ASSORTATIVE MATING BASED ON PHENOTYPE: I. TWO ALLELES WITH DOMINANCE

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IN animals with separate sexes several traits are known to induce some sort of preference for mating between similar individuals (assortment) or preference for mating between dissimilar individuals (disassortment). Assorting, for example, is known to occur in certain bird, mammal (man included) and insect populations.

Traits affecting preferences can be partly subject to environmental control; e.g., odor in mice or size in man. To date, not many instances of simple Mendelian traits affecting preferences have been found. Most examples of such Mendelian factors are associated with pigment color or pattern (MAINARDI 1968; PARSONS 1967).

The model proposed so far to account for the effects of assortative mating are such that gene frequencies remain invariant over time and only the genotypic distribution is altered. The simplest model of this kind, complete assortment with no other disturbance, was treated initially by JENNINGS (1916) and extended by WENTWORTH and REMICK (1916). S. WRIGHT (1921) examined more general situations where, among mating individuals, there is a constant somatic correlation, possibly determined by several loci with equal additive effects and influenced by environment. The case of a single locus, with complete dominance and no environmental effect, is characterized by prescribing a constant correlation, σ , between uniting zygotes the same for all phenotypic pairs. Using heuristic arguments, WRIGHT established a reduction in the proportion of heterozygotes, measured by the "inbreeding coefficient", F , according to the recurrence relation

$F_{n+1} = \frac{\sigma}{2} (1 + F_n)$ so that

$$F_n = \frac{\sigma}{2-\sigma} + \left(F_0 - \frac{\sigma}{2-\sigma}\right) \left(\frac{\sigma}{2}\right)^n.$$

Thus a correlation σ has the same effect as mixed selfing and random mating where a proportion σ of the population is selfing.

In this and a companion paper we shall formulate and analyze several simple models for partial assortment. Sex-dependent selection for mating could be treated in a similar way. The models attempt to take account of such factors as different

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degrees of assorting in the various phenotypes, relative fertilities of matings which occur assortatively compared to those occurring at random, the mechanism and timing of pair formation, the availability of proper mates, etc. We are mainly interested in the evolutionary effects of assortment as a form of "sexual selection".

The models presented in this paper were formulated principally by the first author, the mathematical analysis is primarily due to the second author.

1. AUTOSOMAL TRAITS; MODELS:

In this first paper we consider a number of deterministic models for partial assortment on a pair of Mendelian alleles, A being completely dominant over a . In this series of papers it is assumed that sexual preferences are manifested only in one sex. This covers most known instances where only the females exercise a choice. For convenience of exposition we refer in the following to "females making a choice" in the case of autosomal traits. No differences arise in most of the models if the males are choosing instead. However, this distinction is decisive in the case of sex-linked traits.

Degrees of assortment in the phenotypes are measured by two parameters: α ($0 \leq \alpha < 1$) will be the fraction of dominant females "preferring" to mate with their kind, β ($0 \leq \beta < 1$) that of recessive females preferring their kinds. Note that strict upper inequalities are assumed unless otherwise indicated. Specifying the fraction of dominant and recessive females "preferring" to mate with their own kind does not completely delimit the model. Additional information is needed in describing how the preferences operate.

Specifically, one might assume that the fractions α of dominant and β of recessive females mate only with their own kind if partners are available. Availability will depend on sex ratio, the actual frequencies of the various genotypes and the capability of single males to fertilize large or small numbers of females. Alternatively, one might assume that females try to mate with their own kind and, if not successful, settle for a different partner. Other formulations or variations of the above assumptions can be found in KARLIN and SCUDO (1969), KARLIN (1968), and SCUDO (1967).

Consider first a model in which permanent pairs are formed, the sex ratio is 1:1 and all mature individuals mate. Two sharply contrasting assumptions can be made on the timing of pair formations depending on whether assorting females mate prior to the nonassorting ones or after. Denote by X , Y , and Z the frequencies of AA , Aa and aa genotypes, respectively. When assortment takes place first, a fraction $\alpha(X + Y)$ of the dominant females form pairs with an equal fraction of dominant males. The same occurs for a fraction βZ of recessive males and females. The remaining individuals whose proportions are $(1 - \alpha)(X + Y)$ and $(1 - \beta)Z$, mate at random. The resulting relative frequencies of the mating types are recorded in Table 1-2. We may "normalize" back to gene frequencies simply by dividing the proportions in the random part of the matings by $(1 - \alpha)(X + Y) + (1 - \beta)Z$ (Case 1). A model of this type has been considered by

O'DONALD (1960). As another possibility, suppose that the delay in pairing causes a decrease in the fertility of the females mating at random. A simple way to reflect this circumstance is to assume that the contribution to the next generation of the part of the population undergoing random mating is $\{(1-\alpha)(X+Y) + (1-\beta)Z\}^2$ instead of $(1-\alpha)(X+Y) + (1-\beta)Z$ (Case 2). This is a very simple way to account for a situation in which the smaller the proportion of individuals who delay mating the more their relative fertility will be impaired.

Consider now an alternative formulation in which random-mating females pair first. The remaining $\alpha(X+Y)$ dominant and βZ recessive females will attempt to form pairs with the free males. The proportions of males available to the assorting females are $(X+Y)\{\alpha(X+Y) + \beta Z\}$ dominants and $Z\{\alpha(X+Y) +$

TABLE 1-2

Population array for assortment according to an autosomal, Mendelian pair of alleles in a population with a 1:1 sex ratio forming permanent bonds
Fractions α of the dominant females and β of the recessive females assort first; the remaining individuals mate at random

Mating types	Assorting	Frequencies	Random mating
AA $\times$ AA	$\alpha \frac{X^2}{X+Y}$		$(1-\alpha)^2 X^2$
AA $\times$ Aa	$2\alpha \frac{XY}{X+Y}$		$2(1-\alpha)^2 XY$
AA $\times$ aa			$2(1-\alpha)(1-\beta)XZ$
Aa $\times$ Aa	$\alpha \frac{Y^2}{X+Y}$		$(1-\alpha)^2 Y^2$
Aa $\times$ aa	...		$2(1-\alpha)(1-\beta)YZ$
aa $\times$ aa	βZ		$(1-\beta)^2 Z^2$

Case 1: recursion relations when no reduction in fertility occurs in the random mating females

$$X' = \alpha \frac{(X+Y/2)^2}{X+Y} + \frac{(1-\alpha)^2 (X+Y/2)^2}{R} \quad (1.1)$$

$$Y' = \alpha Y \frac{X+Y/2}{X+Y} + \frac{2(1-\alpha)(X+Y/2)\{\frac{1}{2}(1-\alpha)Y + (1-\beta)Z\}}{R} \quad (1.2)$$

$$Z' = \beta Z + \alpha \frac{Y^2}{4(X+Y)} + \frac{\{\frac{1}{2}(1-\alpha)Y + (1-\beta)Z\}^2}{R} \quad (1.3)$$

where $R \equiv 1 - \alpha + (\alpha - \beta)Z$

Case 2: the average fertility of the random mating females, relative to the assorting ones, is reduced to R .

