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A MODEL FOR THE EVOLUTION OF DIOECY AND GYNODIOECY

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> There is much difficulty in understanding why hermaphrodite plants should ever have been rendered dioecious. [CHARLES DARWIN, 1877]

Comparative evidence suggests that dioecy in flowering plants has evolved independently in a number of different groups, from an hermaphrodite or monoecious ancestral condition (Lewis 1942; Westergaard 1958). It is obvious that at least two gene mutations are necessary to transform an hermaphrodite or monoecious species into one with separate sexes; one mutation must affect ovule production, and the other the production of pollen. Studies of sex determination in several species have given results which support the view that two mutations with these effects have indeed been involved (Westergaard 1958). Since it is extremely unlikely that two such mutations should occur and become established simultaneously, it seems likely a priori that the evolution of dioecy from hermaphroditism has involved an intermediate type of population that contained both hermaphrodites and some male-sterile or female-sterile plants. The former condition is known as gynodioecy, the latter as androdioecy. Androdioecy is extremely rare in nature (Darwin 1877), and some reasons why this is to be expected are given later in this paper (see also Lloyd 1975). It thus seems most likely that dioecy has usually evolved from gynodioecy. This was originally suggested by Darwin (1877) and has been discussed recently by Carlquist (1966), Ross (1970), Lloyd (1974*a*, 1974*b*, 1975), and Arroyo and Raven (1975). This idea is given further support by the fact that the relatives of dioecious species are not infrequently gynodioecious, though gynodioecy is in general a rare condition. Some examples are given in Section V below. Another example is *Silene vulgaris*, a gynodioecious species related (but not closely) to the dioecious species *S. alba* and *S. dioica*; *Rumex hastatus* and *R. lunaria* are also gynodioecious (Smith 1967).

In the present paper we explore a model for the evolution of dioecy from the hermaphrodite condition, via gynodioecy, using both analytical methods and computer calculations of population trajectories. Previous theoretical treatments of the evolution of dioecy include Lewis (1942), Lloyd (1974*a*, 1974*b*, 1975), Ho and Ross

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(1974), and Ross and Weir (1976). An analysis of the conditions for the spread of mutations affecting male and female fertility has not previously been attempted. This and a study of how these conditions relate to the selective pressures influencing the evolution of dioecy are the main points of the present paper. We imagine that the initial step taken by a species that is evolving dioecy is the occurrence of a mutation abolishing pollen production, i.e., a male-sterility factor. This can be either dominant or recessive. The conditions for the establishment of such a gene in a population, which would then be gynodioecious, are discussed in Section II, where we also show that a female-sterility factor is unlikely to become established. In the rest of the paper we discuss the fate of a mutation at a second locus, introduced into a gynodioecious population and modifying hermaphrodites in the direction of increased maleness. We show how dioecy can evolve by this mechanism. One of the main questions which we explore is: Why are the different genes determining sex grouped together in one region of one chromosome in most species? Our results suggest that there may often be a "linkage constraint" such that a modifier mutation of the sort described above would be eliminated from a gynodioecious population unless it occurred at a locus that is fairly closely linked to the gene controlling the gynodioecy.

Dioecy and gynodioecy have often been regarded in the botanical literature as outbreeding devices (Lewis 1942; Baker 1959). In order to examine this idea we have incorporated partial self-fertilization and inbreeding depression into our models. The role of these factors in the maintenance of gynodioecy and androdioecy has also been investigated by Lloyd (1975, 1976). Darwin (1877) felt that redistribution of reproductive resources as a consequence of failure of pollen or ovule production—causing, respectively, increased seed or pollen production—was an important factor in the evolution of dioecy and gynodioecy. This idea is a feature of many previously published models of the evolution of dioecy and has recently been explored in detail by Charnov et al. (1976) for the random mating case. It will be seen that, while this type of effect is almost certainly essential for the evolution of dioecy, inbreeding effects probably also play a major role.

