A STUDY OF MORPH-RATIO CLINES

by

John A. Endler

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Any mistakes or omissions, of course, are entirely my own (as is the rest of the thesis!).

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John A. Endler
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SUMMARY

Geographic variation is a characteristic phenomenon of the majority of continental species. This study investigates the causes of regional differentiation within species, in particular the causes of sharp differentiation in the absence of barriers to gene flow and in the absence of spatially abrupt environmental changes. The problem is approached using single locus and oligogenic models involving a major gene whose genotypes are subject to one or more smooth selection gradients, and one or more minor genes which affect the fitness and mating patterns of the major genotypes. The effects of varying levels of selection and gene flow (including their absence), and varying deme sizes, are investigated using experimental populations of Drosophila melanogaster populations and computer simulation.

The major findings of this study are (1), gene flow may not be as important a contradifferentiating factor as it was often previously believed; (2), morphotones, or abrupt spatial changes in morph or gene frequency, may develop in the absence of barriers to gene flow, in the absence of spatially sharp environmental changes, and may develop along smooth selection gradients which are gentle enough so that it may be difficult to measure them in the field; (3), conversely, morphotones must not be interpreted as evidence for sexual, genetic, or physiographic isolation, secondary contact, or abrupt changes in the environment in the vicinity of the clinal discontinuity, in the absence of other evidence; (4), genetic sampling drift alone cannot explain large scale "area effects"; (5), simple models of coadaptive

modifiers in clines may produce any of the effects of interpopulation crosses and hybrid zones observed in the literature; and (6), certain combinations of coadaptive modifiers in clines may lead to hybrid zone effects and to the evolution of sexual isolating mechanisms. A general model of geographic differentiation and speciation mechanisms, and their interrelationships, are presented (figure 1-2). All the paths in the diagram are genetically possible, considering the effects of as few as four genes.

The results were not analytically precise, but they elucidate the requirements and some of the problems to be expected in studies of geographic differentiation. Without a thorough geographic study of the population biology and history of a species, one cannot say by what mechanisms populations have differentiated.

A STUDY OF MORPH-RATIO CLINES

> Those forms which possess in some considerable degree the character of species, but which are so closely similar to some other forms, or are so closely linked to them by intermediate gradations, that naturalists do not like to rank them as distinct species, are in several respects the most important to us. (Darwin, 1859, p. 47).

Chapter 1, Introduction

Geographic variation is found in nearly every group of organisms, and a given species, Rassenkreis, or Artenkreis may exhibit one or more of the following structural patterns:

1. Disjunction; one or more populations are geographically isolated from the rest of the taxon.
   a. disjunct undifferentiated: disjunct populations similar.
   b. disjunct differentiated: one or more of the disjunct populations are divergent from the others.
2. Overlap; one or more groups of populations are partially or wholly sympatric with others, without hybridizing within the area of sympatry.
3. Hybrid zones; narrow belts, with greatly increased variability in fitness and morphology, separating distinct groups of rather uniform sets of populations.
4. Conjunction; series of distinct but contiguous groups of populations which are not separated by hybrid zones.
5. Gradation; series of gradually changing contiguous populations.

Partly after Mayr (1963) and Huxley (1939). See figure 1-1.

Examples of each include (1), Bufo Microscaphus (Stebbins, 1966); (2), Ensatina eschscholtzi (Stebbins, 1948); (3), Crinia laevis (Littlejohn, et al., 1971); (4), Ambystoma tigrinum (Conant, 1958, Stebbins, 1966); and (5), Pseudacris triseriata (Conant, 1958, Stebbins, 1966).

FIGURE 1 - 1

THE DISTRIBUTION OF A HYPOTHETICAL SPECIES

SHOWING SEVERAL SUBSPECIES DISTRIBUTION TYPES

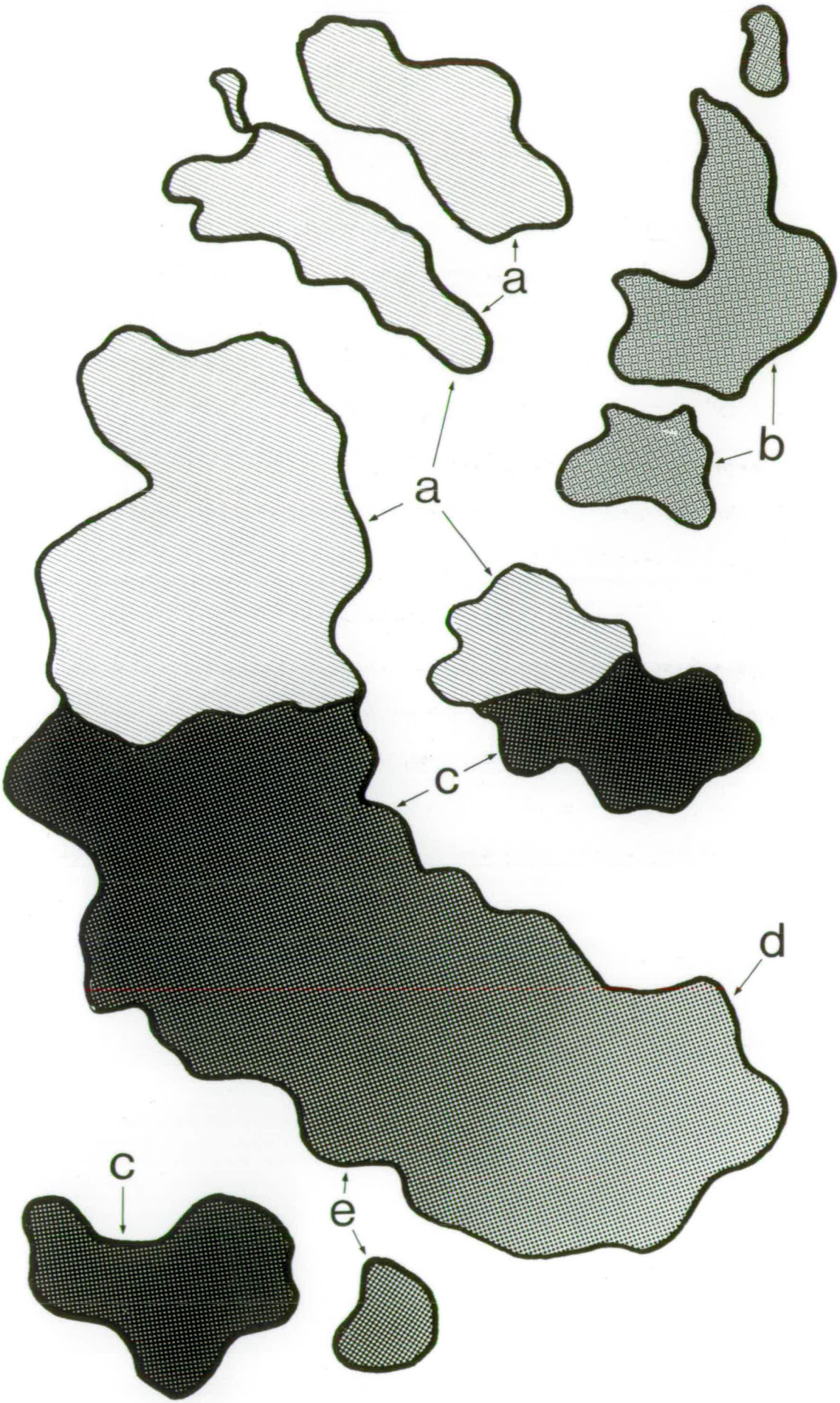
a. subspecies a shows several disjunct but not differentiated populations, and is in conjunction with subspecies c.

b. subspecies b is disjunct and differentiated from the rest of the species. It also shows some disjunct, but not differentiated populations.

c. subspecies c is in conjunction with a, but in gradation with subspecies d. A cline goes from c to d. Subspecies c shows three disjunct not differentiated populations, however, the population labeled e resembles the centre of the c - d cline. If only the disjunct e and c populations were present, we might think that e was disjunct differentiated from c.

d. subspecies d is the extreme part of the cline from c.

All of these distribution types may be found in almost any book of distribution maps in animals and plants.


a
a
b
a
c
d
c
e

The phenomenon of disjunction, or complete geographic isolation, is of considerable interest because it is almost universally believed to be a fundamental requirement for speciation. For that reason it has been thoroughly studied (Mayr, 1942, 1954, 1963, Huxley, 1942, Huxley, et al., 1954). The classical example of speciation in disjunct populations is that of Darwin's finches (Lack, 1947).

Overlap between groups of populations is usually presumed to mean, at least in the area concerned, that species-level isolating mechanisms have evolved, allowing the groups to invade each other's ranges without producing hybrids (Mayr, 1942, 1963). When no series of geographically contiguous populations unite them, the overlapping groups are regarded as biological species. When overlap is present in a continuous Rassenkreis - ring species - the status of the overlapping populations is open to dispute. For a discussion see Mayr (1942, 1963), Huxley (1940, 1942), and Rensch (1959).

Hybrid zones have been studied in greater detail in plants (Anderson, 1949, Stebbins, 1950, Grant, 1963) than in animals (Sibley, 1961, Short, 1965, Mayr, 1942, 1963, Remington, 1968), and represent a testing ground for any isolating mechanisms evolved in disjunction (Mayr, 1942, 1963), or by other means (Huxley, 1939, White, 1968). The fate of such zones will be either to broaden (introgressive hybridization), or, ultimately to disappear (completion of speciation). A classical example is the zone between the hooded and carrion crows (Meise, 1928).

Conjunction and gradation are distinguished by the slopes of clines connecting their component populations. A cline is a

gradation in a measurable character (Huxley, 1938); the steepness of a cline is a measure of the extent of geographic differentiation. Although conjunction and gradation are characteristic patterns of the majority of continental Rassenkreise (Mayr, 1942, 1963, Rensch, 1959, Udvardy, 1969, see also table 1-1), these clinal classes of geographic variation have received relatively little attention from students of adaptation. It is agreed that conjunct subspecies represent responses to local environments (Mayr, 1942, 1963, Huxley, 1940, 1942, Huxley, et al., 1954, Dobzansky, 1951, Grant, 1963), but lack of geographic isolation is thought to make them unimportant in speciation (Mayr, 1942, 1949, 1963). Gradation has been particularly neglected, probably because its presence is presumed to mean that gene flow is too strong, or environmental differentiation too weak to allow appreciable geographic differentiation of the genome to take place (Dobzansky, 1941, 1951, 1970, Mayr, 1940, 1942, 1949, 1954, 1963, Stebbins, 1950, Grant, 1963).

Are conjunction and gradation patterns really unimportant in speciation? Ring species and other extensively differentiated Rassenkreise have been known for a long time (Timofeeff-Ressovsky, 1940, Mayr, 1940, 1942, 1963, Fitch, 1941, Miller, 1931, 1941, 1951, Fox, 1951, Huxley, 1940, 1942, Rensch, 1959). In many cases the extremes do not successfully interbreed. If some catastrophy or change in climate resulted in the extinction of the intermediate populations, the survivors would be good biological species (Huxley, 1942, Rensch, 1959). But is full geographic isolation necessary? With few exceptions speciation has proceeded more rapidly in the centres of continents compared to more broken up areas (Dillon, 1970, Darlington, 1957).

Disjunction of range is rare in the oceans, and there is a dearth of evidence for geographic barriers (other than mere distance) playing any role in the speciation of pelagic and benthic marine organisms (Carter, 1959, Mayr, 1954b). The topographic and climatic diversity of the terrestrial world certainly provides many opportunities for classical allopatric speciation, but it does not exclude the possibility of, nor does it belittle the relative importance of speciation within a spatially continuous Rassenkreis.

Table 1-1 illustrates the frequency of types of geographic variation found within the ranges of the extensively studied North American amphibians and reptiles. Within the limits of accuracy of mapping and styles of taxonomy it should indicate general trends (see appendix 1, Rensch, 1959, Stebbins, 1966, and Udvardy, 1969). The frequency of disjunct populations which have <u>not</u> differentiated sufficiently to be given subspecific rank (1.a.) exceeds the frequency of the differentiated disjunct populations (1.b.). If allopatric differentiation was prevalent, then we would expect relatively more differentiated than undifferentiated disjunct populations, other factors being equal. Allowing for the fact that the contiguous class (4) may contain some unrecognized hybrid zones (3), we see that the number of species showing areal differentiation among spatially continuous populations is greater than those showing disjunct differentiation. Again, for the usual arguments about gene flow and differentiation (Mayr, 1963, Dobzansky, 1970), we would expect the disjunct differentiated class to exceed the continuous differentiated class.

TABLE 1 - 1

PATTERNS OF VARIATION IN NORTH AMERICAN AMPHIBIANS AND REPTILES

(Data from Conant, 1958, and Stebbins, 1966)
(See appendix 1)

Number of species, parts of ranges with subspecies showing:	Amphibians	Reptiles	Total
1. Disjunction,			
a. Undifferentiated			
in monotypic species	34	23	57
in polytypic species	28	48	76
total	62	71	133
b. Differentiated			
in polytypic species	24	25	49
2. Overlap	1	2	3
3. Hybrid zones	4	4	8
4. Conjunction	44	100	144
5. Gradation	16	33	49
Conjunction + gradation	60	133	193

Note: a polytypic species may fall into more than one category, as does the species shown in figure 1-1, see appendix 1.

Jain and Bradshaw (1966), Ehrlich and Raven (1969), and Antonovics (1971) review field evidence for extensive differentiation evolved without disjunction, and the experiments of Streams and Pimentel (1961), Thoday and his Colleagues (Thoday, 1958, Thoday and Boam, 1959, Thoday and Gibson, 1962, 1970, Millicent and Thoday, 1961), Dobzansky and his colleagues (Dobzansky and Spassky, 1967, Dobzansky, et al., 1969, 1970), and of the present author (chapter 3, Endler, 1973), show that quite marked differentiation may evolve quickly in spite of high levels of gene flow. This is supported by theoretical investigations by Jain and Bradshaw (1966), Slatkin (1971), Sokal and

Schnell (1971), and the present author (Chapter 2, Endler, 1973). Basically, it is geographical differentiation that is required to initiate the process of speciation (Mayr, 1942). Allopatry, per se may not be the exclusive prerequisite for speciation.

Huxley (1939), Timofeeff-Ressovsky (1940), and Mayr (1942, 1963) briefly mention the possibility of speciation taking place within a continuous parental species range. This form of speciation has been called semigeographic by Mayr (1942, 1963), semisympatric by Cain (1954), stasipatric by White et al., 1967, White, 1968, and Key (1968), and parapatric by Smith (1965, 1969) and Bocquet (1969). The diversity of names for the process stems partly from the confusion of several types of distributional patterns, and partly from the confounding of several modes of speciation (figure 1-2).

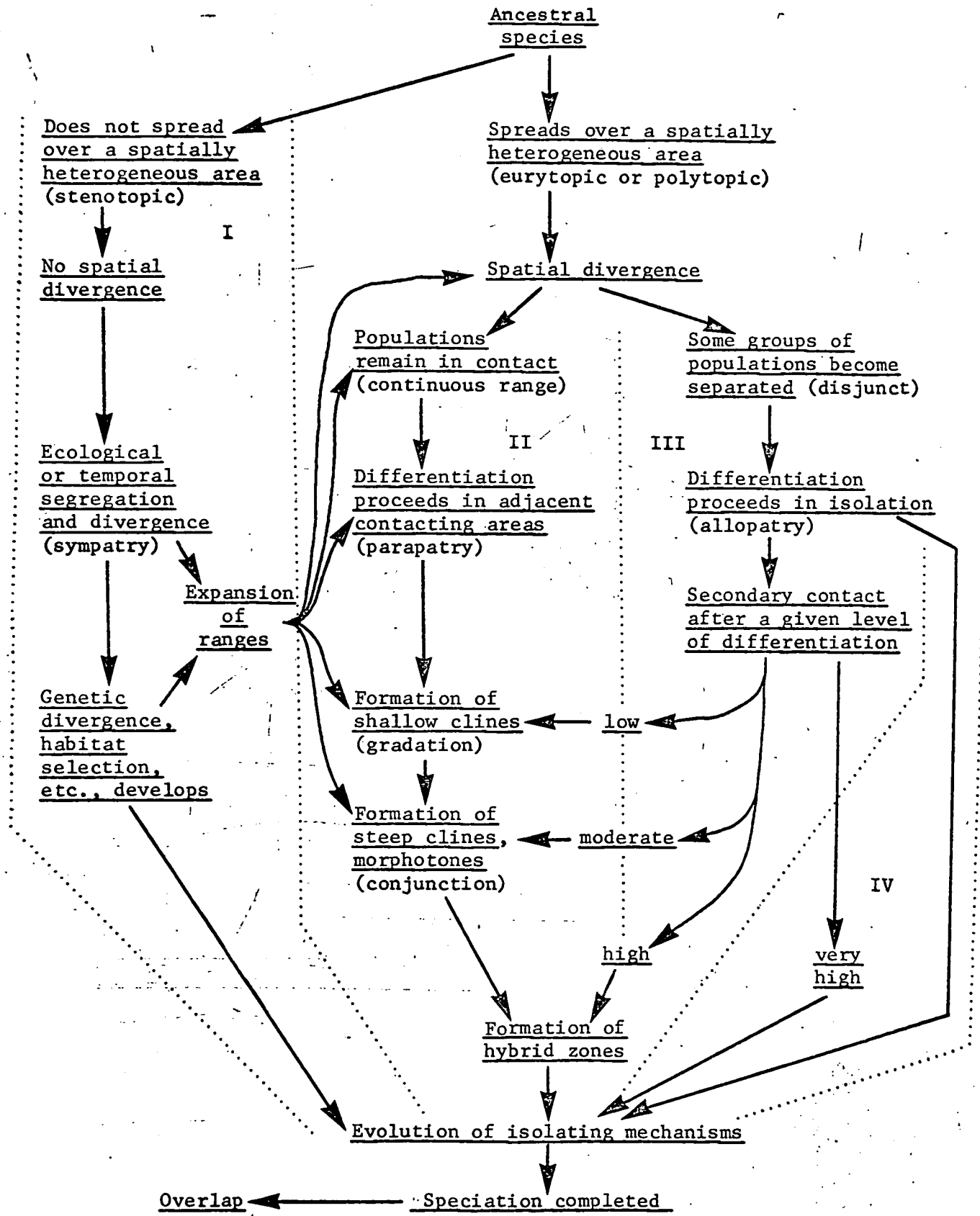
The steepest clines are often, but by no means always, associated with hybrid zones. Mayr (1963) distinguishes, in theory, two kinds of hybrid zone; those resulting from primary intergradation, and those resulting from secondary intergradation. Primary intergradation assumes that the species or Rassenkreis has not been subject to disjunction, but merely local differentiation of populations to that gentle clines become steeper (Mayr, 1963, Huxley, 1939, Fisher, 1930, figure 1-2-II). Although Huxley (1939, 1942) and Fisher (1930) suggest that the steepening of clines may give rise to isolating mechanisms (see below), Mayr (1963) is skeptical. In any case a steep cline should not be assumed to be a hybrid zone unless there is some evidence for increased variability of fitness and morphology in the steepest part of the cline compared to the flatter portions, and beyond that due to random effects. Until then, it should be included in the conjunct class. Primary intergradation is thus a synonym for clinal variation.

Figure 1 - 2

THE INTERRELATIONSHIPS

AMONG

PATTERNS OF GEOGRAPHIC VARIATION AND MODES OF SPECIATION



I: Sympatric speciation

II: Parapatric speciation

III: Allo-parapatric speciation

IV: Allopatri speciatio

Secondary intergradation is expected when two populations of a species or Rassenkreis have differentiated in disjunction, and have expanded to secondary contact and interbreeding as a result of changes in climate or adaptability (Mayr, 1963). If sufficient differentiation has occurred in isolation, interbreeding may result in the formation of unbalanced gene complexes in the hybrids (see especially Dobzansky, 1941 and Mayr, 1954). The consequent hybrid breakdown gives the characteristic increased variability of hybrid zones. This is secondarily hypothesized to result in a stable belt of intergrades (Mayr, 1942, 1963, Dobzansky, 1940, 1941, 1951, 1970, Goodhart, 1963). However, if there has not been enough differentiation during disjunction, the hybrids formed during secondary contact will not be "unbalanced" enough to produce a hybrid zone, and the zone of intermediates will expand until the geographic pattern is indistinguishable from conjunction or gradation (Mayr, 1963, figure 1-2,III, and chapter 2, below). In the field, all that we can measure is the steepness of clines and the variability of the inhabitants at various portions of the clines; without knowing the complete history of the area it is not possible to say whether a species has undergone primary or secondary intergradation. (See also Endler, 1973). It would therefore seem advisable to adopt a more descriptive and precise terminology (page 1-1) for geographic variation.

Figure 1-2 integrates and clarifies the classification of several modes of speciation postulated by various authors. There are four main classes: (I), sympatric; (II), parapatric; (III), allo-parapatric, and (IV), allopatric.

In sympatric speciation (I), there is neither spatial segregation nor spatial divergence. All members of the species in statu nascendi are within the "cruising range" of each other, and speciation is

postulated to take place through ecological or temporal segregation, host choice, habitat selection, etc. (Mayr, 1942, 1963, Cain, 1953, Maynard-Smith, 1966).

In parapatric speciation (II), both spatial segregation and spatial differentiation initiate the process, and lead to the evolution of isolating mechanisms between groups of geographically distinct but continuous populations. Although the differentiating groups of populations are in contact at their borders, this is not a form of sympatric speciation (Cain, 1953, Key, 1968), because only a very small fraction of the members of a given group are within "cruising range" of the others. Isolation is by distance rather than by geographical, ecological, or temporal factors (II, figure 1-2, Huxley, 1939, Timofeeff-Ressovsky, 1940, Mayr, 1942, White, et al., 1967, White, 1968, Smith, 1965, 1969). Thus parapatric speciation (II) is neither a special case of sympatric (I, as suggested by Key, 1968), nor of allopatric speciation (see Smith, 1969), but a process in its own right.

Parapatric speciation has been confused (as in Key, 1968) with allo-parapatric speciation (III). Allo-parapatric speciation differs from true parapatric speciation (II) in that the initial stages of spatial segregation and genetic divergence take place in disjunction, or allopatrically (figure 1-2, III), although speciation is completed in parapatry (Mayr, 1942, Dobzansky, 1941, Key, 1968). This is Key's modification of White's concept (White, et al., 1967, White, 1968) of stasipatric speciation. Allo-parapatric speciation (III) differs from allopatric speciation (IV) in that speciation is not completed until secondary contact is made, and in that the secondary contact does not necessarily lead to speciation, i.e. reversion to clinal variation is possible (II and III, figure 1-2).

In allopatric speciation (IV), all, or almost all divergence, segregation, and development of isolating mechanisms takes place while the differentiating population are geographically disjunct (Mayr, 1942, 1963). Disjunct populations will not revert to clinal variation if the geographic barrier breaks down, and secondary contact is not required to complete speciation in allopatric speciation (IV) (Mayr, 1942, 1963).

There are many branches in the paths to speciation, and therefore many possible alternate histories to a given geographic pattern. Most of the sympatry-allopatry controversy may be explained by the multifarious interpretations of each speciation mode (table 1-2), and because each author viewed, so to speak, a different portion of the diagram (figure 1-2).

TABLE 1 - 2

THE SYNONYMY OF THE MODES OF SPECIATION ILLUSTRATED IN FIGURE 1-2

I: Sympatric Mayr, 1942, 1963, Cain, 1953, 1954, Maynard-Smith, 1966, and other authors.

II: Parapatric Smith, 1965, 1969, Bocquet, 1969; =Stasipatric of White, et al., 1967, White, 1968; =Semigeographic of Mayr, 1942, 1963; =Semisympatric of Cain, 1954; and concepts of Huxley, 1939, 1942, and Timofeeff-Ressovsky, 1940. Misnamed "Sympatric" by Ford, 1964, 1971 (preoccupied by Sympatric Mayr, 1942.)

III: Allo-parapatric des. nov.; =Stasipatric of Key, 1968 (preoccupied by Stasipatric White, et al., 1967); =Semigeographic of Mayr, 1942, 1963; some concepts of Dobzansky, 1941, 1951.

IV: Allopatric Mayr, 1942, 1963; =Geographic of Mayr, 1942, 1963; =Geographic of Cain, 1953, 1954, and other authors.

Figure 1-2 indicates that clines are at the crossroads of several modes of geographic variation and speciation. If this is a reasonable picture of natural processes it suggests that clines would bear closer scrutiny. There are basically two questions to be investigated: (1), may sharp geographic differentiation evolve across a spatially and genetically continuous series of populations? (2), can the resulting steep clines give rise to hybrid zones?

In figure 1-2 we can discern two conditions which favour the development of clines; secondary contact of formerly disjunct differentiated populations (III), and implied spatial differences in the environments of continuous series of populations (II).

These may be further subdivided into the following:

TABLE 1 - 3

THE CAUSES OF CLINES

1. Secondary contact of populations which have differentiated in disjunction;
   a. by chance.
   b. adaptively.

2. Chance differentiation among continuous groups of populations;
   a. by recurrent mutation.
   b. by genetic sampling drift.
   c. by combinations of chance factors.

3. Adaptive differentiation among continuous groups of populations distributed along environmental gradients.

4. Adaptive differentiation among continuous groups of populations distributed across abrupt spatial changes in the environment.

Each of these possible causes has been invoked by various authors to explain different clinal phenomena, and often by different authors to explain the same phenomenon. Examples include clines in (1.a.), Cepaea, Goodhart (1963); (1.b.), Paradisaea, Mayr (1942); (2.a.), Cricetus, Timofeeff-Ressovsky (1940); (2.b.), Linanthus, Epling and

Dobzansky (1942); (3), Schizoporella, Schopf and Gooch (1971); and (4), Peromyscus, Dice (1939, 1940, 1941, 1947). In almost all cases the interpretation of the cline is open to considerable dispute (for example, Mayr, 1942, Gain and Currey, 1963a,b, 1968, Goodhart, 1963, Wright, 1965§ and Wolda, 1969a, for Cepaea nemoralis). Further examples are found in table 5-1, chapter 5).

There are many conditions classically favouring the development of clines, but the requirements for very steep clines are not at all clear. Secondary contact could result in a steep cline initially, but, under the influence of gene flow, it would soon decay into a shallow cline unless there was enough "hybrid breakdown" to form a hybrid zone, which bypasses question 1. There is considerable dispute about the possibility of nonadaptive differentiation (for an unusually unbiased view and review see Huxley, 1942 and 1954), but there is nothing to prevent gene flow from "swamping out" any chance spatial differentiation; nonadaptive clines would not be expected to be stable either. Gene flow is usually expected to prevent anything but smooth clines over smooth environmental gradients, and even to prevent sharp spatial differentiation across abrupt environmental boundaries, unless the magnitude of gene flow is not very great (Mayr, 1940, 1942, 1954, 1963, Dobzansky, 1940, 1941, 1951, 1970, Stebbins, 1950, Simpson, 1953, Grant, 1963, and many others). Thus, according to the classical view, we are left with either very sharp environmental differences and reduced gene flow, or a period of complete allopatry, to allow any extensive differentiation.

Many steep clines are indeed associated with a distinct environmental boundary. The best examples are associated with populations of small mammals living on soils of sharply contrasting colours (Dice, 1939a,b,c, 1940a,c, 1941, 1949, Dice and Blossum, 1939, Blair, 1940,

1943b, 1950), and in populations of grasses living on and off abandoned heavy-metal mines (Jain and Bradshaw, 1966, McNeilly and Antonovics, 1968, 1971, Antonovics and Bradshaw, 1970). Such clines are characterized by segments which are very much steeper than the rest of the cline; the flat portions between the local steepenings were called "steps" by Huxley (1939). It is usually assumed that the steepest parts of clines correspond with some abrupt environmental change if there is no evidence for secondary contact (see especially Mayr, 1963, and almost any discussion of geographic variation).

However, there are a number of situations in which local steepenings do not correspond with a known spatial change in the environment, as in Peromyscus, (Sumner, 1932) and Amathes (Kettlewell and Berry, 1961, 1969), or no abrupt spatial changes can be detected in the environment at all, as in Linanthus (Epling and Dobzansky, 1942), Lebistes (Haskins, et al.,1961), Cepaea (Cain and Currey, 1963a, 1968), and Partula (Clarke, 1968, 1971). In general, sharp environmental differences do not always result in steps, and steep clines are not always associated with distinct environmental heterogeneity or secondary contact. This suggests that, like the problem of "primary" and "secondary intergradation", there is not a unique one-to-one correspondence between the outcomes of postulated causes and the clines we observe in nature.

The primary questions to be considered in the present study of clines are:

1. may sharp geographic differentiation evolve across a spatially and genetically continuous series of populations?
   a. does gene flow really prevent spatial differentiation?
   b. do stepped clines require stepped environments?
   c. why is there not always a unique one-to-one correspondence between local steepenings and environmental changes?
2. can steep clines give rise to hybrid zones?

Chapter 2

PROPERTIES OF CLINES

"The gradient results partially from the dispersal of variants (genes) from one center toward another with lowered frequency of occurrence. The extreme groups in a gradient are, so to speak, partially isolated by their own population pressures. But the gradient also results from many local intermediate frequencies established in balance with intermediate environments."
(Alden H. Miller, 1941, p. 377).

"Spatial or geographic separation is an invaluable adjunct to racial differentiation, even though absolute discontinuity in population is not." (Alden H. Miller, 1951, p. 618).

Section 2.1. On Gene Flow

Gene flow counteracts the factors which favour geographic differentiation among populations (table 1-3), and the balance between these forces determines the slopes of the resulting clines (Fisher, 1930, 1950, Wright, 1931, 1943, 1969, Haldane, 1932, 1948, Huxley, 1939, 1942, Dobzansky, 1941, 1951, 1970, Mayr, 1942, 1963, Stebbins, 1950, Womble, 1951, Rensch, 1959, and others). Gene flow may be defined as the movement of genes and gene complexes into, and their establishment in, allochthonous gene pools. Gene flow must be distinguished from migration and from dispersal (table 2-1); neither migration nor dispersal necessarily lead to establishment of new genes or gene arrangements in a given gene pool.

Migration may be defined as the relatively long distance movements made by large numbers of individuals in approximately the same

Table 2 - 1

THE RELATIONSHIP BETWEEN MIGRATION, DISPERSAL, AND GENE FLOW

Characteristic	Migration	Dispersal	Gene Flow
1. Timing:	Periodic, or sporadic.	Continuous.	Continuous.
2. Time period:	Within a generation.	Within and between generations.	Between generations.
3. Units:	Groups of individuals.	Individuals.	Genes and gene arrangements.
4. Mode of travel:	Large numbers of individuals together in time and space.	Individuals separately.	Via individuals separately.
5. Distance travelled:	Very large.	Usually small.	Usually small.
6. Direction:	Unidirectional, or with a very strong directional bias.	Random and nondirectional or with a weak directional bias.	Random and nondirectional, or with a weak directional bias.
7. Establishment (successful breeding in the settling place):	None, or rarely*.	Sometimes*.	Always.
8. Habitat of new place already occupied by the same species:	Seasonally, or irregularly**	Not necessarily**.	Always.
9. Effect:	Avoidance of seasonally unfavourable habitat or climate.	Occupation of all available habitat, and reduction of local extinction.	Reduction of local inbreeding and of geographic variation.

Notes:

* If establishment it is also gene flow.
** May include range extensions of the species.

direction at approximately the same time, and is usually followed by a regular return migration, as in many butterflies (Williams, et al., 1942), fish (Heape, 1931, Hasler, 1954, Hagen, 1967, Harden Jones, 1968), amphibians (Twitty, 1961, 1966, Heusser, 1969), birds (Swarth, 1920, Heape, 1931, Landsborough Thompson, 1942, Van Tyne and Berger, 1961, Welty, 1962), and mammals (Heape, 1931), including lemmings (Kalela, et al., 1961). It is very common for migrating species to return to the same breeding locality although they travel long distances each year; this philopatry may apply to within the same few decimeters of the birthplace, as in Taricha torosa (Twitty, 1961, 1966), Bufo bufo (Heusser, 1969), Uria aalge (Southern, et al., 1965), or to the same stream segment, as in many species of Salmo (Harden Jones, 1968). Thus migration may be very great, but dispersal very small (table 2-1).

Dispersal may be defined as the roughly random and non-directional small scale movements made by individuals rather than groups, continuously rather than periodically, as a result of their daily activities (after Elton, 1927, Allee, et al., 1949, Andrewartha and Birch, 1953, and Johnson, 1969; see table 2-1). For the estimates of dispersal which have been made for various species of animals and plants, the relationship between a given distance and the probability that a given individual will travel that distance is leptokurtotic (Allee, et al., 1949, Andrewartha and Birch, 1953, Archimovitsch, 1949, Bateman, 1947a,b,c, 1950, Blair, 1943, 1960, Barber, 1965, Burla, et al., 1950, Colwell, 1951, Dice and Howard, 1951, Den Boer, 1971, Dobzansky and Wright, 1943, 1947, Ehrlich, 1961, 1965, Epling and Lewis, 1952, Gerking, 1959, Griffiths, 1950,

Hagen, 1967, Haskins, et al., 1961, 1971, 1972, Hewitt and Ruscoe, 1971, Hewitt, pers. comm., 1971, Howard, 1949, 1960, Jain and Bradshaw, 1966, Jenkins and Hassett, 1951, C.G. Johnson, 1969, R.F. Johnson, 1961, Kerster, 1964, Kerster and Levin, 1968, Lamb et al., 1971, Lamotte, 1951, Levin and Kerster, 1968, Miller, 1947, Mitchell, 1970, Mook, 1971, Roberts and Lewis, 1955, Rodeheffer, 1940, Wadley and Wolfenbarger, 1944, Wolfenbarger, 1946, and others). In other words, relative to the movement expected if individuals move at random, many more individuals than expected move small distances or do not move at all, fewer than expected move intermediate distances, and (in some cases) slightly more than expected move long distances (figure 2-1). The absolute numbers moving long distances are small, and their occurrence infrequent, for they are at the "tails" of the distribution, and for reasons to be discussed, are unlikely to become established.

Release experiments must be interpreted with caution; the disturbance caused by the introduction of large numbers of individuals into a population is likely to increase the activity of both aliens and residents. In addition, the increased crowding may increase the dispersal rate, inflating the estimate of the ordinary dispersal of the animals. The aliens are also more likely to move than the established population. (Dobzansky and Wright, 1943, 1947, Andrewartha and Birch, 1953, Johnson, 1969, Narise, 1968, Sakai, 1964, Narise [1968], Wallace, 1966a, 1968a,b, 1970, Den Boer, 1971, personal observations, and much of the literature on small mammal ecology). In general, the results of field studies and release experiments indicate that the vast majority of individuals move small distances relative to body size, during true dispersal.

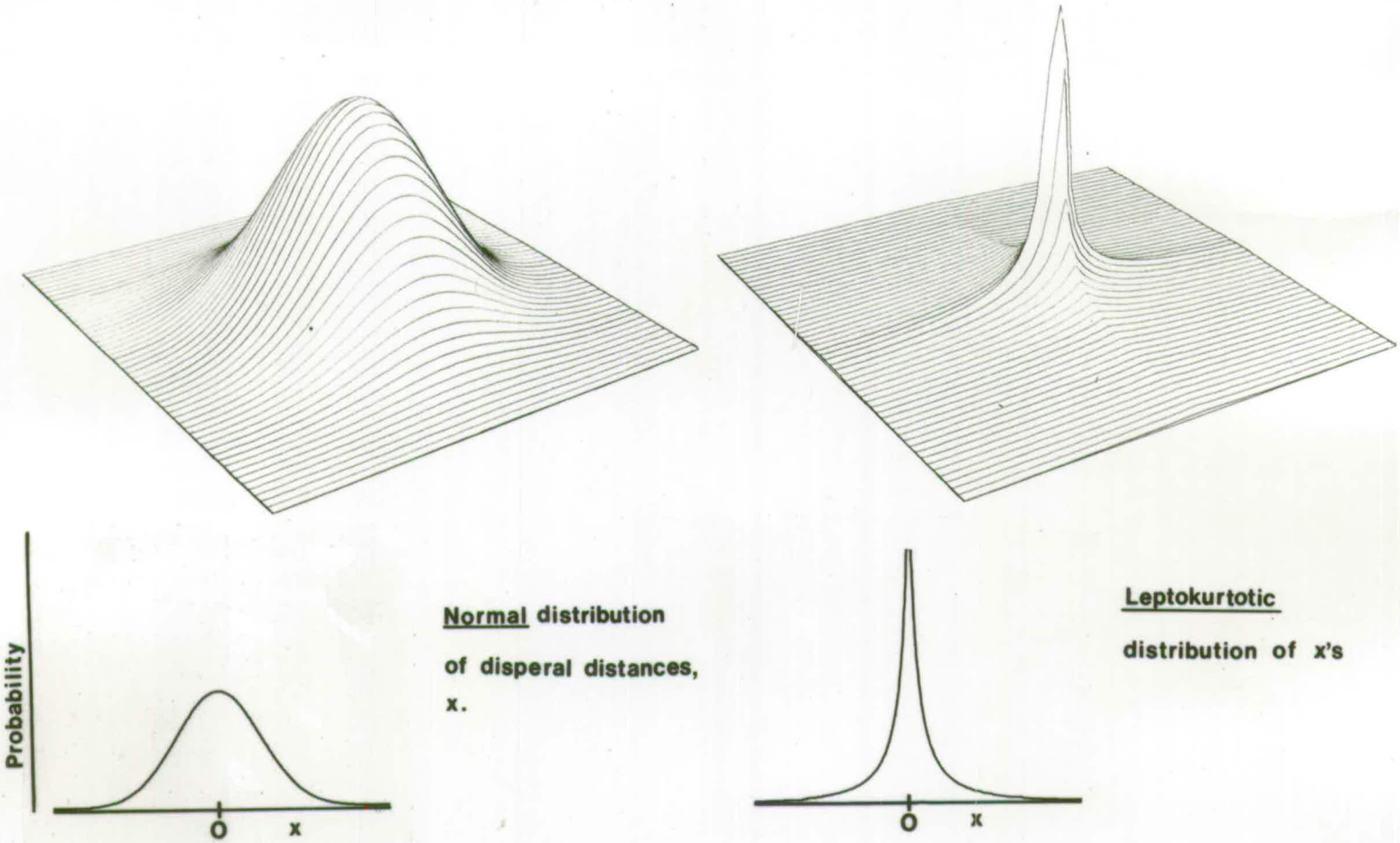

Probability
0
x
Normal distribution
of disperal distances,
x.
0
x
Leptokurtotic
distribution of x's

Figure 2-1

The average distances moved by individuals, or the average dispersal from a release point, can give a misleading picture if they are assumed to represent gene flow. Movement into a new area does not necessarily result in the establishment of the immigrant genes. (Epling, 1947, Kerster, 1964, Ehrlich and Raven, 1969). Field studies on small mammals (Anderson, 1964, 1969, Anderson and Hill, 1965, Blair, 1951, Calhoun, 1963, Christian, 1970, Crowcroft and Rowe, 1963, Dice, 1940a,b, Errington, 1946, 1963, Ingles and Biglione, 1952, Reimer and Petras, 1967, Selander, 1970), and Lepidoptera (Ehrlich, 1961, 1965, 1969, Labine, 1966, Kettlewell, et al., 1969) provide evidence that aliens, if they mate at all, are much less likely to reproduce successfully than natives for many reasons, including social ostracism, unfamiliarity with the new habitat (a "psychological barrier", Mayr, 1942), and slightly different microhabitat and microclimatic tolerances (see also Muller, 1952, Bush, 1969, and Lack, 1968). In addition to ethological and ecological factors, aliens may be genetically incompatible with the residents, further reducing the effective gene flow relative to dispersal. In seed plants, local differences in reproductive physiology (which are not necessarily genetic) may affect the ability of pollen from different areas to penetrate to the ovules. Small differences in the time taken from contact to fertilization could exclude the slower forms (Haldane, 1932).

Even if aliens and natives were equal in all other respects, individuals are more likely to mate, and are more likely to mate sooner, with nearby individuals than with those further away on the basis of probabilities of random encounter, If single matings are the rule this greatly favours close neighbors over aliens (Labine, 1964).

In the Great Tit (Perrins, 1965), the Kittiwake (Coulson and White, 1968), the Lesser Black-backed Gull (Brown, 1967), and the Manx Shearwater (Perrins, 1966), there is evidence that young from earlier matings have a greater probability of survival than those of later matings (see also Lack, 1966, 1968, and Labine, 1964, 1966). If this is generally true, then aliens which succeed in mating will be at an additional disadvantage because their offspring will hatch after the natives, if fertilization-egg laying times were equal and also equal mean arrival times. Immigrants from longer distances could have the additional problem of smaller food reserves which might further delay or reduce the efficiency of reproduction, and may also arrive later (Heape, 1931, Van Tyne and Lerger, 1961, Welty, 1962, Lack, 1966, 1968). This timing effect could also be effective in plants; pollen from neighboring individuals is likely to reach a given female earlier than pollen from the more distant populations, reducing or excluding gene flow from the more distant plants (Haldane, 1932, Stebbins, 1950, Grant, 1963, Briggs and Walters, 1972). Finally, the comparative rarity of introduced genes means that they may be lost by chance in the first few generations (Wright, 1931, Fisher, 1930). Gene flow is probably considerably more spatially restricted than dispersal, and is certainly more restricted than migration.

The exceptions, such as the Lincoln Sparrow (Miller and McCabe, 1935), many duck species (Salomonsen, 1955, Welty, 1962, Udvardy, 1969), and many marine organisms with one or more pelagic stages (Eckman, 1953, Hubbs, 1957, Sears, 1959, Udvardy, 1969) do not usually exhibit well marked geographic races (but, see Mayr, 1954b). If they do, strong selection seems to be operating (for example, in Echinus, Nichols, 1962). However, dispersal may be restricted even

in organisms that are apparently subject to passive transport. Directional factors such as oceanic currents or prevailing winds, may favour dispersal to particular areas. Harden Jones, in a splendid review of fish migration (1968), points out that there is evidence that the oceanic currents may actually be used by several fish species (some salmon, herring, and possibly eels) to return to their birth-place to spawn! (See also, I.C.E.S., 1969). Passive transport does not necessarily mean random transport; by analogy, passengers are not dispersed at random by trains, a variety of cues tell them when to get on and off.

Genetic measures of gene flow also present difficulties. Selander (1970) has pointed out that using the Wahlund effect (see Wallace, 1968b) to estimate the amount of subdivision of populations (e.g. Rasmussen, 1964, Petras, 1967, Anderson, 1964), or simply the distribution of gene frequencies (Wright, 1943b, Lewontin, 1962, Lewontin and Dunn, 1960), can yield misleading results because of the presence of "silent" or "null" alleles, or of excessive heterozygote frequencies, or as a consequence of lumping samples from different times of year. In addition, such measures are very sensitive to the quadrat size in which the genotype frequencies are calculated (see Pielou, 1969, and Wright, 1951). Calculating the relation between allelism and distance (Wright, et al., 1942, Wallace, 1966b) is very likely to give an inflated estimate of mean gene flow distance because the"tail" of the leptokurtotic distribution of dispersal distances ensures that a given allele will be present over a broad area although its frequency will be high only over a very small area; about 95% of all movements are over short distances. There is no substitute for following the dispersal and mating of undisturbed individuals

(Howard, 1949), or of introduced markers in undisturbed natural populations (Haskins, et al., 1961). Wright and Dobzhansky (1943) mention that the frequency of orange in Drosophila pseudoobscura in their experimental area was above the average natural frequency one year after their famous experiment. Unfortunately they did not study the rate of spread in subsequent years; that would have given a far superior measure of gene flow than the published experiment.

An additional factor must be considered. Very few organisms live as continuously distributed populations. They are usually clumped in subareas of favourable microclimate and microhabitat, separated by subareas of low population density. Breeding areas are usually even more localized (Allee, et al., 1949, Anderson, 1964, Andrewartha and Birch, 1953, Blair, 1943, 1947, 1950, Christian, 1970, Dice, 1940a,b, Diver, 1940, Dobzhansky, 1941, Ehrlich, 1961, 1965, 1969, Ehrlich and Mason, 1966, Ferrell, 1966, Gerking, 1959, Grant, 1963, Hagen, 1967, Haskins, et al., 1961, Haskins, pers. comm., 1971, Hesse, et al., 1937, Hewitt and John, 1970, Hewitt, pers. comm., 1971, Kettle, 1951, Howard, 1949, Marshall, 1948, Mayr, 1942, 1963, Miller, 1941, 1947, 1951, 1956, Timofeeff-Ressovsky, 1940, White, 1968, White, et al., 1964, 1967, Wolda, 1969a,b, Udvardy, 1969, Voipio, 1952, and many other field studies). This clumping amplifies the ecological and ethological restrictions to gene flow, and reduces the number of settling places where establishment would be possible in the absence of other limits. The exceptions, for example, some plants (especially palaearctic grasses and conifers), are either not sharply differentiated spatially, or the differentiation can be shown to result from intense selection pressures (Stebbins, 1950, Grant, 1963, 1971, Jain and Bradshaw,

1966, Antonovics, 1972, Briggs and Walters, 1972). Generally, gene flow is restricted both in distance and in the location of successful establishment to a reticulation of favourable localities, and is spatially restricted compared with dispersal and migration.

In summary:

1. Dispersal is restricted compared with migration.
   a. there is usually a return migration.
   b. birthplace philopatry is common.
2. Dispersal is usually leptokurtotic or very restricted.
3. Gene flow is restricted compared with dispersal.
   a. gene flow requires establishment of alien genes.
   b. numbers and frequency of long distance travellers are very low.
   c. random encounters favour the nearby as mates, or the nearby as earlier mates.
   d. ethological reduction of aliens' reproduction.
   e. ecological and physiological reduction of aliens' reproduction.
   f. chance loss of introduced genes or gene arrangements.
4. Microdistribution is patchy.
   a. in population size or density.
   b. in breeding or egg laying sites.

These factors favour geographic differentiation, because they increase genetic isolation between areas (Wright, 1940b, Mayr, 1942), even though the movement among these areas may seem large. The deme concept (Gilmour and Gregor, 1939, Huxley, 1939, Gregor and Watson, 1961) although originally conceived for plants, is a good approximation to the population structure and gene flow patterns of most animals.

Section 2.2 A Model for the Study of Clines

In approaching the problem of distinct geographical differentiation in genetically continuous areas it will be helpful to consider models as well as field data. "Models can be viewed as selective approximations, which, by the elimination of incidental detail, allow some fundamental, relevant, or interesting aspects of the real world to appear in some generalized form." (Haggett and Chorley, 1967). A good model should be suggestive, or "one with implications rich enough to suggest novel hypotheses and speculations in the primary field of investigation." (Black, 1962). "A model must be simple enough for manipulation and understanding by its users, representative enough in the total range of implications it may have, yet complex enough to represent accurately the system under study (Chorafas, 1965). Finally, it should be reapplicable to the real world, that is, not only describe, but predict in a manageable way (Haggett and Chorley, 1967). Thus "a bad model would be heavily symbolic, present an overly formalized view of reality, be much oversimplified, represent an attempt to erect a more exact structure than the data allows, and be used for inappropriate prediction." (Haggett and Chorley, 1967, after Kaplan, 1964).

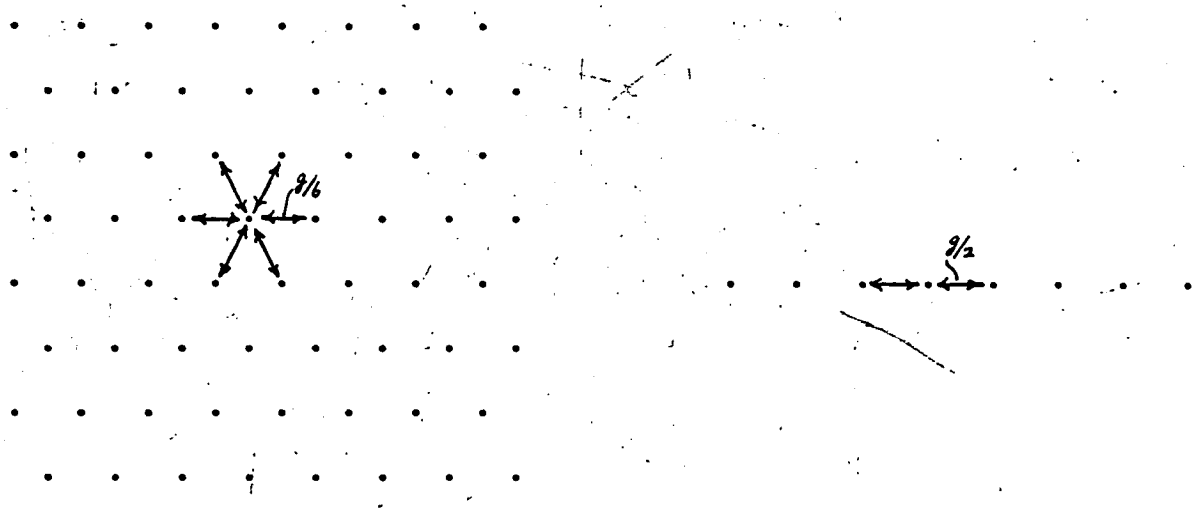
At the simplest level, geographic variation is characterized by spatial differences in gene frequencies. "An analysis of the formation of the geographical races and species ought to begin with a study of the behaviour of the single characters distinguishing the different forms from each other. Only subsequently can one study the interaction of the unit-character in the complex systems representing the types with which taxonomy is primarily concerned" (Dobzansky,

1933, p. 124). For a polymorphic character (sensu Ford, 1940, 1945), a morph-ratio cline is a spatial gradient in genotype or gene frequencies. There are numerous examples in nature, although few have been analyzed throughly (see marked papers in the reference list and Chapter 5). Morph-ratio clines will be useful models in the study of the causes of distinct geographical differentiation.

In order to understand the properties of morph-ratio clines it will be helpful to use a model of population structure which is flexible and takes account of what is known of natural populations. The remaining part of this section describes the assumptions and mechanics of the model. Section 2.3 discusses some salient considerations which are too often neglected in cline studies; section 2.4 presents the results for genetic sampling drift; section 2.5 the results for environmental gradients; section 2.6, for abrupt environmental changes, and section 2.7 the results for secondary contact. Finally, a brief summary is provided in section 2.8. Results of the models were obtained by simulation.

We will consider a diploid species distributed as a series of demes. A deme may be regarded as a spatially discrete breeding unit; an effectively panmictic aggregate of organisms lasting for at least one breeding session, and connected by gene flow with the neighboring demes before and after reproduction. The deme model is similar to Wright's neighborhood concept (Wright, 1940a,b. 1943, 1946, 1969), Dobzansky and Wright, 1943, 1947), but takes account of the microdistribution and limited gene flow patterns observed in nature (section 2.1). The demes will be assumed to make up a hexagonal grid in two dimensions; each deme has six adjacent neighbors (fig. 2-2a). This has been shown to be a better approximation for two

dimensional dispersal than the square grid (Cole and King, 1968, Chorley and Haggett, 1967). A linear transect in which each deme has two adjacent neighbors will also be considered (fig. 2-2b). The amount of gene flow per generation will be represented by g, the fraction of each deme which is exchanged with the other demes (g is used rather than m because m may imply migration). Thus g represents the probability that a given individual in deme x will leave deme x. Gene flow will be to adjacent demes only, for the reasons discussed in section 2.1; this type of model has been termed the "stepping stone" model by Kimura (1958).



a, hexagonal array (size 8 x 8) b, transect (size: d = 8)

Figure 2 - 2. Distribution of demes in the author's models for size (d) 8.
Arrows represent gene flow between a given deme and its neighbors.

Each deme is assumed to be initially polymorphic for an autosomal locus with two alleles, A and a, and will be assumed to have initially a 50% frequency (p) of gene A. For the purposes of the subsequent models, the initial gene frequencies are not important

because only simple selective models will be considered. Each deme will have a population size of N. There is a dearth of evidence for a relationship between gene frequency and population size (Thoday, 1963, Tamarin and Krebs, 1969), other than that resulting from genetic sampling drift (Wright, 1931, 1969), although it is theoretically possible (Clarke, 1972). Therefore population size, N, in each deme will be supposed to remain temporally constant.

Equilibrium (if any) will be attained after many generations of the sequence: mating, selection (if present), and gene flow. Gene frequencies at equilibrium, $\hat{p}$, are measured after gene flow; this will bias the models in favour of the effects of gene flow (see, for example, generation 50 in figure 2-10). The final gene frequencies were obtained by simulations on an I.C.L. 4-75 computer at the Edinburgh Regional Computing Centre, using programmes written in IMP language (see appendix 2, and Whitfield, 1969). Section 2-3 will deal with three important preliminary points. Sections 2-4 through 2-7 will discuss the clines that may result from genetic sampling drift, environmental gradients, environmental boundaries, and secondary contact, and will review the models of other workers. Data from the field will be discussed wherever relevant material has been published.

Section 2.3. Preliminary Considerations

2.3.1. Nomenclature. Several terms have been used to describe spatially sharp geographic differentiation in clines; steps (Huxley, 1939, 1942, and others), local steepenings (Clarke, 1966, and others), subspecies boundaries (Mayr, 1942, and most taxonomists, see also Bigelow, 1965 and Remington, 1968), genetic discontinuity (Crosby, 1969), secondary contact or intergradation (inappropriate, see chapter 1), hybrid zone (inappropriate, see chapter 1), or simply steep clines (most studies of geographic variation). The problem with most of them is that they imply a particular cause, or the existence of a particular genetic structure. For example Huxley's term step implies a discontinuity as does Crosby's term. The term step was originally intended to refer to a relatively stable portion of a cline, and not to the part separating the stable areas (Huxley, 1939). Local steepening may be taken to mean a cline in the process of steepening locally as well as being locally steep. Other terms are rather ponderous. Morphotone will be used as a purely descriptive term to denote a relatively abrupt spatial change in morph or gene frequency, just as Ecotone is used to denote a sharp transition in ecology (Allee, et al., 1949).

2.3.2 Measurement. How can one quantitatively describe sharp spatial differentiation, or quantitatively describe the distinctness of a morphotone? This is a statistically unsolved problem. Wright (1931, 1943, 1946, 1965, 1969) and most recent workers studying differentiation under genetic sampling drift (see section 2.4) have used the differentiation ratio, $\sqrt{F} = \sigma_p / \sqrt{(\bar{p}(1-\bar{p}))}$, or its equivalent, where σ_p

is the between-deme (or between-neighborhood) standard deviation of gene frequency and $\bar{p}(1-\bar{p})$ is the expected between deme standard deviation given the mean of all the demes' gene frequencies, $\bar{p}$. The greater the differentiation of gene frequencies, the larger is this ratio. However, given an area (say) which has differentiated into two subareas of high and low gene frequency (fig. 2-3a), it is possible to rearrange the location of the demes without changing the gene frequencies so that there is no spatial differentiation, but the differentiation ratio has the same value (figure 2-3b). A high differentiation ratio does not necessarily imply clines (Wright, 1943). An additional problem is that the differentiation ratio is dependent upon the mean gene Frequency of the sample.

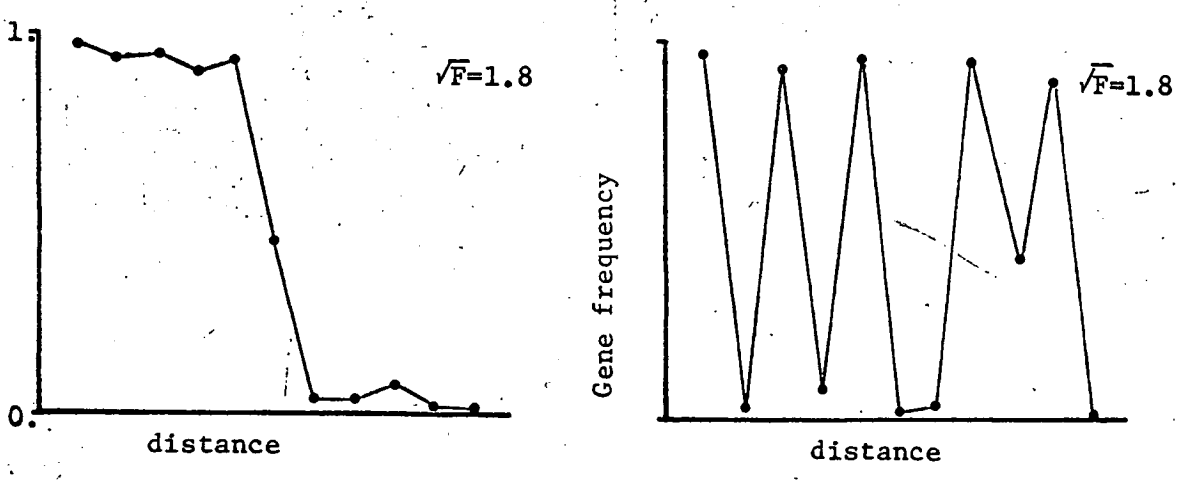


Figure 2-3. The difficulty of the differentiation ratio ($\sqrt{F}$).

Kimura and Weiss (1964), also working with random drift, have suggested measuring the decrease of genetic correlation with distance. If groups of demes have changed in gene frequency together, then the decrease of genetic similarity with distance will not be as rapid, for distances less than the radii of the differentiated areas, than it would be if there was no differentiation. Further away, the genetic

correlation should fall off more rapidly with distance. Unfortunately, for this measurement to work at all, the differentiated groups must be roughly equally spaced as the spatial correlations must be done between all possible pairs of demes; irregular spacing of differentiated groups of demes may not appear different, by this measure, from randomly differentiated single demes. There is no substitute for mapping the geographical distribution of gene frequencies in each deme and working from there.

It is possible to define an arbitrary threshold gene or morph frequency, and then calculate the mean area of groups of adjacent demes or samples beyond the threshold. If demes have differentiated independently the mean area of these groups will be very small; if groups of demes or neighborhoods have differentiated together the mean size will be large. Although a dependable measure, it is as arbitrary as the thresholds chosen, and requires extensive sampling. Furthermore, it does not reveal the presence of morphotones.

The slope of a cline is a measure of the sharpness of differentiation between the two areas it separates, but it is not always clear at what portion of the cline on which to do the calculation. A cline may be sigmoid, as in several colour and pattern morphs of Tisiphone abeona (Lepidoptera) in eastern Australia (Lucas, 1969), figure 2-4.

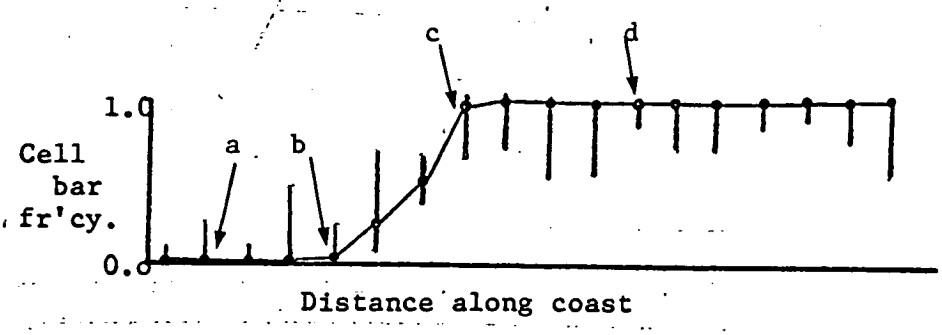


Fig. 2-4. A Sigmoid Cline (Lucas, 1969)

Clearly, taking the average slope between points a and d would indicate a flatter cline than if the slope was calculated between b and c. If samples were roughly equally spaced, then a crude measure could be the slope of the steepest part of the cline, taking only adjacent samples which are significantly different from each other. However, this looses information, and may give improper results if there is much between (adjacent) sample variation, as in the purple shell frequency cline in Partula taeniata described by Clarke (1968), fig. 2-5.

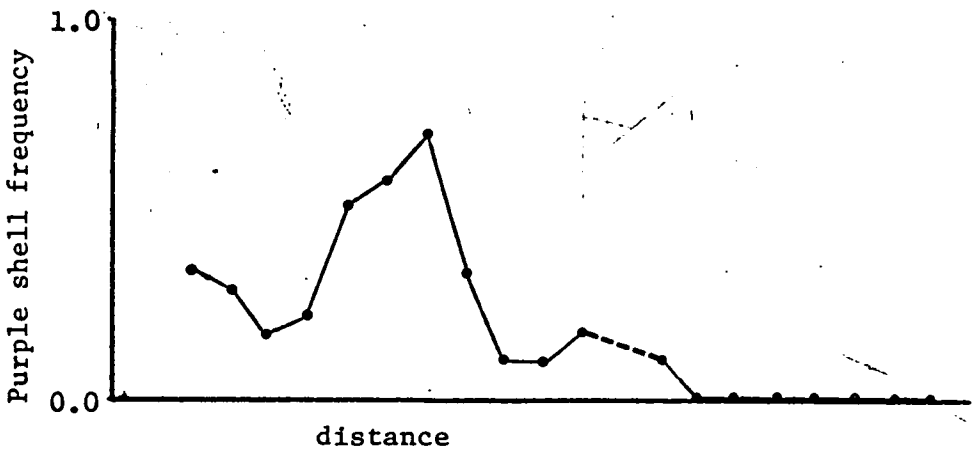


figure 2-5. A cline with high between sample variation (Clarke, 1968).

Specifying a morphotone is particularly difficult if the rapid change-over appears to take place between only two adjacent sample points, as in the banded shell frequency in Partula taeniata (Clarke, 1968), and yellow shell frequency in Cepaea nemoralis (Wolda, 1969b), figure 2-6.

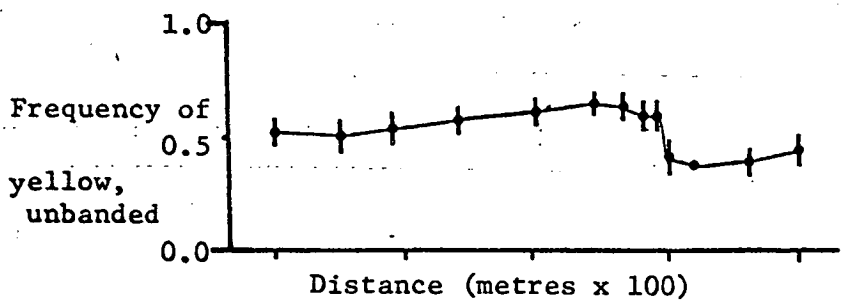


Figure 2-6. A morphotone between two sample points only (Wolda, 1969b)

For this study of the causes of morphotones, the criterion described by Matthews will be sufficient. By the Matthew's criterion, a morphotone falls into one of three classes: (I), "not obvious"; (II), "obvious", and (III), "bloody obvious" (Matthews, 1966).

2.3.3 Gene and Genotype frequencies. Unless one knows the genetics of a polymorphism, all one can measure are polymorph frequencies. An important consequence of diploidy and the Mendelian relationships in populations is that a small spatial change in gene frequency may be accompanied by a large morphotone in genotype or phenotype frequency. The data for the cline in melanism of Amathes glareosa (Kettlewell and Berry, 1961, 1969, Kettlewell, et al., 1969) provide a good example, figure 2-7.

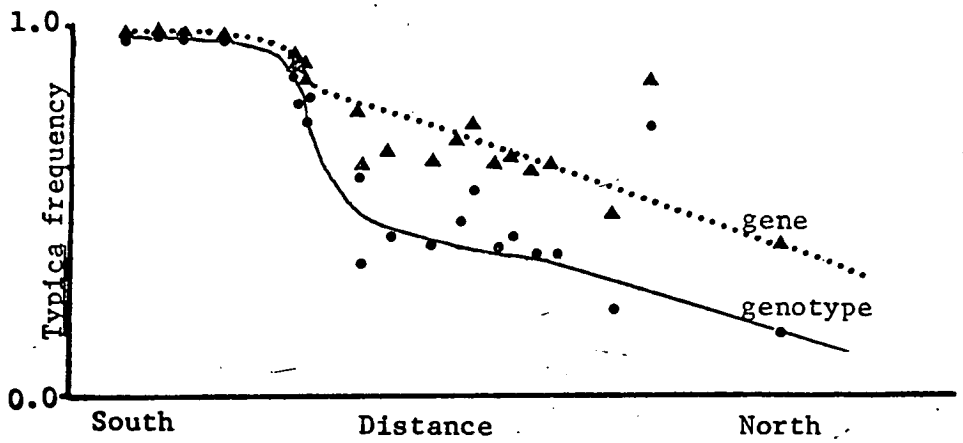


Figure 2-7. Morph frequencies and "gene" frequencies in Amathes glareosa (Kettlewell and Berry, 1961, 1969).

As a simple numerical example, consider a smooth cline for the gene frequency of a bi-allelic locus. The corresponding clines in genotype frequency (assuming Hardy-Weinburg ratios) show a very steep segment in some areas, and a gentle slope in others (figure 2-8).

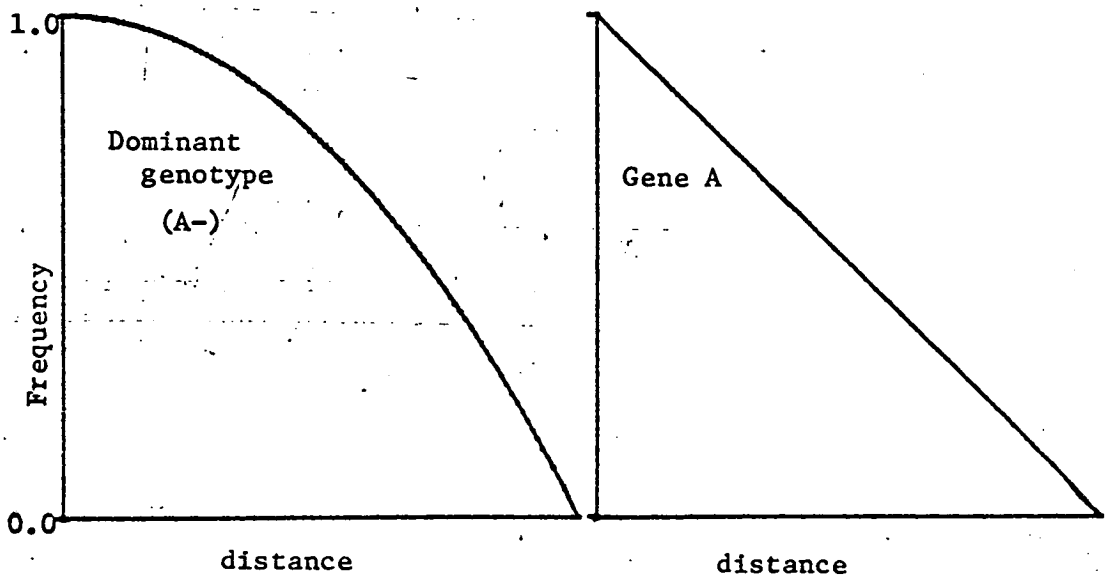


Figure 2-8. Genotype and gene frequencies of a single locus.

Thus genotypes may be differentiated strongly among nearby demes even though gene frequencies are not.

Section 2.4. Genetic Sampling Drift.

The relative importance of genetic sampling drift in natural populations is the subject of a controversy which is remarkable for its temporal stability and its lack of resolution. Huxley (1942) presents one of the most unbiased discussions of Wright's famous theory (Wright, 1931, 1940a,b. 1969); more recent, but biased, views may be found in Ford (1964, 1971), Sheppard (1967), Manwell and Baker (1970), and Wright, (1969).

The fundamental assumption of the genetic drift hypothesis, exactly equal mean survival rates among the genotypes, is often made when one does not know, or cannot measure any selective differences, or one has not measured and cannot conceive of any reason for selective differences. Examples include discussions of polymorphisms for flower colour in Linanthus parryae (Epling and Dobzansky, 1942), for shell colour and pattern in Cepaea nemoralis (Lamotte, 1951, Dobzansky, 1941, Mayr, 1942), and almost all instances of protein polymorphisms. However, there are differences in soils in the range of Linanthus, although brushed aside by Epling and Dobzansky (1942), and seed storage makes the effective population size large for drift (Epling, et al., 1960). Differential selection has been demonstrated for the polymorphisms in Cepaea (Cain and Sheppard, 1954), and in some protein polymorphisms (e.g.Koehn, 1969, Schopf and Gooch, 1971). However:

> "Very often, a mode of selection is inferred from an observed pattern of polymorphisms in natural populations. Thus if the same pair of alleles are found in uniform frequencies over wide distribution range of the species, it is claimed that natural selection is actively maintaining these frequencies. If, on the other hand, different alleles are fixed in different local populations, or if there is a cline, these are often considered to be the result of local adaptation of these alleles. Furthermore, if the frequencies of alleles are uniform within each locality but different among localities, this is also assumed to indicate some form of 'balancing selection'.
>
> Actually, selection can be invoked to explain any pattern of polymorphism in natural populations. Often, such presumed selection is used to refute the neutral polymorphism theory." (Kimura and Maruyama, 1971, p. 125).

Of course, the same criticism may be leveled at those assuming that the variation is a result of genetic drift in the absence of other evidence. Although the fundamental assumption of the genetic drift hypothesis is unlikely to hold for many generations, it is still important to ask the question, can genetic drift give rise to stable morphotones?

Wright (1931, 1940a,b, 1943a, 1946, 1965, 1969) has shown that spatially limited gene flow combined with genetic sampling drift can give rise to marked "differentiation". By "differentiation" he means that the frequency distribution of gene frequencies throughout the (theoretical) species range is rectangular to U-shaped (fig. 2-9), and adjacent neighborhoods may have greatly differing gene frequencies (Wright, 1931, 1943, and implicit in all his subsequent papers, and in papers quoting Wright). As pointed out in section 2.3, this does not necessarily mean that areas containing many neighborhoods or demes have differentiated from other such areas, but only that high and low gene frequencies are at least as common as intermediate gene frequencies. Thus clines, and clines containing morphotones are not necessarily found in species exhibiting a high degree of "differentiation" in Wright's sense, although they are more likely in

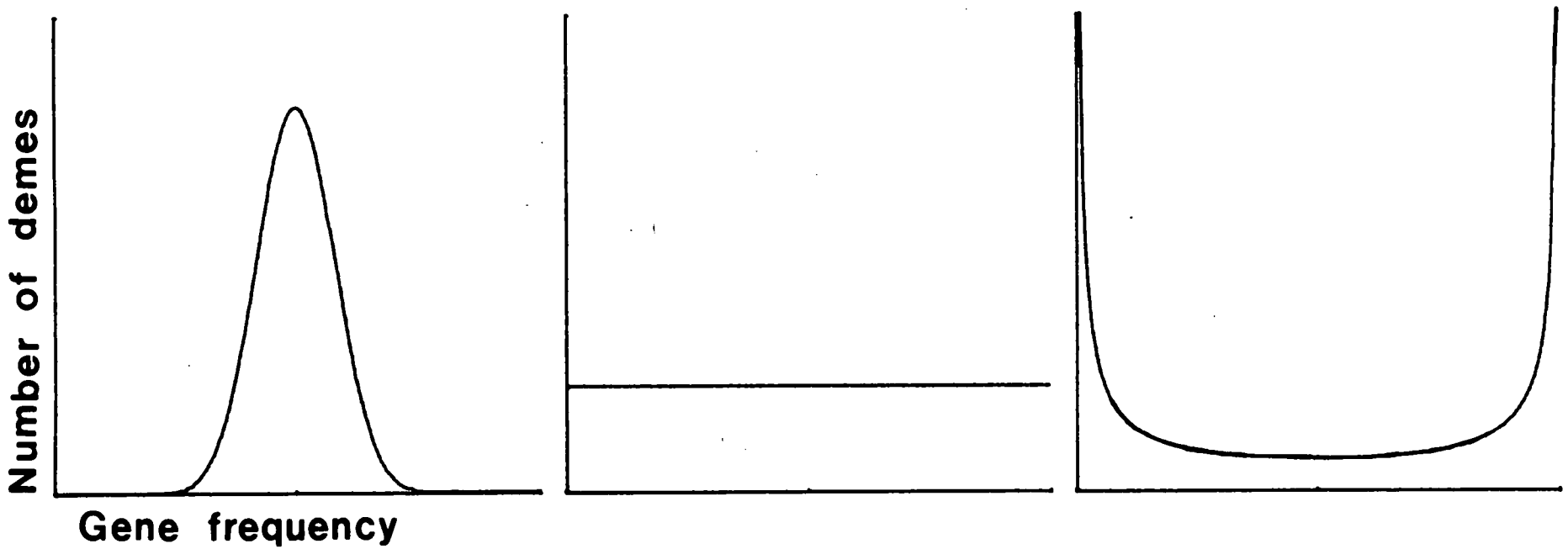

Number of demes
Gene frequency
"LITTLE" DIFFENTIATION
"IMPORTANT" DIFFERENTIATION
"SIGNIFICANT" DIFFERENTIATION

Figure 2-9

such species.

The gene frequency, p'_x, in deme x, after gene flow, is the mean of the gene frequencies of the residents and the aliens contributing to the breeding group, weighted by the gene flow rate, or:

$$p'_x = \underset{\text{residents}}{p_x (1 - g)} + \underset{\text{aliens}}{\bar{p}\, g} = p_x - g (p_x - \bar{p}) \qquad (1),$$

where g is the fraction of the deme or neighborhood which is replaced by aliens during one generation of gene flow, p_x is the gene frequency of the deme before gene flow, and $\bar{p}$ is the gene frequency of all breeding aliens. (Gene flow is represented by g and not m because m may connotate migration, see section 2.1). From (1), the change in a given deme's frequency, Δp_x, per generation, is:

$$\Delta p_x = -g (p_x - \bar{p}) \qquad (2),$$

if there is no differential emigration among genotypes (after Wright, 1931). Thus, both the rate of gene flow, g, and the difference between the gene frequencies of aliens and natives, changes the gene frequency after gene flow; Δp_x will be small if either g or $(p_x - \bar{p})$, or both are small. Any differences among demes participating in gene flow increases their Δp's, reducing $(p - \bar{p})$ the following generation. Therefore demes participating in gene flow tend to become more similar to each other than those not exchanging genes, or those exchanging genes at a lower rate. Aliens come primarily from adjacent neighborhoods or demes; those from longer distances are rare and contribute little to $\bar{p}$ (section 2.1). As a result adjacent demes may be similar but distant demes may be very different. If gene flow can come from anywhere in the species range with equal probability, as in the

Squaw Duck (Salomonsen, 1955), $\bar{p}$ is equal to the mean gene frequency of the species, $\bar{p}_s$. In such a case, if a deme's gene frequency (p_x) happens to be markedly different from $\bar{p}_s$, then ($p_x - \bar{p}_s$) and Δp_x will be large, and in subsequent generations, p_x will approach $\bar{p}_s$ (in the absence of differentiating factors) as gene flow reduces the differentiation of that deme. As long as gene flow is spatially restricted, isolation by distance allows populations to remain differentiated.

To include the effects of genetic sampling drift, a drift term is added to (2):

$$\Delta p_x = -g (p_x - \bar{p}) + \delta(p_x, N_x) \qquad (3).$$

The drift term, $\delta(p_x, N_x)$, is a randomly determined variable with a mean of zero, and a variance of $p_x(1-p_x)/2N_x$ where N_x is the effective population size of deme or neighborhood x, and p_x is the deme's gene frequency before genetic drift (Wright, 1931, 1943, 1946). Thus the greatest variation in changes in gene frequency occurs by chance in small populations with intermediate gene frequencies, in addition to the effects of gene flow. We are here assuming that genetic drift takes place before gene flow, and the measurement of the new gene frequency is made after gene flow. Equation (3) also assumes that the gene frequency of the natives which do not emigrate is the same as the deme's gene frequency before gene flow. This commonly made assumption gives rise to an inaccuracy which will be discussed later in this section.

The mathematics of the joint effects of genetic sampling drift and gene flow are complicated and require many oversimplifications, but the results of differing models agree well. Wright (1943, 1946) considered two models, an "island" model and a continuous model with

isolation by distance. In the "island" model the hypothetical species is distributed among a large number of demes, each of which exchanges genes with the entire species at a rate g; i.e. $\bar{p} = \bar{p}_s$. In the continuous model the species is distributed continuously in one or two dimensions, and each individual is considered to be at the centre of a (uni- or bi-variate) normal distribution of parent-offspring distances from which its parents may have been drawn at random - the neighborhood. Rohlf and Schnell (1971) simulated Wright's continuous model. Kimura and Weiss (1964) studied one, two, and three dimensional stepping stone models in which a species is distributed among a square grid of demes connected by gene flow, to adjacent demes only, at rate g per generation, and long distance gene flow ($\bar{p} = \bar{p}_s$) at a rate g_s per generation. Kimura and Maruyama (1971) and Maruyama (1971) considered a stepping stone model like that of Kimura and Weiss (1964), but with no long distance gene flow and a peculiar form of recurrent mutation. The present author ran simulations of the stepping stone model with no mutation and the demes arranged in a hexagonal grid (section 2.2.2. for description).

In all models differentiation is greatest when few individuals move, and the distance travelled is small compared to the total distribution of the hypothetical species (see Wright, 1943; and figures 2-10 through 2-20). Wright's "island" model yields the least "differentiation" (smallest differentiation ratio) among the models for comparable rates of gene flow. "Important differentiation" (all gene frequency classes equally common, or a rectangular distribution of commonness) may evolve if the number of individuals participating in gene flow, $N_x g$, is less than 5, and "significant differentiation" (gene frequency classes in a U-shaped distribution) may evolve if $N_x g$ is less than 0.5 (Wright, 1943). This is because small g reduces

the effect of gene flow, and small N increases the random differentiation by genetic drift.

In Wright's 2-dimensional continuous or neighborhood model, "important differentiation" may evolve if the effective size of a neighborhood is less than 100 individuals and the ratio of the area occupied by the species to the average area occupied by a neighborhood, ($A_s/A_x = K$,) is greater than 10^7. Effective size (N) and area (A) of a neighborhood are directly related because a constant density is assumed. For smaller effective neighborhood sizes "important differentiation" may evolve for smaller K. "Significant differentiation" requires larger K and smaller effective neighborhood size, For example, if the effective size is 20 individuals, "important differentiation" may occur if K>10, and "significant differentiation" if $K>10^5$, but if N=50 individuals, "important differentiation" does not occur unless $K>10^3$ and "significant differentiation" not unless K is extremely large. Wright (1943, 1946, 1969) does not give any indication that steep clines will evolve in the neighborhood model, but the simulations of Rohlf and Schnell (1971) indicate that clines may be formed. Rohlf and Schnell (1971) do not give a vertical (gene frequency) scale in the maps which they present, so it is difficult to see whether any morphotones are present. In continuous models differentiation measured by the differentiation ratio or its equivalent is greater for smaller neighborhoods and for more neighborhoods per species area (1/K) (Wright, 1943, 1946, 1969, Rohlf and Schnell, 1971); both N and K are measures of the amount and distance of gene flow and the magnitude of the drift factor.

Analogous results are found in stepping stone models. Kimura and Maruyama (1971) and Maruyama (1971) find that if $N_x g$ is less than 1,

pronounced "differentiation" (as measured by the differentiation ratio) of demes may evolve. If $1<N_x g<4$, then "differentiation" is less pronounced. Kimura and Maruyama (1971) also ran some simulations. The details are not presented, but apparently the grid was a square grid of 20 by 20 demes arranged on a torus (doughnut). Deme sizes (N_x) and gene flow rates (g) were not given, but their product ($N_x g$) was 4.0 and 0.25 for two published simulations. It is not clear how the demes were started with respect to gene frequencies, and apparently results were not printed out by their computer unless the mean gene frequency ($\bar{p}_s$) rose above 0.10. Rohlf and Schnell (1971), and the simulations of the present author (figure 2-10 to 2-20), demonstrate that the changes in the first few generations are likely to affect the pattern of areal differentiation in future generations; the results could be artifacts of poorly chosen random numbers or biases in starting gene frequencies. Furthermore, when a deme's gene frequency became 0, the lost allele was introduced again! It was not said what the reintroduction of the lost allele did to the deme's gene frequency, and one cannot tell because N_x is not given. The two maps Kimura and Maruyama (1971) present show the gene frequency pattern of groups of "6.25" demes; apart from the dubious method of lumping "6.25" demes, this pooling obscures adjacent-deme differences in gene frequencies, which could be less than implied. In addition to losing details of the distribution of gene frequencies, lumping data from several demes is prone to bias; the pooled variation is dependent upon the quadrat size (size of the grouping) and the placing of the quadrat boundaries relative to the actual boundaries (if any) of the differentiated areas (see Pielou, 1969, and the ecological literature). It is therefore difficult to interpret Kimura and

Maruyama's (1971) simulations in relation to other models and field studies, although their critical $N_x g$ for marked "differentiation" is similar to other workers' results. Unfortunately, in most natural populations the number of individuals breeding in allochthonous demes per generation ($N_x g$) is likely to be greater than 1 (see references of section 2-1), so the application of the theory is limited.

In Wright's (1943, 1946), Rohlf and Schnell's (1971), Kimura and Maruyama's (1971), and Maruyama's (1971) models, the "differentiation" of demes or neighborhoods is very much greater for a species distributed as a narrow series or linear series of demes or neighborhoods compared to broad two dimensional areas for a given rate of gene flow. This is simply because a given individual's parents on average, come from more neighborhoods in a two dimensional habitat than in a narrow linear range, i.e., $\bar{p}$ is closer to $\bar{p}_s$ in an area compared to a line. Kimura and Weiss (1964) also considered "3-dimensional" distributions of demes. At first sight this is biologically unsound (even in the sea, distribution and breeding is usually restricted to one depth), but in the "3-dimensional" model each deme has 6 adjacent neighbors, hence is comparable with a species distributed in two dimensions in a hexagonal grid of demes (fig. 2-2). Kimura and Weiss (1964) find that differentiation is smaller in "3 dimensions" compared with two or one, as is expected since gene flow can come from 6 instead of four or two neighbors.

Wright (1943, 1946, 1951, 1969), and Kimura and Weiss (1964) also considered models in which both long and short distance gene flow occurred. In both models the long range gene flow was assumed to come from a random sample of the species (or $\bar{p} = \bar{p}_s$), and the long range rate g_s was found to set a limit to the amount of random

"differentiation" possible for all g. Wright found that long range gene flow reduces the "differentiation" of subgroups if their sizes are less than $1/g_s$. As g_s is likely to be very small (section 2-1), and must actually introduce a new or rare gene or gene arrangement, the effect of long range gene flow in natural populations is likely to be very small. In addition, long range gene flow is unlikely to be representative of a species ($\bar{p} \neq \bar{p}_s$, refs. in Section 2.1 and Christian, 1970). Kimura and Weiss (1964), using the relationship between gene frequency correlation and distance as an index of differentiation, found that the correlation between a given deme and all demes x steps apart is approximately:

$$r_1(x) \doteq \exp\left(-x\sqrt{4g_s/g}\right), \qquad \text{for large x and } g_s << g << 1,$$

for a linear series of demes, and:

$$r_x(x) \doteq \left(\exp\left(-x\sqrt{4g_s/g}\right)\right)/\sqrt{x} \qquad \text{for large x and } g_s << g << 1,$$

for two dimensions, and:

$$r_3(x) \doteq \left(\exp\left(-x\sqrt{6g_s/g}\right)\right)/x \qquad \text{for large x and } g_s << g << 1$$

for "three dimensions". Thus the similarity between any two demes falls off rapidly with distance for larger g_s and smaller g. The correlation at a given distance, x, is greater for larger short range gene flow rate, g, and for small g_s. Similar results were found by Bodmer and Cavalli-Sforza, (1968). Unfortunately Kimura and Weiss's (1964) formulae are designed for small g ($g \leq 0.10$) and give unrealistic results for very small g_s. It is possible that 10^{-7} to 10^{-5} are reasonable values for g_s, but g is likely to be greater than 0.10 in natural populations. An additional problem is that the formulae

are only good for large x; it is the changes over short to intermediate distances which are of interest from the point of view of raciation and speciation.

It is not clear from any of the models whether morphotones may form as a result of random genetic drift; they only show that demes or neighborhoods may become different in spite of gene flow, depending upon the relative values of the gene flow and sampling drift terms in equation (3). To enquire whether morphotones could form regularly as a result of genetic sampling drift and gene flow, I have carried out a series of simulations for a hexagonal array of demes (fig. 2-2, figs. 2-10 through 2-20). In a given generation mating period, and in a given deme of size N the offspring genotype numbers were determined by drawing numbers at random from a rectangular distribution with a range of 0 - 1, inclusive. Random numbers were generated by the "power residue" method, the method that is least subject to periodicity (I.B.M., 1959). If a given number falls below p^2 it is treated as an AA homozygote, if between p^2 and $p^2 + 2p(1-p)$, it is a heterozygote, and if above $p^2 + 2p(1-p)$ it is an aa homozygote. The genotypes may then participate in gene flow. (The program will be found in appendix 2).

Figure 2-10 illustrates a typical run of 1000 generations for 2500 demes arranged in a 50 by 50 hexagonal grid. Each deme consists of 100 breeding individuals*. Total gene flow, g, in this simulation

---

*This and the following simulations are on a much larger geographical scale than those of Rohlf and Schnell (1971) and Kimura and Maruyama (1971), and were done before either paper was published (in 1969-1970). Rohlf and Schnell's area would fit comfortably in 1/25 of the area shown in fig. 2-10. Kimura and Maruyama's simulation of 20 x 20 demes is on a torus, and they do not give population size, so comparison is difficult. If their N were the same, their area would fit in 4/25 of the present area.