$$NX' = \alpha \frac{(X+Y/2)^2}{X+Y} + (1-\alpha)^2 \left(X + \frac{Y}{2}\right)^2 \quad (2.1)$$

$$NY' = \alpha Y \frac{X+Y/2}{X+Y} + 2(1-\alpha) \left(X + \frac{Y}{2}\right) \left\{(1-\alpha) \frac{Y}{2} + (1-\beta)Z\right\} \quad (2.2)$$

$$NZ' = \beta Z + \alpha \frac{Y^2}{4(X+Y)} + \left\{(1-\alpha) \frac{Y}{2} + (1-\beta)Z\right\}^2 \quad (2.3)$$

where $N \equiv 1 - R(1 - R)$

βZ recessives. It is easy to see that if $\alpha > \beta$ there will be a deficiency of a fraction $(\alpha - \beta) Z (1 - Z)$ of dominant males; $(\alpha - \beta) Z (1 - Z)$ dominant females will, therefore, be unable to pair according to their preference (Table 3-4). The reverse occurs when $\alpha < \beta$ (Table 5-6). Two simple contrasting assumptions can be made for the mismatched individuals. The remaining females could "change their mind" and pair with the "wrong" males. If these reverting females and the assorting ones have the same fertility as those who mate at random, the appropriate recurrence system connecting genotype frequency over successive generations is obtained by imposing the normalization described in cases 3 and 5 of the tables. One might assume on the other hand, that mismatched females do not contribute at all to the next generation while the assorting individuals have the same mean fertility as the nonassorting individuals. The appropriate recursion relations are given in cases 4 and 6 of the tables. Some similar models have been

TABLE 3-4

Population array in the case of permanent bonds and 1:1 sex ratio when assortment occurs after random mating; when $\alpha > \beta$, $(\alpha - \beta) Z (1 - Z)$ recessive males and as many assorting dominant females are left mismatched

Mating types	Random Mating	Assorting	Frequencies	Mismatched
AA $\times$ AA	$(1 - \alpha)X^2$	$\frac{X^2}{X + Y} \{ \alpha - (\alpha - \beta)Z \}$		
AA $\times$ Aa	$2(1 - \alpha)XY$	$\frac{2XY}{X + Y} \{ \alpha - (\alpha - \beta)Z \}$		
AA $\times$ aa	$(2 - \alpha - \beta)XZ$			$(\alpha - \beta)XZ$
Aa $\times$ Aa	$(1 - \alpha)Y^2$	$\frac{Y^2}{X + Y} \{ \alpha - (\alpha - \beta)Z \}$		
Aa $\times$ aa	$(2 - \alpha - \beta)YZ$			$(\alpha - \beta)YZ$
aa $\times$ aa	$(1 - \beta)Z^2$	βZ		

Case 3: mismatched dominant females pair with recessive males with relative fertilities unaffected

$$X' = \left(1 + \frac{\beta Z}{1 - Z} \right) \left(X + \frac{Y}{2} \right)^2 \quad (3.1)$$

$$Y' = \left(X + \frac{Y}{2} \right) \left\{ \left(1 + \frac{\beta Z}{1 - Z} \right) Y + 2(1 - \beta) Z \right\} \quad (3.2)$$

$$Z' = \beta \left\{ Z + \frac{Y^2}{4(1 - Z)} \right\} + (1 - \beta) \left(Z + \frac{Y}{2} \right)^2 \quad (3.3)$$

Case 4: mismatched individuals do not contribute to the next generation.

$$NX' = \left(\frac{f}{X + Y} + 1 - \alpha \right) \left(X + \frac{Y}{2} \right)^2 \quad (4.1)$$

$$NY' = fY \frac{X + \frac{Y}{2}}{X + Y} + (1 - \alpha) \left\{ X \left(\frac{Y}{2} + Z \right) + \frac{Y}{2} \right\} + (1 - \beta) Z \left(X + \frac{Y}{2} \right) \quad (4.2)$$

$$NZ' = \beta Z + f \frac{Y^2}{4(X + Y)} + (1 - \alpha) \frac{Y}{2} \left(Z + \frac{Y}{2} \right) + (1 - \beta) Z \left(Z + \frac{Y}{2} \right) \quad (4.3)$$

where $N = 1 - (\alpha - \beta) Z (1 - Z)$ and $f = \alpha - (\alpha - \beta) Z$

discussed by SCUDO (1967), KALMUS and SMITH (1967) and KARLIN (1968).

The above models are based on the underlying assumption that assorting does not affect the process of "finding" mates. In noncolonial populations, especially at a rather low density, assorting individuals will have more difficulty in finding a mate than random-mating ones. A formulation for this effect assumes that the contributions of the mating types to the following generations are proportional to the products of the actual densities of the individuals who can take part in the mating. A fraction α of the dominant females are barred from mating with recessives, and a fraction β of the recessives are prohibited from mating with dominants. It is also assumed that fertility is directly proportional to the number of matings which occur in analogy with the dynamics of elastic collisions of particles. The frequencies of the mating types are exhibited in Table 7. This kind of model will be called "mass action"; it is formally identical to familial selection (HALDANE 1921). The special case at hand has already been examined, together with more general models of familial selection, by NABHOLZ (1967).

In many populations with neither permanent bonding nor extensive paternal care males are often present in abundance. We might accordingly assume that assorting causes an unequal contribution from the male genotypes without influencing in any way the fertility of the females. The resulting frequencies of the mating types are then as in Table 8. Models of this kind have been first applied to disassortment by WATTERSON (1959) who called them "asymmetric" since the contributions to the following generation are proportional to the actual numbers of the genotypes only in the female sex. The problem of the initial increase in gene frequencies for similar cases has also been considered by PARSONS (1962).

