#### Lecture Handout 11

Sexual selection: the genotype proportions of reproductively mature males and females are not the same as the actual genotypes or mating proportions among successful mates. Often involves male competition or female choice.

Mate success in many species is strongly affected by male competition or female choice (noted by Darwin). These may lead to extreme morphological or behavioral traits in males (eg size difference in elephant seals). Or to post-mating, sperm competition in *Drosophila*. Or to females choosing males with "good genes".

**Sexually antagonistic genes**: genes that have opposite selective effects in females and males. Usually selection favors one genotype in one sex for one component of fitness but favors other genotypes in the other sex for a different componet of viability. In this case, different genes may be involved (so doesn't have to involve pleiotropic effects in a single gene).

Red queen process or evolutionary arms race.

Positive assortative mating: similar individuals mate more often than randomly expected. Results in no change in allele frequency but an increase of homozygosity (as in inbreeding).

Negative assortative mating: individuals with unlike genotypes or phenotypes tend to mate with each other more often than randomly expected.

#### **Negative assortative mating:**

model assumes that a certain proportion of the population (R) mates assortatively, having dominant x recessive matings, while the rest of the population (1-R) mates at random.

### **Negative assortative mating:**

**TABLE 4.4** The frequency of mating types and the proportion of progeny produced for a general negative-assortative mating model.

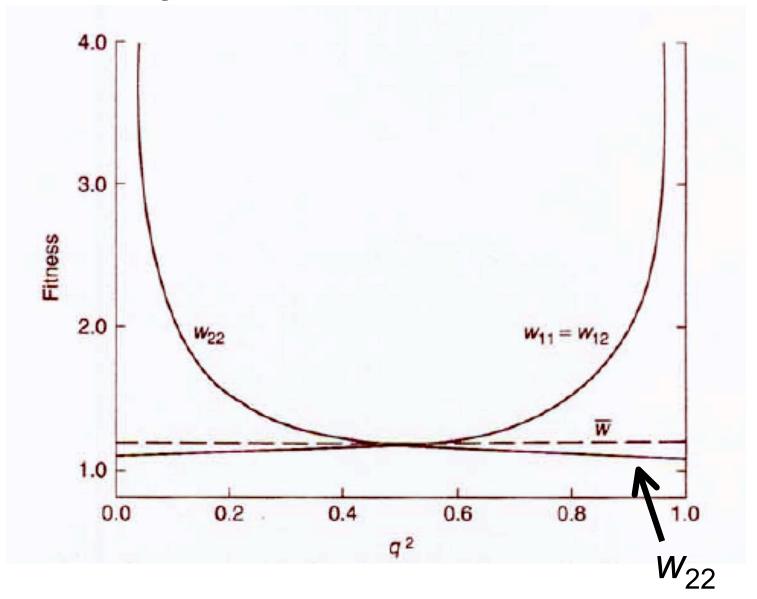
| Mating type            | Frequency      | Progeny    |                             |                                |
|------------------------|----------------|------------|-----------------------------|--------------------------------|
|                        |                | $A_1A_1$   | $A_1A_2$                    | $A_2A_2$                       |
| $A_1$ -× $A_1$ -       | $(P+H)^2(1-R)$ | $p^2(1-R)$ | pH(1-R)                     | $\tfrac{1}{4}H^2(1-R)$         |
| $A_1 - \times A_2 A_2$ | 2(P+H)Q(1-R)+R |            | $2pQ(1-R)+rac{pR}{P+H}$    | $HQ(1-R)+rac{HR}{2(P+H)}$     |
| $A_2A_2 \times A_2A_2$ | $Q^2(1-R)$     |            |                             | $Q^2(1-R)$                     |
|                        | 1              | $p^2(1-R)$ | $2pq(1-R) + \frac{pR}{P+H}$ | $q^2(1-R) + \frac{HR}{2(P+H)}$ |

### Rare male mating advantage?

A form of frequency dependent mating

Controversial: are the observations due to problems with observer bias, experimental design, or data analysis?

## Frequency dependent selection



# Female choice, MHC, and negative assortative mating

MHC: recognizes pathogens and initiates immune responses.