I. MODEL AND ASSUMPTIONS

Formulation of the Model

Throughout this paper we assume an infinitely large population which is partially self-fertilizing and which is segregating for loci affecting ovule and pollen production. We define the following parameters for individuals of genotype i: z_i = the frequency of i adults in a given generation; b_i = the mean pollen output of an i individual; e_i = the mean ovule production of an i individual. Provided that the genotype i is not male-sterile, a nonzero fraction s_i of its ovules is assumed to be fertilized by its own pollen. The same value, s, is assigned to all genotypes that produce pollen. The pollen used in selfing is assumed to be a negligible fraction of the pollen output of a plant. The last parameter required is the inbreeding depression, δ. We write $1 - \delta$ for the mean probability of survival of a zygote produced by self-fertilization, relative to that of a zygote produced by outcrossing. We assign a fixed value to δ for a given population, independent of the genotype of the zygote and its parents and of the overall composition of the population. This is clearly in general only a realistic

procedure for an equilibrium population. The introduction of a gene which affects the level of outcrossing will change the value of δ; we would normally expect δ to be positively correlated with the mean level of outcrossing of the population. In our computer runs, where we treat δ as a constant during the evolution of dioecy or gynodioecy, we are therefore probably erring on the conservative side as far as estimating the selection pressure in favor of outbreeding is concerned. Furthermore, this procedure ignores the genetic basis of inbreeding depression and in particular the effects of linkage between the loci we are studying and those responsible for the inbreeding depression. On account of the complex genetic control of inbreeding depression, this is probably the best that can be done in the way of modeling. Our results should provide an accurate account of the possible equilibria in the systems we study and should also be reasonably accurate with regard to the introduction of a rare mutant gene into a population at equilibrium. There seems no reason to suppose that our qualitative conclusions will be seriously affected by this simplification in the representation of inbreeding depression.

Definition of Fitness

In the rest of this paper a fundamental concept is the "fitness" of a genotype. For a partially self-fertilized population, we can define w_i, the net fitness of genotype i, as the fraction of the gametes present among adults of the next generation which are contributed by individuals of genotype i, divided by the fraction of the total number of gametes contained in i adults in the present generation. The mean fitness of the population on this definition is always one; w_i can be obtained as follows.

Among the gametes that go to form adults of the next generation, the fraction contributed through the female gametophytes produced by type i individuals is:

$$[s_i(1-\delta)e_i z_i + (1-s_i)e_i z_i]/W, \tag{1a}$$

where

$$W = T + U, \quad T = \sum_k s_k(1-\delta)e_k z_k, \; U = \sum_k (1-s_k)e_k z_k.$$

The female "fertility" of genotype i, w_{fi}, is this quantity divided by z_i. The male fertility, w_{mi}, can be calculated in a similar way, noting that the pollen contributions have to be normalized so that the total contribution of gametes through male gametophytes is equal to that through female gametophytes. We therefore have:

$$w_{mi} = \left[\frac{Ub_i}{V} + s_i(1-\delta)e_i\right]/W, \tag{1b}$$

where

$$V = \sum_k b_k z_k.$$

Since all zygotes contain an equal contribution of genetic material from male and female gametes, we obtain the net fitness of genotype i as

$$w_i = \tfrac{1}{2}(w_{mi} + w_{fi}). \tag{1c}$$

In the next part of this paper we show how this definition of fitness can be used to obtain conditions for the evolution of gynodioecy and androdioecy; we then go on to discuss the evolution of dioecy. Before doing this, however, it is useful to illustrate the concept by considering the case of a single locus with two alleles, A_1 and A_2. Let the frequencies of A_1A_1, A_1A_2, and A_2A_2 among the adults of a given generation be z_1, z_2, and z_3 respectively. Using equations (1a), (1b), and (1c), fitnesses w_1, w_2, and w_3 can be assigned to these genotypes. The frequencies of alleles A_1 and A_2 are p_1 and p_2, respectively. The gene frequencies in the next generation can be seen to be given by

$$p'_1 = z_1 w_1 + \tfrac{1}{2} z_2 w_2, \tag{2a}$$

$$p'_2 = \tfrac{1}{2} z_2 w_2 + z_3 w_3. \tag{2b}$$

If A_2 is fully dominant to A_1, then $w_2 = w_3$ so that equation (2b) becomes

$$p'_2 = p_2 w_2. \tag{3}$$

Hence at equilibrium in this case we have $w_1 = w_2 (= w_3) = 1$, i.e., both phenotypes contribute equally to the next generation (see also Lloyd 1975).