TABLE 5-6

The same as in Table 3-4 but for $\alpha < \beta$; now $(\beta - \alpha) Z (1 - Z)$ recessive females and as many dominant males are mismatched

Case 5: no effect on fertility

$$X' = \left(1 + \alpha \frac{Z}{1-Z}\right) \left(X + \frac{Y}{2}\right)^2 \quad (5.1)$$

$$Y' = \left(X + \frac{Y}{2}\right) \left\{ \left(1 + \frac{\alpha Z}{1-Z}\right) Y + 2(1-\alpha) Z \right\} \quad (5.2)$$

$$Z' = \alpha \left\{ Z + \frac{Y^2}{4(1-Z)} \right\} + (1-\alpha) \left(Z + \frac{Y}{2} \right)^2 \quad (5.3)$$

Case 6: mismatched individuals have null fertility

$$NX' = \left(\frac{\alpha}{X+Y} + 1 - \alpha \right) \left(X + \frac{Y}{2} \right)^2 \quad (6.1)$$

$$NY' = \alpha Y \frac{X+Y/2}{X+Y} + (1-\alpha) \left\{ X \left(\frac{Y}{2} + Z \right) + \frac{Y}{2} \right\} + (1-\beta) Z \left(X + \frac{Y}{2} \right) \quad (6.2)$$

$$NZ' = Z \{ \alpha + (\beta - \alpha) Z \} + \frac{\alpha Y^2}{4(X+Y)} + (1-\alpha) \frac{Y}{2} \left(Z + \frac{Y}{2} \right) + (1-\beta) Z \left(Z + \frac{Y}{2} \right) \quad (6.3)$$

where $N = 1 - (\beta - \alpha) Z (1 - Z)$

TABLE 7

Population array and recursion system for the "mass action" model, taking into account the "chance of mating"

Mating type	Frequencies
AA × AA	X^2
AA × Aa	$2XY$
AA × aa	$(2 - \alpha - \beta)XZ$
Aa × Aa	Y^2
Aa × aa	$(2 - \alpha - \beta)YZ$
aa × aa	Z^2
Total	$N \equiv 1 - (\alpha + \beta) Z(X + Y) = 1 - (\alpha + \beta) Z(1 - Z)$

$$NX' = \left(X + \frac{Y}{2}\right)^2 \tag{7.1}$$
$$NY' = \{Y + (2 - \alpha - \beta)Z\} \left(X + \frac{Y}{2}\right) \tag{7.2}$$
$$NZ' = \left(Z + \frac{Y}{2}\right)^2 - \frac{\alpha + \beta}{2} YZ \tag{7.3}$$

2. AUTOSOMAL TRAITS; RESULTS:

I. Model 1-2:

In the case of permanent pairs, with assorting females mating first and no reduction in fertility for the random-mating females (Table 1, Case 1), it is easy to

TABLE 8

Population array and recursion formulae for the "asymmetric model"

Mating types	Assorting	Frequencies	Random mating
AA × AA	$\frac{\alpha X^2}{X + Y}$		$(1 - \alpha)X^2$
AA × Aa	$\frac{2\alpha XY}{X + Y}$		$2(1 - \alpha)XY$
AA × aa	...		$(2 - \alpha - \beta)XZ$
Aa × Aa	$\frac{\alpha Y^2}{X + Y}$		$(1 - \alpha)Y^2$
Aa × aa	...		$(2 - \alpha - \beta)YZ$
aa × aa	βZ		$(1 - \beta)Z^2$

$$X' = \left(\frac{\alpha}{X + Y} + 1 - \alpha\right) \left(X + \frac{Y}{2}\right)^2 \tag{8.1}$$
$$Y' = \frac{\alpha XY}{2(X + Y)} + \frac{Y}{2} + (1 - \alpha) X \left(\frac{Y}{2} + Z\right) + (1 - \beta) Z \left(X + \frac{Y}{2}\right) \tag{8.2}$$
$$Z' = \beta Z + \alpha \frac{Y^2}{4(X + Y)} + \left\{(1 - \alpha) \frac{Y}{2} + (1 - \beta) Z\right\} \left(\frac{Y}{2} + Z\right) \tag{8.3}$$

verify that the gene frequency, $1 - q = p = X + Y/2$, is invariant over time. Using this fact, it suffices to analyze only one of the recursion equations, say (1.2), which can be rewritten as

$$Y' = \frac{\alpha p Y}{p + \frac{Y}{2}} + 2p(1 - \alpha) \frac{(1 - \beta)q + \frac{\beta - \alpha}{2} Y}{1 - \alpha p - \beta q + (\beta - \alpha) \frac{Y}{2}} \equiv f(Y). \quad (1.4)$$

Of course, p, q are positive constants.

The frequency of Aa in the n th generation will be $Y_n = f^{(n)}(Y_0)$ where $f^{(n)}(\cdot)$ denotes the n th functional composition of $f(\cdot)$; this notation will be used throughout. By direct verification we find that $f(Y)$ is concave and $f(0) > 0$. It follows that $f(Y) = Y$ admits a unique solution, $\hat{Y}$, in $(0, 1)$ and Y_n converges to $\hat{Y}$. This can be computed as the unique root in $(0, 1)$ of the cubic

$$-\left(\frac{\beta - \alpha}{4}\right) Y^3 - \frac{1}{2} (1 - \alpha p - \beta q) Y^2 + (1 - \beta)(1 - \alpha) p^2 Y + 2(1 - \alpha)(1 - \beta)q = 0.$$

Alternatively, by substituting $Y = 2(1 - \mathbf{F})pq$, $Y' = 2(1 - \mathbf{F}')pq$ in (1.4), we obtain an equivalent recurrence relation for the inbreeding coefficient

$$\mathbf{F}' = 1 - \frac{\alpha(1 - \mathbf{F})}{1 + (1 - \mathbf{F})q} - (1 - \alpha) \frac{1 - \beta + (1 - \mathbf{F})(\beta - \alpha)p}{1 - \alpha p - \beta q + (\beta - \alpha)(1 - \mathbf{F})pq} \quad (1.5)$$

and similar analysis to that for (1.4) applies. Thereby, we obtain a family of equilibria, depending on the initial gene frequency p . The frequency of heterozygotes is eventually reduced to $2(1 - \hat{\mathbf{F}})pq$, where unlike the situation of standard inbreeding, $\hat{\mathbf{F}}$ depends on p .

The special case $\alpha = \beta$ is of interest as it corresponds to the usual version of mixed assortative and random mating (O'DONALD 1960). The above recursion relation assumes the form

$$Y' = p \frac{2(1 - \beta) p(1 - p) + Y\{1 - p(1 - \beta)\}}{p + \frac{Y}{2}}. \quad (1.6)$$

This transformation is of the linear fractional type. The expression of Y_n in terms of Y_0 is given by

$$\frac{Y_n - v_1}{Y_n - v_2} = K^n \frac{Y_0 - v_1}{Y_0 - v_2} \quad (1.7)$$

(see, e.g., KARLIN 1966, p. 296) where

$$\begin{aligned} v_1 &= p^2(\beta - 1) + p[(1 - \beta)\{p^2(1 - \beta) + 4q\}]^{1/2} \\ v_2 &= p^2(\beta - 1) + p[(1 - \beta)\{p^2(1 - \beta) + 4q\}]^{1/2} \end{aligned} \quad (1.8)$$

and

$$K = \frac{v_2\{2(1 - \beta)pq - v_1\}}{v_1\{2(1 - \beta)pq - v_2\}}.$$

Thus Y_n converges to $v_1 \equiv \hat{Y}$ at the geometric* rate

$$\lambda = \frac{8\beta p^2}{(2p + \hat{Y})^2}$$

which also depends on gene frequency.