Mice can detect MHC differences in other mice, and females choose mates that differ in MHC. (controversial)

Model results in stable polymorphism and excess of heterozygotes.

# Female choice, MHC, and negative assortative mating

**TABLE 4.5** The mating types for a MHC gene, giving the number of alleles that males differ from females, relative mating preference, and the relative mating frequencies

| Mating type<br>Female × male | Number of different alleles | Mating preference | Mating frequencies          |
|------------------------------|-----------------------------|-------------------|-----------------------------|
| $A_1A_1 \times A_1A_1$       | 0                           | 1-s               | $P_{11}^2(1-s)/w_{11}$      |
| $A_1A_1 \times A_1A_2$       | 1                           | 1-hs              | $P_{11}P_{12}(1-hs)/w_{11}$ |
| $A_1A_1 \times A_2A_2$       | 2                           | 1                 | $P_{11}P_{22}/w_{11}$       |
| $A_1A_2 \times A_1A_1$       | 0                           | 1-s               | $P_{11}P_{12}(1-s)/w_{12}$  |
| $A_1A_2 \times A_1A_2$       | 0                           | 1-s               | $P_{12}^2(1-s)/w_{12}$      |
| $A_1A_2 \times A_2A_2$       | 0                           | 1-s               | $P_{12}P_{22}(1-hs)/w_{12}$ |
| $A_2A_2 \times A_1A_1$       | 2                           | 1                 | $P_{11}P_{22}/w_{22}$       |
| $A_2A_2 \times A_1A_2$       | 1                           | 1-hs              | $P_{12}P_{22}(1-hs)/w_{22}$ |
| $A_2A_2 \times A_2A_2$       | 0                           | 1-s               | $P_{22}^2(1-s)/w_{22}$      |

#### **Gametic selection:**

Meiotic drive or segregation distortion: gametes produced from heterozygotes are not in equal proportions, ie don't conform to Mendelian segregration.

Is an example of "selfish genes" that interfere with other genes to increase their own frequency. Generally subject to counterbalancing selection.

#### Meiotic drive or segregation distortion.

Example: t locus in mouse chromosome 17

- alleles are lethal when homozygous
- but heterozygous males produce large majority of sperm with the *t* allele.

**Self-incompatibility alleles**: prevent self fertilization in plants, result in the absence of germination or growth of pollen on the stigma of flowers from the same or genetically similar plants. Two types:

- (1) Gametophytic self-incompatibility (GSI): pollen must have different allele from female plant (leads to obligate heterozygosity).
- (2) Sporophytic self-incompatibility (SSI): results from genotype of the male parent.

### Self-incompatibility alleles

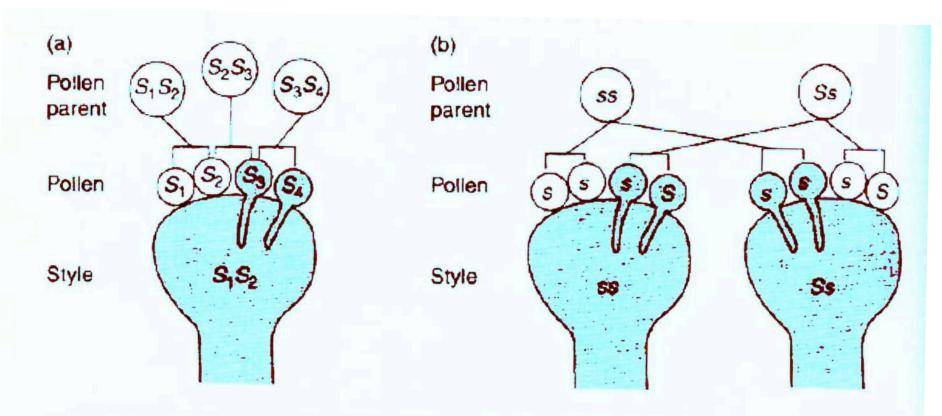
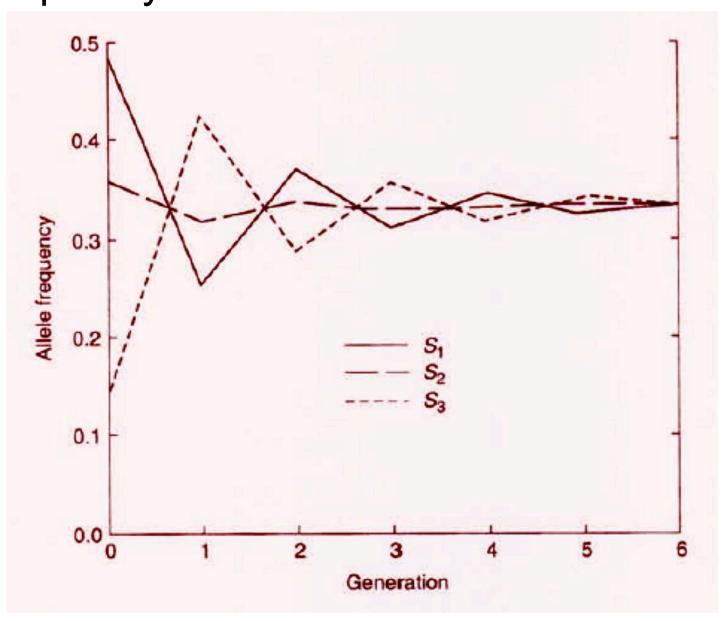