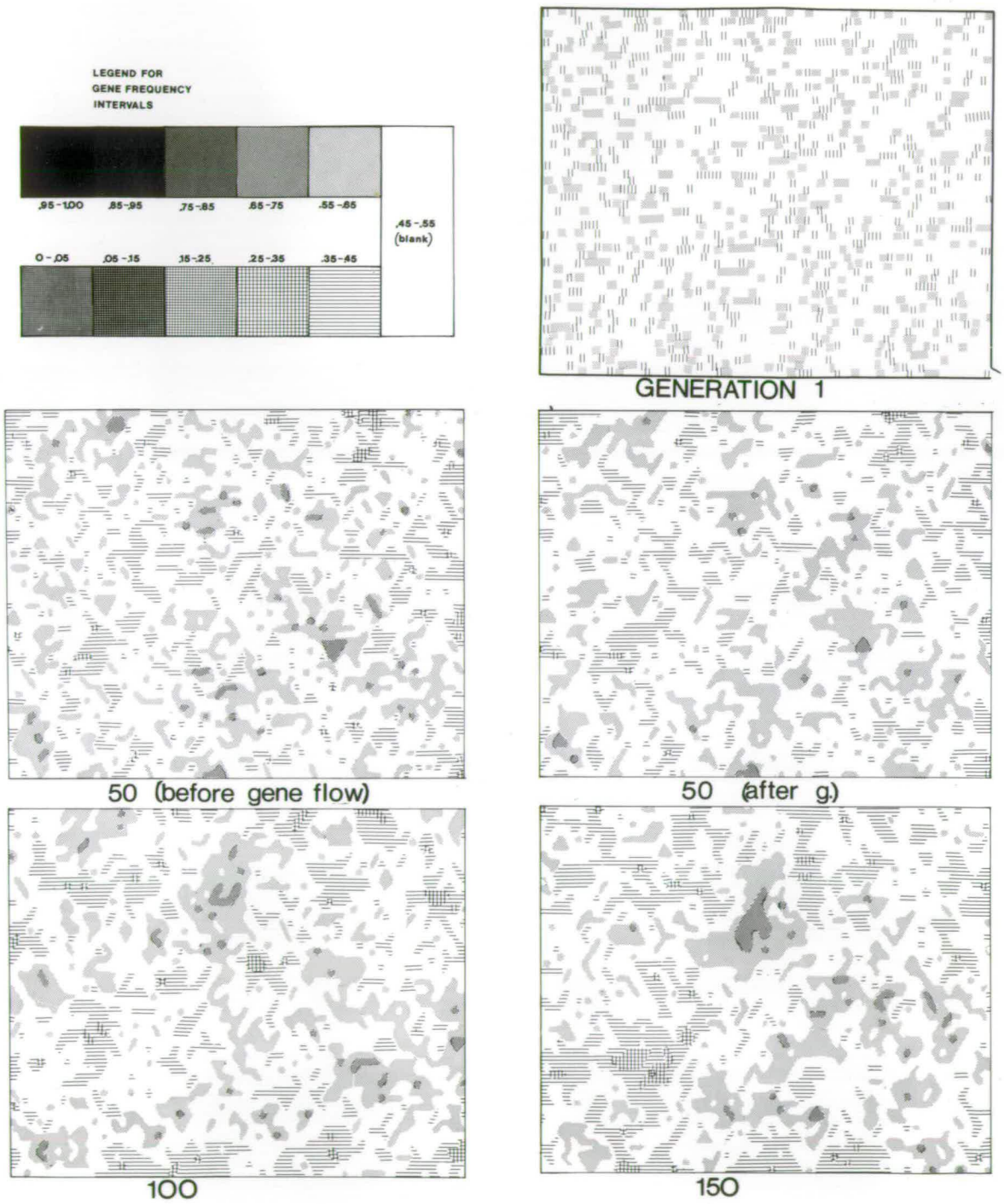
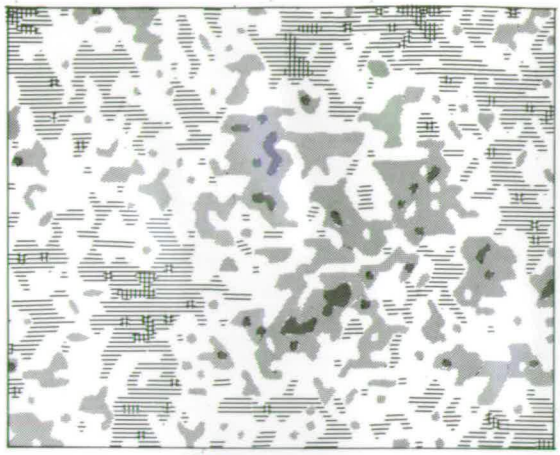
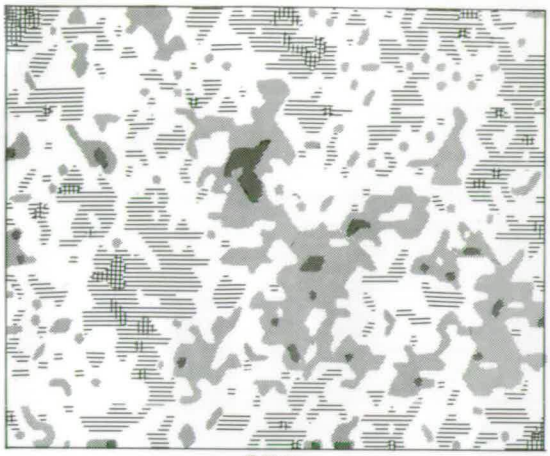


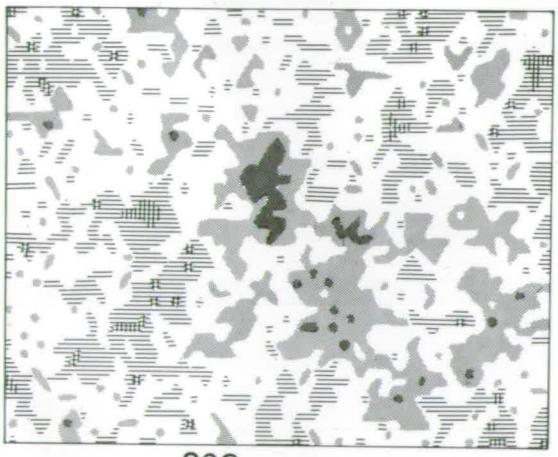
FIGURE 2 - 10. Random genetic drift in a 50 x 50 hexagonal array of demes, each with N=100, g=0.20. All demes started with a gene frequency of 0.5. The legend also applies to figures 2-15,20,36.



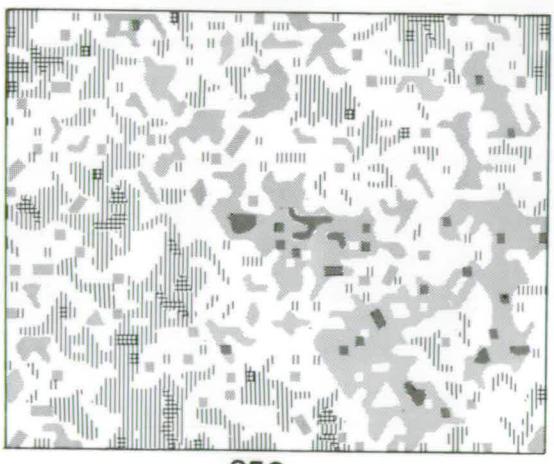
200



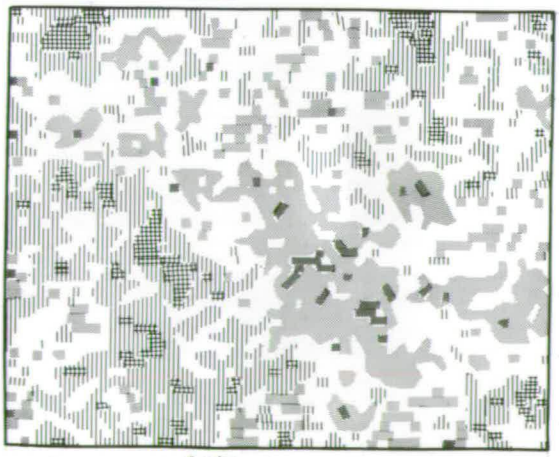
250



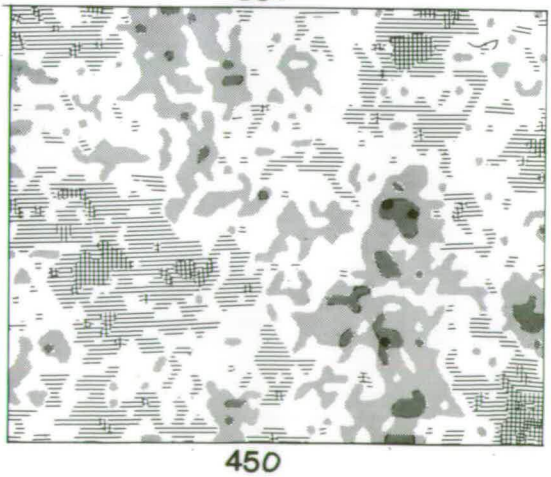
300



350



400



450

FIGURE 2 - 10, continued.

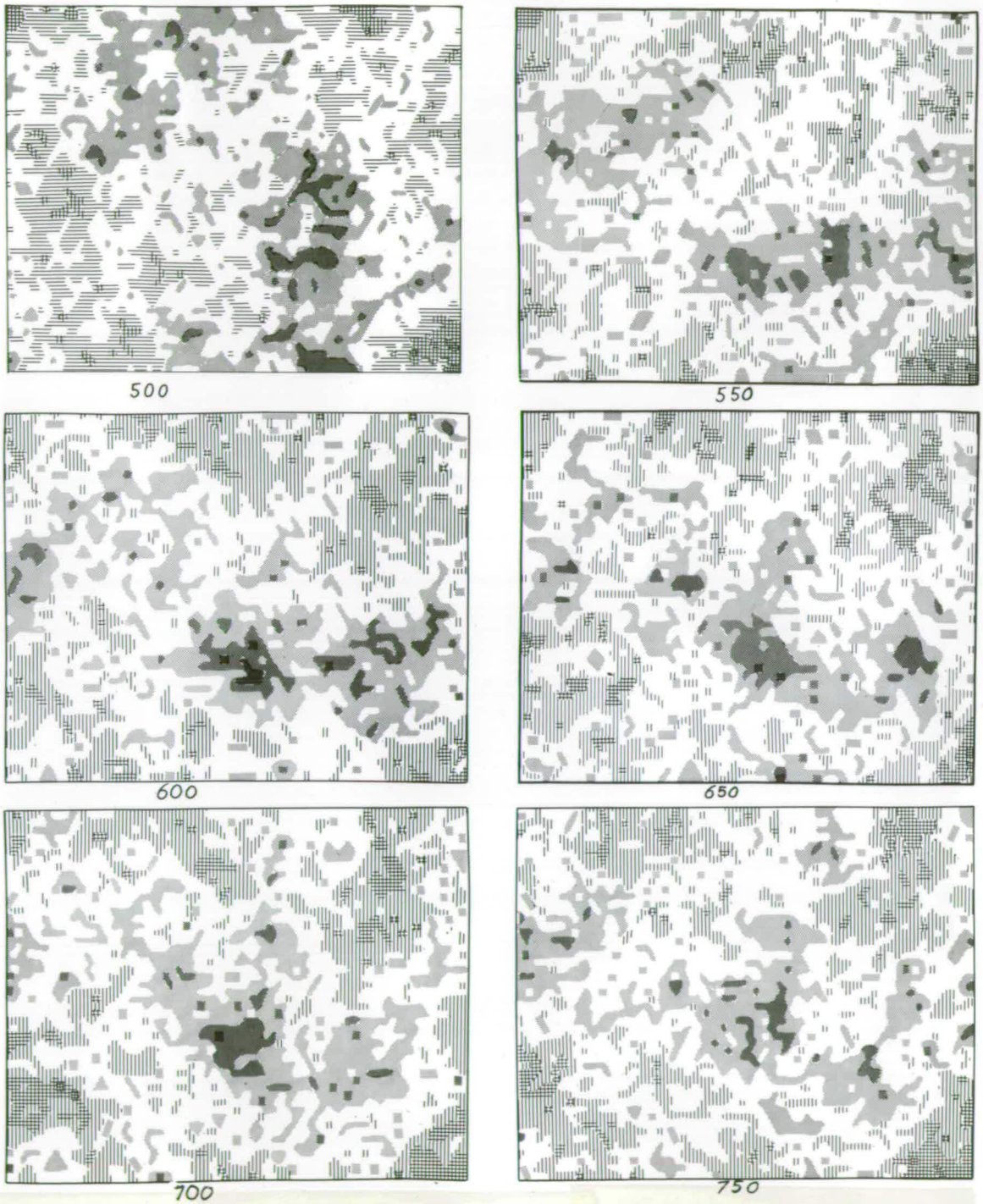
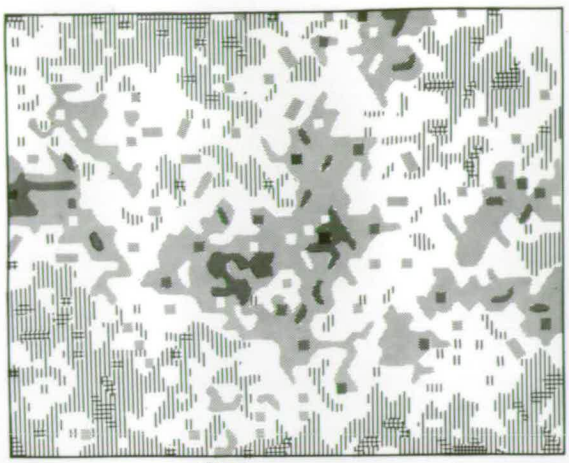
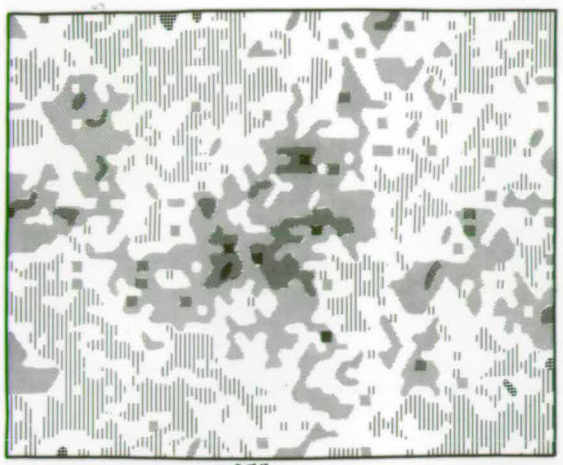
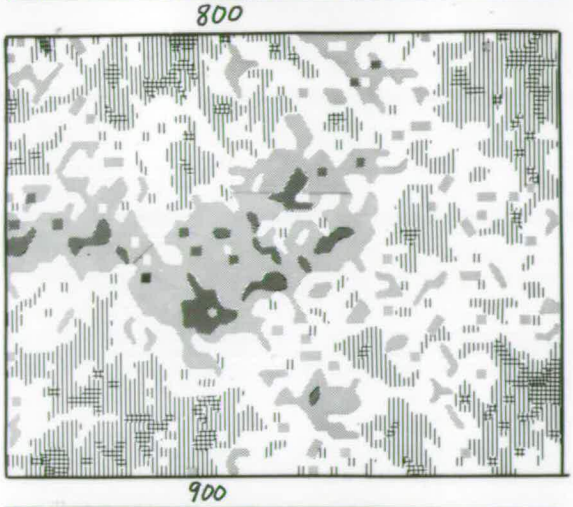
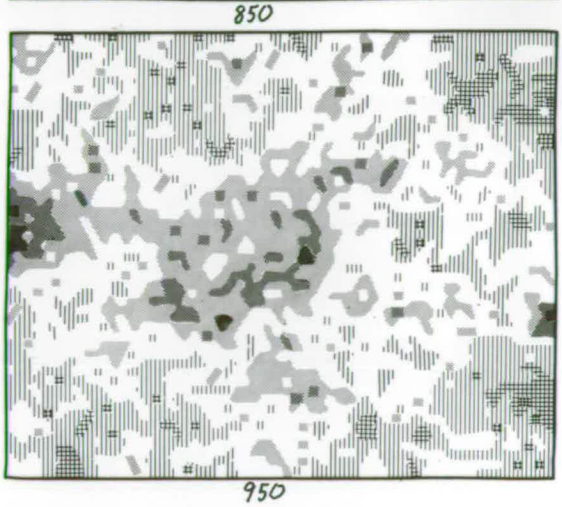


FIGURE 2 - 10, continued. Rows and columns were inadvertently exchanged in the printouts used for these illustrations between generations 500 and 550. This has no effect on the conclusions.









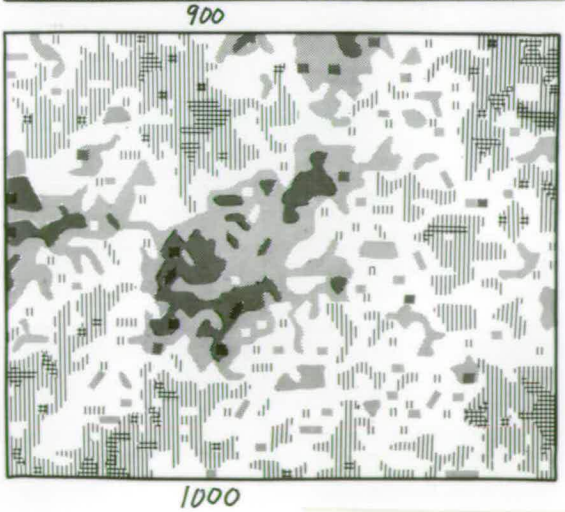


FIGURE 2 - 10, continued.

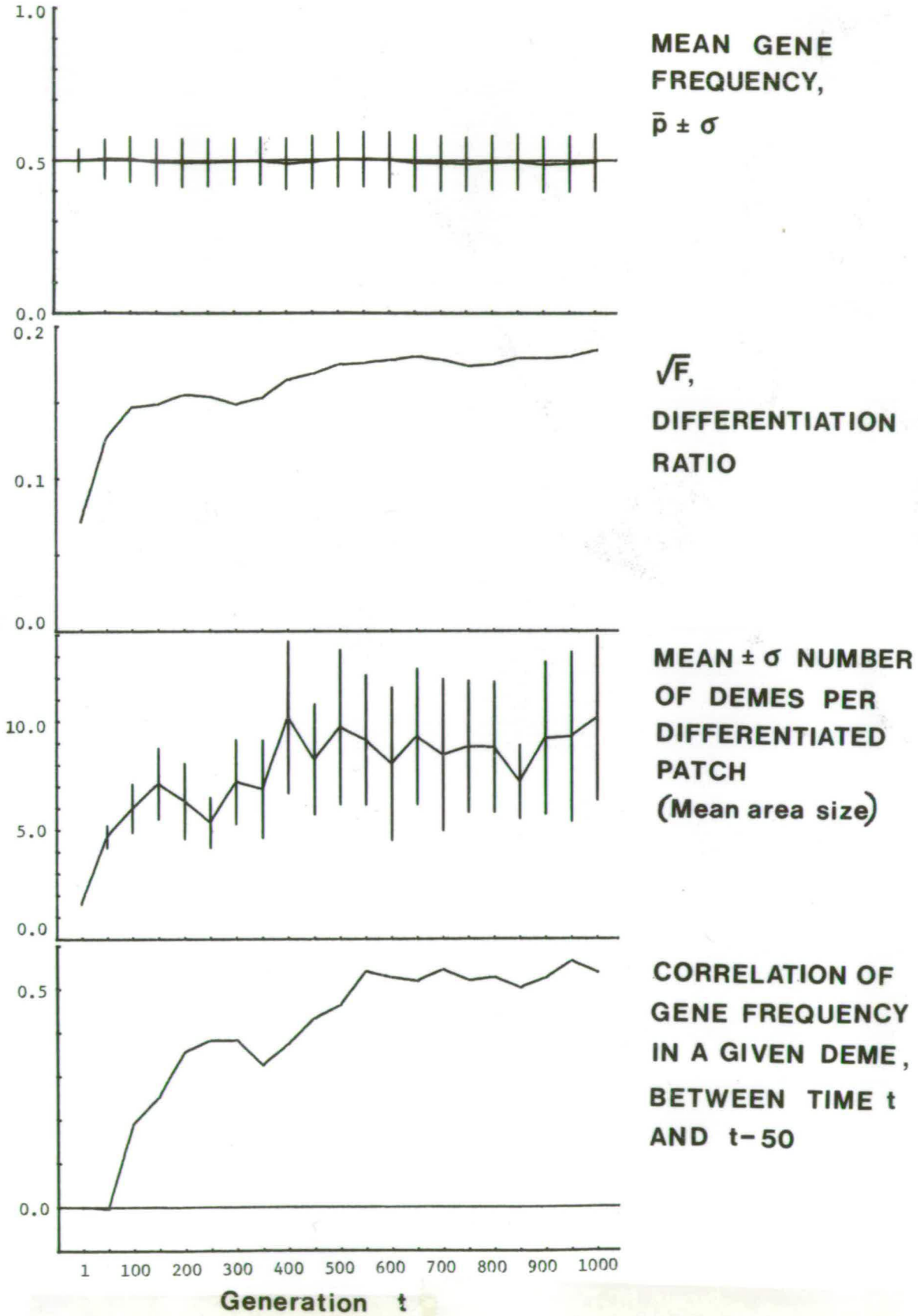


FIGURE 2 - 11. Measures of differentiation for the simulation shown in figure 2 - 10.

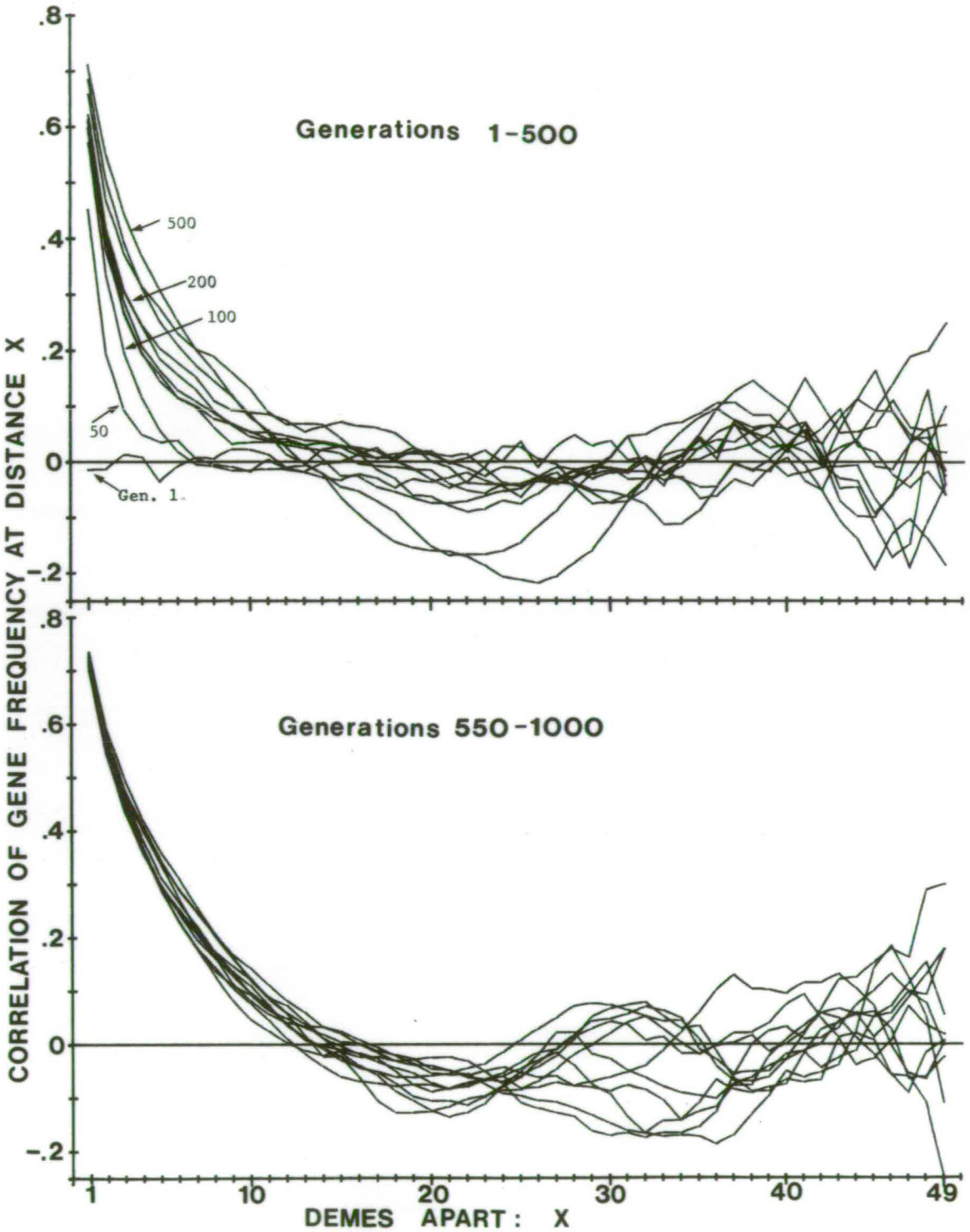


FIGURE 2 - 12. Correlation of gene frequencies of demes which are x steps apart, simulation of figure 2 - 10.

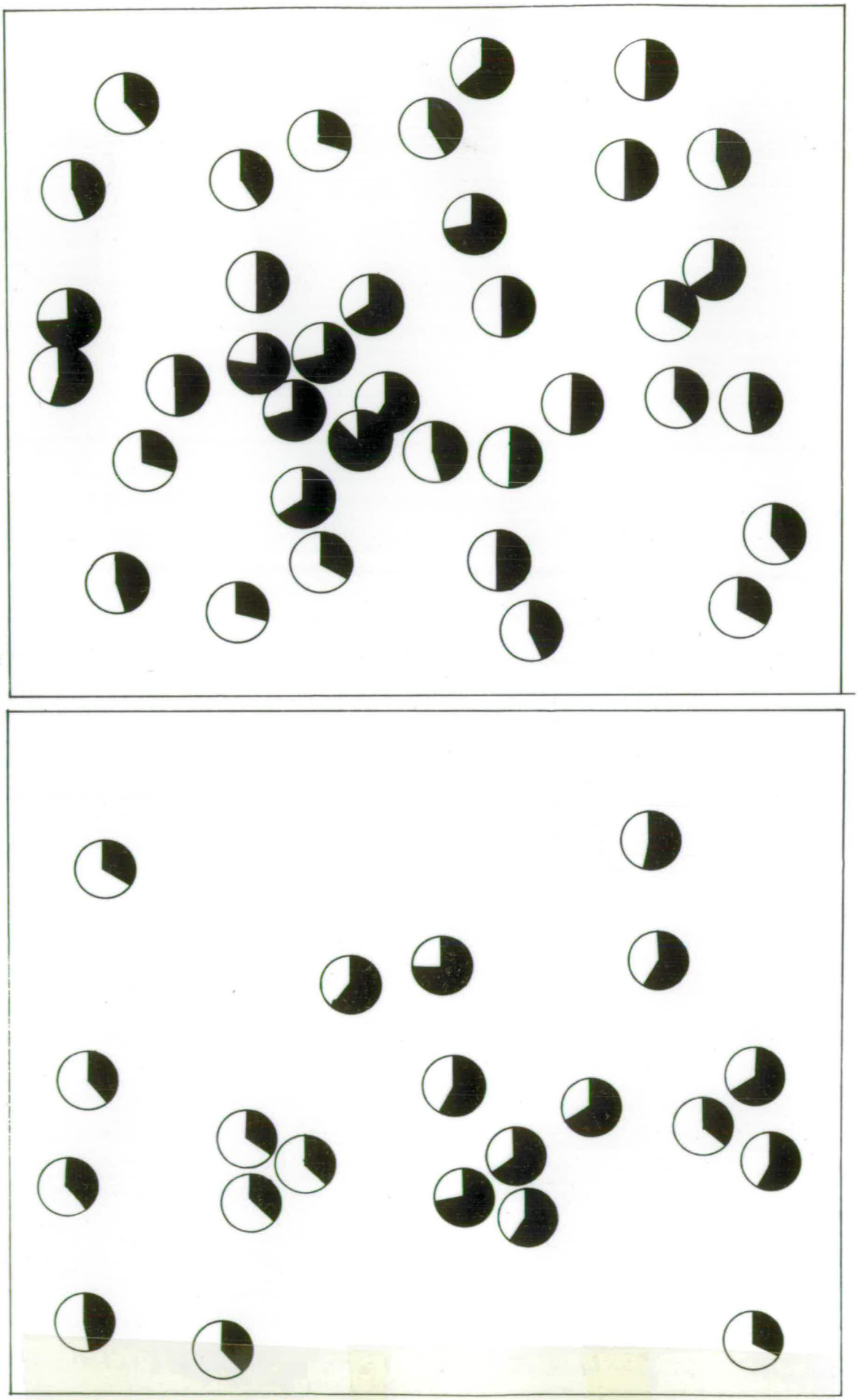


FIGURE 2 - 13. Random samples of 7 demes taken at (below) generation 200 and (above) generation 1000.

was g= 0.20, i.e., adjacent demes exchanged g/6= 0.03333 of their breeding population each generation; thus $N_x g$= 2.0. Natural populations may have smaller deme sizes, but many will have larger g.

As in Rohlf and Schnell's (1971) simulations, the changes in the first few generations determine the major features of the spatial distribution of gene frequencies. Subsequent generations change more slowly. The various measures of differentiation for this simulation are shown in figures 2-11 and 2-12, and demonstrate the gradual reduction in rate of differentiation in time. Clines are formed between generations 50 and 100, and their location remains relatively constant for many hundreds of generations. The broad picture of highs and lows is not too different between generations 250 and 1000.

If the maps of figure 2-10 represented a natural population, and sampling points were located at random over the map, but were widely spaced, it would appear that stable clines were present in the species (Fig. 2-13). Samples taken 1000 generations previous to the most recent sample might be even more thinly scattered, especially if from fossil or subfossil material. Data from subfossil Cepaea nemoralis and C. hortensis going back to about 1000 generations before the present-day samples from the same area show patterns of microgeographical variation which are not inconsistent with the present simulation (Cain, 1971, Currey and Cain, 1968). Other simulations also yield long lasting areas of generally high and low gene frequencies. This particular simulation was chosen for illustration because it shows, by chance, a saddle-shaped surface of gene frequencies. If this was observed in the field it would be very tempting to ascribe the ridge of high gene frequency to an environmental factor cutting diagonally across the study area. One would

rule out random genetic drift as a factor because the sampling points would yield large numbers of individuals (Cain and Currey, 1963a,b, 1968, Clarke, 1968, etc.).

However, a detailed survey would show that the clines are not smooth, nor is their detail stable from generation to generation. A morphotone may form one generation by chance, but will be quickly reduced or disappear in the next generation, although the overall cline may last hundreds of generations. Unless samples are taken over many successive generations at closely adjacent localities a stable pattern may seem to persist. The peak in the upper left centre of figure 2-10 lasted for about 500 generations before becoming small, although the boundary of the area fluctuated markedly each generation.

The patterns shown in figure 2-10 are similar to some of the microgeographic variation described by Cain and Currey (1963a, 1968), Arnold, 1968, Carter, 1968, Wolda, 1969a,b, and Jones, 1971 for Cepaea, and by Clarke, (1968, 1971) for Partula, and would qualify as area effects if detailed sampling were not carried out. An area effect is a stable microdistribution pattern in which areas larger than the species' panmictic unit are relatively uniform in gene or genotype frequencies, and are separated by stable morphotones which do not correspond with any known environmental factor (After Cain and Currey, 1963a).

The effect of various levels of gene flow (g) on random differentiation is shown in figure 2-14. Each simulation in the figure uses the same set of random numbers to allow direct comparison of the effects of gene flow, and other sets yield very similar results. Each deme is given a size of N= 50 individuals in a 30 by 30 hexagonal grid of 900 demes. As the mathematical models (and common sense)

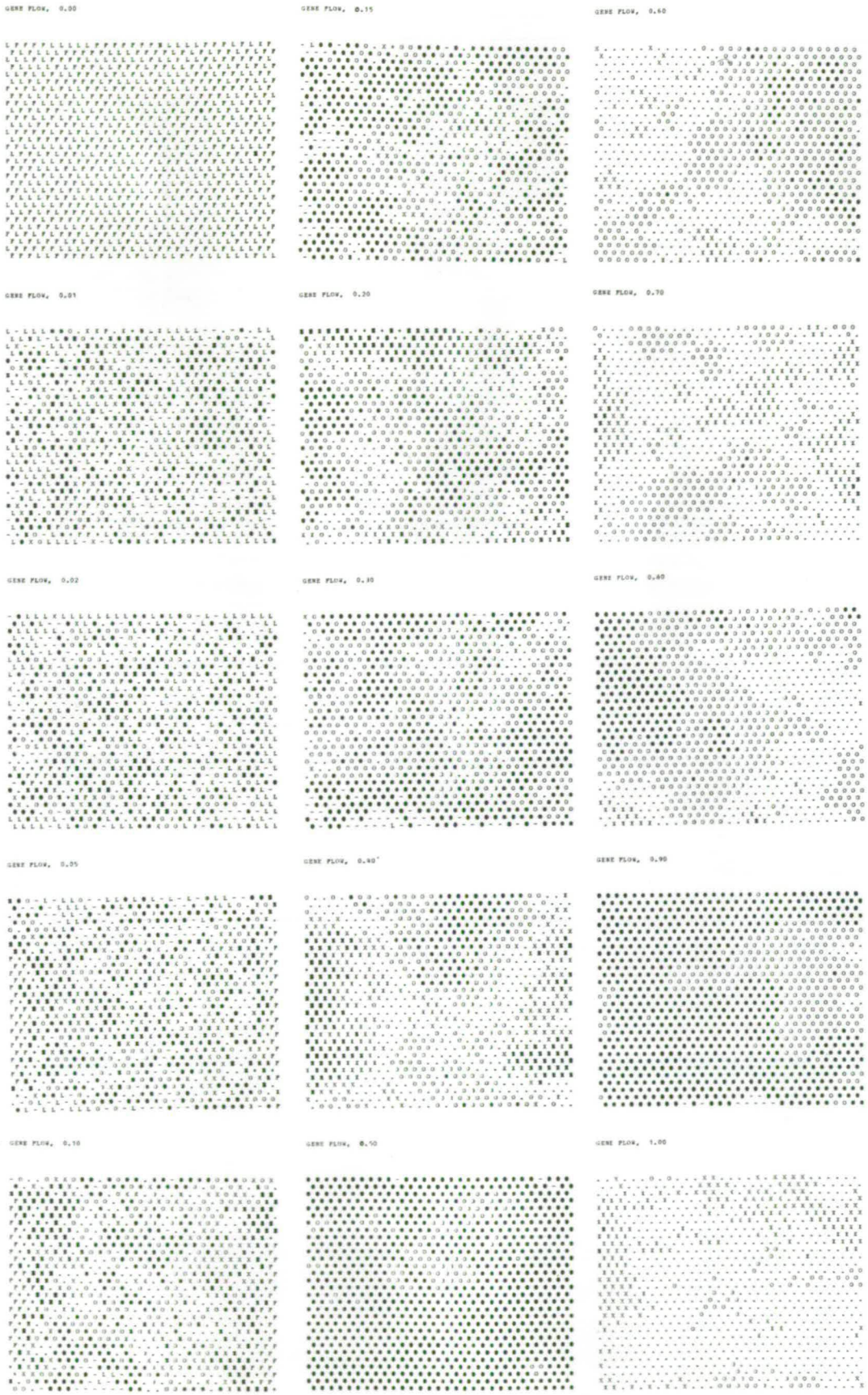


FIGURE 2–14

LEGEND:

Gene frequencies in each deme

Gene frequencies at generation 500 for various g and N=50.

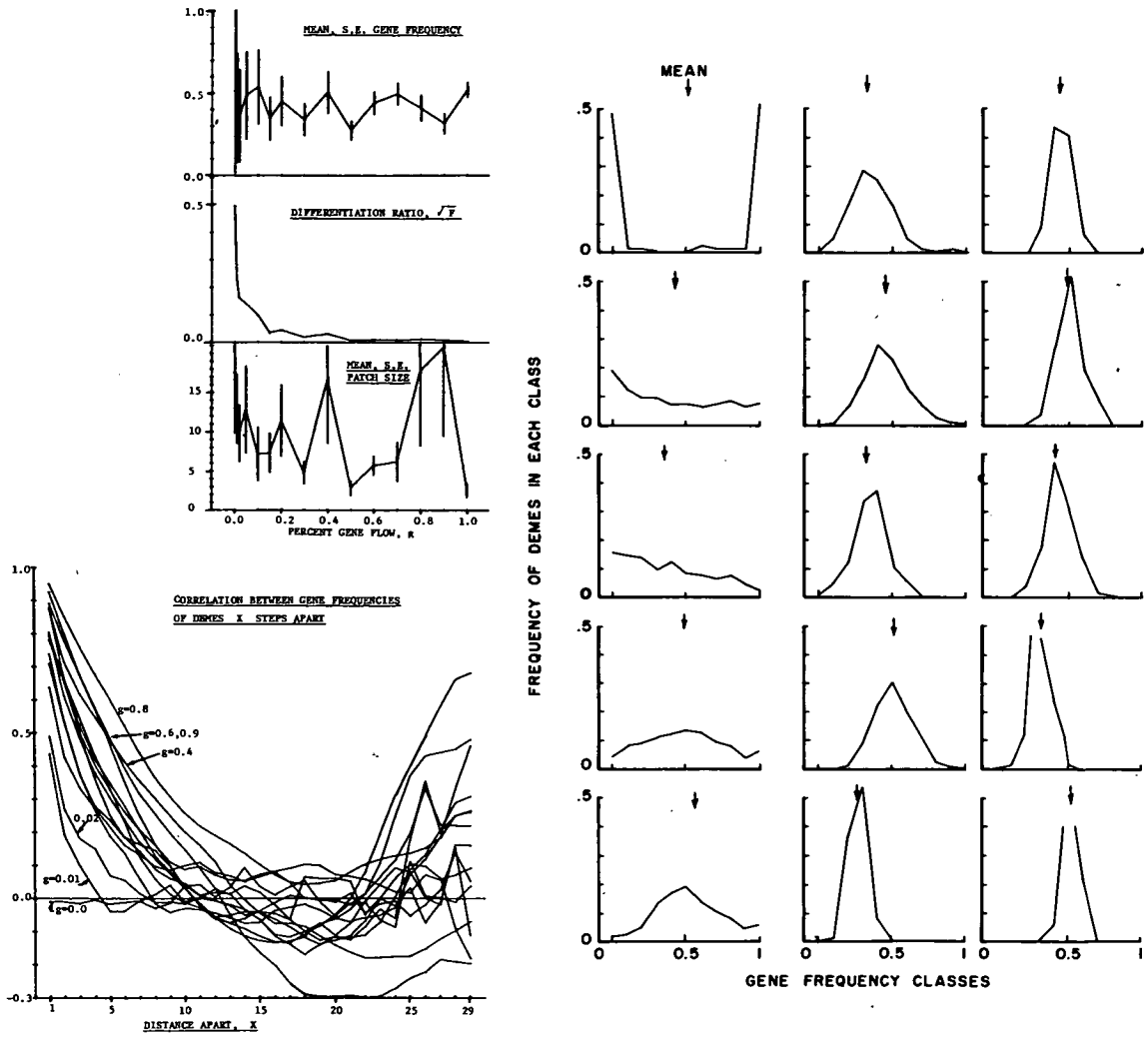


FIGURE 2 - 14, continued. Measures of differentiation at various levels of gene flow. The panels for frequency of each gene frequency class are arranged as in the maps.

predict, decreased gene flow results in greater variation in gene frequencies among demes - the "differentiation" of Wright and others- but not necessarily differentiation of areas. There seems to be an optimal level of gene flow at which areal differentiation is greatest. For the 30 by 30 hexagonal array of demes of 50 individuals each the optimal g is about 0.30. On the basis of mean differentiated area size near equilibrium, (see section 2.32, p. 2-16) the number of individuals breeding in allochthonous demes (Ng) which give rise to maximum areal differentiation is always greater than the maximum Ng which favours "differentiation" as measured by the differentiation ratio. At the optimal rate, gene flow keeps the similarity of nearby demes high but permits areas to diverge. If the rate is lower then each individual deme will fluctuate too much for gene flow to act as a "cohesive force" (Dobzansky, 1941, Mayr, 1942); if the gene flow is too great it will "swamp out" local differentiation (Mayr, 1942, etc.) In none of the models did stable morphotones evolve, although clines were steepest at optimal Ng.

Uniform density, or spatially uniform deme size (N) is extremely unlikely in nature (Allee, et al., 1949, Andrewartha and Birch, 1953, Southern, 1966b, Pielou, 1969, and almost any issue of Ecology). Variation in species density may be exhibited in two ways: (1), barriers to dispersal, and (2), inter-deme variation in population size. Effective deme size is reduced near barriers because dispersing residents may not be replaced by aliens. One would expect a priori more differentiation in species with spatially variable deme size compared to constant N, because the total effective population size of the species is smaller (Gadgil, 1971, Wright, 1940a), and the local variation in N increases the between-deme variation in genetic drift rate (Wright, 1931, 1940a,b, 1969).

Figure 2-15 is a hypothetical example of the effect of barriers. Similar results are obtained with other parameters. The demes or groups of demes near barriers differentiate more strongly than those with gene flow from all six neighbors. As in the previous models areal differentiation may evolve without barriers, but is stronger and more stable when barriers are present. Of special interest is that a group of demes which differentiates near a barrier, more than others away from the barrier, can act as a centre of differentiation, giving rise to a differentiated area which does not correspond to the barrier. Barriers need not restrict gene flow from all directions to be effective as differentiating agents; even restriction in one direction accelerates the process.

Data from the microgeographical distribution of the grasshopper Myrmeleottetix maculatus provides excellent material for investigating the effect of variable deme size. Like many grasshoppers (White, 1968, White, et al., 1967), Myrmeleottetix has a very limited dispersal and a very patchy distribution (Hewitt and Brown, 1970, Hewitt and John, 1970a,b, Hewitt and Ruscoe, 1971). Dr. Godfrey Hewitt, who has been studying "B" chromosome clines in M. maculatus very kindly mapped its fine distribution on the Ben Goginen mine in Wales for the author, and provided unpublished information on the dispersal rates of these annual grasshoppers. Using all available data, Dr. Hewitt's study area was transformed into a series of demes of varying sizes in a hexagonal grid to be used in simulations (fig. 2-16). The distance between demes was estimated to be 8 yards and g=0.20 as an approximation. A typical result after 500 generations is found in figure 2-17, and the results for the high density areas in two more simulations are found in figure 2-18. The simulations show that large spatial

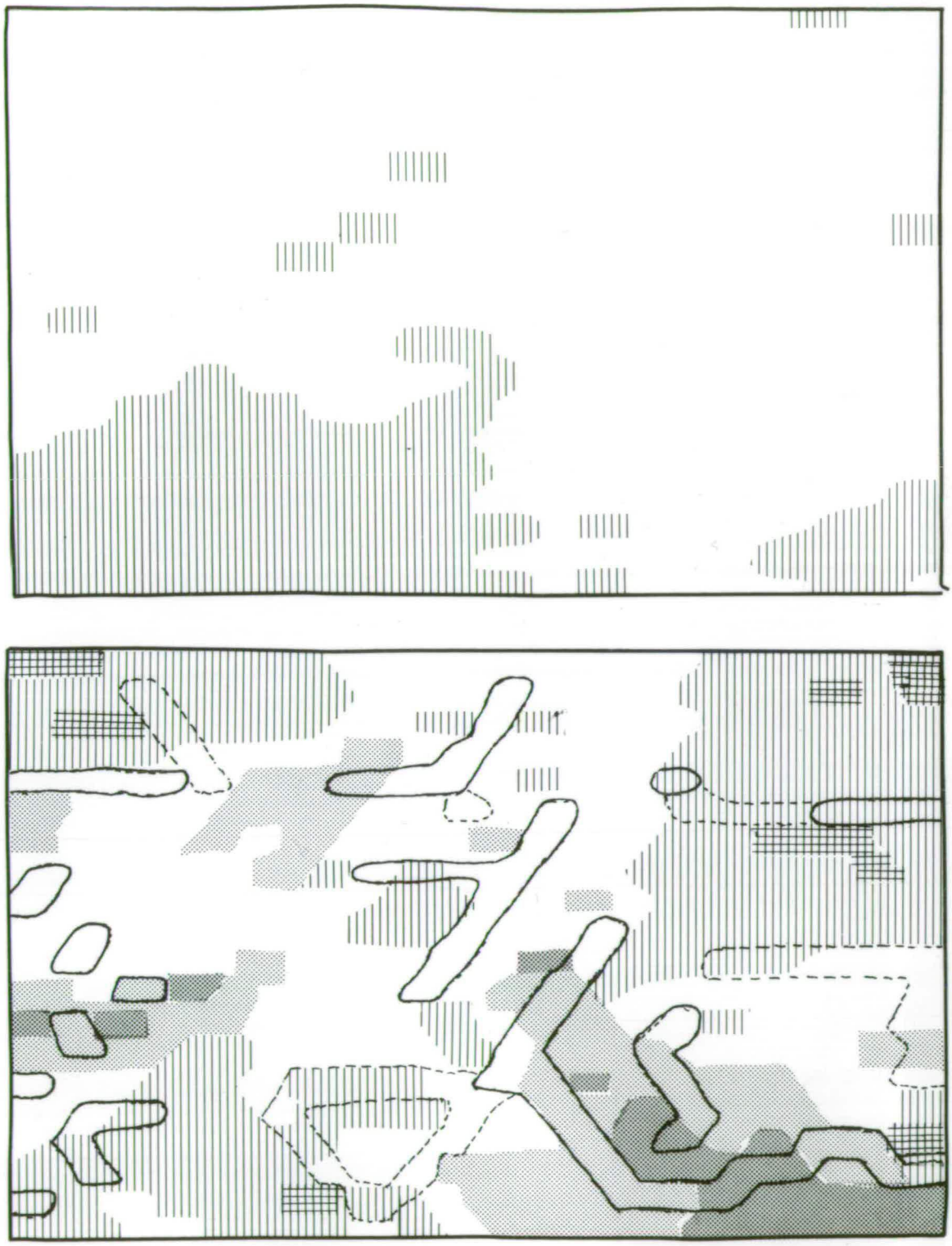


FIGURE 2 - 15. A 400 DEME (20 by 20) RANDOM GENETIC DRIFT SIMULATION

Upper map: 250 GENERATIONS, N= 100, q= 0.20

Lower map: 250 GENERATIONS, N= 100, inside dotted lines N= 75, inside solid lines N=25, q= 0.20, same set of random numbers as upper map

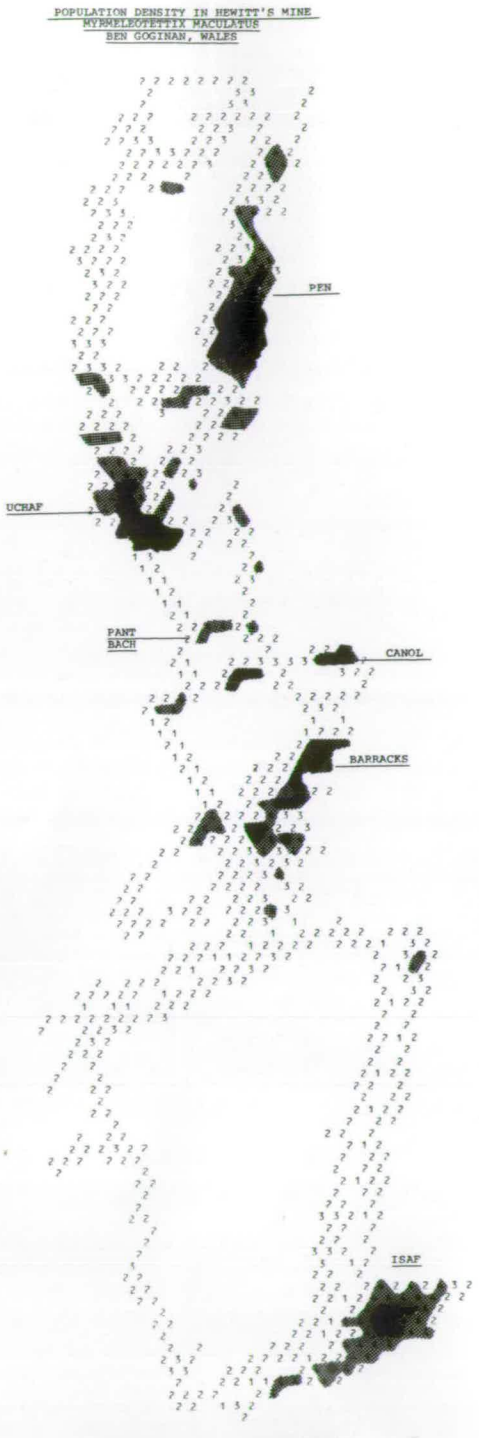


FIGURE 2 - 16

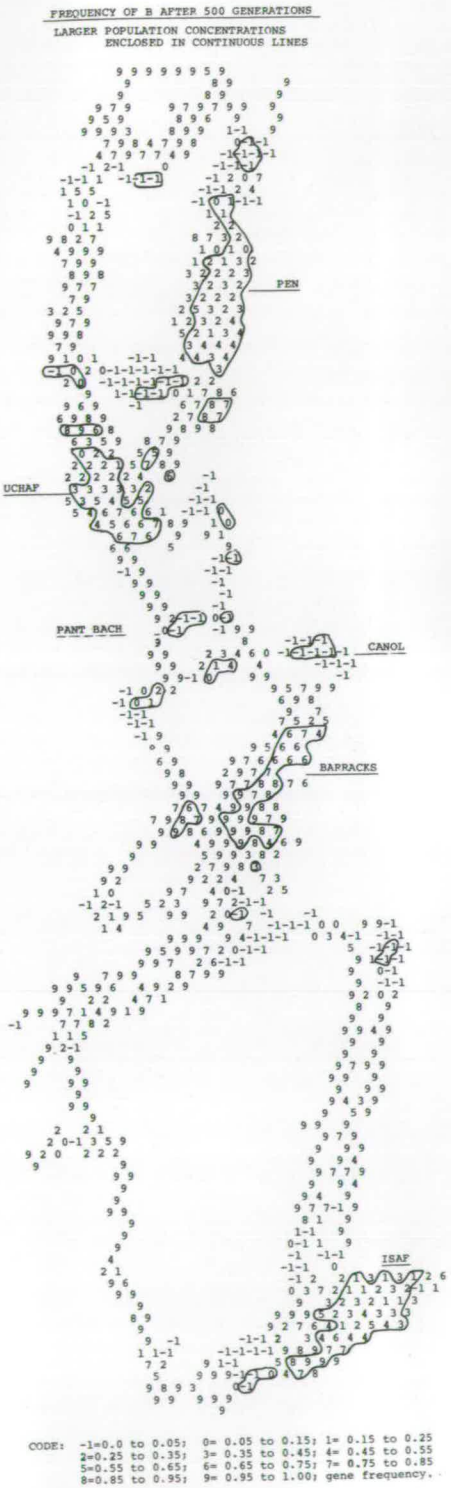


FIGURE 2 - 17

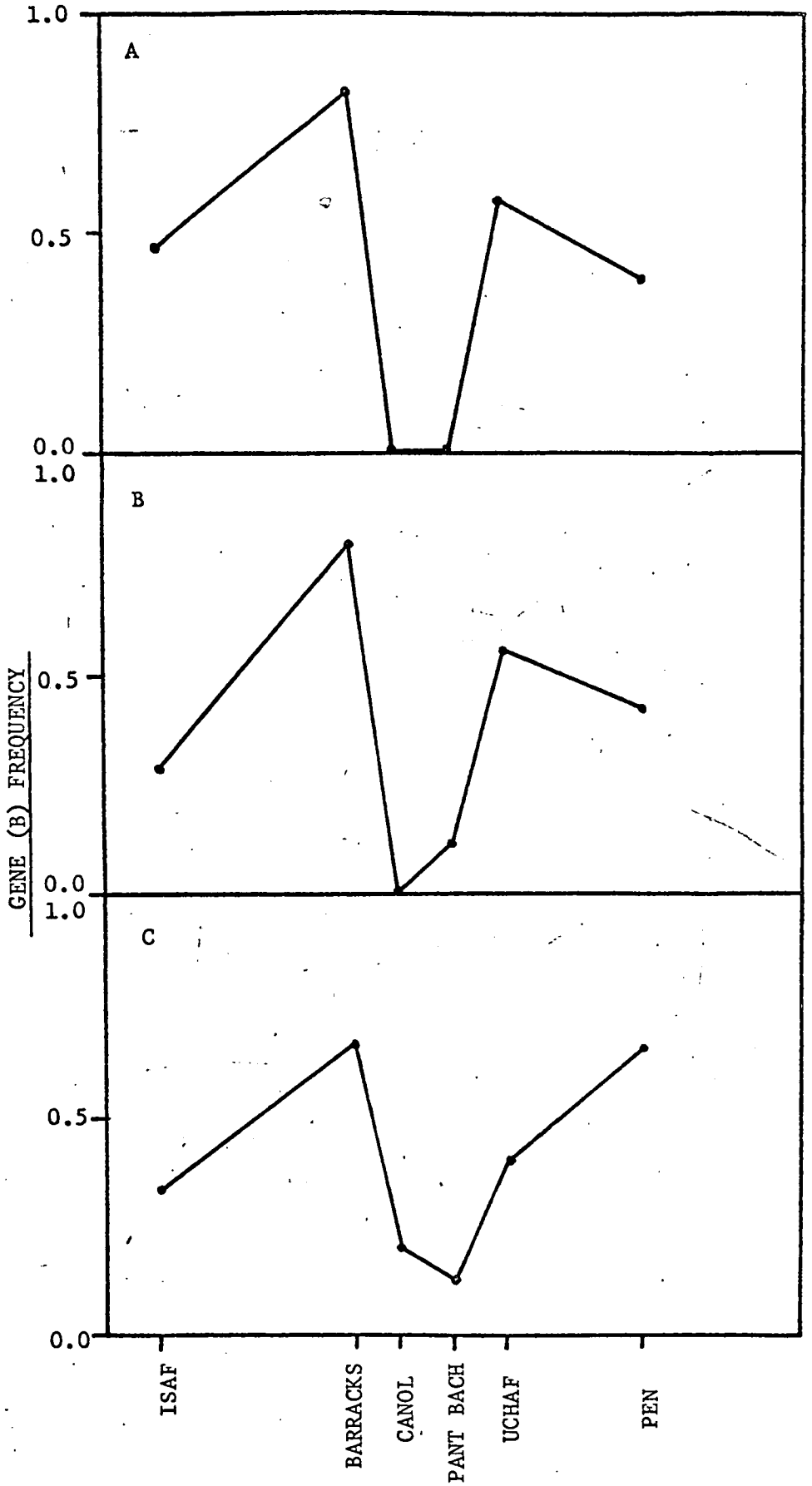


FIGURE 2-18. Three random genetic drift simulations of Hewitt's Welsh mine with resident Myrmeleotettix maculatus. A, from fig. 2-17,; B,C, two more simulations.

differences in gene frequencies can evolve as a result of genetic drift and gene flow; groups of small demes may become very different from adjacent large demes, and large populations separated by small demes may become significantly different from each other. Large populations do not necessarily "swamp out" small ones even though they may be sending out greater numbers of individuals. The greatest differentiation occurred in the centre of the mine, and not at the periphery of the distribution as one might expect. It is at the centre (Uchaf) where the greatest reticulation of barriers and variation in deme size is found.

In all of the simulations of random genetic drift and gene flow which the author has run, the mean area size of strongly differentiated areas (using the most generous thresholds) did not become larger than about 15 demes, even after many hundreds of generations with optimal Ng. Given the estimated dispersal rates (which may be overestimates, section 2-1) of Cepaea (Lamotte, 1951, Murray, 1964, Goodhart, 1962, 1963, Cain and Currey, 1968) and Partula (Clarke, 1968), this would mean area effects of 30,000m^2 and 4700m^2 respectively. The area effect for Brown Shell in Cepaea nemoralis, in the Marlborough Downs covers an area of about 6km^2 (from map of Cain and Currey, 1963a), and for purple shell in Partula taeniata about 3km^2 (from map of Clarke, 1968). Thus, although random genetic drift may produce area effects, it produces them on a much smaller scale than is actually observed in nature (1km^2 = $10^6 m^2$).

In the simulations of the present author, and of Rohlf and Schnell (1971), the extent of differentiation by random genetic drift was very much below that observed in area effects, except for very low g. For intermediate g the range of gene frequencies was about 0.3

to 0.7. Adjacent demes rarely had a difference in gene frequency greater than about 0.3, and such differences were usually not associated with large differences nearby, or in regular patterns. This is in marked contrast to area effects, which show a great range in gene frequencies, and regular patterns of between sample gene frequency differences greater than 0.3 and often as much as 0.6 (Cain and Currey, 1963, Clarke, 1968, 1971, Arnold, 1968, Carter, 1968). In the simulations such large differences were only obtained with very low gene flow, which was associated with a lack of areal differentiation.

Random genetic drift is not the only source of random variation in gene frequencies among demes or neighborhoods. An additional factor is the sampling error of gene flow. All previous models and simulations have assumed that gene flow is deterministic and that all random changes in gene frequencies resulted from sampling error between parents and zygotes. The variance of gene flow was assumed to be zero (implicitly), and the gene frequency of the nonemigrating natives was assumed to be exactly equal to all natives before gene flow. However, emigrating natives may not be a representative sample of their parental deme by chance. This would be especially true for small N_x and g. Therefore p_x in$\delta(p_x,N_x)$ may be different from p_x in the $-g(p_x-\bar{p})$ term in formula (3). Similarly, $\bar{p}$ may not be representative of the mean of the demes providing aliens to deme x. In addition, variation about the mean gene flow rate, g, will cause fluctuation in the effect of gene flow on Δp_x each generation. Thus it is necessary to introduce an additional drift term into equation(3).

$$\Delta p_x = -g(p_x-\bar{p}) + \delta(p_x,N_x) + \gamma(g,p_x,N_x,\bar{p}) \qquad (4),$$

The gene flow drift term, $\gamma(g,p_x,N_x,\bar{p})$, is a randomly determined variable with a mean zero and a variance which would be a function of $g(1-g)/N_x$, $gp_x(1-p_x)/2N_x$, and the gene flow sampling variances of the immigrants making up $\bar{p}$. Apparently no one has investigated the effect of sampling error of gene flow. Disregarding this additional factor results in a consistent underestimate of the ability of random processes to produce differentiation; Δp_x depends upon the relative magnitude of gene flow and all drift factors.

Simulations including gene flow drift as well as genetic sampling drift were run using several sets of parameters. Each individual in each deme, each generation, was assigned a number drawn at random from a rectangular distribution of values between 0 and 1, inclusive. The individual's number not only determined its genotype (probability p^2, $2p(1-p)$, or q^2) as in the earlier simulations, but also whether or not it left the parental deme (probability g or (1-g)), and if it left, to which of the neighboring demes it reproduced in (probability g/6). The method is described in appendix 2. Figure 2-20 illustrates a typical result for a 50 by 50 hexagonal grid of demes, compared with an otherwise identical simulation with deterministic gene flow (fig. 2-19). Deme size (N) is 10 and gene flow rate is g= 0.20. Although differentiation is greater with stochastic gene flow, the mean and maximum area size and extent of differentiation is still very much smaller than naturally occurring area effects. In some cases the differentiation into areas was actually less for stochastic gene flow compared with the same set of random numbers for deterministic gene flow.

We must therefore look for factors additional to random drift to account for morphotones.

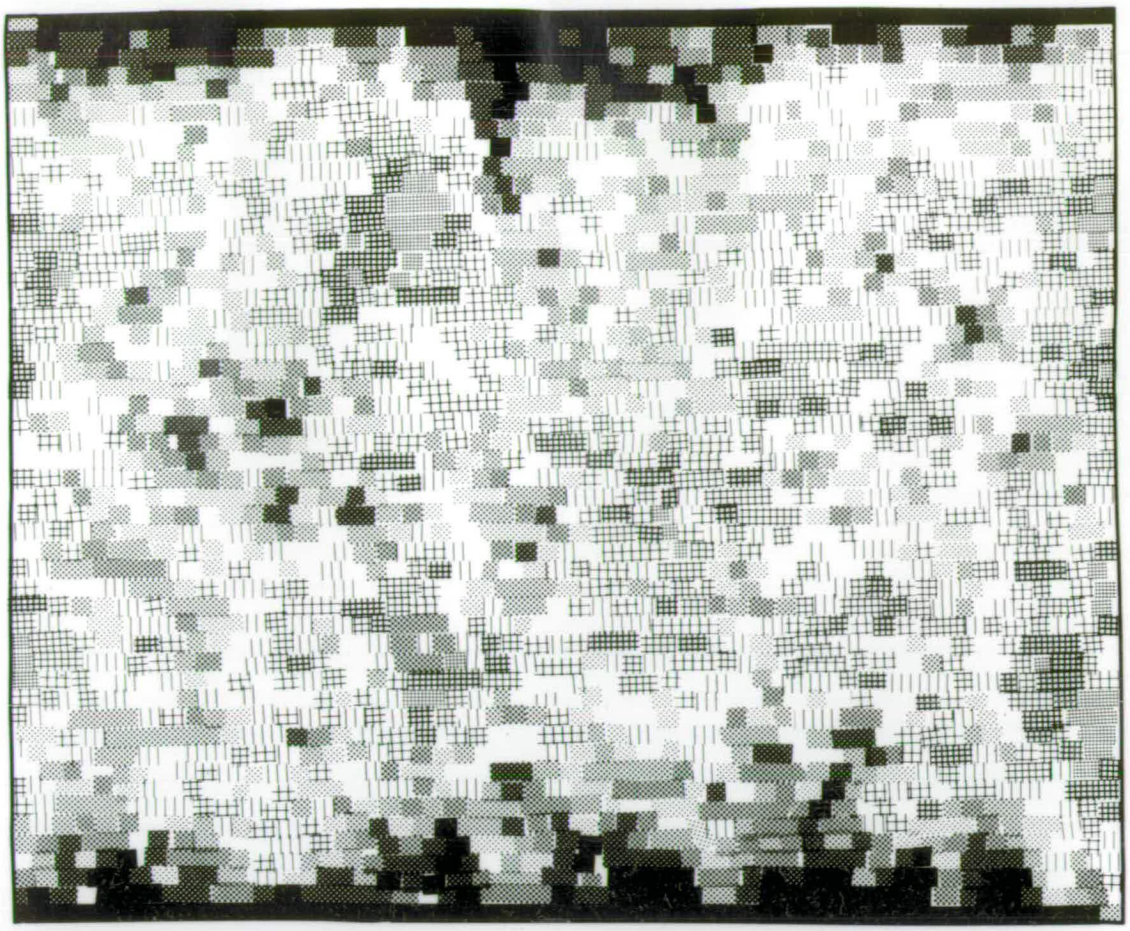


FIGURE 2 - 19. Genetic drift with N=10, g=0.20, 50 x 50 demes.

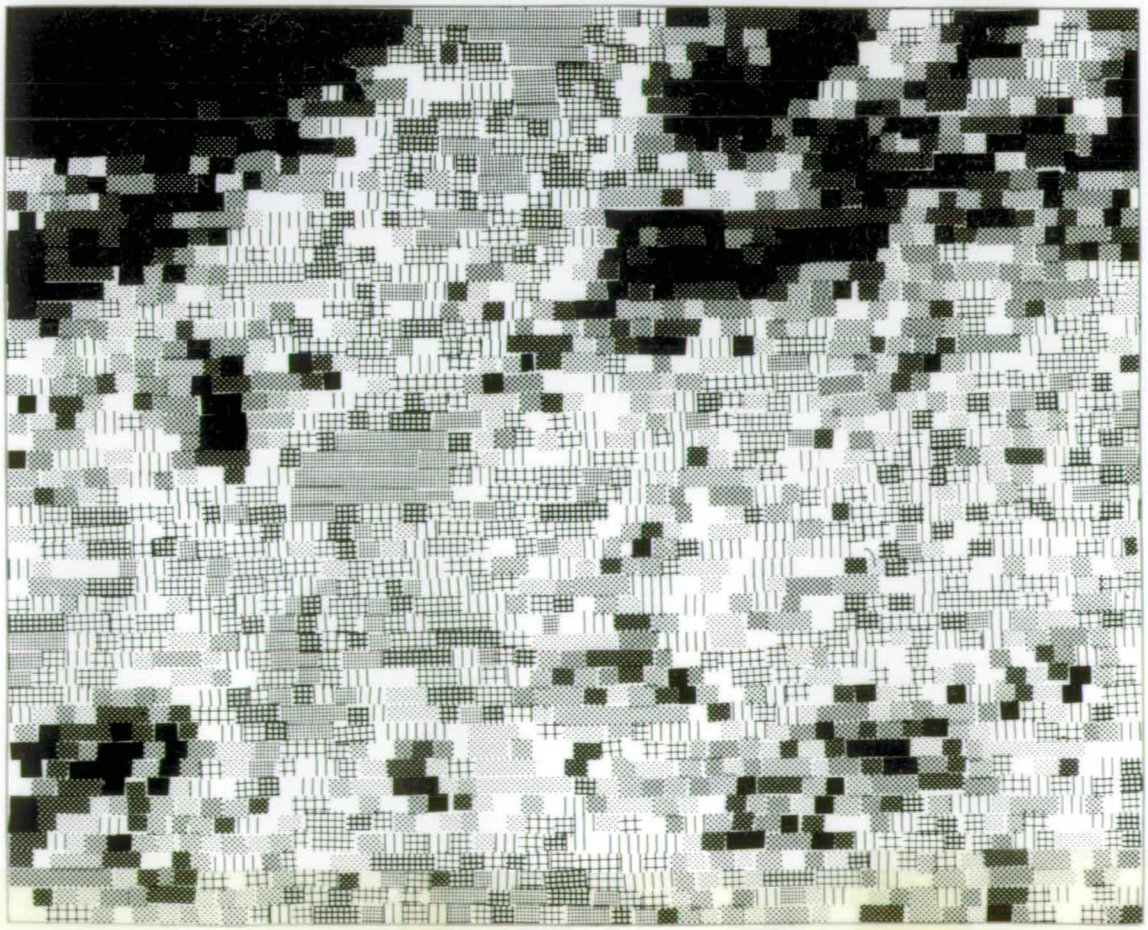


FIGURE 2 - 20. Same as above, but with additional gene flow drift. Legend same as in figure 2 - 10.

Section 2.5. Environmental Gradients

It is most unlikely that all genotypes will have exactly equal mean survival and reproductive rates for more than a short period of time. Under conditions of random genetic drift and gene flow, what happens in a few generations may determine the general spatial pattern of gene frequencies for the next thousand generations or more (section 2.4). Therefore an environmental factor which is geographically variable within a species range, but only acts once or twice in hundreds of generations, is sufficient to maintain areal differentiation of gene frequencies, although it would have to act more continuously to maintain morphotones. This makes it extremely difficult to distinguish in practice between selection and genetic drift as causes of areal differentiation and area effects which are less than about 15 demes or neighborhoods in area. From the point of view of raciation and speciation, however, it is the larger differentiated areas which are of interest, and one would expect that most environmental factors would act more continuously.

The majority of environmental factors are found in gradients rather than in two or more spatially distinct zones separated by abrupt changes (Allee, et al., 1949, Theinemann, 1950, Andrewartha and Birch, 1953, Clarke, 1953, Geiger, 1966). Broadly speaking, a species has a tolerance, and within that a preference, for a particular zone on an environmental gradient, and its maximum abundance is found at some intermediate optimum position (Figure 2-21a, Lenz, 1931, Vouk, 1939, Thienemann, 1950). The range of tolerance of a species is its ecological valence (Theinemann, 1950), or its ecological amplitude (Terborgh, 1971). The abundance curve of a species on a

gradient reflects the spatial pattern of its survival and reproductive rates. The probability of survival from zygote to successful reproduction will hereafter be referred to as the probability of absolute survival, survival value, or fitness (relative fitness will always be referred to as relative fitness). Absolute survival is at a maximum at the optimum position on the gradient, and decreases with increasing distance from the optimum, forming the survival or fitness curve (figure 2-21a). The area under the survival curve is directly related to the mean absolute survival of the species as a whole.

Similarly, a given individual will have its own range of tolerance and optimum on a gradient; a function of the various genetic and environmental factors operating during its lifetime. The species valence is made up of the aggregate of individual valences and fitness curves. Levins (1962, 1963) terms the shape and the area under the aggregate survival curve the "fitness set".

In a polymorphic species with two or more morphs which are affected by an environmental gradient, the species valence may be made up of two or more major groups of individual valences, corresponding to the morphs (figure 2-21b,c,d). If the mean valence and optima of the morphs are roughly the same, and all individuals are very sensitive to the position on the gradient (small individual valences), the species will be stenotopic (figure 2-21b). If the mean optima of the morphs are the same, but the action of the environmental factor is not so strong (large individual valences), the species will be eurytopic (figure 2-21c). If the morphs have different mean optimum positions, and different morph frequencies are found in different areas, then the species will be amphitopic or polytopic

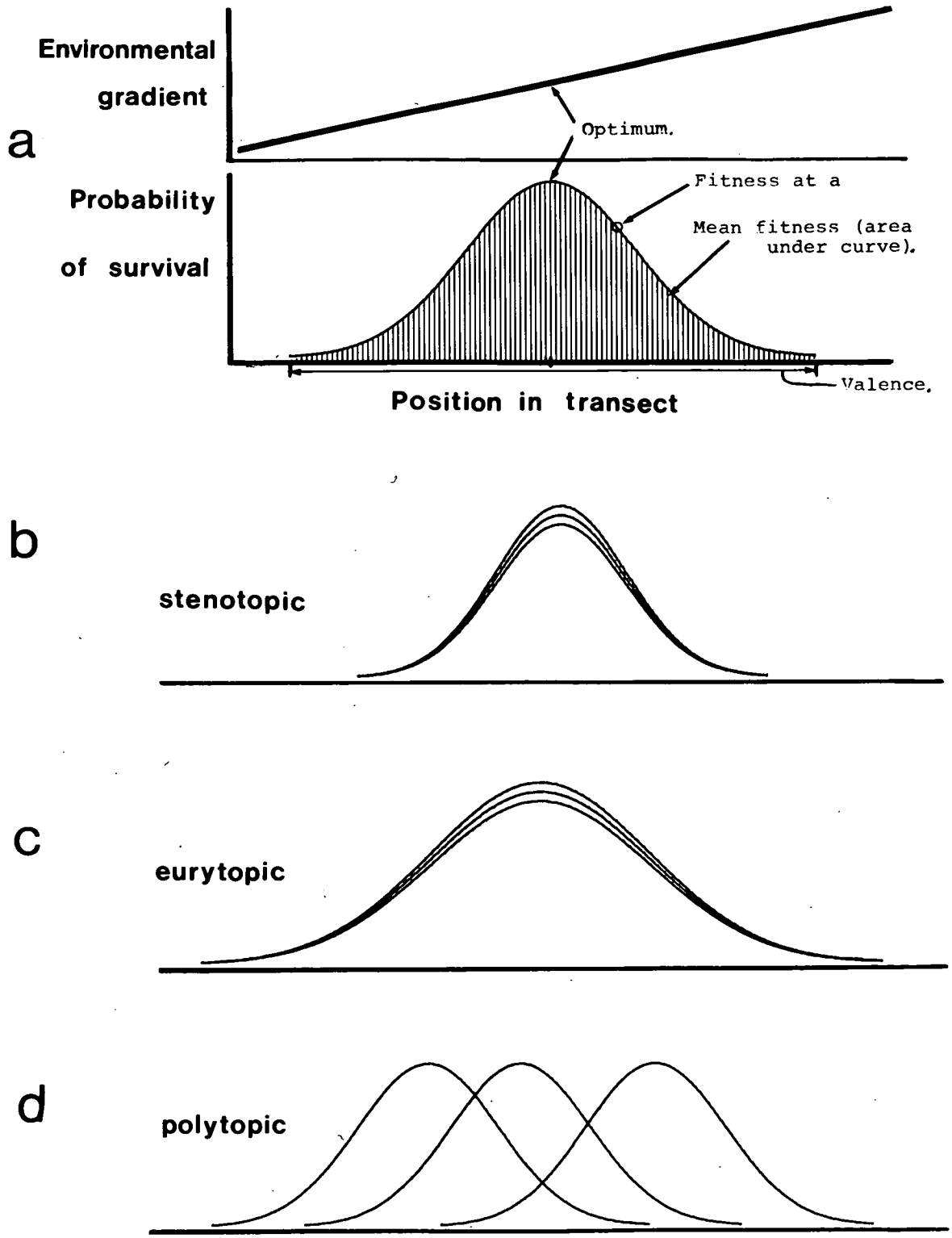


FIGURE 2 - 21. Fitness curves on an environmental gradient.

(figure 2-21d).. It is in amphitopic and polytopic species that environmentally induced clines are most likely to be found. (For a recent discussion of stenotopy, eurytopy, amphitopy, and polytopy, and the species range, see Udvardy, 1969).

The spatial survival curve of a given genotype may or may not correspond with the other genotypes. In a "neutral" character, or a character which is not affected by the gradient considered, all genotypes will have identical fitness curves; the same optima, valences and mean survival. Any difference in one or more of these three characteristics of the fitness curve results in differential survival among the genotypes, or selection. Figure 2-22 illustrates a few of the many possible conditions giving rise to selection, and the resulting equilibrium configurations in the absence of gene flow along the gradient.

Let $W_1(x)$, $W_2(x)$, and $W_3(x)$ be survival functions of position on the gradient, x. Let W_1 be the fitness function for genotype AA, W_2 for Aa, and W_3 for aa.

In figures 2-22a through d, examples are given for genotypes with equal optimum positions; the species is stenotopic or eurytopic with respect to the gradient considered. In 2-22a and b, the genotypes differ in mean probability of survival only (the area under their W curves); the implicit assumption in the classical population genetics formulae. In figure 2-22a the heterozygote has an identical mean survival to homozygote AA ($W_1(x) = W_2(x)$ for all x), and the mean survival of recessives is less than AA or Aa. In this situation, gene A would eventually become fixed throughout the species range. In figure 2-22b the heterozygote has a greater mean fitness than either homozygote ($W_2(x) > W_1(x)$ and $W_2(x) > W_3(x)$ for all x). This

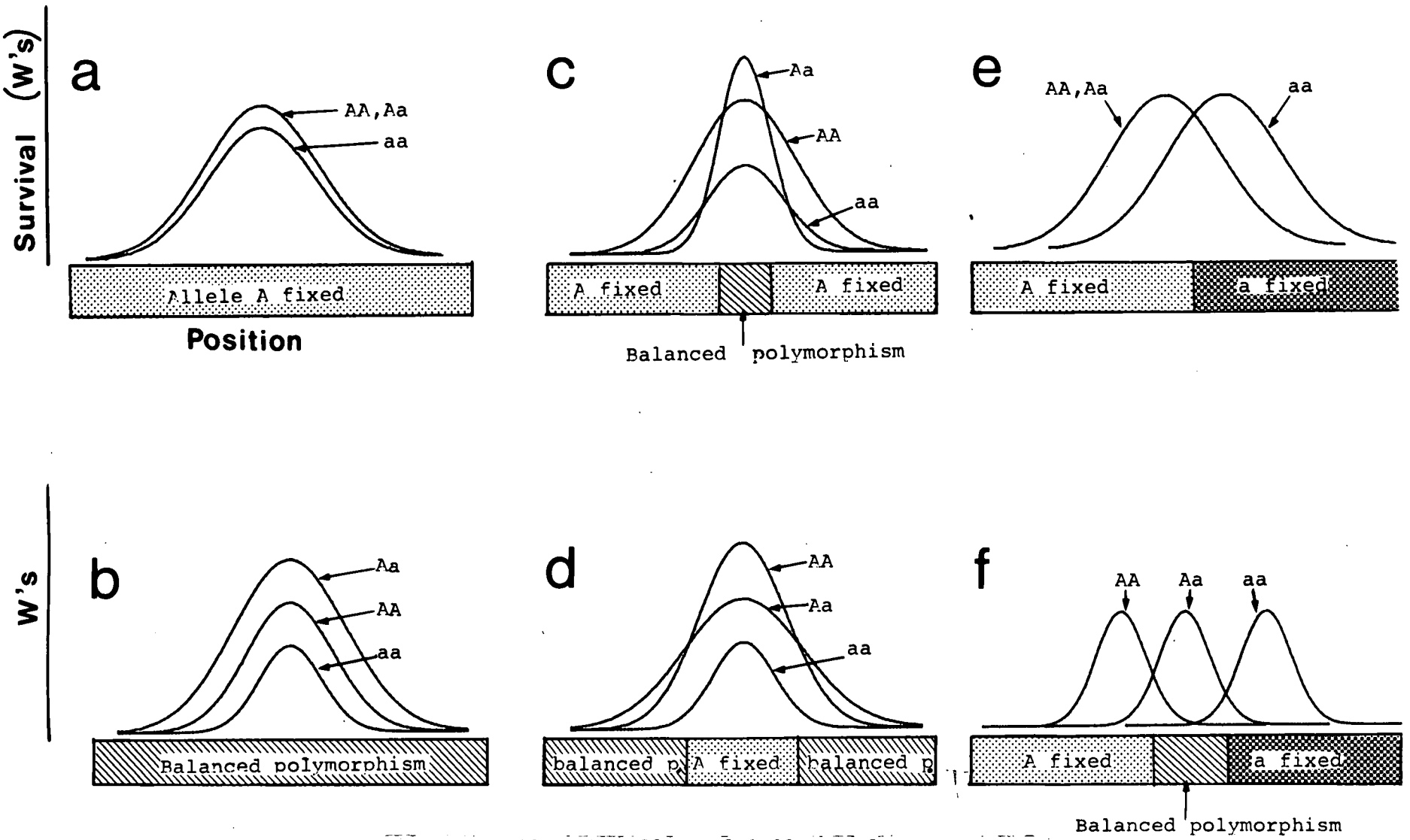


FIGURE 2 - 22. The effect of differing fitness curves.

will give rise to a balanced polymorphism throughout the species range.

In figure 2-22c and d the genotypes differ in valence as well as in mean probability of survival. In figure 2-22c the heterozygotes' mean survival is less than the homozygotes, but has greater probability of survival at or near its optima than do the homozygotes. This will result, in this case, if fixation of gene A near the periphery of the species range, but balanced polymorphism in the central part of the species range. Fixation at the periphery of species range is thus not necessarily a result of small population size, and, or, of restricted gene flow (as suggested by Mayr, 1963). In figure 2-22d the valence of the heterozygote is greater than either homozygote, and has a lower optimal survival than one homozygote. This will result in a balanced polymorphism at the periphery of the species range, and near fixation of gene A (in this case) near the centre. Such would be the case, if, for example, heterozygotes were better adapted for a wide range of conditions, but did not do as well as homozygotes at the optima.

In figure 2-22e and f two examples are given with differing optimum positions (polytopic species). In figure 2-22e the optima are relatively close together. This will give rise to an area fixed for gene A adjacent to an area fixed for gene a. In figure 2-22f the optima are further apart, and are distant enough so that the absolute fitness of the heterozygote is considerably greater than either homozygote in the centre of the species range. This will give rise to an area fixed for A adjacent to an area polymorphic for A and a, which is adjacent to an area fixed for gene a.

In general, if a given individual is a genotype with a greater

overall survival (often observable as greater area under its abundance curve in nature), and is physically close to its optimum position on the gradient, it will have a greater absolute probability as well as greater relative fitness compared with other genotypes in amphitopic and polytopic species. If it is not close to the genotypic optimum, its relative fitness in a given locality may be less than one or more than the other genotypes in the same locality, even though its mean fitness (area under fitness curve) may be greater (figure 2-22c,d,e,f). Overall mean survival values do not predict the outcome of a polymorphism without reference to the geography of local survival values; neither does calculation of genotype fitness at one locality necessarily predict the fitness at other localities.

If a cline is to form it is most likely to form in the region between the genotypic optima, where net genic selection changes direction. Such a position will be called the null point (np). In many species it is difficult to measure differential fitness among the genotypes; this suggests that the genotypic optima are spatially close to one another, and the fitness curves are similar, giving rise to gentle differential selection gradients (figure 2-23a). When the differences in measureable selection are much greater, the optima are likely to be further apart (figure 2-23b). Figure 2-24 illustrates eight ways in which absolute survival values of three genotypes may be arranged on a gradient.

A. In the dominant gradient model the absolute fitness of AA(W_1) and Aa (W_2) are equal in each deme, and form a selection gradient of different slope from that of genotype aa (W_3), as in figure 2-24a. Such would be the case if, for example, the probability of survival of dominants increased in a transect up a mountainside whilst the probability of survival of recessives decreased. Kimura (1958) analyzed this model mathematically, using relative fitnesses.

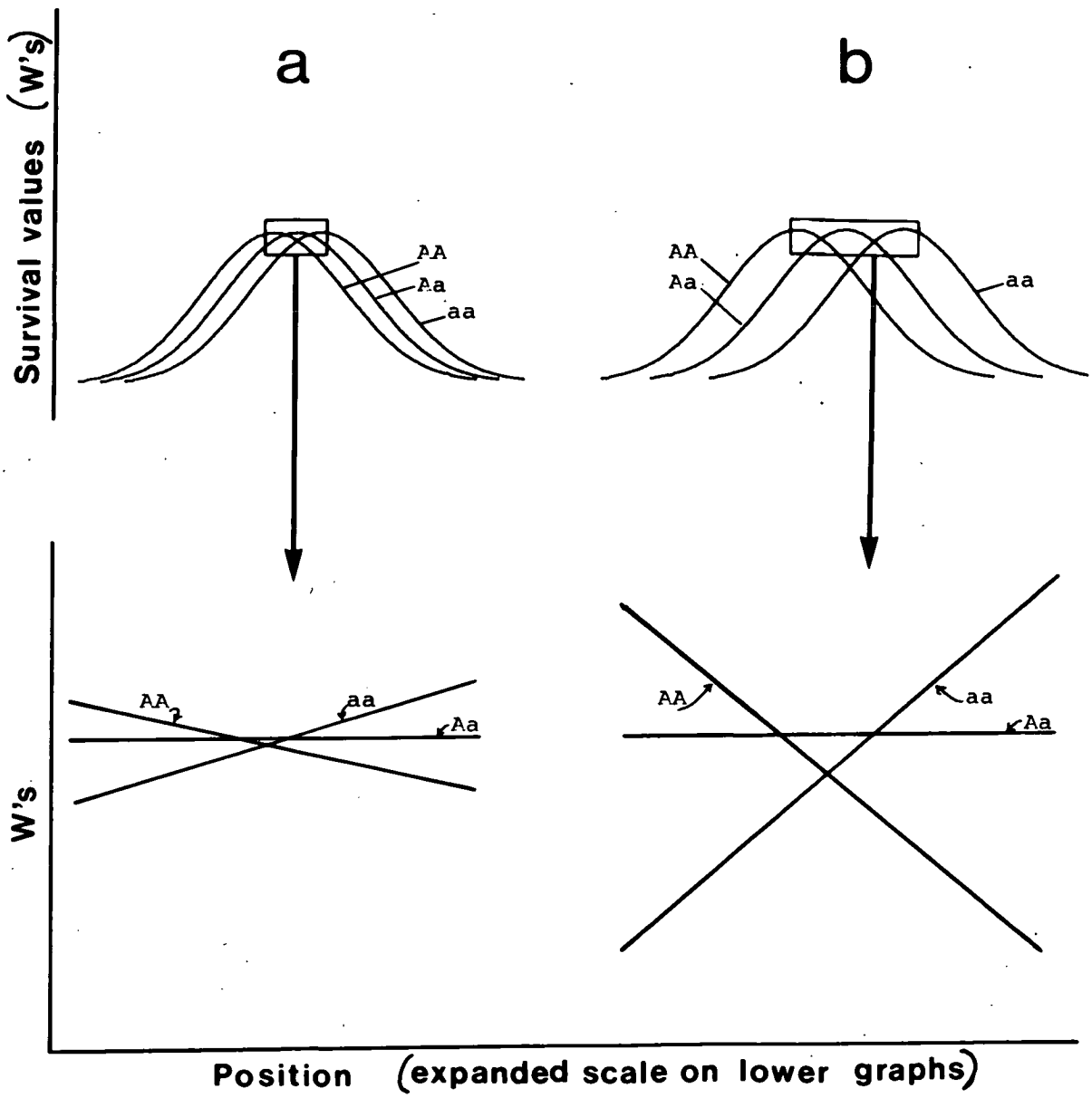


FIGURE 2 - 23. Weak and strong selective differences.

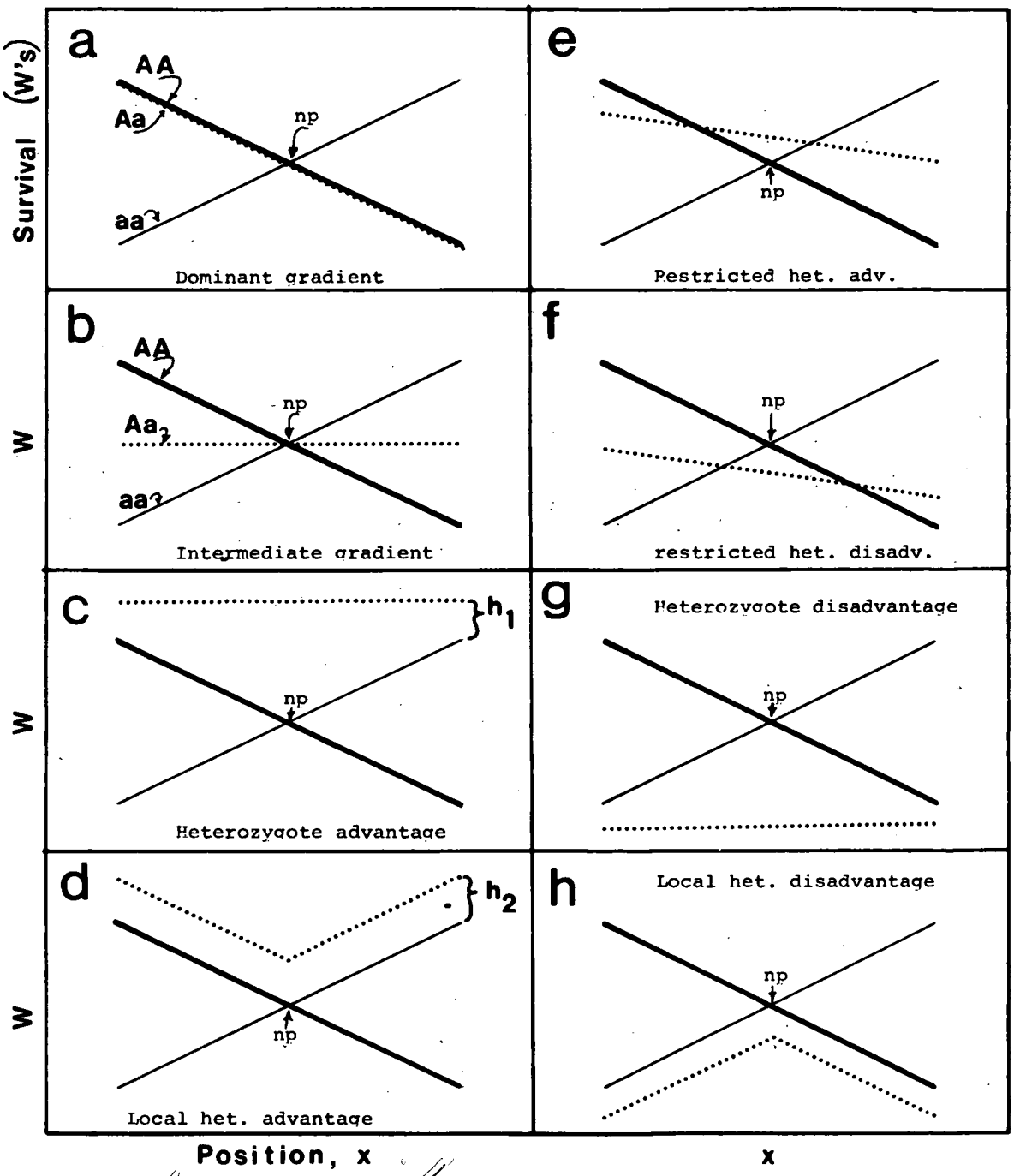


FIGURE 2 - 24. Models A through H.