When $\beta = 1$, (1.6) reduces to

$$Y' = \frac{Yp}{p + \frac{Y}{2}} \quad (1.10)$$

which, by simple manipulation, leads to

$$Y_n = \frac{2pY_0}{nY_0 + 2p} \quad (1.11)$$

and so $Y_n \rightarrow 0$, $X_n \rightarrow p$, $Z_n \rightarrow q$ at an algebraic* rate (cf. JENNINGS 1916).

In the same case of permanent pairs, when the relative mean fertility of all random-mating females reduces to R (Table 1-2, Case 2), the gene frequency is no longer invariant over time. In fact, its change from generation to generation has the form

$$p' = p \frac{1 - (1 - \alpha)(1 - R)}{1 - R(1 - R)} \quad (2.4)$$

Analysis of (2.4) reveals the following results:

- i) if $\alpha > \beta$, then $p_n \uparrow 1$ as $n \rightarrow \infty$, i.e., the population tends slowly towards fixation of the dominant gene, the rate of convergence being algebraic;
- ii) If $\alpha < \beta$, then $p_n \downarrow 0$ as $n \rightarrow \infty$, and the approach to fixation of the gene is geometrically fast at the rate

$$\lambda = \frac{1 - (1 - \alpha)\beta}{1 - (1 - \beta)\beta}$$

per generation;

- iii) if $\alpha = \beta$, gene frequency is invariant over time. The complete time-dependent behavior of, say, Y_n is easily worked out by considering the recursion relation

$$\{1 - \alpha(1 - \alpha)\} Y' = \alpha Y \frac{p}{p + \frac{Y}{2}} + 2(1 - \alpha)^2 p(1 - p)$$

to which the analysis used in case (1.4) applies.

II. Models 3-6:

In the other model with permanent pairs, mating occurs in three stages: non-assorting females mate first, assorting females later find mates if proper partners are available, and finally the remaining females pair off with the remaining males of different phenotype. The corresponding system of recursion relations has two distinct expressions according to whether α exceeds β or vice versa (Tables 3-4

* By algebraic convergence we mean that $n|Y_n - \hat{Y}|$ has a finite, nonzero limit as n tends to infinity, i.e., $\lim_{n \rightarrow \infty} n|Y_n - \hat{Y}| = b$ where $0 < b < \infty$. Thus, for instance, a reduction of $1 - X_n$ by 1 percent requires about 100 generations. By geometric convergence we mean that $\lim_{n \rightarrow \infty} [|Y_n - Y|/\lambda^n] = a$ where $0 < a < \infty$ for some λ , $0 < |\lambda| < 1$.

and 5-6). If all females have the same mean fertility irrespective of the stage of mating (cases 3 and 5), then gene frequency is again an invariant over time.

When $\alpha > \beta$ (Case 3), the recursion relations for zygote frequencies depend only on β . When $\alpha < \beta$ the recursion relations maintain the same functional form with α substituted throughout for β . Thus the timing for pair formation, when not affecting fertility, introduces a curious symmetry. When random mating occurs first, the effect of assorting is completely determined by the *smaller* of the two assorting parameters. When the order is reversed the behavior of the system depends on both parameters.

Cases 4 and 6 of the model assume that assorting females which are not able to find a proper mate do not contribute at all to the next generation. Consider first the case $\alpha > \beta$; it is convenient to rewrite the recursion relations (4.1)–(4.3) in terms of $p = (X + Y/2) = 1 - q$ and Z , yielding

$$p' = p \frac{1 - \frac{\alpha - \beta}{2} Z}{1 - (\alpha - \beta) Z(1 - Z)} \quad (4.4)$$

$$Z' = \frac{\beta Z + (1 - \alpha)q^2 + (\alpha - \beta)qZ + \frac{\alpha - (\alpha - \beta)Z}{1 - Z} (q - Z)^2}{1 - (\alpha - \beta) Z(1 - Z)} \quad (4.5)$$

From (4.4) we infer that p is increasing when $(\alpha - \beta) Z(1/2 - Z) > 0$ or equivalently

$$p' > p \text{ if and only if } 0 < Z < 1/2. \quad (4.6)$$

Let $\hat{p}$ be the equilibrium gene frequency obtained by solving (4.5) with $Z = Z' = 1/2$.

It can be proved (see Appendix 1) that starting from any Z , p satisfying $0 < Z < 1/2$, $\hat{p} < p < 1$ (the vertically shaded region in the deFinetti diagram of Figure 1), the system converges towards fixation of the dominant allele.

By the same reasoning, for any initial composition, Z , p , satisfying $1/2 < Z < 1$, $0 < p < \hat{p}$ (horizontally shaded region in Figure 1), the system will converge towards the fixation of the recessive allele.

An invariant curve located in the white area of the diagram, and having a complex analytical form, separates the domains of convergence to the two trivial equilibria; on it the system moves towards the unstable interior polymorphism. While convergence to the pure recessive state occurs at a geometrically fast rate, convergence to the pure dominant state always occurs at an algebraic, or much slower rate (cf. case 1).

Equilibrium zygote frequencies are more easily calculated in terms of Y , ($\hat{Z} = 1/2$, $\hat{X} = 1/2 - \hat{Y}$), where $\hat{Y}$ is the unique root in $(0, 1)$ of the quadratic

$$2(1 + \beta) Y^2 + 2(2 - \alpha - \beta) Y - 2 + \alpha + \beta = 0, \quad (4.7)$$

In the case $\alpha < \beta$ the relation connecting gene frequencies over successive generations is

$$p' = p \frac{1 - \frac{\beta - \alpha}{2} Z}{1 - (\beta - \alpha) Z(1 - Z)} \quad (6.4)$$

FIGURE 1.—deFinetti diagram illustrating the stability properties of case 4 ($\alpha = .2$, $\beta = .1$ in this example). The curve in the white region, not drawn to scale, separates the domains of attraction of the two pure states; it has a complex analytical determination. Convergence to each fixation is proved for the two shaded regions (Appendix 1).

which is the same as (4.4) with α , β interchanged. The nontrivial equilibrium is

$$\dot{Z} = 1/2, \dot{X} = 1/2 - \dot{Y} \text{ where } \dot{Y} \text{ is the unique solution in } (0,1) \text{ of} \\ 2(1 + \alpha) Y^2 + 2(2 - \alpha - \beta) Y - 2 + \alpha + \beta = 0. \quad (6.5)$$

An analysis parallel to the case $\alpha > \beta$ applies to the present one.