Figure 4.10. Diagrams of pollen tube growth for (a) gametophytic and (b) sporophytic incompatibility.

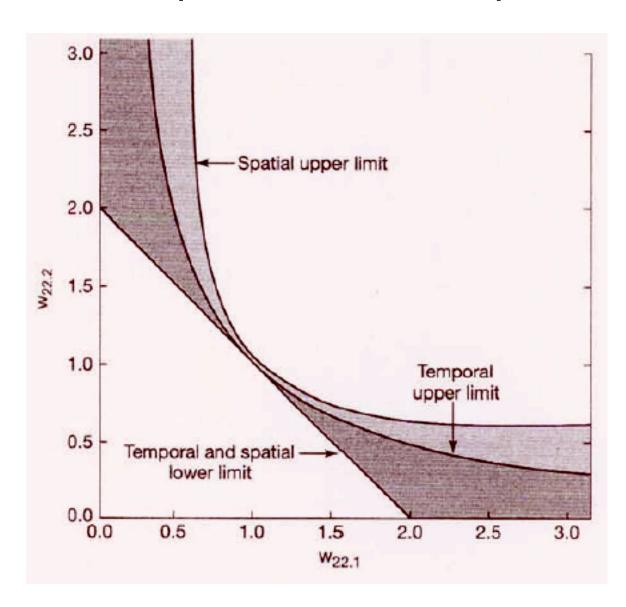
# GSI system with *n* alleles: equilibrium frequency for all alleles is 1/*n*.



Ecological genetics Assumes that selective values are environmentally dependent and that the environment may vary over time and space.

May vary due to predators, prey, parasites or hosts; physical environmental factors; or samespecies density effects

# Spatial or temporal variation: Fitness varies over space or time, respectively



Habitat selection: Individuals prefer ecological niches in which they have

better fitness

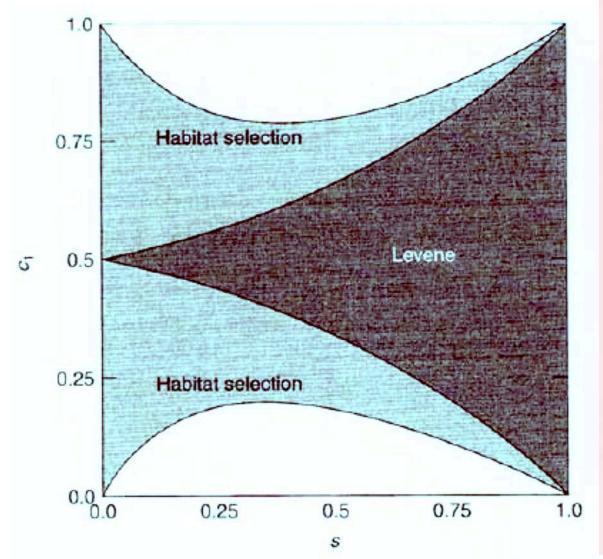
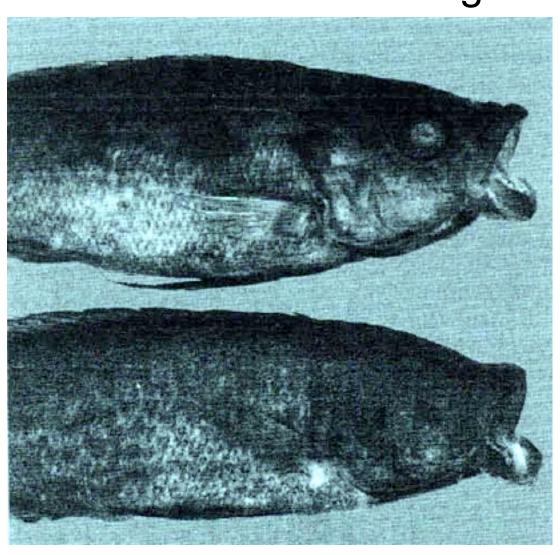