B. In the intermediate gradient model the selection gradients of each genotype are different, and at some point (the null point) all fitnesses are equal (figure 2-24b). For example, this would happen if the position of the optimum for heterozygotes was located somewhere between the homozygote optima and the genotypes had similar fitness curves; in other words, in a transect up a mountainside in the vicinity of the optima, the survival of homozygote aa might decrease and the survival of Aa and AA might increase with distance at differing rates. Fisher (1950) and Slatkin (1971) analysed special cases mathematically, using relative fitnesses. In terms of absolute survival values, heterozygotes in Slatkin's model were everywhere exactly intermediate between the homozygotes at the same location ($W_2(x) = [W_1(x) + W_3(x)]/2$. Slatkin's model (1971) is the opposite extreme to Kimura's (1958) model.*

C. In the heterozygous advantage model the selection gradients of homozygotes (W_1 and W_3) have different slopes, and the probability of survival of heterozygotes is always greater than either homozygote by a minimum value h_1 (figure 2-24c). This would happen, if, for example, the optimum and mean fitness of heterozygotes was greater than either homozygote, and the heterozygote optimum was between the homozygote optima. The special case of heterozygotes having a constant fitness equal to 1.0 and $W_3(x) = 1 - W_1(x)$ has been considered mathematically by Clarke (1966).

D. In the local heterozygous advantage model the probability of survival of heterozygotes is always greater than either homozygote in the same position by a constant amount h_2 (figure 2-24d).

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*Slatkin's (1971) mathematical study duplicates some of the author's work, but was done independently.

E. In the restricted heterozygous advantage model the survival gradients are all different as in B, but in a restricted zone the heterozygotes have a greater probability of survival than either homozygote (figure 2-24e). This is probably one of the most realistic models as it is unlikely that at any one location all fitnesses could be exactly equal. This model applies to almost all cases in which positions of optimum survival or, and, valences of each genotype are unequal.

F. In the restricted heterozygous disadvantage model the survival curves are all different as in B and E, but in a restricted zone the heterozygotes have a poorer absolute probability of survival than have either of the homozygotes at the same position (figure 2-24f). This is also a fairly realistic model, as in E, but the optimum and mean survival of heterozygotes is smaller, It could also happen if the position of the optima of the heterozygote was nearer to one homozygote, its overall mean fitness low, but its ecological amplitude high. The same situation would give model E if the mean fitness of Aa was greater.

G. In the heterozygous disadvantage model the absolute survival of heterozygotes is always less than either homozygote (figure 2-24g). This would happen, if, for example, the mean fitness of heterozygotes was less than either homozygote, or if the optimum position of heterozygotes was far from either homozygote, but its valence high. It would be expected in contact zones between incipient species, or in cases where the "alleles" are actually chromosome arrangements (White, et al., 1964, 1967).

H. In the local heterozygous disadvantage model the probability of survival of heterozygotes is always less than either homozygote in the same position (figure 2-24h). This would happen if, for example,

the optimum position of heterozygotes was between the optima of the homozygotes, and the mean fitness and valence of heterozygotes was low. Such would be the case in the earlier stages of the development of pre- or post- mating sexual isolation.

I. An additional model is the frequency-dependent model (not illustrated), in which the probability of survival of each genotype is related to both its position on the gradient and its frequency at that position, by the relationship:

$$W_i(x) = 1 - s\ (U_i - f_i(x)\ ),\quad i = 1, 2, 3:$$

where U_i is the frequency of genotype i, whose "focal frequency", $f_i(x)$, depends upon the genotype's position, x, on the gradient, and s is the "strength" of the frequency-dependent relationship. The focal frequency is the optimum genotype frequency for a given position, or the genotype frequency at which the probability of survival is at a maximum for that position. Thus model I incorporated Clarke's (1964) model of frequency-dependent selection. Focal frequencies must not be confused with the optimum position on the gradient. In the frequency-dependent model (model I), the optimum position for a given genotype is the location on the gradient with the maximum focal frequency for that genotype.

Figures 2-25, 2-26, and 2-27 illustrate the equilibrium clines of each of the models with 40% gene flow (g=0.40) in a linear series of d=50 demes, each with a population size of N=100 individuals. I_1, I_2, and I_3 will represent the difference in absolute fitness between adjacent demes for genotypes AA, Aa, and aa, respectively. In the illustrated models I of a given genotype will be constant throughout the deme series; we will be examining only a small portion of the fitness curves, (fig. 2-23).

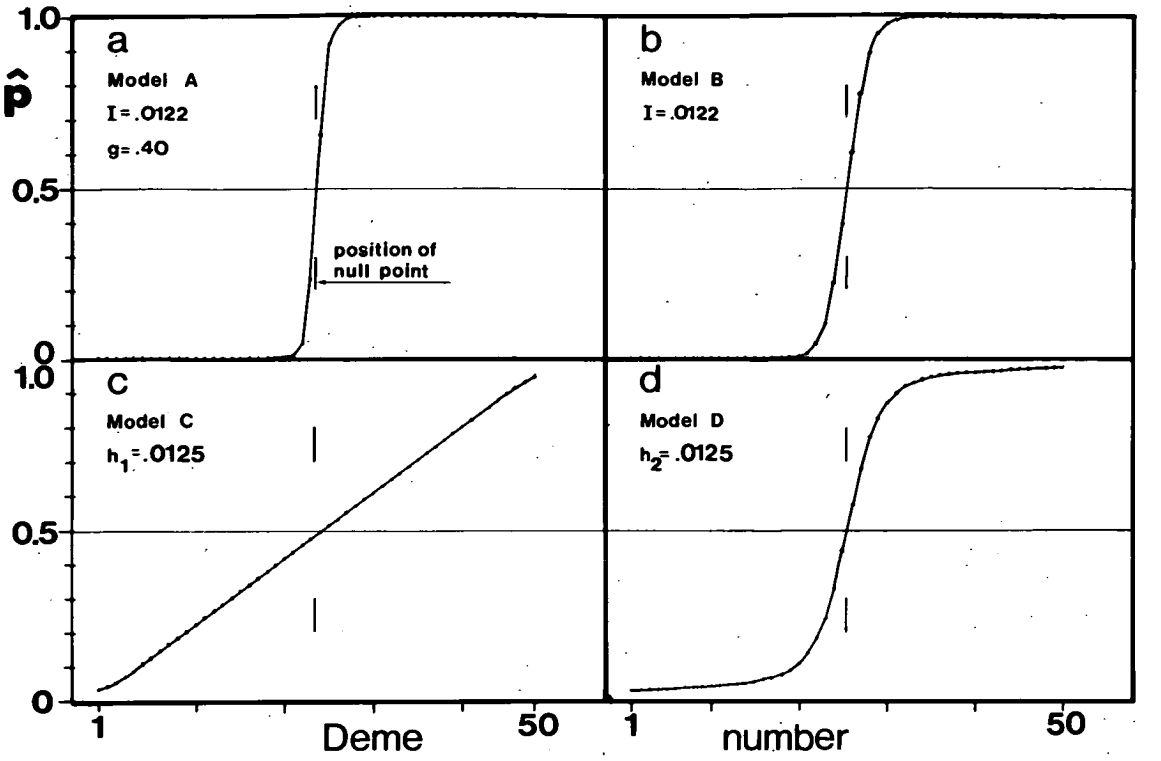


FIGURE 2-25

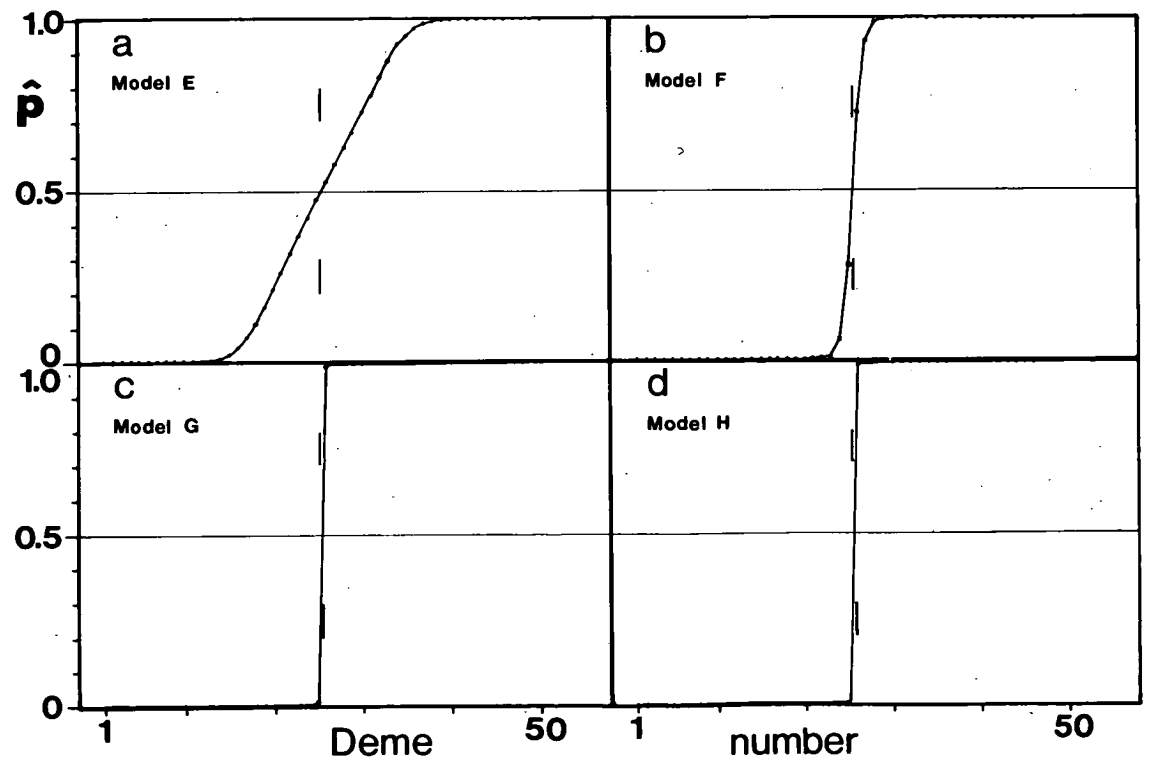


FIGURE 2-26

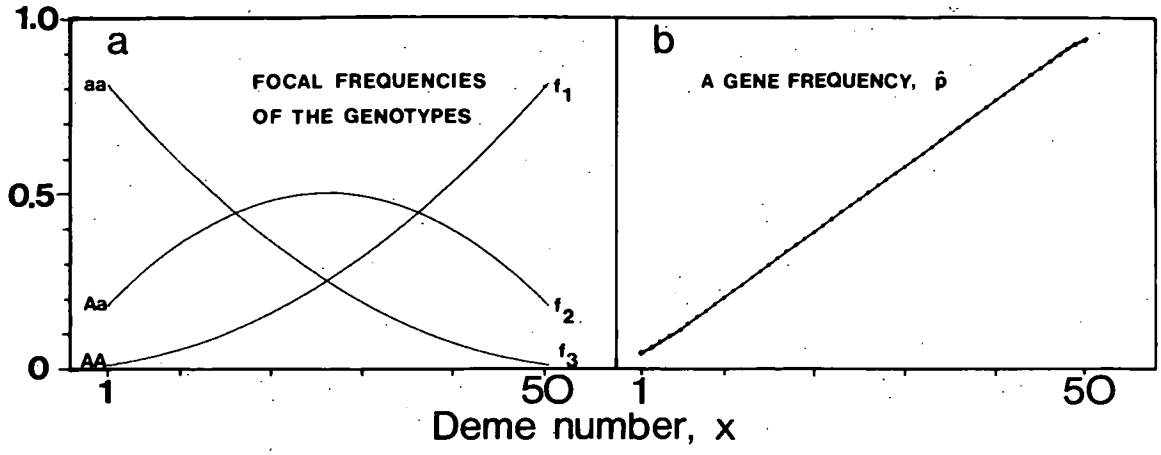


FIGURE 2-27
MODEL I

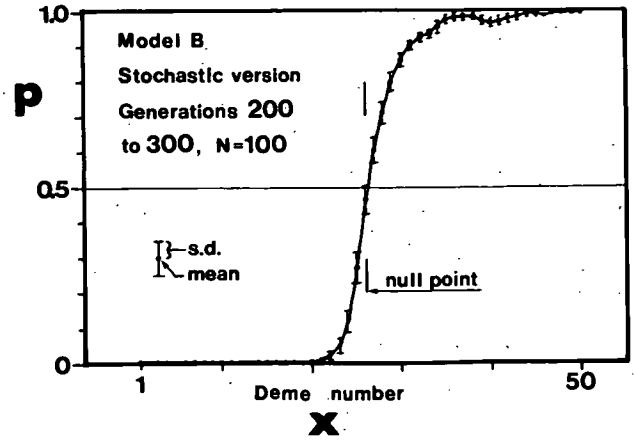


Figure 2-28

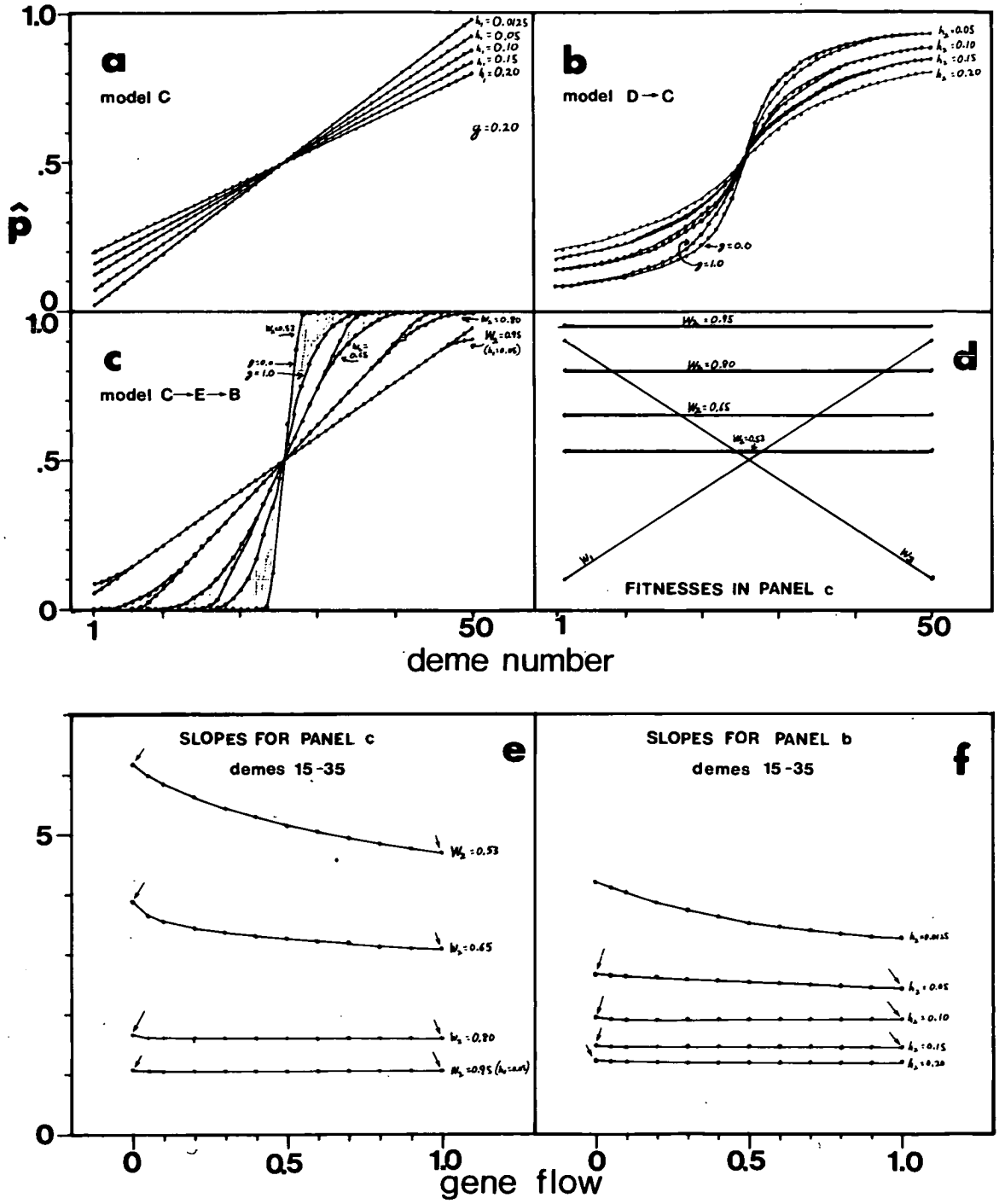

a
model C
b
model D→C
c
model C→E→B
d
FITNESSES IN PANEL c
e
SLOPES FOR PANEL c
demes 15-35
f
SLOPES FOR PANEL b
demes 15-35
p̂
deme number
gene flow
1.0
.5
0
1
50
5
0.5

FIGURE 2-29

Deterministic and Monte Carlo simulations were carried out to obtain the equilibrium clines, using a wide variety of selection and gene flow parameters. The programmes are found in Appendix 2. Because the Monte Carlo runs did not differ significantly from the deterministic simulations, only the latter will be illustrated. A stochastic run for model B is shown in figure 2-28 for comparison.

The dominant gradient model (A) produces a cline with a well defined morphotone in the vicinity of the null point (figure 2-25a). As found in Kimura's continuous model (1958), the steepest part of the cline is at a gene frequency slightly above 0.4. The intermediate gradient model (B) produces a very similar cline which is more symmetrical and slightly steeper (figure 2-25b). If the fitnesses of heterozygotes are always exactly intermediate between the homozygotes in the same deme (as in Slatkin, 1971), the steepest part of the cline goes through $\hat{p}= 0.5$ at the null point. The more similar W_2 to one of the homozygote fitness curves, the further the equilibrium gene frequency at the null point (np) will be from 0.5; if W_2 is more similar to W_1 then $\hat{p}_{np}$ will be closer to 0.4, and if W_2 is more like W_3 then $\hat{p}_{np}$ will be closer to 0.6. The steepest part of the cline always corresponds to the null point in models A and B. The form of the cline is similar for other values of N, g, and I_1, I_2, I_3. Clines are steeper for larger I (figure 2-31a), or steeper selection gradients.

The heterozygous advantage model (C) produces a straight linear cline (figure 2-25c) as shown by Clarke (1966). Its slope depends upon the amount of heterozygous advantage (h_1, figure 2-24c), and the slopes (I) of the homozygotes' selection gradients. The slope of the cline is steeper if either or both homozygotes have steeper selection gradients (larger I), and is more gentle if h_1 is larger (figure 2-29a). The strength of heterozygous advantage, h_1 has a

greater effect on the slope of a model C cline than any other parameter.

The local heterozygous advantage model (D) produces a cline with a morphotone in the vicinity of the null point as do the gradient models (figure 2-25d). The cline becomes more gentle, and the morphotone less distinct as h_2 increases, even for large I, as in model B (figure 2-29c). As h_2 approaches zero model D approaches one of the gradient models, depending upon the form of the fitness curves (fig. 2-29b).

The restricted heterozygous advantage model (E) and the restricted heterozygous disadvantage model (F) produce clines with morphotones as in the gradient models (figure 2-26a and b). Model F produces steeper clines and more distinct morphotones than does model E as one might expect, but the differences are not very great. As in all models the steeper the selection gradient, the steeper the clines. As h_1 decreases, model E approaches one of the gradient models, depending upon the shapes of the fitness curves (figure 2-29c).

The heterozygous disadvantage model (G) produces the steepest cline and most distinct morphotone of any of the models, as one would expect from the low fitness of heterozygotes (figure 2-26c). The local heterozygote disadvantage model (H) also produces very distinct morphotones (figure 2-26d). The effect of different slopes of selection gradients is less than in models A through F.

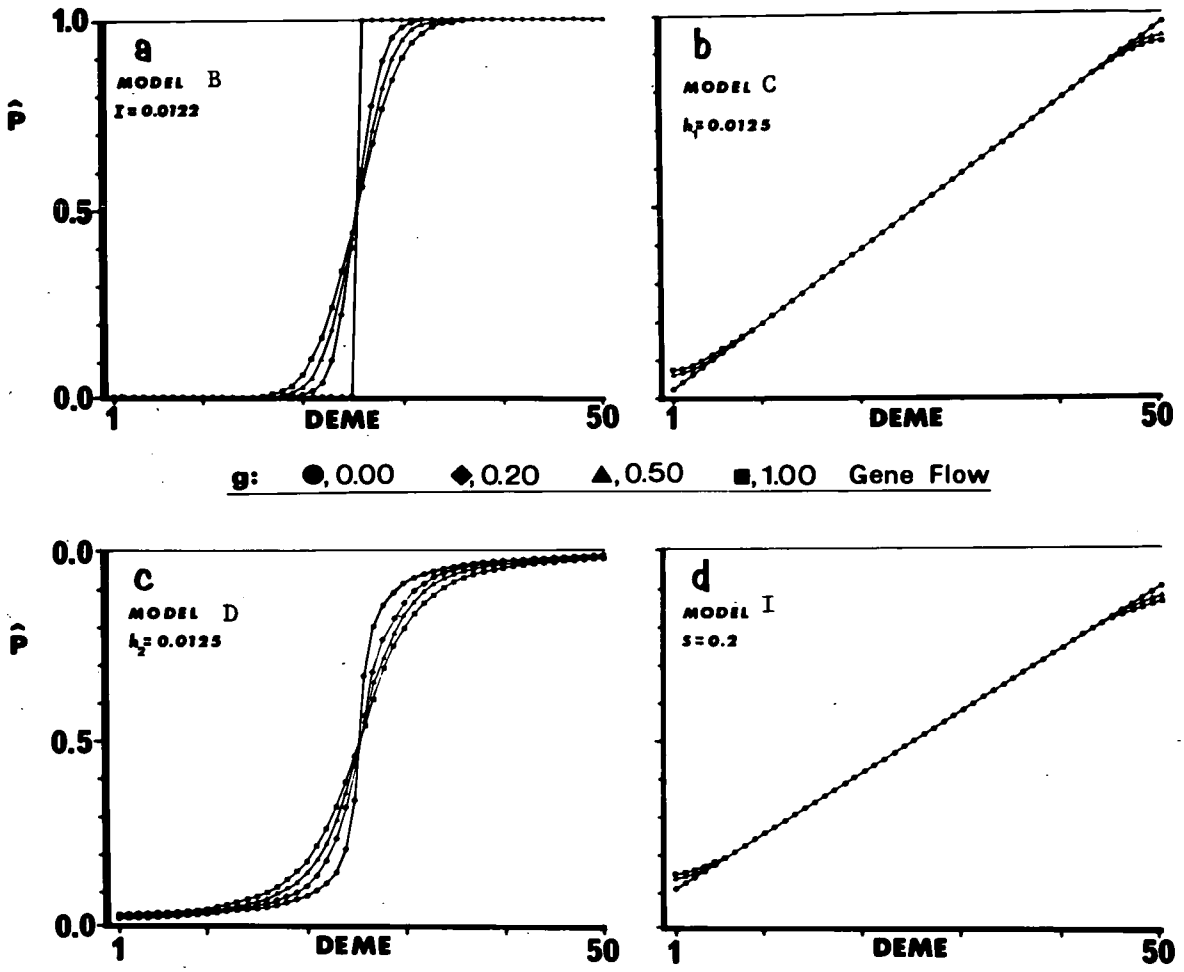
The frequency-dependent model (I) produces a cline which depends upon the spatial arrangement of the focal frequencies (f_1, f_2, f_3) among the demes. If the focal frequencies are arranged as the Hardy Weinberg ratios in a given position on the gradient and the Hardy-Weinberg ratios are arranged as in a linear series of gene frequencies,

(fig. 2-27a) then a linear cline is formed (figure 2-27b). The steeper the slopes of the focal frequencies the steeper the clines, as in the previous models. The strength of frequency-dependence (s) affects only the resistance of the cline to the effects of gene flow.

The nine models produce basically two kinds of clines; a sigmoid cline with a well marked morphotone (models A, B, D, E, F, G, H), and a smooth linear cline (models C, and I with focal frequencies arranged as in figure 2-27a). All clines would be expected to be made smoother and more gentle by the effects of gene flow. The effects of varying levels of gene flow (g) are shown in figures 2-30, 2-31, for four models and both classes of clines.

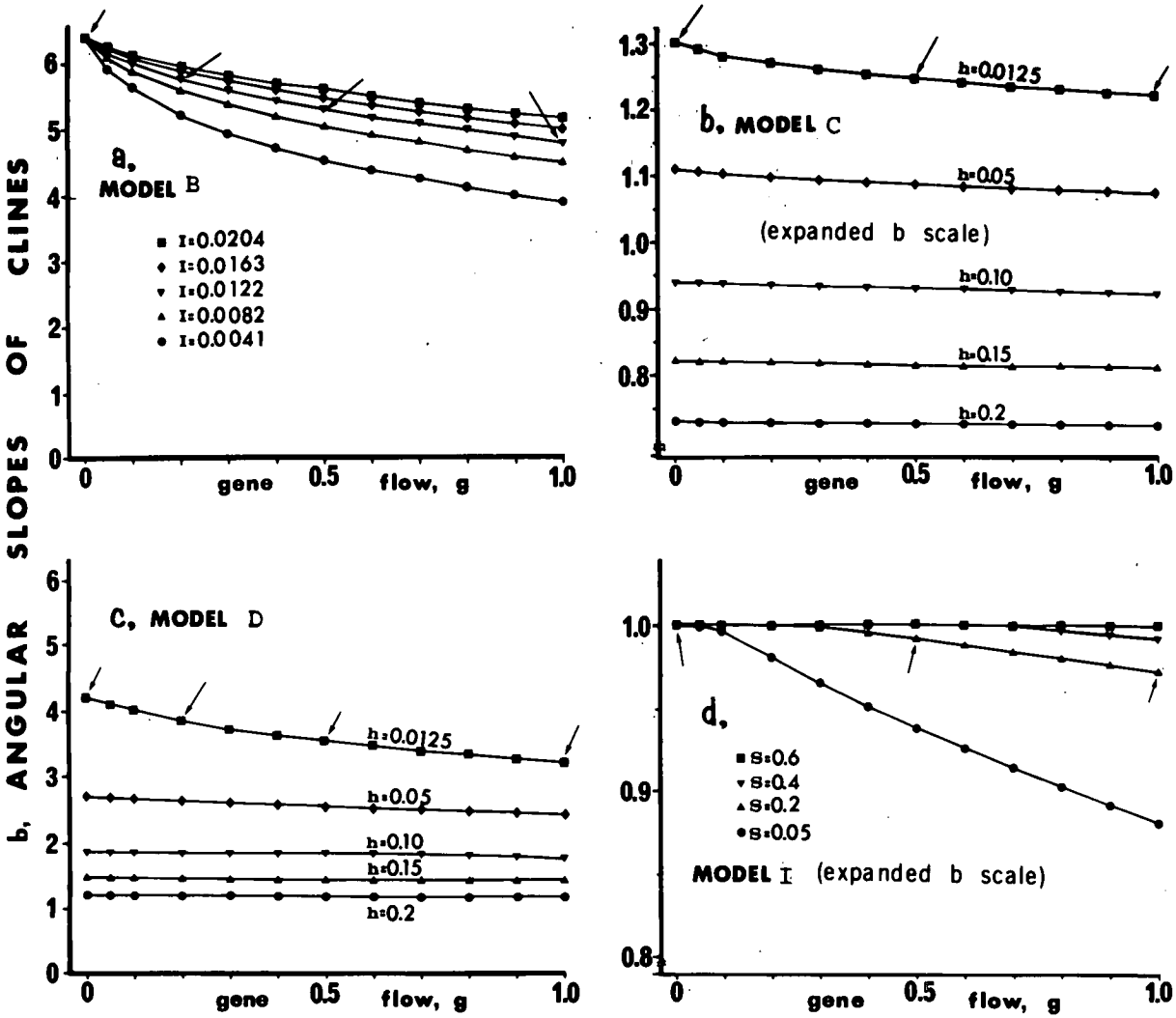
Figure 2-30 and 2-31 (from Endler, 1972, appendix 3) illustrate the effect of various amounts of gene flow on the intermediate gradient model (B), the heterozygote advantage model (C), the local heterozygote advantage model (D), and the frequency-dependent model (I), for d=50 demes and N=100 individuals per deme. Similar results were obtained for other values of d and N.

The intermediate gradient model (B) produces a cline with a well defined morphotone for all but the weakest selection gradients (figures 2-30a and 2-31a). The greatest effect of gene flow on slope is found at low levels of gene flow and weak selection gradients (low g and I's). As the slopes of one or more selection gradients increase the attenuation of slope due to gene flow is progressively reduced (figure 2-31a). For very weak gradients (I's very small) the differentiation into two adjacent areas of high and low gene frequency may be reduced, but for moderate to strong selection gradients the differentiation may be very sharp, even for 100% gene flow (g= 1.0 in figure 2-30a). It should be emphasized that in this and in



EQUILIBRIUM CLINES AND LEVELS OF GENE FLOW IN FOUR MODELS

FIGURE 2 - 30



CLINAL SLOPE vs. GENE FLOW AND SELECTION INTENSITY IN FOUR MODELS

FIGURE 2 - 31

the other models there is no sharp environmental change (figure 2-24).

The heterozygous advantage model (C) produces a roughly linear cline for all levels of gene flow except near the edges of the deme series (figure 2-30b). For a given selection gradient ($I_1 = I_3 = 0.0125$, $I_3 = 0.0$ in figure 2-30 b) there is negligible change of slope for increased gene flow)figure 2-30b, 2-31b). Gene flow has a slightly greater effect when the cline in the absence of gene flow is nearly flat. Random fluctuations in a natural cline following this model would probably obscure any differences due to gene flow. The amount of heterozygous advantage (h_1) is the major determinant of clinal slope in model C.

The local heterozygous advantage model (D) produces a cline with a morphotone in the vicinity of the null point as in the gradient models (figure 2-30c). For small values of local heterozygous advantage, h_2, the clines and morphotones for various values of g approach those of the gradient models. As a result of the morphotone, the effect of gene flow is more noticeable than it is in model C, especially for weak selection (small h_2 in figure 2-31c), but the clines produced are nearly as insensitive to the effects of gene flow as is model B. Like the gradient model, most of the attenuating effect of gene flow takes place for small values of gene flow ($0<g<0.3$), and progressively decreases for the same changes around large values of g. However, for very large magnitudes of gene flow, there is still a well defined morphotone (figures 2-30c and 2-31c).

The frequency-dependent model (I) produces a cline similar to model C in form and properties (figure 2-30d). For moderate to strong selection intensities ($s>0.1$) the effect of gene flow is very small, but for very weak selection ($s<0.1$) the cline may become noticeably

flattened for the largest levels of gene flow (figure 2-31d).

Models B, C, E, F, and G are distinguished primarily on the position of the heterozygote's fitness curve. Rearranging the models in order of decreasing mean fitness of heterozygotes, with the same homozygote fitness curves, we have C, E, B, F, and G (figure 2-32). With decreased heterozygote mean fitness the slope of the resulting clines is steeper, morphotones are more distinct, and sensitivity to gene flow greater.

On the other hand, the effect of assortative mating is very much less. Figure 2-33 illustrates the effect of various levels of positive assortative mating on model A. The assortative mating model is that of O'Donald (1960). There is almost no change in the gene frequency cline when assortative mating is varied between 0 and 100% although the genotype-frequency clines become steeper.

In all the models the effect of gene flow is small, and does not prevent the formation of morphotones on smooth environmental gradients. Differentiation into two areas of high and low gene frequency can evolve along smooth selection gradients with differences in absolute survival between adjacent demes as little as I=0.0008 in spite of considerable gene flow (figure 2-31a). Such fitness gradients would be difficult to measure in natural populations. Large amounts of selection and sharp environmental changes are not required to allow morphotones to evolve.

The same conclusions apply to demes arranged on a hexagonal array in two dimensions, except that the effect of a given level of gene flow (g) is much less. In the linear series of demes a given deme consists of a fraction g of aliens after gene flow, and adjacent demes exchange g/2 individuals each generation. In the hexagonal array of

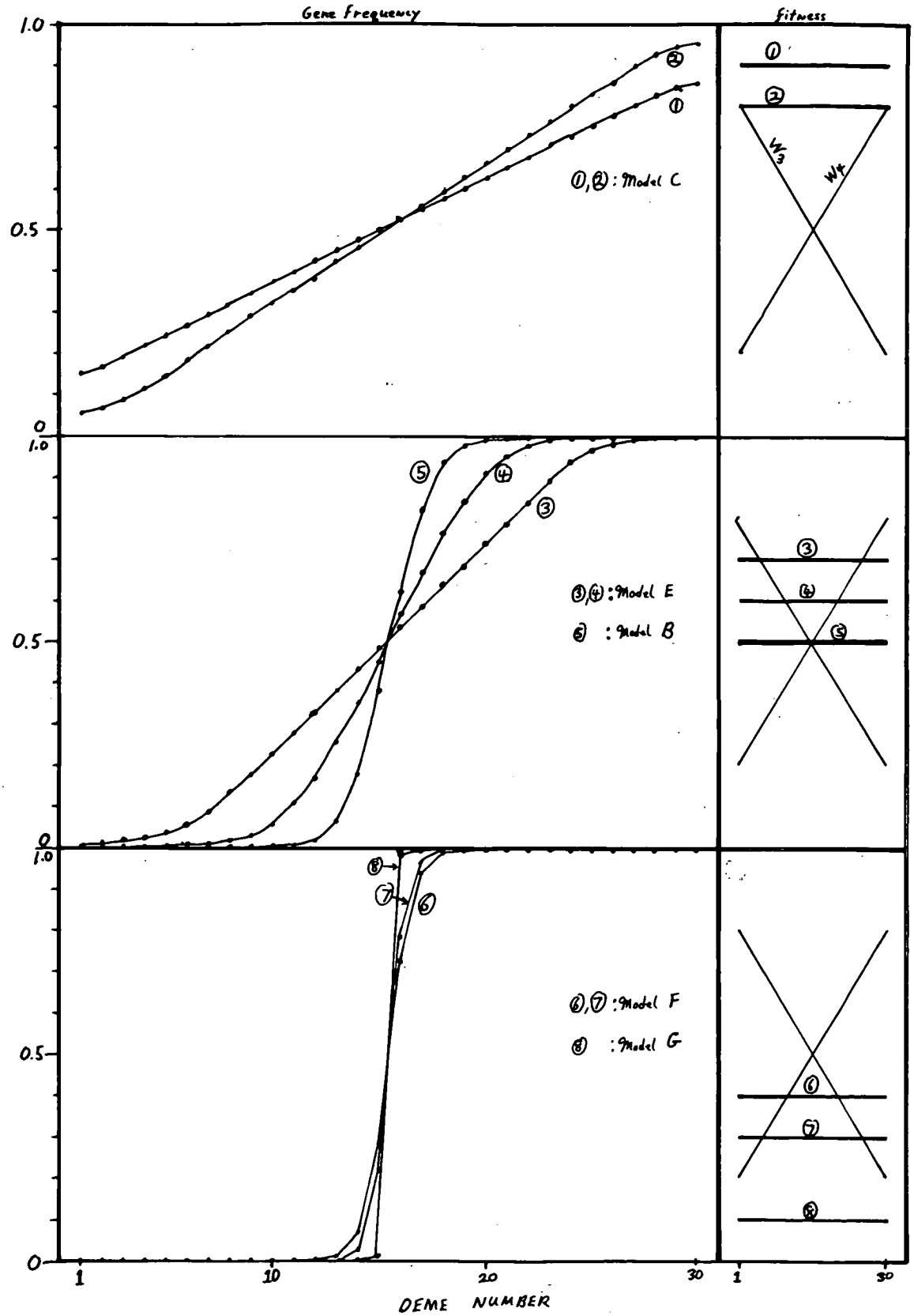


FIGURE 2 - 32. The interrelationships among the models. The horizontal line in the right-hand panels represents various levels of the fitness of heterozygotes. It need not be level.

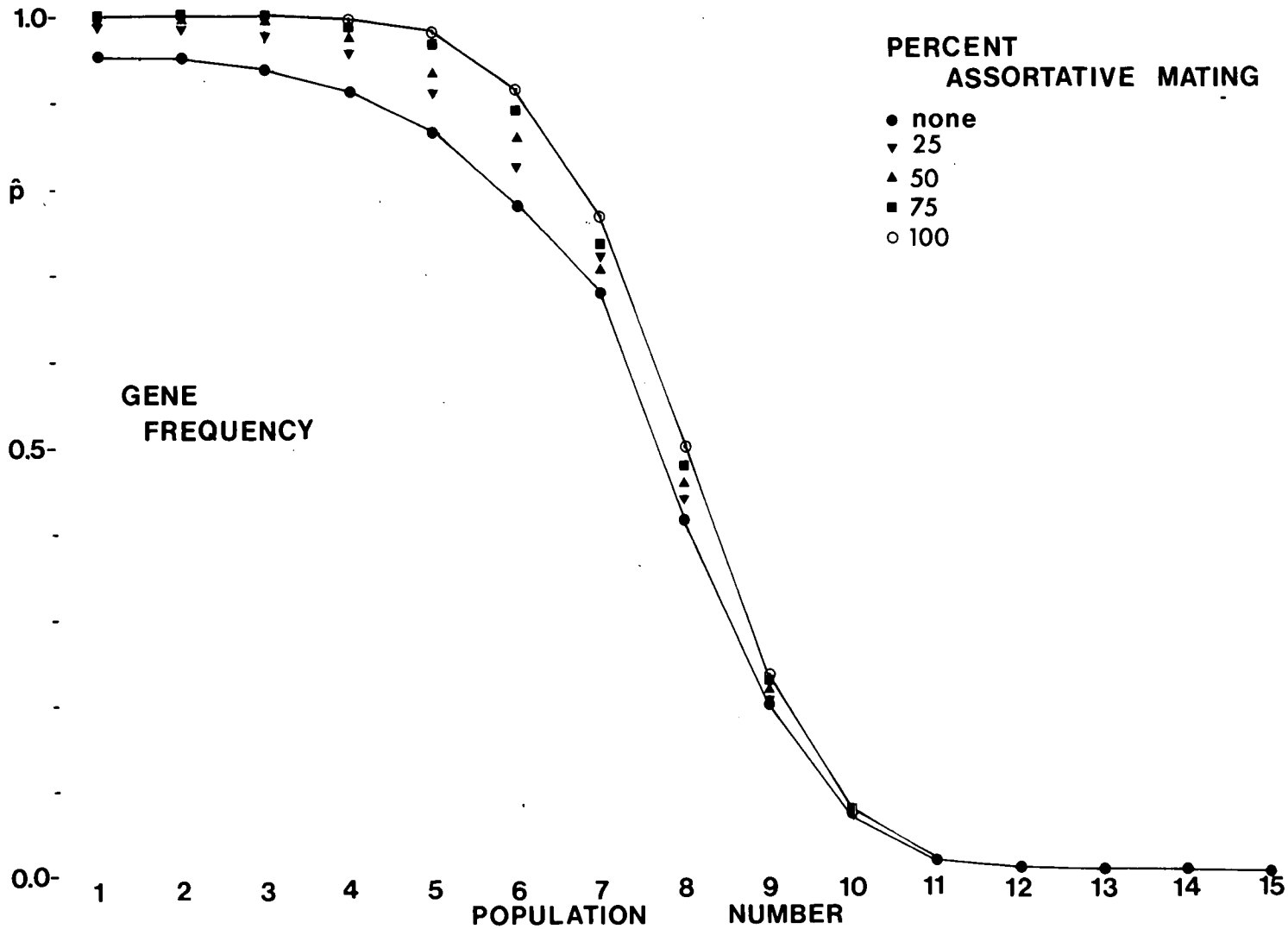

1.0-
p̂
0.5-
0.0-
GENE
FREQUENCY
PERCENT
ASSORTATIVE MATING
none
25
50
75
100
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
POPULATION NUMBER

FIGURE 2-33

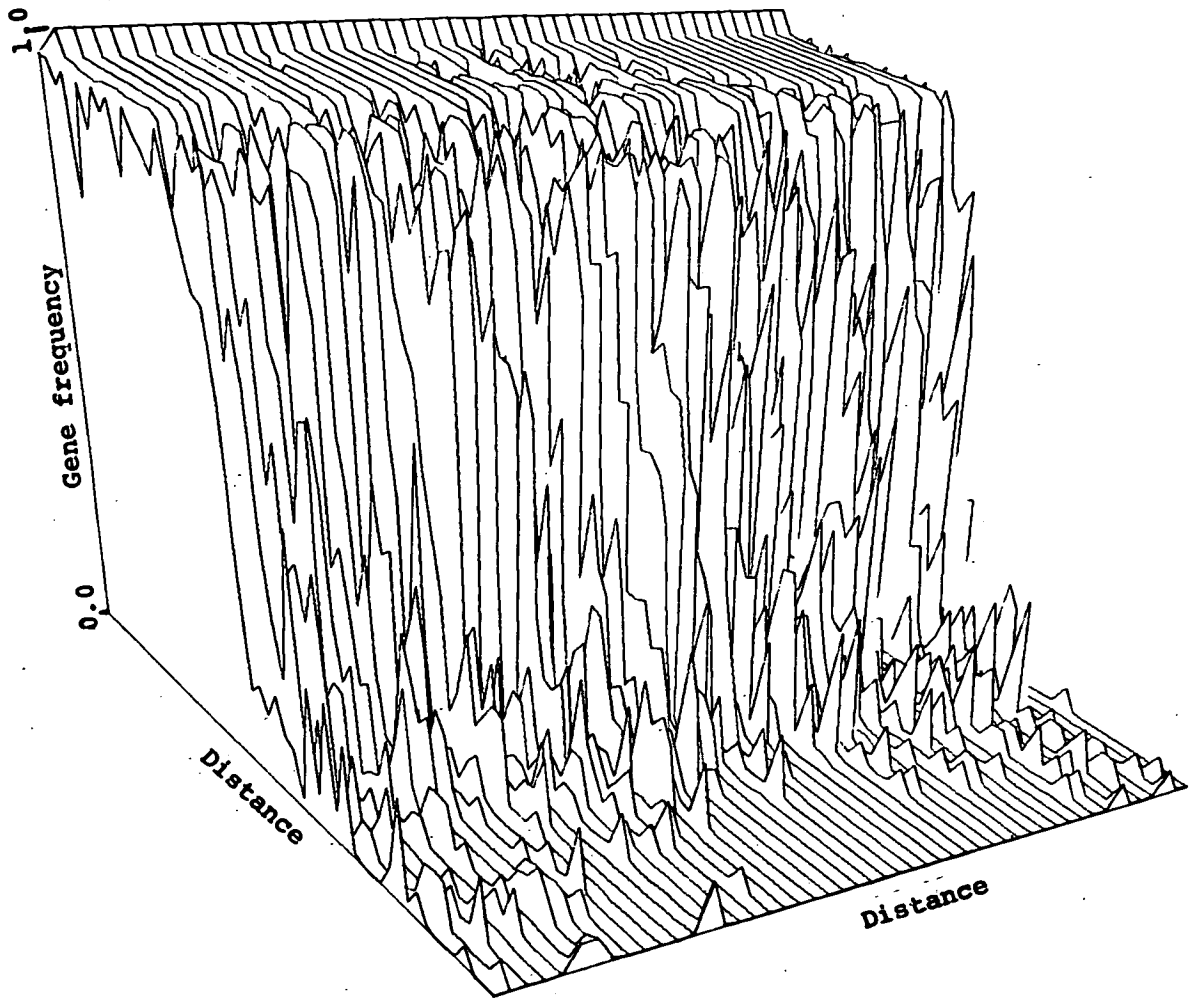


Figure 2 - 34

A TWO-DIMENSIONAL MONTE CARLO CLINE SIMULATION

demes a given deme also consists of g aliens after gene flow, but adjacent demes exchange only g/6 individuals. Thus, along a linear transect through a hexagonal array of demes, along an axis of gene flow, the gene flow along the transect is g/6 compared to g/2 if the transect were along a linear series. Thus the slope of a cline for g=0.6 (for example) in the two dimensional grid, is equivalent in slope and conformation to a cline on a linear series with g=0.20. Figure 2-34 illustrates the result of model B ($I_1 = I_3 = 0.025$, $I_2 = 0$) with g=0.20, and each deme containing 10 individuals. Figure 2-34 is a Monte Carlo simulation.

The conclusions also apply to more than one locus, even if the loci are linked. Using model B ($I_1=I_3=0.0015$, $I_2=0$) in a two-locus model for both loci independently, the fitness gradients for locus A were made to go from left to right and the fitness gradients for locus B were made to go from top to bottom on the hexagonal array of demes. Alleles A and B were assumed to be dominant and noninteractive and the probability of survival of a given phenotype was determined by the product of the fitness of phenotype with respect to locus A and locus B in the phenotype's deme. Recombination was 0.001, and initially all demes had 50% of each gene, but only "coupling" chromosomes. The resulting gene frequency clines are found in figure 2-35 and the phenotype frequencies in figure 2-36. It can be seen that the low level of recombination does not prevent the formation of perpendicular clines for both loci and independent morphotones in each cline. The four phenotypes (and the chromosomes) segregate spatially into four distinct areas (figure 2-36) with very distinct morphotones between them. There are no sharp changes in the environment at any part of the area. All selection gradients are smooth; W_1 ranges from

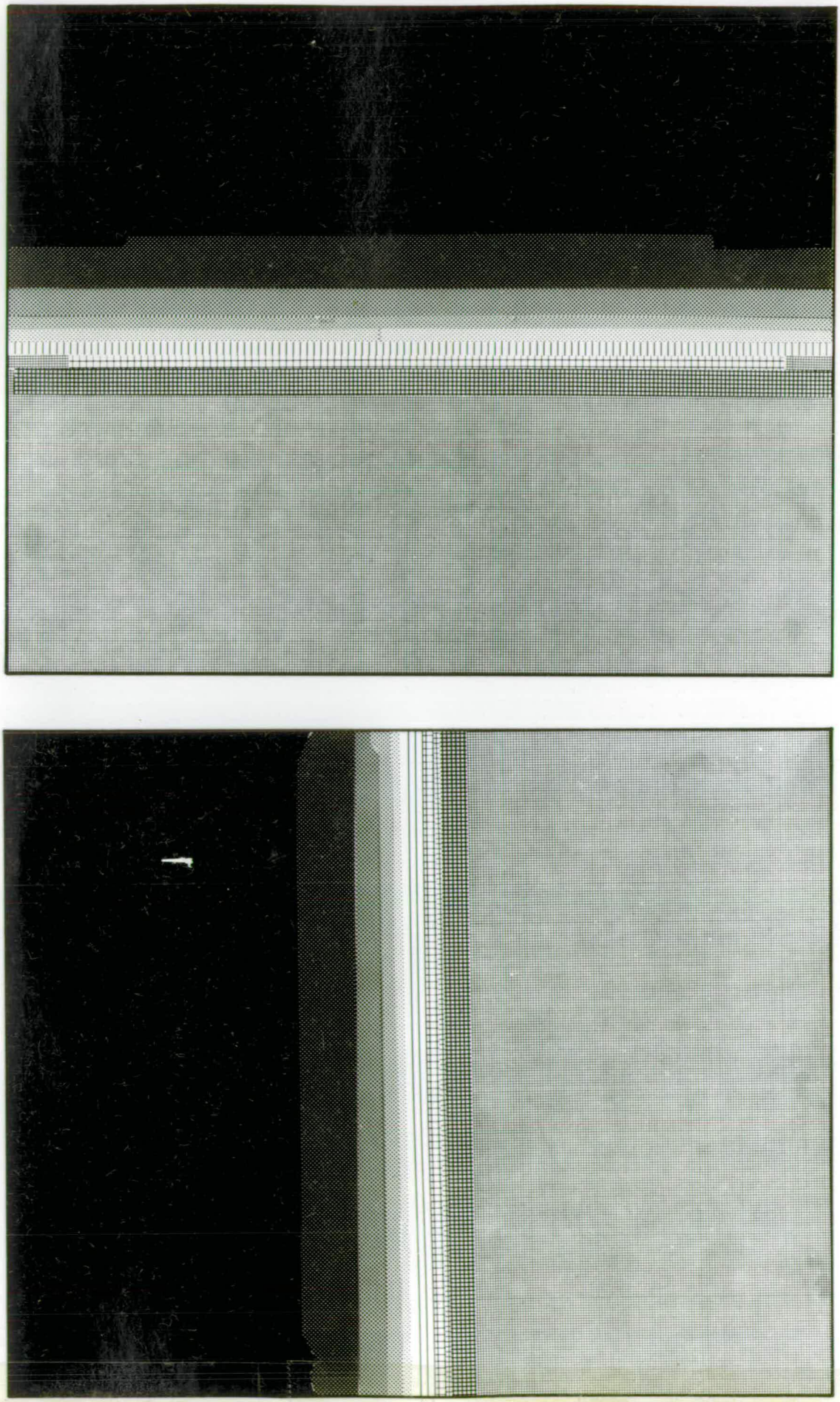


FIGURE 2 - 35. Gene A (above) and gene B (below) frequency clines in a two-locus model with 0.1% recombination. Legend as in figure 2 - 10.

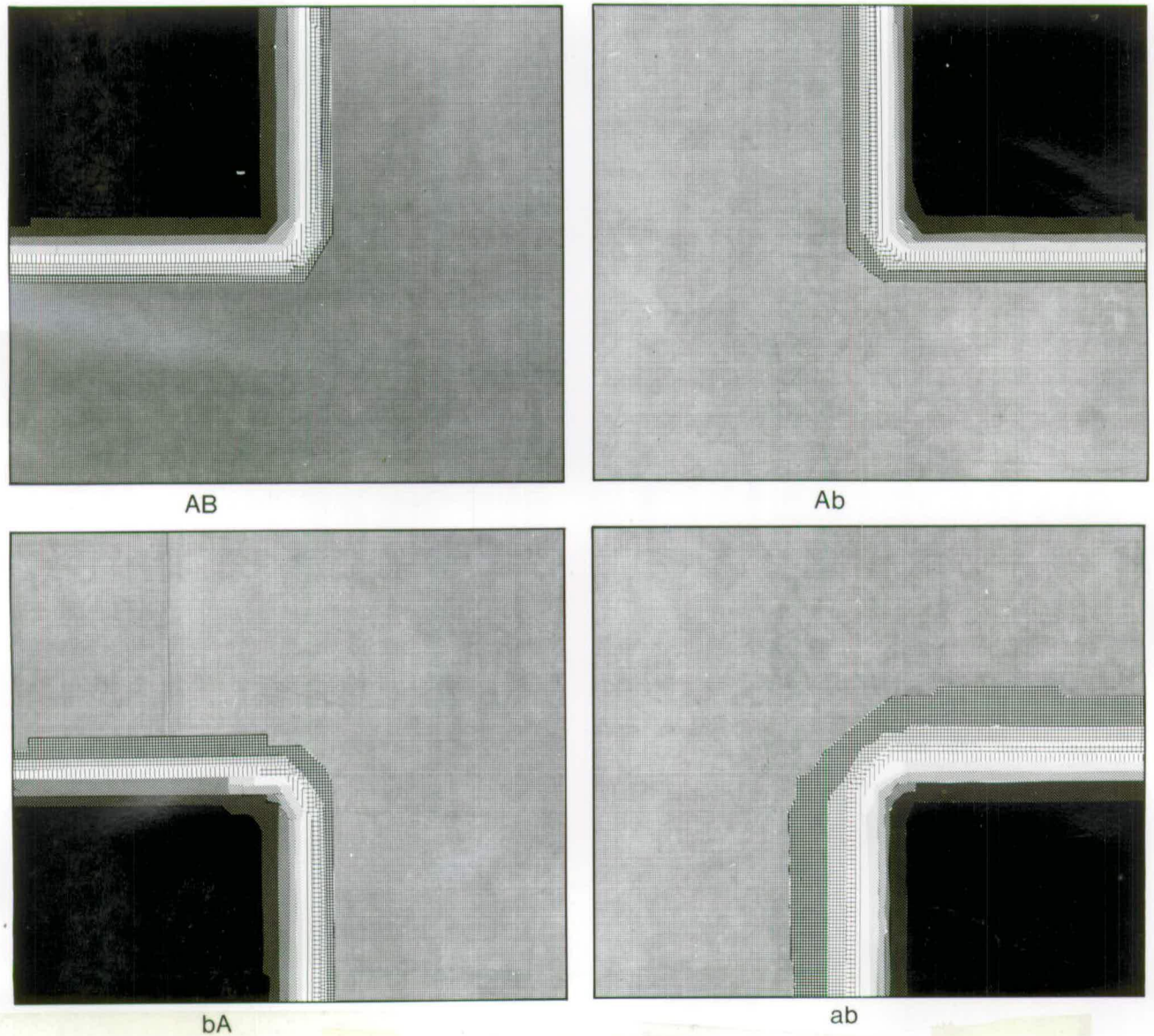


FIGURE 2 - 36. Phenotype frequency clines in the two locus model. Legend as in figure 2 - 10.

0.45 through 0.55, W_2 is constant and equal to 0.50, and W_3 ranges from 0.55 through 0.45, from left to right for locus A, and from top to bottom for locus B. Such changes in selection would be extremely difficult to detect in a natural population. This is one possible explanation for the apparently independent area effects for linked loci observed in Cepaea (for example Cain and Currey, 1963a, 1968, Wolda, 1969a,b, Jones, 1971) and Partula (Clarke, 1968, 1971). There is no reason, of course, why these area effects should result from selection following model B; there are at least seven other models which would yield the same spatial pattern! (A,D,E,F,G,H, and I if focal frequencies are not arranged for a linear change in gene frequency, see also section 2.6)

If all components of selection are known, the steepest part of a cline, or its morphotone (if present) will fall in the vicinity of the null point. However, in many cases we may not be able to measure all components of fitness. This is an especially difficult problem in interpreting natural clines because the morphotone expected as a result of one component of fitness may be shifted away from the known null point, or may be destroyed, by additional selective factors.

Taking model A as an example, consider a linear series of d=30 demes, each with N=100 individuals, g=0.20, $W_1=W_2$, and $I_1=I_2=I_3=0.01$. (I = difference in selection between adjacent demes). Let W_1 and W_2 range from 0.75 through 0.46 and W_3 range from 0.25 through 0.54, giving a null point at deme 26. As expected this yields a sigmoid cline with a morphotone centred around deme 26. (Curve a, figure 2-37a). Now, suppose that for some reason, allele A has a dominant deleterious effect throughout the deme series which is independent and additional to the clinal selection. As the deleterious effect of A is increased the morphotone is shifted away from the position predicted on the

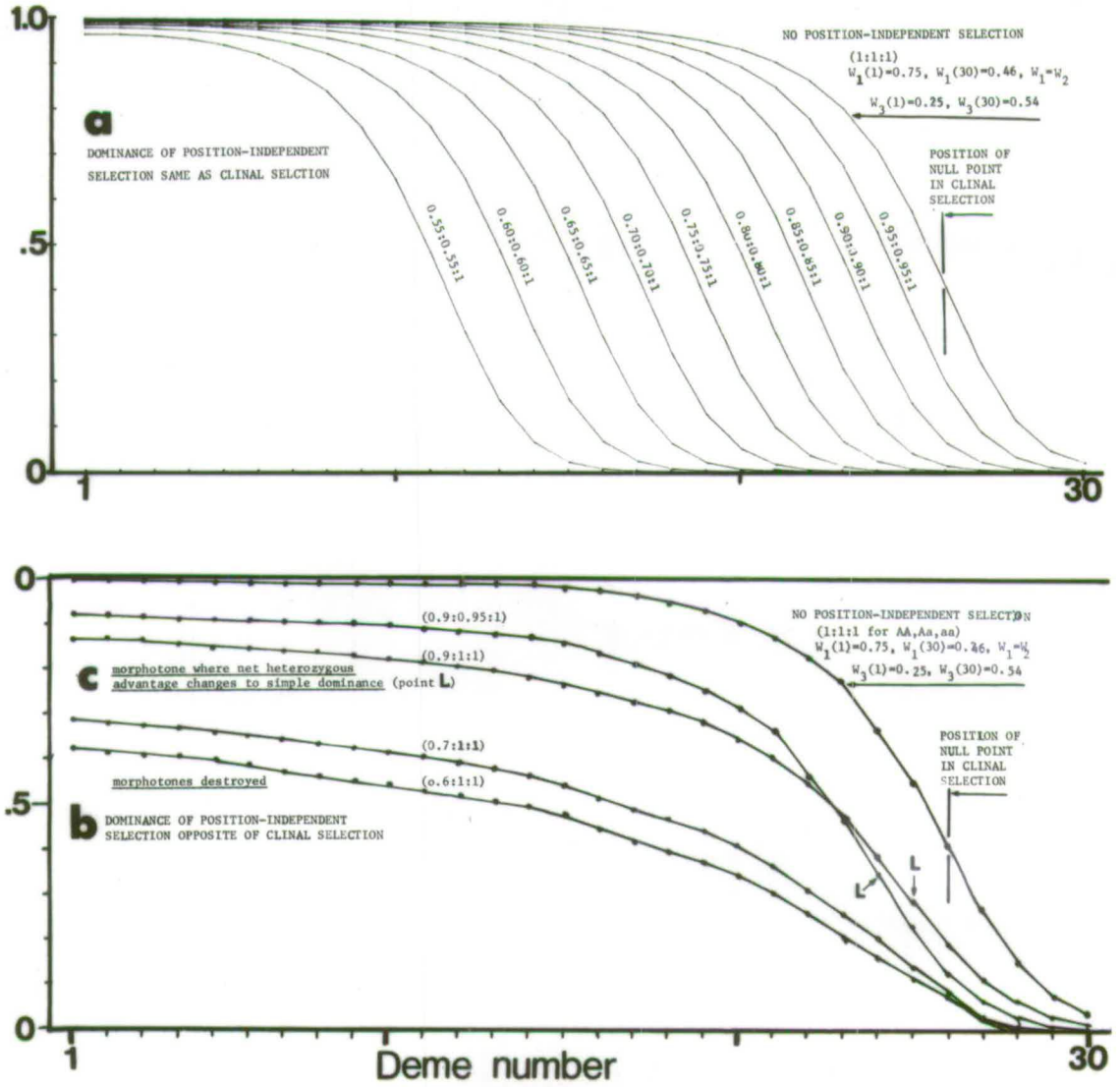

1.0
.5
0
1
30
a
DOMINANCE OF POSITION-INDEPENDENT
SELECTION SAME AS CLINAL SELCTION
NO POSITION-INDEPENDENT SELECTION
(1:1:1)
$W_1(1)=0.75$, $W_1(30)=0.46$, $W_1=W_2$
$W_3(1)=0.25$, $W_3(30)=0.54$
POSITION OF
NULL POINT
IN CLINAL
SELECTION
0.55:0.55:1
0.60:0.60:1
0.65:0.65:1
0.70:0.70:1
0.75:0.75:1
0.80:0.80:1
0.85:0.85:1
0.90:0.90:1
0.95:0.95:1
0
.5
0
1
30
Deme number
(0.9:0.95:1)
(0.9:1:1)
c morphotone where net heterozygous
advantage changes to simple dominance (point L)
(0.7:1:1)
(0.6:1:1)
morphotones destroyed
b DOMINANCE OF POSITION-INDEPENDENT
SELECTION OPPOSITE OF CLINAL SELECTION
NO POSITION-INDEPENDENT SELECTION
(1:1:1 for AA,Aa,aa)
$W_1(1)=0.75$, $W_1(30)=0.46$, $W_1=W_2$
$W_3(1)=0.25$, $W_3(30)=0.54$
POSITION OF
NULL POINT
IN CLINAL
SELECTION
L
L

FIGURE 2-37

basis of the null point of clinal selection only (figure 2-37a). The morphotone is shifted in the direction of the deleterious gene in proportion to the reduction of fitness of AA and Aa. The shifting is greater if the slope of selection gradients are smaller.

Now suppose the deleterious effect is only found in homozygotes (AA). In this case the morphotone is destroyed (figure 2-37b) as the overall selection pattern follows model C; heterozygous advantage throughout the deme series.

Now suppose the deleterious effect is exactly intermediate in dominance; heterozygotes have a smaller probability of survival than aa homozygotes (aside from clinal selection), and the probability of survival of AA genotypes is half that of heterozygotes. Again the morphotone is destroyed (figure 2-37c). This condition results in a combination of models B and C; in the demes to the left of deme 18 there is heterozygous advantage, but heterosis disappears to the right of deme 18. The spatial change from heterozygous advantage to intermediate dominance is gradual, yet can, under certain conditions, give rise to a morphotone at the changeover point. This additional cause of morphotones may also be extracted from the models of Levins and MacArthur (1966) and Slatkin (1971).

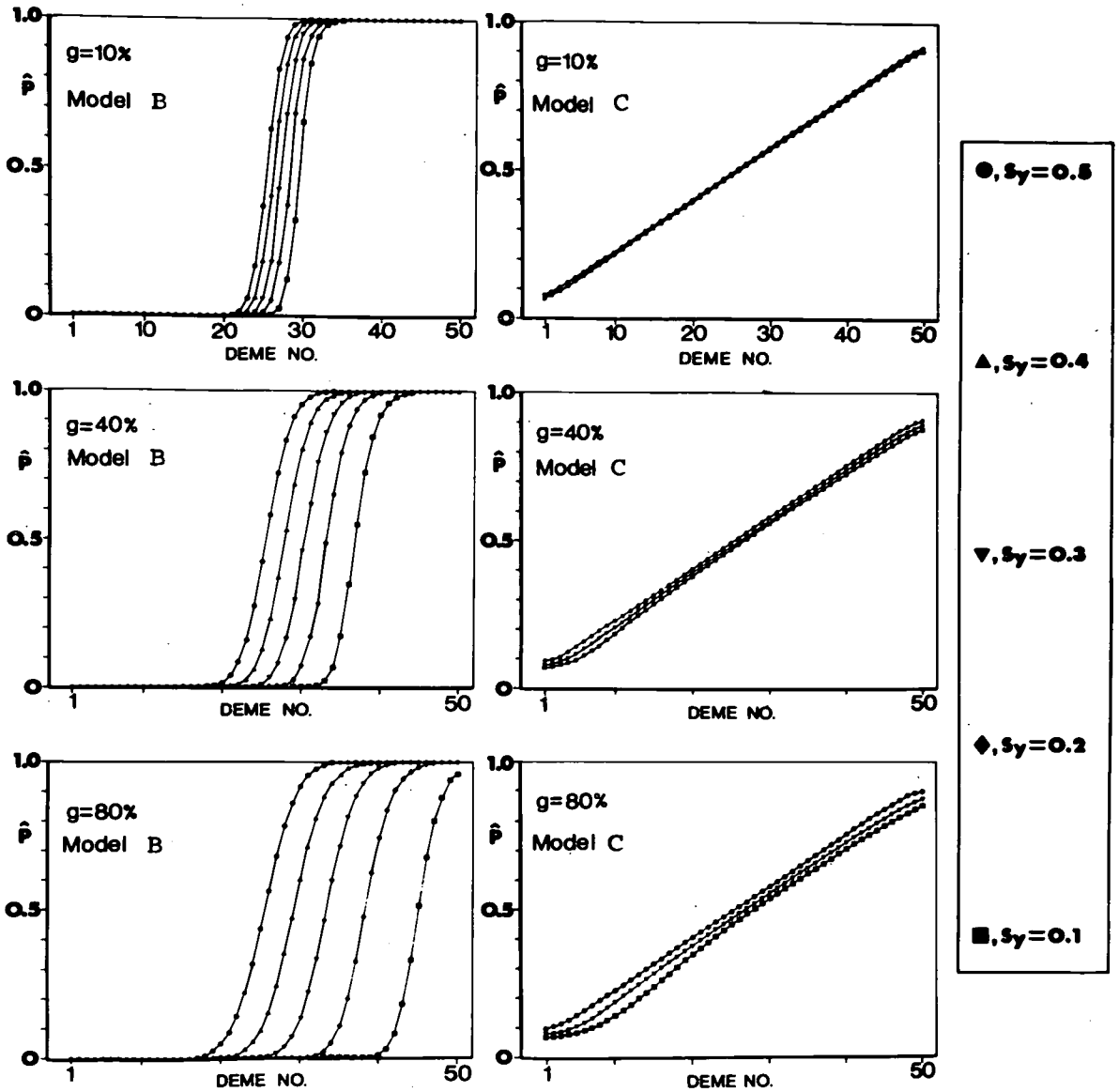
It is therefore necessary to measure all components of selection in order to make progress in explaining a given natural cline.

Even if all components of the probability of survival of each genotype have been mapped, a morphotone may still not correspond to a known null point as a result of asymmetry in gene flow. Dispersal is not always exactly nondirectional (Allee, et al., 1949, Haskins, et al., 1961, Johnson, 1969). For example, Kerster (1964) found a small displacement in the mean position of Sceloporus olivaceus lizards after all phases of dispersal. To explore the effect of a biasing

environmental factor, such as wind or stream flow, may have on a cline, an asymmetry parameter, sy, was introduced into the simulations. For the linear series of demes a fraction sy·g of each genotype move into the deme on the left, and a fraction (1-sy)·g move to the deme on the right of the parental deme. The parameter sy is a coefficient of asymmetry and varies from 0 through 1, and represents perfect symmetry when sy=0.500. In all previous models sy was assumed to be 0.500.

Figure 2-38 illustrates the effect of symmetrical gene flow and several degrees of asymmetry on models B and C (from Endler, 1973). The results for other models are very similar. For a given asymmetry of gene flow (sy) the entire cline is shifted in the direction of the dispersal bias in proportion to the given degree of total gene flow (g). A greater asymmetry (sy more different from 0.500) will result in an increased shifting effect for each dispersal value (g), but has little effect on the slope of the cline. Thus an asymmetry in gene flow may shift the geographic location of a morphotone between differentiated areas without effecting the extent of the differentiation (Endler, 1973). The amount of shifting for various levels of gene flow and asymmetry is summarized in figure 2-39. If the slopes of the fitness gradients are smaller the amount of shifting will be greater than if the slopes are steeper. If the gradients are quite gentle, even a small asymmetry will shift a model B morphotone through many demes. A morphotone may not therefore, correspond even with an overall null point.

Up to now we have been assuming that all demes are of equal population size (N). Any variation in population size affects the amount of gene flow between demes since the number of moving individuals is $N_x g$; a large deme will "export" more individuals than a small deme even if individuals from both have the same probability of



EQUILIBRIUM CLINES ($\hat{p}$ Vs. DEME NUMBER) AND ASYMMETRY (s_y) OF GENE FLOW (g) IN MODEL B AND MODEL C.

FIGURE 2 - 38

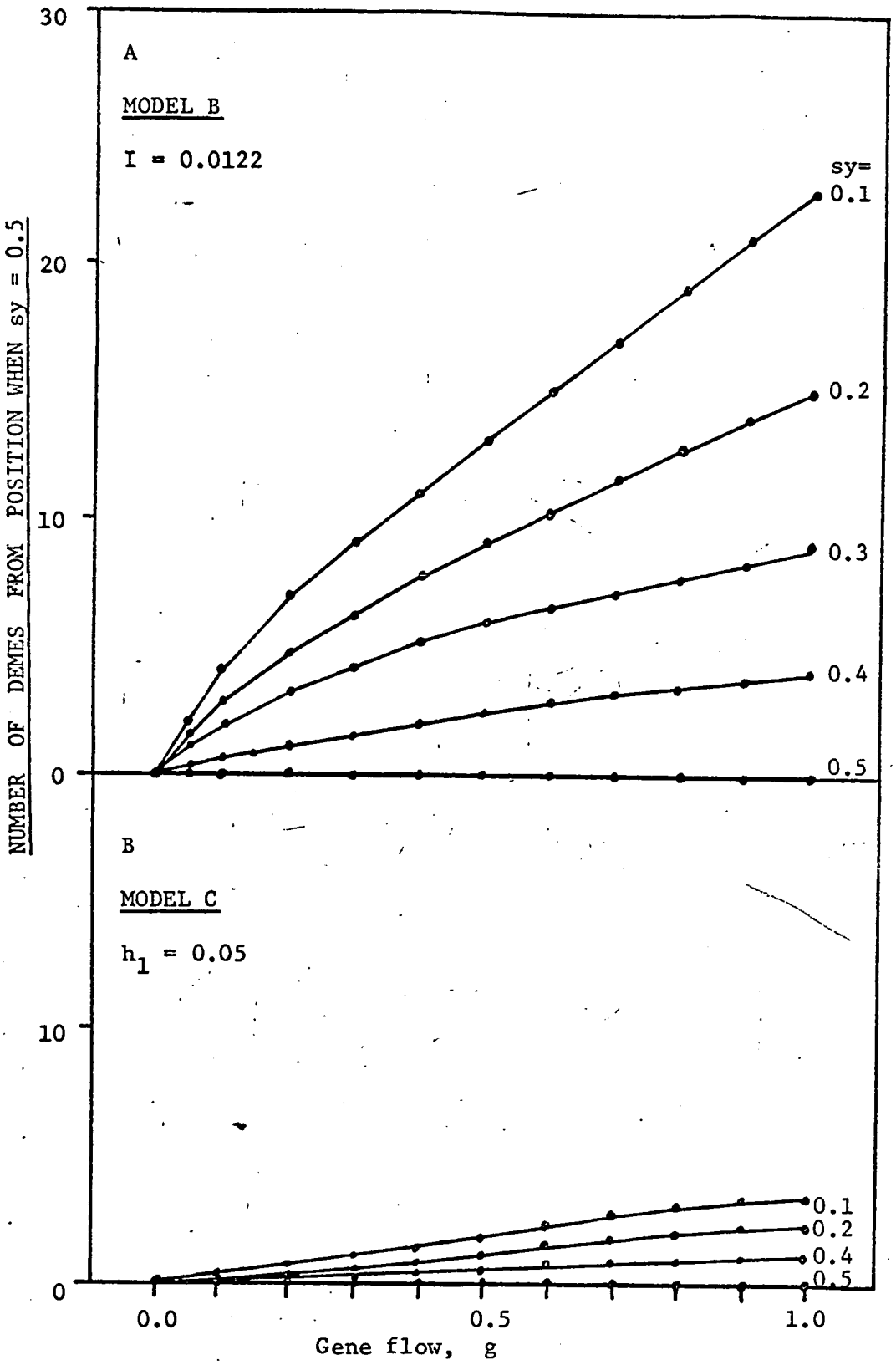


FIGURE 2-39. Shifting of position at which the cline crosses $\hat{p}$=0.50 A, model B; B, model C.

leaving (g). This is aside from possible crowding effects (Section 2.1, Cain and Currey, 1968, Saekai, et al., 1958, Narise, 1968, Christian, 1970). Thus the effect of demes with low population size is equivalent to a partial barrier to gene flow. The effects of actual partial barriers to gene flow and variation in population size are very similar, and only the effects of variable population size will be illustrated. As correctly pointed out by Womble (1951) the effect of a partial barrier is to increase the differentiation across the barrier (figure 2-40, see also section 2.4) in proportion to the "strength" of the barrier. As the zone of small population size is reduced relative to the rest of the deme series the differentiation becomes greater, and in some cases (Models A, B, E, F) a morphotone may be formed (figure 2-40a). Models with heterozygous advantage, however, are remarkably insensitive to barriers to gene flow (figure 2-40b). Perhaps this would explain the numerous cases of absence of morphotones in the vicinity of barriers, as in the House Sparrow (Marshall, 1948a,b, Johnston, 1956a,b, Ferrell, 1966), Amathes glareosa (Kettlewell and Berry, 1961, 1969), Partula taeniata (Clarke, 1968), and Lebistes reticulatus (Haskins, et al., 1961.) Models E and F are intermediate between C and the others in sensitivity to barriers.

Partial barriers have an interesting property; if a morphotone is likely to form within a certain critical range of a partial barrier, then it will be "attracted" to the barrier, otherwise it will be unchanged and a second, smaller morphotone will form at the barrier (figure 2-40). If several loci are responding to one or more environmental gradients, and all gradients are going in roughly the same direction (say $\pm$ 20°) but positions of optima and null points are different, then clines will not be concordant. However, given one partial barrier across the average axis, (and no heterozygous advan-

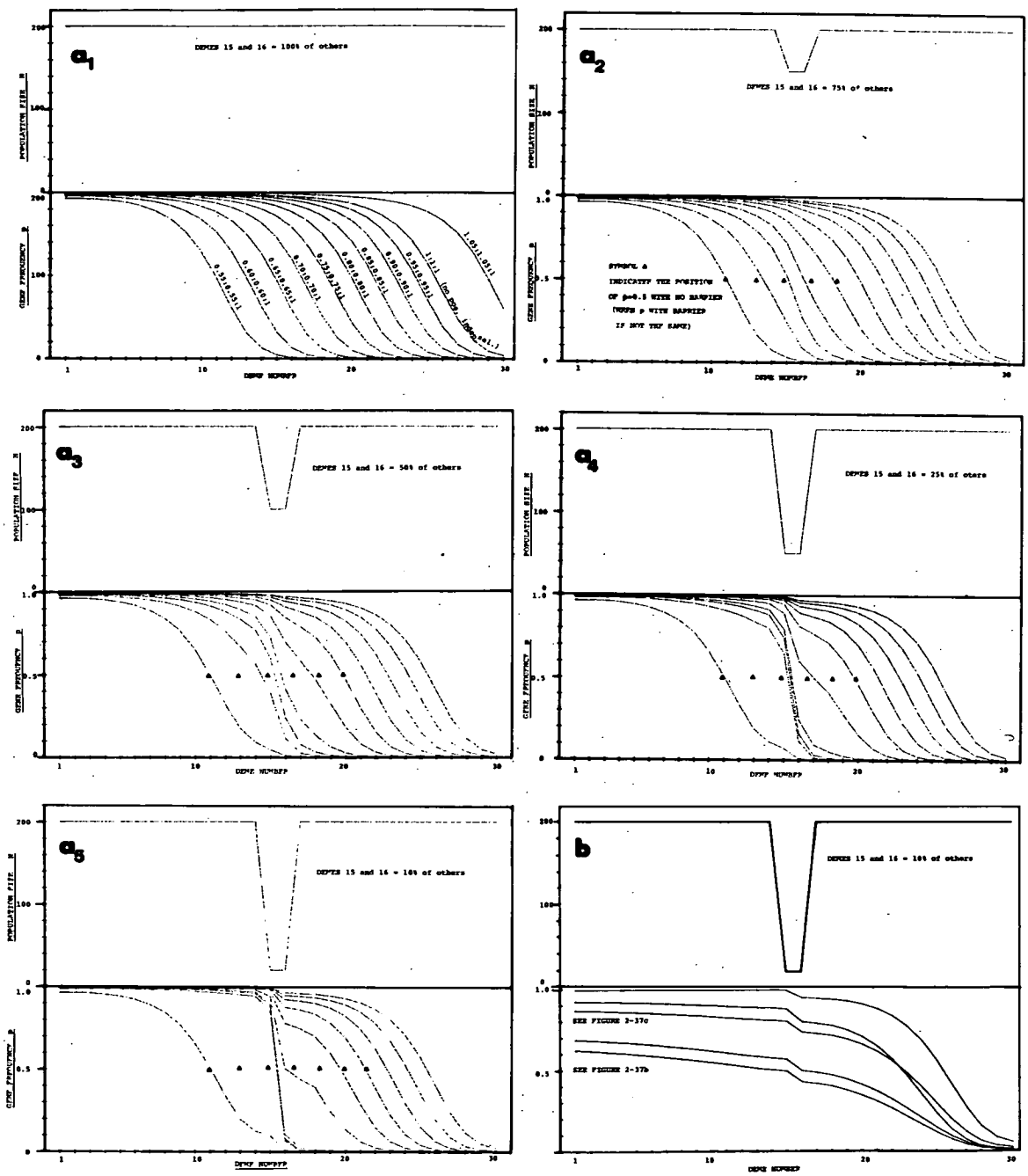


FIGURE 2 - 40. As in figure 2-37, showing the effect of a barrier.

tage) all clines with null points sufficiently near the barrier will form morphotones at the barrier, and so will be concordant. Only the clines with null points too far away will not be concordant. This could easily be happening in the clines for five separate polymorphic characters in the butterfly Tisiphone abeona described by Lucas (1969). All five clines are sigmoid and all but "dull red ocellar surround" show morphotones in roughly the same position. Lucas mentions in passing that the breadth of the species range contracts in that region.

Although partial or complete barriers need not be the cause of morphotones, there are many examples in which morphotones do correspond to barriers. Among them are (Cain and Currey, 1963a, Kettwell and Berry, 1961, 1969, Lucas, 1969, Wolda, 1969b). Even the area effect for yellow shell in the Marlborough Downs falls in this category as the sharpest part of the cline in morph frequency corresponds to the limit of a major area of land available to Cepaea (Cain and Currey, 1963a).

Terborgh (1971) has pointed out that distribution of species on environmental gradients will take on a special truncated form if the species exhibit competitive exclusion. Similarly, if genotypes exclude one another, or reduce one anothers' probability of survival in the same deme, their abundance surves will be truncated relative to their fitness curves (figure 2-41). This will either steepen existing morphotones, or form them where they are absent (figure 2-42a). If A is dominant to gene a, then only one morphotone will form, but if heterozygotes are intermediate in fitness or in optimum position, then two morphotones will form (figure 2-42b). Morphotones resulting from competitive exclusion will form at one or more null points between pairs of genotypes rather than at the null point for net genic selection.

Lastly, morphotones may form as the result of interaction among loci (Clarke, 1966); this will be discussed in chapter 3.

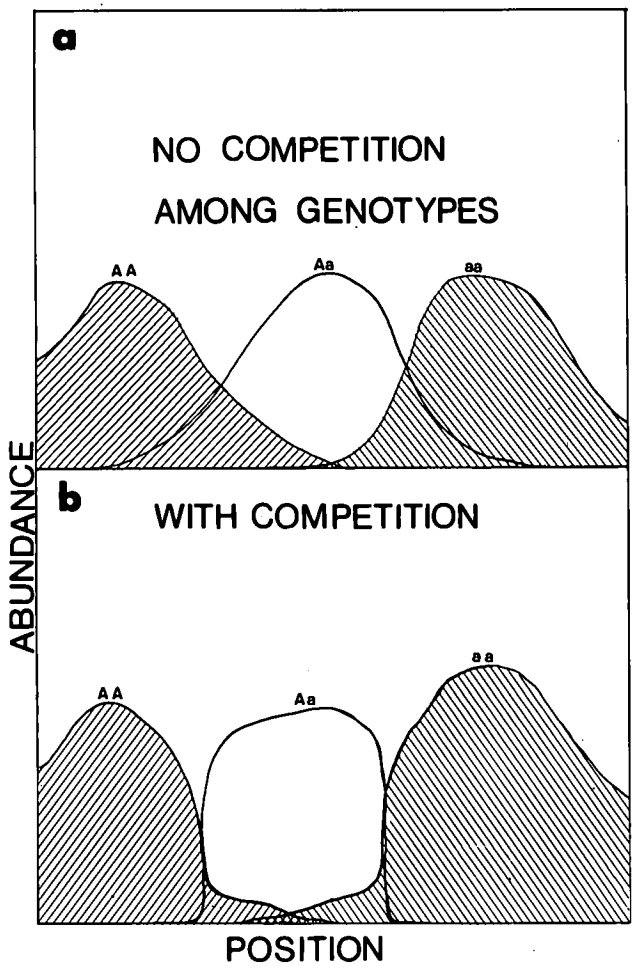


FIGURE 2-41

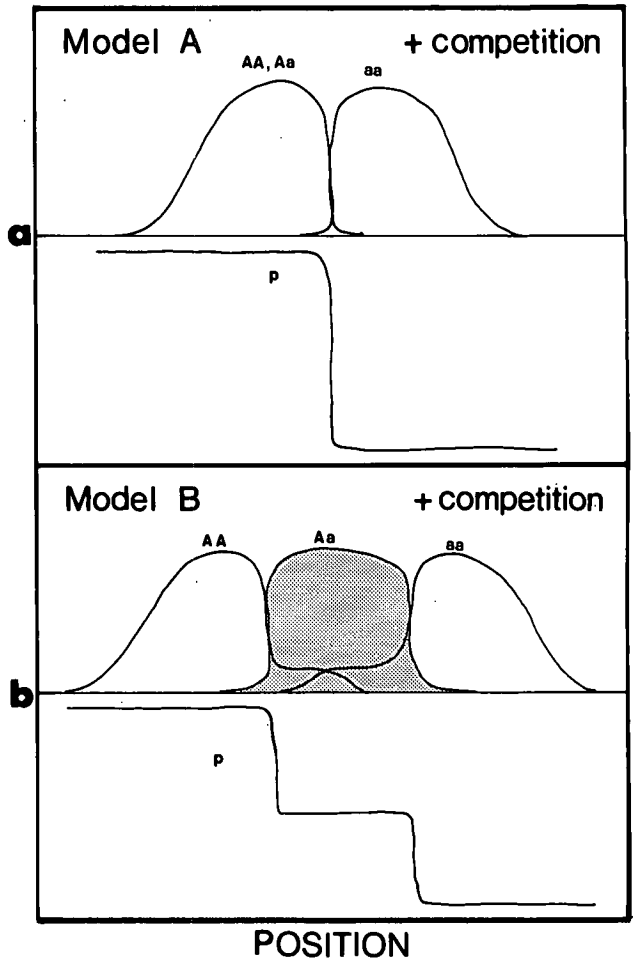


FIGURE 2-42

The relationship between an environmental factor and the probability of a zygote's survival to successful reproduction is not necessarily a linear one. For example, the relationship between temperature and death rates of several insects and some vertebrates may be exponential or sigmoid (Andrewartha and Birch, 1953, Uvarov, 1931). For factors such as atmospheric moisture, the situation may even be more complex. If the critical factor of an animal is evaporation rate per unit area, the rate itself depends upon temperature, barometric pressure, and the constellation of water vapour pressures at varying distances from the animal at a given moment. If the relationship between survival and the evaporation rate is also nonlinear, animals may find their survival probabilities very different over short distances, even though the temperature and the measureable relative humidity change only slightly. In addition, the nonlinear relationship itself may change with variation in a third or fourth factor. The relationship between death rate of Calandra oryzae and saturation deficit x time is exponential at low temperatures and sigmoid at high temperatures (Birch, 1945). Microclimatology is discussed by Geiger (1966), and an excellent review of survival rates under differing environmental conditions may be found in Uvarov (1931), and Andrewartha and Birch (1953). Differences between species described by Birch (1945) and Uvarov (1931) suggest ways in which polymorphs may differ on a smaller scale. It is quite possible that many of the cases of morphotones in the absence of environmental changes may simply reflect our ignorance of the relationship between measureable environmental factors, actual environmental factors, and rates of survival and reproduction collectively known as fitness.

There are many ways in which morphotones may form in the absence

of sharp environmental changes, but a smooth environmental gradient does not necessarily mean the absence of an abrupt change in relationship.

Section 2.6. Spatially abrupt environmental changes.

When one finds a morphotone in a continuous natural population, the most obvious causal factor to look for is a spatially sharp change in the environment; an ecotone near the morphotone. Indeed, even if ecotones are not found in the vicinity, it is often assumed that some "cryptic" environmental factor changes abruptly near the morphotone. As has been demonstrated by Haldane (1948), Jain and Bradshaw (1966), Hanson (1966), Livingstone (1969), Cook (1971), and Slatkin (1971), abrupt environmental changes do not have to be large to produce steep clines, although the relative amount of gene flow is critical. Clines resulting from abrupt environmental changes have been analysed most thoroughly and clearly by Jain and Bradshaw (1966), and the conclusions of other authors are not noticeably different. All published models incorporating ecotones (op.cit.) may be regarded as modifications of Models A, B, C, etc, with added ecotones; these will be named A', B', etc.

The equilibrium clines for models A', B', and D' through H' are very similar to models A, B, and D through H (figure 2-43, 2-44); all form morphotones at the ecotone provided that gene flow is not great and selection is moderate to strong (op. cit.). If selection and gene flow are both strong, then the clines resulting from the modified models are not distinguishable from the environmental gradient models (section 2.5) with moderate selection and most levels of gene flow (compare figure 2-44 with figures 2-25 and 2-26). When selection is weak, however, only a weak cline may be formed (figure 2-45, see also Jain and Bradshaw, 1966, Hanson, 1966, Livingstone, 1969, and Slatkin, 1971), even for low levels of gene flow.