Finally when $\alpha = \beta$, gene frequency becomes invariant over time and only one of the recursion relations is independent. That for Y can be rewritten as

$$Y' = \frac{\beta p Y}{p + \frac{Y}{2}} + 2(1 - \beta) p(1 - p),$$

and the same reasoning as for case 1 applies.

While case 1 is rather similar to cases 3 and 5 there is a large difference between case 2 and cases 4 and 6. In the former, the relative magnitude of α , β determines the stability of the pure states; in the latter, both pure states are stable (provided $\alpha, \beta > 0$) and the magnitudes of α , β only affect the domains of attraction.

III. Mass Action Model:

The recursion relations for the "mass action model" (Table 7) become, in terms of the variables Z and $p \equiv 1 - q$,

$$p' = p \frac{1 - aZ}{1 - 2aZ(1 - Z)} \quad (7.4)$$

$$Z' = \frac{q^2 - 2aqZ + 2aZ^2}{1 - 2aZ(1 - Z)}, \text{ where } a = \frac{\alpha + \beta}{2}. \quad (7.5)$$

The unique interior equilibrium is

$$Z = \frac{1}{2}$$

$$\hat{p} = 1 - \frac{\alpha + \sqrt{(2-\alpha)(1-\alpha)}}{2}. \quad (7.6)$$

The value of $\hat{p}$ depends in an almost linear form on α , its values ranging from $\sim .293$ to $.5$ as α ranges from 0 to 1. Paraphrasing the same argument for cases 4-6, it is easy to prove that starting from an initial set of frequencies satisfying $0 < Z < \frac{1}{2}$, $\hat{p} < p < 1$ fixation of A always takes place; while from $\frac{1}{2} < Z < 1$, $0 < p < \hat{p}$ fixation of a occurs. Identical considerations as for model 4-6 apply in ascertaining the rates of convergence and the exact domains of attractions. The principal difference is that the parameters α , β enter model 7 only through their sum.

IV. *Model 8: The asymmetric model* (Table 8) allows for an excess of males so that assorting does not decrease the chance of a female to be fertilized. The recursion formula for gene frequency achieves the simple form

$$p' = p(1 + \frac{\alpha - \beta}{2} Z) \quad (8)$$

from which the following inferences are easy:

- i) when $\alpha > \beta$ the only stable state for the population is that with only dominants ($X = 1, Y = Z = 0$); convergence to this state takes place, as usual, at an algebraic rate;
- ii) when $\alpha < \beta$ pure recessive is the only stable state, and it is approached geometrically fast, at the rate

$$\lambda = 1 + \frac{\alpha - \beta}{2};$$

- iii) when $\alpha = \beta$ the situation is again that of the standard case of mixed assortative and random mating (formulae (1.4) to (1.11)).

3. SEX-LINKED TRAITS

While for autosomal traits no distinction is usually made regarding the sex that performs the choice, in the case of sex-linked traits such a distinction must be made and has some interesting consequences.

As an example, we examine the "asymmetric model" in which males are assumed to be in large excess such that the propensity for assortment does not appreciably reduce the chance of a female to be fertilized. There are four alternatives of this model depending on which sex is heterogametic and which sex exercises the choice.

It is convenient in the following to refer to diploids and haploids; where the latter will usually be the heterogametic sex. We now denote by X and Y ($X + Y = 1$) the frequencies of A and a haploids, respectively; by U , V and W ($U + V +$

$W = 1$) the frequencies of the AA, Aa and aa diploids; by $p = 1 - q = U + V/2$ the gene frequency in diploids.

Consider first the case in which the females are haploid (heterogametic), choose, and all have the same chance of being fertilized. Table 9 gives the frequencies of the mating types and the recurrence equations connecting gene frequencies in successive generations.

Only three of the five equations are independent; it is convenient to work in terms of X , $p = U + V/2$ and W , so that

$$X' = \frac{\alpha p X}{1 - W} + (1 - \alpha) p X + (1 - \beta)(1 - X)p \quad (9.6)$$

$$p' = \frac{X}{2} + \frac{\alpha p X}{2(1 - W)} + \frac{1 - \alpha}{2} p X + \frac{1 - \beta}{2} (1 - X)p \quad (9.7)$$

$$W' = \{\beta + (1 - \beta)(1 - p)\}(1 - X). \quad (9.8)$$

Therefore, the following relation is immediate

$$p' = (X + X')/2. \quad (9.9)$$

This formula aids in establishing that (9.1-9.3) admits at most one interior equilibrium, given by

$$\hat{p} = \hat{X} = \frac{\alpha - \beta - \beta(1 - \beta)}{(\alpha - \beta)(1 - \beta)}, \quad \hat{W} = \frac{(1 - \alpha)\beta^2}{(\alpha - \beta)^2} \quad (9.10)$$

TABLE 9

Asymmetric model for a sex-linked, dominant trait with choice in the female, heterogametic (haploid) sex

Now X and Y denote the frequencies of A and a haploids; U , V and W the frequencies of AA, Aa and aa zygotes, respectively

Mating types	Assorting	Frequencies	Random mating
AA $\times$ A	$\frac{\alpha XU}{U + V}$		$(1 - \alpha)XU$
Aa $\times$ A	$\frac{\alpha XV}{U + V}$		$(1 - \alpha)XV$
aa $\times$ A	...		$(1 - \alpha)XW$
AA $\times$ a	...		$(1 - \beta)YU$
Aa $\times$ a	...		$(1 - \beta)YV$
aa $\times$ a	βY		$(1 - \beta)YW$
$X' = \left\{ \frac{\alpha X}{U + V} + (1 - \alpha)X + (1 - \beta)Y \right\} \left(U + \frac{V}{2} \right)$			(9.1)
$Y' = \beta Y + \frac{\alpha VX}{2(U + V)} + \{ (1 - \alpha)X + (1 - \beta)Y \} \left(\frac{V}{2} + W \right)$			(9.2)
$U' = \left\{ \frac{\alpha X}{U + V} + (1 - \alpha)X \right\} \left(U + \frac{V}{2} \right)$			(9.3)
$V' = \frac{\alpha XV}{2(U + V)} + (1 - \alpha)X \left(\frac{V}{2} + W \right) + (1 - \beta)Y \left(U + \frac{V}{2} \right)$			(9.4)
$W' = \beta Y + (1 - \beta)Y \left(\frac{V}{2} + W \right)$			(9.5)

which is admissible if and only if

$$\alpha > \beta(2 - \beta) . \quad (9.11)$$

Local linear analysis (Appendix 2) proves that:

- i) the polymorphic solution (9.10–9.11), whenever valid, is locally stable;
- ii) fixation at A ($p = X = 1, W = 0$) is always locally unstable;
- iii) fixation at a ($p = X = 0, W = 1$) is locally unstable when the interior polymorphism exists, stable otherwise.