Equations (2a) and (2b) can be used to investigate the initial spread of a rare gene. For example, if A_2 is introduced into an A_1A_1 population, the fitness of the new type can be approximated by giving the summation terms (T, U, and V) in equations (1) the values appropriate for the original type. The condition for the spread of A_2 is that this fitness must exceed one.

II. THE EVOLUTION OF GYNODIOECY AND ANDRODIOECY

Lewis (1942) suggested that most cases of gynodioecy are due to cytoplasmic inheritance and therefore cannot be considered as precursors of dioecy. It is now known that single-gene inheritance of male sterility can be found in gynodioecious species (Baker 1966, and personal communication; Jain, Boussy, and Hauptli 1978; Arroyo and Raven 1975; see also Lloyd 1974*a*). Genes with such effects are known in many genetically well-studied species (Jain 1959), so it cannot be true that they never occur in natural populations. Their role in the evolution of dioecy is further supported by genetical studies of sex determination in several species (Westergaard 1958). In this part of the paper, we present some models for the evolution of gynodioecy (and androdioecy) based on single-gene inheritance, which are in some respects a generalization of the earlier models of Lloyd (1974*a*) and of Valdeyron et al. (1973). Lloyd (1975, 1976) has already published an analysis of the equilibrium conditions for gynodioecy and androdioecy in partially selfing populations. We will present our own treatment of this problem as a preliminary to Section III. The main addition to Lloyd's work is the derivation of the conditions for the spread of a rare male- or female-sterility gene.

Gynodioecy

Gynodioecy is here assumed to have evolved from a completely hermaphrodite condition as a result of a single gene mutation. The hermaphrodites are homozygous A_1A_1, and a mutant allele A_2 arises which we assume to be either completely

dominant or completely recessive and which causes a complete loss of pollen production. Let the male-sterile (female) individuals have a mean ovule production of $1 + k$ relative to that of the hermaphrodites. It is assumed here that s and k remain constant as the frequency of females increases in the population, i.e., there is a sufficiently large number of pollinator visits to each flower that seed set in outcrosses is not affected by the presence of females in the population. Lloyd (1974*a*, 1974*b*, 1974*c*, 1975) has considered the effect of restricted pollinator visits on the equilibria in some models of gynodioecy. Since, as will be shown later, the frequency of females is rarely high, this factor can be neglected for present purposes. In any case it will not affect the condition for the initial spread of A_2. This can be determined by applying the method of the previous section. We find that A_2 will spread if and only if:

$$1 + k > 2(1 - s\delta). \tag{4}$$

Equation (4) shows that a male-sterility gene can invade an hermaphrodite population even when $k = 0$, i.e., when females have the same ovule production as hermaphrodites. This requires $s\delta > 0.5$, i.e., both very strong inbreeding depression and a high rate of selfing. (This condition is, however, less severe than that derived by Valdeyron et al. [1973], who assumed that inbreeding depression affects ovule production only.) It seems likely that abolition of pollen production might increase ovule production as a result of reallocation of the resources available for reproduction. Darwin (1877) suggested that this type of effect, which he called "compensation", might play a role in the evolution of gynodioecy and dioecy.

If there is no selfing or inbreeding depression, equation (4) requires $k > 1$ for the establishment of A_2, i.e., that, as is intuitively obvious, ovule production must be more than doubled in compensation for the loss of pollen production; such a degree of compensation seems improbable. In general, the higher the value of $s\delta$, the lower the value of k that permits gynodioecy to evolve. The most favorable conditions for the establishment of gynodioecy occur when there are fairly high levels of selfing and inbreeding depression and an increased ovule production by females. For example, with s and δ both equal to 0.5, a k value greater than 0.5 is required, compared with 1 in the random-mating case.

The frequency of females at equilibrium in the case of completely dominant or recessive inheritance of male sterility is, by the method of Section I,

$$\hat{Z} = (k + 2s\delta - 1)/2(k + s\delta). \tag{5}$$

This corresponds to the formulae of Lloyd (1974*a*, 1975, 1976).

It can easily be seen that $\hat{Z}$ is nonzero only when equation (4) is satisfied and that it is an increasing function of k and $s\delta$, i.e., the higher the selfing rate and inbreeding depression and the greater the degree of compensation, the higher the frequency of females. Some examples are given in table 1. It also follows directly from equation (5) that $\hat{Z} < \frac{1}{2}$, so that females can never exceed hermaphrodites in frequency in an equilibrium population. Unless k is high, females will always be rather rare (see table 1). These conclusions are identical with those of Lloyd (1975, 1976) and accord with observations on gynodioecious species (Darwin 1877; Lloyd 1976).