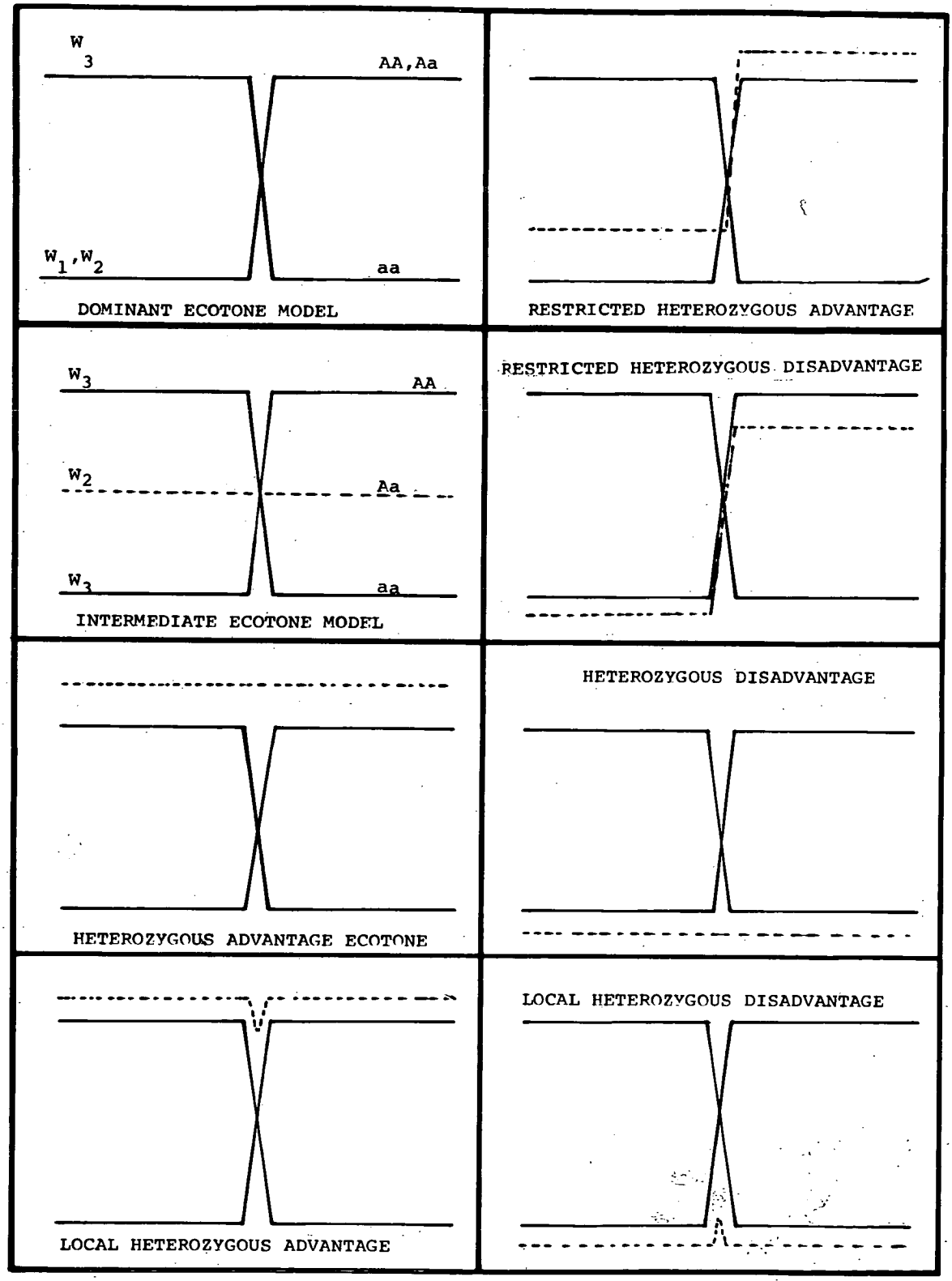


FIGURE 2 - 43. Ecotone models A' through H'.

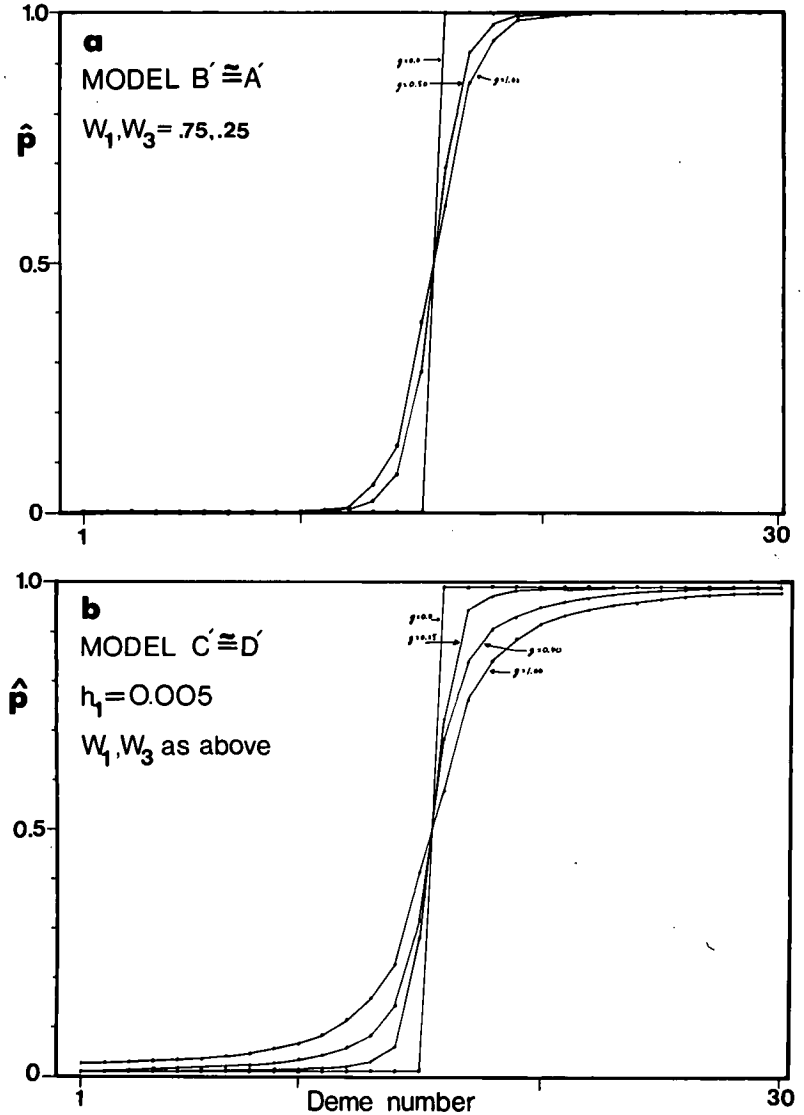


FIGURE 2–44

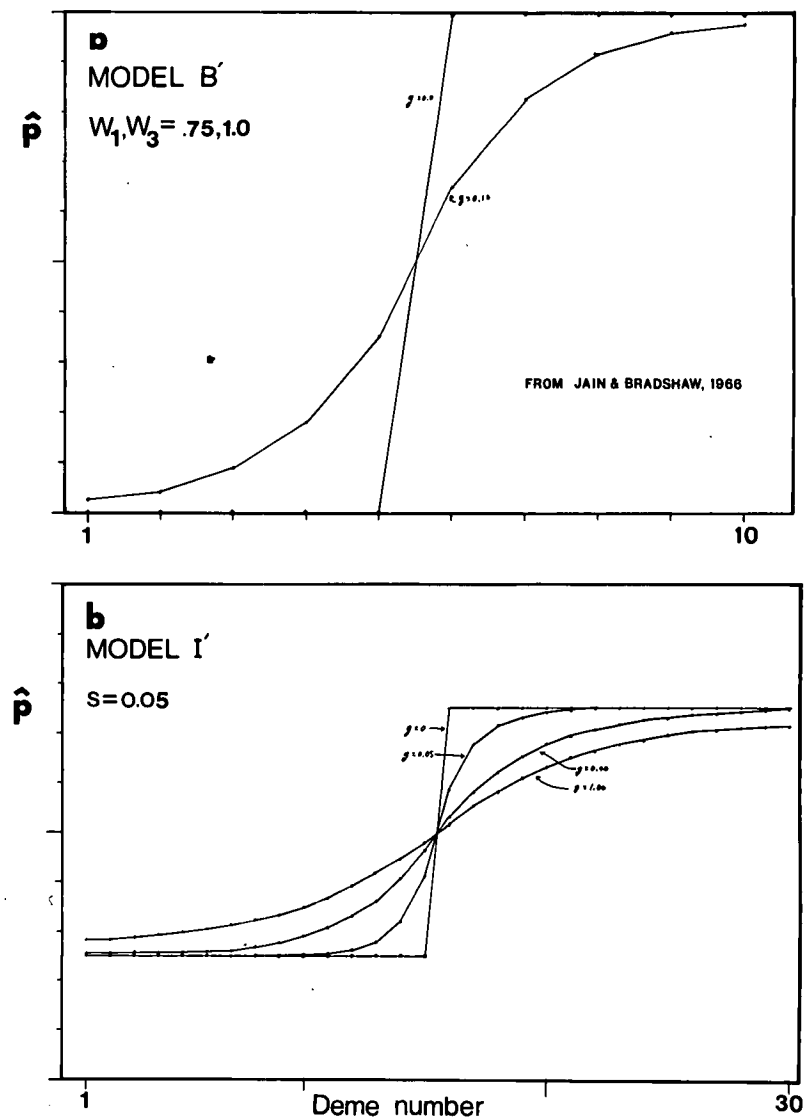


FIGURE 2–45

Models C' and I' produce different clines from models C and I. A model C' cline has a morphotone, especially if the amount of heterozygote advantage (h_1) is small (figure 2-44b). However, if h_1 is very large, the morphotone will be small enough to be buried in the sampling error of a natural cline following this model. Model I' with focal frequencies which change suddenly at a certain position in the transect may only result in a morphotone cline if the strength of the frequency-dependence (s) is high and if gene flow (g) is small. If this is not the case, then morphotones will only form for extremely low levels of gene flow (figure 2-45b). Generally, model I' morphotones are less sensitive to the dedifferentiating effects of gene flow if the focal frequencies change through a large number of values,suddenly over a short distance.

Clines resulting from sharp environmental changes differ from environmental gradient clines cheifly in their comparative sensitivity to the leveling effect of gene flow. For low levels of gene flow, morphotones associated with ecotones are more distinct than those associated with environmental gradients (compare figure 2-44 with 2-25). However, differentiation accross an ecotone rapidly becomes "swamped" at higher levels of gene flow, while the same gene flow would have little effect on a cline resulting from an environmental gradient. This is particularly true for weak selection accross an ecotone, where the cumulative effects of selection do not build up slowly with distance. Clines resulting from environmental gradients are less sensitive to the effects of gene flow than ecotone clines because aliens are less likely to come from demes with drastically different selective regimes. Furthermore, in gradient clines, a given deme will be subject to gene flow from demes

with, say, reduced selection against one genotype, and gene flow from demes on the other side with increased selection against the same genotype. If the gradient is reasonably smooth gene flow from up and down the gradient will be self-cancelling; the mean gene frequency of aliens will not be appreciably different from the residents (Endler, 1973). This is not the case in the vicinity of ecotones.

If two sets of demes occupy different spatially constant environments, separated by an ecotone, then the operation of an additional selective factor will not shift the cline as it does if the fitness curves have nonzero slopes (see page 2-51 and figure 2-37). It will simply shift the mean gene frequency of the cline up or down, depending upon the action of the position-independent selection. This can be seen in Jain and Bradshaw's (1966) simulations with "asymmetrical selection". If, however, there is a slight gradient in one or more of the fitness curves in addition to the ecotone (figure 2-46a), then, in some cases, the morphotone may be shifted (figure 2-46b). As in models A-I (section 2.5), if the dominance patterns of the distance-dependent and distance-dependent selection components are different, then the morphotone may be destroyed or greatly reduced.

An asymmetry in gene flow will shift an ecotone-caused morphotone, but has little effect on the amount of differentiation. A large asymmetry may greatly reduce or destroy a morphotone, and the reduction in differentiation is directly related to the amount of asymmetry (Jain and Bradshaw, 1966 and the author's simulations). The amount of reduction in differentiation found by Jain and Bradshaw (1966) is, however, unexpectedly large. In their simulations only

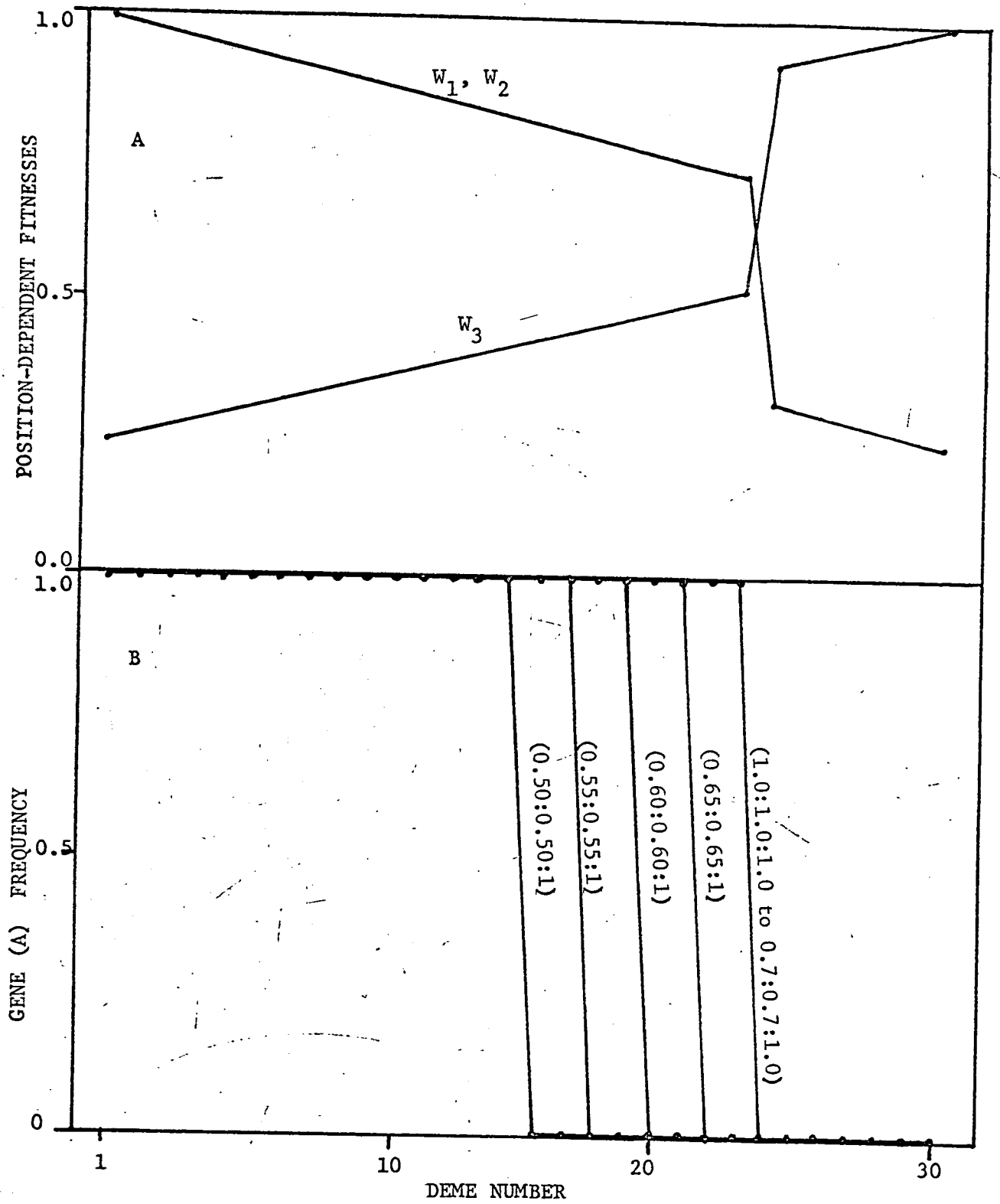


FIGURE 2-46. The effect of position-independent selection on Model A with an added sharp change in fitness. A, fitnesses before the position-indpendent mortality; B, the clines (g=0.0) for several different levels of position-independent fitness. (position-independent fitnesses are W_1:W_2:W_3).

10 demes were used, and the ecotone was placed between 5 and 6. As soon as the asymmetry reached a certain level, the morphotone was shifted off the edge of their deme series; if they had, say, 20 or 30 demes, the entire cline would be visible, and the apparant reduction in differentiation not so great. (See figure 2-38 for a comparable situation in model B, sy=0.1).

Jain and Bradshaw (1966) also discuss the effect of a difference in generation time on each side of the ecotone. The effect is equivalent to an asymmetry in gene flow in which the dispersal bias is in the direction of the faster population replacement rates. The effect of unequal generation time is marked if one deme group has a generation length 5 times longer than the other, and can accentuate, or completely counteract other factors. Unfortunately results are not given for more realistic differences in generation time.

On a gentle environmental gradient, however, it is possible for a morphotone to be shifted away from the null point by a small difference in generation time, just as a small asymmetry (sy) on a gentle gradient can cause shifting (section 2.5) This might be an important factor in certain poikilotherms (such as insects) distributed on temperature gradients; temperature effects development rate, which in turn affects generation time (in bivoltine and polyvoltine species), yielding a cline in generation time along the gradient. If the associated fitness gradients are not too steep, any morphotone will be shifted towards the warmer temperature. If the selection is in the form of an ecotone, then such a difference in generation time may destroy or reduce the morphotone.

Ecotones will often result in morphotones, but there are many conditions in which expected morphotones do not appear.

Section 2.7 Secondary Contact

One of the most common explanations of morphotones, especially in the earlier literature, is that of secondary contact (about 30 references). The basic idea is that two formerly isolated populations coming together will somehow maintain a very steep cline. This is supposed to be aided, in many theories, by hybrid inviability and infertility consequent upon genetic incompatability between the two groups. The presence of increased variation in the zone of contact is supposed to be "proof" of secondary contact. But such increased variation is expected in all areas of intermediate gene frequencies, simply because the expected sampling variance of gene frequency p is $p(1-p)/2N$.

Usually it is assumed that there is no difference between the two populations' selective environments, at least in the area of contact, but often it is assumed that this is true throughout the species range. Apart from this being an unwarranted assumption, it will simply not work. If two groups meet each other, and there is no selective restraint on invading the other's territory, each will spread smoothly into the other's range and the morphotone produced at the moment of contact will dissolve, leaving only a smooth, gradually falling cline. Figure 2-47 is an example of a simulation of secondary contact between a set of demes with a gene frequency of 100% and another with a frequency of 0%. The morphotone is almost gone by generation 50 with only 20% gene flow. As in section 2.4, the neutral cline takes a longer time to decay to zero slope. This is because a given deme receives gene flow from demes with smaller gene frequencies, others with the same gene frequency, and still

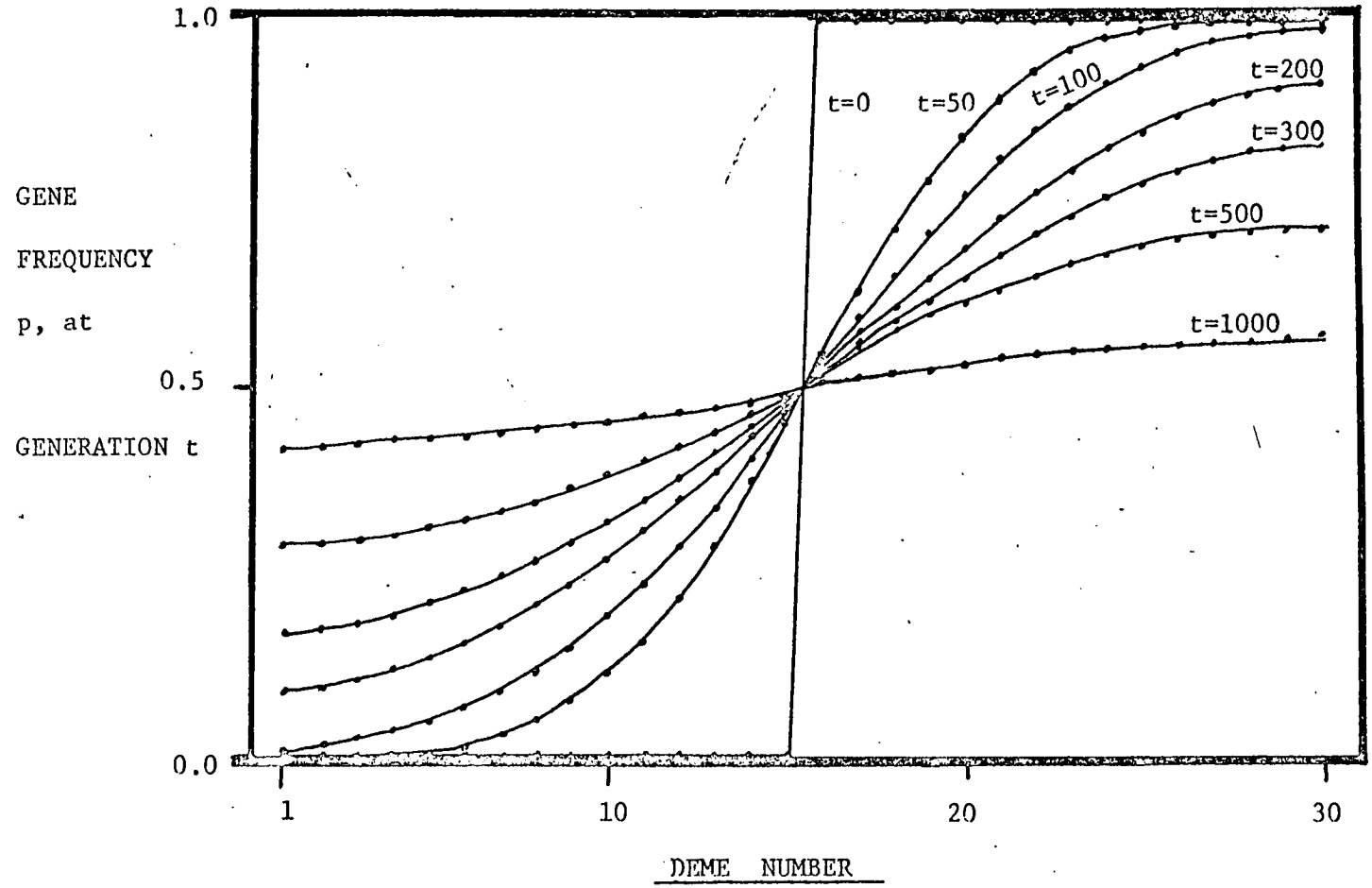


FIGURE 2-47. The effect of secondary contact with no selective difference among demes. Demes 1 through 15 were started with p=0.0 and demes 16 through 30 were started with p=1.0. Gene flow, g=0.20. Gene frequency clines shown for generations 0, 50, 100, 200, 300, 500, and 1000. The results are identical for 100% assortative mating among the genotypes, although the genotype frequency clines remain steeper for a longer period of time.

others with larger gene frequencies, keeping the difference in gene frequency between residents and all aliens small. Secondary contact of neutral characters will result only in a long lasting cline, not in a stable morphotone.

Another possibility is that the populations coming into secondary contact compete for a common limiting resource. In this case one phenotype will always exclude the other, creating a morphotone at the contact zone. However, the resulting morphotone is not stable in position, and will eventually travel to one edge of the species range and disappear. Exactly the same thing happens if heterozygotes are at a disadvantage compared to the homozygotes; an equilibrium which is unstable in time will not be stable in space.

Another possibility, though still unrealistic, is that the characters are selectively neutral in the contact region, but there are differences in more central portions of each group's ranges. Nagylaki (1966) simulated equilibria under these conditions and found that morphotones are only formed if the neutral zone is narrow compared with the panmictic diameter of the species.

Stable morphotones will only form if there is some spatial difference in probabilities of survival among genotypes; secondary contact by itself is not sufficient to maintain a cline with a sharp change in morph, genotype, or gene frequency. The conditions favouring and determining the positions of morphotones are the same regardless of whether polymorphism arises from secondary contact, or is already present throughout the species range. Primary and secondary intergradation cannot be distinguished by observing the properties of a cline.

Section 2.8 Summary of Chapter 2

1. Gene flow is restricted compared to migration and dispersal; the gross movements of animals gives an exaggerated picture of the movement of genes. The great localization of movement provides opportunity for isolation by distance, and the restriction of available habitat and breeding sites suggests that the deme model is a better approximation to population structure than the continuous model.

2. The morph-ratio cline, or gradient in morph, genotype, or gene frequency is utilized as a model in the study of clines. The model is examined by means of computer simulation and simple graphical methods. The term morphotone is used to denote a marked spatial change in morph, genotype, or gene frequency.

3. Stochastic influences on gene frequency, sampling error in mating, and in gene flow, may produce long lasting clines, but are not capable of producing stable morphotones. "Area effects" may apparently result from genetic drift if sampling methods do not give a complete and precise mapping of the microgeographical variation.

4. Nine models of the effects of environmental gradients on clines are presented. Seven of them (A,B,D,E,F,G,H,) and combinations of them produce well marked morphotones, even if selection is below the practical limits of field measurement and gene flow is high. There are a great variety of conditions which can yield the same kind of cline; there is no single simple explanation of geographic variation. The operation of position-independent factors, asymmetry in gene flow, partial barriers to gene flow, and differences in generation time may shift any morphotones produced by position dependent factors away from the position expected, or may destroy the morphotones.

5. The nine models are modified by the addition of sharp changes in fitness, or ecotones. All modified models (A'B'C'D'E'F'G'H'I') produce morphotones, but they are much more sensitive to the attenuating effects of gene flow than the environmental gradient models (A-I). Ecotone models are not subject to shifting of their morphotones, shifting factors simply reduce the sharpness of the morphotone, or destroy it.

6. Secondary contact is not sufficient by itself to maintain a stable morphotone although it may result in a moderately long lasting cline. The distinction between primary and secondary intergradation cannot be made by observing the properties of a cline.

7. It is impossible to interpret a natural cline without knowing the geography of absolute survival values (the shape of the fitness curves), and the extent of gene flow. It is exceedingly important to sample properly. There is no single simple explanation of geographic variation.

Chapter 3

EXPERIMENTAL CLINES

> "There is no way toward understanding of the mechanisms of macroevolutionary changes, which require time on geological scales, other than through understanding of microevolutionary processes observable within the span of a human lifetime, often controlled by man's will, and sometimes reproducible in laboratory experiments." Th. Dobzhansky (1951, p. 16)

Section 3.1. Introduction

In order to study the effects of known selection and gene flow, a series of clines were set up in experimental polymorphic populations of *Drosophila melanogaster*. Fruit flies are very convenient because they are easy to handle and raise in large numbers, and have a very short generation time (9 days minimum). *Drosophila* have the added distinction of well-known genetics.

Bar, a duplication of the first chromosome, was chosen as the polymorphic character because all genotypes, including heterozygotes, are distinguishable. In *D. melanogaster* the normal eye is round or ovoid; males have about 740, and females have about 780 ommatidia. Hemizygous *Bar* males have only 90 and homozyous *Bar* females only 70. In both, the eyes are much reduced to a narrow vertical bar-shaped cluster of ommatidia. Heterozygotes (females only as *Bar* are sex linked) have about 360 ommatidia, and their eyes are reduced to a maize kernel or sometimes a kidney-shaped cluster with a flattened outer surface (Lindsley and Grell, 1968, Bridges and Bream, 1944). It is therefore possible to calculate exactly the gene frequency of

Bar in the population.

Section 3.2. The Base Population

Bar (with a small adjacent segment of the first chromosome) was introduced into a large outbred normal ("wild type") population in order to create a polymorphic population that was not a heterogeneous mixture of two different gene complexes.

The outbred normal population was derived from Prof. A. Robertson's "Standard-Kaduna" population at the Institute of Animal Genetics, Edinburgh. Robertson's Kaduna cage has contained several thousand flies for many years and is in genetic equilibrium with respect to many loci (Robertson, personal communication, 1969). Two large egg samples were obtained from Kaduna by leaving 500 ml. beakers, filled with sterile Drosophila medium, in Robertson's population cage for 24 hours. The beakers were transferred to a new cage in the Department of Zoology, University of Edinburgh, and allowed to produce a replicate population. All my flies, in Kaduna, and subsequently in the experiments, were kept in the same "warm room" at 25°±1°C. Kaduna's food was provided in uncovered milk bottles (65mm. diameter by 160mm. high) filled with about 250 ml. of Edinburgh cornmeal-molasses-agar Drosophila medium. Ten bottles were always present in the cage. One new bottle of food was added, and one old bottle removed, every 4-5 days. The new Kaduna population was allowed to breed for about 10 generations before the introduction of Bar.

Bar and a small segment of the adjacent X-chromosome was introduced into Kaduna by recombination and backcrossing, according to the scheme in figure 3-1. The first part is based upon a method

FIGURE 3 - 1

INTRODUCTION OF BAR INTO KADUNA

Mutants used:

Chromosome I (X): w = white; sn = singed; B = Bar

II,III: Xa = Xasta, a marked (notched wing) translocation and two inversions. Homozygotes are lethal, and there is almost no (ch. II,III) recombination in heterozygotes (Robertson, pers. comm., 1969).

IV: Ci^W = Cubitis interruptis of Wallace

K will indicate Kaduna chromosomes. + will indicate "wild" chromosomes from mutant stocks. Y indicates the Y chromosome.

Cross I: $\frac{wsnB}{Y}\ \frac{+}{+}\ \frac{+}{+}\ \frac{+}{+}$ by $\frac{+}{+}\ \frac{Xa}{+}\ \frac{+}{+}\ \frac{Ci^W}{Ci^W}$

Cross II: $\frac{K}{Y}\ \frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}$ by $\frac{wsnB}{+}\ \frac{Xa}{+}\ \frac{+}{+}\ \frac{Ci^W}{+}$

Females collected every 4 hours. (all crosses)

Cross III: $\frac{wsnB}{Y}\ \frac{Xa}{K}\ \frac{+}{K}\ \frac{Ci^W}{K}$ by $\frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}$

B Ci^W female offspring only.

Cross IV: $\frac{K}{Y}\ \frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}$ by $\frac{wsnB}{K}\ \frac{K}{K}\ \frac{K}{K}\ \frac{Ci^W}{K}$

Cross V: $\frac{B}{Y}\ \frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}$ by $\frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}\ \frac{K}{K}$

B males only
149 of these recombinants found.

Cross VI: Kaduna males by Bb females

4 bottles, each with 35 BY and 200 Kaduna females.

4 bottles.

Cross VII: B males by Bb,bb, K females 8 bottles.

Cross VIII: B, K, males by BB,Bb, K females 10 bottles.

To new population cage, Bar-Kaduna

suggested by Prof. A. Robertson (Personal communication, 1969). The technique ensured that Kaduna and Bar-Kaduna never fell below about 5000 adults. The average size of Bar-Kaduna remained about 8000.

Section 3.3. The Cline Method

Five sets (A through E) of 15 demes (vials) each were made up from the Bar-Kaduna population two generations after the last backcross. Initially each deme in each set contained 50 pairs of flies with a Bar gene frequency of 50%. These were allowed to mate and produce offspring for one generation (generation 0) before selection and gene flow were started. This and all subsequent generations were raised in 30mm. by 100mm. glass vials, stoppered with cotton-wool, and containing roughly 17ml. of Edinburgh Drosophila medium. Generations were discrete.

Each deme was subject each generation to the following sequence (figure 3-2):

a. Collection and scoring of emerging adults for each of six days, from 7:00 a.m. to 11:30 a.m.; males and females into separate fresh vials.

b. Artificial selection (if any) on each genotype.

c. Populations after selection reduced to N=50 pairs per deme, holding genotype frequencies constant.

d. Gene flow (if any) for each genotype separately.

e. Mating for 24 hours in fresh vials.

f. Egg laying for 4 days in fresh vials.

g. Removal of parents from egg vials.

h. Development into the adults of the next generation.

EXPERIMENTAL TECHNIQUE FOR THE GENERATION OF CLINES

(Only the first three demes are shown)

a. Parents in fresh vials, (Natural selection on offspring larvae).

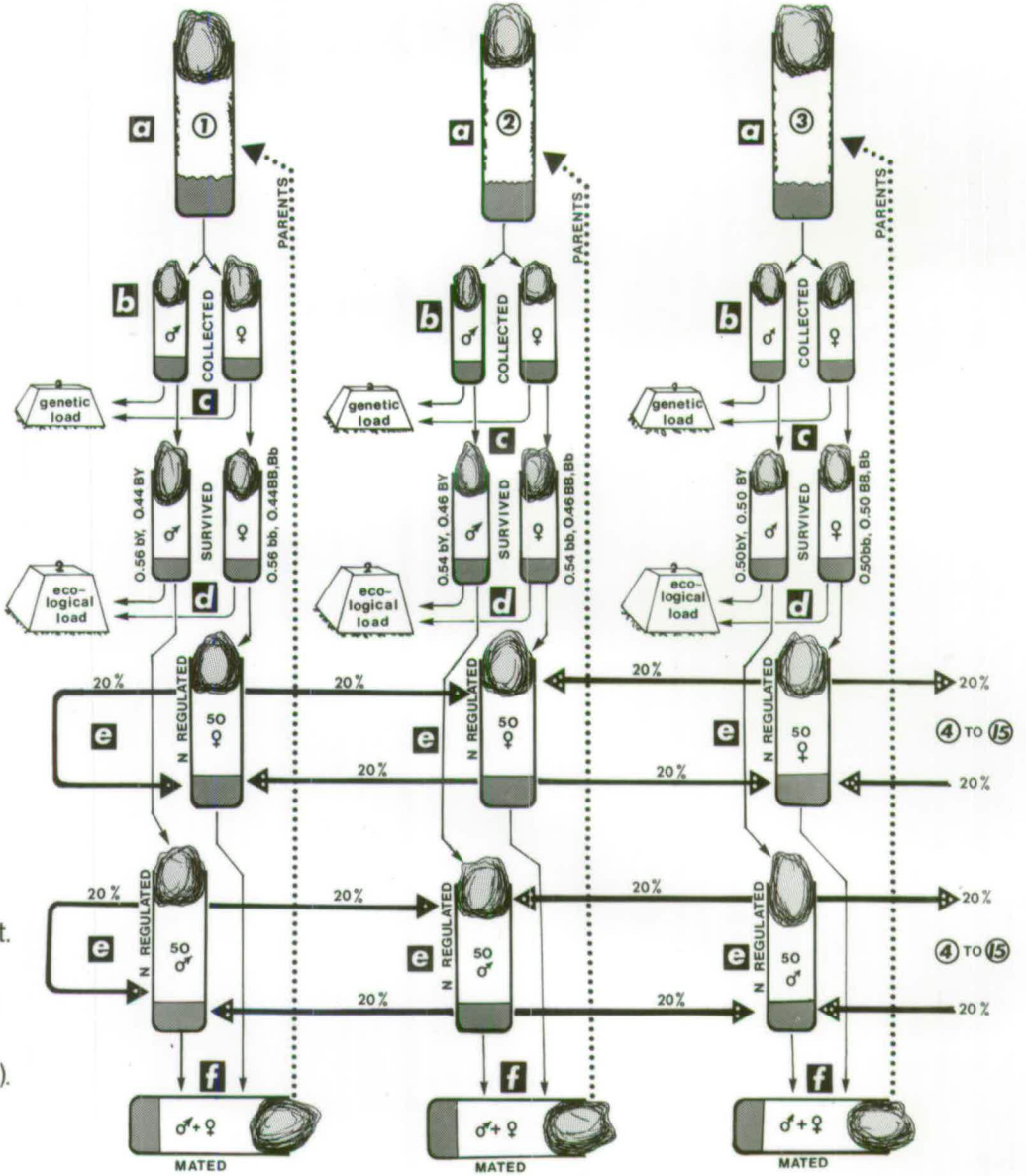
b. Adults collected and separated by sex.

c Artificial selection.

d Population regulation; Numbers reduced to 50 in each sex, holding genotype frequencies constant.

e 40% Gene flow; 20% of each genotype to both adjacent demes (end emigrants return).

f Mass mating within demes.



Differences in fitness among the genotypes resulted in a second period of selection, henceforth called natural selection, against Bar during period h.

The arrangement of artificial selection and gene flow in the five sets was as follows:

Set A, artificial selection, gene flow.

Set B, artificial selection only (control for gene flow).

Set C, artificial selection, gene flow.

Set D, artificial selection only (control for gene flow).

Set E, gene flow only (control for artificial selection).

In order to produce clines, the artificial selection was imposed in the form of a gradient of fitness along each deme series (except E), with an increment in selection between adjacent demes of I = 0.04. The demes in sets A, B, C, and D, were subject to the following survival values:

Deme x	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
$W_1(x)$, $W_2(x)$	.42	.46	.50	.54	.58	.62	.66	.70	.74	.78	.82	.86	.90	.94	.98
$W_3(x)$	.58	.54	.50	.46	.42	.38	.34	.30	.26	.22	.18	.14	.10	.06	.02

Thus in each deme (x), a fraction $W_1(x)$ of Bar genotypes (males and females), a fraction of $W_2(x)$ of heterozygotes (females), and a fraction $W_3(x)$ of normal genotypes (males and females) were selected to be the parents of the next generation. Bar is treated as a dominant for the purposes of artificial selection. The null point in artificial selection was located at deme 3 because a preliminary estimate suggested that this selection pattern would uniformly counteract the natural selection against Bar, centering the resulting clines near deme 8. Artificial selection was continued

throughout generations 1-35, except for a suspension in generation 10, and selection on females only in generation 18.

Gene flow was accomplished in each deme by removing g=0.40 of each genotype from a given deme and placing one-half of these emigrants (i.e. 20% of N) into the deme on the left and placing the remaining half of the emigrants into the deme on the right (figure 3-2). Thus adjacent demes exchanged 20% of their members and a given deme contained 40% immigrants after gene flow each generation. The would-be emigrants from the end demes, 1 and 15, were returned to the demes from which they came. The manipulations thus correspond to model A, Chapter 2.

Section 3.4. The Drosophila Clines

The gene frequency clines for the last 10 generations are shown in figure 3-3, and the slopes of the clines for all generations are found in figure 3-4a. Table 3-0 and figure 3-6 give the gene frequencies in each deme in each generation. The total numbers emerging in each generation are found in appendix 4, along with the numbers of parents from the deme, and the slope of a cline was estimated by the regression of gene frequency on deme number for the set concerned (See also, Endler, 1973).

The response to selection (sets A through D) was quite marked for the first five or six generations; thereafter there was little change in the configurations of the clines. All slopes became significantly different from zero at generation 1, with the exception of set E (no selection), which reached significance briefly during generation 4. Thereafter the slopes of sets A through D remained significantly different from zero and E insignificant. There is no consistent or significant difference between the selective clines

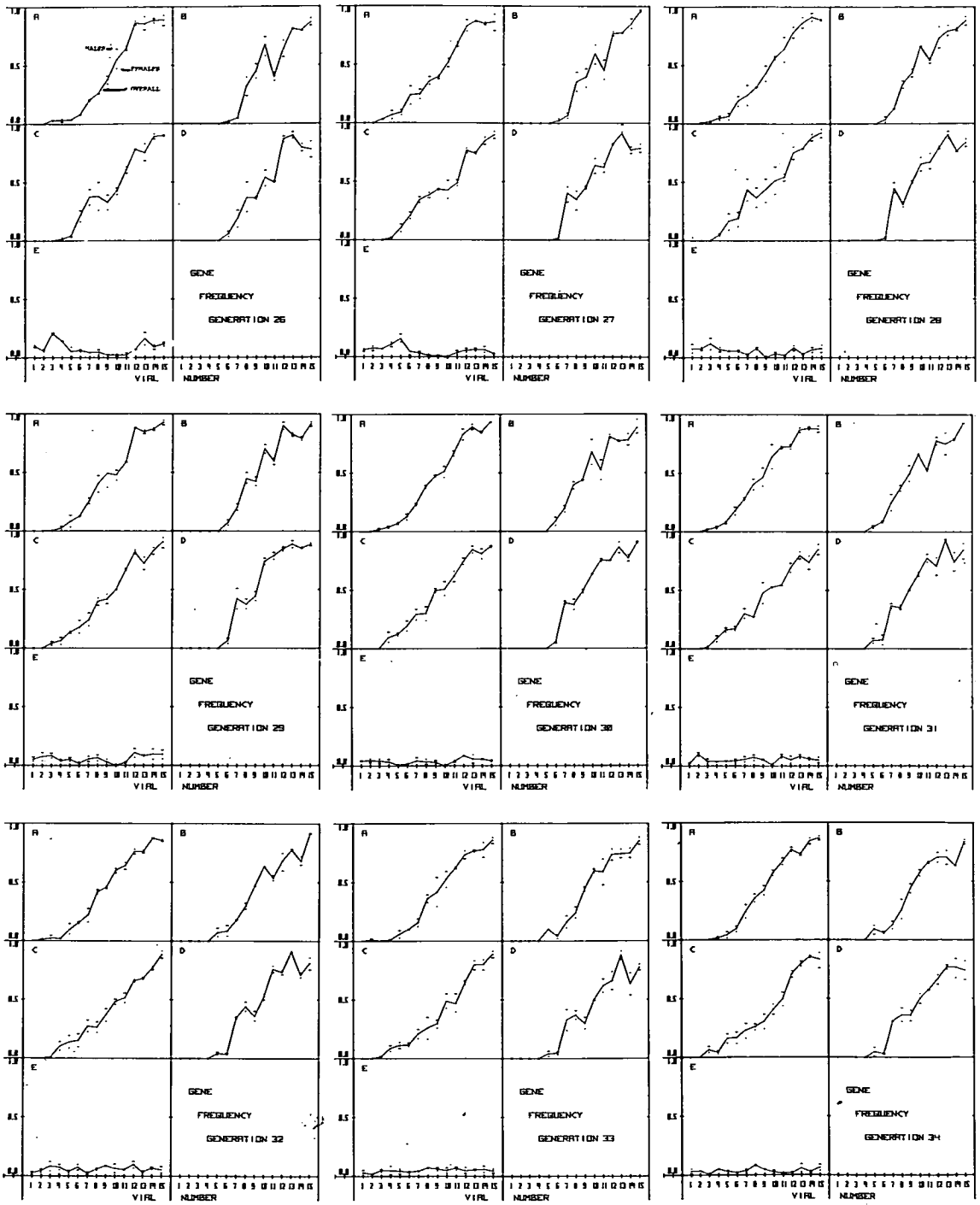
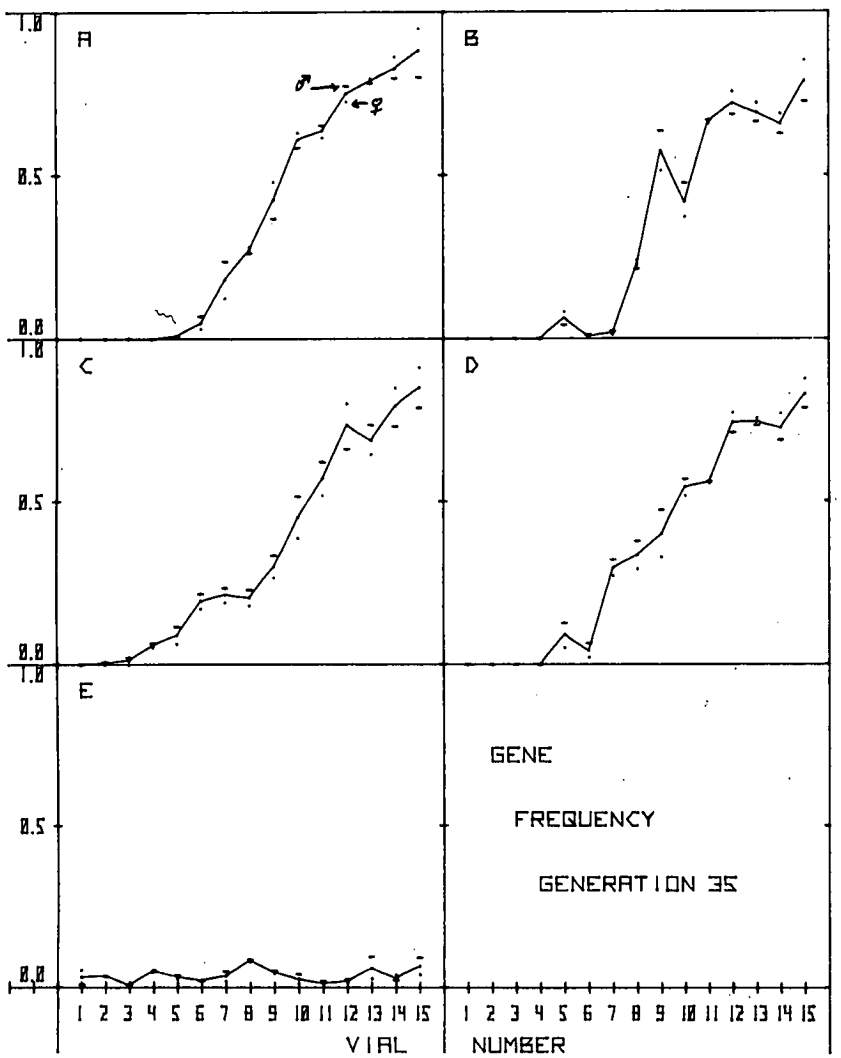


FIGURE 3 - 3. Bar gene frequency clines for generations 26 - 35.



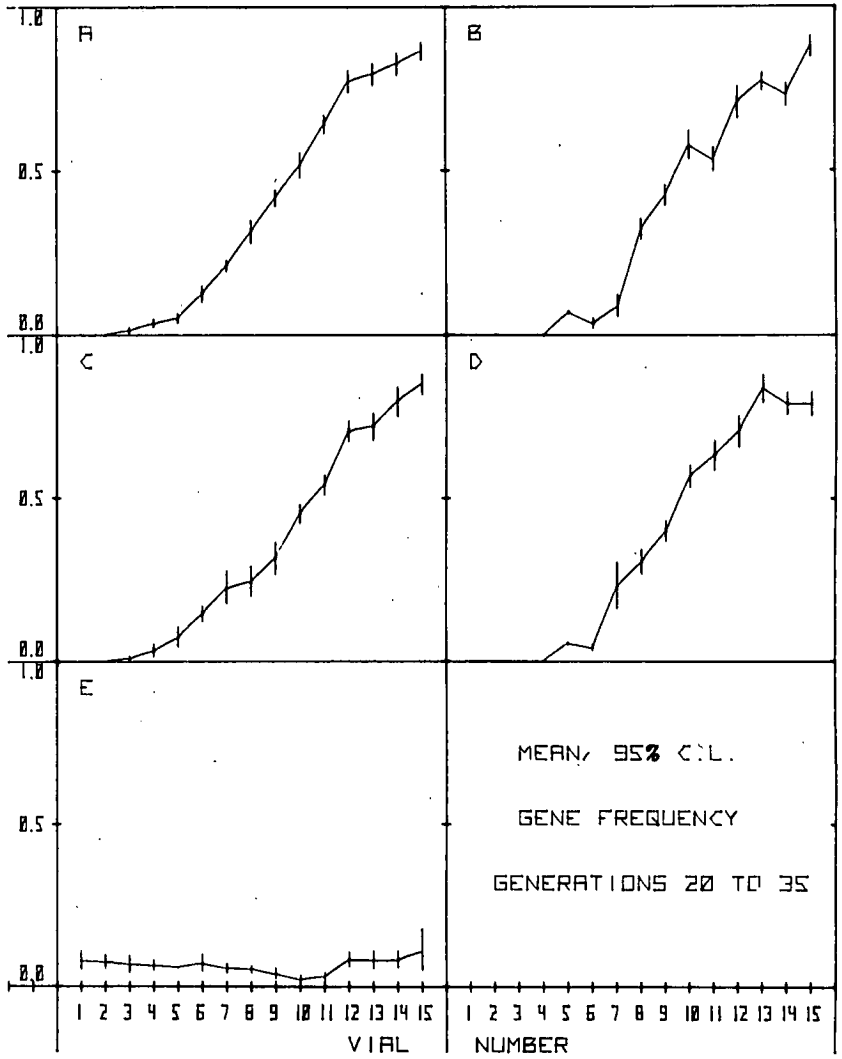


FIGURE 3 - 3, continued.

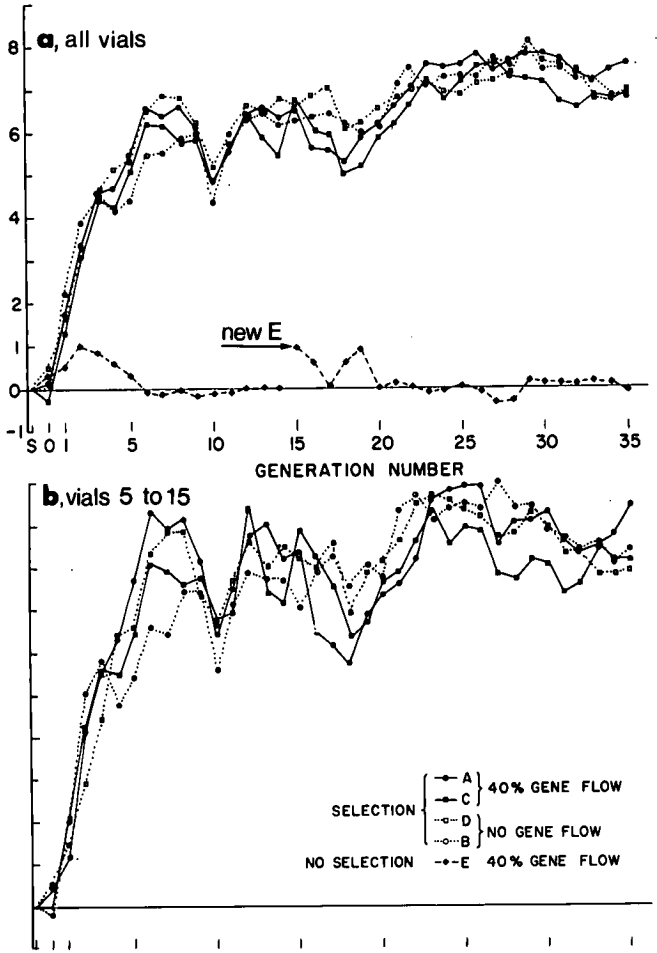


FIGURE 3–4
SLOPES OF THE CLINES

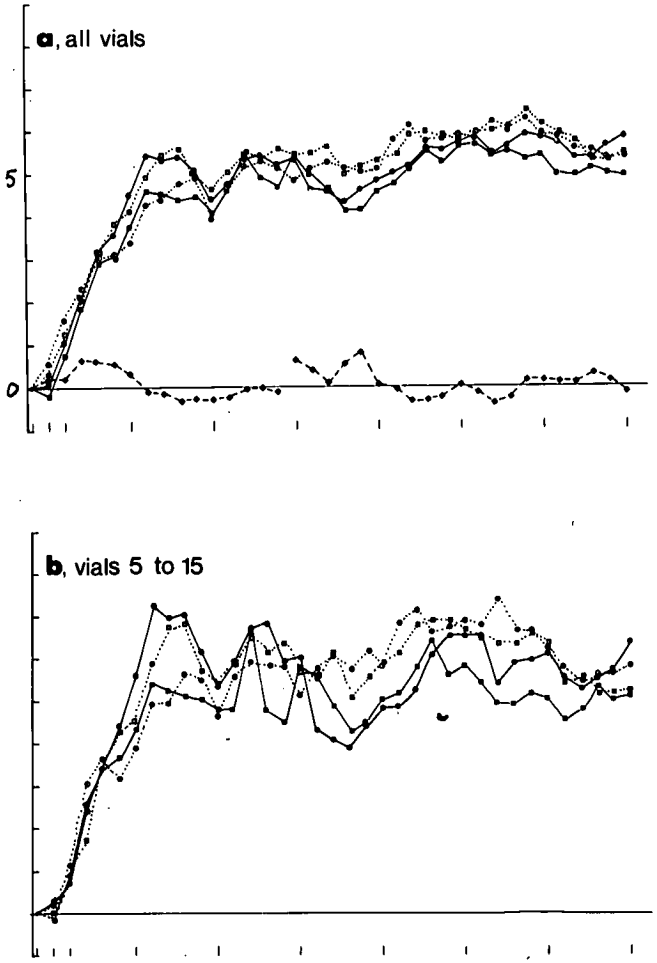


FIGURE 3–5
SLOPES [ANGLES]

SET A, SELECTION, GENE FLOW

GEN. NO.	VIAL NUMBER 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
S	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500
0	0.355	0.360	0.335	0.415	0.330	0.305	0.288	0.380	0.450	0.377	0.370	0.325	0.365	0.395	0.350
1	0.198	0.242	0.236	0.405	0.356	0.394	0.413	0.367	0.347	0.409	0.380	0.408	0.516	0.511	0.446
2	0.162	0.177	0.159	0.242	0.189	0.254	0.302	0.345	0.328	0.377	0.401	0.495	0.519	0.539	0.667
3	0.057	0.068	0.040	0.027	0.099	0.141	0.212	0.218	0.187	0.365	0.502	0.469	0.574	0.644	0.533
4	0.027	0.018	0.006	0.062	0.017	0.058	0.106	0.119	0.219	0.291	0.396	0.510	0.513	0.490	0.630
5	0.000	0.000	0.002	0.000	0.013	0.016	0.019	0.078	0.173	0.336	0.391	0.560	0.614	0.607	0.648
6	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.061	0.280	0.330	0.586	0.648	0.672	0.684	0.802
7	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.153	0.248	0.353	0.510	0.587	0.683	0.739	0.738
8	0.000	0.000	0.000	0.000	0.000	0.000	0.006	0.135	0.268	0.404	0.475	0.595	0.672	0.776	0.764
9	0.000	0.000	0.000	0.000	0.000	0.000	0.096	0.168	0.290	0.344	0.517	0.566	0.645	0.715	0.690
10	0.000	0.000	0.000	0.000	0.000	0.000	0.011	0.085	0.347	0.292	0.430	0.506	0.575	0.543	0.452
11	0.000	0.000	0.000	0.000	0.000	0.000	0.014	0.126	0.328	0.336	0.445	0.473	0.642	0.576	0.649
12	0.000	0.000	0.000	0.000	0.000	0.000	0.013	0.197	0.292	0.435	0.478	0.561	0.688	0.635	0.833
13	0.000	0.000	0.000	0.000	0.000	0.000	0.043	0.137	0.341	0.404	0.508	0.509	0.627	0.728	0.870
14	0.000	0.000	0.000	0.000	0.000	0.019	0.076	0.267	0.407	0.405	0.413	0.638	0.586	0.713	0.793
15	0.000	0.000	0.000	0.000	0.000	0.019	0.140	0.245	0.378	0.434	0.470	0.602	0.697	0.698	0.799
16	0.000	0.000	0.000	0.014	0.040	0.082	0.107	0.301	0.426	0.416	0.517	0.531	0.600	0.674	0.586
17	0.000	0.000	0.000	0.004	0.026	0.141	0.142	0.292	0.412	0.525	0.455	0.570	0.646	0.583	0.610
18	0.000	0.000	0.000	0.010	0.021	0.143	0.224	0.249	0.427	0.482	0.371	0.565	0.572	0.583	0.605
19	0.000	0.000	0.000	0.048	0.047	0.122	0.168	0.270	0.355	0.375	0.519	0.622	0.652	0.635	0.695
20	0.000	0.000	0.000	0.067	0.014	0.166	0.216	0.304	0.370	0.391	0.538	0.628	0.659	0.701	0.771
21	0.000	0.000	0.004	0.077	0.046	0.167	0.223	0.308	0.425	0.534	0.553	0.727	0.720	0.729	0.781
22	0.000	0.000	0.033	0.068	0.045	0.168	0.154	0.359	0.502	0.567	0.636	0.759	0.735	0.779	0.828
23	0.000	0.000	0.071	0.027	0.087	0.068	0.194	0.250	0.402	0.478	0.727	0.809	0.862	0.834	0.891
24	0.000	0.004	0.030	0.050	0.026	0.076	0.188	0.228	0.346	0.408	0.696	0.763	0.800	0.840	0.923
25	0.000	0.000	0.006	0.039	0.025	0.082	0.186	0.160	0.331	0.434	0.680	0.820	0.821	0.854	0.866
26	0.000	0.000	0.032	0.033	0.038	0.080	0.205	0.263	0.378	0.550	0.645	0.862	0.856	0.885	0.889
27	0.000	0.000	0.034	0.069	0.091	0.240	0.246	0.357	0.395	0.517	0.677	0.828	0.873	0.852	0.866
28	0.000	0.005	0.021	0.050	0.063	0.195	0.243	0.312	0.428	0.363	0.637	0.781	0.862	0.913	0.891
29	0.000	0.000	0.004	0.025	0.082	0.127	0.257	0.407	0.493	0.480	0.591	0.883	0.850	0.873	0.928
30	0.000	0.000	0.017	0.037	0.069	0.123	0.231	0.381	0.471	0.511	0.666	0.827	0.892	0.845	0.933
31	0.000	0.000	0.018	0.038	0.080	0.179	0.279	0.404	0.463	0.639	0.724	0.727	0.871	0.883	0.880
32	0.000	0.010	0.023	0.019	0.095	0.156	0.221	0.416	0.457	0.599	0.641	0.760	0.761	0.876	0.857
33	0.000	0.011	0.002	0.009	0.058	0.099	0.156	0.361	0.414	0.527	0.624	0.734	0.767	0.778	0.858
34	0.000	0.000	0.002	0.016	0.043	0.094	0.239	0.354	0.422	0.571	0.667	0.769	0.732	0.845	0.867
35	0.000	0.000	0.014	0.000	0.009	0.049	0.181	0.272	0.425	0.608	0.634	0.748	0.788	0.826	0.881

SET B, SELECTION ONLY

GEN. NO.	VIAL NUMBER 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
S	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500
0	0.320	0.320	0.436	0.375	0.361	0.450	0.400	0.305	0.426	0.381	0.390	0.347	0.385	0.298	0.435
1	0.024	0.208	0.365	0.261	0.301	0.343	0.316	0.329	0.359	0.374	0.382	0.434	0.470	0.532	0.459
2	0.180	0.150	0.252	0.228	0.239	0.256	0.210	0.238	0.360	0.497	0.430	0.479	0.647	0.677	0.652
3	0.057	0.051	0.124	0.153	0.120	0.126	0.126	0.180	0.408	0.604	0.363	0.473	0.630	0.612	0.587
4	0.029	0.004	0.066	0.091	0.054	0.107	0.157	0.169	0.473	0.488	0.321	0.330	0.529	0.486	0.539
5	0.037	0.000	0.071	0.078	0.003	0.099	0.191	0.204	0.394	0.421	0.311	0.298	0.556	0.630	0.533
6	0.016	0.000	0.016	0.038	0.000	0.036	0.073	0.389	0.451	0.538	0.397	0.329	0.638	0.720	0.604
7	0.011	0.000	0.019	0.011	0.000	0.004	0.197	0.411	0.379	0.464	0.453	0.407	0.736	0.647	0.567
8	0.000	0.000	0.025	0.008	0.000	0.000	0.152	0.241	0.433	0.462	0.482	0.401	0.668	0.730	0.663
9	0.000	0.000	0.011	0.000	0.000	0.010	0.276	0.127	0.474	0.496	0.447	0.366	0.636	0.664	0.786
10	0.000	0.000	0.000	0.016	0.000	0.000	0.206	0.081	0.252	0.324	0.303	0.272	0.547	0.525	0.513
11	0.000	0.000	0.000	0.029	0.000	0.000	0.330	0.120	0.356	0.390	0.431	0.513	0.616	0.693	0.674
12	0.000	0.000	0.000	0.000	0.000	0.000	0.302	0.214	0.248	0.472	0.455	0.615	0.657	0.765	0.709
13	0.000	0.000	0.000	0.000	0.000	0.000	0.308	0.298	0.372	0.541	0.463	0.663	0.563	0.766	0.769
14	0.000	0.000	0.000	0.000	0.000	0.000	0.203	0.271	0.327	0.472	0.398	0.687	0.562	0.739	0.708
15	0.000	0.000	0.000	0.000	0.000	0.032	0.143	0.334	0.331	0.409	0.409	0.579	0.626	0.695	0.625
16	0.000	0.000	0.000	0.000	0.000	0.014	0.167	0.299	0.335	0.411	0.497	0.566	0.716	0.742	0.695
17	0.000	0.000	0.000	0.000	0.000	0.005	0.107	0.261	0.254	0.527	0.488	0.639	0.748	0.750	0.639
18	0.000	0.000	0.000	0.000	0.000	0.000	0.123	0.420	0.306	0.553	0.525	0.526	0.697	0.677	0.686
19	0.000	0.000	0.000	0.000	0.000	0.000	0.030	0.214	0.287	0.438	0.479	0.457	0.676	0.687	0.697
20	0.000	0.000	0.000	0.000	0.000	0.006	0.045	0.297	0.444	0.446	0.473	0.589	0.674	0.596	0.733
21	0.000	0.000	0.000	0.000	0.000	0.019	0.035	0.309	0.406	0.498	0.534	0.709	0.747	0.663	0.930
22	0.000	0.000	0.000	0.000	0.000	0.015	0.022	0.329	0.428	0.533	0.644	0.724	0.821	0.701	0.912
23	0.000	0.000	0.000	0.000	0.000	0.031	0.071	0.355	0.389	0.558	0.478	0.611	0.803	0.681	0.925
24	0.000	0.000	0.000	0.000	0.000	0.050	0.048	0.350	0.347	0.555	0.474	0.676	0.849	0.753	0.873
25	0.000	0.000	0.000	0.000	0.000	0.045	0.036	0.390	0.342	0.559	0.478	0.644	0.843	0.746	0.918
26	0.000	0.000	0.000	0.000	0.000	0.020	0.047	0.319	0.446	0.672	0.400	0.637	0.810	0.798	0.866
27	0.000	0.000	0.000	0.000	0.000	0.021	0.065	0.354	0.394	0.595	0.454	0.768	0.773	0.851	0.960
28	0.000	0.000	0.000	0.000	0.000	0.035	0.128	0.342	0.434	0.666	0.543	0.732	0.794	0.808	0.883
29	0.000	0.000	0.000	0.000	0.000	0.074	0.204	0.448	0.426	0.703	0.600	0.900	0.826	0.794	0.916
30	0.000	0.000	0.000	0.000	0.000	0.087	0.195	0.394	0.437	0.676	0.524	0.808	0.772	0.779	0.885
31	0.000	0.000	0.000	0.000	0.043	0.087	0.249	0.373	0.493	0.665	0.527	0.778	0.752	0.789	0.927
32	0.000	0.000	0.000	0.000	0.072	0.085	0.178	0.299	0.474	0.636	0.537	0.683	0.778	0.681	0.920
33	0.000	0.000	0.000	0.000	0.099	0.040	0.159	0.237	0.440	0.595	0.588	0.735	0.740	0.747	0.851
34	0.000	0.000	0.000	0.000	0.093	0.061	0.125	0.256	0.437	0.571	0.659	0.702	0.707	0.634	0.840
35	0.000	0.000	0.000	0.000	0.064	0.008	0.018	0.226	0.572	0.417	0.662	0.718	0.690	0.656	0.787

SET C, SELECTION, GENE FLOW

GEN. NO.	VIAL NUMBER 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
S	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500
0	0.373	0.373	0.406	0.470	0.470	0.356	0.354	0.375	0.275	0.331	0.312	0.296	0.474	0.415	0.367
1	0.298	0.324	0.369	0.387	0.348	0.321	0.278	0.358	0.417	0.385	0.303	0.445	0.481	0.539	0.490
2	0.181	0.216	0.234	0.293	0.201	0.208	0.297	0.391	0.385	0.330	0.331	0.564	0.656	0.426	0.671
3	0.093	0.074	0.061	0.069	0.103	0.162	0.169	0.303	0.271	0.314	0.287	0.500	0.644	0.468	0.710
4	0.025	0.043	0.070	0.033	0.062	0.113	0.148	0.056	0.267	0.303	0.436	0.403	0.467	0.428	0.651
5	0.010	0.010	0.066	0.036	0.035	0.081	0.117	0.230	0.288	0.376	0.469	0.484	0.594	0.513	0.683
6	0.000	0.006	0.057	0.094	0.034	0.047	0.145	0.282	0.337	0.401	0.581	0.597	0.519	0.718	0.878
7	0.000	0.012	0.049	0.070	0.046	0.049	0.139	0.315	0.379	0.337	0.521	0.534	0.653	0.701	0.839
8	0.000	0.023	0.010	0.083	0.021	0.143	0.092	0.254	0.314	0.348	0.392	0.468	0.538	0.701	0.877
9	0.000	0.003	0.022	0.117	0.112	0.061	0.103	0.196	0.341	0.280	0.433	0.506	0.604	0.661	0.893
10	0.021	0.000	0.004	0.000	0.023	0.031	0.090	0.173	0.226	0.161	0.231	0.363	0.548	0.452	0.821
11	0.000	0.000	0.000	0.000	0.004	0.196	0.097	0.255	0.290	0.181	0.348	0.473	0.619	0.761	0.668
12	0.000	0.000	0.000	0.000	0.000	0.051	0.088	0.139	0.173	0.265	0.601	0.645	0.713	0.792	0.820
13	0.000	0.000	0.000	0.000	0.035	0.123	0.222	0.251	0.246	0.378	0.504	0.575	0.634	0.737	0.735
14	0.000	0.000	0.000	0.018	0.082	0.165	0.112	0.222	0.297	0.402	0.475	0.603	0.611	0.678	0.750
15	0.000	0.000	0.000	0.011	0.051	0.088	0.141	0.190	0.371	0.418	0.526	0.734	0.730	0.775	0.856
16	0.000	0.000	0.000	0.053	0.031	0.038	0.097	0.252	0.318	0.469	0.556	0.651	0.649	0.647	0.820
17	0.000	0.000	0.000	0.078	0.078	0.113	0.052	0.160	0.291	0.447	0.559	0.577	0.615	0.671	0.734
18	0.000	0.000	0.000	0.051	0.049	0.073	0.063	0.188	0.226	0.326	0.438	0.514	0.522	0.598	0.578
19	0.000	0.000	0.010	0.045	0.057	0.084	0.069	0.136	0.194	0.342	0.503	0.620	0.516	0.542	0.650
20	0.000	0.000	0.011	0.017	0.043	0.125	0.064	0.115	0.221	0.423	0.410	0.575	0.599	0.651	0.772
21	0.000	0.000	0.019	0.039	0.026	0.101	0.160	0.141	0.233	0.340	0.518	0.629	0.573	0.674	0.805
22	0.000	0.000	0.009	0.014	0.020	0.051	0.184	0.144	0.215	0.427	0.603	0.684	0.679	0.677	0.833
23	0.000	0.000	0.027	0.000	0.000	0.116	0.183	0.199	0.223	0.450	0.589	0.761	0.742	0.827	0.850
24	0.000	0.000	0.008	0.000	0.019	0.116	0.181	0.196	0.278	0.436	0.530	0.673	0.702	0.811	0.787
25	0.000	0.000	0.000	0.000	0.053	0.177	0.233	0.241	0.216	0.371	0.509	0.678	0.774	0.920	0.872
26	0.000	0.000	0.000	0.014	0.040	0.214	0.373	0.379	0.329	0.425	0.609	0.781	0.753	0.889	0.899
27	0.000	0.000	0.000	0.017	0.102	0.208	0.343	0.384	0.432	0.426	0.486	0.766	0.742	0.847	0.899
28	0.000	0.000	0.000	0.051	0.168	0.190	0.435	0.368	0.438	0.512	0.544	0.753	0.789	0.885	0.923
29	0.000	0.000	0.042	0.065	0.137	0.177	0.240	0.400	0.419	0.500	0.670	0.822	0.724	0.833	0.908
30	0.000	0.000	0.000	0.095	0.123	0.192	0.293	0.299	0.496	0.506	0.616	0.742	0.845	0.810	0.871
31	0.000	0.000	0.011	0.087	0.162	0.171	0.300	0.273	0.477	0.527	0.544	0.689	0.797	0.740	0.849
32	0.000	0.000	0.008	0.102	0.137	0.151	0.272	0.264	0.369	0.483	0.514	0.662	0.680	0.770	0.885
33	0.000	0.000	0.014	0.085	0.112	0.114	0.208	0.259	0.295	0.488	0.468	0.647	0.797	0.801	0.888
34	0.000	0.000	0.063	0.043	0.161	0.167	0.230	0.258	0.305	0.405	0.496	0.709	0.796	0.857	0.837
35	0.000	0.004	0.012	0.058	0.089	0.195	0.215	0.206	0.299	0.450	0.571	0.736	0.688	0.794	0.851

SET D, SELECTION ONLY

GEN. NO.	VIAL NUMBER 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
S	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500
0	0.338	0.319	0.358	0.380	0.339	0.280	0.470	0.420	0.440	0.385	0.380	0.460	0.295	0.375	0.455
1	0.249	0.235	0.246	0.350	0.376	0.196	0.397	0.419	0.488	0.369	0.437	0.423	0.381	0.479	0.489
2	0.127	0.154	0.159	0.237	0.286	0.294	0.367	0.348	0.458	0.375	0.434	0.517	0.519	0.554	0.569
3	0.027	0.067	0.059	0.128	0.119	0.116	0.198	0.202	0.542	0.320	0.355	0.549	0.611	0.558	0.612
4	0.008	0.008	0.051	0.070	0.096	0.011	0.141	0.216	0.381	0.394	0.297	0.560	0.680	0.626	0.580
5	0.007	0.014	0.000	0.035	0.108	0.000	0.174	0.277	0.291	0.336	0.331	0.545	0.650	0.580	0.688
6	0.041	0.002	0.000	0.009	0.052	0.000	0.102	0.205	0.467	0.524	0.430	0.661	0.869	0.731	0.667
7	0.026	0.000	0.000	0.000	0.000	0.000	0.065	0.301	0.385	0.569	0.500	0.723	0.926	0.733	0.636
8	0.018	0.000	0.000	0.000	0.000	0.000	0.061	0.291	0.374	0.571	0.422	0.625	0.984	0.662	0.721
9	0.016	0.000	0.000	0.000	0.000	0.000	0.193	0.541	0.340	0.466	0.381	0.572	0.882	0.633	0.686
10	0.000	0.000	0.000	0.000	0.000	0.008	0.070	0.377	0.199	0.305	0.301	0.337	0.934	0.515	0.529
11	0.000	0.000	0.000	0.000	0.000	0.000	0.172	0.335	0.102	0.481	0.448	0.506	0.896	0.566	0.654
12	0.000	0.000	0.000	0.000	0.000	0.000	0.211	0.175	0.213	0.417	0.663	0.574	0.922	0.560	0.772
13	0.000	0.000	0.000	0.000	0.000	0.000	0.217	0.237	0.332	0.442	0.573	0.517	0.950	0.563	0.720
14	0.000	0.000	0.000	0.000	0.000	0.000	0.216	0.337	0.292	0.465	0.627	0.565	0.908	0.698	0.731
15	0.000	0.000	0.000	0.000	0.000	0.115	0.193	0.309	0.241	0.457	0.593	0.597	0.875	0.723	0.725
16	0.000	0.000	0.000	0.000	0.000	0.108	0.222	0.503	0.266	0.500	0.628	0.693	0.844	0.747	0.716
17	0.000	0.000	0.000	0.000	0.000	0.059	0.119	0.232	0.485	0.529	0.666	0.821	0.846	0.728	0.654
18	0.000	0.000	0.000	0.000	0.000	0.080	0.147	0.299	0.341	0.512	0.605	0.760	0.749	0.615	0.512
19	0.000	0.000	0.000	0.000	0.000	0.109	0.084	0.196	0.306	0.500	0.488	0.603	0.751	0.794	0.583
20	0.000	0.000	0.000	0.000	0.000	0.041	0.141	0.205	0.390	0.523	0.586	0.618	0.638	0.848	0.646
21	0.000	0.000	0.000	0.000	0.000	0.034	0.094	0.213	0.387	0.569	0.593	0.587	0.641	0.842	0.748
22	0.000	0.000	0.000	0.000	0.000	0.028	0.118	0.245	0.442	0.580	0.583	0.589	0.791	0.916	0.809
23	0.000	0.000	0.000	0.000	0.000	0.028	0.112	0.259	0.402	0.490	0.637	0.659	0.874	0.871	0.798
24	0.000	0.000	0.000	0.000	0.000	0.034	0.053	0.227	0.365	0.526	0.660	0.556	0.893	0.806	0.796
25	0.000	0.000	0.000	0.000	0.000	0.036	0.090	0.258	0.315	0.540	0.542	0.720	0.889	0.763	0.779
26	0.000	0.000	0.000	0.000	0.000	0.055	0.188	0.366	0.362	0.539	0.500	0.868	0.898	0.795	0.781
27	0.000	0.000	0.000	0.000	0.000	0.013	0.402	0.349	0.449	0.637	0.623	0.819	0.911	0.770	0.785
28	0.000	0.000	0.000	0.000	0.000	0.020	0.445	0.310	0.500	0.655	0.670	0.795	0.905	0.765	0.838
29	0.000	0.000	0.000	0.000	0.000	0.065	0.423	0.376	0.445	0.742	0.709	0.848	0.889	0.855	0.887
30	0.000	0.000	0.000	0.000	0.000	0.054	0.390	0.374	0.490	0.635	0.754	0.750	0.865	0.773	0.905
31	0.000	0.000	0.000	0.000	0.071	0.075	0.365	0.353	0.506	0.641	0.775	0.709	0.927	0.742	0.844
32	0.000	0.000	0.000	0.000	0.042	0.038	0.344	0.440	0.360	0.524	0.760	0.735	0.908	0.710	0.812
33	0.000	0.000	0.000	0.000	0.041	0.045	0.329	0.369	0.299	0.500	0.616	0.660	0.878	0.632	0.776
34	0.000	0.000	0.000	0.000	0.048	0.032	0.307	0.362	0.359	0.500	0.573	0.667	0.768	0.766	0.745
35	0.000	0.000	0.000	0.000	0.091	0.043	0.295	0.337	0.402	0.546	0.560	0.744	0.746	0.727	0.831

SET E, GENE FLOW ONLY

GEN. NO.	VIAL NUMBER 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
S	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500	0.500
0	0.360	0.350	0.466	0.415	0.490	0.430	0.381	0.405	0.525	0.450	0.439	0.409	0.475	0.440	0.400
1	0.347	0.304	0.391	0.340	0.368	0.417	0.391	0.364	0.497	0.371	0.310	0.357	0.428	0.427	0.368
2	0.201	0.189	0.238	0.285	0.221	0.258	0.300	0.186	0.339	0.280	0.333	0.359	0.358	0.324	0.303
3	0.118	0.171	0.209	0.124	0.166	0.157	0.112	0.134	0.128	0.187	0.250	0.301	0.171	0.204	0.312
4	0.112	0.081	0.044	0.206	0.111	0.120	0.105	0.095	0.134	0.156	0.187	0.198	0.185	0.106	0.178
5	0.071	0.096	0.042	0.165	0.127	0.106	0.081	0.049	0.063	0.085	0.191	0.109	0.104	0.109	0.157
6	0.052	0.058	0.102	0.231	0.116	0.049	0.076	0.069	0.050	0.031	0.128	0.045	0.092	0.177	0.034
7	0.104	0.028	0.128	0.140	0.143	0.098	0.009	0.027	0.056	0.053	0.037	0.076	0.075	0.134	0.071
8	0.180	0.074	0.113	0.215	0.067	0.039	0.012	0.023	0.078	0.088	0.020	0.037	0.062	0.085	0.130
9	0.113	0.093	0.106	0.049	0.029	0.031	0.030	0.052	0.083	0.033	0.078	0.041	0.082	0.013	0.083
10	0.081	0.034	0.019	0.009	0.078	0.000	0.070	0.000	0.043	0.004	0.012	0.008	0.009	0.006	0.073
11	0.065	0.017	0.000	0.004	0.035	0.041	0.017	0.000	0.014	0.007	0.000	0.000	0.000	0.016	0.053
12	0.017	0.004	0.000	0.000	0.013	0.006	0.000	0.004	0.015	0.008	0.000	0.000	0.000	0.006	0.013
13	0.004	0.000	0.000	0.002	0.000	0.017	0.000	0.005	0.000	0.005	0.000	0.000	0.000	0.000	0.008
14	0.000	0.003	0.000	0.000	0.004	0.008	0.065	0.087	0.049	0.000	0.000	0.000	0.000	0.000	0.000
(NEW SET E SET UP FOR GENERATION 15)															
15	0.247	0.237	0.235	0.238	0.214	0.259	0.403	0.403	0.332	0.220	0.318	0.322	0.398	0.397	0.306
16	0.215	0.278	0.133	0.211	0.235	0.286	0.206	0.321	0.295	0.166	0.262	0.360	0.207	0.272	0.370
17	0.122	0.331	0.106	0.185	0.267	0.194	0.157	0.183	0.245	0.130	0.070	0.110	0.325	0.192	0.222
18	0.066	0.287	0.068	0.065	0.140	0.180	0.147	0.071	0.134	0.105	0.224	0.240	0.243	0.169	0.158
19	0.121	0.090	0.027	0.030	0.039	0.153	0.091	0.053	0.147	0.072	0.035	0.190	0.251	0.194	0.157
20	0.239	0.096	0.031	0.105	0.122	0.218	0.086	0.082	0.040	0.062	0.041	0.103	0.128	0.195	0.171
21	0.133	0.090	0.050	0.094	0.151	0.145	0.089	0.048	0.037	0.009	0.048	0.121	0.160	0.151	0.093
22	0.131	0.086	0.044	0.079	0.133	0.078	0.111	0.061	0.058	0.028	0.005	0.100	0.096	0.067	0.053
23	0.150	0.209	0.073	0.052	0.069	0.093	0.066	0.052	0.052	0.026	0.027	0.090	0.075	0.050	0.181
24	0.096	0.099	0.126	0.053	0.049	0.056	0.064	0.036	0.049	0.015	0.010	0.104	0.080	0.089	0.061
25	0.102	0.122	0.218	0.089	0.051	0.054	0.124	0.028	0.020	0.054	0.062	0.136	0.196	0.172	0.079
26	0.098	0.059	0.208	0.142	0.052	0.039	0.045	0.046	0.024	0.023	0.023	0.070	0.162	0.081	0.115
27	0.059	0.074	0.069	0.106	0.156	0.048	0.036	0.012	0.013	0.002	0.037	0.057	0.062	0.062	0.030
28	0.075	0.076	0.118	0.068	0.056	0.054	0.025	0.077	0.000	0.033	0.018	0.082	0.030	0.066	0.078
29	0.053	0.073	0.084	0.041	0.052	0.017	0.054	0.063	0.031	0.000	0.022	0.109	0.082	0.096	0.095
30	0.042	0.046	0.041	0.033	0.008	0.024	0.041	0.035	0.030	0.000	0.038	0.086	0.057	0.057	0.042
31	0.020	0.089	0.040	0.039	0.042	0.043	0.036	0.073	0.036	0.009	0.082	0.053	0.080	0.060	0.050
32	0.027	0.064	0.083	0.076	0.038	0.075	0.025	0.058	0.088	0.065	0.056	0.087	0.033	0.068	0.055
33	0.032	0.017	0.069	0.068	0.064	0.035	0.064	0.071	0.063	0.050	0.069	0.067	0.055	0.058	0.046
34	0.032	0.033	0.007	0.050	0.032	0.021	0.034	0.081	0.066	0.025	0.013	0.019	0.057	0.029	0.063
35	0.037	0.028	0.060	0.037	0.025	0.025	0.028	0.074	0.031	0.010	0.010	0.021	0.013	0.021	0.050

TABLE 3 - 0. Gene frequencies in each vial, in each generation.

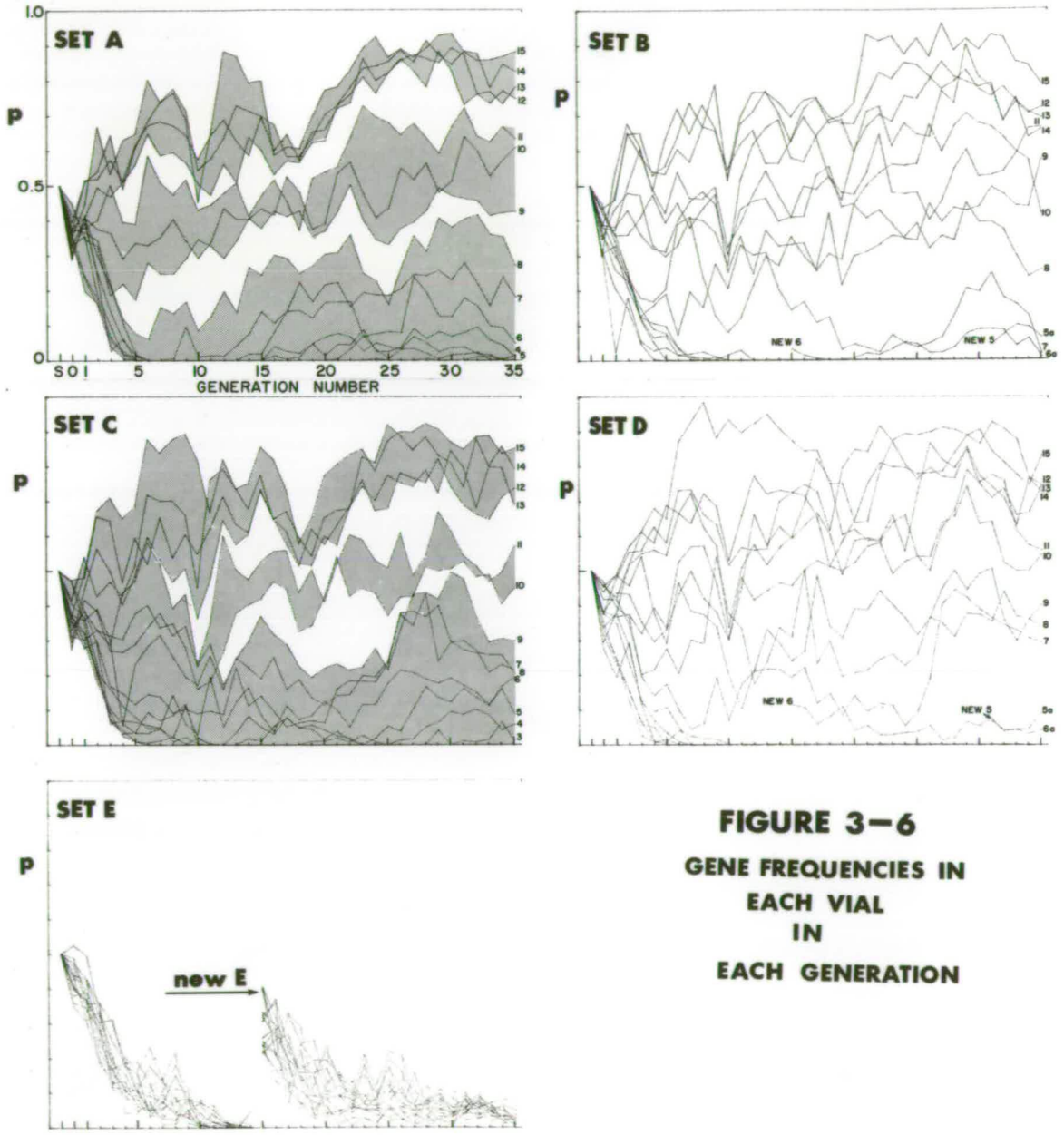


FIGURE 3–6

GENE FREQUENCIES IN EACH VIAL IN EACH GENERATION

with 40% gene flow (A and C), and those without gene flow (B and D) from generation to generation.

Set E (no selection) lost Bar from almost all demes by generation 14. It was therefore set up anew from Bar-Kaduna for generation 15 (figure 3-4). It nearly lost all Bar genes again by the end of the experiment.

During the first few generations, vials 1 through 6 in sets A through D lost Bar as a result of the natural and artificial selection against Bar phenotypes. These vials remained phenotypically normal in all subsequent generations in sets B and D (no gene flow). The vials in the same positions of sets A and C were subject to gene flow from vials with Bar genes present, and Bar could reinvade as it gradually adapted to the new environment of the selection gradient (figures 3-3 and 3-6, see also section 3.7). Thus the clines in sets A and C were slightly different from B and D from the early generations.

As a control for this historical factor, replicates of the vials of positions 6 of sets B and D were set up and given one generation of gene flow in generation 15, and from these, replicates of position 5 were given one generation of gene flow in generation 31. The fate of these extra vials is shown in figure 3-6b and d.

Excluding the monomorphic vials, 1 through 4, from calculation of the clines' slopes, to make the replicates more comparable, still shows no significant or consistent differences between replicates with gene flow and those replicates without gene flow (figure 3-4b).

The effect of gene flow becomes just detectable of one considers the slopes of the clines calculated on gene frequencies which have be been transformed into angles (figure 3-5). The angular transformation weights differences between extreme frequencies more than differences of similar magnitude at intermediate

frequencies, and reduces the dependence of expected variance on gene frequency (Fisher and Yates, 1948, Sokal and Rohlf, 1968, Rohlf and Sokal, 1968). The dedifferentiating effect of gene flow is still small, and could easily be obscured in field studies of clines with similar parameters.

Section 3.5. Differential Coadaptation

Two striking phenomena are apparent in the sets with selection (Sets A - D, figure 3-6), the gradual increase in Bar gene frequency, and gaps in the domains of variation of gene frequency in certain areas.

During the early generations the Bar gene frequency in most vials declined or remained roughly constant. During later generations, however, Bar gene frequencies in most of the vials increased at a regular rate. In the replicates with gene flow (A and C), invasion of monomorphic wild vials by Bar genes was followed by a slow increase of their gene frequency, succeeded by further invasion down the cline. The effect is rather like that of the spread of an advantageous gene described by Fisher (1937), but if Bar is advantageous in these vials why was it lost from them in the first six to ten generations? Why do the extra vials 6 and 5 in sets B and D remain roughly constant whilst their equivalents in the sets with gene flow increase markedly after about generation 20?

The two replicates with gene flow (A and C) show another interesting phenomenon. In both, vial 12 has a consistently higher gene frequency than vial 11 from as early as generation 8. Vial 9 in both replicates is associated with a second gap in the

domain of gene frequency variation. In set A vial 9 is at a consistently higher gene frequency than vial 8 from generation 4, and in set C vial 9 is consistently lower than vial 10. Vials 12-15 appear to belong to a group of demes which vary together, but separately from a group of vials at intermediate frequency. The intermediate group seems distinct from a third group of demes with low Bar gene frequency. These groups are shown connected by shading in figure 3-6. Sets B and D show no such gaps, except for the difference between fixed and unfixed demes. There does not appear to be any objective way of testing the significance of this observation, but the consistency of the effect (over 20 generations), and the fact that the gaps occur in the same position in both clines is highly suggestive.