The inequality (9.11) divides the parameter space into two regions. a) When $0 < \beta(2 - \beta) < \alpha < 1$ the frequencies of the genotypes appear to converge from any nontrivial initial condition towards the polymorphic equilibrium; the rate of convergence is geometric. b) When $0 < \alpha < \beta(2 - \beta) < 1$ the system, if not at fixation of A, converges towards fixation of a, also at a geometric rate. c) On the boundary between the two regions, $\alpha = \beta(2 - \beta) \neq 0$, fixation of a will occur at an algebraic rate.

Special extreme values of α, β must be treated separately. Thus, in particular if $\beta = 0, \alpha > 0$, the only stable situation will be fixation at A which is approached at an algebraic rate. When $\alpha = 1$ and $\beta < 1$ fixation of A is again the only stable situation, being approached at an algebraic rate. Details of these and other cases are discussed in Appendix 3.

If the diploid males exercise the choice in mating while all haploid (heterogametic) females have the same chance of being fertilized we obtain exactly the same results as for Model 9, but with α, β interchanged. Thus both cases of female heterogamety have, as stable situations, either a polymorphism or the fixation of the recessive allele, depending on the relative magnitudes of the assorting parameters. The two cases are symmetric with respect to the choosing sex and the intensities of assortment.

Let us turn next to the case of male heterogamety in which all females are fertilized and assume first that the females exercise the choice. The frequencies of the mating types and the recursion relations for genotypic frequencies are set forth in Table 10.

Observe that in this case the frequency of the A haploids is the same as the frequencies of the A gene in the diploids of the previous generation. The only nontrivial equilibrium of system (10) is easily checked to be

$$\hat{X} = \hat{p} = \frac{\beta - \alpha(2 - \alpha)}{(\beta - \alpha)(1 - \alpha)} \quad (10.6)$$

$$\hat{U} = \hat{p}\{\alpha + (1 - \alpha)\hat{p}\} \quad \hat{V} = 2(1 - \alpha)\hat{p}(1 - \hat{p}) \quad \hat{W} = \frac{\alpha^2(1 - \beta)}{(\beta - \alpha)^2}$$

and is valid only when

$$\beta > \alpha(2 - \alpha) . \quad (10.7)$$

A local linear analysis yields the following results:

- i) the interior equilibrium (10.6–10.7) is always unstable;

ii) fixation of A is always stable; it is approached geometrically fast at the rate

$$\lambda = \text{Max} \left\{ \beta, \frac{1 + \sqrt{9 - 8\alpha}}{4} \right\}.$$

TABLE 10

Population array and recursion formulae in the "asymmetric model" for a sex-linked trait, with choice in the homogametic (diploid) females

Mating types	Assorting	Frequencies	Random mating
AA × A	αU		$(1 - \alpha)XU$
Aa × A	αV		$(1 - \alpha)XV$
AA × a	...		$(1 - \alpha)YU$
Aa × a	...		$(1 - \alpha)YV$
aa × A	...		$(1 - \beta)XW$
aa × a	$W\beta$		$(1 - \beta)YW$
$X' = U + \frac{V}{2}$ (10.1)			
$Y' = \frac{V}{2} + W$ (10.2)			
$U' = \{\alpha + (1 - \alpha)X\} \left(U + \frac{V}{2} \right)$ (10.3)			
$V' = \frac{V}{2} + (1 - \alpha)UY + (1 - \beta)WX$ (10.4)			
$W' = \beta W + (1 - \beta)WY + \frac{1 - \alpha}{2}VY$ (10.5)			

TABLE 11

Comparison of the stability properties of the asymmetric model in the four cases of sex-linked traits

Each case is specified by the ploidy of the female and of the choosing sex. Thus the first two entries in the first row mean: females are heterogametic and also the choosing sex. For each case two regions of the parameter space are distinguished. The stability of a solution is indicated by +, its instability by — and 0 denotes the nonexistence of the polymorphic solution.

Ploidy Females	Choosing sex	Fixation of A	Equilibrium states polymorphism	Fixation of a
$0 < \beta(2 - \beta) < \alpha < 1$				
1	1	—	+	—
2	1	+	—	+
$0 < \alpha < \beta(2 - \beta) < 1$				
1	1	—	0	+
2	1	+	0	—
$0 < \alpha(2 - \alpha) < \beta < 1$				
1	2	—	+	—
2	2	+	—	+
$0 < \beta < \alpha(2 - \alpha) < 1$				
1	2	—	0	+
2	2	+	0	—

- iii) Fixation of a is stable if and only if the interior equilibrium exists. When convergence occurs it is geometrically fast at the rate

$$\lambda = \frac{\frac{1}{2} + \alpha + \sqrt{9/4 + \alpha(1 - \alpha) - 2\beta}}{2}.$$

Thus, for general values of the parameters, when inequality (10.7) holds, both fixations are stable. Which gene fixes depends on what side of the interior solution the starting point is located. When the reverse of (10.7) is true, fixation of A is the only stable situation. Note that for $\beta = 1$, $\alpha < 1$, the stability of the trivial solutions reverse such that fixation of a is stable, convergence taking place at an algebraic rate. This result, which looks paradoxical at first sight, is wholly symmetrical to that for Model 9. It further exemplifies the qualitative difference between complete and incomplete assortment.

The fourth alternative where heterogametic males choose and all females have the same chance of being fertilized, gives the same results as the model of (10) with the parameters interchanged.

The stability results among the four cases of this model are summarized in Table 11.

DISCUSSION

Most previously considered models on mating preferences treat partial assortative mating in a manner such that no change in gene frequency occurs. We have presented a few models which incorporate more of the mechanics of assortment. It is important to emphasize that the mere specification of the intensities for assortment in the various phenotypes is not enough to predict its effect. Throughout, our analysis concentrates on the case in which preferences are exercised by the individuals of one sex only. Various possible effects of assortment have been accounted for in the case of autosomal traits. For example, it has been assumed that assortment might influence the timing of fertilization, and this in turn the fertility of the females. The models also incorporate a measure of the availability of partners of a desired phenotype. Except for very special cases, gene frequency changes from generation to generation and converges towards fixation. Some cases of complete assortment have also been briefly discussed (Appendix 3), primarily to emphasize qualitative differences from partial assortment.

In nongregarious animals assortment might also reduce the chance of meeting a desirable partner. This feature has been taken into account by the usual mass action model. In this case gene frequency is never an invariant over time.

Another possibility is the presence of an excess of males which assures equal chances of fertilization to assorting and nonassorting females. This property has been built into the model by assuming that males possess unlimited fertility. Except for a special case, changes in gene frequency will occur.

