Exercise sheet 5: A model for the maintenance of sexual reproduction

Sex, Ageing and Foraging Theory

In exercise sheet 4, you investigated evolution at L loci under purifying selection in: a population composed of asexual individuals (specifically where the probability σ of reproducing sexually was fixed to zero for all individuals, $\sigma = 0$); and a populations of sexuals (where $\sigma = 1$ for all individuals). Here, we extend this model to investigate when asexuality can and cannot invade a population of sexuals. To do so, we allow for the probability σ of reproducing sexually to also evolve, i.e. each individual i now has a probability σ_i of reproducing sexually. Each haploid individual is thus characterised by $L + 1$ genetic loci: (1) one locus coding for the probability σ of reproducing sexually where two alleles segregate, one for sexual ($\sigma = 1$) and one for asexual reproduction ($\sigma = 0$); and (2) L loci under purifying selection, at each of which a wild type and deleterious mutation can segregate (as in Ex sheet 4).

The life cycle is composed of five steps. (1) First, each adult female i either reproduces sexually with probability σ_i or asexually with probability $1 - \sigma_i$ according to her allele at the locus for sexual reproduction. If a female reproduces sexually, it mates at random with a male in the population. (2) Each female produces a Poisson-distributed number of offspring with mean f_0. Before mutation, an offspring produced asexually is an exact copy of its parent (e.g. if the parent has the allele for $\sigma = 0$ and 3 deleterious alleles at the L loci under purifying selection, its offspring also has the allele for $\sigma = 0$ and 3 deleterious alleles), while an offspring produced via sexual reproduction is a recombined version of its mother and father, assuming each locus segregates independently (i.e., at each locus, the inherited allele is a copy of the mother with probability $1/2$ or of the father with probability $1/2$). (3) Mutation from the neutral allele to the deleterious allele occurs with probability u at each of the L loci under purifying selection (we assume there is no constant mutation at the locus for sexual reproduction). (4) An offspring survives with a probability that depends on density-dependent competition and on the number of deleterious mutations it carries. Specifically, we assume that the probability ω_i that an offspring i with k_i deleterious mutations survives to adulthood is,

$$\omega_i = \frac{(1-s)^{\left(k_i/K\right)^{\epsilon}}}{1+\gamma n_t}, \tag{1}$$

when there are n_t adults in the population. There are two extra parameters compared to eq. (8) of Ex sheet 4 to capture epistasis (see item (b) below). When $\epsilon = 1$ and $K = 1$, we recover eq. (8) of Exercise Sheet 4. Finally, (5) the surviving offspring of sexual females becomes an adult male with probability r, or an adult sexual female with probability $1 - r$, so that r is the sex ratio at birth (all offspring of asexual females are also asexual females).

An individual-based simulation program for the life-cycle above has been made available on the course website (lab-mullon.github.io/SAF). We start with 1000 sexual individuals that have no deleterious mutations (i.e. each individual carries the allele for $\sigma = 1$ and the wild-type allele at the L loci under purifying selection). We let the simulation run for 100 generations, which is enough time for the population to reach a distribution of deleterious mutations that no longer changes very much over time. In the 100-th generation, a random sexual female becomes

asexual, i.e. we change the allele σ_i from 1 to 0 for some random i. We let the simulation run for another 100 generations to observe if the asexual lineage invades the sexual resident population.

a. Familiarise yourself with this program. Discuss the biological interpretation of line 133.

b. Plot the survival probability ω_i as a function of the number k_i of deleterious mutations for different strengths of epistasis (e.g. with $\epsilon = 0$, 0.5, 1, 10, and 75) for a fixed value of $K = 50$. Do these plots again but for different K (e.g. with $K = 10$, 50, 100) with a fixed value of $\epsilon = 75$. Interpret these plots and use them to conjecture on the implications of epistasis for the maintenance of sexual reproduction.

c. Run the simulation under no epistasis, $\epsilon = 1$. Do this a few times recording relevant information about the population each replicate (remember it is a stochastic simulation so it is good to a have a few replicates). Run the simulations also with high epistatic effects, $\epsilon = 75$. Do the results differ when $\epsilon = 1$ and $\epsilon = 75$? Does this fit with your expectations formulated in part (b)?

d. Reduce the number L of loci under purifying selection from 200 to 100. Re-run simulations under strong epistasis, $\epsilon = 75$. What can you conclude from these simulations about the effects of the number of loci on the maintenance of sexual reproduction? Why?