If we assume for the moment that the gaps are real this implies that the clines have differentiated into three groups with similar gene frequencies, separated by morphotones, or are in the process of developing morphotones. Morphotones are in fact observed between vials 11 and 12 in generations 25-30 in set A, between 10 and 11 in generations 23-25 in set A, between vials 11 and 12 for 2 or three generations at a time from generation 23-35 in set C, and between vials 9 and 10 from generations 19 to 25 in set C. The clines have a two-morphotone form in a few generations, notably set A in generation 19, and set C in generation 27. Such discontinuities are not long lived, however they do appear to occur at about the same gene frequencies, 0.2-0.3, and 0.65-0.75, regardless of the demes which they involve. This is especially apparent in set C from generations 25-34. Until generation 25 a morphotone was present between vials 9 and 10. In generation 26,

vials 7, 8, and 9 suddenly increased in gene frequency, and a morphotone appeared between vials 6 and 7. After generation 29, vials 7, 8, and 9 fell, and the old gap broadened again. The morphotone was at first centered about a gene frequency of 0.3, next 0.29, and finally at about 0.34.

These observations are difficult to explain on the basis of one-locus selection and gene flow models (chapter 2). If selection were only a function of one locus, then the observed fitnesses and equilibrium gene frequencies would not change slowly with time (figure 3-6). During the investigations described in chapter 2, no model could be found that produced two morphotones. It is most likely, therefore, that the selection gradient for Bar also affected other loci.

It is a moderately old idea that if a given type is favoured in a particular area, then all factors which increase the favoured type's fitness will themselves be favoured, forming a geographical pattern of local adaptive races, characterized by particular gene complexes. Some of the first evidence for the selection for locally adaptive gene complexes may be found in the studies of Timofeeff-Ressovsky (1940), and Dobzansky (1937, 1951). Fisher (1930) and Huxley (1939) were among the first to recognize the importance of this idea in regard to geographical differentiation, for example:

> Whenever two relatively large and uniform areas were separated by regions of relatively rapid environmental change, the effect of selection would be to produce two main types of gene-complex, each stabilized by its own set of modifiers giving maximum harmony and viability. So long as the population is continuous, these will interbreed where they meet. But the recombinations between them being ex hypothesi less well adapted and harmonius then either of the two main complexes,

> will remain restricted to a narrow zone, and will not spread progressively through the population. (Huxley, 1939)

Further evidence and discussion of the idea of coadaptation may be found in Dobzhansky (1939, 1951, 1970), Mayr (1956, 1963), Brncic (1961), Vetukhiv (1953, 1954, 1956), Wallace (1955, 1968b), Moore (1946, 1949a, 1954), and Kuhn (1971). A discussion and critical review of the more circumstantial evidence is found in Clarke (1968).

Huxley's (and others') arguments provide us with two testable predictions: that the gene complex characteristic of a particular environment will be more fit in its own environment than in others, and that offspring of crosses (both natural and experimental) between differently coadapted types will exhibit reduced or more variable fitness compared to the parental types. The theoretical study of coadapted clines by Clarke (1966) provides two additional predictions; slopes of clines may be changed by modifiers, and the spatially restricted spread of modifiers may cause morphotones to form where they are not expected on the basis of one locus theory.

It is of interest to know whether any differential coadaptation has taken place along the experimental clines; whether the Bar phenotypes have become well adapted at the Bar end of the clines (vials 10-15), and the other phenotypes differentially adapted to the other end of the selection gradient. It is of great interest (chapter 1) to enquire whether or not the presence of 40% gene flow has affected the development and extent of any coadaptation which may have evolved during the course of the experiment. The recombination-backcrossing method of introducing Bar into Kaduna should have eliminated any previously Bar coadapting gene complexes.

I will use a modified version of Clarke's (1966) model to give a

more precise theoretical basis to the predictions before going on to describe the results in detail.

Section 3.6. Models of Coadaptation

Consider a locus A with two alleles A and a, and a modifier locus B, also with two alleles B and b. Locus A is subject to selection, and as in chapter 2 we will denote the fitness of the three A genotypes, AA, Aa, and aa, by $W_1(x)$, $W_2(x)$, and $W_3(x)$. Let these be the unmodified fitnesses at each position (x) in the deme series, that is, the fitness of A genotypes when each is also genotype bb. We will assume that the effects of the modifier allele B are dominant, and when present, its effects will be:

genotype		AA	Aa	aa
genotype frequency		p^2	$2pq$	q^2
B genotype	bb	$W_1(x)$	$W_2(x)$	$W_3(x)$
	BB,Bb	$W_1(x)+a$	$W_2(x)+b$	$W_3(x)+c$

where a, b, and c are positive, zero, or negative amounts by which the presence of allele B changes the fitness of each A genotype.

Assuming that allele B is initially present at low frequency, or appears during the course of study by mutation, under what conditions will the modifier (allele B) spread in a given area? Following Clarke (1966), B will spread if the mean fitness of all genotypes BB and Bb is greater than that of all genotypes bb, or if:

$$p^2(W_1(x)+a)+2pq(W_2(x)+b)+q^2(W_3(x)+c) > p^2W_1(x)+2pqW_2(x)+q^2W_3(x)$$

or, in terms of p;

$$p^2(a-2b+c) + p(2b-2c) + c > 0 \qquad (3\text{-}1).$$

Clearly, B will never increase in a given deme if a, b, and c, are all negative, and will always increase if a, b, and c are all positive. As B increases, the observed fitness of each genotype will change with time. This will happen wherever B spreads, including the conditions below.

If a and b are positive, but c is zero, (3-1) becomes:

$$p(a-2b) + 2b > 0 \qquad (3\text{-}2),$$

and B will increase in all demes where allele A is not lost ($p > 0$).

If a and b are positive, but c is negative, then (3-1) is only true if p is greater than some niminum value, or,

$$p > \frac{(b-c) - \sqrt{(b-a)^2 + a(2b-a-c)}}{(b-c) + (b-a)} \qquad (3\text{-}3),$$

following Clarke (1966). Thus B will spread only in areas where allele A is favoured over a, or is above a critical frequency. The location at which B ceases to spread depends upon the mode of modification (the values of a, b, and c), and the local gene frequency.

If a is positive, and both b and c are zero, then (3-1) becomes:

$$pa + 1 > 0 \qquad (3\text{-}4)$$

and B will always spread when A is present in the population.

If a is positive, and both b and c are negative, then (3-1) is only true if p is greater than some critical value, or,

$$p > \frac{(b-c) - \sqrt{(b-a)^2 + a(2b-a-c)}}{(b-c) + (b-a)} \qquad (3\text{-}5),$$

which is the same as (3-3); B will spread only in areas where a minimum gene frequency is exceeded.

If a is positive, b is zero, and c is negative, then (3-1) becomes:

$$p^2(a-c) + p(-2c) + c > 0 \qquad (3\text{-}6),$$

and B will spread when p is given by (3-5).

If a is positive, b is negative, and c positive, B may spread in some areas but not in others. If $(b^2-ac) < 0$, (3-1) will be true for all p, and B will spread throughout the cline. If, however, $(b^2-ac) > 0$, then the spread of B alleles depends upon p as follows. It will spread if

$$p > \frac{(b-c) - \sqrt{b^2-ac}}{2b-a-c} \qquad (3\text{-}6a)$$

$$p < \frac{(b-c) + \sqrt{b^2-ac}}{2b-a-c} \qquad (3\text{-}6b).$$

(Clarke, 1966). If b^2 is sufficiently greater than ac, then B will be absent at intermediate frequencies and present at the ends. The situation is exactly reversed if a and c are negative and b positive. (The latter situation is probably unrealistic.)

Thus there are basically two classes of modifiers, (I), those which have an advantageous or no effect on genotypes, and (II), those which have positive or zero effect on some genotypes, but deleterious effects on others. Type I is expected to be found throughout a cline

(except at initial stages), and type II will be found only in regions where the A genotypes upon which it confers an advantage are also the commoner type. Therefore, if one were to sample amongst a large number of demes with a variety of gene frequencies, and type II modifiers were present, we would expect a positive relationship between gene frequency and the observed fitness of the most common genotype, other fitness factors being equal.

We therefore can conclude that if type II modifiers are present in series of demes along a cline, differential coadaptation can take place; modifiers with different properties will spread in different areas, depending on the form of the cline. As Clarke (1966) has pointed out, the presence of modifiers can affect the slope and form of a cline, and form a morphotone at the critical gene frequency if the cline under modification is very different from the cline without modification.

In Clarke's (1966) example (a type C cline), $W_1(x)=y$, $W_2(x)=1$, and $W_3(x)=1-y$, where $y=kx$, k is a constant, and y varies from 0 to 1.0. From Fisher's (1930) formula, the equilibrium gene frequency as a function of y is simply

$$\hat{p}(y) = y \qquad \text{(unmodified)} \qquad (3\text{-}7),$$

and the slope of the corresponding cline is therefore k. In the regions of the cline where B is fixed, the fitnesses are now y+a, 1+b, and 1-y+c, for genotypes AA, Aa, and aa, respectively. Again, from the equilibrium formula:

$$\hat{p}(y) = \frac{y+b-a}{1+2b-a-c} \qquad \text{(modified)} \qquad (3\text{-}8).$$

The slope of the modified portion of the cline is now

$$\frac{k}{1+2b-a-c} \qquad (3\text{-}9).$$

If (3-7) and (3-8) are very different when (3-7) is very close to the critical gene frequency (3-1 through 3-6), then a morphotone will form in the location corresponding to the critical gene frequency. Using different functions for W_1, W_2, and W_3 (as in chapter 2), it can be shown that differential coadaptation can lead to morphotones in many different kinds of clines.

Let us now consider the effect of crossing individuals from demes that have differing modifier gene frequencies. Assume that the form of the gradients (W's, chapter 2) and the values of a, b, and c permit a cline to develop. The symbols a, b, and c, will now be used to represent the absolute values of the three coefficients of modification.

First, consider that differential coadaptation has taken place between the parents; let c be negative. Then the fitnesses will be:

genotype	AA	Aa	aa
a end of cline (bb)	W_1	W_2	W_3
A end of cline (BB)	W_1+a	W_2+b	W_3-c
F_1 between ends (Bb)	W_1+a	W_2+b	W_3-c
$F_2 = F_1 \times F_1$ (bb,	W_1	W_2	W_3
B-)	W_1+a	W_2+b	W_3-c

The mean fitness and variance of fitness of F_1 individuals will be indistinguishable from the parent in which allele B has become fixed. If B has not become fixed at (in this case) the A end of the cline, then it can be seen that the mean fitness of the F_1 would be

slightly less than the A end parent, and the variance greater. If B has become fixed at the A end, the mean fitness of the F_2 will become slightly lower than the A end parent and the F_1. If B is not fixed at the A end, then the mean and variance of both F_1 and F_2 will be about the same. Similar predictions may be made for other type II modifiers.

There is no reason why a second modifier, C, favouring allele a, should not occur and spread differentially. This is more similar to what Huxley (and others) had in mind when discussing the mechanisms of local adaptation. Let us assume that the second modifier, C, is also a type II modifier, and for simplicity, assume that the effects of C are the inverse of B, and the effects are additive. Other assumptions give similar results. The fitnesses are now:

genotype		AA	Aa	aa
unmodified		W_1	W_2	W_3
A end of cline	(BBcc)	W_1+a	W_2+b	W_3-c
a end of cline	(bbCC)	W_1-a	W_2+b	W_3+c
F_1	(BbCc)	W_1	W_2+2b	W_3
F_2	(bbcc,	W_1	W_2	W_3
	B-cc	W_1+a	W_2+b	W_3-c
	bbC-,	W_1-a	W_2+b	W_3+c
	B-C-)	W_1	W_2+2b	W_3

The overall mean fitness of the F_1 genotypes would now be equal to or greater than that of either parent. If the crosses were taken between demes with A gene frequencies at roughly equal but opposite

differences from 0.5, then the heterozygotes, Aa, would be most abundant in the F_1, and a definite "Hybrid vigour" would be observed. Depending upon the A genotype frequencies, the F_2 overall mean fitness will be equal to or slightly greater than either parental type, but the variance of fitness would be very much increased. The F_2 will be closer to the F_1 in both mean and variance if the modifiers are not fixed in the two demes crossed.

If b is negative the conclusions are very similar, but in some cases the mean fitness of the F_1 and sometimes the F_2 will be less than the parents. This is true even if coefficient c is also negative (for B-cc). The variances will be about the same as for b positive, after taking account of the dependance of V(p) on p.

In the initial stages of spread of a type I modifier, the modifier will be more common in the region where it confers an advantage of the most common genotype (the genotype selected for in that region). Thus if one made a cross across such a cline (or found natural hybrids), one would find the following:

genotypes		AA	Aa	aa
unmodified		W_1	W_2	W_3
A end of cline	(BBcc)	W_1+a	W_2+b	W_3
a end of cline	(bbCC)	W_1	W_2+b	W_3+c
F_1	(BbCc)	W_1+a	W_2+2b	W_3+c
F_2	(bbcc,	W_1	W_2	W_3
	B-cc,	W_1+a	W_2+b	W_2
	bbC-,	W_1	W_2+b	W_3+c
	B-C-)	W_1+a	W_2+2b	W_3+c

The mean fitness of the F_1 would in this case exhibit a definite "hybrid vigour," possibly with reduced variance compared to the parents. The heterotic effect would be large and the variance reduced if the frequency of Aa in the F_1 was high. The mean fitness of the F_2 would probably not be distinguishable from the parents, but the variance would be considerably increased.

In summary; type I modifiers, before their fixation in a cline, will produce "hybrid vigour" in the F_1 and "hybrid breakdown" in the F_2 (increased variance). Type II modifiers, at or near equilibrium, will show much less of an increase of fitness in the F_1 than type I, and will sometimes show a "hybrid inferiority" in the F_2. Type II, like type I, shows an increase in variance of fitness in the F_2.

It is important to note that these predictions hold even if it is not possible to score the fitnesses of each genotype separately.

These conclusions are not, of course, restricted to clines, but may apply to artificial crosses between any two natural populations. One would expect similar, if not greater effects if more than one modifier affected alleles A and a. The effect of more than one modifier can also be to increase the size of any morphotones produced (Clarke, 1966).

Section 3.7. Evidence for Coadaptation Along the Drosophila Clines

We thus have a good explanation for the first two observations. The slow increase of Bar gene frequency in the experiment could be explained by the spread of modifiers of the fitness of Bar phenotypes in the clines, and, or, the increase of modifier frequency in individual demes. It is possible that the reason for the absence of

increase in Bar gene frequency in the extra vials 6 in sets B and D are a result of either (1) the critical Bar gene frequency for Bar phenotype modifiers is higher than vial 6 Bar gene frequency; or, (2), modifiers arising in other vials could not reach vials 6 due to the lack of gene flow; or both.

If we accept that there are in fact two morphotones in both sets A and C, this can be explained if we assume that one or more modifiers decrease the fitness of heterozygotes, compared to their effect on the other genotypes. The constancy of gene frequency at which the apparent morphotones appear, 0.3 and 0.65, suggests the presence of modifiers; these could be the two critical gene frequencies in equation (3-6), or its equivalent for sex linkage.

The work of Moore (1946, 1949a, 1954) and the verbal arguments of Huxley (1939, 1942), Dobzhansky (1951), Maynard-Smith (1958), and Mayr (1963), suggest that there may be two additional factors contributing to changes in fitness of F_2 crosses between "differently coadapted" populations. If a large number of genes have been selected for a long enough time they may come to be very closely integrated. Any chromosomes from differently coadapted regions combining with a given local chromosome may be so disharmonious that the heterozygote does not function properly. This may be expressed in any number of ways including reduction of development rate, reduced viability, reduced fertility, and at the other extreme, sterility or lethality (see especially Moore's work). Any developmental problems in the F_1 heterozygote may be compounded with problems of crossing over if the coadapted groups are held together in inversions (see especially Dobzhansky, 1951, 1970). If the crossover products manage to survive meiosis, they may be subject to the same or worse kinds of developmental problems which may be found in the F_1 (again, see Moore).

In a natural cline, and in the experiments, if two parts of the cline have become differentially coadapted, then in the middle there will be natural F_1, F_1 s parent backcrosses, F_2, and F_2 backcrosses. We might, therefore, if differential coadaptation has taken place, expect to see an increase in developmental problems and an increase in deviations from 1:1 sex ratio in the middle of clines compared to the ends. The latter is to be expected because according to "Haldane's rule" (Maynard-Smith, 1958) in a cross between differently coadapted groups there should be a deficiency in the heterogametic sex as it is more sensitive to "unbalanced" gene complexes.

The sex ratio was calculated from the total number of emerging flies in each vial in each generation. The number of times which the sex ratio deviated significantly from 1:1 expected is found in table 3-1.

Using the data of table 3-1, and dividing each cline into two groups of approximately equal sizes, "ends" (vials 1-4 and 12-15 (8)), and "Middle" (vials 5-11 (7)), we expect, on the null hypothesis, a ratio of 8 "ends" to 7 "middle" instances of significant deviation from 1:1 sex ratio. The results are shown in table 3-2.

Almost all deviations from 1:1 sex ratio are due to female excess. All sets show an excess of significant deviations from 1:1 sex ratio in the middle compared to the ends except set B. Only set A is significant.

It is common knowledge among those who work with Drosophila that females emerge from their pupae earlier than males. This was substantiated in the present study. Since only the first six days of the emergence are counted (day 1 is defined as the day following the first day in which a minimum of 10 flies have emerged from

TABLE 3-1

Frequency of significant deviation from 1:1 sex ratio

(Measured by $X^2 > 3.84$)

Vial	Set A			Set B			Set C			Set D			Set E		
1	0	0	0	2	0	2	1	1	2	0	0	0	5	0	5
2	6	1	7	3	1	4	1	0	1	1	1	2	2	1	3
3	2	0	2	3	2	5	1	0	1	3	1	4	1	0	1
4	1	1	2	5	1	6	1	2	3	2	0	2	4	1	5
5	5	0	5	6	3	9	2	0	2	2	0	2	4	2	6
6	2	0	2	2	0	2	5	0	5	2	0	2	2	1	3
7	5	0	5	1	1	2	3	0	3	1	1	2	5	2	7
8	7	0	7	4	0	4	2	0	2	3	0	3	1	1	2
9	7	1	8	2	0	2	2	2	4	2	0	2	4	1	5
10	6	0	6	3	0	3	2	0	2	4	0	4	4	1	5
11	2	1	3	0	0	0	8	1	9	4	0	4	2	1	3
12	1	0	1	3	0	3	3	0	3	2	1	3	0	2	2
13	3	0	3	1	1	2	4	1	5	1	1	2	2	1	3
14	2	0	2	3	1	4	4	2	6	1	0	1	4	0	4
15	1	0	1	2	1	3	4	0	4	3	0	3	1	0	1

Note: Under each set, the first column is for instances of female excess, the second for male excess, and the third the total.

TABLE 3-2

Data from table 3-1 grouped by "ends" vs. "middle" of clines

Set	"Ends"	"Middle"	X^2	P
A	16	34	8.31	<.001
B	22	18	0.00	n.s.
C	19	24	1.10	n.s.
D	13	18	1.19	n.s.
E	19	22	0.81	n.s.

Calculated on frequencies of female excess only

at least one vial) it is possible that the female excess is at least partly due to an overall late emergence in the centre of the clines compared to the ends.

If there was coadaptive breakdown in the middle of the clines, then one would expect an increase of developmental problems and an increase in developmental time in the middle compared to the ends. In each vial, in each generation the total number of flies was counted each day. Next a "mean day emerging" was calculated by the formula:

$$\text{"Mean day emerging"} = \frac{\sum_{i=1}^{6} iN_i}{\sum_{i=1}^{6} N_i} \qquad (3\text{-}10)$$

where N_i is the number of flies emerging on day i. Table 3-3 shows the number of times each vial, in each set, was the latest of the set to emerge during that generation; the number of times that vial had the highest "mean day emerging."

TABLE 3-3

Frequency that a given vial in a given set is the slowest to emerge (as measured by "mean day emerging")

VIAL	SET A	SET B	SET C	SET D	SET E
1	1	3	0	1	3
2	1	3	2	5	2
3	1	1	2	3	4
4	2	2	2	0	3
5	1	1	5	2	2
6	2	1	6	3	2
7	4	1	0	1	3
8	4	1	3	3	2
9	3	6	3	6	0
10	4	5	2	2	1
11	3	1	4	1	2
12	1	2	0	2	2
13	1	1	2	2	1
14	2	3	3	1	3
15	4	3	0	1	3

TABLE 3-4

Data from table 3-3 grouped by "ends" vs. "middle" of clines

Set	"Ends"	"Middle"	X^2	P
A	13	21	2.54	n.s.
B	18	16	0.02	n.s.
C	11	23	5.20	<.02
D	15	18	0.55	n.s
E	21	12	1.02	n.s

X^2 in table 3-2 and 3-4 include Yates' correction

Table 3-4 compares the data in table 3-3 with respect to "ends" and "middle." In this case the "middle" vials of set C have, on the average, a longer development time compared to the ends. Set A also shows a delayed emergence in the middle, but it is not significant. Sets B and D have a very small X^2, and set E is also not significant.

Another possibility is to look for differences in fitness along the clines. The data each generation is in the form of numbers of parental genotypes (after artificial selection and gene flow) and numbers of offspring (the following generation before selection and gene flow). These were converted to frequencies such that male parents add up to one, female parents add up to one, and all offspring add up to one. Also, a sex ratio, females divided by total, was calculated on the offspring. The data will be represented by the following symbols:

Genotype	Parents	Offspring
Bar males (By)	A	A'
Wild males (by)	B	B'
Bar females (BB)	C	C'
Heterozygotes (Bb)	D	D'
Wild females (bb)	E	E'

In order to get expected zygote frequencies it is necessary to take into account that Bar males are at a disadvantage. Dr. David Noakes kindly studied the behavior of Bar and Wild flies from the clines by observation of single pair courtships. Bar males had a significantly higher courtship time (from the first chase to copulation), and a significantly larger number of breaks in courtship than do wild males. Female genotypes did not show any differences in mating parameters. In order to take account of Bar male mating deficiency when calculating expected offspring frequencies the following experiment was set up. Flies from vials 7, 10 through 15 were collected every 3 hours and stored separately for 3-4 days. Twelve replicates were set up, each with 25 By, 25 by, and 50 bb. These were allowed to mate under the same conditions as in the main experiment, and after 24 hours, females were placed into separate vials for egg laying. The offspring reveal which mating has taken place:

Replicate	1	2	3	4	5	6	7	8	9	10	11	12	Total
By X bb	17	15	11	20	16	14	11	7	8	21	18	13	171
by X bb	28	28	32	25	26	32	36	42	40	27	32	35	383
female dead	5	7	7	5	8	4	3	1	2	2	0	2	46
mating coeff (m)	.38	.34	.26	.44	.38	.30	.31	.14	.17	.43	.36	.27	.31

Except for crosses 8 and 9, the results were fairly uniform. The mean mating fitness of Bar males was 31%, in other words, Bar males fertilized about a third as many females as did wild males. Therefore the effective frequencies of male parents were not A and B, but,

$$\frac{mA}{mA + B} \quad \text{and} \quad \frac{B}{mA + B} \qquad (3\text{-}11),$$

where m is a coefficient of mating fitness. The expected zygote frequencies can then be calculated according to table 3-5.

Table 3-5

PARENT AND OFFSPRING (ZYGOTE) FREQUENCIES

Genotype	Parents	Effective Parents	Offspring Frequencies Expected		Observed
By	A	$\frac{mA}{mA+B}$	$(C + \frac{D}{2})/2$	=a	A'
by	B	$\frac{B}{mA+B}$	$(E + \frac{D}{2})/2$	=b	B'
BB	C	C	$\frac{mA}{2(mA+B)}(C + \frac{D}{2})$	=c	C'
Bb	D	D	$\frac{1}{2(mA+B)}[ma(E+\frac{D}{2})+B(C+\frac{D}{2})]$	=d	D'
bb	E	E	$\frac{B}{2(mA+B)}(E + \frac{D}{2})$	=e	E'

Note: A+B=1, C+D+E=1, a+b+c+d+e=1, and A'+B'+C'+D'+E'=1.

The observed fitnesses are then,

$$W_1(x) = A'/a \qquad W_3(x) = C'/c$$

$$W_4(x) = D'/d \qquad (3\text{-}12).$$

$$W_2(x) = B'/b \qquad W_5(x) = E'/e$$

It is possible that the mating success of Bar or normal males was related to their genotype frequencies such that rare males were at an advantage compared to the more common males. This has been found by Petit (1951, 1954, 1958) and Ehrman (1966, 1967, 1970, Ehrman and Petit,

1968) for Bar and other mutants in Drosophila. This has a plausible behavioural basis because females choose their mates after a complex courtship sequence. It is possible that the females may become "adapted out" to the courtship pattern of the more common male genotype (just as we may become accustomed to, and no longer notice, the noise of heating systems). If a rare type has a different courtship pattern, and begins to court a female, the situation may be equivalent to courtship in the absence of the common males, because they no longer provide courtship stimulus (Manning and Ewing, in conversations, 1972). Not allowing for this effect may result in a spurious frequency-related fitness in females (see table 3-5).

Although experiments were not attempted to test for frequency-dependent mating, it is possible to estimate m directly from the cline data. The following method is subject to many errors, but at least it should reveal any relationship between male frequency and male mating ability.

Assume that the fitness of normal males (by) and normal females (bb) are equal, or $W_2(x)=W_5(x)$. This is reasonable because the sex ratio in Kaduna was never found to be significantly different from 1:1. To help to eliminate random effects the expected numbers of male and female zygotes were taken to be (1-SR) and (SR) respectively, where SR, the fraction of females, is taken from the total number of emerging adults in the vial for which m is being calculated. (The variance of m was found to be slightly higher using SR=0.5). Taking fitnesses relative to normal males, we have from table 3-5 and equations (3-12):

$$
\begin{aligned}
A' &= W_a p(1-SR)(mA+B)/T \\
B' &= \quad q(1-SR)(mA+B)/T \\
C' &= W_c(SR)(mAp)/T \\
D' &= W_d(SR)(mAq+Bp)/T \\
E' &= \quad (SR)(Bq)/T
\end{aligned}
\qquad (3\text{-}13),
$$

where $p = (C + \frac{D}{2})$, $q = (1-p) = (E + \frac{D}{2})$, and T is a normalizing factor such that $A' + B' + C' + D' + E' = 1$.

Taking B' and E', we can now solve for m, which is:

$$
m = \frac{B[B'(SR) - E'(1-SR)]}{AE'(1-SR)} \qquad (3\text{-}14).
$$

(Note also, that solving for W_a using A' and B' gives the correct answer, $W_a = qA'/pB'$; in a sex-linked locus, male mating effects do not effect fitness estimates.)

The mating efficiency coefficient, m, was estimated by (3-14) for each deme in each set in each generation, where both male genotypes were present. There was no evidence for any relationship between m and A, and the mean value of m in each set was rather similar to m estimated in the mating tests, when m was calculated from the cline data (table 3-6).

Observed fitnesses were calculated for each genotype in each vial in each generation, using equations (3-12), with an arbitrary constant mating value of m = 0.31. The mean fitnesses (± 1 S.E.) in each deme, for generations 20 through 35, are shown in figure 3-7. It is apparant that a genotype's fitness is a function of both its position (deme number) and its frequency. In most genotypes, fitness rises sharply in the parts of the artificial selection gradient where the genotype is increasingly selected against, and also where it is very rare. Fitness remains roughly constant and near unity where a given

Table 3 - 6

ESTIMATED MATING COEFFICIENT, m, AND REGRESSION OF m ON A

	SET A	SET B	SET C	SET D	SET E
			Generations 1 - 19		
Mean	0.303	0.456	0.303	0.506	0.500
S.E.	0.034	0.048	0.044	0.077	0.053
N (number of demes)	200	201	232	193	249
$m = K_1 + K_2A = K_3A^2$					
K_1	-0.128	0.466	-0.508	0.420	0.536
K_2	0.918	-0.001	2.124	0.077	-0.183
K_3	-0.841	-0.104	-1.916	0.193	0.101
Significantly different from zero? F(2,N-2)	1.94	0.23	7.00***	0.09	0.06
			Generations 20 - 35		
Mean	0.260	0.280	0.242	0.317	1.241
S.E.	0.032	0.043	0.026	0.041	0.065
N	178	140	173	134	228
K_1	0.111	0.690	0.460	-0.437	2.357
K_2	0.317	-0.721	-0.497	1.325	-23.751
K_3	-0.177	0.543	0.527	-1.117	98.924
F(2,N-2)	1.20	1.20	1.50	2.95	17.39***

*** Significant at P < 0.001

genotype is common and favoured. In sets A and C the minimum observed mean fitness was not at the bar end of the gradient (vials 12 - 15), but in the middle, and fitness appears to increase with increasing as well as decreasing selection and frequency. This was not observed in the earlier generations (0 to 19). The sharp negative frequency-relationship was also not as distinct in the early generations.

To attempt to separate the position-dependent effects from the

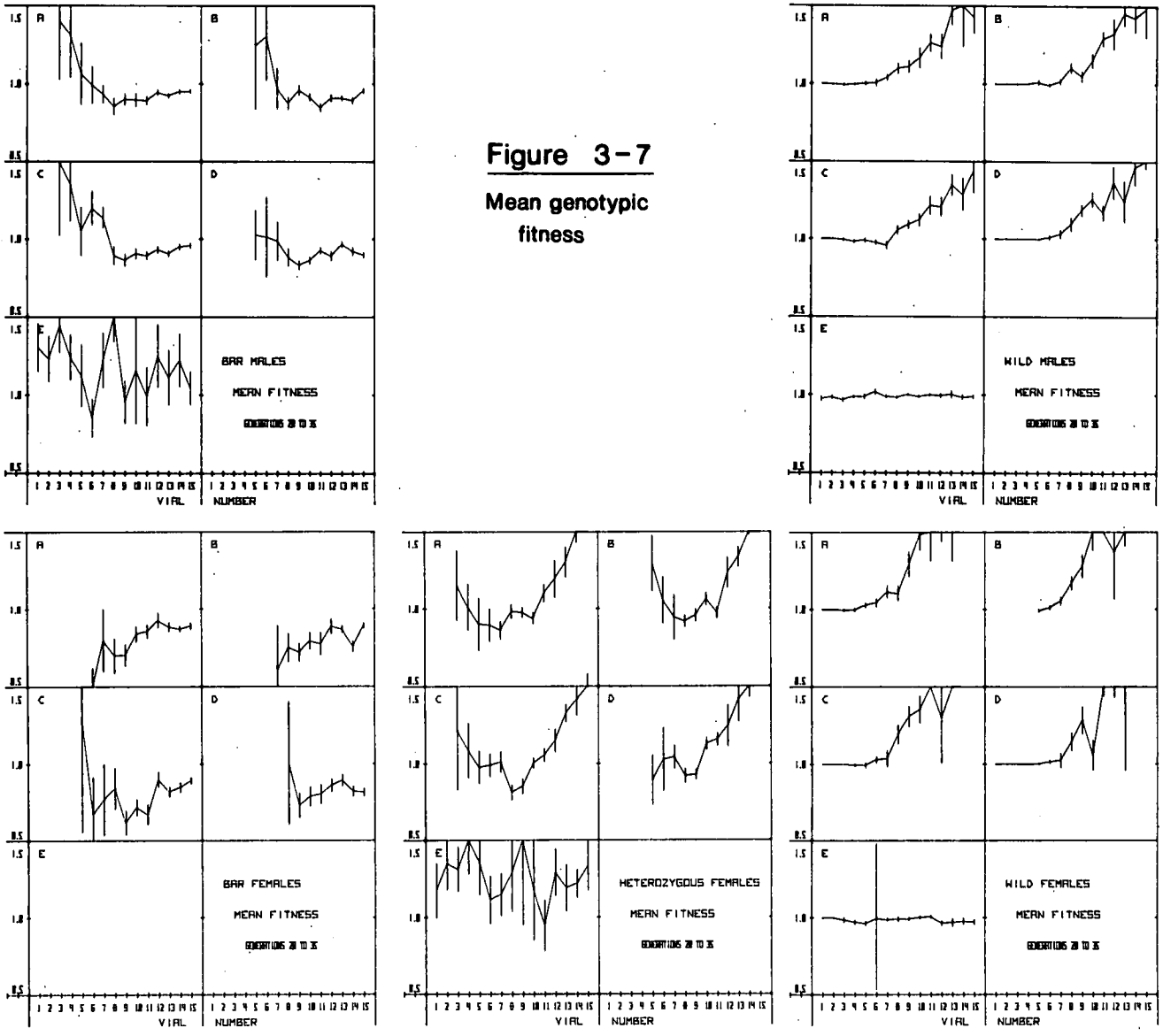


Figure 3-7

Mean genotypic fitness

frequency-dependent effects, curvilinear regression of fitness on expected zygote frequency (m = 0.31) was carried out. Table 3-7 gives the regression lines for each genotype in the later generations (20-35) and figure 3-8 illustrates the frequency-dependent relationship.

Table 3-7

EQUATIONS RELATING FITNESS AND EXPECTED ZYGOTE FREQUENCY (m = 0.31)

Equations are of the form $W = K_1 + aK_2 + a^2K_3$

		SET A	SET B	SET C	SET D	SET E
By	K_1	1.669*	1.679*	1.704*	1.381*	1.633
	K_2	-3.559	-2.989	-3.581	-1.873	-14.632
	K_3	6.275	4.812	6.016	3.475	95.027
by	K_1	2.031*	1.989*	1.942*	1.854*	1.857
	K_2	-3.524	-3.130	-3.071	-2.657	-1.901
	K_3	4.416	3.639	3.754	2.908	2.015
BB	K_1	0.489*	0.881	0.910	0.941	29.642
	K_2	1.590	-0.372	-0.879	-0.498	-5058.
	K_3	-2.109	1.133	2.391	0.972	34736.
Bb	K_1	1.368	1.772	1.368	1.451	2.858*
	K_2	-1.728	-3.783	-1.581	-1.210	-37.529
	K_3	0.617	3.821	-0.474	-3.938	8.889
bb	K_1	2.634*	3.184*	3.587*	3.099*	0.266
	K_2	-5.696	-7.801	-9.264	-7.446	1.550
	K_3	7.224	10.193	12.213	10.195	-1.709

* Asterisk means overall regression is significant at P = 0.05 or better.

Data from generations 20 - 35

The addition of the quadratic term to the regression is not especially helpful in describing the frequency-dependent relationship; considering the variation in fitness among vials and between generations, a simple linear regression is an accurate enough measure. Table 3-8 gives the linear regression equations for both the early and late generations.

Table 3-8

LINEAR EQUATIONS RELATING FITNESS AND ZYGOTE FREQUENCY ($W=K_1 + aK_2$)

Generations 0 - 19

		SET A	SET B	SET C	SET D	SET E
By	K_1	0.970	0.860	1.097	0.732	0.928
	K_2	-0.421	-0.051	-0.857	0.410	-0.389
by	K_1	1.521	1.507	1.436	1.430	1.213
	K_2	-1.196	-1.144	-0.977	-0.928	-0.445
BB	K_1	0.961	0.959	0.842	0.975	1.361
	K_2	-0.920	-0.833	-0.334	-0.596	-4.684
Bb	K_1	0.984	1.139	1.212	1.119	1.037
	K_2	0.026	-0.557	-0.950	-0.428	-0.167
bb	K_1	2.685	2.039	2.282	1.693	0.920
	K_2	-3.747	-2.435	-3.023	-1.546	0.109

Generations 20 - 35

By	K_1	1.164	1.143	1.232	0.991	1.374
	K_2	-0.696	-0.658	-0.911	-0.244	-5.691
by	K_1	1.456	1.451	1.385	1.437	1.027
	K_2	-1.053	-0.986	-0.907	-0.945	-0.079
BB	K_1	0.587	0.694	0.657	0.765	3.4
	K_2	0.742	0.347	0.439	0.162	-30
Bb	K_1	1.345	1.620	1.379	1.626	1.584
	K_2	-1.524	-2.599	-1.691	-0.245	-7.433
bb	K_1	1.950	2.126	2.307	1.931	1.017
	K_2	-2.086	-2.311	-2.953	-2.002	-0.069

Underlined figures are significantly different from zero slope at P = 0.05 or better.

Figure 3-9 illustrates the development of the negative frequency-dependent effect in time. For the first five or six generations the slope (K_2) is positive (and usually insignificant), later it becomes negative, and remains at about the same mean level for the rest of the experiment, though subject to much fluctuation.

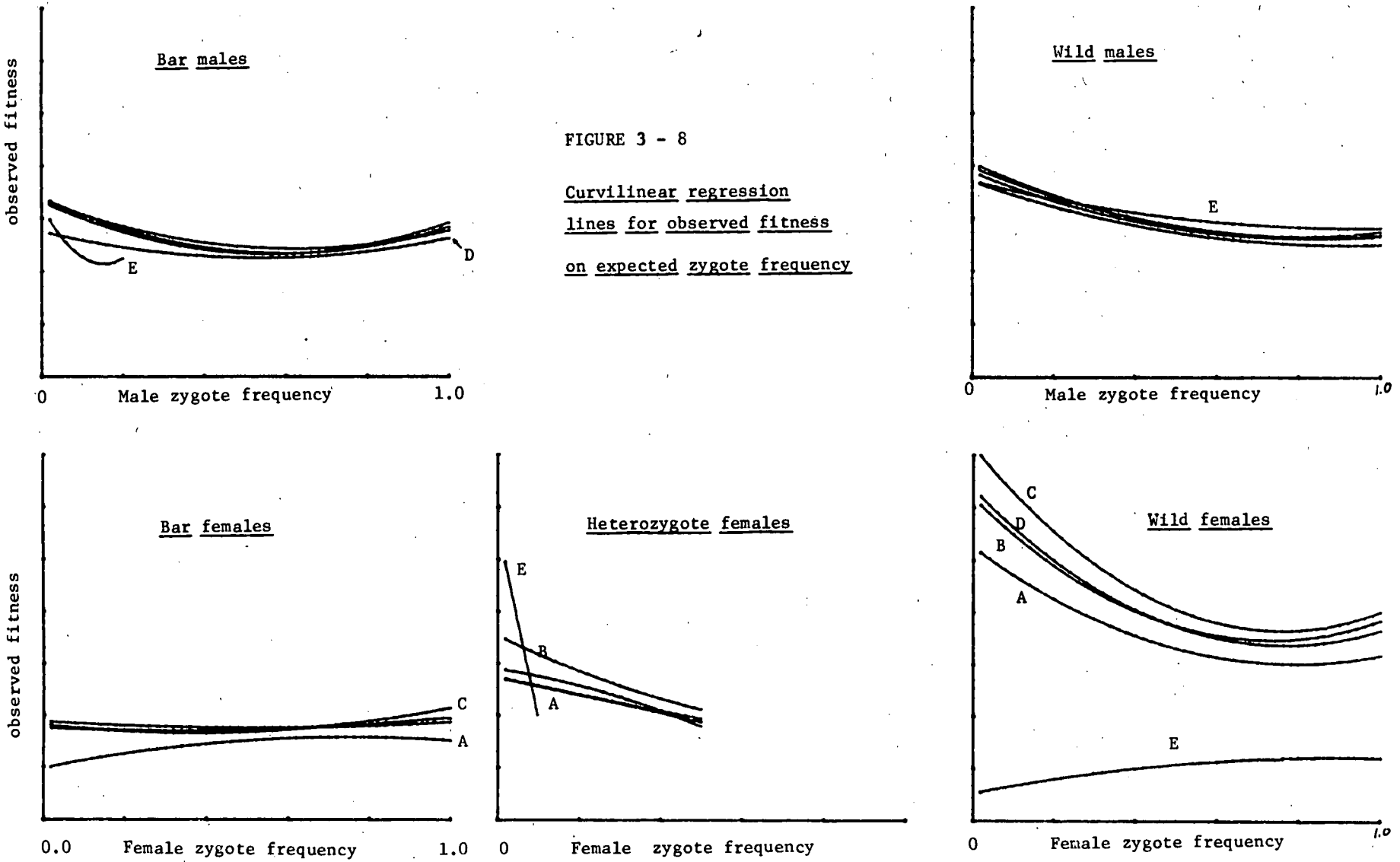


FIGURE 3 - 8

Curvilinear regression lines for observed fitness on expected zygote frequency

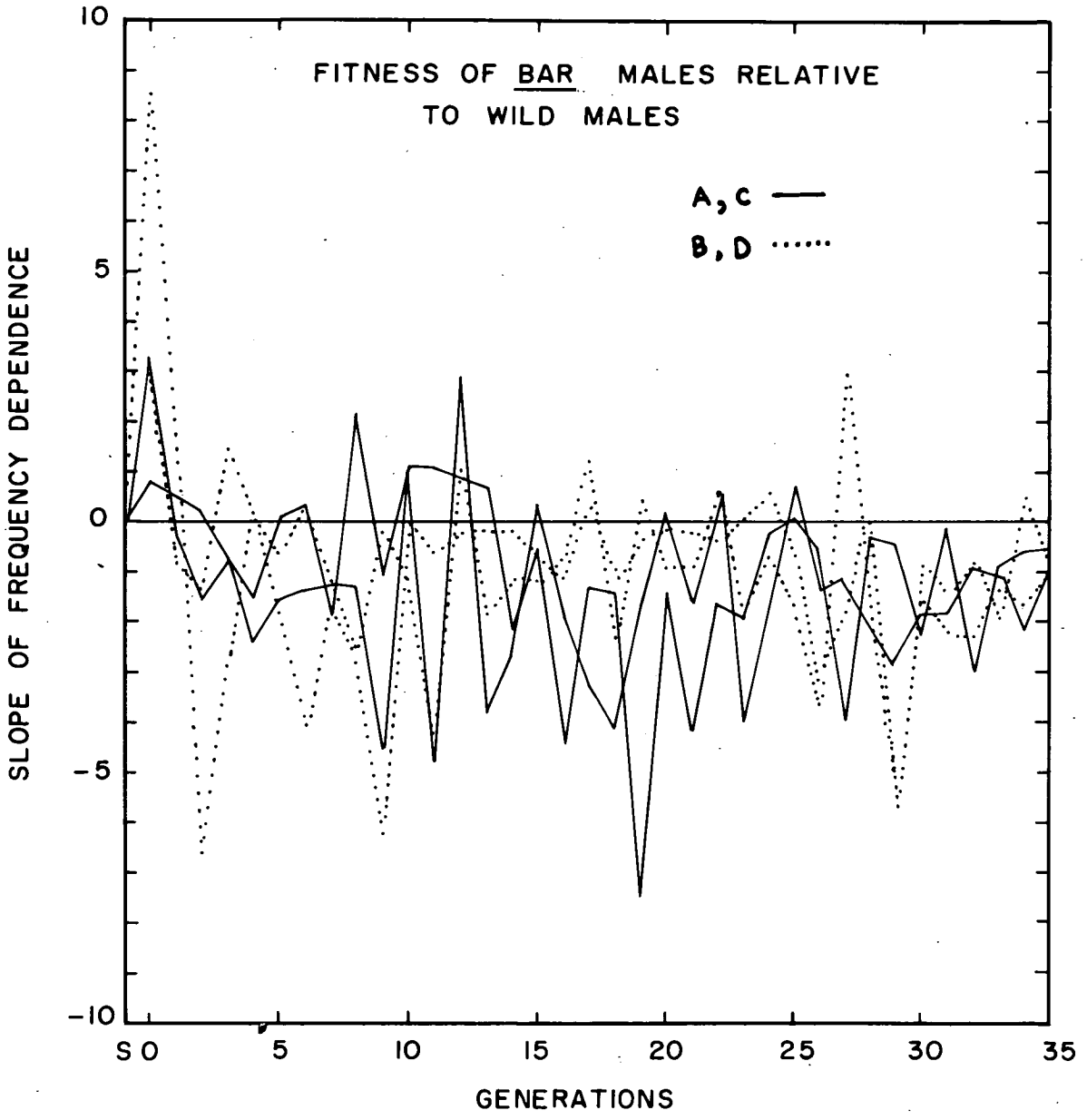


FIGURE 3 - 9. The development of frequency-dependence

There does not seem to be any facile explanation for the negative frequency-dependent effect. That it is found in males as well as in females suggests that it is not a result of a male mating effect. It might be a female mating effect (table 3-5). If rare females were inseminated sooner than the more common ones they might lay eggs earliest, and their offspring might then be subject to less intense competition for food, and hence be subject to less "natural selection" than the bulk of the common genotype larvae. I know of no evidence for earlier mating of rare females, however.

The negative frequency-dependence may also be a result of a Bar "niche" and a Wild "niche" in each deme. If some fraction of the resources used by wild flies cannot be utilized by Bar flies and vice versa, a rare type would have plenty of food, and would do well, whilst the common type would be subject to intense competition. Again this is highly speculative.

The fact that the effect develops in time implies that it is not an artifact of measurement. Whatever the reason for the negative frequency-dependent selection, it is difficult to explain on the basis of one-locus theory.

It would be interesting to know whether there is any position-dependent relationship between expected zygote frequency and fitness. One possible way to examine this is to plot the deviations from expected fitness (expected on the basis of frequency-dependence) at each position in the clines. For brevity I will call these fitness deviations. The prediction equations in table 3-8 were used to calculate fitness deviations for all vials in all sets, in each generation according to:

fitness deviation = Fd_i = (observed fitness) - (expected from table 3-8)

$$\begin{aligned} Fd_1(x) &= A'/a - (aS_1 + I_1) \\ Fd_2(x) &= B'/b - (bS_2 + I_2) \\ Fd_3(x) &= C'/c - (cS_3 + I_3) \\ Fd_4(x) &= D'/d - (dS_4 + I_4) \\ Fd_5(x) &= E'/e - (eS_5 + I_5) \end{aligned} \qquad (3\text{-}13)$$

Where A' and a are given by (3-12), and S_i is the slope and I_i the intercept of the frequency-dependent relationship given in table 3-8, for the i'th genotype.

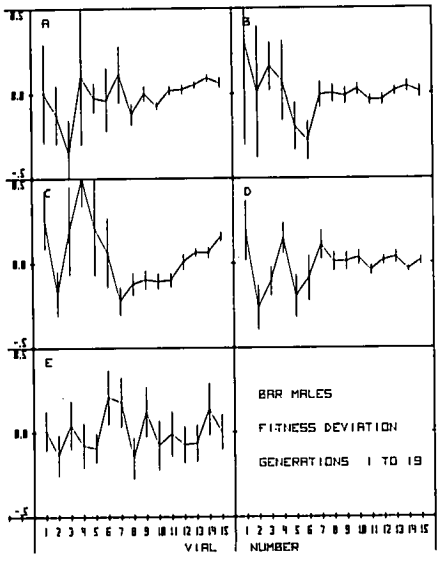
Figure 3-10 shows the fitness deviation for each genotype in each set, averaged for generations 0-19, and 20-35, separately. A positive fitness deviation indicates that more of that genotype survived than expected on the basis of the overall frequency-dependent relationship.

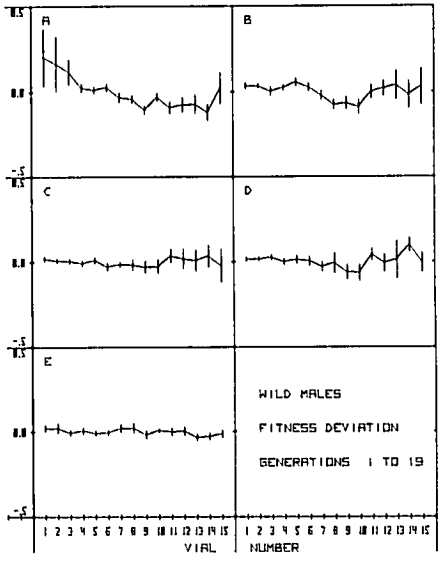
Bar males, Wild males, and Wild females show a distinct reduction in Fd in the centre vials in the later generations, particularly in sets A and C. There is no sign of a consistent effect in the early generations. These same genotypes show an increase in fitness deviation in the end vials in the later generations. Heterozygotes show a distinct position-dependent change in fitness; positive in the Bar region and negative in the wild regions of the clines. Only in females does the effect seem to be the same in replicates with gene flow and without gene flow; in the males the effect appears to be greater in the replicates with gene flow. The effects in females appear to be of greater age than in males.

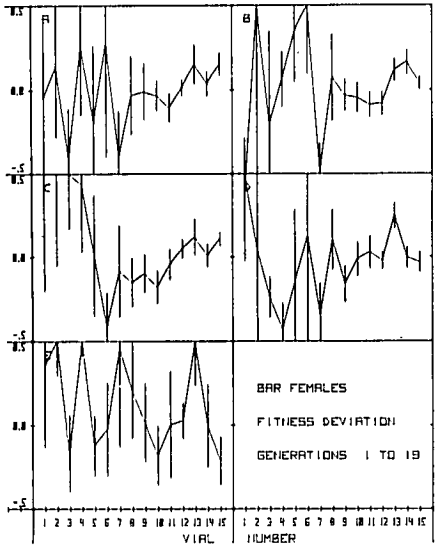
The results are strongly suggestive of the presence of coadaptive modifiers, differentially distributed along the clines. The fact that the effects are greater in the later generations rules

FIGURE

3–10a


A
B
C
D
E
BAR MALES
FITNESS DEVIATION
GENERATIONS 1 TO 19
VIAL
NUMBER


A
B
C
D
E
WILD MALES
FITNESS DEVIATION
GENERATIONS 1 TO 19
VIAL
NUMBER


A
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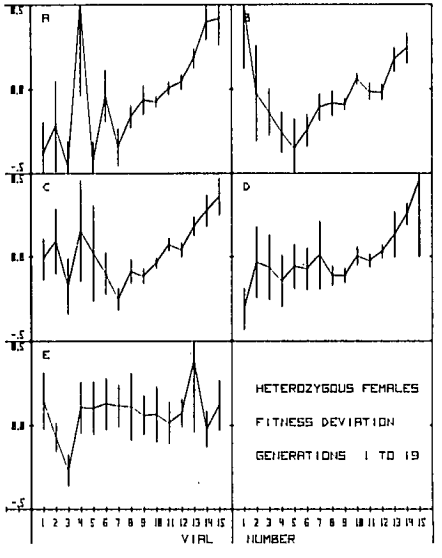
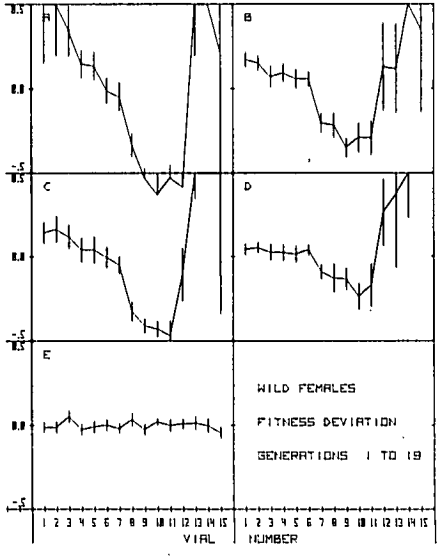
C
D
E
BAR FEMALES
FITNESS DEVIATION
GENERATIONS 1 TO 19
VIAL
NUMBER


A
B
C
D
E
HETEROZYGOUS FEMALES
FITNESS DEVIATION
GENERATIONS 1 TO 19
VIAL
NUMBER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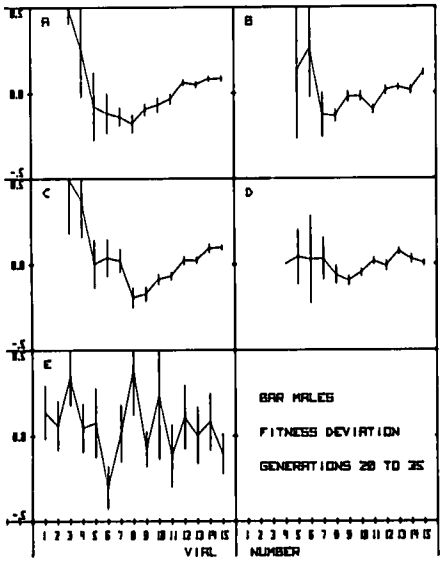
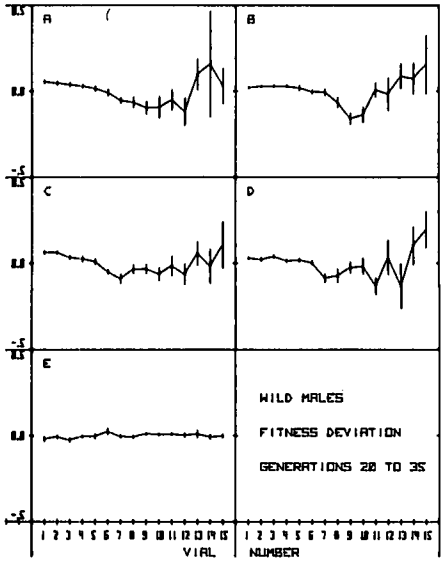
C
D
E
WILD FEMALES
FITNESS DEVIATION
GENERATIONS 1 TO 19
VIAL
NUMBER


A
B
C
D
E
BAR MALES
FITNESS DEVIATION
GENERATIONS 20 TO 35
VIAL NUMBER

FIGURE

3—10 b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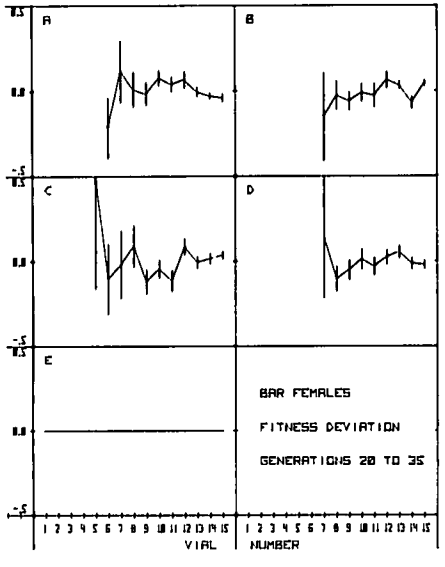
FITNESS DEVIATION
GENERATIONS 20 TO 35
VIAL NUMBER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B
C
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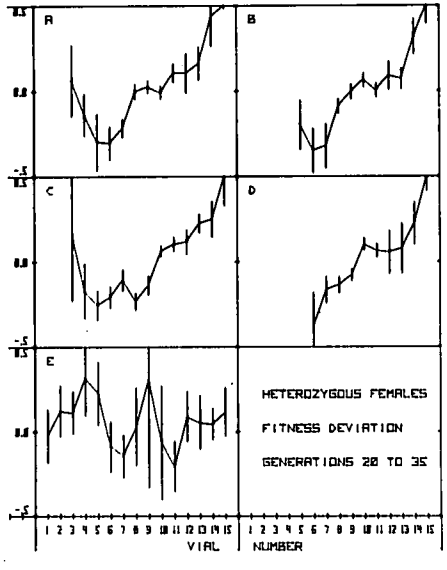
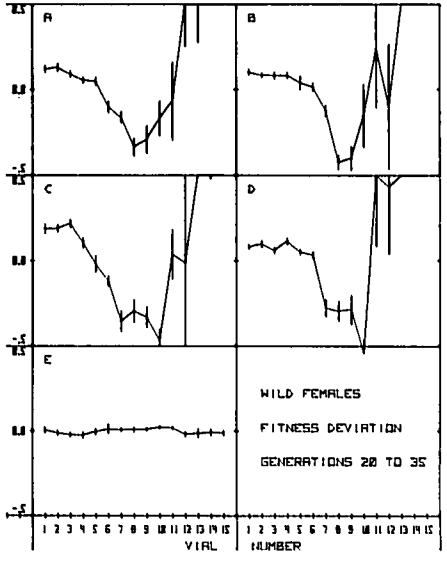
E
BAR FEMALES
FITNESS DEVIATION
GENERATIONS 20 TO 35
VIAL NUMBER


A
B
C
D
E
HETEROZYGOUS FEMALES
FITNESS DEVIATION
GENERATIONS 20 TO 35
VIAL NUMBER


A
B
C
D
E
WILD FEMALES
FITNESS DEVIATION
GENERATIONS 20 TO 35
VIAL NUMBER

out the possibility of artifactual results. However, there is one point worth mentioning. Wherever a particular genotype becomes rare, its expected genotype numbers become less than one. If the "real" fitness of a genotype were 0.5, and the expected number of zygotes was 0.5, on the average 0.25 flies would emerge. Fitnesses are calculated on each total emergence. Therefore if one fly came out the observed fitness would be 2, and if no flies came out, the observed fitness would be zero. If two flies came out the observed fitness would be 4. As this could lead to a spurious negative frequency-dependence, all fitnesses calculated on expected genotype numbers less than 0.9 were excluded from all calculations. The calculations and figure 3-10 take account of this potential problem, yet the mean fitness deviation is still very high when the given genotype is rare. We must therefore conclude that the negative relationship between expected zygote frequency and observed fitness is nonlinear, and increases with increasing extremes in zygote frequency.

If we ignore the vials with low genotype frequency we see that fitness deviations are largest where the genotype frequency is highest, and that heterozygotes have a highest Fd at the Bar end of the clines. This is what one would expect if type II modifiers have spread in the clines.

There does not appear to be any effect of gene flow; in males there is a suggestion that the effect may be stronger in the gene flow replicates (A and C) than in the others. Again, this is expected since coadaptive modifiers could spread from deme to deme.

If coadaptive modifiers have spread between vials (sets A and C), we would expect a higher correlation between adjacent vials of fitness deviation in the sets with gene flow (A and C) than the sets

with selection but no gene flow (B and D). Table 3-9 shows these correlation coefficients.

Table 3-9

Correlation coefficients of fitness deviation between adjacent vials r's are underlined if significant at 0.05 or better.

Genotype	SET A	SET B	SET C	SET D	SET E
		Generations 0 - 19			
By	0.130	0.175	0.406	0.092	0.204
by	0.035	0.256	0.273	0.095	0.170
BB	0.317	0.049	0.191	0.178	0.177
Bb	0.555	0.267	0.389	0.268	0.144
bb	0.576	0.185	0.356	0.647	0.345
		Generations 20 - 35			
By	0.567	0.140	0.475	0.326	-0.100
by	0.150	0.117	0.349	0.176	0.096
BB	0.357	0.203	0.272	0.252	0.000
Bb	0.350	0.260	0.391	0.222	-0.075
bb	0.278	0.377	0.098	-0.069	0.246

The selective sets with gene flow (A,C) in the latter generations do indeed have a higher correlation among adjacent vials than those without. The effect is greatest for the non-wild genotypes. This is what one would expect if only wild-coadapting modifiers were present in the original Kaduna population.

An experiment was set up specifically to test for the possibility of coadaptation. Virgin flies were collected every 3 hours from the clines at generation 33. For each set the following procedure was adopted. Flies were taken from vials 4, 7, 11, and 14. Thus four points were chosen symmetrically about the p=0.50 location (vial 9) far apart (4 and 14) and close together. These will be referred to as the "far" and "near" vials, respectively. The following crosses were then set up:

"far" X "far"	4 X 4 14 X 14	parents
	4 X 14 14 X 4	F_1
"near" X "near"	7 X 7 11 X 11	parents
	7 X 11 11 X 7	F_1

Each cross (8 per set) consisted of 50 males and 50 females at approximately the same genotype frequencies of the vials they came from. These were assorted on the basis of random mating expectation and placed together in single pairs to mate for 48 hours. The flies were then collected together and placed in egg laying chambers without ether. Egg laying chambers were patterned after David Briscoe's; they consisted of a plastic beaker with a hole opened in the bottom, plugged with cotton-wool, and upended over a 5 cm. plastic petri dish filled with yeasted Edinburgh Drosophila medium. Eggs were collected at two 24-hour intervals, simply by replacing the petri dish at the bottom while attracting the flies to light at the other end of the beaker. No escapes were made during this process. From each cross four batches of 200 egs (two days of egg collecting) were placed by means of a flattened needle onto the medium in the larvae vials. The larvae vials were weighed before and after putting in fresh medium, and each contained 5.00±0.05 gm. of medium. All were stoppered with weighed plugs of cotton-wool.

As in the main experiment flies were collected for six days. In addition to the usual scoring of genotypes and counting the numbers that emerged each day, the vials were weighed before and after the flies were removed. Thus data is available on the biomass as well as the total numbers emerging from each replicate of each cross. Virgins of the F_1 and parental crosses were crossed to themselves to form the F_2 by the same method.

FIGURE 3 - 11

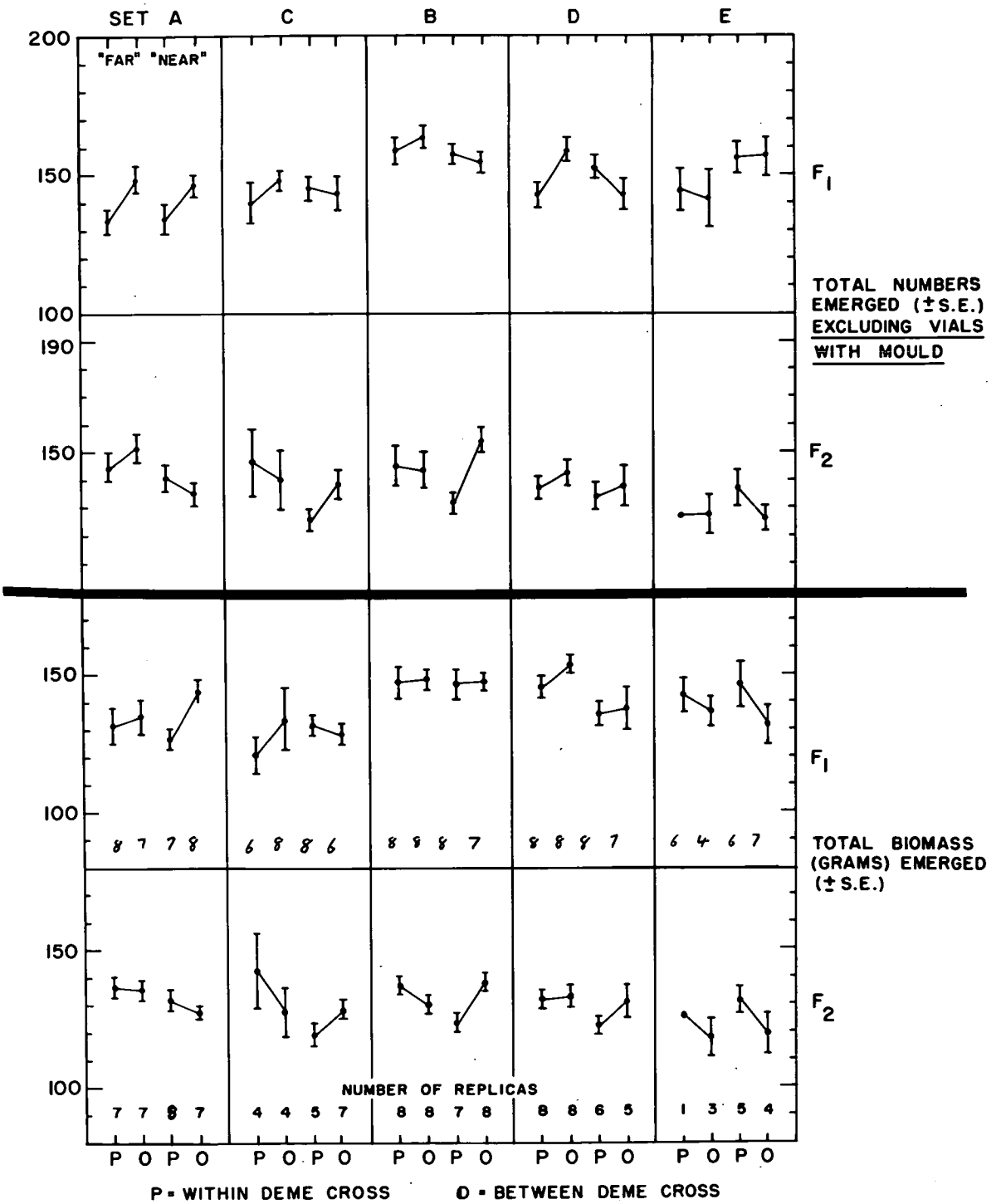


FIGURE 3 - 12

Figures 3-11, 3-12 show the result of each cross for numbers and for biomass. The data may be found in appendix 4.

Figure 3-11 shows the numbers emerging. In all the "far" crosses (except for those of Set E) the mean number emerging in the F_1 is greater than the parents raised under the same conditions. This is not true for the "near" crosses. There is no consistent result for either "far" or "near" in the F_2.

Figure 3-12 shows the biomass emerging. Again, in all the "far" crosses from replicates with artificial selection (A - D) there is a consistent increase in biomass emerged in the F_1 compared to the parents. This is true for the "near" crosses in A, B, and D, but not in C. The F_2 is lower than the parents in sets A, C, and B, but not D, in the "far". The opposite is true for the "near". Set E resembles the other sets in its lack of concordance.

In summary, the following suggest the presence of coadaptive modifiers in the experimental Drosophila clines:

1. Increase in Bar gene frequency after initial loss in sets A and C.
2. Gaps and constancy of gene frequency at which they appear.
3. Set A shows a significant excess of females in the centre of the clines compared to the ends.
4. Set C (and possibly set A) shows a significant delay in emergence in its centre compared to its ends.
5. Development of frequency-dependence betwee fitness and zygote frequency in time.
6. Position-dependent fitness and deviations in fitness, of the sort predicted by theory.
7. Correlation between adjacent vials of adjusted fitness is higher in gene flow replicates than in replicates without gene flow.

8. Cross cline crosses, when made far enough apart on the clines, produce more flies than parents under controlled conditions (F_1, "far").

9. Cross cline crosses, when made far enough apart, F_1 produce a greater weight of flies than parents or F_2 under controlled conditions.

Section 3.8. Conclusion for Chapter 3

The most obvious result of all experiments is that the effect of 40% gene flow is difficult to detect. Differentiation with respect to Bar and probably several other loci proceeded rapidly with little regard to whether the demes exchanged 40% of their members each generation or not. The results are consistent with the presence of both type I (points 1, 7, 8, 9) and type II (2 - 9) modifiers.

Chapter 4

Parapatric Speciation

> ".. with the fuller knowledge of the facts of variation we now possess, I think it may be shown that natural selection is, in some probable cases at all events, able to accumulate variations in infertility between incipient species." A. R. Wallace, (1898, Ch. 7).

The model with coadaptive modifiers presented in the last part of chapter 3 has some interesting consequences with respect to speciation mechanisms.

In the areas between differentially coadapted regions, populations will contain various combinations of genotypes with no, one, and two (or more) coadaptive modifiers. Under certain conditions this leads directly to "hybrid breakdown" (chapter 3). Any additional effects of unbalanced gene complexes will decrease the fitness of the more heterozygous individuals relative to the parental types (for example, Moore, 1949a).

As suggested by Wallace (1889), Dobzansky (1937, 1940) and Mayr (1940, 1942, 1955, 1963) any postmating disadvantage of hybrids will favour genes that promote assortative mating; assortative mating avoids the "wastage" of reproductive energy on ill-adapted genotypes.

Consider, as in chapters 2 and 3, a single locus A with two alleles A and a. The genotypes AA, Aa, and aa have the usual fitnesses, W_1, W_2, and W_3. Let there be another, independent, locus, D, with two alleles D and d. Let the presence of allele D in one of the A genotypes cause positive assortative mating with respect to A

genotypes, and random mating with respect to all other loci. Following O'Donald (1960) let the presence of allele D in an A genotype cause assortative mating with a frequency m and random mating with a frequency (1-m). Thus if D were fixed in a given population, and AA mates assortatively with AA, Aa with Aa, and aa with aa, the genotype frequencies of the zygotes of the next generation (D', H', and R') would be:

$$\begin{aligned} &AA: && D' = p^2 (1-m) + m(p-H/4) \\ &Aa: && H' = 2pq(1-m) + m(H/2) \\ &aa: && R' = q^2 (1-m) + m(q-H/2) \end{aligned} \qquad (4\text{-}1)$$

where p is the gene frequency of allele A, q = (1-p), and H is the heterozygote frequency of the parents (O'Donald, 1960).

More generally:

$$\begin{aligned} D' &= p^2(1-m) + mf_1 \\ H' &= 2pq(1-m) + mf_2 \\ R' &= q^2(1-m) + mf_3 \end{aligned} \qquad (4\text{-}2)$$

where f_1, f_2, and f_3 are determined by the type of assortative mating.

When D is segregating in a population with a gene frequency i, and j=(1-i), let the three D genotypes (DD, Dd, and dd) have the frequencies U, V, and Y. Let the effects of D be dominat. The mating frequencies will then be as in table 4-1, and the offspring frequencies will be as follows:

	AA	Aa	aa	Total
DD	i^2D'	i^2H'	i^2R'	i^2
Dd	$i(j+V/2)D'+iYp^2$	$i(j+V/2)H'+iY2pq$	$i(j+V/2)R'+iYq^2$	2ij
dd	$j(V/2)D' + jYp^2$	$j(V/2)H' + jY2pq$	$j(V/2)R' + jYq^2$	j^2

ASSUMING THAT MATING FOLLOWS (4-1).

If selection is only with respect to the A locus, and genotypes AA, Aa, and aa have the fitnesses W_1, W_2, and W_3, then it can be shown that D will spread to fixation if:

$$mY(pq-H/4)\ [W_1+W_3-2W_2] > 0 \qquad (4-3)$$

or simply,

$$[W_1+W_3-2W_2] > 0 \qquad (4-4)$$

Similar results may be found with other systems of positive assortative mating. This is exactly what is expected; if heterozygotes are at a disadvantage compared to homozygotes, then any genes causing a reduction of heterozygote frequency by changes in mating frequencies will be favoured over other genes not causing positive assortative mating.

Can differential coadaptation lead to a combination of A locus fitnesses which fulfill condition (4-4)?

Consider the model of chapter 3 with two modifier loci B and C. We will now add locus D and find the conditions for parapatric speciation. Let allele B have a gene frequency r, and b have a gene frequency s=(1-r). Let allele C have a gene frequency t, and c have a gene frequency u=(1-t). Let all four loci segregate independently and all but A mate at random. Then, as in chapter 3, let the fitnesses of the A genotypes be as in table 4-2.

TABLE 4 - 1. PARENTS' MATING FREQUENCIES, ASSORTATIVE MATING GENE (D) SEGREGATING

Mating type		AA by AA	AA by Aa	Aa by Aa	AA by aa	Aa by aa	aa by aa
DD by DD	U^2	U^2K_1	U^2K_2	U^2K_3	U^2K_4	U^2K_5	U^2K_6
DD by Dd	2UV	$2UVK_1$	$2UVK_2$	$2UVK_3$	$2UVK_4$	$2UVK_5$	$2UVK_6$
Dd by Dd	V^2	V^2K_1	V^2K^2	V^2K_3	V^2K_4	V^2K_5	V^2K_6
DD by dd	2UY	$UY[K_1+D^2]$	$UY[K_2+2DH]$	$UY[K_3+H^2]$	$UY[K_4+2DR]$	$UY[K_5+2HR]$	$UY[K_6+R^2]$
Dd by dd	2VY	$VY[K_1+D^2]$	$VY[K_2+2DH]$	$VY[K_3+H^2]$	$VY[K_4+2DR]$	$VY[K_5+2HR]$	$VY[K_6+R^2]$
dd by dd	y^2	Y^2D^2	$Y^2 2DH$	Y^2H^2	$Y^2 2DR$	$Y^2 2HR$	Y^2R^2

$K_1 = D^2(1-m)+mD$

$K_2 = 2DH(1-m)$

$K_3 = H^2(1-m)+mH$

$K_4 = 2DR(1-m)$

$K_5 = 2HR(1-m)$

$K_6 = R^2(1-m)+mR$

Genotype Frequencies

AA: D
Aa: H
aa: R

DD: U
Dd: V
dd: Y

Loci A and D not linked.

TABLE 4-2 — Mean fitnesses of modified A genotypes

Modifier	AA	Aa	aa	frequency
bbcc	W_1	W_2	W_3	s^2u^2
Bbcc, BBcc	W_1+a	W_2+b	W_3+c	$2rsu^2$, r^2u^2
bbCc, bbCC	W_1+d	W_2+e	W_3+f	$s^2 2tu$, s^2t^2
BbCc, BBCc, BbCC, BBCC	$W_1+k_1(a+d)$	$W_2+k_2(b+e)$	$W_3+k_3(c+f)$	$2rs2tu$, $r^2 2tu$, $2rst^2$, r^2t^2

From table 4-2 the overall fitness of each of the A genotypes is:

$$W_1 = W_1 + a(1-s^2)[u^2+k_1(1-u^2)]+d(1-u^2)[s^2+k_1(1-s^2)]$$

$$W_2 = W_2 + b(1-s^2)[u^2+k_2(1-u^2)]+e(1-u^2)[s^2+k_2(1-s^2)]$$

$$W_3 = W_3 + c(1-s^2)[u^2+k_3(1-u^2)]+f(1-u^2)[s^2+k_3(1-s^2)] \qquad (4-5)$$

From (4-3) and (4-4) a gene (D) causing assortative mating with respect to locus A will spread if:

$$[W_1+W_3-2W_2]+(1-s^2)[u^2(a\_c-2b)+(1-u^2)(ak_1+ck_3-2bk_2)]+$$

$$(1-u^2)[s^2(d+f-2e)+(1-s^2)(dk_1+fk_3-2ek_2)]> 0 \qquad (4-6)$$

Or, more simply, if $k_1=k_2=k_3=1$, then (4-5) becomes:

$$[W_1+W_3-2W_2]+(1-s^2)[a+c-2b]+(1-u^2)[d+f-2e]> 0 \qquad (4-7)$$

There are therefore a large number of conditions which favour the spread of assortative mating genes in a cline affected by modifiers. Modifiers which are especially favourable for parapatric speciation include those which have no or negative effects on heterozygotes (b, and, or, e zero or negative), and modifier genes which interact with other such genes such that k_2 is small or zero. The restrictions of (4-6) are somewhat relaxed if the unbalanced gene complexes of hybrids are subject to developmental or physiological problems; this reduces W_2.

We may find a stable hybrid zone (such as in Heliconius erato, Turner, 1971, or the Hooded and Carrion Crows, Meise, 1928) if the modifiers permit a cline with a morphotone to develop, but do not fulfill equation (4-6). It is therefore not possible to predict the outcome of secondary contact; even if there has been marked divergence in the two types, if they can still interbreed when they meet after isolation, whether or not they fuse depends upon the interaction of the major and minor components of fitness.

If only type I modifiers (chapter 3) are present, assortative mating genes can still spread; (4-6) is then the condition for sympatric speciation because, then a, b, c, d, e, and f are all positive, type modifiers will spread throughout the cline. This is analogous to Maynard-Smith's (1966) conditions, but with more than two environments. White (1968, White, et al., 1967) discussed one form of "stasipatric" speciation in which a new chromosomal rearrangement appears in a local patch within the species zone, and is partially or wholly sterile with the other chromosome arrangements. It then is supposed to spread (assuming it is not lost by chance or selection) through all or part of the existing species range. Since it is isolated from the rest of the species by the inviable hybrids a new species has formed. If we substitute type I modifier genes for chromosome arrangements in the

argument, and if the modifiers fulfill (4-6), then the process is more likely, although it will not be as rapid. If all modifier coefficients are positive (type I), however, there are fewer combinations of modifiers which fulfill (4-6) compared to type II modifiers. Therefore parapatric speciation is more likely than sympatric or "stasipatric" speciation.

As discussed in chapter 2, the effect of a partial barrier is to enhance the effect of differentiation along a cline. This applies to minor (modifier) genes as well as to major genes. The effect of a barrier would not be to change (4-6), but it might increase the rate of the process by allowing two zones to become even more sharply differentiated than they might be in its absence. As postulated by Mayr (1963) and others increased isolation may allow greater differentiation of the entire genome, reducing W_2 for any inter group crosses. We thus might expect to see more species pairs coming together in mutually inferior habitats than in more constant salubrious environments. However, this would result from any of the other forms of geographic speciation; population size in a contact zone cannot be used as a clue to what speciation process has occurred.

Summary of chapter 4.

The presence of coadaptive modifiers in a cline can lead to the evolution of assortative mating if the modifiers result in a net deficiency of major gene heterozygote fitness. Depending upon the type of modifiers present, either parapatric or sympatric speciation is possible, according to condition (4-6). Parapatric speciation is more likely than sympatric speciation because the restrictions of (4-6)

are not as severe for type II modifiers. Restriction of gene flow (partial barriers) does not lead directly to speciation, but may accelerate the process of parapatric speciation.

Chapter 5

Concluding Remarks

> "Any success in solving the problems in the present study is due partly to firsthand knowledge of the complex topographical and ecological situations along with extensive collecting at critical localities."
> Sibley (1950, p. 109).

The questions posed in chapter 1 were,

1. Can sharp geographic differentiation occur across a spatially and genetically continuous series of populations?
   a. Why do morphotones often not correspond sharp spatial environmental changes? Do morphotones require ecotones?
   b. Does gene flow really prevent spatial differentiation?
2. Can clines give rise to "hybrid zones"?

Chapter 2 has dealt with question 1. Morphotones can form in a cline through a continuous series of demes connected by gene flow. Morphotones, or spatially sharp changes in gene and genotype frequency, need not form at the same geographical position as ecotones; in fact ecotones are not required for morphotones to evolve. Gene flow is not nearly as strong a dedifferentiating factor as has often been supposed. For gene flow to have much of an effect selection must be extremely weak, and mean gene dispersal distance very large. Geographic differentiation can be strong with respect to one locus responding adaptively to a selection gradient even though there may be a continual and uninterrupted flow of genes among the component demes. If more than one locus responds directly to the same selection gradient, then several loci may become sharply differentiated geograhically.

Chapter 3 provided further evidence that the effect of gene flow can be very small or immeasureable, and suggested a mechanism for the formation of "hybrid zones" in clines.

Coadaptive modifiers which increase the fitness of a given genotype (or phenotype) will spread only where that genotype is common if the same modifier has a deleterious effect when present in one or more of the other genotypes. Different modifiers may have different effects, hence, different modifiers will spread in different parts of a cline, leading to differential coadaptation. The results of the experiments suggest that at least the initial stages of differential coadaptation have evolved a "hybrid zone" from a smooth cline, as outlined in figure 1-2 (chapter 1). Whether a cline is smooth, steep, has or has not a morphotone, and exhibits great variability of intermediates, depends upon the selective forces and the action of the modifier genes, not necessarily simply on the history of population structure.

Chapter 4 briefly illustrates how the effect of coadaptive modifiers on a cline may lead to speciation, or to some level of assortative mating. Given a morphotone or even a "hybrid zone" (a zone of increased variability of fitness, see chapter 1) assortative mating may or may not evolve. It is quite possible for a "hybrid zone" to be stable but not evolve sexual isolating mechanisms, as in the hooded and carrion crows, as long as the effects of fitness modifiers do not follow condition (4-6). If the right modifiers are present parapatric speciation may result. If other kinds of modifiers are present, or appear in a cline with a "hybrid zone", then it may decay into a smooth cline. For the same reason, experimental interpopulation crosses may (e.g. Vetukiv, 1953) or may not (e.g. McFarqhar and Robertson, 1963) show hybrid vigour or hybrid breakdown.

We therefore find that figure 1-2 (chapter 1) has a firm genetic basis, and that all four modes of differentiation and speciation are possible. The least restrictive conditions for speciation are those for allopatric speciation, the most restrictive for sympatric, with allo-parapatric and parapatric speciation falling in between. Thus the three forms of geographic speciation would be expected to be more common in nature than sympatric. To distinguish among the former modes, a thorough understanding of natural clinal phenomena is necessary.

The interpretation of clinal phenomena in natural populations is difficult because they require many parameters to be estimated. There are many possible spatial patterns of selection and gene flow that can produce a given cline structure; the actual geography of natural selection and gene flow must be worked out before an attempt to explain a natural cline in terms of a model or conceptual framework (Endler, 1973, and see chapter 2). In order to explain a cline, especially a cline with one or more morphotones, the following kinds of data are required:

1. The geography of population structure must be known.
   a. The neighborhood size, or number of breeding individuals per deme must be estimated.
   b. Mean gene flow rate per deme per generation must be measured.
   c. The distance among demes must be measured.
   d. Any systematic biases in gene flow direction must be measured.

2. An accurate description of the cline must be made.
   a. The genetics of the clinal character must be known or worked out.
   b. The genotype and gene frequencies at each deme must be calculated at each deme; distance between sample points or quadrat size should be of the order of the neighborhood diameter.

3. Selection coefficients should be measured at as many sample points as possible, using a dependable measure such as the genotype frequencies of very young juveniles to breeding adults.

4. Data on development time, sex ratio , etc. (measures of coadaptive breakdown) should be gathered for as many sample points as possible.

5. Replicated crosses between and within different parts of the cline should be made to test for differential coadaptation.

6. Environmental measurements should be made at as many sample points as possible since microclimatic differences may be large over short distances (chapter 2).

7. The relationship between an environmental gradient and selection should be worked out or known.

For requirements 1 and 2, causa scientiae patet; one should know what one is trying to explain, see also chapter 2, and Bradshaw (1962). Probably the best way of obtaining samples is to first measure dispersal distances and estimate the neighborhood area, then take samples along transects at intervals corresponding roughly to the neighborhood diameter. If each sample area is too large then the form of the cline will be obscured (Bradshaw, 1962, Pielou, 1969), and if smaller than the neighborhood area the inter-sample variation may be large and the cline form also obscured. See Pielou (1969) and Southwood (1966) for a further discussion of the effects of differing quadrat size in sampling spatial phenomena.

Requirements 3 through 6 follow from the results of chapters 2 and 3. Unless one has a detailed map of selection coefficients for each genotype it will be impossible to give a unique explanation for the observed cline; several patterns of selection can result in the same cline shape. If the gene flow distance is not known it is even more difficult; a given cline shape can result from one of many selective models, and for any given selective model it will not be known whether the cline results from a given degree of selection and gene flow, weaker gene flow and stronger selection, or vice versa.

Requirements 1 through 3 are needed to explain a cline by a one-

locus model. If and only if no one-locus model will fit the cline, then data from requirements 4 and 5 can be used to test whether or not differential coadaptation has taken place. In order to prevent the coadaptive hypothesis from becoming a "dustbin" explanation one should not invoke it unless one-locus models provide a poor fit to the data.

Requirement 6 can be used to enquire whether or not any environmental variable is associated with the clines. For example Hewitt and Ruscoe (1971) found a temperature gradient associated with clines in B-chromosomes in the grasshopper Myrmeleotettix maculatus. Requirement 7 is used to relate the environmental gradient to fitness. As mentioned in chapter 2, Birch (1945) worked out in detail the percent mortality in Calandra oryzae and Rhizopertha dominica beetles as a function of temperature and amount of available moisture. If one then measures an environmental gradient in both these factors one should be able to predict the clines. If Birch's data had been on two genotypes of a given species a fairly precise prediction of clines would be possible. The relationships do in fact work for predicting which species is found in a given set of conditions. It is necessary to emphasize that there is no reason why there should be a linear relationship between a fitness gradient and an environmental gradient (see chapter 2); one may be able to measure fitness, but not environment in the same cline, and one may be able to find a relationship between one genotype and the environment, but the error in estimation may be too large to detect a relationship in the other genotypes, see chapter 2.

If any of the required data are not gathered it will be impossible to provide a unique biological explanation of a cline, unless it is associated with an unusual historical, physiographic, or climatic

situation. Most of the published studies of clines suffer from this problem, as can be seen in table 5-1.

As an example, consider Bishop's work on the cline in melanism in the moth Biston betularia (Bishop and Harper, 1970, Bishop, 1972). This is the most complete study of a cline which has come to my attention, yet there is not enough data to give one explanation (table 5-1). Requirements 1 to 3 have been partially met, although his sample points are too far apart. He estimated that 95% of the moths move less than 5 km. in 24 hours, yet his sample points are much more widely spread. Selection due to visual predation was estimated at several localities, and found to be roughly linear with distance from Liverpool. However, as he says, there are other forms of selection than visual predation, and his estimates are incomplete.