It seems unlikely that assorting could occur without one or more of the above factors in operation. Thus, unless these effects balance perfectly, gene frequencies would be expected to change over time.

In all cases of assortment for autosomal traits treated, the change in gene

frequencies is always in the direction of a pure population. In some of the models there is an unstable polymorphism, and according to the initial composition, either allele can be fixed. Usually convergence to the pure recessive state takes place at a geometric rate while that to a pure dominant state occurs more slowly, at an algebraic rate. This class of models could apply to the establishment of partial reproductive isolation between populations.

In other models, no interior polymorphism exists and only one of the pure states is stable. The same occurs when only one phenotype assorts, in models in which an interior polymorphism would otherwise exist. Assorting can thus produce gene substitutions and the resulting net loss in reproductive efficiency will often be smaller than that for ordinary selection with comparable effects. At the limit, due to excess of males, evolutionary processes could occur at no loss at all in reproductive efficiency. Also, the same rates of approach to pure states apply to the initial increase in gene frequencies, i.e., the initial increase in the frequency of a recessive, when occurring, takes place at an algebraic rate, that of a dominant at a geometric one.

The above discussion and analysis allows us to draw the following, seemingly robust conclusion: assortment for dominant autosomal genes will almost always have an effect similar to natural selection in the disruptive or dominant case but it will never produce a stable polymorphism.

To give an idea of the peculiar situation produced by assortment for dominant, sex-linked genes we treated the "asymmetric model" for such cases. Each model has now four specifications according to which sex is heterogametic and which sex exercises the choice.

The first difference from the autosomal case is that the attainment of pure states (or the initial increase) occurs at a geometric rate both for the recessive and the dominant allele. Thus, if other conditions were the same, partial assortative mating would produce substitutions of sex-linked alleles in a much shorter time than for autosomal alleles.

Also, assorting for autosomal genes will produce an effective genetic isolation in populations only with respect to immigrants carrying a variant which is dominant to the established one. Genetic isolation against recessive variants will be much weaker, their elimination occurring at an algebraic rate. On the contrary, genetic isolation for sex-linked traits will have roughly the same effectiveness in both directions.

An even more striking difference occurs in both cases of female heterogamety (females or males exercising the choice). When the intensities of assortment in the two phenotypes exceed some appropriate difference in relative magnitude, stable polymorphisms will result. Thus assortment for sex-linked traits can, and almost always will, produce many of the usual effects of natural selection.

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SUMMARY

Most models previously considered for assortment were such that no change in gene frequencies occurred. In this paper a class of models for partial assortative mating is analyzed, taking account of some behavioral and ecological characteristics. Specifically, factors such as different propensities for assortment in the various phenotypes, the mechanism and timing of pair formation and their effects on the reproductive performance of the pair and the availability of proper mates are considered. It is demonstrated quite generally that assortment for dominant traits cannot lead to the establishment of stable polymorphism. In all these cases gene frequency tends to fixation and which fixation is stable relates to the ecological assumptions of the model.—In contrast to the above conclusion, *stable* polymorphism can occur in cases of assortative mating based on a dominant, sex-linked trait.

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APPENDIX 1

PROOF OF THE STABILITY PROPERTIES OF MODEL 4

We prove that if $0 < Z_0 < \frac{1}{2}$, $\hat{p}' < p_0 < 1$, then $p_n \rightarrow 1$, and $Z_n \rightarrow 0$. Clearly $Z_0 < \frac{1}{2}$ always implies $p'_0 > p_0$ hence it suffices to show that $Z'_0 < \frac{1}{2}$.

Consider the relation

$$N(Z' - \frac{1}{2}) = \beta Z + \frac{\{\alpha - (\alpha - \beta)Z\}(q - Z)^2}{1 - Z} + (1 - \alpha)q^2 \\ + (\alpha - \beta)qZ - \frac{1}{2} + \frac{\alpha - \beta}{2} Z(1 - Z) \quad (1)$$

where $N = 1 - (\alpha - \beta)Z(1 - Z)$. Differentiating its right side with respect to Z and simplifying yields

$$(\alpha - \beta)(\frac{1}{2} - q + Z) + \beta \left\{ \frac{1 + Z - 2q}{1 - Z} + \frac{(q - Z)^2}{(1 - Z)^2} \right\}.$$

The coefficient of $\alpha - \beta > 0$ is positive for $Z \geq Z_0$ since then $\frac{1}{2} - q + Z \geq \frac{1}{2}(1 - Y_0) > 0$. The coefficient of β is also positive since $1 + Z - 2q \geq 1 - Z_0 - Y_0 \geq 0$ for $Z \geq Z_0$.

Thus the right hand side of (1) is increasing in Z (for $Z \geq Z_0$) and, setting $Z = \frac{1}{2}$, we have

$$N(Z'_0 - \frac{1}{2}) \leq \beta/2 + (\alpha + \beta)(q - \frac{1}{2})^2 + (1 - \alpha)q^2 + \frac{\alpha - \beta}{2} q - \frac{1}{2} + \frac{\alpha - \beta}{8} \equiv Q(q) \quad (2)$$

The quadratic equation $Q(q) = 0$ determines $\hat{q}$ and an easy argument verifies that for $q < \hat{q}$ we have $Q(q) < 0$. Thus $Z'_0 < \frac{1}{2}$ which implies $p''_0 > p'_0 > p_0 > \hat{p}$. The argument can be repeated thereby confirming that p_n increases and its limit is necessarily 1.

APPENDIX 2

STABILITY PROPERTIES FOR THE SEX-LINKED CASE IN TABLE 9

The linearized equations for system (9.1-9.3), in the neighborhood of the trivial equilibrium $E_1 = (X = 1, p = 1, W = 0)$ are

$$X' = P + \beta X - \alpha W \\ P' - \frac{X'}{2} = \frac{X}{2} \\ W' = \beta X,$$

leading to the characteristic equation

$$C(\lambda) \equiv \lambda\{-\lambda^2 + \lambda(\beta + \frac{1}{2}) - \alpha\beta + \frac{1}{2}\} = 0.$$

But since $C(1)C(-1) > 0$, provided $\beta > 0$ and $\alpha < 1$, we infer that one root of $C(\lambda)$ always exceeds 1 in magnitude. This allows us to prove that E_1 is locally unstable.

The stability of $E_2 = (X = 0, p = 0, W = 1)$ is difficult to discern from linear analysis. The associated characteristic equation has the form

$$C(\lambda) \equiv \lambda\left\{\lambda^2 - \lambda\left(\frac{1-\beta}{2} + \delta\alpha\right) - \frac{1-\beta}{2}\right\} = 0$$

where δ is an unknown parameter

$$\delta = \frac{P}{W} = \frac{U + V/2}{U + V},$$

P, U and V being both infinitesimal. It transpires that the critical value of δ is actually $(2 - \beta)^{-1}$ but the issue of local stability cannot be settled in any direct manner.