In the case of a dominant male-sterility gene, the only possible genotype with A_2 is $A_1 A_2$, regardless of the level of self-fertilization, so that the equilibrium genotype frequencies follow directly from equation (5). In the more plausible case of a recessive

TABLE 1

FATE OF A MALE-STERILITY GENE INTRODUCED INTO A POPULATION OF HERMAPHRODITES WITH POLLEN OUTPUT = 1.0 AND OVULE OUTPUT = 1.0

SELFING RATE (s)	INBREEDING DEPRESSION (δ)	OVULE OUTPUT OF ♀ $(1 + k)$	EQUILIBRIUM FREQUENCY OF GENE	EQUILIBRIUM FREQUENCY OF FEMALES
		A, Recessive Gene		
.7	.8	1.0	.3137	.1071
		1.1	.4095	.1667
		1.5	.5648	.2925
		2.1	.6394	.3675
.7	.7	1.0	0	0
		1.1	.2259	.0678
		1.5	.5061	.2424
.7	.4	1.5	0	0
		1.7	.3406	.1326
0	0	2.1	.2164	.0455
		2.5	.4242	.1667
		B, Dominant Gene		
.7	.8	1.0	.0536	.1071
		1.1	.0833	.1667
0	0	2.1	.0227	.0445
		2.5	.0833	.1667

male-sterility gene, it is necessary to consider the recurrence relations relating the frequency of A_2A_2 (females) to the genotype frequencies in the previous generation. Let Y be the frequency of A_1A_2 individuals and Z the frequency of A_2A_2 individuals. Then it is easy to show that

$$WZ' = \frac{[Z(1+k) + \frac{1}{2}(1-s)Y]Y}{2(1-Z)} + \frac{s(1-\delta)Y}{4}, \tag{6}$$

where $W = (1 - Z)(1 - s\delta) + Z(1 + k)$.

At equilibrium, equation (5) shows that $W = (1 + k)/2$. (This is to be expected from the facts that the equilibrium fitnesses of the three genotypes are the same and that the females contribute only through their ovules.) Substituting into equation (6) and rearranging, we obtain a quadratic equation for the equilibrium frequency of A_1A_2:

$$\frac{(1-s)}{2\hat{Z}(1-\hat{Z})}\hat{Y}^2 + \left[\frac{1+k}{1-\hat{Z}} + \frac{s(1-\delta)}{2\hat{Z}}\right]\hat{Y} - (1+k) = 0, \tag{7}$$

where $\hat{Z}$ is given by equation (5).

Table 1 shows the phenotypic composition and gene frequencies of some equilibrium populations with dominant and recessive male-sterility. Computer calculations showed that in all these cases the equilibria are globally stable. In those cases in table

1 where s and δ were nonzero, or in the random mating case with A_2 dominant, equilibrium was reached in a matter of a few hundred generations after the introduction of the male-sterility gene at a frequency of 0.00025. As might be expected, however, the frequency of a recessive male-sterility gene increased extremely slowly in a random mating population, and the gene remained rare for many thousands of generations. Gynodioecy due to recessive male-sterility is thus unlikely to be established in random mating populations unless finite population size effects can be invoked.

Androdioecy

The case of a dominant or recessive gene causing female sterility can be treated in a way similar to the above. The pollen production of the males relative to that of the hermaphrodites is written here as $1 + K$. The condition for the initial spread of a female-sterile gene is

$$1 + K > 2(1 - s\delta)/(1 - s). \tag{8}$$

When there is partial self-fertilization ($s > 0$), it is immediately apparent that this condition is more stringent than for the case of gynodioecy (eq. [4] above). Furthermore, for constant δ, the larger s the larger the minimum value of K required to establish androdioecy. This is in contrast to the case of gynodioecy, where with nonzero inbreeding depression, self-fertilization reduces the minimum increase that is necessary in ovule production. The reason for this effect is simply that the pollen produced by the males (female steriles) is used only in outcrosses; the higher the level of self-fertilization, the less this pollen can contribute to the next generation. We conclude that androdioecy is most likely to be established in outcrossing populations and cannot be established in any population unless the pollen production of the males is at least doubled relative to that for hermaphrodites. This conclusion was also reached by Lloyd (1975). Furthermore, a recessive gene for female sterility will encounter the same difficulty in establishing itself in an exclusively outcrossing population, as noted above for a recessive male-sterility gene. These considerations may be among the reasons for the rarity of androdioecy in nature.