Carbonaria is a dominant, so as far as visual selection is concerned, considering his measured fitness measurements, the cline should follow model A. However, the cline is not as strongly S-shaped. From his fitness values we can see that the null point in (visual) selection should fall between Eastham Ferry and Howarden. The models (chapter 2) predict that the gene frequency of a dominant gene should be between 40 and 50% in the vicinity of a null point in selection. However the observed cline goes through 40-50% between Loggerheads and Llanbear, many kilometres southwest of Howarden. From chapter 2 there are three possible explanations. There could be a deleterious effect of typica at some stage of the life cycle which is regardless of locality, and which is not found in carbonaria; perhaps typica is more sensitive to the effects of general insecticide or other pollutant levels—this position independent selection would shift the cline away from Liverpool as is observed (see chapter 2). The absolute survival value gradients (which can be calculated from Bishop's data) have gentle slopes; only a small deleterious effect of

TABLE 5 - 1. PUBLISHED CLINE STUDIES AND POSSIBLE EXPLANATIONS

Legend: 1-7: data requirements (chapter 5) Rg.:restricted gene flow in vicinity of morphotone
A-I: cline models (chapter 2) Ect.:one or more ecotone model (A'-I') explanations
Cd.: coadaptation cline model possible (chapter 3)
X : data collected, or possible explanation. I: data incomplete or a crude estimate
P : possible, but no evidence in paper for reduction in N or g in vicinity or morphotone

			DATA											POSSIBLE EXPLANATIONS											
	SPECIES	REFERENCES	1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd.
Misc.	Misc. animals	Remington (1968)	I	I	I	I	I	W	I	I		I	I	X	X	X	X	X	X	X	X	X	X	X	X
Ectoprocta	Schizoporella errata	Schopf and Gooch, 1971, Gooch and Schopf, 1971					X	W				W			X	X	X	X	X	X			X	X	X
Isopod	Jaera albifrons	Bocquet, 1969					I	W	I	I	I	I	I	P	X	X	X	X	X	X	X	X	X	X	X
Insects	Misc. insects	Thorpe, 1930,'31 Hubbell, 1956 Sammeta & Levins 1970	I	I	I	I	I	W				I		X	X	X	X	X	X	X	X	X	X	X	X
Hemiptera	Philaeneus spumarius	Halkka, 1962-1964 Halkka and Mikkola,1965, Halkka et al., 1966-1967	X	X	I		X	W			I	X		P										X	X
Orthoptera	Myrmeleotettix maculatus	John and Hewitt, 1965, Hewitt & John, 1967, 1970 Hewitt & Ruscoe, 1971, Hewitt & Brown, 1970	X	X	X	X	X	X				X		P	X	X	X	X	X	X			X		X
	"Morabra" scurra group	White et al., 1964, 1967, White, 1968	I	I			X	W						X	X	X	X	X	X	X	X	X	X	X	X

Lepidoptera

SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
		1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd.
Arphia conspersa	Willey & Willey, 1967, '71	I	I	I		X	W				X		X	X	X	X	X	X	X			X	X	X
Acraea Johnsoni	Hale Carpenter, 1932					X	W	I			I	I	P	X	X	X	X	X	X			X	X	X
Argynis callippe	Hovanitz, 1943					X	W				X			X	X	X	X	X	X	X	X	X	X	X
Amathes glareosa	Kettlewell, 1961, Kettlewell & Berry, 1961, 1969,	I		I	X	I	W	I		I	I		P	X	X	X	X	X	X			X	X	X
Biston betularia	Clarke & Sheppard, 1966, Bishop & Harper, 1970, Bishop, 1971	X	X	X	I	X	W	I			X	I	P	X	X	X	X	X	X			X		X
Coenonympha tullia	Ford, 1954						W				X		P	X	X	X	X	X	X	X	X	X	X	X
Colias spp.	Hovanitz, 1944a, 1944b, 1953, Watt, 1968	X			I	X	W				I	I		X	X	X	X	X	X			X	X	X
Euphydryas editha	Ehrlich, 1961, 1965, Labine, 1964, 1966, Ehrlich & Mason, 1966	X	X	X	X	I	X	I			X		P	X	X	X	X	X	X			X	X	X
Heliconius spp.	Turner, 1971	I		I		I	W	I	I	I	I			X	X		X	X	X	X	X	X		X
Limenitis spp.	Platt & Brower, 1968	I				I	W	I	I		X		P	X	X		X	X	X	X	X	X		X
Lymantria dispar	Goldschmidt, 1934						W	I	I	X	I		P	X	X	X	X	X	X	X	X	X	X	X
Maniola jurtina	Ford, 1964, 1971, Dowdswell & Ford, 1953, Creed, et al., 1971	X	I	I			X	I			I		P	X	X		X	X	X	X	X	X		X
Oeneis chryxus	Hovanitz, 1940	I					W	I			X		X	X	X	X	X	X	X	X	X	X	X	X
Papilio glaucus	Brower & Brower, 1962	I	I			I	W	I			I		P	X	X	X	X	X	X	X	X	X	X	X

	SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
			1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd
Coleptera	Papilio dardanus	Ford, 1964, 1971, Clarke & Sheppard, 1962					X	W			X	X	X	P	X	X	X	X	X	X	X	X	X	X	X
	Phigalia pedarina	Lees, 1971						W				X			X	X	X	X	X	X	X	X	X	X	X
	Tisiphone abeona	Lucas, 1969	I				X	W				X		X	X	X		X	X	X			X	X	X
	Zygaena ephialates	Bovey, 1941					I	W				I		X	X	X	X	X	X	X	X	X	X	X	X
	Harmonia axyrides	Komai, 1954, Komai et al., 1950, Komai & Hosino, 1951					X	W	I			X		X	X	X	X	X	X	X	X	X	X	X	X
	Harmonia axyrides, Adonia varigatus, Coccinella quinquipunctata	Dobzhansky, 1933					X	W	I			I		X	X	X	X	X	X	X	X	X	X	X	X
	Adalia bipunctata	Creed, 1971						W				X	I		X	X	X	X	X	X	X	X	X	X	X
	Carabus mollis	Lindroth, 1949, 1953, Den Boer, 1971, Haeck, 1971	I	I	I	X	X	W				X		P	X	X	X	X	X	X	X	X	X		X
	Bembidon gropei																								
Hymenoptera	Apis mellifera	Alpatov, 1929						W				X		X	X	X	X	X	X	X	X	X	X	X	X
	Cynips group	Kinsey, 1937, Goldschmidt, 1937						W				I			X	X	X	X	X	X	X	X	X	X	X
	Hoplitis albifrons	Michener, 1957						W				I		P										X	X

	SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
			1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd
Diptera	Drosophila pseudoobscura	Dobzhansky, 1951, 1970, 1933, 1947, 1948, 1969	X	X	X		X	W	I		X	I		X	X	X	X	X	X	X			X	X	X
	Drosophila robusta	Stalker & Carson, 1947, 1948	I				X	W				I		P										X	X
	Drosophila persimilis	Spiess, 1950					X	W	I		X	X			X	X	X	X	X	X			X	X	X
Mollusca	Amadora trapezia	O'Gower & Nicol, 1968					X	W				X		P	X	X	X	X	X	X			X	X	X
	Acmaea spp.	Test, 1946	I		I			W				I		P	X	X	X	X	X	X	X	X	X	X	X
	Cepaea nemoralis	Wolda, 1969a,b	X	I	I	I	X	X				X		P	X	X		X	X	X			X		X
	Cepaea nemoralis Cepaea hortensis	Cain, 1971, Carter, 1968, Arnold, 1968 Cain & Currey, 1963a, c, 1968, Currey & Cain, 1968 Day & Dowdswell, 1968, Goodhart, 1963 Clarke & Murray, 1962	X	X	X	X	X	X			I	I		P	X	X	X	X	X	X			X		X
	Cepaea vindobinensis	Jones, 1972	X	X	X		X	X	I		I	I			X	X		X	X	X			X	X	X
	Nucella spp.	Clench, 1954						W				X			X	X	X	X	X	X	X	X	X	X	X
	Partula	Clarke, 1968, Clarke & Murray, 1969, 1971	X	X	X		X	X		X	X			P	X	X		X	X	X	X	X	X	X	X
Pisces	Anoplarchus purpurescens	Johnson, 1971					X	W				X	I	P	X	X	X	X	X	X	X	X	X	X	X
	Catastomus clarkii	Koehn, 1969					X	W				X	I	P	X	X	X	X	X	X	X	X	X	X	X
	Cod, Herring, Plaice	Frydenberg, et al., 1965, I.C.E.S., 1969					X	W			I	I		P	X	X	X	X	X	X	X	X	X	X	X

	SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
			1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd.
Urodela	Gasterosteus aculeatus	Heuts, 1947	X			X	X	W				X		P	X	X		X	X	X	X	X	X	X	X
	Gasterosteus aculeatus	Hagen, 1967, Miller & Hubbs, 1969, Hagen & McPhail, 1970	X	X	X	X	X	X	I		I	X		X	X	X		X	X	X			X		X
	Notropis spp.	Gilbert, 1961, Hubbs, 1943	I				I	W		X	X	I		X	X	X	X	X	X	X	X	X	X		X
	Pimephalis promelas	Merritt, 1972					X	W				X	X	P	X	X	X	X	X	X	X	X	X	X	X
	Desmognathus spp.	Dunn, 1943	X		X			W	I			X			X	X	X	X	X	X	X	X	X		X
	Plethodon, spp.	Hairston, 1950, Hairston & Pope, 1948, Highton, 1962,1971, Jaeger, 1971, Williams et al., 1968	X	I	I	I	I	W	I	I		X		X	X	X		X	X	X	X	X	X	X	X
Anura	Bufo regularis	Low, 1968						W		X	X	I		P	X	X	X	X	X	X	X	X	X	X	X
	Crinia, spp.	Littlejohn et al., 1971, Moore, 1954	I	I	I			W	I					P	X	X		X	X	X	X	X	X		X
	Pseudophryne spp.	Woodruff, 1972	X					W?	X	X	X	X			X	X		X	X	X	X	X	X		X
	Rana pipiens complex	Moore, 1944-1957	I	I	I	I	X	W	I	X	X	X	I		X	X	X	X	X	X	X	X	X	X	X
Reptilia	Gerrhonotus spp.	Fitch, 1938	I					W				X		X	X	X	X	X	X	X	X	X	X		X
	Thamnophis spp.	Fitch, 1941, Fox, 1951						X				X		X	X	X	X	X	X	X	X	X	X		X
Aves	Anser coerulescens	Seiger & Dixon, 1969	I	X	I	I	X	W							X	X	X	X	X	X	X	X	X	X	X
	Aphelocoma spp.	Pitelka, 1951	I					W				I			X	X	X	X	X	X	X	X	X		X
	Colaptes, spp.	Short, 1965, Johnson, 1965	X	I	I			I							X	X	X	X	X	X	X	X	X	X	X

SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
		1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd
Coryus spp.	Meise, 1928, Mayr, 1942, 1963	I					W		I	X	I			X	X		X	X	X	X	X	X		X
Dicruridae	Mayr & Vaurie, 1948	I					W				X		X	X	X	X	X	X	X	X	X	X	X	X
Junco, spp.	Miller, 1931	I	I				W				X			X	X	X	X	X	X	X	X	X		X
Melospiza melodia	Marshall, 1948a,b, Johnson, 1956a,b, Miller, 1947, Ferrell, 1966	X	X	X	X		X				X	I	P	X	X	X	X	X	X			X		X
Oenanthe spp.	Mayr & Stresseman, 1950						W				I		P	X	X	X	X	X	X	X	X	X	X	X
Motacilla flava	Sammalisto 1956, 1957, 1958	X	I	I	I	I	W	I			X		X	X	X	X	X	X	X			X	X	X
Paradisaea apoda	Mayr, 1942, 1963, 1940						W	I					X	X	X	X	X	X	X	X	X	X	X	X
Parus spp.	Huxley, 1939a, Kluijver, 1951, Mayr, 1963, Snow, 1954						I				X		X	X	X	X	X	X	X	X	X	X	X	X
Pachycephala pectoralis	Galbraith, 1956						W	I			I		X	X	X	X	X	X	X	X	X	X	X	X
Passer domesticus	Jones, 1970	I					W	I			X		X	X	X	X	X	XX	X			X	X	X
Passerella iliaca	Swarth, 1920	I		I			W				X		X	X	X	X	X	X	X	X	X	X		X
Passerella lincolni	Miller & McCabe, 1935	I	I	I			W				I		P	X	X	X	X	X	X	X	X	X		X
Pipilio eurythropthalamus	Sibley, 1950	I	I	I	I	I	W				X		X	X	X	X	X	X	X	X	X	X	X	X
Quiscalus quiscala	Huntington, 1952	I	I	I			W	I	I		X			X	X	X	X	X	X	X	X	X		X
Tipisphone spp.	Chapin, 1948	I					W				I			X	X	X	X	X	X	X	X	X	X	X

Mammalia

SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
		1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd.
Uria aalge	Southern, 1939, 1951, 1962, 1966, Southern & Reeve, 1941, Southern et al., Storer, 1952	X	I	I	I	I	X	I			X		X	X	X		X	X	X			X		X
Zonotrichia spp.	Miller, 1949	I					W				X		P	X	X		X	X	X	X	X	X	X	X
Bubalus caffus	Christy, 1929	I	I	I			I				X			X	X	X	X	X	X	X	X	X	X	X
Cricetus cricetus	Gershenson, 1945a, b	I	I	I	I		W	I			I		X	X	X	X	X	X	X	X	X	X		X
Heliosciurus spp.	Ingoldby, 1927						I				I		X	X	X	X	X	X	X	X	X	X	X	X
Homo sapiens	McCullough & Giles, 1970	I				X	I				X	I	P	X	X	X	X	X	X			X	X	X
Homo sapiens	Livingstone, 1969	X	X	X	X	X	X	I	I	I	I		X	X	X	X	X	X	X			X	X	X
Mus musculus	Ursin, 1952	X	X	X			W				X		X	X	X	X	X	X	X	X	X	X		X
Mus musculus	Selander, 1970, Levin, et al., 1969, Lewontin, 1962, Lewontin & Dunn, 1960, Lidicker, 1972, Petras, 1967, Reimer & Petras, 1967, Anderson, et al., 1964, Anderson, 1964, Crowcroft & Rowe, 1963	X	X	X	I	X	X	X			I		P	X	X	X	X	X	X	X	X	X	X	X
Neotoma albigula	Blair, 1954							I			X		P	X	X							X	X	X
Peromyscus spp.	Benson, 1933, Blair, 1940-1953, Dice, 1939-1949, Dice & Blossum, 1937,	X	X	X	X	I	W	I		X	X	X I	P									X	X	X

SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
		1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd.
Peromyscus supp. (continued)	Dice & Howard,1949, Hall & Hoffmeister, 1942, Hayne, 1950, Howard, 1949, Hooper, 1941, Murie,1933, Pasmussen, 1964																							
Peromyscus polionotus	Sumner, 1926-1932	X			I	I	W	I	I		X	I		X	X	X	X	X	X			X	X	X
Peromyscus polionotus	Selander et al.,1971	X	I	X		X	W				X		P	X	X	X	X	X	X			X	X	X
Sciurus variegatoides	Harris, 1937						W				X			X	X	X	X	X	X	X	X	X	X	X
Sciurus vulgaris	Voipio, 1950-1970, Voipio & Hissa,1970	X	I	I		I	W				X		X	X	X	X	X	X	X	X	X	X	X	X
Thonomys bottae	Ingles & Biglioni, 1952, Vaughan,1967	I	I	I	I		W				X			X	X	X	X	X	X	X	X	X		X
Vulpes vulpes	Voipio, 1950,1956					I	W				X		P	X	X	X	X	X	X	X	X	X	X	X
Vulpes fulva	Cowan, 1938,Cross, 1941					I	W				I		X	X	X	X	X	X	X	X	X	X	X	X
Ursus americanus	Cross, 1941					I	W				I			X	X	X	X	X	X	X	X	X	X	X
Plants																								
Agrostis tenuis, A. tenuis, and Anthoxanthum odoratum	Jain & Bradshaw, 1966, Aston & Bradshaw, 1966,McNeilley & Antonovics, 1968, McNeilley, 1969, Antonovics & Bradshaw, 1970, Antonovics, 1968a,b, 1972	X	X	X	X	X	X	I	X	X	X	I										X	X	X
Anthoxanthum odoratum	Snaydon, 1970	X	X	X	X		I	I			X											X	X	X
Avena barbata	Clegg & Allard, 1972, Hamrick & Allard, 1972	I	I	I		X	W	X			I				X		X	X				X		X
Clarkia biloba	Roberts & Lewis, 1955, Lewis,1953	X	I	I	I	I	X						X	X	X	X	X	X	X	X	X	X	X	X

Plants

SPECIES	REFERENCES	DATA											POSSIBLE EXPLANATIONS											
		1a	1b	1c	1d	2a	2b	3	4	5	6	7	Rg.	A	B	C	D	E	F	G	H	I	Ect.	Cd
Dactylus marina	Benson & Borrill, 1969	I				X	W				X			X	X	X	X	X	X	X	X	X	X	X
Eucalyptus spp.	Barber, 1965	X	X	X	X		X	I		I	X			X	X		X	X	X			X		X
Juniperus virginica	Flake et al.,1969	X	X	X			W				X			X	X	X	X	X	X	X	X	X	X	X
Justica simplex	Joshi & Jain, 1964	X				I	W				I		P	X	X	X	X	X	X	X	X	X	X	X
Linanthus parryae	Epling & Dobzhansky, 1942, Wright,1943, Epling et al.,1960	X	X	X	X	X	X	I		I	I		X	X	X		X	X	X			X		X
Lotus corniculatus	D.A. Jones, 1972					X	W				X	I		X	X	X	X	X	X	X	X	X	X	X
Mimulus guttatus	Allen & Sheppard, 1971	X	X	X	X	X	W				X	X										X	X	X
Pinus rigida	Leydig & Fryer,1972	I	I	I	I	I	W	I			X	I		X	X	X	X	X	X	X	X	X	X	X
Plantago maritima	Gregor, 1938						X	I		I	X	I		X	X	X	X	X	X	X	X	X	X	X
Quercus spp.	Muller, 1952	X	I	I	I		W	I	I				P	X	X	X	X	X	X	X	X	X	X	X
Spergula arvensis	New, 1958					I	W				X		P									X	X	X

Note: not all the clines cited are morph-ratio, for example, Maniola jurtina. The same kind of data and explanations could be applied to other types of clines.

typica or a small bias in dispersal (the second possible explanation) could displace the cline over several demes (chapter 2). In fact Bishop found a small bias in dispersal towards the southwest, just what would be expected to shift the cline downwind from Liverpool. It would be interesting to do a similar transect to the northeast of Liverpool; if it is dispersal bias then the cline would shift towards Liverpool, and if a deleterious effect of typica (or increased pesticide resistance of carbonaria) the cline would be shifted to the northeast. A third possibility is a local reduction of gene flow and, or, in population size between Loggerheads and Llanbear. As mentioned in chapter 2, this would attract any morphotones in the vicinity. This seems unlikely as the cline does not show a morphotone (Matthews type I). In any case there is no data on population size in that area.

Clarke and Sheppard (1966) found evidence for a general (position independent) decreased fitness of carbonaria homozygotes relative to heterozygotes. This would change the observed fitness gradients of Bishop (1972) into a model C, or heterozygote advantage cline, which is linear rather than s-shaped, as observed. Clarke and Sheppard (1966) also found that the frequency of a third gene, insularia, also a dominant melanic form, was correlated with the carbonaria frequency. If fitness gradients for all three forms were worked out the expected clines would be different from that assuming only a two allele model. The position at which the cline passes through 40 to 50% would not necessarily be the same as was observed in the typica and carbonaria gradients.

Bishop (1972) ran some simulations like that of the present author. He chose a model C system with 27 demes (spaced 2 km. apart), g=0.25 (stated as 0.20 in another part of his paper) and with h_1=0.05, 0.10, and 0.15. He ran his simulations for 160 generations and had

all demes with the same population size, in spite of different estimates from his capture-recapture data. He had no direct evidence for heterozygous advantage. He used the observed selection gradient for $W_2(x)$, and $W_3(x)$, and assumed $W_1(x)=W_2(x)-h_1$. The results of the model did not fit the observed clines very well. This does not rule out model C as an explanation as selection was not measured completely (h_1 not known, other components of selection not known), and as he points out, his inter deme distances were too close. His data suggested that, in fact, 5 km. would have been better than 2 km. He allowed for this by increasing the mean distance of gene flow; allowing longer distance gene flow. This improved the fit, but is, unfortunately an a posteriori adjustment. The population structure should have been worked out in greater detail before setting up the simulation. Aside from the incorrect interdeme distance, not allowing for differing deme size could explain the poor fit.

Bishop (1972, Bishop and Harper, 1970) has data on some of the selection gradients of the type of requirements 6 and 7, but does not make use of it. His maps show a patchwork of urbanisation and rural areas, and his transect crosses over several urban patches outside Liverpool. Both the selection gradients (Bishop, 1972) and the dispersal pattern (Bishop and Harper, 1970) are perturbed in the vicinity of the large urban patches. Dispersal does not fall off smoothly with distance, but goes up again near the urban patch (Bishop and Harper, 1970) and the frequency of carbonaria rises locally in urban areas. The work of Hamrick and Allard (1972) is an excellent example of taking account of local fluctuations in the environment, but they fall down in the other requirements of cline analysis. It is clear that a careful mapping of population structure and fitness gradients are the fundamental requirements for any proper cline study.

Difficulties of explanation are even in greater in investigating a pattern of species ranges; one cannot tell whether a pair of closely related species which are partially overlapping have evolved allopatrically or parapatrically. Almost all alleged examples of allopatric speciation can also be interpreted on the basis of parapatric speciation models (figure 1-2). For example the Leptodactylid frogs Pseudophryne dendyi, P. bibroni, and P. semimarmorata are contiguously distributed in southern Australia (Woodruff, 1972). The frogs are identical except in colour and show extremely sharp hybrid zones with reduced viability and increased developmental problems in the hybrid zones. The frogs are postulated by Woodruff to have separated sometime in the Pleistocene and diverged, and more recently come together to form their present state. However the few differences among the three "species" could be explained on the basis of a few colour-controlling genes, and the hybrid zone effect explained on the basis of a few coadaptive modifiers as in chapter 3, together with clinal selection patterns described in chapter 2.

The major findings of this study of morph-ratio clines are (1), gene flow may not be as important as contradifferentiating factor as it was often previously believed; (2), morphotones, or sharp changes in morph or gene frequency, may develop in the absence of barriers to gene flow, in the absence of spatially sharp environmental changes, and may develop along smooth selection gradients which are gentle enough so that it may be difficult to measure them in the field; (3), conversely, morphotones must not be interpreted as evidence for sexual or physiographic isolation, secondary contact, or sharp changes in the environmental in the vicinity of the clinal discontinuity (see also Endler, 1973); (4), drift alone cannot explain large scale "area effects"; (5), simple models of coadaptive modifiers

in clines may produce any of the effects of interpopulation crosses discussed in the literature (F_1 "vigour", F_2 "breakdown" and increased variance, etc.); and (6), certain combinations of coadaptive modifiers in clines may lead to hybrid zone effects and even to the evolution of sexual isolating mechanisms. All the paths shown in figure 1-2 are genetically possible with as few as four genes.

This study has raised more questions than it has solved. Prediction of clines is still imprecise (I cannot, for example, say exactly how much gene flow will produce a given natural cline, for example), but it is less imprecise than methods of estimating many of the parameters listed in requirements 1-7. At least the study has clarified the kinds of data which are required in a reasonably complete cline study, and the kinds of problems which may be expected in investigating patterns of geographical differentiation. Without a thorough geographic study of the population biology and history of a species, one cannot say by what mechanisms populations have differentiated.

LITERATURE CITED

A BIBLIOGRAPHY OF CLINES AND RELATED SUBJECTS

* Natural clines.
T Theoretical clines.

Allee, W.C., A.E. Emerson, O. Park, T. Park, and K.P. Schmidt,
1949, Principles of Animal Ecology. W.B. Saunders and Co., Philadelphia.

* Allen, W.R., and P.M. Sheppard,
1971, Copper tolerance in some Californian populations of the monkey flower, Mimulus guttatus. Proc. Roy. Soc. Lond. B. 177:177-196.

* Alpatov, W.W.,
1929, Biometrical studies on variation and races of the Honey Bee, (Apis mellifera L.), Quart. Rev. Biol. 4(1):1-58

* Anderson, E.,
1949, Introgressive Hybridization. New York: John Wiley and Sons, Inc.

* Anderson, G.W.,
1970, Haemoglobin and protein variation in three species of Littorina. Ophelia, 8:267-273.

Anderson, P.K.,
1964, Lethal alleles in Mus musculus: Local distribution and evidence for demes. Science 145(3628):177-178.

Anderson, P.K.,
1969, unpublished data referred to in Ehrlich and Raven, 1969.

Anderson, P.K., and J.L. Hill,
1965, Mus musculus: Experimental induction of territory formation. Science 148:1753-1755.

Anderson, P.K., L.C. Dunn, and A.B. Beasley,
1964, Introduction of a lethal allele into a feral house mouse population. Amer. Nat. 98:57-64.

Andrewartha, H.G., and L.C. Birch,
1954, The Distribution and Abundance of Animals. Chicago: University of Chicago press.

* Antonovics, J.
1968a, Evolution in closely adjacent plant populations, V. Evolution of self-fertility. Heredity 23(2):219-238.

* Antonovics, J.
1968b, Evolution in closely adjacent plant populations, VI. Manifold effects of gene flow. Heredity 23(4):507-524.

* Antonovics, J.
1971, The effects of a heterogeneous environment on the genetics of natural populations. American Scientist 59(5):593-599.

* Antonovics, J. and A.D. Bradshaw
1970, Evolution in closely adjacent plant populations, VIII. Clinal patterns at a mine boundary. Heredity 25(3):349-362.

Archimowitsch, A.
1949, Control of Pollination in the Sugar Beet. Bot. Rev. 15:613-628.

* Arnold, R.W.
1968, Studies on Cepaea, VII, Climatic selection in Cepaea nemoralis (L.) in the Pyrenees. Phil. Trans. Roy. Soc. Lond. B. 253:549-593.

* Aston, J.L. and A.D. Bradshaw
1966, Evolution in closely adjacent plant populations. II. Agrostis stolonifera in maritime habitats. Heredity 21(4):649-664.

Baker, R.R.
1968, A possible method of evolution of the migrating habit of butterflies. Phil. Trans. Roy. Soc. Lond. B. 253:309-341.

* Barber, H.N.
1965, Selection in natural populations. Heredity 20(4):551-572.

Bartholomew, G.A.
1957, The role of physiology in the distribution of terrestrial vertebrates. In C.L. Hubbs, editor, Zoogeography, Publ. (51), Amer. Assoc. Adv. Sci., 1958, pp. 81-85.

Bateman, A.J.
1947a, Contamination of seed crops. I. Insect Pollination. J. Genetics 48(2):257-275.

Bateman, A.J.
1947b, Contamination in seed crops. II. Wind Pollination. Heredity 1(2):235-246.

Bateman, A.J.
1947c, Contamination in seed crops. III. Relation with isolation distance. Heredity 1(3):303-336.

Bateman, A.J.
1950, Is gene dispersion normal? Heredity 4(3):353-363.

Beall, G.
1941, The monarch butterfly, Danaus archippus Fab. II. The movement in southern Ontario. Canad. Field Nat. 60:123-137.

* Beall, G. and C.B. Williams
1945, Geographic variation in the wing length of Danaus plexippus (Lep. Rhopalocera). Proc. Roy. Entomol. Soc. Lond. 20:65-76.

* Benson, M, and M. Barrill,
1969, The significance of clinal variation in Dactylis marina Barrill. New Phytol. 68:1159-1173

* Benson, S.B.
1933, Concealing coloration among some desert rodents of the southwestern United States. Univ. Calif. Publ. Zool. 40(1):1-69.

Bigelow, R.S.
1965, Hybrid zones and reproductive isolation. Evolution 19(4):449-458.

Birch, L.C.
1945, The mortality of the immature stages of Calandra oryzae (small strain) and Rhizopertha dominica Fab. in wheat of different moisture contents. Aust. J. Exp. Biol. Med. Sci. 23:141-145

* Birdsell, J.B.
1950, Some implications of the genetical concept of race in terms of spatial analysis. Cold. Spr. Harb. Symp. Quant. Biol. 15:259-311, discussion p. 311-314.

* Bishop, J.A., and P.S. Harper
1970, Melanism in the moth Gonodontis bidentata: A cline within the Merseyside conurbation. Heredity 25:449-456.

T* Bishop, J.A.
1972, An experimental study of the cline in industrial melanism in Biston betularia (L.)(Lepidoptera) between urban Liverpool and rural North Wales. J. Animal Ecol. 41(1):209-243.

* Blair, W.F.
1940, A Study of Prarie Deer-Mouse Populations in Southern Michigan. Amer. Midl. Nat. 24(2):273-305.

Blair, W.F.
1943a, Criteria for species and their subdivisions from the point of view of genetics. Ann. New York. Acad. Sci. 44(2):179-188.

* Blair, W.F.
1943b, Populations of the Deer-Mouse and associated small mammals in the Mesquite associations of southern New Mexico. Contr. Lab. Vert. Biol. Univ. Mich. (21):1-40.

* Blair, W.F.
1947, Estimated frequencies of the buff and grey genes (G,g) in adjacent populations of Deer-mice (Peromyscus maniculatus) living on soils of different colors. Contr. Lab. Vert. Biol. Univ. Mich. (36):1-16.

* Blair, W.F.
1950, Ecological factors in speciation of Peromyscus. Evolution 4(4):253-275.

* Blair, W.F.
1951a, Population structure, social behavior and environmental relations in natural populations of the Beach Mouse, Peromyscus polionotus leucocephalus. Contr. Lab. Vert. Biol. Univ. Mich. (48):1-47.

* Blair, W.F.
1951b, Interbreeding of natural populations of vertebrates. Amer. Nat. 85(820):9-30.

Blair, W.F.
1954, A Melanistic race of the White-throated packrat. (Neotoma albigula) in Texas. J. Mammal. 35(2):239-242.

Blair, W.F.
1960, The Rusty Lizard, a Population Study. Austin: University of Texas Press.

Black, M.
1962, Models and Metaphors. Ithaca, N.Y.: Cornell U. Press.

Bocher, T.W.
1967, Continuous variation and taxonomy. Taxon. 16:255-258

* Bocquet, C.
1969, Le Probleme des formes apparentees a distribution contigue. Bull. Zool. Soc. France 94(4):517-526.

T Bodmer, W.F. and L.L. Cavalli-Sforza
1968, A Migration-Matix Model of Random Genetic Drift. Genetics 59(4):565-592.

* Bovey, P.
1941, Contribution a L'etude Genetique at Biogeographique de Zygaena ephialtes L. Rev. Suisse Zool. 48:1-90.

Bradshaw, A.D.
1962, The taxonomic problems of local geographic variation in plant species. In D. Nichols, editor, Taxonomy and Geography Syst. Assoc. Lond. Publ. (4):7-16.

Bridges, C.B., and K.F. Brehme
1944, The mutants of Drosophila melanogaster. Carnegie Inst. Wash. Publ. (7):1-257.

* Brooks, J.L.
1957, The species problem in freshwater animals. In, E. Mayr, editor, The Species Problem, Publ. (50), Amer. Assoc. Adv. Sci., pp. 81-123.

Brncic, D.
1961, Integration of the genotype in geographic populations of Drosophila pavani. Evolution 15(1):92-97.

Brown, R.G.B.
1967, Breeding success and population growth in a colony of Herring and Lesser Black-backed gulls, Larus argentatus and L. fuscus. Ibis 109(4):502-515.

Burla, H., A.B. DaCunha, A.G.F. Cavalcanti, Th. Dobzhansky, and C. Pavan
1950, Population density and dispersal rates in Brazilian Drosophila willistoni. Ecology 31(3):393-404.

Burt, W.H.
1954, The subspecies category in mammals. Syst. Zool. 3:99-104.

Bush, G.L.
1969, Sympatric host race formation and speciation in frugiverous flies of the genus Rhagoletis (Diptera, Tephritidae). Evolution 23(2):237-251.

Cain, A.J.
1953, Geography, ecology, and coexistance in relation to the biological definition of the species. Evolution 7:76-83.

Cain A.J.
1954, Animal Species and Their Evolution. New York and London: Hutchinson's University Library.

* Cain, A.J.
1971, Colour and banding morphs in subfossil samples of the snail Cepaea. In, E.R. Creed, editor, Ecological Genetics and Evolution, pp. 65-92. Oxford:Blackwells Scientific Publications.

* Cain, A.J. and J.D. Currey
1963a, Area effects in Cepaea. Phil. Trans. Roy. Soc. Lond. B. 246:1-81.

Cain, A.J. and J.D. Currey
1963b, The Causes of Area Effects. Heredity 18:467-471.

* Cain, A.J. and J.D. Currey
1963c, Area effects in Cepaea on the Larkhill Artillery Range, Salisbury Plain. J. Linn. Soc. (Zool) Lond. 45:1-15.

* Cain, A.J. and J.D. Currey
1968, Studies on Cepaea. III. Ecogenetics of a population of Cepaea nemoralis (L.) subject to strong area effects. Phil. Trans. Roy. Soc. Lond. B. 253:447-482.

* Carson, H.L. and H.D. Stalker
1947, Gene arrangements in natural populations of Drosophila robusta Sturtevant. Evolution 1(3):113-133.

Carter, G.S.,
1959, Evolution in the Deep Sea. In, M. Sears, editor, Oceanography, Publ. (67), Amer. Assoc. Adv. Sci., 1961, pp. 229-237.

* Carter, M.A.
1968, Studies on Cepaea. II. Area effects and visual selection in Cepaea nemoralis (l.) and Cepaea hortensis. Phil. Trans. Roy. Soc. Lond. B. 253:397-446.

* Chapin, J.P.
1948, Variation and hybridization among the paradise flycatchers of Africa. Evolution 2(1):111-126

Chorley, R.J. and P. Haggett
1967, Models in Geography. London: Methuen and Co. Ltd.

Chorofas, D.N.
1965, Systems and Stimulation. New York. (Quoted in Haggett and Chorley, 1967).

Christian, J.J.
1970, Social Subordination, population density, and mammalian evolution. Science 168(3927):84-90.

* Christy, C.
1929, The African Buffaloes. Proc. Zool. Soc. Lond. 1929, paper (36):445-462.

Clarke, B.C.
1964, Frequency-dependent selection for the dominance of rare polymorphic genes. Evolution 18(3):364-369.

T Clarke, B.C.
1966, The evolution of morph-ratio clines. Amer. Nat. 100(914): 389-402.

* Clarke, B.C.
1968, Balanced polymorphism and regional differentiation in snails. In, E.T. Drake, editor, Evolution and Environment. pp. 351-368.

Clarke, B.C.
1972, Density-dependent Selection. Amer. Nat. 108(947):1-13.

* Clarke, B.C., C. Diver, and J.J. Murray,
1968, Studies on Cepaea, VI. The spatial and temporal distribution of phenotypes in a colony of Cepaea nemoralis (L.). Phil. Trans. Roy. Soc. Lond. B. 253:519-548.

* Clarke, B.C., and J. J. Murray,
1962, Changes of gene frequency in Cepaea nemoralis. Heredity 17:445-465.

* Clarke, B.C., and J.J. Murray,
1969, Ecological genetics and speciation in land snails of the genus Partula. Biol. J. Linn. Soc. Lond. 1(1):31-42

* Clarke, B.C., and J.J. Murray,
1971, Polymorphism in a Polynesian land snail, Partula suturalis vexillium. In R. Creed, editor, Ecological Genetics and Evolution, pp.51-64, Oxford: Blackwells Scientific Publications.

Clarke, C.A., and P.M. Sheppard,
1962, Disruptive selection and its effects on a metrical character in the butterfly Pipilio dardanus. Evolution 16:244-226.

* Clarke, C.A., and P.M. Sheppard,
1966, A local survey of the distribution of industrial melanics found in the moth Biston betularia and estimates of the selective values of these in an industrial environment. Proc. Roy. Soc. Lond. B. 165:424-439.

Clarke, G.L,
1954, Elements of Ecology. New York: John Wiley and Sons, Inc.

* Clausen, J., D.D. Keck, and W.M. Hiesey,
1940, Experimental studies on the nature of species, I. Effects of varied environments on western North American plants. Carnegie Institute of Washington Publication (520):1-452.

* Clausen, J., D.D. Keck, and W.M. Hiesey,
1947, Heredity of geographically and ecologically isolated races. Amer. Nat. 81(797):114-133.

* Clegg, M.T., and R.W. Allard,
1972, Pattern of Genetic differentiation in the slender wild oat, Avena barbata. Proc. Nat. Acad. Sci. U.S. 69(7):1820-1824.

* Clench, W.J.,
1954, Subspecies and clines: The occurrence of clines in molluscan populations. Syst. Zool. 3:123-125.

* Conant, R.,
1958, A Field Guide to Reptiles and Amphibians of the United States and Canada east of the 100th Meridian. Boston (U.S.A.): Houghton Mifflin Co.

Cole, J.P., and C.A.M. King,
1970, Quantitative Geography. New York: John Wiley and Sons, Inc.

Colwell, R.N.,
1951, The use of radioactive isotopes in determining spore distribution patterns. Amer. J. Bot. 38(7):511-523.

Cook, L.M.,
1961, The edge effect in population genetics. Amer. Nat. 95(884): 295-307.

T Cook, L.M.,
1971, Coefficients of Natural Selection. Hutchinson's University Library, Hutchinson and Co., Ltd., London.

Coulson, J.S., and E. White,
1958, Observations on the breeding of the Kittiwake. Bird Study, Oxford 5(2):74-83.

* Cowan, I.M.,
1938, Geographic distribution of color phases of the red fox and black bear in the Pacific Northwest. J. Mammal. 19:202-208.

* Creed, E.R.,
1971, Melanism in the two-spot ladybird, Adalia bipunctata in Great Britain. Chapter 7 in E.R. Creed, editor, Ecological Genetics and Evolution. Oxford: Blackwells Scientific Publications.

* Creed, E.R., W.H. Dowdswell, E.B. Ford, and K.G. McWhirter,
1970, Evolutionary studies on Maniola jurtina (Lepidoptera: Stayridae): The "boundary phenomenon" in southern England 1961 to 1968. Chapter 9 in M.K. Hecht and W.C. Steere, editors, Essays in Evolution and Genetics in honour of Theodosius Dobzhansky. Amsterdam: North-Holland Publishing Company.

* Crenshaw, J.W., Jr., and W.F. Blair,
1959, Relationships in the Pseudacris nigrita complex in southern Georgia. Copeia 1959(3):215-222.

Crosby, J.L.,
1940, High proportion of homostyle plants in populations of Primula vulgaris. Nature 145(3678):672-673.

T* Crosby, J.L.,
1970, The evolution of genetic discontinuity: computer models of the selection of barriers to interbreeding between subspecies. Heredity 25(2):253-297.

* Cross, E.C.,
1941, Color phases of the red fox (Vulpes fulvus) in Ontario. J. Mammal. 22:25-39.

Crowcroft, P., and F.P. Rowe,
1963, Social organization and territorial behaviour in the wild house mouse (Mus musculus L.). Proc. Zool. Soc. Lond. 140:517-531.

* Cuellar, H.C.,
1971, Levels of genetic compatability of Rana areolata with south-western members of the Rana pipiens complex (Anura: Ranidae). Evolution 25(2):399-409.

* Currey, J.D., and A.J. Cain,
1968, Studies on Cepaea, IV. Climate and selection of banding morphs in Cepaea from the climatic optimum to the present day. Phil. Trans. Roy. Soc. Lond. B. 253:483-498.

* Dale, F.H.,
1940, Geographic variation in the meadow mouse in British Columbia and southeastern Alaska. J. Mammal. 21:332-340.

* Darlington, P.J., Jr.,
1957, Zoogeography: The Geographical Distribution of Animals. New York: John Wiley and Sons, Inc.

Darwin, C.,
1859, On the Origin of Species by Means of Natural Selection or the Preservation of Favoured Races in the Struggle for Life. London: John Murray (Facsimile of the first edition published by Harvard University Press, Cambridge, 1964).

* Day, J.C.L., and W.H. Dowdswell,
1968, Natural selection in Cepaea on Portland Bill. Heredity 23 (2):169-188.

Den Boer, P.J.,
1971, On the dispersal power of Carabid beetles and its possible significance. Pages 119-137 of Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Bird Station, Wijster, Nov. 3-5, 1969, P.J. Den Boer, editor. Misc. Papers 8(1971) Landbouwhogeschool, Waginingen, The Netherlands.

* Dice, L.R.,
1931, The occurrence of two subspecies of the same species in the same area. J. Mammal. 12:210-213.

* Dice, L.R.,
1939a, Variation in the Cactus-mouse, Peromyscus eremicus. Contr. Lab. Vert. Genet. Univ. Mich. (8):1-27.

Dice, L.R.,
1939b, An estimate of the population of deer mice in the black hills of South Dakota and Wyoming. Contr. Lab. Vert. Genet. Univ. Mich. (10):1-5.

* Dice, L.R.,
1939c, Variation in the Deer Mouse (Peromyscus maniculatus) in the Columbia basin of Southeastern Washington and Adjacent Idaho and Oregon. Contr. Lab. Vert. Genet. Univ. Mich. (13):1-14.

* Dice, L.R.,
1940a, Ecologic and genetic variability within species of Peromyscus. Amer. Nat. 74:212-221.

Dice, L.R.,
1940b, Speciation in Peromyscus. Amer. Nat. 74(753):289-298.

* Dice, L.R.,
1940c, Intergradation between two subspecies of Deer-mouse (Peromyscus maniculatus) across North Dakota. Contr. Lab. Vert. Genet. Univ. Mich. (13):1-14.

* Dice, L.R.,
1941, Variation in the Deer-mice (Peromyscus maniculatus) on the sand hills of Nebraska and adjacent areas. Contr. Lab. Vert. Genet. Univ. Mich. (15):1-19.

Dice, L.R.,
1947, Effectiveness of selection by owls of deer-mice (Peromyscus maniculatus) which contrast in color with the background. Contr. Lab. Vert. Biol. Univ. Mich. (34):1-20.

* Dice, L.R.,
1949, Variation of Peromyscus maniculatus in parts of western Washington and adjacent Oregon. Contr. Lab. Vert. Biol. Univ. Mich. (44):1-34.

* Dice, L.R., and P.M. Blossum,
1937, Studies of mammalian ecology in southwestern North America with special reference to the colors of desert mammals. Carnegie Institute of Washington Publication (485):1-129.

Dice, L.R., and W.E. Howard,
1951, Distance of dispersal by Prarie Deermice. Contr. Lab. Vert. Biol. Univ. Mich. (50):1-15.

Dillon, L.S.,
1956, Wisconsin climate and life zones in North America. Science 123(3168):167-176.

Dillon, L.S.,
1970, Speciation and changing environment. American Zoologist 10(1):27-39.

Diver, C.,
1938, The plant carpet in relation to animal distribution. Proc. Linn. Soc. Lond. 150:124-135.

Diver, C.,
1940, On the problem of closely related species living in the same area. Pages 303-328 in J.S. Huxley, editor, The New Systematics. Oxford: Oxford University Press.

* Dobzhansky, Th.,
1933, Geographic variation in Lady Beetles. Amer. Nat. 67(709): 97-126.

Dobzhansky, Th.,
1940, Speciation as a stage in evolutionary divergence. Amer. Nat. 74(753):312-321.

* Dobzhansky, Th.,
1941, 1951, Genetics and the Origin of Species (Second and Third editions). New York: Columbia University Press.

* Dobzhansky, Th.,
1948, Genetics of natural populations. XVI. Altitudinal and seasonal changes provided by natural selection in certain populations of Drosophila persimilis. Genetics 33:158-176.

* Dobzhansky, Th.,
1970, Genetics of the Evolutionary Process. New York: Columbia University Press.

* Dobzhansky, Th., W.W. Anderson, and O. Pavlovsky,
1966, Genetics of natural populations. XXXVIII. Constancy and change in populations of Drosophila pseudoobscura in Western United States. Evolution 20(3):418-427.

* Dobzhansky, Th., and C. Epling,
1944, Contributions to the genetics, taxonomy and ecology of Drosophila pseudoobscura and its relatives. Carnegie Inst. Wash. Publ. 544.

Dobzhansky, Th., H. Levene, and B. Spassky,
1971, Effects of selection and migration on geotactic and phototactic behaviour in Drosophila III. Proc. Roy. Soc. B. 180:21-41.

Dobzhansky, Th., and B. Spassky,
1967, Effects of selection and migration on geotactic and phototactic behaviour of Drosophila I. Proc. Roy. Soc. B. 168:27-47.

Dobzhansky, Th., B. Spassky, and J. Sved,
1969, Effects of selection and migration on geotactic and phototactic behavious in Drosophila II. Proc. Roy. Soc. B. 173:191-207.

Dobzhansky, Th., and S. Wright,
1941, Genetics of natural populations. V. Relations between mutation rate and accumulation of lethans in populations of Drosophila pseudoobscura. Genetics 26:23-51.

Dobzhansky, Th., and S. Wright,
1943, Genetics of natural populations. X. Dispersion rates in Drosophila pseudoobscura. Genetics 28:304-340.

Dobzhansky, Th., and S. Wright,
1947, Genetics of natural populations. XV. Rate of diffusion of a mutant gene through a population of Drosophila melanogaster. Genetics 32:303-324.

Doggett, H., and B. N. Mojisu,
1968, Disruptive selection in crop development. Heredity 23(1): 1-23.

* Dubinin, N.P., and G.G. Tiniakov,
1946, Inversion gradients and natural selection in ecological races of Drosophila funebris. Genetics 31:537-545.

* Dunn, E.R.,
1943, Lower categories in herpetology. Ann. N.Y. Acad. Sci. 44(2):123-131.

Ehrlich, P.,
1961, Intrinsic barriers to dispersal in checkerspot butterfly. Science 134(3472):108-109.

* Ehrlich, P.R.,
1965, The population biology of the butterfly Euphydryas editha. II. The structure of the Jasper Ridge colony. Evolution 19(3):327-336.

Ehrlich, P.R.,
1969, unpublished data quoted in Ehrlich and Raven, 1969.

* Ehrlich, P.R., and L.G. Mason,
1966, The population biology of the butterfly Euphydryas editha. III. Selection and the phenetics of the Jasper Ridge colony. Evolution 20(2):165-173.

Ehrlich, P.R., and P.H. Raven
1969, Differentiation of populations. Science 165(3899):1228-1232.

Ehrman, L.,
1966, Mating success and genotype frequency in Drosophila. Anim. Beh. 14:332-339.

Ehrman, L.,
1967, Further studies on genotype frequency and mating success in Drosophila. Amer. Nat. 101(921):415-424.

Ehrman, L.,
1970, Simulation of the mating advantage of rare Drosophila males. Science 167:905-906.

Ehrman, L., and C. Petit,
1968, Genotype frequency and mating success in the willistoni species group of Drosophila. Evolution 22:649-658.

Ekman, S.,
1953, Zoogeography of the Sea. London: Sidgwick and Jackson.

Elton, C.,
1927, Animal Ecology. London: Sidgwick and Jackson.

T Endler, J.A.,
1973, Gene flow and population differentiation, Science 179:243-250

* Epling, C.,
1947, Actual and potential gene flow in natural populations. Amer. Nat. 81(797):104-113.

* Epling, C., and Th. Dobzhansky,
1942, Genetics of natural populations. VI. Microgeographical races in Linanthus parryae. Genetics 27:317-332.

* Epling, C., H. Lewis, and F.M. Ball,
1960, The breeding group and seed storage: a study in population dynamics. Evolution 14: 238-235.

Errington, P.L.,
1946, Predation and vertebrate populations. Quart. Rev. Biol. 21(2,3): 144-177, 221-245.

Errington, P.L.,
1963, Muskrat Populations. Iowa State University Press, Ames, Iowa.

* Ferrell, G.T.,
1966, Variation in blood group frequencies in populations of song sparrows of the San Francisco bay region. Evolution 20(3):369-382.

T* Fisher, R.A.,
1930, The Genetical Theory of Natural Selection. Oxford: Oxford Clarendon Press.

T Fisher, R.A.,
1937, The wave of advance of advantageous genes. Ann. Eugen. 7:355-369.

T Fisher, R.A.,
1950, Gene frequencies in a cline determined by selection and diffusion. Biometrics 6:353-361.

Fisher, R.A., and F.B. Yates,
1948, Statistical Tables for Biological, Agricultural, and Medical Research. Edinburgh: Oliver and Boyd.

* Fitch, H.S.,
1939, A systematic account of the Alligator Lizards (Gerrhonotus) in the Western United States and Lower California. Amer. Midl. Nat. 20(2):381-424.

* Fitch, H.S.,
1941, A biogeographical study of the Ordinoides Artenkreis of garter snakes (genus Thamnophis). Univ. Calif. Publ. Zool. 44(1):1-150.

* Flake, R.H., E. VonRudloff, and B.L. Turner,
1969, Quantitative study of clinal variation in Juniperus virginiana using terpenoid data. Proc. Nat. Acad. Sci. U.S. 64:487-494.

* Ford, E.B.,
1940, Polymorphism and Taxonomy. Pages 493-513 in J.S. Huxley, editor, The New Systematics, Oxford: Oxford University Press.

Ford, E.B.,
1945, Polymorphism. Biol. Rev. 20(2):73-88.

* Ford, E.B.,
1949, Early stages in allopatric speciation. Pages 304-314 in G.L. Jepson, G.G. Simpson, and E. Mayr, editors, Genetics, Palaeontology and Evolution. Princeton: Princeton Univ. Press.

* Ford, E.B.,
1954, Problems in the evolution of geographic races. Pages 99-108 in J.S. Huxley, A.C. Hardy, and E.B. Ford, editors, Evolution as a Process. London: George Allen and Unwin, Ltd.

* Ford, E.B.,
1964, 1971, Ecological Genetics (Second and Third editions). London: Methuen and Company (Chapman and Hall).

* Fox, W.,
1951, Relationships among the garter snakes of the Thamnophis elegans Rassenkreise. Univ. Calif. Publ. Zool. 40(5):485-530.

* Frydenberg, O., D. Moller, G. Naevdal, and K. Sick,
1965, Haemoglobin polymorphism in Norwegian cod populations. Hereditas 53:257-271.

Gadgil, M.,
1971, Dispersal: Population consequences and evolution. Ecology 52(2):253-261.

* Galbraith, I.C.J.,
1956, Variation, relationships, and evolution in the Pachycephala pectoralis superspecies (Aves, Muscicapidae). Bull. Brit. Mus. (Nat. Hist.) Zool. 4:133-222.

Geiger, R.,
1961, The Climate Near the Ground (Translation of the second edition). Cambridge (U.S.A.): Harvard University Press.

Gerking, S.D.,
1959, The restricted movement of fish populations. Biol. Rev. 34(2):221-242.

* Gershenson, S.,
1945a, Evolutionary studies in melanism in the Hamster (Cricetus cricetus L.), I: Distribution of black hamsters in the Uhur and Bachkirian Soviet Socialist Republics (U.S.S.R.). Genetics 30: 233-251.

* Gershenson, S.,
1945b, Evolutionary studies of melanism in the Hamster (Cricetus cricetus L.), II: Seasonal and annual changes in the frequency of black hamsters. Genetics 30:233-251.

* Gilbert, C.R.,
1961, Hybridization versus intergradation: An inquiry into the relationship of two cyprinid fishes. Copeia 1961(2):181-192.

Gilmour, J.S.L., and J.W. Gregor,
1939, Demes: A suggested new terminology. Nature 144(3642):333.

* Glover, R.S.,
1959, Biogeographical boundaries: The shapes of distributions, in, M. Sears, editor, Oceanography, Publ. (67), Amer. Assoc. Adv. Sci., 1961, pp. 201-228.

* Goldschmidt, R.,
1934, Lymantria. Bibliographica Genetics, The Hague, 11:1-186.

* Gooch, J.L., and T.J.M. Schopf,
1971, Genetic variation in tha marine Ectoproct Schizoporella errata. Biol. Bull. 141(2):235-246.

* Goodhart, C.B.,
1963, "Area effects" and non-adaptive variation between populations of Cepaea (mollusca). Heredity 18:459-465.

* Grant, V.,
1957, The plant species in theory and practice, in, E. Mayr, editor, The Species Problem, Publ. (50), Amer. Assoc. Adv. Sci., pp. 39-80.

* Grant, V.,
1963, The Origin of Adaptations. New York: Columbia University Press.

* Gregor, J.W.,
1938, Experimental taxonomy II. Initial population differentiation in Plantago maritima L. of Britain. New. Phytol 37(1):15-49.

* Gregor, J.W.,
1939, Experimental taxonomy IV. Population differentiation in North American and European sea plantains allied to Plantago maritima L. New. Phytol. 38(4):293-322.

* Gregor, J.W., and P.J. Watson,
1961, Ecotypic differentiation: Observations and reflections. Evolution 15(2):166-173.

Griffiths, D.J.,
1950, The liability of seed crops of perennial rye grass (Lolium perenne) to contamination by wind-borne pollen. J. Agr. Sci. 40(1):19-38.

Grinnell, J.,
1914, Barriers to distribution as regards birds and mammals. Amer. Nat. 48(568):248-254.

* Gulik, J.T.,
1905, Evolution, Racial and Habitudinal. Carnegie Institution of Washington Publ. (25):i-vi, 1-269.

Haeck, J.,
1971, The immigration and settlement of Carabids in the New Ijsselmeerpolder. Pages 33-52 in P.J. Den Boer, editor, Dispersal and dispersal power in Carabid Beetles, a symposium held at the Biological Station, Wijster, Nov. 3-5, 1969, Misc. Papers 9(1971). Landbouwhogeschool, Waginingen, The Netherlands.

* Hagen, D.W.,
1967, Isolating mechanisms in Threespine Stiklebacks. J. Fish. Res. Bd. Canada 24(8):1687-1692.

* Hagen, D.W., and J.D. McPhail,
1970, The species problem within Gasterosteous aculeatus on the Pacific Coast of North America. J. Fish. Res. Bd. Canada 27(1): 147-155.

Haggett, P. and R.J. Chorley,
1967, Models, Paradigms and the New Geography. Pages 19-41 in R.J. Chorley and P. Haggett, editors, Models in Geography. London: Mathuen and Co., Ltd.

* Hairston, N.G.,
1950, Intergradation in Appalachian salamanders of the genus Plethodon. Copeia 1950(4):262-273.

* Hairston, N.G., and C.H. Pope,
1948, Geographic variation and speciation in Appalachian salamanders (Plethodon jordani group). Evolution 2(3):266-278.

* Hale Carpenter, G.D.,
1932, The forms of Acraea johnsoni, Godm. (Lep. Nymphalidae). Trans. Roy. Entomol. Soc. Lond. 80(2):251-268.

T Haldane, J.B.S.,
1932, The Causes of Evolution. London: Longmans Green and Co., Ltd.

T Haldane, J.B.S.,
1948, The theory of a cline. J. Genet. 48(3):277-284.

*T Haldane, J.B.S.,
1956, The theory of selection for melanism in Lepidoptera. Proc. Roy. Soc. B. 145:303-306.

* Halkka, O.,
1964, Geographical, spatial, and temporal variability in the balanced polymorphism of Philaenus spumarius. Heredity 19(3): 383-401.

* Halkka, O., and E. Mikkola,
1965, Characterization of clines and isolates in a case of balanced polymorphism. Hereditas 54:140-148.

* Halkka, O., L. Heinonen, M. Raatikainen, and A. Vasarainen,
1966, Crossing experiments with Philaenus spumarius (Homoptera). Hereditas 56:306-312.

* Halkka, O., M. Raatikainen, A. Vasarainen, and L. Heinonen,
1967, Ecology and ecological genetics of Philaenus spumarius (L.) (Homoptera). Ann. Zool. Fenn. 4:1-18.

* Halkka, O., M. Raatikainen, and J. Vilbaste,
1967, Mode of balance in the polymorphism of Philaenus spumarius (L.). Ann. Acad. Scient. Fenn. A. IV (107):1-16.

* Hall, E.R., and D.F. Hoffmeister,
1942, Geographic variation in the canyon mouse, Peromyscus crinitus. J. Mammal. 23:51-65.

* Hamrick, J.L., and R.W. Allard,
1972, Microgeographical variation in allozyme frequencies in Avena barbata. Proc. Nat. Acad. Sci. U.S. 69(8):2100-2104.

T Hanson, W.D.,
1966, Effects of partial isolation (distance), migration, and different fitness requirements among environmental pockets upon steady state gene frequencies. Biometrics 22(3):453-468.

Harden Jones, F.R.,
1968, Fish Migration. London: Edward Arnold.

* Harris, M.P., Jr.,
1937, Revision of Sciurus variegatoides, a species of Central American ground squirrel. Misc. Publ. Mus. Zool. Univ. Mich. (38):1-39.

* Haskins, C.P., E.F. Haskins, J.J.A. McLaughlan, and R.E. Hewitt,
1961, Polymorphism and population structure in Lebistes reticulatus, an ecological study. Pages 320-395 in W.F. Blair, editor, Vertebrate Speciation. Austin: University of Texas Press.

Hasler, A.D.,
1954, Odour perception and orientation in fishes. J. Fish. Res. Bd. Canada 11:107-129.

Hasler, A.D., and W.J. Wisby,
1958, The return of displaced Largemouth Bass and Greed sunfish to a "home" area. Ecology 39(2):289-293.

* Hayne, D.W.,
1950, Reliability of laboratory-bred stocks as samples of wild populations, as shown in a study of the variation of Peromyscus polionotus in parts of Florida and Alabama. Contr. Lab. Vert. Biol. Univ. Mich. (46):1-56.

Heape, W.,
1931, Emigration, Migration and Nomadism (prepared and edited by F.H.A. Marshall). Cambridge: W. Heffer and Sons, Ltd.

Hesse, R., W.C. Allee, and K.P. Schmidt,
1937, Ecological Animal Geography. New York: John Wiley and Sons, Inc.

Heusser, H.,
1969, Die Lebensweise der Erdkrote, Bufo bufo (L.): Das Orientierungsproblem. Rev. Suisse Zool. 76:443-518.

* Heuts, M.J.,
1947, Experimental studies on adaptive evolution in Gasterosteus aculeatus L. Evolution 1(1):89-92.

* Hewitt, G.M., and B. John,
1967, The B-chromosome system of Myrmeleotettix maculatus. (Thunb.). III. The statistics. Chromosoma (Berl.) 21(2):140-162.

* Hewitt, G.M., and B. John,
1970, The B-chromosome system of Myrmeleotettix maculatus. (Thunb.). IV. The dynamics. Evolution 24(1):169-180.

* Hewitt, G., and F.M. Brown,
1970, The B-chromosome system of Myrmeleotettix maculatus. V. A steep cline in East Anglia. Heredity 25(3):365-371.

* Hewitt, G., and C. Ruscoe,
1971, Changes in microclimate associated with a cline for B-chromosomes in Myrmeleotettix maculatus (Thunb.) (Orthoptera: Acrididae). J. Anim. Ecol. 40:753-765.

* Highton, R.,
1962, Revision of North American salamanders of the genus Plethodon. Bull. Fla. St. Mus. Biol. Sci. 6:235-367.

* Highton, R.,
1971, Distributional interactions among eastern North American salamanders of the genus Plethodon. Pages 139-188 in P.C. Holt, editor, The Distributional History of the Biota of the Southern Appalachians, Res. Div. Monogr. (4), Virginia Polytechnic Inst.

T Holgate, P.,
1964, Genotype frequencies in a section of a cline. Heredity 19:507-509.

* Hooper, E.T.,
1941, Mammals of the lava fields and adjoining areas in Valencia County, New Mexico. Misc. Publ. Mus. Zool. Univ. Mich. (51):7-47.

* Hovanitz, W.,
1940, Ecological color variation in a butterfly and the problem of "protective coloration." Ecology 21(3):371-380.

* Hovanitz, W.,
1943, Geographic variation and racial structure of Argynnis callippe in California. Amer. Nat. 77(772):400-425.

* Hovanitz, W.,
1944a, Genetic data on the two races of Colias chrysothene in North America and on a white form in each. Genetics 29:1-30.

* Hovanitz, W.,
1944b, The distribution of gene frequencies in wild populations of Colias. Genetics 29:31-60.

* Hovanitz, W.,
1949, Increased variability in populations following natural hybridization. Pages 339-355 in G.L. Jepson, G.G. Simpson, and E. Mayr, editors, Genetics, Palaeontology and Evolution. Princeton: Princeton Univ. Press.

* Hovanitz, W.,
1953, Polymorphism and evolution. Pages 238-251 in J. Brown and J.F. Danielli, editors, Evolution, symposium no. 7, symposia of the Society for Experimental Biology.

Howard, W.E.,
1949, Dispersal, amount of inbreeding, and longevity in a local population of prarie deermice on the George Reserve, southern Michigan. Contr. Lab. Vert. Biol. Univ. Mich. (43):1-50.

* Hubbell, T.H.,
1954, Subspecies and clines: The naming of geographically variant populations, or what is all the shouting about? Syst. Zool. 3: 113-121.

* Hubbell, T.H.,
1956, Some aspects of geographic variation in insects. Ann. Rev. Ent. 1:71-88.

* Hubbs, C.L.,
1943, Criteria for subspecies, species and genera, as determined by researches on fishes. Ann. N.Y. Acad. Sci. 44(2):109-121.

* Hubbs, C.L.,
1955, Hybridization between fish species in nature. Syst. Zool. 4(1):1-20.

Huxley, J.S.,
1938, Clines: An auxiliary taxonomic principle. Nature 142(3857): 219-220.

* Huxley, J.S.,
1939a, Clines: An auxiliary method in taxonomy. Bijdragen tot de Dierkunde, Leiden 27:491-520 (Feestnumber 1838-1938).

Huxley, J.S.,
1939b, in A discussion of subspecies and variation, by the Association for the study of systematics. Proc. Linn. Soc. Lond. 151:105-106.

* Huxley, J.S.,
1940, Towards the new systematics. Pages 1-46 in J.S. Huxley, editor, The New Systematics. Oxford: Oxford University Press.

* Huxley, J.S.,
1942, Evolution: The Modern Synthesis. London: George Allen and Unwin, Ltd.

* Huxley, J.S., A.C. Hardy, and E.B. Ford,
1954, Evolution as a Process. London: George Allen and Unwin, Ltd.

I.B.M. (International Business Machines)
1959, Random Number Generation and Testing. I.B.M. Data Processing Techniques C20-8011-0.

* I.C.E.S. (International Council for the Exploration of the Sea)
1969, (In press, 1971), Special Meeting on Identification of Fish Stocks, Dublin, September, 1969. Charlutealund Slot, Denmark, Denmark. (Notes kindly supplied by J.L. Jamie).

* Ingles, L.G., and N.J. Biglione,
1952, The contiguity of the ranges of two subspecies of Pocket Golphers. Evolution 6(2):204-207.

* Ingoldby, C.M.,
1927, Some notes on the African squirrels of the genus Heliosciurus. Proc. Zool. Soc. Lond. 1927 paper (30):471-487.

T* Jain, S.K., and A.D. Bradshaw,
1966, Evolutionary divergence among closely adjacent plant populations. I. The evidence and its theoretical analysis. Heredity 21(3):407-441.

* Jaeger, R.G.,
1971, Competitive exclusion as a factor influencing the distribution of two species of terrestrial salamanders. Ecology 52(4): 632-637.

* James, F.C.,
1970, Geographic size variation in birds and its relationship to climate. Ecology 51(3):365-390.

* Jameson, D.L., W. Taylor, and J. Mountjoy,
1970, Metabolic and morphological adaptation to heterogeneous environments of the Pacific Tree Toad, Hyla regilla. Evolution 24(1):75-89.

Jenkins, D.W., and C.C. Hassett,
1951, Dispersal and flight range of subarctic mosquitos marked with radiophosphorous. Canad. J. Zool. 29(3):178-187.

* Jepson, G.L., G.G. Simpson, and E. Mayr,
1949, Genetics, Paleontology and Evolution. New Jersey: Princeton University Press. (Reprinted 1963 by Atheneum Press.)

* John, B., and G.M. Hewitt,
1965, The B-chromosome system of Myrmeleotettix maculatus. II. The statics. Chromosoma (Berl.) 17(2):121-138.

Johnson, C.G.,
1969, Migration and Dispersal of Insects by Flight. London: Methuen and Co.

* Johnson, M.S.,
1971, Adaptive lactate dehydrogenase variation in the crested blenny, Anoplarchus. Heredity 27(2):205-226.

* Johnson, N.K.,
1969, Review, Three papers on variation in Flickers (Colaptes) by Lester L. Short, Jr. Wilson Bulletin 81(2):225-230.

* Johnston, R.F.,
1956a, Population structure in Salt Marsh song sparrows, Part I. Environment and seasonal cycle. Condor 58(1):24-44.

* Johnston, R.F.,
1956b, Population structure in Salt Marsh song sparrows, Part II. Density, age structure and maintenance. Condor 58(4):254

* Jones, D.A.,
1972, On the polymorphism of Cyanogenesis in Lotus corniculatus L., IV: The Netherlands. Genetica 43:394-406.

* Jones, J.S.,
1972, Ph.D. Thesis, University of Edinburgh, Department of Zoology, Edinburgh, Scotland.

* Joshi, B.C., and S.K. Jain,
1964, Clinal variation in natural populations of Justica simplex. Amer. Nat. 96(899):123-125.

Kalela, O., T. Koponen, E.A. Lind, U. Skaren, and J. Tast,
1961, Seasonal change of habitat in the Norwegian Lemming (Lemmus lemmus L.). Annales. Acad. Scient. Fennicae. Helsinki, Series A, Section IV Biologia 55:1-72.

Kaplan, A.,
1964, The Conduct of Inquiry. San Francisco: W.H. Freeman and Co.

Kerster, H.W.,
1964, Neighborhood size in the rusty lizard, Sceloporus olivaceus. Evolution 18(3):445-457.

Kettle, D.S.,
1951, The spatial distribution of Culicoides impunctatus Goet. under woodland and moorland conditions and its flight range through woodland. Bull. Ent. Res. 42:239-291.

* Kettlewell, H.B.D.,
1961a, Geographical melanism in the Lepidoptera. Heredity 16(4): 393-402.

* Kettlewell, H.B.D.,
1961b, Selection experiments on melanisms in Amathes glareosa Esp. Lepidoptera. Heredity 16(4):414

* Kettlewell, H.B.D., and R.J. Berry,
1961, The study of a cline, Amathes glareosa and its melanic f. edda Staud. (Lep.) in Shetland. Heredity 16(4):403-414.

* Kettlewell, H.B.D., and R.J. Berry,
1969, Gene flow in a cline. Amathes glareosa Esp. and its melanic f. edda Staud (Lep.) in Shetland. Heredity 24(1):1-14.

* Kettlewell, H.B.D., R.J. Berry, C.J. Cadbury, and G.C. Phillips,
1969, Differences in behaviour, dominance and survival within a cline. Amathes glareosa Esp. (Lep.) and its melanic f. edda Staud. in Shetland. Heredity 24(1):15-25.

* Key, K.H.L.,
1968, The Concept of Stasipatric Speciation. Syst. Zool. 17:14-22.

T Kimura, M.,
1958, A gene frequency cline determined by selection and migration. Ann. Report. Nat. Inst. Genet. Mishima-shi, Japan (9):84-86.

T Kimura, M., and G.H. Weiss,
1964, The stepping stone model of population structure and the decrease of genetic correlation with distance. Genetics 49:561-576.

T Kimura, M., and T. Maruyma,
1971, Pattern of neutral polymorphism in a geographically structured population. Genet. Res. Cambridge (England) 18:125-131.

* Kinsey, A.C.,
1937, An evolutionary analysis of insular and continental species. Proc. Nat. Acad. Sci. U.S. 23(1):5-11.

* Kiriakoff, S.G.,
1947, Le Cline, une nouvelle categorie systematique intraspecifique. Bull. et Ann. Soc. Ent. Belgique 83:130-140.

* Kluijver, H.N.,
1951, The population ecology of the Great Tit, Parus Major L. Ardea 39:1-135.

* Koehn, R.K.,
1969, Esterase heterogeneity: Dynamics of a polymorphism. Science 163(3870):943-944.

* Komai, T.,
1954, An actual instance of microevolution observed in an insect population. Proc. Japanese Acad. 30(10):970-975.

* Komai, T., M. Chino, and Y. Hosino,
1950, Contribution to the evolutionary genetics of the lady-beetle Harmonia, I: Geographic and temporal variation in the relative frequencies of the elytral pattern types and in frequency of the elytral ridge. Genetics 35:589-601.

* Komai, T., and Y. Hosino,
1951, Contribution to the evolutionary genetics of the lady-beetle Harmonia, II: Microgeographic variation. Genetics 36:382-390.

Kuhn, D.T.,
1971, Coadaptation of the Payne inversion with a previously unrelated genetic background in Drosophila melanogaster. Evolution 25(1):207-213.

Labine, P.A.,
1964, Population biology of the butterfly Euphydras editha. I. Barriers to multiple insemination. Evolution 18(2):335-336.

Labine, P.A.,
1966, The population biology of the butterfly Euphydras editha. IV. Sperm precedence-a preliminary report. Evolution 20:580-586.

Lack, D.,
1947, Darwin's Finches. Cambridge (England): Cambridge U. Press.

Lack, D.,
1949, The Significance of Ecological Isolation. Pages 299-314 in G.L. Jepson, G.G. Simpson and E. Mayr, Genetics, Paleontology and Evolution. Princeton (U.S.A.): Princeton U. Press.

Lamb, K.P., E. Hassan, and D.R. Scoter,
1971, Dispersal of Scandium-46 labelled Pantorhytes weevils in Papuan Cacao plantations. Ecology 52(1):178-182.

* Lamotte, M.,
1951, Recherches sur la structure génétique des populations naturelles de Cepaea nemoralis (L.). Bull. Biol. Suppl. 35:1-239.

Lansborough Thompson, A.,
1942, Bird Migration (Second edition). London: Witherby.

* Lees, D.R.,
1971, The distribution of melanism in the Pale Brindled Beauty moth, Phigalia pedaria in Great Britain. Chapter 8 in E.R. Creed, editor, Ecological Genetics and Evolution. Oxford: Blackwells Scientific Publications.

Lerner, I.M.,
1954, Genetic Homeostasis. Edinburgh: Oliver and Boyd.

Levin, B.R., M.L. Petras, and D.I. Rasmussen,
1969, The effect of migration on the maintenance of a lethal polymorphism in the house mouse. Amer. Nat. 103(934):647-661.

Levin, D.A., and H.W. Kerster,
1968, Local gene dispersal in Phlox. Evolution 22(1):130-139.

Levin, D.A., and H.W. Kerster,
1971, Neighborhood structure in plants under diverse reproductive methods. Amer. Nat. 105(944):345-354.

Levin, D.A., H.W. Kerster, and M. Niedzlek,
1970, Pollinator flight dispersion and its effect on pollen flow. Evolution 25:113-118.

T Levene, H.,
1953, Genetic equilibrium when more than one ecological niche is available. Amer. Nat. 87:331-333.

T Levins, R.,
1962, Theory of fitness in a Heterogeneous Environment. I. The fitness set and adaptive function. Amer. Nat. 96(891):361-373.

T Levins, R.,
1963, Theory of fitness in a Heterogeneous Environment. II. Developmental flexibility and nich selection. Amer. Nat. 97(893): 75-90.

T Levins, R., and R. MacArthur,
1966, The maintenance of genetic polymorphism in a spatially heterogeneous environment: Variations on a theme by Howard Levene. Amer. Nat. 100(918):585-589.

* Lewis, H.,
1953, The mechanisms of evolution in the genus Clarkia. Evolution 7:1-20.

Lewontin, R.L.,
1962, Interdemic selection controlling a polymorphism in the house mouse. Amer. Nat. 96(887):65-78.

Lewontin, R.L., and L.C. Dunn,
1960, The evolutionary dynamics of a polymorphism in the house mouse. Genetics 45:705-722.

* Ledig, F.T., and J.H. Fryer,
1972, A pocket of variability in Pinus rigida. Evolution 26(2): 259-266.

* Lidicker, W.A.,
1962, The nature of subspecies boundaries in desert rodents and its implications for subspecies taxonomy. Syst. Zool. 11(4): 160-171.

Lidicker, W.Z.,
1972, Talk on mice in enclosures at University of Edinburgh, Department of Zoology.

Lindsley, D.L., and G.H. Grell,
1968, Genetic variation in Drosophila melanogaster. Carnegie Institute of Washington Publication (627):1-472.

* Lindroth, C.H.,
1949, Die fennoskandischan Carabidae III. Allgemeiner Teil. Zugleich eine biogeographisch Prinzipdiskussion. Goteborgs Kugl. Vetenskapsoch Vitterhetes-Samholles Handlingar 6, Ser. B 4:1-911, Stockholm.

* Lindroth, C.H.,
1953, Some attempts toward experimental zoogeography. Ecology 34(4):657-666.

Lindroth, C.H.,
1971, Biological investigations on the new volcanic island Surtsey, Iceland. Pages 65-69 of Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Biol. St., Wijster, Nov. 3-5, 1969, P.J. Den Boer, editor. Misc. Papers 8(1971) Landbouwhoge-school, Waginingen, The Netherlands.

* Linsdale, J.M.,
1938, Bird life in Nevada with reference to modification in structure and behavior. Condor 40(4):173-180.

* Littlejohn, M.J., and R.S. Oldham,
1968, Rana pipiens complex: Mating call structure and taxonomy. Science 162(3857):1003-1005.

* Littlejohn, M.J., G.F. Watson, and J.J. Laftis-Hills,
1971, Contact hybridization in the Crinia laevis complex (Anura: Leptodactylidae). Aust. J. Zool. 19:85-100.

T* Livingstone, F.B.,
1969, An analysis of the A. B. O. blood group clines in Europe. Amer. J. Physical Anthropology 31(1):1-10.

Lloyd, M.,
1967, "Mean Crowding" J. Anim. Ecol. 36(1):1-30.

* Lotsy, J.P.
1916, Evolution By Means of Hybridization. The Hague: Martinus Nijhoff.

* Lucas, A.M.,
1969, Clinal variation in pattern and color in coastal populations of the butterfly Tisiphone abeona (Donovan) (Lepidoptera: Satyrinae). Aust. J. Zool. 17:37-48.

* Marshall, J.T., Jr.,
1948a, Ecologic races of song sparrows in the San Francisco bay region. Condor 50(5):193-215.

* Marshall, J.T., Jr.,
1948b, Ecologic races of song sparrows in the San Francisco bay region. Condor 50(6):233-256.

Mather, K.,
1941, Variation and selection of polygenic characters. J. Genet. 41(2):159-193.

Matthews, G.V.T.,
1966, Book reviews on salmon orientation: A book by Hasler and a thesis by Croot. Anim. Beh. 14(4):593-594. (A.D. Hasler, 1966, Underwater Guideposts, U. of Wisconsin press: C. Groot, 1965, On the orientation of young sockeye salmon (Oncorhynchos nerka) during their seasonal migration. Beh. Suppl. 14:1-198.)

* Maynard-Smith, J.,
1958, The Theory of Evolution. Harmondsworth: Penguin Books, Ltd.

T Maynard-Smith, J.,
1966, Sympatric speciation. Amer. Nat. 100(916):637-650.

T Maynard-Smith, J.,
1970, Genetic polymorphism in a varied environment. Amer. Nat. 100(938):487-490.

* Mayr, E.,
1940, Speciation phenomena in birds. Amer. Nat. 74(752):249-278.

* Mayr, E.,
1942, Systematics and the Origin of Species, From the Viewpoint of a Zoologist. New York: Columbia University Press.

* Mayr, E.,
1947, Ecological factors in speciation. Evolution 1(4):263-288.

* Mayr, E.,
1949, Speciation and Systematics. Pages 281-298 in G.L. Jepson, G.G. Simpson and E. Mayr, Genetics, Paleontology and Evolution. Princeton: Princeton University Press.

Mayr, E.,
1954a, Change of Genetic Environment and Evolution. Pages 157-180 in J.S. Huxley, A.C. Hardy and E.B. Ford, editors, Evolution as a Process. London: George Allen and Unwin, Ltd.

* Mayr, E.,
1954b, Geographic speciation in tropical Echinoids. Evolution 8(1):1-18.

Mayr, E.,
1955, Integration of genotypes: Synthesis. Cold Spring Harbor Symposium Quant. Biol 2

* Mayr, E.,
1963, Animal Species and Evolution. Cambridge (U.S.A.): The Belknap Press.

* Mayr, E., and E. Stresseman,
1950, Polymorphism in the Chat genus Oenanthe (Aves). Evolution 4(4):291-300.

* Mayr, E., and C. Vaurie,
1948, Evolution in the Family Dicruridae (birds). Evolution 2(3): 238-265.

Meijer, J.,
1971, Immigration of Arthropods into the new Lannerszee Polder. Pages 53-64 of Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Biol. St., Wijster, Nov. 3-5, 1969, P.J. Den Boer, editor. Misc. Papers 8(1971) Landbouwhogeschool, Waginingen, The Netherlands.

* Meise, W.,
1928a, Die Verbreitung der Aaskrahe (Formenkreise Corvus corone L.). J. Ornith. 76:1-203.

* Meise, W.,
1928b, Rassenkreuzungen an den Arealgrenzen. Verh. d. Deut. Zool. Ges. 32:96-104.

Merchant, H.,
1972, Estimated population size and home range of the salamanders Plethodon jordani and Plethodon glutinosus. J. Wash. Acad. Sci. 62:248-257.

Merrell, D.J.,
1967, A comparison of the estimated size and the "effective size" of breeding populations of the Leopard frog Rana pipiens. Evolution 22:274-283.

* Merrell, D.J.,
1970, Migration and gene dispersal in Rana pipiens. American Zoologist 10(1):47-52.

* Merritt, R.B.,
1972, Geographic distribution and enzymatic properties of L.D.H. enzymes in the fathead minnow, Pinephales promelas. Amer. Nat. 106(948):173-184.

* Michener, C.D.,
1947, A Character Analysis of a Solitary Bee, Hoplitis albifrons (Hymenoptera: Megachilidae). Evolution 1(3):172-185.

* Miller, A.H.,
1931, Systematic revision and natal history of the American Shrikes (Lanius). Univ. Calif. Publ. Zool. 38(2):11-242.

* Miller, A.H.,
1941, Speciation in the avian genus Junco. Univ. Calif. Publ. Zool. 44(3):173-434.

Miller, A.H.,
1947, Panmixia and population size with reference to birds. Evolution 1(3):186-190.

* Miller, A.H.,
1949, Some concepts of hybridization and intergradation in wild populations of birds. Auk. 66(4):338-342.

* Miller, A.H.,
1951, An analysis of the distribution of the birds of California. Univ. Calif. Publ. Zool. 50(6):531-644.

* Miller, A.H.,
1956, Ecologic factors that accelerate formation of races and species of terrestrial vertebrates. Evolution 10(3):262-277.

* Miller, A.H., and T.T. McCabe,
1935, Racial differentiation in Passerella (Melospiza) lincolni. Condor 37(3):144-160.

* Miller, R.R., and C.L. Hubbs,
1969, Systematics of Gasterosteus aculeatus, with particular reference to intergradation and introgression along the Pacific Coast of North America: A commentary on a recent contribution. Copeia 1969(1):52-69.

Millicent, E., and J.M. Thoday,
1961, Effects of disruptive selection IV, gene flow and divergence. Heredity 16:199-217.

Mitchell, R.,
1970, An analysis of dispersal in mites. Amer. Nat. 104(938): 425-431.

Mook, J.H.,
1971, Observations on the colonization of the new Ijsselmeer-Polders. Pages 13-31 of Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Biol. St., Wijster, Nov. 3-5, 1969. P.J. Den Boer, editor. Misc. Papers 8(1971) Landbouwhogeschool, Waginingen, The Netherlands.

* Moore, J.A.,
1944, Geographic races in Rana pipiens Schreber of eastern North America. Bull. Amer. Mus. Nat. Hist. 82:349-369.

* Moore, J.A.,
1946, Incipient intraspecific isolating mechanisms in Rana pipiens. Genetics 31:304-326.

* Moore, J.A.,
1949a, Patterns of evolution in the Genus Rana, in G.L. Jepson, G.G. Simpson, and E. Mayr, editors, Genetics, Palaeontology and Evolution. Princeton: Princeton University Press, pages 315-338.

* Moore, J.A.,
1949b, Geographical variation of adaptive characters in Rana pipiens Schreber. Evolution 3(1):1-24.

* Moore, J.A.,
1950, Further studies on Rana pipiens racial hybrids. Amer. Nat. 84:247-254.

* Moore, J.A.,
1954, Geographic and genetic isolation in Australian amphibia. Amer. Nat. 88(839):65-74.

* Moore, J.A.,
1957, An embryologist's view of the species concept. Pages 325-338 of E. Mayr, editor, The Species Problem, Publ. (50), Washington D.C.: American Association for the Advancement of Science.

Morista, M.,
1959, Measuring the dispersal of individuals and analysis of the distributional patterns. Mem. Fac. Sci. Kyushu Univ. Ser. E, 2:215-235.

* Muller, C.H.,
1952, Ecological control of hybridization in Quercus: A factor in the mechanism of evolution. Evolution 6(2):147-161.

* Murie, A.,
1933, The ecological relationship of two subspecies of Peromyscus in the Glacier Park region, Montana. Occ. Pap. Mus. Zool. Univ. Mich. (270):1-17.

* McCullough, J.M., and E. Giles,
1970, Human cerumen types in Mexico and New Guinea: A humidity-related polymorphism in "Mongoloid" peoples. Nature 226(5244): 460-462.

McFarquhar, A.M., and F.W. Robertson,
1963, The lack of evidence for co-adaptation in crosses between populations of Drosophila subobscura Coll. Genet. Res. Camb. 4: 104-131.

* McNeilly, T.,
1968 Evolution in closely adjacent plant populations. III. Agrostis tenuis on a small copper mine. Heredity 23:99-108.

* McNeilly, T., and J. Antonovics,
1968, Evolution in closely adjacent plant populations. IV. Barriers to gene flow. Heredity 23(2):205-218.

Narise, T.,
1958, in Sackai et al., 1958.

Narise, T.,
1968, Migration and competition in Drosophila. I. Competition between wild and vestigial strains of Drosophila melanogaster. Evolution 22(2):301-306.

* New, J.,
1958, A population study of Spergula arvensis. I. Two Clines and their significance. Ann. Bot. N.S. 22(88):457-477.

Nichols, D.,
1962, Differential selection in populations of a Heart Urchin. In D. Nichols, editor, Taxonomy and Geography, Syst. Assoc. Lond. Publ. (4):105-118.

O'Donald, P.,
1960, Assortative mating in a population in which 2 alleles are segregating. Heredity 15:389-396.

* O'Gower, A.K., and P.I. Nicol,
1968, A latitudinal cline of Haemoglobin in a bivalve mollusc. Heredity 23:485-492.

* Parkes, K.C.,
1955, Sympatry, allopatry and the subspecies in birds. Syst. Zool. 4(1):35-40.

Palmen, E.,
1971, Translocations of insects in the archipelago of the southern coast of Finland. Pages 71-76 in Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Biol. Station, Wijster, Nov. 3-5, 1969, P. J. Den Boer, editor. Misc. Papers 8(1971) Landbouwhogeschool, Wageningen, The Netherlands.

Parslow, J.L.F.,
1967, Changes in status among breeding birds in Britain and Ireland. British Birds 60(5):177-202.

Parsons, P.A.,
1963, Migration as a factor in natural selection. Genetica 33: 184-206.

* Peabody, F.E., and J.M. Savage,
Evolution of a Coast Range Corridor in California and its Effects on the Origin and Dispersal of living Amphibians and Reptiles. In C.L. Hubbs, editor, Zoogeography, Publ. (51), Amer. Assoc. Adv. Sci., 1958, pages 159-186.

Pearson, K.,
1897, The Chances of Death, and Other Studies in Evolution. London: Edward Arnold (two volumes).

Perrins, C.M.,
1965, Population fluctuations and clutch size in the great tit, Parus major L. J. Anim. Ecol. 34:601-647.

Perrins, C.M.,
1966, Survival of young Manx Shearwaters, Puffinius puffinus in relation to presumed date of hatching. Ibis 108(1):132-135.

Petit, C.,
1951, Le role de l'isolement sexuel dans l'evolution des populations de Drosophila melanogaster. Bull. Biol. France Belgique 85:392-418.

Petit, C.,
1958, Le determinism génétique et psycho-physiologique de la competition sexuelle chez Drosophila melanogaster. Bull. Biol. France Belgique.

Petras, M.L.,
1967, Studies of natural population of Mus. I. Biochemical polymorphisms and their bearing on breeding structure. Evolution 21:259-274.

Pielou, E.C.,
1969, An Introduction to Mathematical Ecology. Wiley-Interscience, New York.

* Pitelka, F.A.,
1951, Speciation and ecologic distribution in American jays of the genus Aphelocoma. Univ. Calif. Publ. Zool. 50(3):195-464.

* Platt, A.P., and L.P. Brower,
1968, Mimetic versus disruptive coloration in intergrading populations of Limenitis arthemis and astyanax butterflies. Evolution 22 (4):699-718.

T Prout, T.,
1968, Sufficient conditions for multiple niche polymorphism. Amer. Nat. 102:494-496.

Rand, A.L.,
1948, Glaciation, a factor in speciation. Evolution 2(4):314-321.

Rasmussen, D.I.,
1964, Blood group polymorphism and inbreeding in natural populations of the deer mouse Peromyschs maniculatus, Evolution 18(2): 219-229.

Reimer, J.D. and M.L. Petras,
1967, Breeding structure in the house mouse, Mus musculus in population cages. J. Mammal. 48:87-99.

* Remington, C.L.,
1968, Suture-zones of hybrid interaction between recently joined Biotas, Evolutionary Biology 2(8):321-428.

* Rensch, B,
1938, Some problems of geographical variation and species formation. Proc. Linn. Soc. Lond. 150:275-285.

* Rensch, B.,
1959, Evolution above the Species Level,London: Methuen and Co., Ltd.

Richardson, R.N.,
1969, Migration and Enzyme Polymorphisms in Natural Populations of Drosophila. Japan J. Genetics. 44(suppl. I):172-179.

Richter, C.J.J.,
1971, Some aspects of aerial dispersal in different populations of Wolf Spiders with special reference to Pardosa amentata (Araneae, Lycosidae) p.81-91 of Dispersal and Dispersal Power in Carabid Beetles, a symposium held at the Biological Station, Wijster, Nov. 3-5, 1969, P.J. Den Boer, Editor, Misc. Papers 8(1971), Landbouhogescholl, Waginigen, The Netherlands.

* Roberts, M. R. and H. Lewis,
1955, Subspeciation in Clarkia biloba. Evolution 9(4): 445-454.

Rodenheffer, I.A.,
1940, The movement of marked fish in Douglas Lake, Michigan. Pap. Mich.Acad. Sci.Arts Lett. 26(2):265-280.

T Rohlf, F.J., and G.D. Schnell,
1971, An investigation of the isolation by distance model. Amer. Nat. 105(944):295-324.

* Ruibal, R.,
1955, A study of altitudinal races in Rana pipiens, Evolution 9 (3):322-338.

Sakai, K., T. Narise, Y. Hiraizumi, and S. Iyama,
1958, Studies on migration in Drosophila melanogaster, Evolution 12(1):93-101.

Salomonsen, F.,
1955, The evolutionary significance of bird-migration. Det. Kongelige Danske Videnskabernes Selskab Biologiske Meddeleser 22(6): 1-62.

* Sammalisto, L.,
1956, Secondary intergradation of the blue-headed and grey-headed wagtails (Motatilla flava flava L. and Motacilla flava thumbergi Billb.) in south Finland Ornis Fennica 33(1): 1-19.

* Sammalisto, L.,
1957, The effect of the Woodland-open-peatland edge on some peatland birds in south Finland. Ornis Fennica 34(3):81-89.

* Sammalisto, L.,
1958, Interracial hybridization as an adaptation mechanism in the Fennoscandian Yellow Wagtail (Motacilla flava L.) population. Annales Academiae Scientarum Fennicae, Helsinki Series A, Section IV Biologia 41:1-46.

* Sammeta, K.P.V. and R. Levins.
1970, Genetics and Ecology. Ann. Rev. Genet. 4(3017):469-488.

Schneider, F.,
1962, Dispersal and Migration. Ann. Rev. Ent. 7:233-242.