Local analysis of the interior equilibrium (9.4) leads to the characteristic equation

$$C(\lambda) \equiv -\lambda^3 + \lambda^2(\beta + \frac{1}{2}) + \lambda\left\{\frac{1}{2} - \frac{(3-2\beta-\alpha)\beta(\alpha-\beta)}{2\alpha(1-\beta)^2}\right\} \\ - \frac{1}{2} \frac{(\alpha-\beta)(1-\alpha)\beta}{\alpha(1-\beta)^2} = 0:$$

Trivially $C(-1) > 0$, $C(0) < 0$

$$C(1) = \frac{\beta(1-\alpha)}{\alpha(1-\beta)^2} \{\beta(2-\beta) - \alpha\}$$

and

$$C(\frac{1}{2}) = \frac{1}{4} \left\{ 1 + \beta - \frac{(5-2\beta-3\alpha)\beta(\alpha-\beta)}{\alpha(1-\beta)^2} \right\}.$$

Standard perturbation arguments prove that $C(1/2) > 0$ while, obviously, $C(1) < 0$, provided $\beta(2 - \beta) < \alpha < 1$. Therefore, *whenever an interior polymorphism exists, it is locally stable*. In this case it follows from the general theory of iteration of functions that E_2 is unstable.

APPENDIX 3

SEX-LINKED CASE FOR SOME SPECIAL VALUES OF THE PARAMETERS (MODEL 9)

i) $\beta = 0, \alpha > 0$.

Equation (9.6) becomes

$$X' = p + \alpha \frac{W}{1 - W} pX \quad (1)$$

and, therefore, $X' > p$. It follows from (9.9) that

$$p_{n+2} \geq \frac{p_n + p_{n+1}}{2} \text{ for all } n. \quad (2)$$

Consequently $p_n > \min(p_1, p_0) \equiv \lambda$ for all n , i.e., p_n is bounded away from zero.

Now if W_n does not tend to zero, inspection of (1), using the fact that $\alpha > 0$, shows that

$$X_{n+1} \geq \gamma p_n \text{ for some } \gamma > 1, \text{ for infinitely many } n. \quad (3)$$

If (3) is satisfied for a given n_0 then because of (9.9) and (2) we deduce that

$$p_{n_0+1} \geq \frac{\gamma+1}{2} \lambda \text{ and, consequently, } p_{n_0+2} \geq C\lambda \text{ for } C = \frac{\gamma+3}{4}.$$

Referring to (2) we see that

$$p_n \geq C\lambda \text{ for all } n \geq n_0 + 2.$$

Repeating this argument we infer, provided (3) holds for infinitely many n , that ultimately

$$p_n \geq C^k \lambda \quad (4)$$

for n sufficiently large and any prescribed k . But $C > 1$ and (4) is absurd since p_n is bounded by 1 which implies that (3) is impossible.

Therefore, the only tenable inference is that $W_n \rightarrow 0$. It follows that $X_{n+1} - p_n \rightarrow 0$ and, consequently, $X_n \rightarrow 1, p_n \rightarrow 1$. The rate of convergence is easily established to be algebraic.

ii) $\alpha = 0, \beta > 0$.

It is easy to check that $X' < p$ and, therefore,

$$p_{n+1} < \frac{p_n + p_{n-1}}{2} \text{ for all } n.$$

Appropriate adaptation of the argument for case i) leads to the conclusion that $p_n \rightarrow 0, X_n \rightarrow 0$ and $W_n \rightarrow 1$, where convergence occurs at a geometric rate.

iii) $\alpha < 1, \beta = 1$.

Equation (9.6) becomes

$$X' = \frac{1 - (1 - \alpha)W}{1 - W} pX.$$

From this, since $p < 1 - W$, we infer that $X_{n+1} < X_n$ for all n , and hence $X_n \rightarrow 0$. Consequently $p_n = \frac{1}{2}(X_n + X_{n-1})$ also decreases to zero and $W_n \rightarrow 1$. The rate of convergence is geometric.

iv) $\alpha = 1, \beta < 1$.

By an argument more complicated than that of case (iii) it can be proved that $X_n \rightarrow 1, p_n \rightarrow 1$ and $W_n \rightarrow 0$, at an algebraic rate.

v) $\alpha = \beta = 1$.

The recurrence relations (9.1-9.3) become

$$X_{n+1} = \frac{p_n X_n}{1 - W_n}, \quad p_{n+1} = \frac{X_n}{2} + \frac{p_n X_n}{2(1 - W_n)}, \quad W_{n+1} = 1 - X_n \quad (1)$$

yielding

$$\frac{X_{n+1}}{X_n} = \frac{1}{2} \left(1 + \frac{X_n}{X_{n-1}} \right). \quad (2)$$

By the change of variables $u_n = X_{n+1}/X_n$, (2) becomes

$$u_{n+1} = \frac{1}{2} + \frac{1}{2} u_n$$

giving

$$u_n = 1 - \left(\frac{1}{2}\right)^{n-2} (1 - u_1) \quad (3)$$

where $u_1 = X_1/X_0$. Note that since $p_n < 1 - W_n$, $u_n \leq 1$.

Inserting (3) in (2) we get

$$X_{n+1} = X_n \{1 - \left(\frac{1}{2}\right)^{n-1} (1 - u_1)\}$$

leading to the explicit expression

$$X_n = X_0 \prod_{k=1}^{n-1} \{1 - \left(\frac{1}{2}\right)^{k-1} (1 - u_1)\}. \quad (4)$$

The product in (4) converges since $\sum_{k=1}^{\infty} (1 - u_1) \left(\frac{1}{2}\right)^{k-1} < \infty$. It follows that $\lim_{n \rightarrow \infty} p_n = \lim_{n \rightarrow \infty} X_n = \lim_{n \rightarrow \infty} (1 - W_n)$ exist where the limit values depend on the initial genotype frequencies. The limit value can be expressed in terms of theta functions.

It is worth remarking that for model 10, $\alpha = \beta = 1$, we get again a family of equilibria but the limiting values of genotype frequencies are simply $U_n \rightarrow U_0 + V_0$, $W_n \equiv W_0$.

vi) $\alpha = \beta = 0$.

This is the classic random-mating, sex-linked character. The diploid gene frequency is given, in the n^{th} generation, by

$$p_n = \frac{1}{4} p_0 + \frac{1}{2} p_1 + \frac{2}{3} (p_0 - p_1) \left(-\frac{1}{2}\right)^n.$$