§4.2 Selection

Changes in gene frequency

One of the conclusions of the previous section was that the existence of selective differences among genotypes generally leads to changes in gene frequencies. Assuming Hardy-Weinberg proportions, the frequency p' of A_1 in any generation is related to its frequency p in the previous generation by

$$p' = \frac{w_{11}p^2 + w_{12}pq}{w_{11}p^2 + 2w_{12}pq + w_{22}q^2} = f(p) \qquad (2.1)$$

where the w_{ij} are defined in Section 4.1. The change Δp in the frequency of A_1 id thus

$$\Delta p = pq\frac{w_{11}p + w_{12}(1-2p) - w_{22}q}{w_{11}p^2 + 2w_{12}pq + w_{22}q^2}. \qquad (2.2)$$

Clearly $\Delta p = 0$ whenever $p = 0$ or $p = 1$, corresponding to fixation of A_2 or A_1. Δp is also zero when

$$p = p^* = \frac{w_{12} - w_{22}}{(w_{12} - w_{22}) + (w_{12} - w_{11})}. \qquad (2.3)$$

The evolution of the process is obtained by iterating the transformation law (2.1). The following classical results are readily established (cf. Figure 2.1 below) independent of the initial p $(0 < p < 1)$.

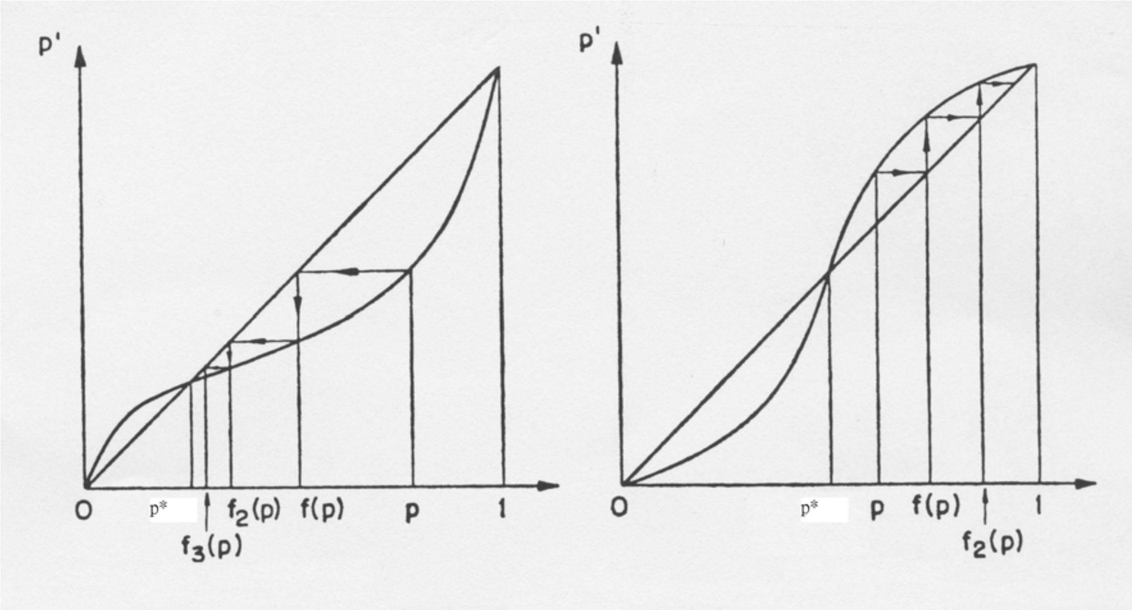
$$\lim_{n\to\infty} f^n(p) = \lim_{n\to\infty} f(f^{(n-1)}(p)) = 1 \text{ (=0) when } w_{11} \ge w_{12} > w_{22} \quad (w_{22} \ge w_{12} > w_{11}),$$

$$\lim_{n\to\infty} p_n = p^* = \frac{w_{12} - w_{22}}{w_{12} - w_{11} - w_{22}} \quad \text{when} \quad w_{12} > \max(w_{11}, w_{22}).$$

In the case $\min(w_{11}, w_{22}) > w_{12}$

$$\lim_{n\to\infty} p_n = 1 \text{ for } p > p^*, = 0 \text{ for } p < p^*.$$

Figure 2.1 shows what happens to $f_{(n)}(p)$ in graphical form. The rigorous details are easily supplied.



$w_{12} > w_{11},\ \ w_{22}$ $w_{12} < w_{11},\ \ w_{22}$

Figure 4.2.1

The equilibrium p^* is of great importance biologically because it entails the simultaneous existence at an equilibrium involving all genotypes. Thus when the heterozygote is the most fit of the three genotypes a stable **polymorphism** (with all forms) will be maintained. The model of heterozugote advantage (also called the principle of overdominance) has been central to the development of theories on the existence of genetic variability.

Perhaps the best-known example of this situation in man concerns the phenomenon of sickle-cell anaemia. The maintenance of high frequencies for both the sickle cell gene and its normal allele in certain East African tribes appears to be due to a selective advantage of heterozygotes brought about by the increased resistance of such heterozygotes to malaria.