* Schopf, T.J.M. and J.L. Gooch,
1971, Gene frequencies in a marine Ectoproct: cline in natural populations related to sea temperatures. Evolution 25(2):286-289.

T* Seiger, M.B. and R.D. Dixon,
1969, A computer simulation study of the effects of two behavioural traits in Genetic structure of semi-isolated populations. Evolution 24:90-97.

* Selander, R.K.,
1970, Behavior and genetic variation in natural populations, Amer. Zoologist 10(1): 53-66.

* Sealander, R.K., M.H. Smith, S.Y. Yang, and W.E. Johnson.
1971, Biochemical polymorphism and systematics in the genus Peromyscus, I, Variation in the Old-field mouse, Peromyscus polionotus. Studies in Genetics (Austin)6(7103): 49-90.

* Short, L.,
1965, Hybridization in the Flickers (Colaptes) of North America. Bull. Amer. Mus. Nat. Hist. 129:307-428.

* Sibley, C.G.,
1950, Species formation in the Red-eyed Towhees of Mexico. Univ. Calif. Publ. Zool. 50(2):109-194.

* Sibley, C.G.,
1954, Subspecies and Clines: The Contribution of Avian Taxonomy, Syst. Zool. 3:105-110, 125.

Sibley, C.G.,
1961, Hybridization and Isolating mechanisms in, W. F. Blair, Editor, Vertebrate Speciation, p. 68-88. Austin: University of Texas Press.

Simpson, G.G.,
1952, Probabilities of dispersal in geological time. Bull. Amer. Mus. Nat. Hist. 99:163-176.

* Simpson, G.G.,
1953, The Major Features of Evolution, Columbia University Press, New York.

T Slatkin, M.,
1971, Ph.D. Thesis, Harvard Univ. Biology Dept., Cambridge, U.S.A.

Smith, H.M.,
1965, More Evolutionary Terms. Syst. Zool. 14:57-58.

Smith, H.M.,
1967, The perspective of subspecies in animal evolution. The Biologist 49:43-51.

Smith, H.M.,
1969, Parapatry: Sympatry or Allopatry? Syst. Zool. 18(2): 254-255.

* Snaydon, R.W.,
1962, Microdistribution of Trifolium repens L. and its relation to soil factors. J. Ecology 50(1):133-144.

* Snaydon, R.W.,
1970, Rapid population differentiation in a mosaic environment, I, the response of Anthoxanthum odoratum populations to soils. Evolution 24(2):257-269.

* Snow, D.W.,
1954, Trends in geographical variation in Palaearctic members of the genus Parus. Evolution 8(1):19-28.

Sokal, R.R. and T.J. Crovello,
1970, The Biological Species Concept, a Critical Evaluation. Amer. Nat. 104:127-153.

* Southern, H.N.,
1939, The status and problem of the Bridled Guillemot. Proc. Zool. Soc. Lond. A(3):31-41.

* Southern, H.N.,
1951, Change in status of the Bridled Guillemot after 10 years. Proc. Zool. Soc. Lond. 121:657-671.

* Southern, H.N.,
1962, Survey of the Bridled Guillemots, 1959-1960, Proc. Zool. Soc. Lond. A 138:455-472.

* Southern, H.N., R. Carrick, and W.G. Potter,
1965, The Natural History of a Population of Guillemots (Uria aalge Pont.) J. Anim. Ecol. 34:649-665.

* Southern, H.N.,
1966, Distribution of Bridled Guillemots in East Scotland over 8 years. J. Anim. Ecol. 35:1-11.

* Southern, H.N., and E.C.R. Reeve,
1941, Quantitative studies in the geographical variation of birds-The common Guillemot (Uria aalge Pont.). Proc. Zool. Soc. Lond. 111 (16):255-276.

Southwood, T.R.E.,
1962, Migration of terrestrial arthropods in relation to habitat. Biol. Rev. 37:171-214.

Southwood, T.R.E.,
1966, Ecological Methods. London. Methuen and Co. Ltd.

* Spiess, E. B.,
1950,Experimental populations of Drosophila persimilis from an altitudinal transect of the Sierra Nevada. Evolution 4(1):14-33.

Spurway, H.,
1954, Communication to J.B.S. Haldane printed in J.B.S. Haldane, 1954, The Statics of Evolution, pp. 109-121, in J.S. Huxley, A.C. Hardy and E.B.Ford, Evolution as a Process, London: George Allen and Unwin, Ltd.

* Stalker, H.D. and H.L. Carson,
1947, Morphological variation in natural populations of Drosophila robusta Sturtevant. Evolution 1(4):237-248.

* Stalker, H.D. and H.L. Carson,
1948, An altitudinal transect of Drosophila robusta Sturtevant. Evolution 2(4):295-305.

* Stebbins, G.L.,
1950, Variation and Evolution in Plants, New York: Columbia Univ. Press.

* Stebbins, R.C.,
1949, Speciation in salamanders of the Plethodontid genus Ensatina. Univ. Calif. Publ.Zool. 48(6):377-526.

* Stebbins, R.C.,
1951, Amphibians of Western North America. Berkeley: University of California Press.

* Stebbins, R.C.,
1954, Amphibians and Reptiles of Western North America. New York: McGraw-Hill.

* Stebbins, R.C.,
1966, A field guide to the Western Reptiles and Amphibians. Boston (U.S.A.): Houghton Mifflin Co.

* Storer, R.W.,
1952, A comparison of variation, behaviour, and evolution in the sea bird genera Uria and Cepphus. U. Calif. Publ. Zool. 52(2): 121-222.

Streams, F.A. and D. Pimentel,
1961, Effects of immigration on the evolution of populations. Amer. Nat. 95(883):201-210.

* Sumner, F.B.,
1926, An analysis of Geographia variation in mice of the Peromyscus polionotus group of Florida and Alabama. J. Mammal. 7(3):149-184.

* Sumner, F. B.,
1929a, The analysis of a concrete case of intergradation between two subspecies. Proc. Nat. Acad. Sci. U.S. 15(2):110-120.

* Sumner, F. B.,
1929b, The analysis of a concrete case of integradation between two subspecies. II. Additional data and interpretation. Proc. Nat. Acad. Sci. U.S. 15(6):481-493.

* Sumner, F. B.,
1932, Genetic, distributional, and evolutionary studies on the subspecies of deer mice (Peromyscus). Bibliolgraphica Genetica 9:1-106.

* Swarth, H.S.,
1920, Revision of the avian genus Passerella, with special reference to the distribution and migration of the races in California. Univ. Calif. Publ. Zool. 21(4):75-224.

Tamarin, R.H., and C. J. Krebs,
1969, Microtus population biology. II. Genetic changes at the trasferrin locus in fluctuating populations of two vole species. Evolution 23(2):183-211.

Tantawy A.O, abd G.S. Mallah,
1961, Studies on natural populations of Drosophila. I. Heat resistance and geographical variation in Drosophila melanogaster and D. simulans. Evolution 15(1):1-14.

Terborgh, J.,
1971, Distribution on Environmental Gradients: Theory and a preliminary interpretation of distributional patterns in the Avifauna of the Cordillera Vilcabamba, Peru. Ecology 52(1):23-40.

* Test, A. R.,
1946, Speciation in limpets of the genus Acmaea. Contr.Lab.Vert.Biol. Univ.Mich.(31):1-23.

Thienemann, A.,
1950, Verbreitungsgeschichte der Sußwassertierwelt Europas; Versuch einer historichen Tiergeographie der europaischen Binnengewasser. Die Binnengewasser XVIII 809 pp.

Thoday, J.M.,
1958, Effects of disruptive selection: the experimental production of a polymorphic population. Nature 181:1124-1125.

Thoday, J.M., and T. B. Boam,
1959, Effects of disruptive selection II, polymorphism and divergence without isolation. Hereidty 13:204-218.

Thoday, J.M. and J.B. Gibson,
1962, Isolation by disruptive selection. Nature 193(4821):1164-1166.

* Thompson, D.H.,
1930, Variation in fishes as a function of distance. Trans. Ill. State Acad. Sci. 23:276-281.

* Thorpe, W.H.,
1930, Biological races in insects and allied groups. Biol.Rev. 5(3): 177-212.

Thorpe, W. H.,
1931, Biological races in insects and their significance in Evolution. Ann.Appl.Biol. 18:406-414.

* Thrurow, G.R.,
1961, A salamander color variant associated with glacial boundaries. Evolution 15(3):281-287.

* Timofeeff-Ressovsky, N. W.,
1940, Mutations and Geographic Variation, pp. 73-136 in J.S. Huxley, Editor, The New Systematics ,Oxford Univ. Press.

* Turner, J.R.G.,
1971, Studies of Müllerian mimicry and its evolution in Burnet moths and Heliconid butterflies, pp. 224-260 in E.R. Creed, Editor, Ecological Genetics and Evolution, Oxford: Blackwells Scientific Publications.

Twitty, V.C.,
1961, Experiments on Homing Behavior and Speciation in Taricha. in W.F. Blair, editor, Vertebrate Speciation, pp. 415-459, University of Texas Press, Austin.

Twitty, V. C.,
1966, Of Scientists and Salamanders. W.H. Freeman and Co., San Francisco.

* Udvardy, M.,
1969, Dynamic Zoogeography. New York: VanNostrand-Reinhold Co.

* Ursin, E.
1952, Occurrence of Voles, Mice and Rats (Muridae) in Denmark. With a special note on a zone of intergradation between two subspecies of the house mouse (Mus musculus L.)Vidensk. Medd. Copenhagen. 114:217-244.

Uvarov, B. P.,
1931, Insects and Climate. Trans. Entomol. Soc. Lond. 79(1):;-247.

Van Tyne, J, and A.J. Berger,
1961, Fundamentals of Ornithology. John Wiley and Sons, Inc. New York.

* Vaughan, T.A.,
1967, Two parapatric species of Pocket Gophers. Evolution 21(1): 148-158.

Vetukiv, M.,
1953, Viability of hybrids between local populations of Drosophila pseudoobscura. Proc.Nat.Acad.Sci., U.S. 39:30-34.

Vetukiv, M.,
1954, Integration of the Genotype in local populations of three species of Drosophila. Evolution 8:241-251.

Vetukiv, M.,
1956, Fecundity of hybrids between geographic populations of Drosophila pseudoobscura. Evolution 10:139-146.

* Voipio, P.,
1950, Evolution at the population level with special reference to game animals and practical game management. Paper on Game Research, Helsinki 5:1-176.

* Voipio, P.,
1952, Subspecies Boundaries and Geodynamics of Populations in Mammals and Birds. Ann. Zool. Soc. "Vanamo". Helsinki. 14(4):1-32.

* Voipio, P.,
1956, The Biological Zonation of Finland as reflected in Zootaxonomy. Ann. Zool.Soc. "Vanamo", Helsinki. 18(3):1-36.

* Voipio, P.,
1957, Über die Polymorphie von Sciurus vulgaris L. in Finland. Ann. Zool.Soc. "Vanamo", Helsinki. 18(7):1-24.

* Voipio, P.,
1970, Polymorphism and regional differentiation in the red squirrel (Scirurus vulgaris L.). J. Ann. Zool. Fennici 7:210-215.

Wadley, F.M. and D.O. Wolfenbarger,
1944, Regression of insect density on distance from center of dispersion as shown by a study of the smaller European Bark Beetle. J. Agric. Res. 59(7):299-308.

Wallace, B.,
1955, Inter-population hybrids in Drosophila melanogaster. Evolution 9(3):302-316.

Wallace, B.,
1966, On the dispersal of Drosophila. Amer.Nat. 100(916):551-563.

Wallace, B.,
1966, Distance and the Allelism of lethals in a tropical population of Drosophila melanogaster. Amer.Nat. 100(916):565-578.

Wallace, B.,
1968a, On the dispersal of Drosophila. Amer. Nat. 102(923):85-87.

* Wallace, B.,
1968b, Topics in Population Genetics. New York: W.W. Norton and Co., Inc.

Wallace, B.,
1970, Observations on the microdispersion of Drosophila melanogaster in M.K. Hecht and W.C. Steere, Editors, Essays in Evolution and Genetics in honor of Theodosius Dobzansky, p. 381-300, North-Holland Publishing Co., Amsterdam.

* Watt, W.B.,
1968, Adaptive significance of pigment polymorphism in Colias butterflies. I. Variation of melanin pigment in relation to thermoregulation. Evolution 22(3):437-458.

Welty, J.C.
1962, The Life of Birds. W.B. Saunders and Co., Philadelphia.

* White, M.J.D.,
1968, Models of Seciation. Science 157(3819):1065-1070.

* White, M.J.D.,R.E. Blackith, R.M. Blackith, and J. Cheney,
1967, Cytogenetics of the Viatica group of Morabine Grasshoppers I The Coastal Species. Aust. J. Zool. 15:263-302.

* White, M.J.D., H.L. Carson, and J. Cheney,
1964, Chromosomal races in the Australian grasshopper Moraba viatica. Evolution 18(3):417-429.

Whitfield, H.,
1969, Programming in IMP, revised edition, Dept. of Computer Science, University of Edinburgh and International Computers, Ltd., Edinburgh EMAP/41.5/0029.

* Willey, R.B. and R. L. Willey,
1967, Barriers to gene flow in natural populations of grasshoppers, I. The Black Canyon of the Gunnison River and Arphia conspersa. Psyche 74(1):42-57.

* Willey, R. B. and R.L. Willey,
1971, Barriers to gene flow in natural populations of grasshoppers, II. Maintenance of narrow hybrid zones between morphs of Arphia conspersa on Black Mesa, Colorado. Psyche 78(4): 330-349.

Williams, C. B.,
1930, The Migration of Butterflies. Edinburgh: Oliver and Boyd.

Williams, C. B.,
1958, Insect Migration, Collins, London.

Williams, C. B., G. F. Cockbill, M.E. Gibbs, and J. A. Downes,
1942, Studies in the migration of Lepidoptera. Trans. Roy. Entomol. Soc. Lond. 92(1):101-283.

* Williams, E.E., R. Highton, and D.M. Cooper,
1968, Breakdown of polymorphism of the Red Backed Salamander on Long Island. Evolution 22(1):76-86.

T* Womble, W. H.,
1951, Differential systematics. Science 114(2961):315-322.

* Wolda, H.,
1969a, The fine distribution of morph frequencies in the snail Cepaea nemoralis near Groningen. J. Anim. Ecol. 38:305-318.

* Wolda, H.,
1969b, Stability of a strong cline in morph frequencies of the small Cepaea nemoralis (L). J. Anim. Ecol. 38:623-635.

* Woodruff, D.S.,
1972, The evolutionary significance of hybrid zones in Pseudophryne (Anura:Leptodactylidae). Ph.D. Thesis, University of Melbourne, Dept. of Zoology.

Wolfenbarger, D.O.,
1946, Dispersion of Small Organisms, Distance Dispersion Rates of Bacteria, Spores, Seeds, Pollen, and Insects; Incidence Rates of Diseases and Injuries. Amer. Midl. Nat. 35(1):1-152.

* Worthington, E. B.,
1940, Geographical differentiation in fresh waters with special reference to fish. pp. 287-302 in J.S. Huxley, editor, The New Systematics. Oxford University Press.

T Wright, S.,
1931, Evolution in Mendelian populations. Genetics 16:97-159.

T Wright, S.,
1940a, The statistical consequences of Mendelian heredity in relation to speciation. pp.161-183 in J. S. Huxley, Editor, The New Septematics, Oxford University Press.

T Wright, S.,
1940, Breeding structure of populations in relation to speciation. Amer. Nat, 74(752):232-248.

T Wright, S.,
1943a, Isolation by distance. Genetics 28:114-138.

T* Wright, S.,
1943b, An analysis of local variability of flower color in Linanthus parryae. Genetics 28:139-156.

T Wright, S.,
1946, Isolation by distance under diverse systems of mating. Genetics 31:39-59.

Wright, S.,
1949, Adaptation and Selection. pp. 365-389 in G.L. Japson, G.G. Simpson and E. Mayer, editors, Genetics, Paleontology Evolution. Princeton University Press.

T Wright, S.,
1951, The genetical structure of populations. Ann.Eugen. 15:323-354.

T Wright, S.,
1965a, The interpretation of population structure by F-statistics with special reference to systems of mating. Evolution 19(3): 395-420.

T* Wright, S.,
1965b, Factor interaction and linkage in evolution. Proc.Roy.Soc.B. 162:80-104.

Wright, S.,
1968, Dispersion of Drosophila melanogaster. Amer.Nat.102(923):81-84.

T Wright, S.,
1969, Evolution and the Genetics of Populations, Volume 2, The Theory of Gene Frequencies. University of Chicago Press, Chicago.

Wright, S., Th. Dobzansky, and W. Hovanitz,
1942, Genetics of natural populations. VII. The allelism of lethal in the third chromosome of Drosophila pseudoobscura. Genetics 27:363-394.

ADDENDA TO REFERENCE LIST

* Briggs, D, and S.M. Walters,
1972, Plant Variation and Evolution, London: Weidenfield and Nicholson, Ltd.

Cain, A.J., and P.M. Sheppard,
1954, Natural selection in Cepaea, Genetics 39:89-116

Calhoun, J.B.,
1963, The Ecology and Sociology of the Norway Rat, U.S. Dept. Health, Ed. Welfare., Publ. Health Serv., Bethseda 8:1-288

* Haskins, C.P.,
1971, 1972, letters to the author and conversations.

T Kimura, M,
1953, The "stepping stone" model of a population. Ann. Rept. Nat. Inst. Genet. Japan, Mishima-shi, 3:63-63

Lack, D.,
1966, The Natural Regulation of Animal Numbers, Oxford University Press.

Lack, D.,
1968, Population Studies in Birds, Oxford University Press.

Lenz, F.,
1931, Quoted in Theinemann, 1950.

T Maruyama, T.,
1971, Spread of a gene substitution in a geographically structured population. Amer. Nat. 105(943):253-266

Rohlf, F.J., and R.R. Sokal,
1968, Statistical Tables, San Francisco: W. H. Freeman and Co.

Sakai, K.,
1964, Contribution to the problem of species colonization from the viewpoint of competition and migration. Pages 215-141 in, H.G. Baker and G.L. Stebbins, edotors, The Genetics of Colonizing Species, New York: Academic Press.

* Sheppard, P.M.,
1967, Natural Selection and Heredity, London: Hutchinson's University Library.

Sokal, R.R., and F.J. Rohlf,
1968, Biometry. San Francisco: W. H. Freeman and Co.

Thoday, J.M.,
1963, Correlation between gene frequency and population size. Amer. Nat. 97(897):409-412

Thoday, J.M., and J.D. Gibson,
1970, The probability of isolation by disruptive selection, Amer. Nat. 104(937):219-230.

Vouk, A.,
1931, Quoted in Theinemann, 1950.

Wallace, A.R.,
1889, Darwinism, An Exposition of the Theory of Natural Selection, with Some of its Applications. London: The MacMillan Co., Ltd.

Appendix 1

Frequency of Distribution Types in North American Amphibians and Reptiles

The table following lists each of the Amphibians and Reptiles found in North America north of Mexico, along with the distribution phenomena they show.

Column I indicates whether or not a given species is monotypic (M) or polytypic (P), and if polytypic, how many subspecies are present (in parenthesis). Column II is ticked if the species has part of its range disjunct from the remainder of the species, but the disjunct population is not subspecifically distinct from other populations. Column III is ticked if one or more subspecies is not anywhere in contact with other subspecies. Column IV is ticked if one or more of the subspecies is involved in a hybrid zone. Column V is ticked if subspecies are contiguously distributed; clines are too steep to show in distribution maps. Column VI is ticked if one or more subspecies grade smoothly into other subspecies.

Herpetologists have long been interested in biogeographical phenomena, and very detailed distributional information is known for most of the North American Amphibians and Reptiles. It is my own experience in the western United States, where one would expect more patchiness of distribution owing to complex topography, that the maps are remarkably reliable. Ranges are not, of course, as continuous as the maps imply. Maps showing individual capture records as well as

shading in correct habitats are preferable to the usual outline maps. It is customary to draw a continuous range or continuous shading unless disjunctions are greater than about 10 Km, or the populations are separated by very unsuitable habitat. As a working definition of disjunction, for the purposes of the table, a species was not regarded as including a disjunct population unless it was separated by more than 10 Km, or what could be seen on the distribution maps of Conant (1958) and Stebbins (1966). Problems were solved by reference to Stebbins (1951, 1954), Wright and Wright (1949), Smith (1946), Bishop (1943), and the literature. The only borderline distribution was Long Island (New York) - at one end of this spindle-shaped island the distance to the mainland is about 2 Km. Probably only a few snakes would cross regularly. Long Island populations were regarded as disjunct. For a further discussion of distribution maps and species ranges see Stebbins (1966), and Chapter 4 of Udvardy (1969).

Column	I	Monotypic or Polytypic
	II	Disjunct but not differentiated
	III	Disjunct differentiated
	IV	Hybrid zone
	V	Continguous ranges
	VI	Clinal subspecies

SALAMANDERS	I	II	III	IV	V	VI
Dicamptodon ensatus	M	x				
Ambystoma gracile	P(2)	x			x	
A. macrodactylum	P(5)	x			x	
A. tigrinum	P(8)		x		x	
Rhyacotriton olympicus	P(2)					x
Taricha torosa	P(2)	x	x			
T. rivularis	M					
T. granulosa	P(2)	x			x	
Hydromantes brunus	M					
H. platycephalus	M					
H. shastae	M					
Ensatina eschscholtzi	P(7)	x	x	x	x	x
Plethodon vehiculum	M	x				
P. dunni	M					
P. elongatus	M					
P. stormi	M					
P. larselli	M					
P. neomexicanus	M					
P. vandykei	P(2)	x	x			
Batrachoceps wrighti	M					
B. attenuatus	M	x				
B. pacificus	P(3)		x			
Aneides ferreus	M	x				
A. lugubris	M	x				
A. flavipunctatus	P(2)		x			
A. hardyi	M					
Cryptobranchus alleganiensis	P(2)	x	x			
N. maculosus	P(3)			x	x	
N. [illegible]ri	P(2)			x	x	
N. lewisi	M					
N. punctatus	P(2)				x	
Siren lacertina	M					
S. intermedia	P(3)					
Pseudobranchus striatus	P(5)					
Amphiuma means	P(2)				x	
Ambystoma annulatum	M					
A. cingulatum	P(2)		x			
A. jeffersonianum	P(x)	x				
A. laterale	P(x)	x				
A. texanum	M					
A. mabeei	M					
A. talpoideum	M	x				
A. opacum	M	x				
A. maculatum	M	x				
Diemictylus viridescens	P(4)	x			x	x
D. perstriatus	M					
D. meridionalis	M					
Desmognathus fuscus	P(5)				x	x

SALAMANDERS (continued)	I	II	III	IV	V	VI
D. planiceps	M					
D. ochrophaeus	P(2)		x			
D. ocoee	M					
D. perlapsus	M					
D. monticola	P(2)		x			
D. quadrimaculatus	M					
D. wrighti	M					
D. aeneus	P(2)		x			
Leurognathus marmoratus	P(5)				x	
Plethodon cinereus	P(3)	x	x			
P. dorsalis	P(2)		x			
P. richmondi	P(4)	x			x	
P. welleri	P(2)				x	
P. glutinosus	P(2)	x			x	
P. wehrlei	P(2)	x			x	
P. yonahlosse	M					
P. ouachitae	M					
P. caddoensis	M					
P. jordani	P(7)	x	x		x	
Hemidactylum scutatum	M	x				
Stereochilus marginatus	M					
Gyrinophilus porphyritucus	P(3)				x	
G. danielsi	P(3)	x			x	
G. palleucus	M	x				
Pseudotriton montanus	P(4)	x	x		x	
P. ruber	P(4)	x			x	
Aneides aeneus	M					
Eurycea bislineata	P(3)				x	x
E. longicauda	P(3)				x	x
E. lucifuga	M	x				
E. multiplicata	P(2)				x	
E. tynerensis	M					
E. neotenes	P(3)		x		x	
E. troglodytes	M					
E. nana	M					
Typhlotriton spelaeus	M					
Typhlomolge rathbuni	M					
Haedeotriton wallacei	M					
Manculus quadridigitatus	M	x				
Phaeognathus hubrichti	M					
SALAMANDER TOTALS	88 sp. 47 M. 41 P. 120 sbsp.	30*	18	3	29	8

* of which 12 are monotypic

ANURANS	I	II	III	IV	V	VI
Scaphiopus intermontanus	M					
S. bombifrons	M	x				
S. hammondi	M	x				
S. couchi	M	x				
Ascaphus truei	M	x				
Gastrophryne olivacea	P(2)		x			
Eleutherodactylus augusti	P(2)	x	x			
Bufo boreas	P(4)		x		x	x
B. canorus	M					
B. microscaphus	P(2)	x	x			
B. woodhousei	P(4)	x				
B. hemiophrys	M	x				
B. cognatus	M	x				
B. speciosus	M					
B. debilis	P(2)				x	
B. retiformis	M					
B. alvarius	M					
B. punctatus	M					
Pseudacris triseriata	P(3)	x			x	x
Pternohyla foidiens	M					
Acris crepitans	P(2)				x	x
Hyla regilla	M	x				
H. wrightorum	M	x				
H. arenicolor	M	x				
H. californiae	M					
Rana sylvatica	M					
R. aurora	P(2)					x
R. cascadae	M	x				
R. pretiosa	M	x				
R. pipiens	P(4)	x			x	x
R. muscosa	M	x				
R. boylei	M	x				
R. catesbeiana	M	x				
R. tarahumarae	M					
Scaphiopus holbrooki	M	x				
S. hurteri	M					
Leptodactylus labialis	M					
Eleutherodactylus ricordi	M	x				
Syrrhophus marnocki	M					
S. campi	M					
Bufo americanus	P(3)					
B. terrestris	M					
B. houstonensis	M					
B. valliceps	M					
B. quercicus	M					
B. marinus	M					
Acris gryllus	P(2)					
Hyla crucifer	P(2)	x			x	
H. cinerea	P(2)				x	x
H. andersoni	M	x				

ANURANS (continued)	I	II	III	IV	V	VI
H. femoralis	M					
H. squirella	M					
H. versicolor	P(3)				x	
H. avivoca	P(2)				x	
H. septentrionalis	M	x				
H. gratiosa	M					
H. ocularis	M					
Smilisca baudini	M					
P. nigrata	P(2)				x	
P. clarki	M					
P. brimleyi	M					
P. brachyphona	M					
P. ornata	M					
P. streckeri	P(2)	x	x			
Gastrophryne carolinensis	M	x				
Hypopachus cuneus	M					
Rana heckscheri	M					
R. grylio	M					
R. virgatipes	M	x				
R. clamitans	P(2)	x			x	
R. septentrionalis	M	x				
R. palustris	M	x				
R. areolata	P(5)	x	x		x	x
ANURANS TOTALS:	73 sp.	32*	6	1	15	8
	53 M.					
	20 P.					
	52 sbsp.					
	0.71					

*of which 22 are monotypic

TURTLES	I	II	III	IV	V	VI
Chelydra serpentina	P(2)				x	x
Kinosternon flavescens	P(3)	x				
K. sonoriense	M	x				
Clemmys marmorata	P(2)	x				x
Chrysemys picta	P(4)	x			x	x
Pseudemys scripta	P(4)				x	x
P. concinna	P(5)				x	
Terrapene ornata	P(2)					x
Gopherus agassizi	M					
Trionyx spiniferus	P(4)	x			x	x
T. muticus	M					
Macroclemmys temmincki	M					
Sternothaerus odoratus	M	x				
S. carinatus	M					
S. minor	P(2)				x	
S. depressus	M					
Kinosternon bauri	P(2)				x	
K. subrubrum	P(3)	x			x	
Clemmys guttata	M	x				
C. muhlenbergi	M	x				
C. insculpta	M	x				
Terrapene carolina	P(4)				x	
Malaclemys terrapin	P(7)				x	x
Graptemys geographica	M	x				
G. barbouri	M					
G. pulchra	M					
G. kohni	M					
G. pseudogeographica	P(3)				x	x
G. versa	M					
G. oculifera	M					
G. flavimaculata	M					
G. nigrinoda	M					
P. floridana	P(3)				x	
P. rubriventris	M	x				
P. nelsoni	P(2)		x			
Deirochelys reticularia	P(3)				x	
Emydoidea blandingi	M	x				
Gopherus polyphemus	M					
G. berlandieri	M					
T. ferox	M					
TURTLES TOTALS:	40 sp. 23 M. 17 P. 55 sbsp. 1.37 sbsp/sp.	13*	1		13	8

*of which 8 are monotypic

LIZARDS	I	II	III	IV	V	VI
Phyllodactylus xanti	P(2)					x
Coleonyx variegatus	P(5)	x			x	
C. brevis	M					
Dipsosaurus dorsalis	P(2)				x	
Sauromalus obesus	P(4)				x	
Holbrookia maculata	P(3)				x	
H. texana	P(3)				x	
Callisaurus draconoides	M					
Uma notata	P(2)				x	
U. inornata	M					
U. scoparia	M					
Crotaphytus collaris	M(2)				x	
C. wislizenii	P(2)		x			
Sceloporus scalaris	M	x				
S. jarrovi	M					
S. poinsetti	M					
S. magister	P(8)	x			x	
S. clarki	P(2)				x	
S. orcutti	P(2)				x	
S. occidentalis	P(4)		x		x	
S. undulatus	P(8)	x			x	x
S. virgatus	x					
S. graciosus	P(2)	x	x			
Uta stansburiana	P(4)				x	x
Urosaurus graciosus	P(2)					x
U. ornatus	M					
U. microscutatus	M					
Streptosaurus mearnsi	M					
Phrynosoma cornutum	M	x				
P. coronatum	P(2)				x	
P. platyrhinos	P(2)				x	x
P. douglassi	P(6)	x			x	
P. m'calli	M					
P. modestum	M					
P. solare	M					
Xantusia henshawi	M					
X. vigilis	P(2)	x	x			
X. arizonae	M					
Klauberina riversiana	M					
Eumeces obsoletus	M					
E. multivirgatus	P(2)					x
E. callicephalus	M					
E. skiltonianus	P(3)				x	x
E. gilberti	P(5)	x	x			x
Cnemidophorus hyperythrus	P(3)					x
C. burti	P(3)		x			x
C. neomexicanus	M					
C. inornatus	M	x				
C. uniparens	M	x				
C. velox	M					

LIZARDS (continued)	I	II	III	IV	V	VI
C. sexlineatus	P(2)				x	
C. exsanguis	M					
C. gularis	M					
C. tigris	P(8)	x			x	
C. tesselatus	M	x				
Gerrhonotus multicarinatus	P(3)				x	
G. panamintinus	M					
G. kingi	M					
G. coeruleus	P(4)	x			x	
Anniella pulchra	P(2)				x	
Heloderma suspectum	P(2)				x	
Anolis carolinensis	M				x	
Crotaphytus reticulatus	M					
Holbrookia propinqua	M					
H. lacerata	P(2)		x			
Sceloporus variabilis	P(2)			x		
S. grammicus	M					
S. cyanogenys	M					
S. olivaceus	M					
S. woodi	M					
Lygosoma laterale	M	x				
Eumeces fasciatus	M	x				
E. laticeps	M					
E. inexpectatus	M					
E. brevilineatus	M					
E. tetragrammus	M					
E. anthracinus	P(2)	x	x			
E. septentrionalis	P(2)				x	
E. egregius	P(2)				x	
Neoseps reynoldsi	M					
Ophisaurus ventralis	M					
O. compressus	M					
O. attenuatus	P(2)				x	
Gerrhonotus infernalis	M					
Rhineura floridana	M					
LIZARDS TOTALS:	85 sp. 47 M. 38 P. 118 sbsp. 1.39 sbsp/sp.	17*	8	-	29	9

*of which 7 are monotypic

SNAKES	I	II	III	IV	V	VI
Leptotyphlops humilis	M					
L. dulcis	P(2)					
Charina bottae	P(3)	x	x		x	
Lichanura trivirgata	P(3)				x	
Diadophis punctatus	P(11)	x			x	x
Contia tenuis	M	x				
Heterodon nasicus	P(3)	x			x	x
Phyllorhynchus decurtatus	P(3)				x	x
P. browni	P(2)				x	
Opheodrys vernalis	P(2)	x			x	
O. aestivus	M					
Coluber constrictor	P(8)	x			x	x
Masticophis flagellum	P(7)				x	
M. lateralis	P(2)				x	
M. taeniatus	P(4)	x			x	
M. bilineatus	P(2)		x			
Salvadora hexalepis	P(4)				x	
S. grahamiae	P(2)				x	
Elaphe guttata	P(3)	x	x		x	
E. triaspis	M					
E. subocularis	M					
Arizona elegans	P(7)	x			x	x
Pituophis melanoleucus	P(9)	x	x		x	x
Lampropeltis getulus	P(12)	x	x		x	
L. zonata	P(5)	x			x	x
L. pyromelana	P(3)	x	x		x	
L. triangulum	P(9)	x	x		x	
Thinocheilus leconti	P95)	x			x	
Natrix erythrogaster	P(5)	x	x		x	
Natrix sipedon	P(3)				x	
Storeria occipitomaculata	P(2)	x			x	
Thamnophis rufipunctatus	M	x				
T. sirtalis	P(8)	x	x		x	
T. elegans	P(5)	x	x	x	x	x
T. couchi	P(6)			x		x
T. ordinoides	M					
T. crytopsis	P(2)					x
T. eques	P(2)				x	
T. marcianus	P(2)				x	
T. radix	P(2)	x			x	
T. proximus	P(4)	x			x	
Tropidoclonion lineatum	P(3)	x			x	
Sonora semiannulata	M					
S. episcopa	P(2)				x	
Chionactis occipitalis	P(4)				x	x
C. palarostris	M					
Chilomeniscus cinctus	M					
Ficimia cana	M					
F. quadrarectangularis	M					

SNAKES (continued)	I	II	III	IV	V	VI
Tantilla planiceps	P(8)	x		x	x	
T. nigriceps	P(2)				x	
T. wilcoxi	M					
Oxybelis aeneus	M					
Trimorphodon vandenburghi	M	x				
T. lambda	M					
T. vilkinsoni	M					
Hypsiglena torquata	P(7)	x			x	x
Micururoides euryxanthus	P(2)					x
Sistrurus catenatus	P(3)				x	
Crotalus atrox	M					
C. ruber	M					
C. lepidus	P(2)				x	
C. mitchelli	P(2)	x			x	
C. cerastes	P(3)				x	
C. molossus	P(2)				x	
C. tigris	M					
C. viridis	P(8)				x	
C. scutulatus	M					
C. pricei	P(2)		x			
C. willardi	P(3)	x	x			
Natrix cyclopion	P(2)				x	
N. taxispilota	M					
N. rhombifera	M					
N. fasciata	P(6)	x	x	x	x	x
Clonophis kirtlandi	M					
C. harteri	M	x				
Regina septemvittata	M	x				
R. grahami	M					
R. rigida	M					
Seminatrix pygaea	P(5)				x	
Storeria dekayi	P(4)	x			x	x
Thamnophis butleri	M	x				
T. brachystoma	M					
Virginia striatula	M					
V. valeriae	P(3)		x		x	
Liodytes alleni	M					
Heterodon platyrhinos	M	x				
H. simus	M					
Rhadinaea flavilata	M					
Carphophis amoenus	P(3)	x			x	
Abastor erythrogrammus	M					
Farancia abacura	P(2)				x	
Coniophanes imperialis	M					
Drymobius margaritiferus	M					
Drymarchon corais	P(2)	x	x			
Salvadora lineata	M					
Elaphe vulpina	P(2)		x			
E. obsoleta	P(8)	x			x	
Lampropeltis calligaster	P(2)					
Stilosoma extenuatum	P(3)				x	
Cemophora coccinea	M	c				
Ficimia olivacea	M					

SNAKES (continued)	I	II	III	IV	V	VI
Leptodeira septentrionalis	M					
Tantilla coronata	P(3)	x			x	
Tantilla gracilis	P(2)	x			x	
Micrurus fulvius	P(3)				x	
Agkistrodon contortrix	P(4)				x	x
A. piscivorus	P(2)				x	
Sistrurus miliarius	P(3)				x	
Crotalis horridus	P(2)	x			x	
C. adamanteus	M					
SNAKES TOTALS:	111 sp. 43 M. 68 P. 260 sbsp. 2.34 sbsp/sp.	41*	16	4	58	16

* of which 8 are monotypic

APPENDIX 2

COMPUTER PROGRAMMES USED IN THE CLINE SIMULATIONS

The basic series of steps in each programme is similar to that of the experimental protocol (figure 3-2). The parameters are read in, then the sequence: random mating, selection gene flow, is repeated for a set number of generations (large enough to allow the clines to reach equilibrium or near equilibrium), then the results are printed out. As in the experiment all demes are started with an initial gene frequency of o.50.

Glossary of symbols

Programme A.

TT the number of generations to run, counter T.
V the number of demes, counter I.
NN(I) the population size in deme I.
WA(I) AA
WB(I) the fitness of genotype Aa
WC(I) aa
PP(I) the gene frequency (gene A) in deme I.
LA(I)
LB(I) the number of each genotype emigrating from deme I.
LC(I)
RA(I)
RB(I) the number of each genotype not emigrating from deme I.
RC(I)
G the total fraction involved in gene flow (g, in chapters 2 to 5).

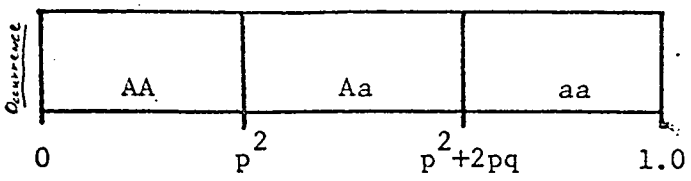
Programme B.

G the number of generations
PL the total fraction of the deme involved in gene flow (g)
RN the total population size of deme I.
R and C the number of demes in each direction; there are R*C demes.
N(I,J) population size in deme (I,J) before gene flow.
D(I,J) population size in deme (I,J) after gene flow.
TNP(I,J) population size * gene frequency (number of A genes) in deme I,J.
P(I,J) gene frequency in deme (I,J).

This programme uses an algebraic simplification of the recurrence equations; one equation for gene flow of genes is used instead of the usual genotype equations to save computing time. (It takes several hours of computing time to do one genetic drift programme).

Subroutine RANDOM is used to produce random numbers by the power residue method (I.B.M., 1959). Random mating is done by giving each of N(I,J) individuals in deme (I,J) a random number from a uniform distribution of real numbers between 0 and 1.0. The rectangular distribution is divided into three segments, corresponding to the three genotypes. The boundaries of the segments are determined by the expected Hardy-Weinberg ratios, or, as in figure A-1.

FIGURE A-1



where p is the gene frequency in deme (I,J) parents, and q=1-p. For a deme size N, N random numbers (N individuals) are drawn by N calls on RANDOM.

PROGRAMME C.

G generations to be run.
PTG generation results to be printed.
L total gene flow (g).
N deme size.
F(K,I,J) is the number of individuals of genotype, destination K, in deme (I,J), where k is as follows:
K=1 to 7, genotype AA
K=8 to 14, genotype Aa
K=15 to 21, genotype aa; and
K=2 to 7, 9 to 14, 16 to 21, will move to one of the six adjacent demes;
K=1,8,15, will not emigrate.
P(I,J) gene frequency in deme (I,J).

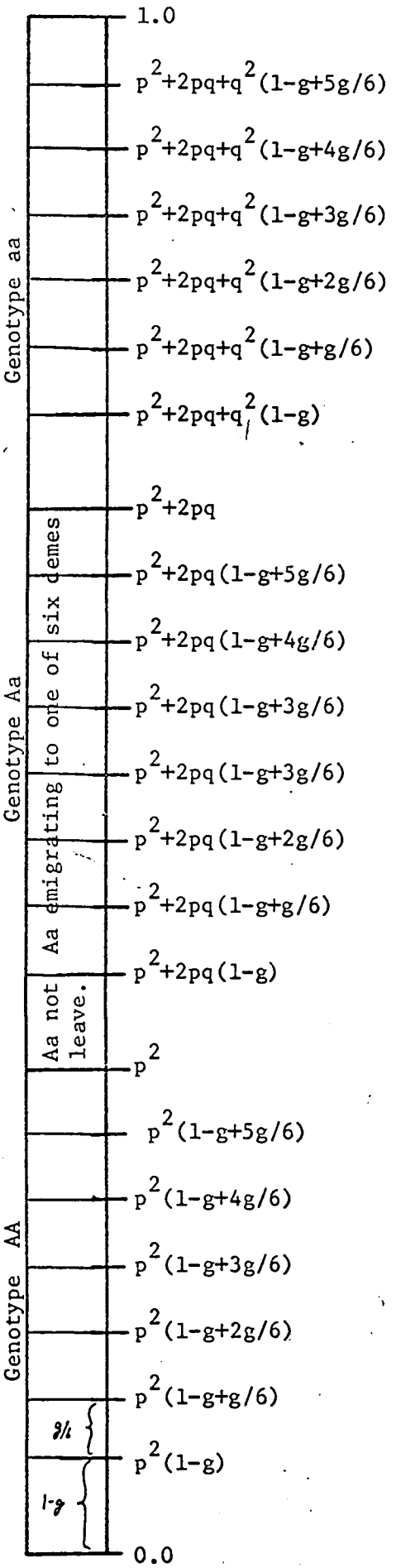
For stochastic mating and gene flow there are still N calls on RANDOM but the uniform distribution is divided up as in figure A-2, into three main segments for each genotype, and each genotype is divided up into a fraction (1-g) not emigrating and a graction (g/6) emigrating to one of the six adjacent demes.

PROGRAMME D.

L gene flow (g)
REC percent recombination between loci A & B
FA(I), FB(J) selection gradients for each direction
N(I,J) population size per deme.
NU(I,J), NV(I,J), NW(I,J), NY(I,J) chromosome frequencies in deme (I,J).

The fitness of A and B genotypes are assumed to be independent (multiplicative).

FIGURE A-2



PROGRAMME A.

The Basic Cline Simulation Model in One Dimension, Deterministic

```
%BEGIN
%INTEGER T,TT,I,V,N
%REAL P,Q,G,A,B,C
READ(TT): READ(V); READ(G)
%REALARRAY PP,RA,RB,RC,AA,BB,CC(1:V),LA,LB,LC(0:V+1),NN(1:V)
%CYCLE I=1,1,V
READ(NN(I)); READ(PP(I))
%REPEAT
%CYCLE I=1,1,V
READ(WA(I)); READ(WB(I)); READ(WC(I))
%REPEAT
NEWLINE; %PRINTTEXT'% GENE FLOW (G)'; PRINT(G,1,4); NEWLINES(2)
%PRINTTEXT'DEME     GENOTYPE FITNESSES     POPULATION SIZE'
NEWLINES(2)
%CYCLE I=1,1,V
WRITE(I,3); PRINT(WA(I),7,1); PRINT(WB(I),4,1); PRINT(WC(I),4,1)
PRINT(NN(I),10,1); NEWLINE
%REPEAT; NEWLINES(4)
%CYCLE T=1,1,TT
%CYCLE I=1,1,V
N=NN(I)
P=PP(I); Q=1-P
A=N*P**2*WA(I)
B=N*2*P*Q*WB(I)
C=N*Q**2*WC(I)
LA(I)=G*A
LB(I)=G*B
LC(I)=G*C
RA(I)=A-2*LA(I)
RB(I)=B-2*LB(I)
RC(I)=C-2*LC(I)
%REPEAT
LA(0)=LA(1); LB(0)=LB(1); LC(0)=LC(1)
LA(V+1)=LA(V); LB(V+1)=LB(V); LC(V+1)=LC(V)
%CYCLE I=1,1,V
A=RA(I)+LA(I-1)+LA(I+1)
B=RB(I)+LB(I-1)+LB(I+1)
C=RC(I)+LC(I-1)+LC(I+1)
PP(I)=(A+(B/2))/(A+B+C)
AA(I)=A
BB(I)=B
CC(I)=C
%REPEAT
%PRINTTEXT'GENERATION '; WRITE(T,4); NEWLINES(2)
%PRINTTEXT'DEME      GENOTYPE NUMBERS   GENE FREQUENCY'; NEWLINES(2)
%CYCLE I=1,1,V
```

G=G/2

```
WRITE(I,3); PRINT(AA(I),7,1); PRINT(BB(I),4,1); PRINT(CC(I),4,1)
PRINT(PP(I),8,4); NEWLINE
%REPEAT
NEWLINE
%REPEAT
%ENDOFPROGRAM
```

PROGRAMME B

Cline Model, Two Dimensions, No Selection, Genetic Drift

```
%BEGIN
%COMMENT SIMPLE DRIFT PROGRAM STOCHASTIC MATING BUT
%COMMENT DETERMINISTIC GENE FLOW
%EXTERNALREALFNSPEC RANDOM(%INTEGERNAME N, %INTEGER I)
%INTEGER I,J,R,C,K,G,X,RN,TD,U,V,T
%REAL L,S,TP,PS,PQ,A,PL
READ(G); READ(R); READ(C)
%INTEGERARRAY N,D(0:R+1,0:C+1)
%REALARRAY P,NP,TNP(0:R+1,0:C+1)
READ(PL); READ(RN)
X=487795
L=PL/6; S=1-(6*L)
%CYCLE J=1,1,C; %CYCLE I=1,1,R
N(I,J)=RN
READ(P(I,J))
%REPEAT; %REPEAT
%CYCLE J=1,1,C
N(0,J)=0; N(R+1,J)=0; NP(0,J)=0;  NP(R+1,J)=0
%REPEAT
%CYCLE I=0,1,R+1
N(I,0)=0; N(I,C+1)=0; NP(I,0)=0; NP(I,C+1)=0
%REPEAT
%CYCLE J=1,1,C
%CYCLE I=1,2,(R-1)
NP(I,J)=N(I,J)*P(I,J)
TD=N(I-1,J-1)+N(I-1,J)+N(I+1,J-1)+N(I+1,J)
D(I,J)=INT(S*N(I,J)+L*(TD+N(I,J-1)+N(I,J+1)))
%REPEAT
%CYCLE I=2,2,R
NP(I,J)=N(I,J)*P(I,J)
TD=N(I-1,J)+N(I-1,J+1)+N(I+1,J)+N(I+1,J+1)
D(I,J)=INT(S*N(I,J)+L*(TD+N(I,J-1)+N(I,J+1)))
%REPEAT
%REPEAT
NEWLINE
%PRINTTEXT'PARAMETERS:  N='; WRITE(RN,3); %PRINTTEXT'   G='
PRINT((6*L),1,4); NEWLINE
%CYCLE T=0,1,G
%CYCLE J=1,1,C
%CYCLE I=1,2,(R-1)
TD=D(I,J)
TP=NP(I-1,J-1)+NP(I-1,J)+NP(I+1,J-1)+NP(I+1,J)
TP=(S*NP(I,J)+L*(TP+NP(I,J-1)+NP(I,J+1)))/TD
P(I,J)=TP
PS=TP*TP; PQ=PS+2*TP*(1-TP)
U=0; V=0
```

PROGRAMME B

```
%CYCLE K=1,1,TD
A=RANDOM(X,1)
%IF A<PS %THEN U=U+1
%IF A>PQ %THEN V=V+1
%REPEAT
TNP(I,J)=N(I,J)*(TD+U-V)/(2*TD)
%REPEAT
%CYCLE I=2,2,R
TD=D(I,J)
TP=NP(I-1,J)+NP(I-1,J+1)+NP(I+1,J)+NP(I+1,J+1)
TP=(S*NP(I,J)+L*(TP+NP(I,J-1)+NP(I,J+1)))/TD
P(I,J)=TP
PS=TP*TP; PQ=PS+2*TP*(1-TP)
U=0; V=0
%CYCLE K=1,1,TD
A=RANDOM(X,1)
%IF A<PS %THEN U=U+1
%IF A>PQ %THEN V=V+1
%REPEAT
TNP(I,J)=N(I,J)*(TD+U-V)/(2*TD)
%REPEAT
%REPEAT
%CYCLE J=1,1,C; %CYCLE I=1,1,R
NP(I,J)=TNP(I,J)
%REPEAT; %REPEAT
%REPEAT
NEWLINE; SELECTOUTPUT(97); NEWLINES(2)
WRITE(T,2); WRITE(RN,5); PRINT((6*L),3,4); NEWLINE
%CYCLE J=1,1,C; %CYCLE I=1,1,R
PRINT(P(I,J),1,4)
%IF (FRACPT(I/10))<0.001 %THEN NEWLINE
%REPEAT; %REPEAT; NEWLINES(2)
NEWLINE
%PRINTTEXT'SIMPLE DRIFT'; NEWLINE
%ENDOFPROGRAM
```

PROGRAMME C

Cline Model, Two Dimensions, No Selection, Genetic Drift and Random Error in Gene Flow

```
%BEGIN
%COMMENT STOCHASTIC MATING, STOCHASTIC GENE FLOW
%EXTERNALREALENSPEC RANDOM(%INTEGERNAME M, %INTEGER I)
%ROUTINESPEC PPRINT
%INTEGER I,J,R,C,T,G,K,KN,X,N,U,V,W,PTG
%REAL TP,PS,PQ,QS,LL,LM,RD,L,S
READ(G); READ(PTG); READ(R); READ(C)
%SHORTINTEGERARRAY F(1:21,0:R+1,0:C+1),GT(1:21)
%REALARRAY P(1:R,1:C),LT(1:21)
READ(L); READ(N)
S=1-L; L=L/6
X=655397
%CYCLE J=0,1,C+1
%CYCLE K=1,1,21; F(K,0,J)=0; F(K,R+1,J)=0; %REPEAT
%REPEAT
%CYCLE I=0,1,R+1
%CYCLE K=1,1,21; F(K,1,0)=0; F(K,I,C+1)=0; %REPEAT
%REPEAT
%CYCLE J=1,1,C; %CYCLE I=1,1,R
P(I,J)=1/2
%REPEAT; %REPEAT
NEWLINE; SELECTOUTPUT (97); NEWLINE
%CYCLE T=1,1,G
%CYCLE J=1,1,C; %CYCLE I=1,1,R
TP=P(I,J)
PS=TP*TP; PQ=2*TP*(1-TP); QS=1-PS-PQ
LM=S*PS; LT(1)=LM
%CYCLE K=2,1,6
LM=LM+(K-1)*L*PS
LT(K)=LM
%REPEAT
LT(7)=PS
LM=PS+S*PQ; LT(8)=LM
%CYCLE K=9,1,13
LM=LM+(K-8)*L*PQ
LT(K)=LM
%REPEAT
LT(14)=PS+PQ
LM=PS+PQ+S*QS; LT(15)=LM
%CYCLE K=16,1,20
LM=LM+(K-15)*L*QS
LT(K)=LM
%REPEAT
LT(21)=1
```

PROGRAMME C

```
%CYCLE K=1,1,21; GT(K)=0; %REPEAT
%CYCLE KN=1,1,N
RD=RANDOM(X,1)
%IF RD>(PS+PQ) %THEN -> C
%IF RD<PS %THEN -> A
LL=LT(7)
%CYCLE K=8,1,14
LM=LT(K)
%IF (LL<=RD<LM) %THEN GT(K)=GT(K)+1
LL=LM
%REPEAT
-> B
A:  LL=0
%CYCLE K=1,1,7
LM=LT(K)
%IF (LL<=RD<LM) %THEN GT(K)=GT(K)+1
LL=LM
%REPEAT
-> B
C:  LL=LT(14)
%CYCLE K=15,1,21
LM=LT(K)
%IF (LL<=RD<LM) %THEN GT(K)=GT(K)+1
LL=LM
%REPEAT
B:  %REPEAT
%CYCLE K=1,1,21
F(K,I,J)=GT(K)
%REPEAT
%REPEAT; %REPEAT
%CYCLE J=1,1,C
%CYCLE I=1,2,(R-1)
U=F(1,I,J)+F(2,I-1,J-1)+F(3,I-1,J)+F(4,I,J-1)+F(5,I,J+1) %C
  +F(6,I+1,J-1)+F(7,I+1,J)
V=F(8,I,J)+F(9,I-1,J-1)+F(10,I-1,J)+F(11,I,J-1)+F(12,I,J+1) %C
  +F(13,I+1,J-1)+F(14,I+1,J)
W=F(15,I,J)+F(16,I-1,J-1)+F(17,I-1,J)+F(18,I,J-1)+F(19,I,J+1) %C
  +F(20,I+1,J-1)+F(21,I+1,J)
P(I,J)=(U+(V/2))/(U+V+W)
%REPEAT
%CYCLE I=2,2,R
U=F(1,I,J)+F(2,I-1,J)+F(3,I-1,J+1)+F(4,I,J-1)+F(5,I,J+1) %C
  +F(6,I+1,J)+F(7,I+1,J+1)
V=F(8,I,J)+F(9,I-1,J)+F(10,I-1,J+1)+F(11,I,J-1)+F(12,I,J+1) %C
  +F(13,I+1,J)+F(14,I+1,J+1)
W=F(15,I,J)+F(16,I-1,J)+F(17,I-1,J+1)+F(18,I,J-1)+F(19,I,J+1)  %C
  +F(20,I+1,J(+F(21,I+1,J+1)
P(I,J)=(U+(V/2))/(U+V+W)
%REPEAT
%REPEAT
%REPEAT; NEWLINE; %COMMENT GENERATIONS
PPRINT
NEWLINES(8); %PRINTTEXT'END OF CALCULATIONS'
NEWLINES(2)
%ROUTINE PPRINT
```

PROGRAMME C

```
NEWLINE
%PRINTTEXT'''GENERATION'; WRITE(T,3); %PRINTTEXT'   N='
WRITE(N,2); %PRINTTEXT',    G='; PRINT((1-S),1,4)
%PRINTTEXT''''; NEWLINES(2)
%CYCLE J=1,1,C
%CYCLE I=1,1,R
PRINT(P(I,J),1,4)
%IF (FRACPT(I/10))<0.001 %THEN NEWLINE
%REPEAT
%REPEAT
NEWLINE
%END
%ENDOFPROGRAM
```

PROGRAMME D

Cline Model, Two Dimensions, Deterministic, Differing Selection Gradients on Two Linked Loci

```
%BEGIN
%COMMENT TWO DIMENTIONAL CLINE SIMULATION WITH TWO LOCI
%INTEGER T,G,I,R,J,C
%INTEGER WND
%REAL TD,TU,TV,TW,TY,F,A,B,AL,AH,BL,BH,RD,REC
%REAL IS,L,S
%REAL UU,VV,WW,YY,TT,TN
READ(G); READ(R); READ(C)
%COMMENT R AND C MUST BE EVEN FOR MIGRATION TO WORK PROPERLY
%REALARRAY D,TNU,TNV,TNW,TNY(1:R,1:C)
%REALARRAY N,NU,NV,NW,NY(0:R+1,0:C+1)
%REALARRAY FA(1:R),FB(1:C)
52:  READ(L); READ(REC)
READ(AL); READ(AH); READ(BL); READ(BH)
READ(WND)
S=(1-6*L)
IS=(AH-AL)/R; FA(1)=AL
%CYCLE I=2,1,R; FA(I)=FA(I-1)+IS; %REPEAT
IS=(BH-BL)/C; FB(1)=BL
%CYCLE J=2,1,C; FB(J)=FB(J-1)+IS; %REPEAT
%BEGIN
%REALARRAY U,V,W,Y(1:R,1:C)
%CYCLE J=1,1,C
N(0,J)=0; N(R+1,J)=0
NU(0,J)=0; NU(R+1,J)=0; NV(0,J)=0; NV(R+1,J)=0
NW(0,J)=0; NW(R+1,J)=0; NY(0,J)=0; NY(R+1,J)=0
%CYCLE I=1,1,R
N(I,J)=100
U(I,J)=1.00; V(I,J)=0.00; W(I,J)=0.00; Y(I,J)=1.00
%REPEAT
%REPEAT
%CYCLE I=0,1,R+1
N(I,0)=0; N(I,C+1)=0
NU(I,0)=0; NU(I,C+1)=0; NV(I,0)=0; NV(I,C+1)=0
NW(I,0)=0; NW(I,C+1)=0; NY(I,0)=0; NY(I,C+1)=0
1: %REPEAT
NEWPAGE
NEWLINES(4)
%PRINTTEXT'ALL POPULATIONS STARTING AT SIZE=100'; NEWLINE
%PRINTTEXT' ALL POPULATIONS STARTING WITH P=0.50, R=0.50   '
%PRINTTEXT' AND CHROMOSOMES AB -- ONLY'; NEWLINES(2)
%PRINTTEXT' RECOMBINATION  R='; PRINT(REC,1,4); NEWLINES(2)
%PRINTTEXT'TOTAL FRACTION MIGRATING '; PRINT((L*6),1,4)
```

PROGRAMME D

```
%PRINTTEXT'  FRACTION MIGRATING IN ONE DIRECTION '
PRINT(L,1,4); NEWLINES(2)
%PRINTTEXT' A DOMINANT SELECTION (SUCCESSIVE ROW CLINE)'
NEWLINES(2)
%CYCLE I=1,1,R; PRINT(FA(I),1,2)
%IF (FRAC PT(I/20))<0.01 %THEN NEWLINE
%REPEAT
NEWLINES(4)
%PRINTTEXT' B DOMINANT SELECTION (WITHIN ROW CLINE)'
NEWLINES(2)
%CYCLE J=1,1,C; PRINT(FB(J),1,2)
%IF (FRAC PT(J/20))<0.01 %THEN NEWLINE
%REPEAT
NEWLINES(6)
%CYCLE J=1,1,C
%CYCLE I=1,2,(R-1)
TN=N(I,J)
NU(I,J)=TN*U(I,J); NV(I,J)=TN*V(I,J)
NW(I,J)=TN*W(I,J); NY(I,J)=TN*Y(I,J)
TD=N(I-1,J)+N(I-1,J+1)+N(I+1,J)+N(I+1,J+1)
D(I,J)=S*TN+L*(TD+N(I,J-1)+N(I,J+1))
%REPEAT
%CYCLE I=2,2,R
TN=N(I,J)
NU(I,J)=TN*U(I,J); NV(I,J)=TN*V(I,J)
NW(I,J)=TN*W(I,J); NY(I,J)=TN*Y(I,J)
TD N(I-1,J)+N(I-1,J+1)+N(I+1,J)+N(I+1,J+1)
D(I,J)=S*TN+L*(TD+N(I,J-1)+N(I,J+1))
%REPEAT
%REPEAT
%END
%CYCLE T=1,1,G
%IF(FRAC PT(T/50))<0.01 %THEN -> 50
%CYCLE J=1,1,C
%CYCLE I=1,2, (R-1)
TN=N(I,J)
TU=NU(I-1,J-1)+NU(I-1,J)+NU(I+1,J)+NU(I+1,J-1)
TV=NV(I-1,J-1)+NV(I-1,J)+NV(I+1,J)+NV(I+1,J-1)
TW=NW(I-1,J-1)+NW(I-1,J)+NW(I+1,J)+NW(I+1,J-1)
TY=NY(I-1,J-1)+NY(I-1,J)+NY(I+1,J)+NY(I+1,J-1)
TU=(S*NU(I,J)+L*(TU+NU(I,J-1)+NU(I,J+1)))/D(I,J)
TV=(S*NV(I,J)+L*(TV+NV(I,J-1)+NV(I,J+1)))/D(I,J)
TW=(S*NW(I,J)+L*(TW+NW(I,J-1)+NW(I,J+1)))/D(I,J)
TY=(S*NY(I,J)+L*(TY+NY(I,J-1)+NY(I,J+1)))/D(I,J)
A=FA(I); B=FB(J); F=(1-A)*(1-B)
RD=REC*(TV*TW-TU*TY)
UU=F*(TU+RD)
VV=F*(TV*(TU+TW)-RD)+B*(1-A)*TV*(TV+TY)
WW=F*(TW*(TU+TV)-RD(+A*(1-B)*TW*(TW+TY)
YY=F*(TU*TY+RD(+TY*(B*(1-A)*TV+A*(1-B(*TW+A*B*TY)
TT=UU+VV+WW+YY
TNU(I,J)=TN*UU/TT; TNV(I,J)=TN*VV/TT; TNW(I,J)=TN*WW/TT
TNY(I,J)=TN*YY/TT
%REPEAT
%CYCLE I=2,2,R
TN=N(I,J)
```

PROGRAMME D

```
TU=NU(I-1,J)+NU(I-1,J+1)+NU(I+1,J)+NU(I+1,J+1)
TV=NV(I-1,J)+NV(I-1,J+1)+NV(I+1,J)+NV(I+1,J+1)
TW=NW(I-1,J)+NW(I-1,J+1)+NW(I+1,J)+NW(I+1,J+1)
TY=NY(I-1,J)+NY(I-1,J+1)+NY(I+1,J)+NY(I+1,J+1)
TU=(S*NU(I,J)+L*(TU+NU(I,J-1)+NU(I,J+1)))/D(I,J)
TV=(S*NV(I,J)+L*(TV+NV(I,J-1)+NV(I,J+1)))/D(I,J)
TW=(S*NW(I,J)+L*(TW+NW(I,J-1)+NW(I,J+1)))/D(I,J)
TY=(S*NY(I,J)+L*(TY+NY(I,J-1)+NY(I,J+1)))/D(I,J)
A=FA(I); B=FB(J);  F=(1-AO*(1-B)
RD=REC*(TV*TW-TU*TY)
UU=F*(TU+RD)
VV=F*(TV*(TU+TW)-RD)+B*(1-A)*TV*(TV+TY)
WW=F*(TW*(TU+TV)-RD)+A*(1-B)*TW*(TW+TY)
YY=F*(TU*TY+RD)+TY*(B*(1-A)*TV+A*(1-B)*TW+A*B*TY)
TT=UU+VV+WW+YY
TNU(I,J)=TN*UU/TT;  TNV(I,J)=TN*VV/TT;  TNW(I,J)=TN+WW/TT
TNY(I,J)=TN*YY/TT
%REPEAT
%REPEAT
- 51
50: %BEGIN.
%REALARRAY U,V,W,Y(1:r,1:C)
%CYCLE J=1,1,C
%CYCLE I=1,2,(R-1)
TN=N(I,J)
TU=NU(I-1,J-1)+NU(I-1,J)+NU(I+1,J)+NU(I+1,J-1)
TV=NV(I-1,J-1)+NV(I-1,J)+NV(I+1,J)+NV(I+1,J-1)
TW=NW(I-1,J-1)+NW(I-1,J)+NW(I+1,J)+NW(I+1,J-1)
TY=NY(I-1,J-1)+NY(I-1,J)+NY(I+1,J)+NY(I+1,J-1)
TU=(S*NU(I,J)+L*(TU+NU(I,J-1)+NU(I,J+1)))/D(I,J)
TV=(S*NV(I,J)+L*(TV+NV(I,J-1)+NV(I,J+1)))/D(I,J)
TW=(S*NW(I,J)+L*(TW+NW(I,J-1)+NW(I,J+1)))/D(I,J)
TY=(S*NY(I,J)+L*(TY+NY(I,J-1)+NY(I,J+1)))/D(I,J)
U(I,J)=TU;  V(I,J)=TV;  W(I,J)=TW;  Y(I,J)=TY
A=FA(I);  B=FB(J);  F=(1-A)*(1-B)
RD=REC*(TV*TW-TU*TY)
UU=F*(TU+RD)
VV=F*(TV*(TU+TW)-RD)+B*(1-A)*TV*(TV+TY)
WW=F*(TW*(TU+TV)-RD)+A*(1-B)*TW(TW+TY)
YY=F*(TU*TY+RD)+TY*(B*(1-A)*TV+A*(1-B)*TW+A*B*TY)
TT=UU+VV+WW+YY
TNU(I,J)=TN*UU/TT:  TNV(I,J)=TN*VV/TT: TNW(I,J)=TN*WW/TT
TNY(I,J)=TN*YY/TT
% REPEAT
% CYCLE I=2,2,R
TU=NU(I-1,J)+NU(I-1,J+1)+NU(I+1,J)+NU(I+1,J+1)
TV=NV(I-1,J)+NV(I-1,J+1)+NV(I+1,J)+NV(I+1,J+1)
TW=NW(I-L,J)+NW(I-1,J+1)+NW(I+1,J)+NW(I+1,J+1)
TY=NY(I-1,J)+NY(I-1,J+1)+NY(I+1,J)+NY(I+1,J+1)
TU=(S*NU(I,J)+1*(TU+NU(I,J-1)+NU(I,J+1)))/D(I,J)
TV=(S*NV(I,J)+L*(TV+NV(I,J-1)+NV(I,J+1)))/D(I,J)
TW=(S*NW(I,J)+L*(TW+NW(I,J-1)+NW(I,J+1)))/D(I,J)
TY=(S*NY(I,J)+L*(TY+NY(I,J-1)+NY(I,J+1)))/D(I,J)
U(I,J)=TU;  V(I,J)=TV:  W(I,J)=TW;  Y(I,J)=TY
A=FA(I);  B=FB(J); F=(1-A)*(1-B)
RD=REC*(TV*TW-TU*TY)
UU=F*(TU+RD)
```

PROGRAMME D

```
VV=F*(TV*(TU+TW)-RD)+B*(1-A)*TV*(TV+TY)
WW=F*(TW*(TU+TV)-RD)+A*(1-B)*TW*(TW+TY)
YY=F*(TU*TY+RD)+TY*(B*(1-A)*TV+A*(1-B)*TW+A*B*TY)
TT=UU+VV+WW+YY
TNU(I,J)=TN*UU/TT; TNV(I,J)=TN*VV/TT; TNW(I,J)=TN*WW/TT
TNY(I,J)=TN*YY/TT
%REPEAT
%REPEAT
NEWPAGE
NEWLINES(2); %PRINTTEXT' GENERATION '; WRITE(T,3)
NEWLINES(2); %PRINTTEXT'AB CHROMOSOME FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*U(I,J))-1),1,0)
%REPEAT
%REPEAT
NEWPAGE; NEWLINES(2); %PRINTTEXT'A- CHROMOSOME FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*V(I,J))-1),1,0)
%REPEAT
%REPEAT
NEWPAGE; NEWLINES(2); %PRINTTEXT'-B CHROMOSOME FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*W(I,J))-1),1,0)
%REPEAT; %REPEAT
NEWPAGE; NEWLINES(2); %PRINTTEXT'-- CHROMOSOME FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*Y(I,J))-1),1,0)
%REPEAT; %REPEAT
NEWPAGE; NEWLINES(2); %PRINTTEXT' A LOCUS FREQUENCIES  P'
NEWLINES(4)
%CYCLE I=1,1,R
NEWLINE
%IF ((FRAC PT(I/2))<0.01) %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*(U(I,J)+V(I,J)))-1),1,0)
%REPEAT
%REPEAT
NEWPAGE; NEWLINES(2); %PRINTTEXT' B LOCUS FREQUENCIES R'
NEWLINES(4)
%CYCLE I=1,1,R; NEWLINE
%IF ((FRAC PT(I/2))<0.01) %THEN SPACE
%CYCLE J=1,1,C
PRINT((INT(10*(U(I,J)+W(I,J)))-1),1,0)
%REPEAT
%REPEAT
NEWPAGE; NEWLINES(4); %PRINTTEXT'AABB PHENOTYPE FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE: %IF (FRAC PT(I/2))<0.01 %THEN SPACE
```

PROGRAMME D

```
%CYCLE J=1,1,C
TU=U(I,J); TV=V(I,J); TW=W(I,J); TY=Y(I,J)
TU=TU*(TU+2*(TV+TW+TY))+2*TV*TW
TV=TV*(TV+2*TY); TW=TW*(TW+2*TY
V(I,J)=INT(10*TV)-1; W(I,J)=INT(10*TW)-1
Y(I,J)=INT(10*TY)-1
PRINT((INT(10*TU)-1),1,0)
%REPEAT; %REPEAT
NEWPAGE; NEWLINES(4); %PRINTTEXT'AAB- PHENOTYPE FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT(V(I,J),1,0)
%REPEAT; %REPEAT
NEWPAGE; NEWLINES(4); %PRINTTEXT'A-BB PHENOTYPE FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT(W(I,J),1,0)
%REPEAT; %REPEAT
NEWPAGE; NEWLINES(4); %PRINTTEXT'A-B- PHENOTYPE FREQUENCIES'
NEWLINES(2); %CYCLE I=1,1,R
NEWLINE; %IF (FRAC PT(I/2))<0.01 %THEN SPACE
%CYCLE J=1,1,C
PRINT(Y(I,J),1,0)
%REPEAT; %REPEAT
%END
51:  %CYCLE J=1,1,C; %CYCLE I=1,1,R
NU(I,J)=TNU(I,J); NV(I,J)=TNV(I,J)
NW(I,J)=TNW(I,J); NY(I,J)=TNY(I,J)
%REPEAT
%REPEAT
%REPEAT
%IF WND=1 %THEN -> 52
NEWLINES(2)
%PRINTTEXT'END OF CALCULATION'; NEWLINE
%ENDOFPROGRAM
```

APPENDIX 3

Endler, J.A., 1973, Gene Flow and Population Differentiation, Science 179:243-250

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SCIENCE

Gene Flow and Population Differentiation

Studies of clines suggest that differentiation along environmental gradients may be independent of gene flow.

John A. Endler

Since the time of Darwin and Wallace there has been considerable interest in how species come to be different in different parts of their geographic ranges. Geographic isolation and spatial differences in environmental factors are thought to lead to the observed geographic differentiation within species, and may finally lead to speciation, when sexual and geographic isolation become complete (*1–3*). Differentiation into species is usually assumed to be impossible without barriers because gene flow is supposed to "swamp out" any differences evolved in response to local environmental factors (*1–9*).

Ehrlich and Raven (*10*), and the proponents of sympatric speciation (*11*) take exception to the view of the dedifferentiating effect of gene flow, and recent experiments (*12, 13*) and theoretical studies (*14, 15*) suggest that gene flow may not have as great an effect as has been postulated. The possibility of parapatric divergence is less commonly discussed (*1, 3, 16, 17*) and is usually assumed to have the same problems that are inherent in sympatric speciation, in particular the difficulty of accounting for the evolution of sexual isolation in the face of considerable gene flow (*1*). The crucial question is how much does gene flow actually retard the development of geographic differentiation within a species (*2*). In this article I present experimental and theoretical evidence suggesting that the effect of gene flow may be small.

Huxley (*17*) defined a cline as a gradient in a measurable character. Relative to the dispersal rate of a species, the slope of a cline between regions is indicative of the extent to which the inhabitants have differentiated. A steep cline means sharp differentiation, as in the pelage colors of the deermouse, *Peromyscus maniculatus* (*18*), and gentle clines mean indistinct divergence between areas, as in the plumages of many duck species (*19*). The basis of subspeciation and speciation is geographic variation in gene frequencies. For a polymorphic character (*20*) a cline is a temporally stable gradient of geotype or gene frequencies.

In spite of the number of clines that have been described (*1, 17, 21, 22*), there is a dearth of natural systems for which all the necessary ecological information has been recorded for each morph along a cline. Therefore I chose to study a model system that could be investigated both experimentally and theoretically. I discuss here a hypotheti-

The article was written while the author was National Science Foundation graduate fellow in the department of zoology, University of Edinburgh, Edinburgh, Scotland. The author's present address is Department of Biology, Princeton University, Princeton, New Jersey 08540.

cal diploid species distributed as a series of discrete breeding units, or demes. A stepping-stone model of dispersal (gene flow per generation to adjacent demes only) is incorporated because, for most field estimates, breeding sites are localized, and individuals dispersing from a given deme are unlikely to move beyond the adjacent demes within one generation. Those individuals that do move longer distances before settling are unlikely to become established and breed in the new area for many reasons (*10, 22–24*). This model is a closer approximation to the discontinuous distribution and limited gene flow patterns observed for many species (*22–24*) than is provided by the neighborhood model (*6*). A deme may be regarded as a spatially discrete breeding unit—an effectively panmictic aggregate of organisms lasting for at least one breeding session and connected by gene flow before and after reproduction. A given deme exchanges a given percentage, g, of its mature or breeding members with the neighboring demes each generation. The model was investigated by experimentation with *Drosophila melanogaster* and by computer simulation.

Experimental Clines in *Drosophila melanogaster*

In order to study the effects of known gene flow and selection, a series of clines were set up in *Drosophila melanogaster*, made polymorphic for *Bar* by introducing this gene and a small segment of the adjacent X chromosome into a large population (approximately 8000) of outbred "normal" flies, originating from Robertson's "Standard Kaduna" population cage. (Standard Kaduna is a large outbred population of *D. melanogaster* collected at Kaduna, North Africa, and maintained for many years.) Five sets (A through E) of 15 demes each were made up from the Bar-Kaduna population. Initially each deme contained 50 pairs of flies with a *Bar* gene frequency of 50 percent. These were allowed to mate and produce offspring for one generation (generation 0) before selection and gene flow were started. This and all subsequent generations were raised in glass vials (30 by 100 millimeters) stoppered with cotton, each vial containing about 17 milliliters of cornmeal-molasses-agar medium. Generations were discrete.

The arrangement of artificial selection and gene flow in the five sets is shown in Table 1. Each deme in each generation was subjected to (i) collection and scoring of emerging adults for each of 6 days; (ii) artificial selection (if any) on each genotype; (iii) reduction of populations to $N = 50$ pairs per deme, genotype frequencies being held constant; (iv) gene flow (if any); (v) a mating period of 24 hours; (vi) an egg-laying period of 4 days; and (vii) a developmental period at $25° \pm 1°C$, ending with emergence of next-generation adults. Differences in fitness resulted in the occurrence of *natural* selection against the *Bar* genotypes during period (vii). This natural selection must be distinguished from the artificial selection of Table 2.

Table 1. The arrangement of selection and gene flow.

Set	Artificial selection	Gene flow	Control
A	Yes	Yes	
B	Yes	No	For gene flow
C	Yes	Yes	
D	Yes	No	For gene flow
E	No	Yes	For artificial selection

To produce clines, the artificial selection was imposed in the form of a gradient across the deme series, with an increment in selection between adjacent demes of $I = 0.04$. The demes in sets A, B, C, and D were subject to the absolute survival values shown in Table 2. The symbol x represents any location of a deme within a series; d is the total number of demes in a series. In each deme, the parents of the next generation were chosen according to Table 2 as follows. Taking males and females separately, a fraction $W_1(x)$, of the total number of flies in the deme in position x consisted of *Bar* genotypes; a fraction $W_2(x)$ consisted of heterozygotes (females only as *Bar* is sex-linked), and $W_3(x)$ consisted of "normal" genotypes. *Bar* is treated as a dominant gene for the purposes of artificial selection. The null point (*25*) in the artificial selection was located at deme 3 because a preliminary estimate suggested that this selection pattern would uniformly counteract the natural selection against *Bar*, centering the resulting clines near deme 8. Artificial selection (Table 2) was continued in generations 1 through 35, except in generation 10 when no selection was made, and in generation 18 when females only were selected.

Table 2. Artificial selection in the experimental clines: proportions of each genotype surviving in each deme.

Deme position (x)	$W_1(x), W_2(x)$	$W_3(x)$
1	0.42	0.58
2	.46	.54
3	.50	.50
4	.54	.46
5	.58	.42
6	.62	.38
7	.66	.34
8	.70	.30
9	.74	.26
10	.78	.22
11	.82	.18
12	.86	.14
13	.90	.10
14	.94	.06
15	.98	.02

Gene flow was accomplished in each deme by removing $g = 0.40$ of each genotype from a given deme and placing one half of these emigrants (that is, 20 percent of N) into the deme on the left and the remaining half of the emigrants into the deme on the right. Thus adjacent demes exchanged 20 percent of their members, and a given deme contained 40 percent immigrants after gene flow each generation. The would-be emigrants from the end demes, 1 and 15, were returned to the deme from which they came.

The clines for gene frequency in generation 35 are shown in Fig. 1, and the slopes of the clines in all generations are shown in Fig. 2. Gene frequencies are calculated on the total number of eclosing adults from each deme, and the slope of a cline is the regression of gene frequency on deme number for the set concerned.

The response to selection (sets A through D) was quite marked for the first five or six generations; thereafter there was little change in the configurations of the clines. All slopes became significantly different from zero at generation 1, with the exception of set E (no selection) which became significantly different from zero in generation 4. Thereafter the slopes of sets A through D remained significantly different from zero and the slope of set E remained insignificant. There is no consistent or significant difference between the selective clines with 40 percent gene flow (A and C) and those without gene flow (B and D) from generation to generation; thus the effect of gene flow in the experiment is not detectable.

Models of Clines

A cline may result from one or more of four basic situations: random genetic sampling drift, secondary contact between formerly isolated populations, spatially discontinuous changes of environment, and continuous environmental gradients. Theory suggests that the slopes of clines produced by genetic drift fall off rapidly with increasing gene flow. For any significant and stable differentiation to evolve as a result of drift the absolute number of dispersing individuals (mN) must be less than one per generation (*6, 9*). This restriction is unlikely to be achieved in nature, and there is a very low probability that all genotypes will have exactly the same mean survival values for even a short period of time; therefore clines produced by genetic drift will not be considered here. Secondary contact between differently structured populations will only produce clines under special circumstances, and will be discussed elsewhere (*26*).

The effects of gene flow on clines resulting from sharp environmental differences have been discussed by several authors (*7, 14, 15, 27*). If there is a large difference in selective effects between two environments, then even large amounts of gene flow are unable to prevent the formation of steep clines (*14, 15*). Given a smoothly changing environmental factor, which is probably more common in nature than are sharp changes (*1, 24, 28*), there are several configurations that will produce a cline; four will be discussed here.

In each of the following models the symbols $W_1(x)$, $W_2(x)$, and $W_3(x)$ represent the probabilities of survival of the three genotypes of an autosomal locus, AA, Aa, and aa, from zygote to reproduction in each deme (*29*). Their values are dependent upon the location in the deme series, x, and form selection gradients along the series. Equilibrium of the A gene frequencies, $\hat{p}$, measured after selection and gene flow, will result from many generations of random mating, selection (W's), and stepping-stone gene flow along the linear series of d demes. The amount of gene flow will be represented by g, the total fraction of immigrants from both adjacent demes within a given deme after gene flow in a given generation. As in the experimental clines ($g = 0.40$, $d = 15$), the would-be emigrants from the end demes (1 and d) return to the deme from which they came (*30*). Thus the models differ from the experimental systems A and C only in that there is no second period of selection (no "natural" selection) before the measurement of gene frequencies. This simplification will bias the models in favor of the attenuating effects of gene flow.

The *gradient model*, 1, as in the experiments, incorporates survival functions, $W_1(x)$, $W_2(x)$, and $W_3(x)$, for genotypes AA, Aa, and aa, respectively, which are dependent only upon the position, x, in the deme series, in which the genotypes were born (Fig. 3a). Such would be the case if, for example, the probability of survival of a particular genotype increased with position along a transect up a mountainside, and the probabilities of survival of the other

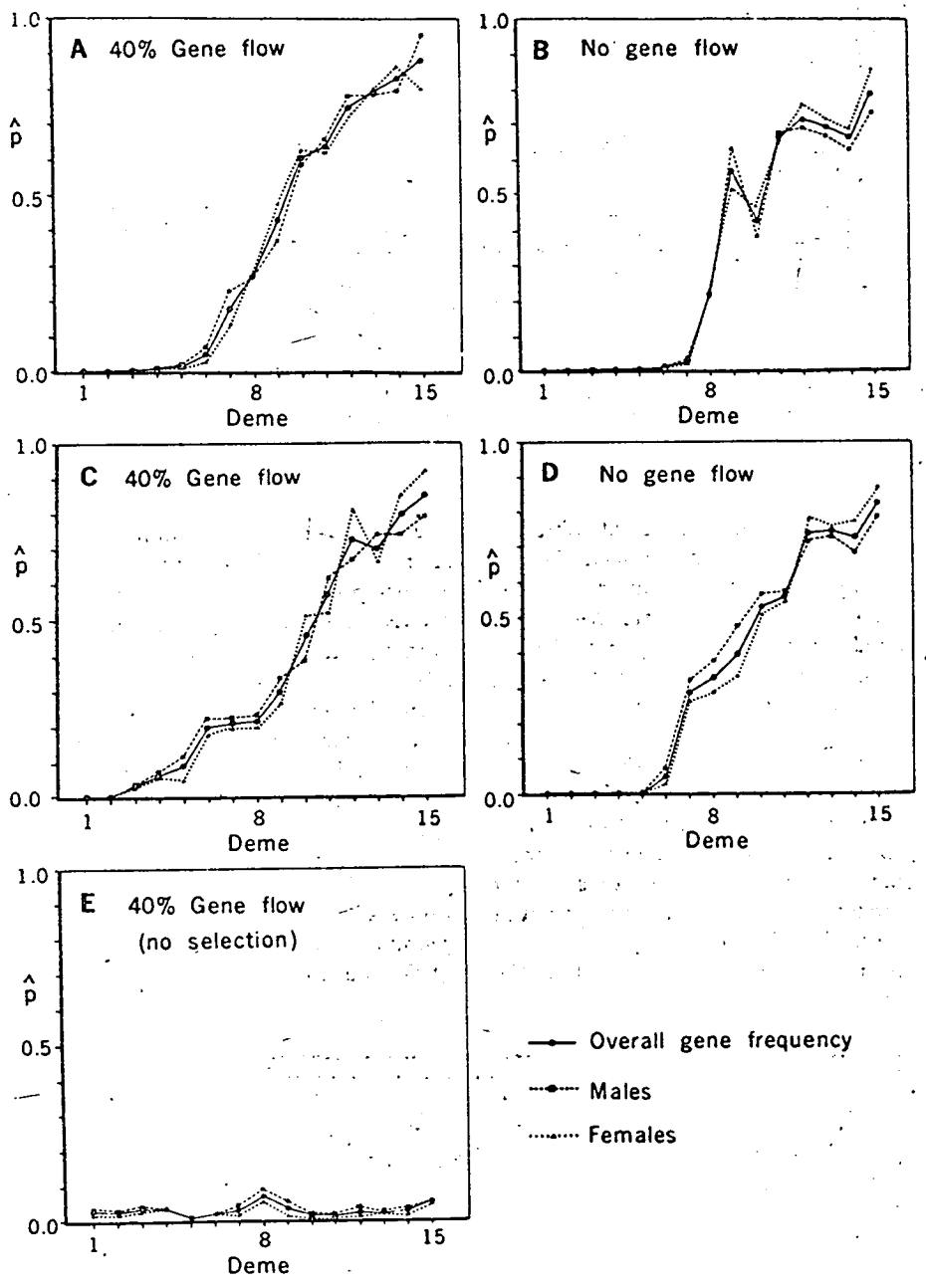


Fig. 1. The experimental clines (of *Drosophila*) showing *Bar* gene frequencies, $\hat{p}$, at generation 35. Generations 20 through 34 differ from 35 only in details. Demes 1 through 6 reached fixation for the "normal" gene within the first few generations as a result of the strong natural selection against *Bar*. These demes became polymorphic again in subsequent generations as a result of gene flow from demes 7 and above in sets A and C, and remained monomorphic in sets B and D with no gene flow. Replicates of demes 6 in sets B and D were subject to one generation of gene flow as in sets A and C during generation 15, and subsequently remained at a low *Bar* frequency. The four selective clines (A through D) are very similar, the fifth, set E, shows no sign of a cline.

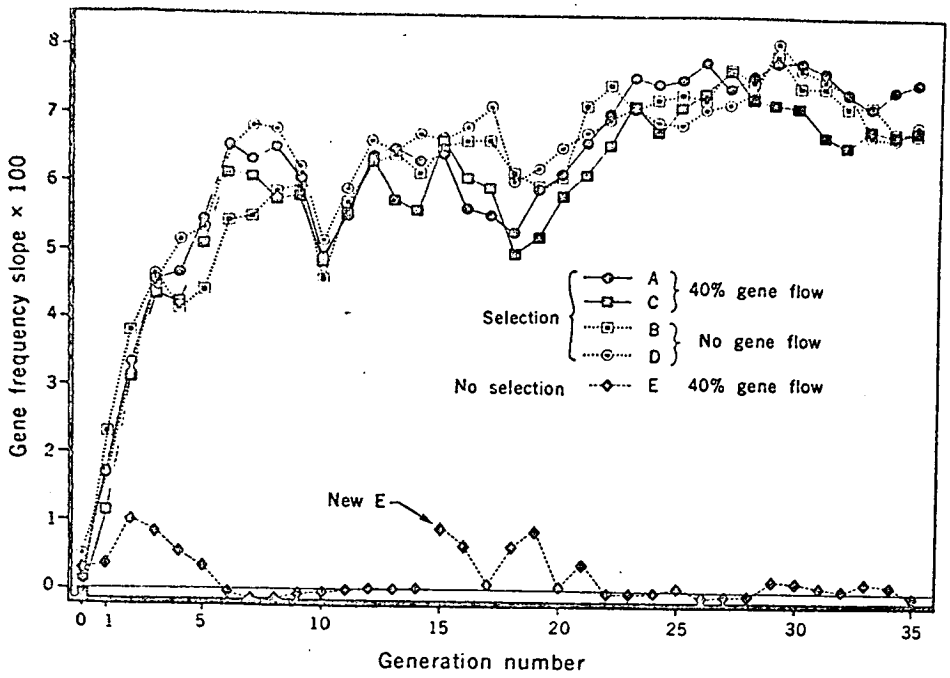


Fig. 2. The slopes of the experimental clines in each generation. The four selective clines remained very similar throughout the experiment; the effect of gene flow is not apparent. The fifth (set E) was reconstituted in generation 15 because almost all of its demes had reached fixation for the "normal" gene by generation 14. Set E was significantly different from zero slope only during generation 4.

two genotypes decreased at different rates along the same transect. This is probably one of the most realistic models. Special cases have been considered mathematically by both Fisher and Kimura (*8*) and by Slatkin (*14*).

The *heterozygous advantage model*, 2, is similar to that of many other authors (*31*). In this model the probabilities of survival of the homozygotes form selection gradients, but the heterozygote has a spatially constant fitness which is always greater than either homozygote by a minimum amount h_1 (see Fig. 3b).

A modification of model 2 is the *local heterozygous advantage model*, 3, in which the heterozygotes' survival is also position-dependent, and always a fixed amount, h_2, greater than either homozygote in the same deme (see Fig. 3c).