Figure 4.17. The region of a stable polymorphism for the Levene model, in which there is no habitat selection (dark shaded) and where there is habitat selection with h = 0.625 (all shaded area between outside curved lines), where c1 is the proportion of niche 1 and s is the selective difference between the two homozygotes.

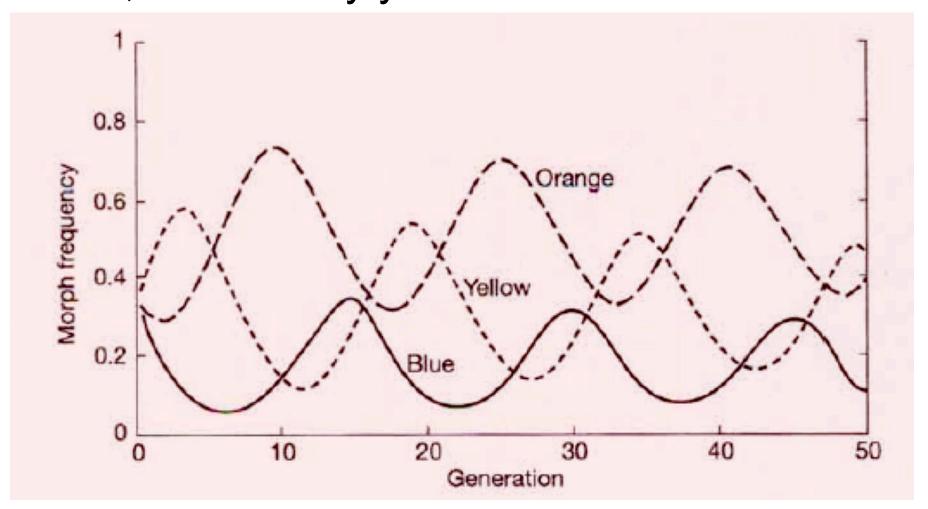
### Frequency dependent selection:

Homozygotes of uncommon alleles have greater or lesser fitness than other genotypes due to their level of frequency in the population.

Positive frequency dependent selection: Selection against lower frequency alleles: eg rare flower type not recognized by pollinator, may lead to faster fixation of common alleles **Negative** frequency dependent selection: Selection against higher frequency alleles: eg "handedness" of scale-eating cichlid



"Rock-scissors-paper" fitness interactions, eg three male color morphs of side-blotched lizards. Dominant orange, mate-guarding blue, and sneaky yellow



Coevolution: evolution that occurs as a result of different species responding to each other. Species may be host and pathogen; predator and prey; competitors; or mutualists.

Host-pathogen interactions: the interactions of pathogens and their hosts may result in frequency-dependent selection; eg if a pathogen type becomes common, host types resistant to that particular pathogen may also increase

#### **Host-pathogen interactions:**

Gene-for-gene change: in plants, widespread matching of host alleles coferring resistance to specific strains of pathogen.

Matching-allele model: Assumes both host and pathogen are polymorphic for two alleles (A and a in host, B and b in parasite), with different genotype fitnesses for the host in response to genotypes in the pathogen.

## Matching-allele model