The equilibrium frequency of males can be determined by the method used in treating gynodioecy and is given by

$$\hat{Z}^* = [(1 + K)(1 - s) - 2(1 - s\delta)]/2K(1 - s\delta). \tag{9}$$

It can easily be shown from this formula that $\hat{Z}^*$ is always less than or equal to $\frac{1}{2}$, and is a decreasing function of s. With high s values, an extremely large K is required for there to be more than a few percent of males in the equilibrium population, e.g., with $s = \delta = 0.5$ and $K = 2.5$, $\hat{Z}^* = 0.067$; K must be greater than 2 in order for androdioecy to become established at all in this case, compared with a k value of 0.5 for the corresponding male-sterile case.

In the following sections of this paper we consider the evolution of dioecy from gynodioecy by examining the fate of a modifier of ovule production introduced into gynodioecious populations at equilibrium. The mutant allele at this second locus can have a total or partial effect in reducing ovule production; it can act only in hermaphrodites or reduce the ovule production of females as well. It can also have an

increased pollen production, by the compensation principle, and can be dominant, recessive, or partially dominant.

III. INTRODUCTION OF A MODIFIER INTO A GYNODIOECIOUS POPULATION

In this section we consider the conditions which determine whether or not a mutation at a second locus can become established in a gynodioecious population at equilibrium.

Conditions for Spread of a Modifier of Arbitrary Effect on Ovule Production

At equilibrium for the first (male-sterile) mutation, both hermaphrodites and females are present. Knowing the frequencies of these forms, as given by equation (5), one can calculate how big an increase in pollen output would be necessary to increase the net fitness of the hermaphrodite, given that its ovule production is decreased. Let the modified hermaphrodites have pollen production $1 + K$ relative to that of the original hermaphrodites and let their ovule production be $1 - k^*$ $(1 \geq k^* \geq 0)$. Then equations (1) and (5) give the following expression for K^*, the value of K that will exactly compensate for the loss in ovule production:

$$K^* = \frac{(1 + s - 2s\delta)}{(k + 2s\delta - s)} k^*. \tag{10}$$

Clearly, an increase in pollen production greater than K^* is a necessary condition for the spread of the mutation at the second locus.

The fact that s and δ are both less than one, together with the condition for the existence of gynodioecy given in equation (4), implies that K^* must be positive. In other words, some increase in pollen production is always required for the modifier gene to spread. When the females have the same ovule production as the hermaphrodites $(k = 0)$, equation (10) shows that $K^* > k^*$ unless $1 + 2s < 4s\delta$, which would require a very high level of inbreeding depression. In most instances of this sort, therefore, we can conclude that a reduction in ovule production cannot be selected for unless it is accompanied by a more than proportionate increase in pollen production. Equation (10) also shows, however, that the higher k, the lower K^*, so that if the ovule production of females is sufficiently high compared with that of hermaphrodites, K^* need not be as high as k. Some cases illustrating these points are given in table 2. One further point is that if a similar calculation is done for a gene of this sort introduced into an hermaphrodite population (eq. [8]), then with the same values of s and δ, a larger compensatory increase in pollen production is always needed than that required by equation (10). The presence of females in the population therefore makes it easier for a modifier reducing female fertility to spread.

These considerations have involved no assumptions about the dominance relations at the loci concerned or about the effect of the modifier gene on the fertility of females. In order to obtain sufficient conditions for the spread of the modifier, it is necessary in general to take these factors, as well as the linkage between the two loci, into account. This is done below for two important cases.