In the *frequency-dependent model*, 4, the probability of survival of each genotype in a deme in location x is related to its frequency in the same deme by:

$$W_i(x) = 1 - s[U_i - f_i(x)]$$

where U_i is the frequency of the ith genotype whose "focal frequency," $f_i(x)$, depends upon the genotype's position, x, in the deme series, and s is the strength of selection. The focal frequency is the optimum genotype frequency for a given deme, or the genotype frequency at which the probability of that genotype's survival is maximized (*32*) (see Fig. 3d).

Deterministic and Monte Carlo simulations of each model were executed on an ICL 4-75 computer, a wide variety of selection and gene flow parameters being used. Because the Monte Carlo simulations did not differ significantly from the deterministic runs, I will discuss only the latter. The deterministic simulations consisted of $d = 50$ demes of $N = 50$ pairs each (similar results were obtained for other values of d and N). Figure 3 indicates the kinds of selection gradients used in the simulations shown in Figs. 4 and 5. Figures 4 and 5 indicate the A gene frequencies, $\hat{p}$, at equilibrium, and the equilibrium slopes of the clines produced by various magnitudes of gene flow (g), and selection strengths in each model. The equilibrium slope is the regression of gene frequency [transformed into angles (*33*)] on deme number, calculated in the central third of the series to minimize edge effects.

Equilibrium Configurations of the Models

The gradient model produces a cline with a very marked local steepening in the vicinity of the null point for all but the very weakest selection gradients (Figs. 4a and 5a). The greatest effect on slope is found at low levels of gene flow coupled with weak selection gradients. As the slope of the selection gradient (*I*) increases the attenuation of

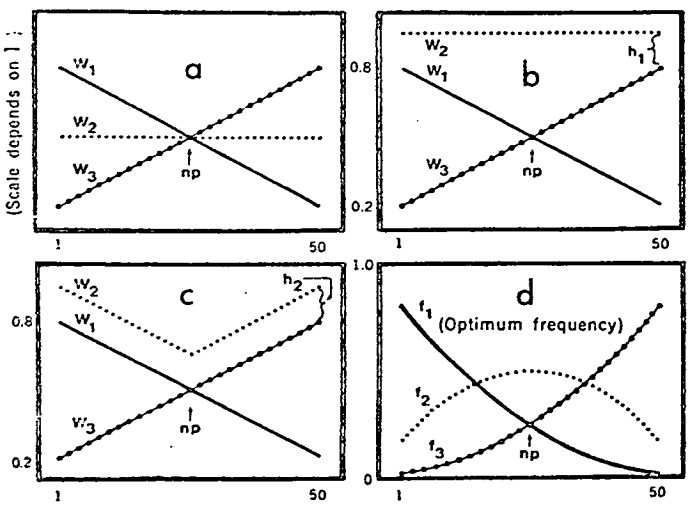


Fig. 3. The modes of selection in the four models shown in Figs. 4 to 6. (a) Gradient model; (b) heterozygous advantage model; (c) local heterozygous advantage model; (d) frequency-dependent model. In (a) through (d) the horizontal axis is the position in the deme series. In (a) through (c) the vertical axis is the absolute survival value for each genotype; W_1 for AA, W_2 for Aa, and W_3 for aa. In (d) the vertical axis is the optimum frequency or focal frequency, f, for each genotype at each deme; any deviation from these frequencies at a given position in the deme series, and the selection against the genotype, increases (see text); np is the null point; I is the increment in selection between adjacent demes; h_1 is the minimum amount by which the spatially constant fitness of the heterozygote is always greater than that of either homozygote; h_2 is the fixed amount by which the fitness of the heterozygote is always greater than the fitness of either of the homozygotes in the same deme.

slope due to dispersal along the cline is progressively reduced (Fig. 5a). For very weak gradients the differentiation may be very sharp, even for 100 percent gene flow ($g = 1.0$ in Fig. 4a). It should be emphasized that in this and the other models there is no sharp environmental change (Fig. 3).

The heterozygous advantage model produces a roughly linear cline (Fig. 4b) as pointed out by Clarke (*31*). For a given selection gradient there is negligible change of slope for increased dispersal (Fig. 5b). Gene flow has a slightly greater effect when the cline (in the absence of gene flow) is nearly flat (not illustrated). Random fluctuations in a natural cline following this model would probably obscure changes due to dispersal.

The local heterozygous advantage model produces a cline with a local steepening in the vicinity of the null point (Fig. 4c) as in the gradient model. For small values of local heterosis, h_2, this model approaches model 1 (gradient) in the clines which it produces. As a result of the local steepening, the smoothing effect of gene flow is more apparent than in model 2, especially for weak selection (h_2 in Fig. 5c), but the clines produced are nearly as insensitive to the effects of gene flow as are the clines in model 1. Like the gradient model, most of the attenuating effect of gene flow takes place for changes in small values ($0 < g < 0.3$), and progressively decreases for the same changes around large values of g. However, for very large amounts of gene flow there is still a marked local steepening (Figs. 4c and 5c).

The frequency-dependent model with distance-dependent focal frequencies (f_1, f_2, and f_3 in Fig. 3d) produces a roughly linear cline if the focal frequencies are arranged as in the Hardy-Weinberg ratios for a linearly increasing series of gene frequencies (Figs. 3d and 4d). For moderate to strong selection strengths ($s > 0.1$) the effect of gene flow is very small, but for very weak selection ($s < 0.1$) the cline may become noticeably flattened for large magnitudes of gene flow (Fig. 5d).

Dispersal is not always random or nondirectional (*22–24, 28, 34*). The process of gene flow may be divided into a nondirectional and a directional component, spatial drift (*35*). To examine the effect that a biasing environmental factor, such as wind or stream flow, may have on a cline, an asymmetry (sy) was introduced into the models. A fraction $sy \cdot g$ emigrate to the deme on the left and a fraction $(1-sy) \cdot g$ emigrate to the deme on the right of the parental deme, where g is the total fraction of moving individuals as before, and sy is a coefficient of asymmetry between 0 and 1. In the previous models and in the experiment, $sy = 0.50$, and asymmetrical gene flow is obtained by varying the parameter sy from 0.50.

Figure 6 illustrates the effect of symmetrical and several degrees of asymmetry on the gradient and heterozygous advantage models. The results for models 3 and 4 are very similar to models 1 and 2, respectively. For a given asymmetry of gene flow (sy), the entire cline is shifted in the direction of the dispersal bias in proportion to the given degree of total gene flow (g). A greater asymmetry (sy more different from 0.50) will result in an increased shifting effect for each dispersal value (g), but has little effect on the slope of the cline. Thus an asymmetry in gene flow may shift the geographic location of a cline between differentiated areas without affecting the extent of the differentiation.

Discussion

Different geographic conditions may cause differing patterns of selection which nevertheless result in very similar cline structures (Figs. 3 and 4). In addition, a given geographic pattern of selection may produce different clinal shapes under differing conditions of dominance of the characters selected, and the type and amount of gene flow (Figs. 4 and 6) (*26*). It is therefore very important that a particular model should not be applied indiscriminately to a given natural cline without specific knowledge of the actual geography of natural selection and gene flow. There is no easy way to explain geographic differentiation.

Ehrlich and Raven (*10*) cite examples of animals and plants which

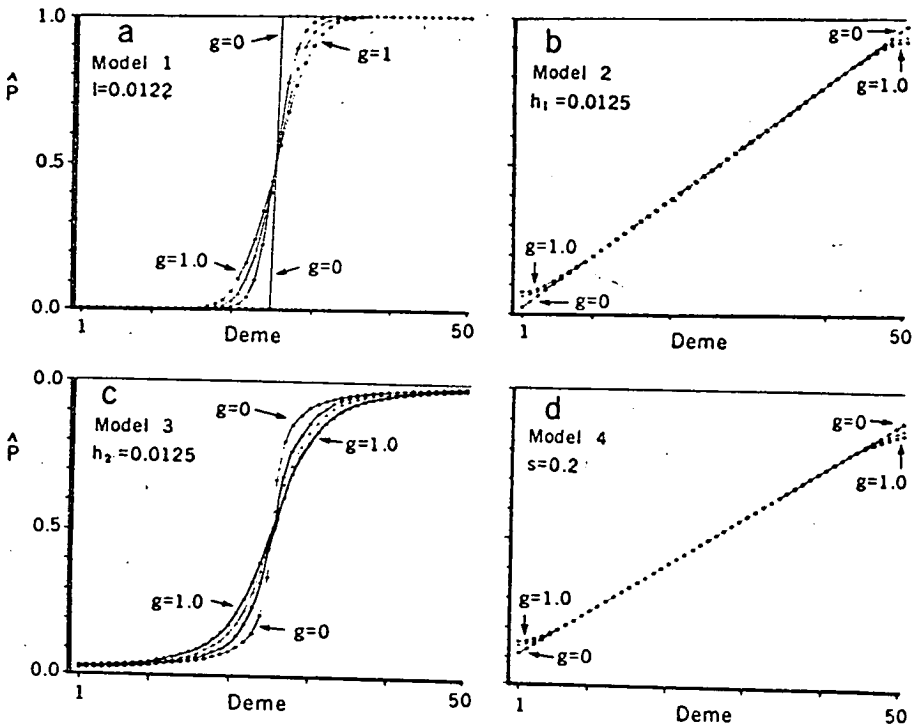


Fig. 4. Equilibrium clines produced by the modes of selection shown in Fig. 3 and various levels of gene flow. (a) Gradient model; (b) heterozygous advantage model; (c) local heterozygous advantage model; (d) frequency-dependent model. The horizontal axis is the position in the deme series and the vertical axis is the A gene frequency, $\hat{p}$, at equilibrium for various levels of gene flow from $g = 0.0$ to $g = 1.00$. A gene flow of 100 percent means that 100 percent of a given deme's population consists of immigrants after gene flow in each generation. In comparison with other experiments (*12*) where 50 percent is the maximum rate of gene flow, 100 percent is possible because there are more than two demes participating in gene flow. The clines produced by models 1 and 3 are very similar, as are the clines produced by models 2 and 4. The local steepening in models 1 and 3 have formed in the absence of sharp environmental changes in selection. All four models show excellent resistance to the attenuating effects of gene flow.

Table 3. The "swamping" effect of gene flow in relation to soil color but not to habitat. N, is sample size. [From Blair (*36*)]

Locality	Soil color	Habitat	Gene frequency	N
3 Miles N. Tularosa	Dark red	Grassy washes	0.567 ± 0.038	108
3 Miles S. Alamogordo	Pinkish gray	Mesquite	.248 ± .024	179
Salinas	Pinkish gray	Grassy	.545 ± .051	55
Lone Butte	Pinkish gray	Mesquite	.365 ± .046	57
White Sands	Creamy white	Gypsum dunes	.241 ± .086	13

have spatially differentiated, apparently in the absence of extensive barriers. They suggest that in most species gene flow is considerably more localized than is commonly thought (*10, 22–24*), and that it will probably prove to be the exception rather than the rule to find species with large amounts of gene flow and little differentiation. This is largely a matter of the difference between gene flow and true dispersal; gene flow requires a period of establishment of the new types into the new demes in addition to their reaching the new areas. There is some evidence that dispersing animals may find it difficult to become established if they move far from their birthplace (*22–24*). Mayr (*1*) and others, however, arguing for the de-differentiating effect of gene flow, cite many examples of spatial differentiation in which gene flow seems to have a marked effect.

One of the more commonly cited examples of the "swamping" effect of gene flow, Blair's study of *Peromyscus maniculatus* (*36*), needs reexamination. In the original paper he not only lists the soil colors and the estimated *gray* gene frequencies, but also the habitat types (see Table 3). Although the estimated *gray* gene frequencies do not correlate with soil color, they correlate very well with the habitat, indicating that gene flow is at least not preventing response to habitat type.

The relative magnitude of selection and gene flow alters the extent to which a given deme's gene frequency is influenced by that of its neighbors (*6, 14, 15, 27, 37*) (Figs. 4 and 5). For example, in one of Thoday's experiments (*12*), although g was 0.50, selection was of the order of 99 percent and differentiation (in this case response to disruptive selection) took place. Similarly, weak selection and strong gene flow is one of Jain and Bradshaw's simulations (*15*) produced poor local differentiation.

It is, however, an oversimplification to state that it is only the relative magnitude of gene flow and selection which affects the steepness of clines; this ignores the effects of spatial patterns of selection and gene flow. In most natural situations individuals are found grouped in favorable habitat patches connected as a network by dispersing individuals (*22–24, 28, 38*). Similarly, environmental factors such as temperature do not exist in two spatial states, but are often found in gradients (*24, 28*). A given deme with a given gene frequency may be subject to gene flow from other demes with higher gene frequencies, still others with lower gene frequencies, as well as from demes with roughly the same gene frequencies. If dispersal is relatively uniform among demes situated on a smooth environmental gradient, the net effect of gene flow will be small in each generation because the increasing effect on gene frequency by gene flow from the demes higher up on the gradient will be counteracted by gene flow from the demes lower down. In terms of Wright's formula (*5*), the mean gene frequency of the immigrants will not differ from the gene frequency of the deme receiving the immigrants on a smooth cline. This neutralizing effect will be effective for all levels of gene flow, hence clines resulting from smooth environmental gradients will be rather insensitive to the attenuating effects of gene flow, as shown by the experimental clines in *Drosophila* and the four models (Figs. 1 through 6). It is therefore possible for local differentiation, even marked differentiation, to occur along a relatively weak environmental gradient; for example, it is possible for differentiation to occur given a difference of $I = 0.008$ between adjacent demes as in Fig. 5a, an amount that might be difficult to measure in the field.

The self-canceling effect of gene flow along an environmental gradient is reduced if there is a rapid spatial change in selection or a large change in slope of the selection gradients causing the . . . This is because, in general, such

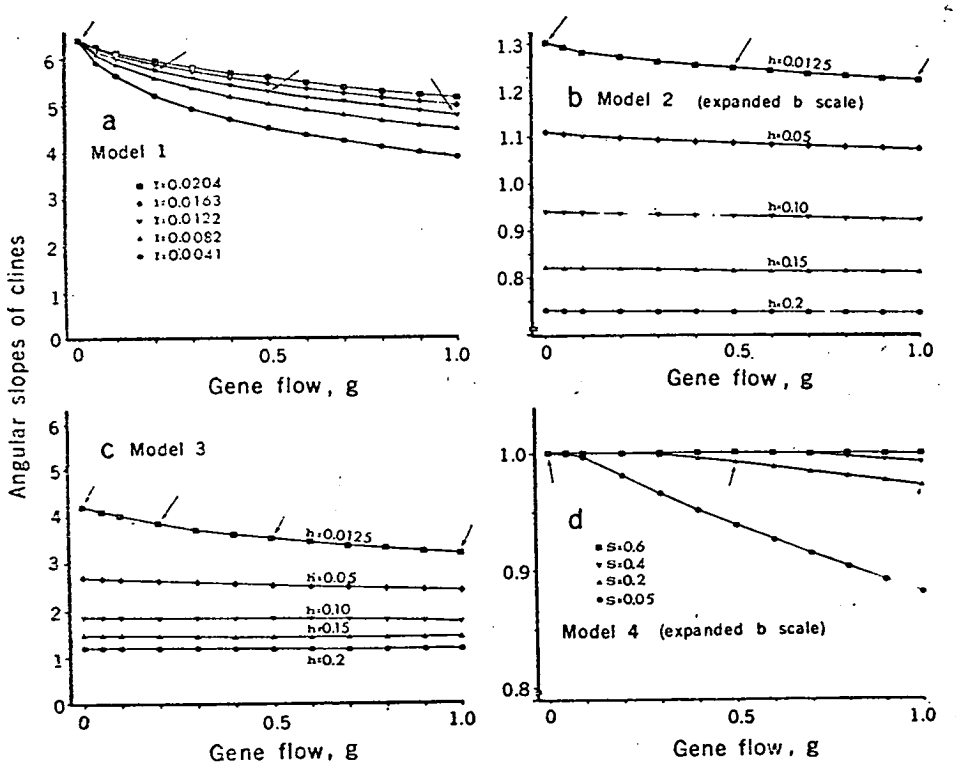


Fig. 5. The relationship between selection strength, gene flow magnitude, and the equilibrium slope of the clines for each model; (a) through (d) as in Fig. 4. The horizontal axis represents the magnitude of gene flow, g. The vertical axis represents the slopes of the clines (see text) at equilibrium. Note the expanded scales in (b) and (d), which are necessary to show any effects of gene flow in models 2 and 4. The parameter I is the strength of the selection gradient, or the difference in selection between adjacent demes; h_1 and h_2 are the strengths of heterosis (see Fig. 3) and s is the strength of frequency-dependence (see text). Arrows mark the slopes of the clines shown in Fig. 4, for comparison. The amount of heterosis (h_1 or h_2) affects the slope of clines more than any differences in gene flow in models 2 and 3. In all four models gene flow has a small effect.

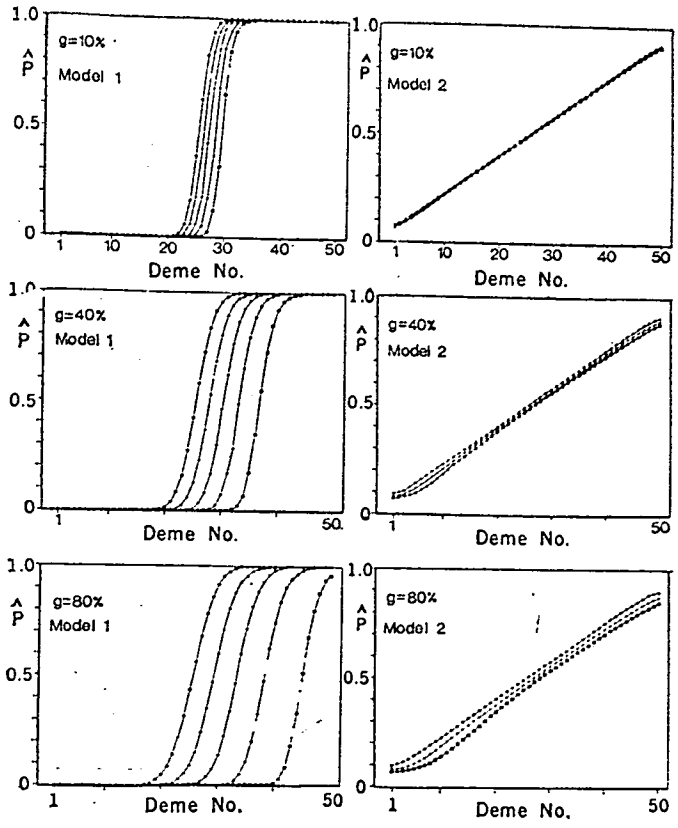


Fig. 6. The effect of an asymmetry in gene flow on the equilibrium clines of models 1 and 2. Horizontal axes represent the positions on the deme series, and the vertical axes represent the equilibrium gene frequencies as in Fig. 4. The three graphs on the left illustrate the effect of three different levels of gene flow, g, on the gradient model, and the three graphs on the right illustrate the same effect for the heterozygous advantage model. In each graph the equilibrium clines for several levels of asymmetry (sy) as well as for completely symmetrical gene flow ($sy = 0.50$) are shown for comparison. For model 1, from left to right, $sy = 0.50$, 0.40, 0.30, 0.20, and 0.10. For model 2 (three graphs on the right), from left to right, $sy = 0.50$, 0.30, 0.10. A greater amount of gene flow (g) makes the system more sensitive to the effects of a given asymmetry of gene flow, and a greater asymmetry (sy different from 0.50) results in a greater shifting of the cline for a given amount of gene flow (g). Model 1 is more sensitive to the shifting effect of asymmetrical gene flow than is model 2, but in both models the steepness of the cline is not noticeably affected by the asymmetry.

conditions increase the difference between the mean gene frequency of the immigrants from the adjacent demes and the gene frequency of the deme receiving the immigrants (*15*), as apparently found in studies of *Peromyscus* (*39*) and *Pachycephala* (*40*). The effect of gene flow may also be noticeable at the ends of a series of demes, and is a form of edge effect (Fig. 4, b and d). If there are very few demes in a cline the effect of gene flow will be very much greater. The greatest possible effect of gene flow is found in the two-deme model (*12, 13, 37*) because there can be no canceling of the effects of gene flow from areas characterized by high and low gene frequencies. Theoretical conclusions from the two-deme model are thus not applicable to species distributed among more than two demes connected by gene flow. With only a few demes the effect of gene flow may also be obvious. In Jain and Bradshaw's simulations with asymmetrical gene flow (*15*) among ten demes, the asymmetry caused a piling up of the dispersing genes at the edge of the series, greatly reducing the sharpness of the differentiation. The cline had been, in effect, shifted off the edge of the deme series. In the models discussed here ($d = 50$ demes, Fig. 6), the edge of the deme series is too far away from the null point to have any appreciable effect, except for extremely asymmetrical gene flow ($sy = 0.10$). In general, provided that differences in selection between adjacent demes (*I*) remain roughly similar, the canceling effect of equidistant but oppositely situated demes will buffer clines against the attenuating effect of gene flow.

Conclusions

There are many possible spatial patterns of selection and gene flow that can produce a given cline structure; the actual geography of natural selection and gene flow must be worked out before an attempt is made to explain a given natural cline in terms of a model.

The results of experimental and theoretical models show that it is possible for local differentiation to evolve parapatrically in spite of considerable gene flow if the selection gradients are relatively uniform. Irregularities in environmental gradients increase the sensitivity of clines to the effects of gene flow in proportion to the increase in the differences in gene frequencies between the emigrants and the demes receiving the immigrants. It is not necessary for a sharp spatial environmental change to be present for distinct differentiation to occur. In some cases even a gentle environmental gradient can give rise to marked spatial differentiation along a genetically continuous series of demes; such environmental differences may be below the practical limits of resolution in field studies. Any asymmetry in gene flow does not lead to dedifferentiation if the environmental gradient is smooth; it merely shifts the position of the transition zone between the differentiated areas from that which would be expected if there were no asymmetry. Abrupt geographic differences in gene, genotype, or morph frequencies should not, therefore, be interpreted as evidence for environmental changes in the immediate vicinity of the steepest part of the cline; neither should they be interpreted as evidence for geographic barriers, sharp environmental differences, or sexual isolation among the differentiated groups of populations when there are no other sources of evidence for these phenomena. Gene flow may be unimportant in the differentiation of populations along environmental gradients.

References and Notes

1. E. Mayr, *Amer. Natur.* 74, 249 (1940); *Systematics and the Origin of Species from the Viewpoint of a Zoologist* (Columbia Univ. Press, New York, 1942); *Animal Species and Evolution* (Harvard Univ. Belknap Press, Cambridge, 1963).
2. Th. Dobzhansky, *Genetics and the Origin of Species* (Columbia Univ. Press, New York, 1970).
3. ———, *Amer. Natur.* 74, 312 (1940); H. M. Smith, *Biologist Phi Sigma Soc.* 49, 43 (1967).
4. G. G. Simpson, *The Major Features of Evolution* (Columbia Univ. Press, New York, 1953); P. J. Darlington, Jr., *Zoogeograph*

The Geographical Distribution of Animals (Wiley, New York, 1957); B. Rensch, *Evolution Above the Species Level* (Methuen, London, 1959); V. Grant, *The Origin of Adaptations* (Columbia Univ. Press, New York, 1963).
5. S. Wright, *Genetics* **16**, 97 (1931).
6. ———, *ibid.* **28**, 114 (1943); *ibid.* **31**, 39 (1946); *Evolution and the Genetics of Populations*, vol. 2, *The Theory of Gene Frequencies* (Univ. of Chicago Press, Chicago, 1969); F. J. Rohlf and G. D. Schnell, *Amer. Natur.* **105**, 295 (1971).
7. J. B. S. Haldane, *J. Genet.* **48**, 277 (1948).
8. R. A. Fisher, *Biometrics* **6**, 353 (1950); M. Kimura, *Annu. Rep. Nat. Inst. Genet. Mishima-City, Japan* **9**, 84 (1958).
9. M. Kimura and G. H. Weiss, *Genetics* **49**, 561 (1964); M. Kimura and T. Maruyama, *Genet. Res.* **18**, 125 (1971).
10. P. R. Ehrlich and P. H. Raven, *Science* **165**, 1228 (1969).
11. For example, J. Maynard-Smith, *Amer. Natur.* **100**, 637 (1966).
12. J. M. Thoday, *Nature* **181**, 1124 (1958); ——— and T. B. Boam, *Heredity* **13**, 204 (1959); E. Millicent and J. M. Thoday, *ibid.* **16**, 219 (1961); J. M. Thoday and J. B. Gibson, *Amer. Natur.* **105**, 86 (1971).
13. F. A. Streams and D. Pimentel, *ibid.* **95**, 201 (1961); Th. Dobzhansky and B. Spassky, *Proc. Roy. Soc. London Ser. B.* **168**, 27 (1967); ———, J. Sved, *ibid.* **173**, 191 (1969); Th. Dobzhansky, H. Levene, B. Spassky, *ibid.* **180**, 21 (1972).
14. M. Slatkin, thesis, Harvard University (1971).
15. S. K. Jain and A. D. Bradshaw, *Heredity* **21**, 407 (1966).
16. Parapatric divergence is divergence between adjacent but genetically continuous populations. See H. M. Smith, *Syst. Zool.* **14**, 57 (1965); *ibid.* **18**, 254 (1969); M. J. D. White, R. E. Blackith, R. M. Blackith, J. Cheney, *Aust. J. Zool.* **15**, 263 (1967); M. J. D. White, *Science* **159**, 1065 (1968); K. H. L. Key, *Syst. Zool.* **17**, 14 (1968).
17. J. S. Huxley, *Nature* **142**, 219 (1938); *Bijdr. Dierk. Leiden* **27**, 491 (1939).
18. F. B. Sumner, *Bibliogr. Genet.* **9**, 1 (1932).
19. F. Salomonsen, *Dan. Biol. Medd.* **22**, 1 (1955).
20. E. B. Ford, *Biol. Rev. Cambridge Phil. Soc.* **20**, 73 (1945).
21. Examples of morph-ratio clines include: H. B. D. Kettlewell and R. J. Berry, *Heredity* **16**, 403 (1961); *ibid.* **24**, 1 (1969); H. B. D. Kettlewell, R. J. Berry, C. J. Cadbury, G. C. Phillips, *ibid.*, p. 15; H. N. Southern, *J. Zool. London Ser. A* **138**, 455 (1966); A. J. Cain and J. D. Currey, *Phil. Trans. Roy. Soc. London Ser. B.* **246**, 1 (1962); A. P. Platt and L. P. Brower, *Evolution* **22**, 699 (1968); O. Haikka and E. Mikkola, *Hereditas* **54**, 140 (1965); B. C. Clarke, in *Evolution and Environment*, E. T. Drake, Ed. (Yale Univ. Press, New Haven, 1968), p. 351; B. C. Clarke and J. J. Murray, in *Ecological Genetics and Evolution*, R. Greed, Ed. (Blackwells, Oxford, 1971), p. 51; J. A. Bishop and P. S. Harper, *Heredity* **25**, 449 (1969); J. A. Bishop, *J. Anim. Ecol.* **41**, 209 (1972); G. Hewitt and F. M. Brown, *Heredity* **25**, 365 (1970); G. Hewitt and C. Ruscoe, *J. Anim. Ecol.* **40**, 753 (1971); H. Wolda, *ibid.* **38**, 623 (1969); F. B. Livingstone, *Amer. J. Phys. Anthropol.* **31**, 1 (1969).
22. C. P. Haskins, E. F. Haskins, J. J. A. McLaughlan, R. E. Hewitt, in *Vertebrate Speciation*, W. F. Blair, Ed. (Univ. of Texas Press, Austin, 1961), p. 320.
23. A. J. Bateman, *Heredity* **1**, 234, 303 (1947); *ibid.* **4**, 353 (1950); R. N. Colwell, *Amer. J. Bot.* **38**, 511 (1951); M. R. Roberts and H. Lewis, *Evolution* **9**, 445 (1955); C. P. Haskins, personal communication; K. P. Lamb, E. Hassan, D. P. Scoter, *Ecology* **52**, 178 (1971). For localized distribution and problem of establishment see also: W. F. Blair, *Ann. N.Y. Acad. Sci.* **44**, 179 (1943); *Evolution* **4**, 253 (1950); L. R. Dice, *Amer. Natur.* **74**, 289 (1940); P. Labine, *Evolution* **20**, 580 (1966); H. Lewis, *ibid.* **7**, 1 (1953); W. Z. Lidicker, personal communication; J. T. Marshall, Jr., *Condor* **50**, 193, 233 (1948); R. K. Sealander, *Amer. Zool.* **10**, 53 (1970); P. Voipio, *Ann. Zool. Fenn.* **15**, 1 (1952); P. K. Anderson, *Science* **145**, 177 (1964).
24. N. W. Timofeeff-Ressovsky, in *The New Systematics*, J. S. Huxley, Ed. (Oxford Univ. Press, Oxford, 1940), p. 73.
25. The null point is the position at which selection changes over from favoring one type to favoring another.
26. J. A. Endler, in preparation.
27. L. M. Cook, *Coefficients of Natural Selection* (Hutchinson Univ. Library, Biological Sciences No. 153, London, 1971); F. B. Livingstone, *Amer. J. Phys. Anthropol.* **31**, 1 (1969).
28. W. C. Allee, A. E. Emerson, O. Park, T. Park, K. P. Schmidt, *Principles of Animal Ecology* (Saunders, Philadelphia, 1949); H. C. Andrewartha and L. C. Birch, *The Distribution and Abundance of Animals* (Univ. of Chicago Press, Chicago, 1954); G. L. Clarke, *Elements of Ecology* (Wiley, New York, 1954); R. Geiger, *The Climate Near the Ground* (translation, Harvard Univ. Press, Cambridge, 1966).
29. Results for autosomal and sex-linked systems do not differ for the models to be discussed, except that, for a given amount of selection, the sex-linked system is less sensitive to the effects of gene flow. This is because the effective gene selection on males in sex-linked loci makes the *net* selection stronger, compared to autosomal loci, for the population as a whole. See C. C. Li, *Population Genetics* (Univ. of Chicago Press, Chicago, 1955) for a good discussion of sex-linkage and selection.
30. The equilibrium configurations are not significantly altered if the emigrants from the end demes do not return, unless the number of demes (d) is very small (J. A. Endler, unpublished data).
31. See, for example, the models of B. C. Clarke [*Amer. Natur.* **100**, 389 (1966)] and those in (*14*).
32. This model incorporates Clarke's model of frequency-dependence; see B. C. Clarke, *Evolution* **18**, 364 (1964).
33. R. A. Fisher and F. Yates, *Statistical Tables for Biological, Agricultural, and Medical Research* (Oliver & Boyd, Edinburgh, 1948); R. R. Sokal and F. J. Rohlf, *Biometry* (Freeman, San Francisco, 1969).
34. See, for example, C. G. Johnson, *Migration and Dispersal of Insects by Flight* (Methuen, London, 1969); J. Antonovics, *Amer. Sci.* **59**, 593 (1971).
35. E. C. Pielou, *An Introduction to Mathematical Ecology* (Wiley-Interscience, New York, 1969).
36. W. F. Blair, *Contrib. Lab. Vertebrate Biol. Univ. Mich.* No. 36, 1 (1947).
37. P. A. Parsons, *Genetica* **33**, 184 (1963).
38. G. Hewitt and B. John, *Chromosoma* **21**, 140 (1967); *Evolution* **24**, 169 (1970); G. Hewitt, personal communication; H. Wolda, *J. Anim. Ecol.* **38**, 305, 623 (1969).
39. L. R. Dice, *Contrib. Lab. Vertebrate Genet. Univ. Mich.* No. 8 (1939), p. 1; *ibid.* No. 15 (1941), p. 1.
40. I. C. J. Galbraith, *Bull. Brit. Mus. Natur. Hist. Zool.* **4**, 133 (1956).
41. I am grateful to the National Science Foundation for a graduate fellowship in support of this study. I thank Prof. Alan Robertson and the Institute of Animal Genetics, University of Edinburgh, for the *Drosophila*, and for kindly providing me with fresh medium throughout the study. Criticism of the manuscript by Professors John Bonner and Jane Potter, Dr. Philip Ashmole, Peter Tuft, Dr. David Noakes, Dr. John Godfrey, Dr. Caryl P. Haskins, and M. C. Bathgate was very welcome. In particular, I thank my supervisor, Professor Bryan C. Clarke, for help and criticism throughout this study. Any errors or omissions are entirely my own. I thank the Edinburgh Regional Computing Center and the Edinburgh University Zoology Department for generous computer time allowances. I will supply, the specially written IMP language program upon request.

APPENDIX 4

DATA FROM THE DROSOPHILA EXPERIMENTS

A4 - A, The Drosophila clines.

The data are arranged as follows: For each generation the total number of parents (after gene flow if any) and the total number of offspring is listed for each vial in each set. For parents and for offspring the arrangement is as follows:

Line 1: Generation number (F0 to F35), parents or offspring.
Lines 2 to 6: Set A, selection, gene flow.
Lines 7 to 11: Set B, selection only.
Lines 12 to 16: Set C, selection, gene flow.
Lines 17 to 21: Set D, selection only.
Lines 22 to 25: Set E, gene flow only.

For each set:

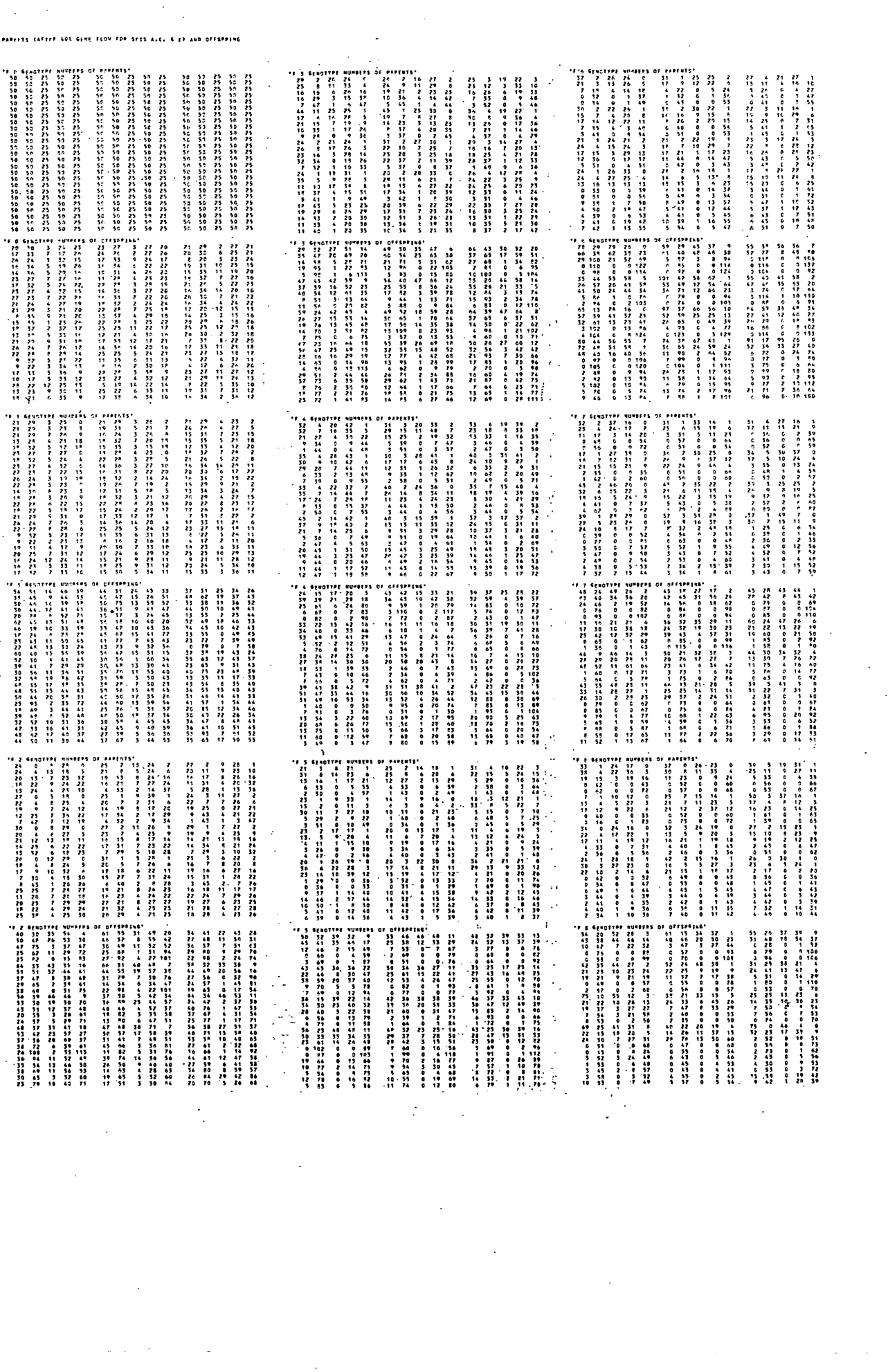
Line 1: vials 15 to 13
Line 2: vials 12 to 10
Line 3: vials 9 to 7
Line 4: vials 6 to 4
Line 5: vials 3 to 1.

For each vial the numbers of each genotype are in the order BY, bY, BB, Bb, and bb.

A4 - B, The Across-cline crosses.

The table is self-explanitory.

PARENTS (AFTER 100 GENE FLOW FOR SEXES A,C, & EP AND OFFSPRING



"F 18 GENOTYPE NUMBERS OF PARENTS"

53	47	33	51	0	56	38	33	50	0	58	45	38	45	2
68	14	22	39	3	47	43	26	42	4	44	41	11	37	9
39	51	14	41	13	26	51	5	27	23	17	54	2	22	25
9	42	0	8	42	8	57	9	4	52	1	68	0	2	63
0	47	0	0	74	0	51	0	0	89	0	85	0	0	92
53	17	39	27	0	41	24	49	29	9	43	18	21	19	1
44	27	33	25	2	28	31	17	37	4	39	28	14	33	5
26	68	4	33	21	17	45	5	42	16	9	53	2	17	37
1	136	0	0	91	0	71	0	0	55	0	79	0	0	100
0	92	0	0	37	0	68	0	0	53	0	94	0	0	86
55	25	31	28	0	56	29	30	30	0	54	33	25	31	2
50	37	24	39	2	33	35	18	46	5	27	43	13	46	9
12	56	7	37	16	34	73	3	20	30	11	77	2	15	32
9	74	0	9	49	13	45	0	7	52	6	79	0	5	77
4	65	0	2	76	0	70	0	0	75	0	64	0	0	65
52	75	20	39	0	49	23	38	24	8	47	13	48	18	0
33	7	27	13	0	42	32	54	37	1	4	2	2	8	1
27	31	9	35	7	10	42	6	18	11	7	12	4	15	30
4	48	0	7	30	0	95	0	0	79	0	87	0	0	59
0	48	0	0	54	0	61	0	0	62	0	27	0	0	61
19	45	0	25	40	20	39	1	20	46	11	36	6	13	34
9	35	2	7	23	15	47	6	12	29	11	57	0	8	24
13	67	1	3	31	14	46	1	4	19	10	54	0	3	18
11	38	0	4	16	11	34	0	4	16	9	33	0	5	15
9	32	2	5	14	8	43	0	7	24	9	34	2	6	23

"F 18 GENOTYPE NUMBERS OF OFFSPRING"

73	43	56	73	22	72	42	29	55	22	77	47	32	68	26
81	61	23	69	31	59	83	20	65	59	52	51	24	39	32
45	51	17	40	39	35	78	5	61	85	29	42	4	56	95
16	94	1	28	66	7	76	0	1	80	0	123	0	5	135
0	89	0	0	128	0	132	0	0	134	0	108	0	0	131
89	48	52	56	12	97	42	69	73	18	52	28	52	36	7
60	43	23	78	28	55	48	24	59	21	55	56	44	54	19
61	58	3	31	63	67	72	14	63	47	14	62	0	12	76
0	129	0	0	92	0	112	0	0	100	0	131	0	0	100
0	124	0	0	84	0	88	0	0	93	0	83	0	0	93
55	48	41	52	15	81	62	43	20	20	58	51	32	81	29
63	37	29	45	58	56	82	20	68	45	94	72	10	58	49
43	115	4	39	92	33	118	2	39	100	5	103	0	19	123
9	84	0	8	75	5	104	0	13	111	9	132	0	8	109
0	110	0	0	118	0	117	0	0	129	0	119	0	0	129
58	57	36	45	36	70	44	38	57	13	77	23	57	45	16
80	35	81	64	7	58	28	24	61	15	59	69	33	71	17
75	53	11	52	41	38	59	2	38	65	74	93	1	11	82
7	70	0	23	111	0	111	0	0	107	0	85	0	0	98
0	99	0	0	117	0	92	0	0	101	0	133	0	0	132
27	117	3	45	143	21	108	5	32	82	21	71	6	52	69
29	43	0	25	59	19	92	22	15	69	14	104	0	21	94
4	82	0	30	46	4	58	0	6	57	15	95	5	27	86
15	65	0	28	53	13	68	2	17	56	5	77	0	10	86
6	67	0	8	66	37	95	8	41	57	9	141	0	19	110

"F 19 GENOTYPE NUMBERS OF PARENTS"

49	0	20	29	0	49	2	18	30	1	46	3	16	34	2
47	5	14	33	3	44	8	15	32	3	39	13	13	27	8
32	18	11	25	13	28	22	5	25	18	18	32	1	23	26
11	39	2	15	33	4	48	0	9	41	1	49	0	1	49
0	50	0	1	49	0	50	0	0	50	0	50	0	0	50
50	0	25	25	0	49	1	24	26	0	48	2	29	20	0
45	5	11	37	2	47	9	15	32	3	39	11	21	25	3
33	17	3	29	20	33	17	2	30	18	11	29	0	11	34
0	50	0	1	50	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
49	1	22	28	0	48	2	19	30	0	45	5	18	30	2
40	7	13	33	3	35	10	11	32	5	36	18	6	30	13
26	23	2	27	19	16	34	2	19	29	19	38	0	14	32
5	45	0	9	40	5	43	0	5	45	1	49	0	5	47
2	48	0	2	48	0	50	0	0	50	0	50	0	0	50
50	0	14	35	0	49	1	30	20	0	49	1	28	22	1
45	5	20	39	3	41	8	16	29	4	38	12	15	32	2
18	19	7	34	9	30	20	2	25	20	17	33	1	9	39
4	44	0	13	37	0	50	0	0	50	0	50	0	0	50
0	50	0	9	50	0	50	0	0	50	0	50	0	0	50
8	47	2	12	38	11	35	1	14	31	14	35	2	15	31
11	34	5	19	28	12	40	2	12	34	6	43	8	12	33
5	45	3	11	40	4	44	5	13	34	7	33	0	10	35
8	35	1	9	30	0	29	0	5	29	5	44	0	6	40
6	41	2	10	35	5	47	0	8	42	8	42	2	12	37

"F 19 GENOTYPE NUMBERS OF OFFSPRING"

106	61	99	45	11	135	99	91	94	21	134	62	100	94	61
107	67	58	76	10	101	85	52	109	53	13	25	9	24	23
60	79	20	65	93	31	84	10	46	66	30	115	1	50	137
21	112	0	37	134	11	143	0	8	154	12	163	0	11	197
0	153	0	0	172	0	201	0	0	234	0	176	0	0	197
66	35	70	50	15	58	28	61	67	8	120	55	59	84	9
67	75	27	59	42	90	95	42	89	62	62	72	24	74	49
16	40	7	24	35	24	90	5	39	86	3	112	0	8	109
0	150	0	0	186	0	116	0	0	98	0	94	0	0	113
0	50	0	0	66	0	57	0	0	77	0	135	0	0	135
71	39	63	74	17	80	56	33	73	34	79	55	24	86	39
84	56	58	83	15	73	78	40	70	33	55	103	16	79	66
39	128	8	33	114	25	176	3	39	136	8	89	0	16	115
5	46	1	9	73	9	84	0	6	94	5	97	0	9	100
2	134	0	2	167	0	184	0	0	193	0	119	0	0	122
39	29	27	44	17	110	42	114	43	0	90	39	81	53	0
73	34	39	37	30	68	83	40	65	32	70	66	34	84	38
21	53	13	72	37	25	98	6	32	79	14	134	0	25	141
11	83	0	25	97	0	163	0	0	170	0	41	0	0	32
0	152	0	0	163	0	77	0	0	95	0	146	0	0	148
21	115	1	46	104	34	82	1	37	122	32	84	6	46	75
34	112	5	32	106	3	107	0	9	95	6	102	6	8	101
12	91	0	18	62	8	76	0	2	85	10	71	1	11	89
26	114	1	30	133	6	146	0	14	172	1	82	0	9	91
6	124	0	2	130	19	158	0	24	162	19	123	3	27	122

"F 20 GENOTYPE NUMBERS OF PARENTS"

49	1	29	21	0	49	1	29	21	0	47	3	22	26	1
46	4	21	29	2	34	8	15	30	5	36	12	12	30	8
23	19	6	30	13	25	25	6	24	21	17	33	2	22	26
10	43	0	13	37	7	43	0	7	43	2	47	0	3	47
2	48	0	1	49	0	50	0	0	50	0	50	0	0	50
50	0	30	20	0	44	1	22	27	0	48	2	20	30	0
41	6	15	31	4	47	2	13	32	5	37	13	10	33	6
24	18	11	25	14	21	29	3	23	24	2	43	0	6	44
0	50	0	1	50	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
48	1	21	29	0	47	1	18	30	0	45	4	18	31	1
43	6	16	31	3	40	10	16	28	4	31	19	10	26	15
23	27	3	21	25	14	34	3	13	36	9	41	0	11	37
6	42	1	7	43	3	47	0	6	43	3	47	0	2	48
2	48	0	2	47	0	50	0	0	50	0	50	0	0	50
45	0	21	29	0	49	1	38	12	0	47	3	28	22	0
47	3	21	27	3	38	12	17	29	3	40	10	12	34	4
18	17	13	19	13	18	32	23	20	28	8	42	0	14	36
8	42	0	16	33	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
11	39	0	14	35	12	39	1	16	34	13	38	0	13	35
9	42	2	12	38	6	50	0	7	51	4	48	0	6	49
5	49	0	6	41	7	41	0	6	34	8	42	1	8	50
6	44	1	7	49	5	45	0	8	41	1	47	0	3	47
3	48	0	5	45	3	46	0	4	44	6	44	1	8	41

"F 20 GENOTYPE NUMBERS OF OFFSPRING"

89	23	66	43	8	38	35	67	57	14	85	43	60	66	16
85	56	42	79	3	44	60	42	52	10	43	70	14	65	37
50	80	16	59	43	35	76	5	64	57	21	81	5	40	66
22	94	0	32	81	2	105	0	7	107	6	100	3	11	95
0	99	0	0	120	0	104	0	0	117	0	123	0	0	103
50	15	42	14	13	54	39	34	66	10	71	37	44	45	7
43	31	40	40	21	28	39	23	36	20	44	59	17	38	24
62	51	18	40	47	30	63	7	40	52	6	98	0	5	70
1	111	0	1	133	0	117	0	0	101	0	121	0	0	119
0	121	0	9	114	0	120	0	0	115	0	84	0	0	89
69	25	60	36	1	68	48	43	58	6	73	55	42	60	11
54	47	42	94	17	50	51	11	51	48	47	81	17	75	40
25	75	7	24	68	9	96	5	79	110	10	137	0	16	118
19	111	0	32	118	7	111	0	8	132	1	136	1	5	121
1	99	0	3	120	0	109	0	0	117	0	93	0	0	97
57	41	47	35	8	71	18	82	23	0	60	25	37	47	20
51	48	51	42	7	63	35	30	59	22	42	44	31	57	20
42	51	7	40	37	23	79	5	34	78	20	105	0	31	95
2	115	1	13	99	0	139	0	0	151	0	144	0	0	119
0	147	0	0	144	0	110	0	0	116	0	84	0	0	102
22	81	0	28	79	18	80	2	33	49	1	92	2	39	42
4	104	1	38	87	4	78	0	6	81	9	111	0	18	96
4	83	0	6	78	11	112	0	21	119	16	104	0	21	102
13	47	4	48	70	15	103	3	30	128	16	132	0	31	121
5	132	0	5	128	15	112	0	17	130	33	72	8	24	85

"F 21 GENOTYPE NUMBERS OF PARENTS"

50	0	30	21	0	48	2	27	25	0	48	2	23	26	0
44	6	21	28	0	39	11	16	33	3	34	16	13	32	4
31	19	5	35	9	23	27	5	29	15	20	30	2	27	22
8	42	1	14	35	9	42	1	11	38	0	49	0	2	48
2	49	1	2	47	0	50	0	0	50	0	50	0	0	50
44	0	38	10	0	48	2	17	32	0	47	3	25	25	0
35	5	24	25	1	25	8	20	27	3	36	14	11	35	4
37	13	10	32	8	26	24	5	29	16	5	43	0	5	44
1	49	0	1	49	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
49	1	28	22	0	48	2	24	24	0	47	3	20	30	1
43	7	15	31	4	41	9	13	33	4	34	16	8	30	12
22	28	6	27	19	13	38	4	16	30	9	41	0	16	34
7	43	0	10	40	4	46	0	7	43	2	48	0	2	47
0	50	0	2	48	0	50	0	0	50	0	50	0	0	50
50	0	28	22	0	49	1	39	11	0	48	2	21	27	1
43	7	27	22	1	45	5	16	31	2	39	11	17	30	3
35	15	6	34	10	20	30	4	23	23	13	37	0	20	30
1	60	1	8	41	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
19	41	0	15	34	8	44	1	19	31	4	46	1	17	31
2	48	0	15	35	3	48	0	7	43	2	48	0	5	45
6	46	0	6	44	4	46	1	7	42	6	44	1	13	37
5	45	0	11	37	8	42	2	14	35	4	46	0	7	43
6	46	0	7	43	8	42	2	7	41	11	39	2	10	39

"F 21 GENOTYPE NUMBERS OF OFFSPRING"

81	37	98	51	2	95	60	27	54	7	82	47	78	66	1
86	62	84	44	8	64	52	43	51	55	71	51	39	67	41
59	72	26	52	52	56	88	5	65	80	38	105	3	51	101
19	88	0	27	61	5	136	0	10	97	7	83	0	18	150
0	136	0	2	132	0	117	0	0	130	0	105	0	8	105
115	9	85	15	0	63	38	55	54	8	55	24	61	34	4
86	52	56	50	10	90	49	33	67	28	96	72	29	76	44
36	49	10	54	31	64	77	2	62	89	6	116	0	5	116
3	142	0	4	117	0	119	0	0	145	0	105	0	0	92
0	111	0	0	120	0	130	0	0	111	0	44	0	0	81
75	20	71	55	2	67	27	56	46	11	57	47	39	52	18
71	37	32	69	13	61	59	41	59	34	61	37	22	68	13
29	83	4	45	77	19	84	0	26	96	30	117	0	28	100
10	82	0	28	97	3	132	0	3	131	6	111	0	4	83
4	117	0	2	139	0	128	0	0	129	0	98	0	0	98
90	38	82	47	5	74	75	93	13	1	55	47	39	48	0
33	20	17	41	9	69	31	26	35	15	46	38	25	39	11
58	77	18	47	57	20	67	3	28	56	8	80	0	19	79
0	76	0	0	80	0	131	0	0	128	0	108	0	0	137
0	112	0	0	107	0	117	0	0	106	0	83	0	0	100
11	100	1	17	91	27	96	0	25	113	24	136	1	54	110
19	88	0	15	97	7	98	0	6	98	0	104	0	4	116
5	124	0	8	139	8	113	0	12	119	15	98	0	14	120
29	135	0	17	107	17	74	0	19	25	12	96	0	15	86
5	110	0	13	114	15	139	0	11	101	15	99	2	26	92

"F 22 GENOTYPE NUMBERS OF PARENTS"

50	0	35	15	0	48	2	34	16	0	48	2	31	16	0
44	6	29	21	0	44	6	24	20	3	38	12	19	27	5
37	13	10	28	10	27	23	7	28	18	21	29	2	24	22
11	39	0	17	33	7	43	0	14	36	2	48	0	4	45
1	49	0	5	44	0	50	0	0	50	0	50	0	0	50
50	0	43	7	0	48	2	23	27	0	38	1	33	17	0
47	3	27	22	1	47	8	17	30	3	40	10	13	31	6
29	15	7	35	8	31	19	1	33	16	4	46	0	4	46
3	47	0	4	46	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	27	23	0	47	3	25	24	0	48	2	19	31	1
44	6	19	29	2	42	7	17	32	2	33	15	12	29	9
27	21	0	26	17	19	31	1	22	28	14	35	0	15	35
8	42	0	11	39	5	45	0	7	43	3	47	0	1	49
1	49	0	2	48	1	49	0	0	50	0	50	0	0	50
50	0	34	16	0	49	1	44	6	0	46	4	25	25	0
45	5	11	32	1	37	5	20	28	3	39	8	21	26	2
34	15	11	28	11	21	29	5	22	22	8	42	0	16	34
8	42	0	4	46	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
8	42	0	5	41	8	42	0	9	39	11	39	0	9	39
5	45	0	8	42	5	45	0	5	46	3	47	0	4	46
1	49	0	2	48	5	45	0	5	45	6	44	0	6	44
11	39	0	5	39	7	39	0	7	37	4	42	0	4	38
4	42	0	4	40	5	45	0	8	42	8	42	0	8	42

"F 22 GENOTYPE NUMBERS OF OFFSPRING"

69	15	71	33	1	74	28	71	31	2	55	22	68	19	9
89	32	73	36	7	59	34	42	55	10	57	39	35	46	76
60	56	21	54	24	37	58	4	65	41	19	73	0	25	88
23	71	1	16	79	9	115	1	12	93	7	73	0	7	67
5	114	0	4	92	0	90	0	8	64	0	130	0	0	115
59	7	51	8	0	73	47	70	42	2	87	20	90	37	4
69	32	55	35	6	58	58	49	37	13	38	37	51	40	76
44	37	7	35	37	34	70	11	31	42	2	91	0	4	83
1	111	0	5	118	0	105	0	0	100	0	99	0	0	113
0	106	0	0	106	0	90	0	0	83	0	79	0	0	95
69	19	69	19	1	74	34	39	35	10	74	50	62	43	3
59	34	49	44	4	74	45	38	69	17	59	65	16	39	43
27	95	9	24	68	19	106	6	27	109	28	132	7	29	91
7	110	1	11	129	1	120	0	8	121	1	103	1	2	111
1	98	0	2	114	0	121	0	0	109	0	107	0	0	98
74	25	71	24	0	113	12	109	16	1	90	23	70	46	4
62	66	58	57	9	79	57	45	74	21	71	51	45	71	23
37	47	18	42	28	27	77	10	45	34	12	92	1	23	80
6	144	1	2	131	0	91	0	0	95	0	138	0	0	112
0	115	0	0	129	0	141	0	0	164	0	171	0	0	154
3	118	0	19	95	7	97	0	14	90	10	79	0	16	83
10	74	0	15	76	0	90	0	2	93	3	65	0	4	104
8	82	0	9	84	8	93	1	4	89	11	77	6	7	83
12	106	0	8	85	19	97	0	19	85	12	102	0	12	103
5	93	0	5	77	8	94	0	17	83	13	74	3	13	75

"F 23 GENOTYPE NUMBERS OF PARENTS"

50	0	34	15	0	48	7	35	15	0	49	1	35	14	0
46	4	29	21	1	43	7	26	22	3	42	8	19	28	2
36	14	12	31	7	27	23	7	25	18	23	27	1	23	26
11	38	1	12	37	9	41	1	8	41	2	48	0	5	44
2	49	0	2	48	1	49	0	1	49	0	50	0	0	51
50	0	43	7	0	48	2	31	19	0	49	1	35	15	0
46	4	30	20	0	44	6	27	21	2	36	8	20	26	5
38	12	6	32	12	27	23	9	26	15	2	48	0	4	46
1	49	0	3	47	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	33	17	0	48	2	34	16	0	47	3	25	25	0
46	4	25	25	1	41	9	16	30	3	35	15	12	28	10
25	25	3	26	21	18	33	5	19	25	12	39	0	13	37
6	44	2	11	37	3	47	0	2	47	1	49	0	3	48
0	50	0	1	48	0	50	0	0	50	0	50	0	0	50
50	0	39	11	0	49	1	43	7	0	49	1	31	19	0
43	7	25	25	1	43	7	18	30	2	41	9	18	29	3
35	15	13	30	7	22	28	5	25	20	10	40	1	18	32
3	47	0	1	49	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
3	47	0	13	37	7	43	0	14	36	8	42	0	13	37
6	44	0	9	42	5	45	0	9	41	3	47	0	3	47
6	46	0	6	44	8	42	1	6	43	10	40	0	7	43
11	39	0	10	40	10	40	0	11	39	9	41	0	9	41
8	42	0	11	39	9	41	1	10	39	11	39	2	14	35

"F 23 GENOTYPE NUMBERS OF OFFSPRING"

116	18	94	25	1	112	21	121	36	11	148	15	109	41	9
108	29	107	25	11	91	28	62	62	12	97	77	39	73	42
60	81	23	79	62	37	76	5	45	95	44	125	1	30	109
13	139	0	14	130	15	119	0	20	135	5	141	0	4	127
13	121	0	15	139	0	119	0	0	129	0	113	0	0	134
96	4	84	23	0	91	48	70	50	14	95	29	94	30	6
89	45	42	63	28	57	36	18	37	42	59	39	24	66	70
38	58	12	36	31	45	72	10	66	55	10	117	0	17	117
5	120	0	5	115	0	106	0	0	141	0	156	0	0	194
0	170	0	0	158	0	156	0	0	156	0	49	0	0	63
89	19	95	26	1	116	25	96	36	5	71	21	64	49	10
97	23	59	58	6	74	38	33	64	27	43	50	29	54	44
30	97	8	35	89	33	109	4	36	104	27	78	0	26	87
22	116	0	19	114	0	113	0	8	144	0	115	0	0	126
6	132	0	2	124	0	138	0	8	141	0	94	0	0	139
89	15	72	43	11	115	13	90	41	0	81	17	93	19	8
85	36	28	69	11	74	43	47	44	12	61	60	24	83	30
38	59	13	38	26	44	81	0	45	87	12	72	0	11	61
8	118	1	11	106	0	55	0	8	58	0	96	0	0	110
0	68	0	0	58	0	146	0	8	130	0	102	0	0	109
10	33	0	0	26	2	68	0	5	35	15	125	0	9	112
16	159	5	14	116	5	164	0	8	161	3	79	0	2	69
6	64	0	6	61	5	55	0	2	54	9	97	0	9	89
14	118	1	16	135	12	116	0	15	108	10	130	0	9	129
9	129	0	21	108	34	139	11	32	106	31	109	0	23	120

"F 24 GENOTYPE NUMBERS OF PARENTS"

50	0	40	10	0	49	1	39	11	0	48	2	38	12	0
49	1	33	16	1	43	7	27	20	3	40	10	18	27	5
31	19	9	29	12	27	29	11	23	22	18	32	5	16	32
13	37	6	12	39	6	44	0	8	44	5	45	0	5	45
2	49	0	2	48	2	48	0	2	48	0	50	0	0	50
59	0	39	11	0	49	1	29	20	0	48	2	39	11	0
46	4	16	30	2	44	6	14	29	7	42	8	13	34	3
32	18	11	29	10	29	21	5	33	12	8	42	0	12	38
4	46	0	4	46	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	37	12	0	49	1	35	14	0	48	2	30	21	0
48	2	22	25	1	42	8	19	28	3	36	14	12	27	11
29	21	9	25	16	22	28	2	23	25	19	31	1	16	33
11	39	0	11	39	5	45	0	4	46	1	49	0	9	50
0	55	0	1	49	1	49	0	0	50	0	50	0	0	50
50	0	34	16	0	49	1	34	16	0	49	1	42	8	0
47	3	12	37	1	44	8	21	28	1	39	11	10	36	4
32	18	16	32	7	27	23	6	29	21	10	38	0	19	37
0	51	0	8	42	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	9	50
9	50	0	10	37	7	45	0	7	40	4	49	1	5	46
3	47	0	4	46	3	47	1	6	45	3	47	0	5	47
4	56	0	2	40	4	51	0	4	45	4	54	0	3	44
5	45	0	8	46	4	46	0	3	47	3	47	0	0	44
7	43	1	6	41	8	42	1	8	40	11	39	1	9	40

"F 24 GENOTYPE NUMBERS OF OFFSPRING"

122	18	124	21	0	108	20	90	34	4	77	27	86	33	0
87	36	82	68	2	41	32	58	47	6	31	61	32	57	43
49	79	10	55	57	7	45	1	35	29	17	87	7	35	82
7	84	0	25	179	4	131	0	8	127	8	98	0	9	97
4	123	0	7	121	1	111	0	0	141	0	123	0	0	120
57	13	65	11	0	94	31	79	13	8	95	24	113	27	0
69	64	78	51	13	44	57	38	38	35	22	28	73	62	12
45	54	8	36	58	46	72	9	67	57	5	111	0	11	91
4	76	0	11	99	0	46	0	0	113	0	103	0	0	121
0	91	0	0	112	0	119	0	0	114	0	105	0	0	72
79	24	79	52	0	48	22	78	21	4	74	34	84	44	12
47	34	55	45	4	52	44	34	54	29	61	59	24	77	68
34	59	8	37	81	18	58	1	27	46	14	63	0	21	57
17	99	0	24	110	2	88	0	2	77	0	118	0	0	122
0	78	0	1	58	0	29	0	8	23	0	60	0	0	88
82	35	99	31	0	74	21	83	52	5	94	12	91	19	2
51	68	32	42	18	87	33	47	16	26	81	56	29	64	74
19	39	9	37	24	21	59	3	40	71	3	72	0	12	83
3	84	0	7	98	0	84	0	0	75	0	97	0	0	118
0	114	0	0	118	0	111	0	0	141	0	112	0	0	107
9	108	0	11	113	14	103	0	13	122	14	110	0	13	111
17	103	0	19	99	2	113	0	1	124	1	113	0	5	122
8	90	0	5	112	4	107	0	8	105	4	98	0	15	96
7	85	0	14	78	8	97	0	9	103	8	105	0	9	118
10	72	3	18	71	18	97	2	21	106	5	54	1	17	51

"F 25 GENOTYPE NUMBERS OF PARENTS"

50	0	41	9	0	49	1	38	12	0	48	2	34	16	0
46	5	30	20	0	41	9	24	23	4	36	14	10	27	5
22	18	7	32	19	19	25	7	28	17	7	32	0	24	24
8	42	2	12	38	4	45	0	8	42	2	48	0	3	47
2	48	0	2	48	1	49	0	1	49	0	50	0	0	50
50	0	43	7	0	49	1	29	21	0	46	2	40	10	0
45	5	30	19	1	39	11	19	27	4	28	7	18	30	3
35	15	5	28	17	38	20	4	33	13	3	47	0	7	41
1	50	0	4	50	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	33	17	0	48	2	32	18	0	47	3	32	18	0
45	5	24	25	1	48	10	18	28	3	37	13	8	31	10
27	15	5	38	17	20	19	1	23	24	15	25	0	19	31
9	35	0	12	37	6	64	0	5	65	1	47	0	1	47
0	41	0	0	44	0	44	0	0	46	0	43	0	0	44
50	0	38	12	0	49	1	36	14	0	49	1	41	8	0
44	5	22	27	1	48	4	20	28	2	40	10	14	32	3
18	13	8	36	7	21	26	1	27	22	6	46	0	11	38
3	47	0	4	46	0	50	0	0	50	0	50	0	9	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
9	41	0	9	41	10	40	0	8	42	11	39	0	13	37
7	45	0	7	43	4	48	0	10	40	3	45	0	2	48
3	47	0	5	44	5	45	0	8	42	3	48	0	11	39
5	45	0	10	40	4	44	0	10	40	4	43	1	9	40
9	43	0	12	38	5	44	2	13	35	4	44	1	13	37

"F 25 GENOTYPE NUMBERS OF OFFSPRING"

62	13	56	17	1	65	15	71	15	2	61	18	74	29	0
64	16	62	22	3	57	20	29	47	8	28	49	18	57	28
34	65	3	38	49	21	57	0	53	76	27	44	1	26	82
9	56	0	7	79	7	61	0	4	93	5	85	0	8	82
1	69	0	0	88	0	77	0	0	74	0	64	0	0	74
68	5	54	13	0	92	31	72	50	8	23	13	79	40	1
68	40	44	62	9	57	58	25	47	28	41	48	37	51	26
26	36	0	34	67	38	53	11	62	35	5	91	0	3	84
7	84	0	3	75	0	105	0	0	77	0	83	0	0	97
0	116	0	0	115	0	94	0	0	113	0	87	0	0	114
81	9	75	38	1	108	7	62	15	1	77	25	49	13	0
54	28	42	37	8	48	46	17	76	29	32	53	15	36	39
26	71	0	24	68	23	48	5	28	50	26	67	0	29	45
74	72	0	27	79	5	49	0	7	81	0	84	0	0	83
0	81	0	0	76	0	77	0	0	92	0	79	0	0	88
91	22	58	47	0	71	16	53	36	10	81	13	100	22	0
84	20	47	27	15	49	36	21	53	19	61	39	41	67	23
28	49	4	38	66	24	65	5	25	42	8	45	0	12	70
3	48	0	9	109	0	110	0	0	91	0	101	0	0	135
0	136	0	0	81	0	94	0	0	105	0	116	0	0	109
10	96	0	11	79	17	64	1	22	65	22	64	1	20	61
14	83	2	23	85	9	58	0	6	90	5	86	0	10	85
1	98	0	8	92	4	78	0	2	95	16	82	0	18	71
4	58	0	10	26	8	99	0	2	69	10	92	1	17	76
28	65	0	23	86	12	70	0	18	72	13	73	0	12	89

"F 26 GENOTYPE NUMBERS OF PARENTS"

50	0	40	10	0	48	2	38	12	0	49	1	39	11	0
47	3	29	21	0	37	5	23	23	3	37	10	11	32	7
19	15	6	29	16	24	23	1	25	24	9	31	0	15	35
10	40	0	8	42	3	45	0	4	46	1	49	0	2	48
2	48	0	2	49	0	50	0	0	50	0	50	0	0	50
50	0	40	10	0	49	1	29	21	0	49	1	32	18	0
46	4	20	29	1	38	11	12	34	3	41	9	20	24	6
23	12	8	27	14	29	21	7	34	9	4	48	0	1	49
5	46	0	2	47	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	36	13	0	48	2	33	16	0	48	2	33	18	0
47	3	21	33	2	38	9	17	27	4	36	13	6	33	11
22	22	7	27	17	20	25	0	26	21	17	31	2	21	26
14	36	0	16	33	4	46	0	8	42	2	48	0	2	48
0	50	0	0	49	0	50	0	0	50	0	50	0	0	49
50	0	27	23	0	49	1	28	21	0	49	1	40	9	0
48	2	31	17	2	39	11	12	34	4	40	10	12	29	3
31	19	3	30	14	23	27	4	26	20	4	39	0	13	38
3	47	0	4	46	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
4	44	0	9	41	10	48	0	11	38	9	41	0	12	39
9	41	1	8	40	4	45	0	7	43	3	47	0	4	46
3	47	0	2	48	4	46	0	8	44	4	45	0	4	46
8	45	0	5	45	5	43	0	7	42	10	43	1	6	44
10	39	0	9	40	10	40	1	7	42	8	42	0	8	42

"F 26 GENOTYPE NUMBERS OF OFFSPRING"

80	15	99	17	0	92	10	62	23	0	79	19	66	14	0
84	15	66	19	1	92	48	64	61	12	55	31	17	65	22
43	42	13	43	44	20	57	6	29	42	14	32	5	21	57
8	91	2	8	65	3	81	0	6	69	2	101	0	10	102
3	83	0	4	68	0	88	0	0	82	0	44	0	0	82
73	14	59	15	0	68	17	51	26	3	82	19	52	27	2
53	40	45	37	9	24	31	10	28	27	57	23	18	30	9
25	24	7	27	19	18	37	16	29	32	4	83	1	6	77
1	93	0	4	105	0	67	0	0	74	0	58	0	0	70
0	75	0	0	109	0	61	0	0	68	0	78	0	0	72
51	6	62	15	0	88	13	82	18	0	80	37	75	32	3
98	28	72	33	7	62	45	52	69	16	52	71	12	64	35
48	75	5	55	62	52	52	9	38	60	47	60	4	54	43
22	63	4	17	55	3	84	0	8	78	0	81	0	5	96
0	68	0	0	84	0	103	0	0	119	0	143	0	0	102
36	23	61	27	0	52	16	44	21	1	65	5	66	20	1
58	7	46	16	2	36	35	19	53	20	54	36	13	44	18
43	69	13	32	46	38	38	3	36	46	28	80	0	24	81
3	58	0	5	69	0	71	0	0	72	0	73	0	0	72
8	96	0	0	98	0	66	0	0	87	0	81	0	0	85
12	81	0	17	69	6	79	3	17	87	17	62	3	11	64
6	91	0	13	79	5	76	0	1	75	1	69	0	5	77
2	71	0	3	72	5	71	0	4	71	3	63	0	8	73
8	83	1	5	85	1	50	0	15	68	13	77	2	17	56
10	70	2	24	44	5	68	0	8	71	8	82	2	17	79

"F 27 GENOTYPE NUMBERS OF PARENTS"

50	0	60	10	0	50	0	41	9	0	49	2	39	11	0
68	2	35	15	0	44	6	25	22	3	41	9	16	30	6
39	14	8	27	9	21	18	7	21	13	10	31	3	12	20
6	36	2	8	11	3	47	1	5	39	3	47	0	3	47
0	50	0	2	48	1	49	0	1	49	0	50	0	0	50
50	0	38	11	0	49	1	36	16	0	49	1	35	17	0
44	6	27	27	1	26	6	8	26	5	45	5	12	24	2
20	7	4	24	4	27	28	12	25	11	4	46	0	4	28
1	49	0	4	46	0	50	0	0	50	0	52	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
49	0	40	11	0	48	2	39	12	0	49	1	36	13	0
46	4	29	20	0	43	7	21	25	3	36	13	12	33	6
35	15	4	31	12	30	19	4	34	13	27	24	3	22	20
17	33	7	19	30	7	43	1	8	39	1	49	0	3	47
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
50	0	35	15	0	43	1	30	19	0	49	1	38	12	0
49	1	37	13	0	37	9	13	34	3	39	11	11	30	4
33	17	8	32	10	30	17	3	26	16	21	29	0	20	30
5	26	6	3	47	0	50	0	0	50	0	50	0	0	50
0	50	0	0	50	0	50	0	0	50	0	50	0	0	50
2	48	0	4	46	2	48	0	4	44	2	48	0	3	42
2	48	0	3	47	1	49	0	7	43	2	48	0	1	48
2	48	0	4	46	3	47	0	4	45	3	46	0	3	46
1	49	1	7	43	3	47	0	4	43	1	49	0	8	42
3	47	0	4	44	2	48	0	6	44	2	47	0	6	44

"F 27 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 28 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 28 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 29 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 29 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 30 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 30 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 31 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 31 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 32 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 32 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 33 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 33 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 34 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 34 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

"F 35 GENOTYPE NUMBERS OF PARENTS"

[illegible]

"F 35 GENOTYPE NUMBERS OF OFFSPRING"

[illegible]

APPENDIX 4 - B

DATA FROM THE ACCROSS-CLINE CROSSES

A,B,C,D,E = Sets A,B,C,D and E.

Numbers after set indicate vial numbers in the original clines.

Cross type	F_1 Set A GENOTYPE NUMBERS	FMASS	MEDIUM	F_2 Set A GENOTYPE NUMBERS	FMASS	MEDIU
14 x 14	47 11 35 23 2	0.108	4.707	54 13 29 27 2	0.125	5.1(
	52 15 44 25 3	0.145	5.048	54 14 40 23 3	0.129	4.9(
	47 16 33 19 4	0.115	4.945	56 28 41 24 7	0.136	4.6(
	45 12 38 22 4	0.117	4.314	62 19 38 34 7	0.149	5.0(
14 x 4	0 50 0 58 20	0.133	4.889	30 43 1 24 46	0.127	4.8(
	0 45 0 50 16	0.116	4.788	13 32 0 23 41	0.125	4.9(
	0 78 0 31 32	0.103	5.442	33 57 2 19 50	0.144	4.9(
	0 83 0 29 34	0.129	4.674	30 61 0 22 48	0.149	4.8(
4 x 14	52 31 0 61 21	0.154	5.163	29 39 17 42 8	0.128	5.1(
	43 31 0 61 18	0.140	4.803	31 43 22 21 15	0.123	5.0(
	48 16 0 62 18	0.139	5.220	49 39 20 45 11	0.141	4.7(
	77 21 0 51 14	0.147	5.098	33 47 15 47 20	0.139	5.2(
4 x 4	0 74 0 0 76	0.148	5.165	0 67 0 0 68	0.131	5.1(
	0 66 0 0 73	0.136	5.144	0 60 0 0 82	0.137	5.1(
	0 71 0 3 62	0.123	4.716	2 73 0 2 84	0.151	4.8(
	0 65 0 0 81	0.163	4.595	0 31 0 1 30	0.070	5.0(
11 x 11	57 18 34 32 8	0.139	5.135	52 22 27 30 13	0.141	4.9(
	44 17 41 26 5	0.122	5.014	36 25 27 35 8	0.131	5.0(
	43 15 28 28 10	0.118	3.918	42 39 28 31 20	0.131	4.8(
	44 14 38 20 13	0.134	5.201	42 25 36 27 5	0.131	5.0(
11 x 7	18 53 9 29 41	0.144	4.860	19 53 5 43 37	0.134	4.7(
	19 62 12 23 49	0.161	5.111	23 38 1 28 33	0.120	4.8(
	57 27 5 39 14	0.136	5.180	11 26 1 23 19	0.082	4.7(
	15 54 5 35 32	0.139	4.952	11 52 2 31 27	0.121	4.5(
7 x 11	51 23 1 60 13	0.149	4.945	34 40 13 34 10	0.127	4.9(
	52 16 5 46 10	0.128	5.066	32 42 12 37 11	0.130	4.8(
	13 62 10 33 36	0.158	4.717	27 35 18 33 22	0.129	4.9(
	46 28 2 51 18	0.137	4.973	36 45 18 24 20	0.132	4.8(
7 x 7	5 64 0 9 51	0.131	4.811	8 56 0 19 44	0.120	4.8(
	9 65 0 40 46	0.132	4.698	6 54 1 22 44	0.119	4.9(
	5 44 0 9 44	0.117	5.264	9 58 0 19 60	0.134	4.6(
	5 57 0 10 48	0.112	4.654	13 64 0 20 61	0.151	4.6(

Cross	F_1 set B							F_2 set B						
14 x 14	61	21	41	25	9	0.148	5.280	35	31	34	40	7	0.132	4.8
	57	30	46	34	6	0.163	5.134	42	21	20	28	7	0.120	5.1
	47	26	50	38	11	0.166	4.948	35	25	31	28	9	0.134	5.0
	47	19	42	46	9	0.152	4.737	24	33	28	29	9	0.139	5.0
14 x 4	0	79	0	67	12	0.145	5.096	36	44	0	32	46	0.144	5.0
	0	77	0	74	13	0.150	5.112	26	45	0	25	48	0.128	5.0
	0	77	0	80	15	0.156	5.076	26	55	0	25	59	0.131	5.1
	0	85	0	76	16	0.157	5.016	14	45	0	26	42	0.122	4.8
4 x 14	48	24	0	55	18	0.134	5.179	28	46	24	45	17	0.142	5.2
	50	21	0	47	30	0.133	4.993	30	38	20	50	13	0.133	5.0
	62	27	0	64	20	0.154	4.834	17	28	15	44	16	0.118	4.9
	57	22	0	65	27	0.155	4.802	21	43	10	33	16	0.122	5.1
4 x 4	0	63	0	0	64	0.114	5.151	0	94	0	0	76	0.144	5.0
	0	84	0	0	70	0.140	5.127	0	69	0	0	78	0.133	4.8
	0	76	0	0	85	0.145	4.904	0	89	0	0	78	0.143	4.8
	0	75	0	0	89	0.151	5.234	0	72	0	0	88	0.151	5.0
11 x 11	38	30	27	47	8	0.135	5.330	37	20	25	43	11	0.122	5.0
	50	18	37	32	7	0.121	5.115	28	22	26	47	4	0.117	5.1
	46	36	33	32	15	0.146	4.975	40	25	20	39	4	0.118	5.0
	54	21	35	36	4	0.136	5.043	31	23	22	30	9	0.116	5.2
11 x 7	6	77	4	35	28	0.145	5.357	26	52	1	23	62	0.150	5.1
	5	64	2	34	37	0.141	5.276	20	61	0	26	53	0.146	5.0
	7	69	3	52	23	0.148	5.001	20	55	1	21	41	0.127	4.8
	8	62	4	48	27	0.149	5.049	24	54	0	16	45	0.135	5.0
7 x 11	19	13	6	17	12	0.055	5.120	32	38	21	49	11	0.130	5.0
	50	15	11	56	16	0.140	5.139	36	43	28	54	7	0.142	5.0
	49	20	12	68	19	0.144	5.031	31	40	35	40	14	0.130	4.6
	53	20	17	58	25	0.164	4.994	35	46	19	42	12	0.151	4.9
7 x 7	12	77	0	32	53	0.168	5.561	15	49	1	28	43	0.128	5.3
	9	76	0	17	58	0.154	5.255	14	43	1	25	59	0.139	4.9
	18	72	0	20	45	0.165	4.829	7	37	2	12	43	0.102	4.8
	11	76	0	15	64	0.146	4.887	5	49	2	23	59	0.125	5.0

	F_1 set C							F_2 set C						
14 x 14	34	28	32	27	10	0.121	4.918	28	28	15	16	11	0.091	5.38
	34	23	22	14	15	0.117	5.131	47	38	17	35	16	0.144	5.64
	45	24	54	19	13	0.128	4.625	20	29	14	19	5	0.086	5.10
	48	27	37	37	19	0.136	4.823	30	17	3	17	12	0.077	5.08
14 x 4	4	77	0	46	26	0.129	4.828	17	55	1	38	53	0.137	5.54
	2	65	0	54	20	0.130	5.238	21	49	5	16	37	0.118	5.18
	3	72	0	49	42	0.087	4.534	11	40	1	14	36	0.090	5.13
	7	74	0	51	28	0.167	4.696	10	41	0	22	37	0.103	5.00
4 x 14	42	19	1	53	22	0.190	4.977	20	33	18	26	20	0.107	5.17
	58	27	2	46	14	0.126	4.827	24	40	27	49	11	0.148	5.39
	58	18	2	51	15	0.123	4.719	19	29	15	31	9	0.097	5.05
	46	20	4	52	20	0.116	4.716	32	15	15	43	8	0.121	5.05
4 x 4	0	64	0	0	55	0.090	4.822	1	61	0	0	60	0.119	5.22
	0	61	0	0	52	0.114	5.038	0	67	0	0	67	0.127	5.14
	0	76	0	0	65	0.124	4.894	0	99	0	0	78	0.181	5.16
	0	64	0	0	62	0.126	4.686	0	33	0	1	35	0.078	5.07
11 x 11	33	37	15	26	27	0.125	4.732	30	48	12	32	16	0.128	5.23
	38	27	15	39	24	0.136	5.008	23	36	13	29	21	0.113	5.25
	35	34	20	44	23	0.142	4.821	9	22	5	32	15	0.085	4.83
	38	23	21	34	16	0.132	5.007	20	32	11	21	15	0.094	5.04
7	7	82	0	17	55	0.140	4.861	7	54	0	27	57	0.131	5.04
	4	70	2	34	42	0.121	4.629	15	61	2	18	59	0.141	5.38
1		69	2	28	51	0.141	4.858	7	51	3	8	59	0.128	5.22
	14	63	3	19	29	0.123	4.943	21	45	4	20	40	0.137	5.38
7 x 11	10	25	1	26	37	0.110	4.946	11	49	4	34	31	0.126	5.04
	15	30	0	16	21	0.093	4.832	17	43	7	32	26	0.115	5.08
	28	29	2	30	38	0.121	4.646	14	61	7	33	39	0.127	4.41
	29	37	0	30	42	0.123	4.503	18	47	8	12	21	0.102	5.13
7 x 7	5	59	2	9	63	0.134	4.824	3	56	0	16	43	0.109	5.31
	10	61	2	16	46	0.121	4.967	8	55	3	13	52	0.126	5.07
	7	84	0	10	59	0.141	4.779	1	50	1	9	57	0.122	5.06
	15	63	3	23	56	0.120	4.873	1	38	0	13	38	0.089	4.93

	F_1 set D							F_2 set D						
14 x 14	36	21	35	25	10	0.139	5.113	38	26	23	40	14	0.128	5.29
	39	24	25	26	14	0.146	5.138	37	27	25	45	8	0.141	5.08
	45	27	35	42	5	0.133	4.409	41	26	24	36	16	0.134	4.53
	44	28	35	34	4	0.142	4.820	52	28	19	41	5	0.143	5.07
14 x 4	0	63	0	47	31	0.154	5.335	28	42	0	27	50	0.145	5.23
	0	77	0	51	30	0.167	5.263	22	47	0	18	59	0.139	5.12
	0	77	0	56	41	0.155	4.114	20	33	0	27	44	0.111	4.75
	0	75	0	47	38	0.144	4.667	25	53	0	18	42	0.126	5.22
4 x 14	55	19	0	60	27	0.152	5.061	26	48	11	42	19	0.140	4.97
	42	18	0	55	30	0.147	5.165	26	44	10	45	18	0.135	5.32
	55	29	0	57	32	0.157	4.618	25	62	17	38	20	0.144	4.86
	53	20	0	58	32	0.152	4.425	27	44	12	41	11	0.128	4.59
4 x 4	0	72	0	0	71	0.142	5.143	0	64	0	0	55	0.126	5.27
	0	74	0	0	72	0.167	5.022	0	61	0	0	73	0.132	5.08
	0	70	0	0	80	0.155	4.694	0	62	0	0	60	0.116	4.77
	0	88	0	0	69	0.137	5.000	0	78	0	0	71	0.136	4.86
11 x 11	49	22	16	20	29	0.137	5.307	26	44	9	29	18	0.122	5.06
	48	20	13	34	25	0.134	4.973	41	32	9	34	8	0.115	5.18
	55	27	13	40	18	0.130	4.483	48	37	12	35	16	0.136	5.15
	65	24	18	37	20	0.129	4.765	30	44	15	44	10	0.125	4.81
11 x 7	26	53	8	26	41	0.168	5.648	23	38	3	23	26	0.105	5.14
	23	40	13	40	34	0.149	5.199	19	56	10	33	36	0.147	5.36
	21	49	6	31	43	0.129	4.153	27	48	11	28	24	0.125	4.69
	20	35	5	22	18	0.103	4.840	16	57	2	37	41	0.144	4.89
7 x 11	38	22	5	37	38	0.143	5.380	23	33	7	24	22	0.134	5.04
	27	33	6	42	27	0.139	5.340	21	51	13	20	18	0.118	5.05
	25	39	3	30	25	0.108	4.592	22	50	14	30	7	0.123	5.40
	51	37	8	35	23	0.126	4.447	14	35	7	29	23	0.108	4.83
7 x 7	22	51	6	33	40	0.153	5.508	17	37	5	25	34	0.115	5.02
	18	58	4	39	28	0.140	5.089	24	35	8	18	17	0.099	5.21
	32	56	4	33	41	0.114	5.260	15	38	4	29	25	0.111	4.66
	34	48	5	42	37	0.145	4.828	31	38	5	39	31	0.123	4.56

	F_1 Set E					F_2 Set E				
14 x 14	0 59	0	6 54	0.126	4.721	5 44	2	6 53	0.104	5.43
	1 55	0	6 64	0.128	4.936	4 33	0	2 43	0.081	5.34
	0 95	0	0 64	0.146	4.722	2 47	0	4 37	0.086	4.66
	1 35	0	8 50	0.090	4.534	2 38	3	5 44	0.084	5.02
14 x 4	1 53	0	4 45	0.113	4.733	4 49	0	7 42	0.092	4.81
	4 95	0	5 66	0.138	4.461	8 52	0	9 46	0.110	5.15
	2 40	0	7 40	0.101	4.353	2 57	0	21 62	0.132	5.07
	5 50	0	6 45	0.133	4.444	1 22	0	7 20	0.053	5.25
4 x 14	0 71	0	0 69	0.144	4.701	0 61	0	0 40	0.080	5.02
	0 54	1	1 62	0.123	4.693	0 56	0	0 69	0.113	4.99
	0 44	0	0 61	0.107	4.519	0 38	0	0 38	0.077	5.33
	0 75	0	0 63	0.141	4.575	0 32	0	0 47	0.084	4.92
4 x 4	0 77	0	3 84	0.165	4.543	1 27	0	4 38	0.078	4.93
	5 71	0	14 71	0.152	4.335	4 54	0	6 63	0.126	5.05
	5 62	0	6 66	0.137	5.503	0 36	2	5 42	0.084	4.88
	2 49	0	5 53	0.110	5.343	2 52	1	5 46	0.100	4.81
11 x 11	0 50	0	4 59	0.211	4.952	1 51	0	1 77	0.126	5.18
	0 73	0	0 83	0.184	4.780	2 41	0	2 43	0.085	5.05
	0 86	0	4 68	0.140	4.616	1 76	0	1 78	0.142	5.08
	0 88	0	0 81	0.140	4.608	1 64	0	0 52	0.115	4.94
11 x 7	2 59	0	1 44	0.115	4.736	1 58	0	0 57	0.118	5.27
	2 67	0	0 74	0.118	4.731	1 45	0	1 62	0.099	5.00
	7 69	0	6 84	0.103	4.415	4 60	0	1 60	0.118	4.97
	1 61	0	1 68	0.121	4.255	3 50	0	2 59	0.114	5.09
7 x 11	0 78	0	2 94	0.156	4.798	3 43	0	3 54	0.108	5.27
	0 86	0	4 78	0.135	5.374	0 35	0	0 31	0.063	5.12
	0 83	0	7 79	0.152	4.694	6 63	0	2 67	0.139	4.92
	0 78	0	2 68	0.136	5.351	0 51	0	2 72	0.103	4.86
7 x 7	0 64	0	0 69	0.132	4.757	0 72	0	4 59	0.133	5.67
	2 51	0	0 49	0.111	5.025	0 51	0	8 52	0.110	5.06
	0 75	0	0 94	0.154	4.721	1 76	0	5 65	0.140	5.11
	1 69	0	1 85	0.128	4.972	0 54	0	2 58	0.117	4.47