number of deleterious mutations increase, the effect of each new mutation is reduced. This is a case of negative epistasis, i.e. the combined effect of deleterious mutations is smaller than what one would expect from their individual effects. (iii) When $\epsilon = 1$, each mutation equally decreases the survival probability by a factor of $1 - s$. This is a case of multiplicative effects and no epistasis, i.e. the combined effect of deleterious mutations is equal to what one would expect from their individual effects. Finally, (iv) when $\epsilon > 1$, the survival probability is weakly affected by a few mutations, but as the number of mutations increase, the effect of each extra mutation is accentuated. This is a case of positive epistasis, i.e. the combined effect of deleterious mutations is greater than what one would expect from their individual effects. A particular case of positive epistasis is when $\epsilon >> 1$ leading to a sharp transition from a survival probability close to 1 to a survival probability close to 0. This sharp transition is centered at the parameter K, as seen in Fig. 2 .

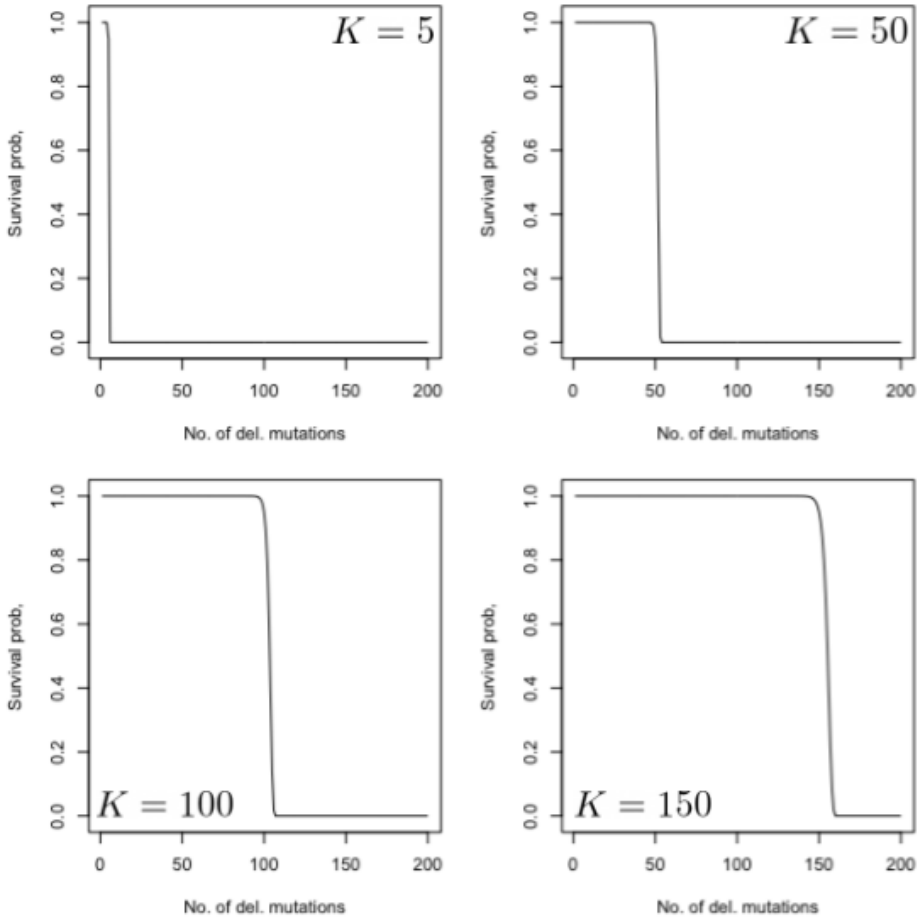

Figure 2: Survival probability as a function of the number of deleterious mutations for different values of K. All plots have $\epsilon = 75$ and $s = 0.05$.

