ANSWERS

1. Female sterility (androdioecy)

The per individual contributions to fitness of the two phenotypes through female gametes are as follows:

Cosexes (phenotype 1), through female gametes: S(1-δ)+1-S

w_1^f is proportional to(1 - S)+ S(1-δ) = 1 – $S\delta$

Males (phenotype 2) produce NO female gametes: $w_2^f = 0$

Males have male fertility 1 + K relative to that of the cosexuals.

For phenotype 1, gametes contributed by selfing amount to another quantity S(1-δ)

When the population is cosexual (males are rare), seeds are available for outcrossing on other cosexuals, in a proportion 1 – S of the ovules produced per individual, and the size of the pollen pool is 1. The ratio of seeds fertilised to pollen is thus 1 – S.

A male mutant enjoys the same ratio of seeds fertilised to pollen.

We thus have the condition for invasion:

$1 - S + 2S(1-\delta) + 1 - S > (1 - S)(1 + K)$

or $1+K > 2(1-S\,\delta)/(1-S)$

QUESTIONS

(i) Females have a higher net fitness than cosexuals if $k > 1 - 2S\delta$. Explain the differences in the result for invasion by males.

- Compared with an outcrossing initial cosexual form, males can invade only if their fertility is doubled (K >1), just like females
- If the initial cosexual is partially selfing, higher increases in male fertility are required
- Other things being equal, invasion of males into partially selfing populations requires a higher fertility advantage than for females

  - Unless $S = 0$ (complete outcrossing)
- This assymmetry occurs because, with partial selfing, ovules are less available to pollen from other individuals, which makes it harder to gain fitness by increasing pollen output

(ii) What does this difference predict in biological terms?

- The theory therefore predicts that androdioecy will be rare
- Dioecy probably doesn't evolve via androdioecy
- The selective forces leading to the evolution of dioecy probably include avoidance of inbreeding, but also re-allocation of reproductive resources.

NOTE that some androdioecious species are known. There are 2 kinds. First, some animals (including *Caenorhabditis elegans*) are polymorphic for males and hermaphrodites, where the hermaphrodites are unable to fertilize the eggs of other hermaphrodites (unlike the cosexuals assumed in our model); the reproductive fitness of these hermaphrodites is not as high as that of cosexuals in our model, so it is less difficult for males to invade populations. Second, some androdioecious plants probably evolved by breakdown of dioecy in response to pollen limitation; if hermaphrodites are modified (mutant) females that can produce some pollen, their pollen output might be expected to be much lower than that of cosexuals, satisfying the condition that K must be high for males to persist alongside hermaphrodites.

2. Male sterility (gynodioecy)

All females are homozygotes for the male-sterility mutation (A_2A_2), because A_2 is assumed to be recessive.

Females (phenotype 2, frequency X) produce 1 + *k* times as many seeds as cosexuals.

Cosexuals (phenotype 1, frequency 1-X) have selfing rate *S* (the mixed mating model)

The progeny produced by selfing suffer reduced fitness, with a fitness δ relative to the fitness of outcrossed progeny (inbreeding depression)

The females have no selfing (or male fertility).

The per individual contributions to fitness of the two phenotypes through female gametes are as follows:

Cosexes, through female gametes: S(1-δ)+1-S

w_1^f=(1 - *S*)+ S(1-δ) = 1 – *S*δ

Females (which produce only female gametes, and have *S*=0): 1+k

TOTAL through female gametes: W = (1-X)[(1 - *S*)+ S(1-δ)] + X(1+k)

The contributions to female fitnesses, dividing by the total, are thus w_1^f=(1 – *S*δ)/W and w_2^f=(1 +k)/W

Cosexes contribute the same total through male gametes, because each sex contributes the same number of gametes to the next generation.

Two things contribute to the individual contributions to male fitness: self-fertilization, and fertilization of outcrossed ovules/eggs of other individuals. Per individual of the cosexual phenotype 1, the contribution through outcrossing to male fitness is thus proportional to [(1-X)(1-S)+ X(1+k)]/(1-X). The contribution through self-pollination for these phenotypes are the same as for female gametes, i.e. *S*(1 - δ). Normalizing by the total gamete contribution *W*, we have:

w_1^m= {*S*(1 - δ) + [(1-S)(1-X)] + X(1+k)]/ (1-X) }/W

and of course w_2^m=0 for females. At equilibrium, $w_1^f + w_1^m = w_2^f$

The quantity *W* is the same for both phenotypes. At equilibrium, we therefore have:

1 – *S* + 2*S*(1 - δ) + [(1-S)(1-X) + X(1+k)]/(1-X) = 1 + k

(1-X)[1 – *S* + 2*S*(1 - δ)] + 1-S + X(1+k) = (1-X)(1 + k)

This gives X = (k+2Sδ -1)/2(k+sδ)

QUESTIONS

(i) Most loss-of function mutations are recessive. Recessive male-sterility mutations can invade in this model because the cosexes reproduce by partial selfing, thus producing A_2A_2 homozygotes

(ii) What else has been assumed in addition to the assumptions listed in the question?

- We assumed
    - that all ovules get fertilized (no pollen limitation),
    - and that selfing of cosexuals' ovules occurs by prior selfing,
    - and that outcrossing occurs by the mixed mating model
    - that females have zero male fertility (the reason for this assumption is that just having reduced pollen output might not lead to much reduction in the proportion of ovules that are self fertilized)

(iii) Why can males sometimes invade such a polymorphic population?

- Ovules present in female individuals can be fertilized through outcrossing, so that the genetic values of pollen production (the fitness contributed per amount of pollen produced) is greater than in the absence of females.

- Recall from the lectures that linkage of a female-suppressing mutation to the locus with the male-sterility mutation makes spread of males more likely.