The accumulation of deleterious mutations in an asexual population is more detrimental in the case of strong epistasis. In such case, the dynamics begins with the Muller's Ratchet quickly "spinning", with very little selection counteracting (see the plateau in Fig.2 for $k_i < K$). Asexuals therefore quickly accumulate mutations, up to the point an additional mutation is lethal. When the Muller's Ratchet passes through the

sharp transition found in highly epistatic interactions, the survival probability is abruptly reduced and asexual individuals are not able to purge away the deleterious mutations. At this moment, the asexual population rapidly goes extinct.

c. Running the simulations with no epistasis ($\epsilon = 1$), an asexual mutant introduced in the population in the 200th generation is able to quickly invade and substitute the sexual population. In very few generations, the population is completely asexual. At this moment, the population size is regulated by the density-dependent competition and the minimum number of deleterious mutations in the population becomes are monotonically increasing function of time. An example of this dynamics is given in Fig. 3.

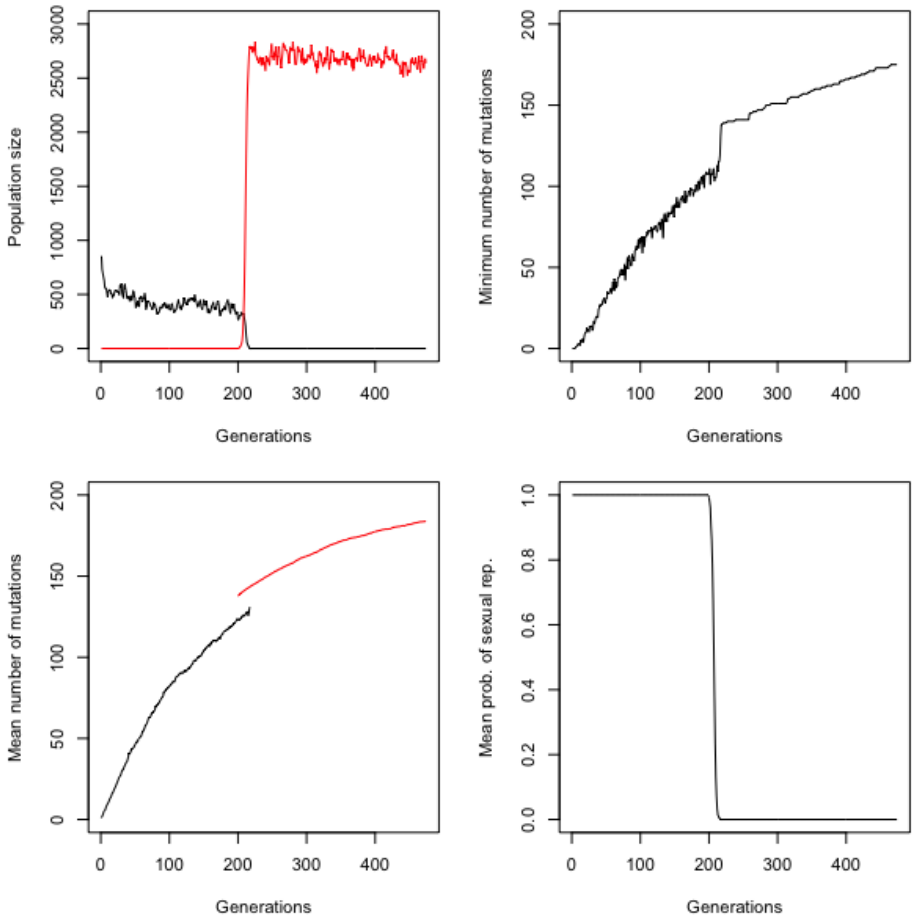

Figure 3: Invasion ans substitution of asexuals in a formerly sexual population. Survival probability with no epistasis, $\epsilon = 1$. At each graph, the black curves represent quantities measured in sexual individuals, while the red curves represent the same quantities measured in asexual individuals, introduced in the 200th generation.

When there is high epistasis ($\epsilon = 75$), asexuals are also able to invade the population because they have the short-scale two-fold advantage over sexuals. However, in the long-run, as asexuals increase in population size, they quickly accumulate deleterious mutations. Due to highly epistatic interactions, this accumulation of deleterious mutations leads to a rapid decay of the survival probability in asexual individuals. Hence, despite invading, they go extinct rather than substituting the sexual population as in the case of no epistasis. One example of this dynamics is found in Fig. 4.

d. By reducing the number of loci, asexuals are able to invade and substitute the sexual population even under

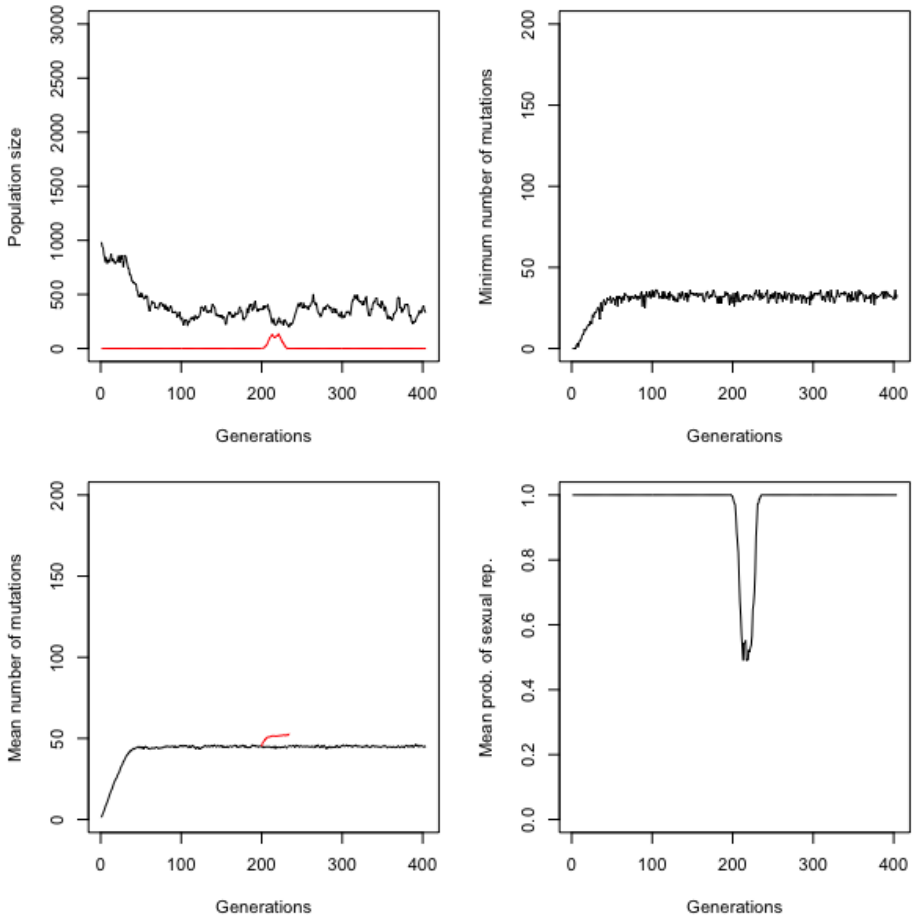

Figure 4: Asexuals fail to invade a sexual population when the survival probability has epistasic effects, $\epsilon = 75$. At each graph, the black curves represent quantities measured in sexual individuals, while the red curves represent the same quantities measured in asexual individuals, introduced in the 200th generation.

high epistatic effects. A large genome increases the efficiency of recombination in sexuals because it reduces the probability that both parents carry simultaneously a deleterious mutation at the same locus. At the same time, it increases the number of deleterious mutation per individual in both populations (given by the product of the rate of mutation per locus and the number of loci, uL). Hence, large genomes favor the sexual individuals over the asexual individuals.