TABLE 2

CALCULATIONS OF THE MINIMUM FRACTION (K^*) BY WHICH THE POLLEN PRODUCTION MUST BE INCREASED TO ENABLE A FEMALE-STERILITY MUTATION TO SPREAD IN A GYNODIOECIOUS POPULATION

SELFING RATE (s)	INBREEDING DEPRESSION (δ)	OVULE OUTPUT OF ORIGINAL ♀	OVULE OUTPUT OF ♀					
			1.0	1.1	1.5	1.8	2.1	2.2
.7	.8	1.0	1.381	1.115	.630	···	.382	···
		.9	1.092	.903	···	···	···	···
		.5	.409	.358	···	···	.160	···
.7	.2	1.0	···	···	···	3.737	···	1.821
0	0	1.0	···	···	···	···	.909	···

A Dominant Modifier Introduced into a Population with Recessive Male-Sterility

If male-sterility is recessive, crossing-over between the two loci will produce some females which carry the modifier gene, even when the modifier is rare. If the modifier reduces the ovule fertility of these females, they will be at a selective disadvantage, and a higher pollen production of the modified hermaphrodites will be required to overcome this disadvantage. Only if the modifier does not reduce the ovule production of females will equation (10) give a sufficient condition for the modifier to spread. In other cases, we may expect the degree of linkage between the loci to affect the conditions; the maximum rate of spread of the modifier will be expected to result when it is introduced in coupling with the male-fertility (A_1) allele at the first locus, and when there is no crossing-over to break down this combination. If the modifier is not sufficiently closely linked to the first locus, it may be eliminated, i.e., there may be a linkage constraint.

These conclusions have been checked by the simulations described in Section IV of this paper. In the case of a dominant modifier gene which abolishes the ovule production of both hermaphrodites and females, an algebraic analysis of its initial spread can be made. As outlined in the Appendix, we can study the properties of the quantity λ_0, which measures the asymptotic value of the ratio between the frequencies of the modifier gene in two successive generations, while it is still rare. The maximum value of λ_0 is attained when R, the frequency of recombination between the two loci, is zero. It can be seen that this maximum value is equal to the fitness of the modified hermaphrodites discussed above. As R increases, λ_0 decreases smoothly and, if its maximum value is greater than one, falls below one when R is greater than some critical value, R_c. For R greater than R_c, the modifier gene will therefore be eliminated. The value of R_c, in terms of the parameters a and b defined in the Appendix, is given by:

$$R_c = (a + b - 1)/\left[a + b - \frac{ab}{1 - a}\right]. \tag{11}$$

When the maximum value of λ_0 is not greatly in excess of one, R_c may be rather small. For example, with $k = 0$, $K = 1.45$, $s = 0.7$, and $\delta = 0.8$, we find $R_c = 0.0675$. There is therefore a selective sieve for closely linked modifiers of this sort.

A Recessive Modifier Introduced into a Population with Dominant Male-Sterility

At equilibrium for a dominant male-sterility gene, there are only two genotypes, homozygotes for the original allele ($A_1 A_1$) and heterozygous females ($A_1 A_2$). When a recessive modifier reducing ovule production is introduced at a low frequency, females homozygous for it are produced only by outcrosses, and therefore will initially be negligible in frequency compared with modified hermaphrodites. The conditions of equation (10) will therefore be sufficient for the modifier to spread, regardless of the linkage value and the effect on females. In particular, there can be no linkage constraint. This conclusion is borne out by the simulation results of Section IV.

IV. RESULTS OF COMPUTER SIMULATIONS

The considerations of Section III suggest that a gene which changes the hermaphrodites into a more male-like form can be selected for provided that the gene also increases pollen output sufficiently. These conclusions were verified by computer calculations of population trajectories. The method used was to start with an array of genotype frequencies. Genotypes with both male and female potential were assumed to devote a proportion s of their ovule output to selfing; the selfed ovules had a probability $1 - \delta$ of survival to maturity compared with outcrossed ovules. Male-sterile genotypes produced all their offspring by fertilization with a common "pollen pool," which was also used for the outcrossed progeny of hermaphrodites. The composition of the pollen pool was determined by considering each of the male-fertile genotypes and calculating the overall frequencies of the four gametes produced by these in aggregate. Different genotypes were allowed to produce differing quantities of ovules and pollen. The new array of genotype frequencies produced by this procedure was used to form the next generation. The runs were started by introducing the modifier allele at a low frequency (0.00025) into an equilibrium gynodioecious population. It was introduced heterozygous and in coupling with the original allele (A_1) at the first locus. The population was then run to equilibrium.

Recessive Male-Sterility Gene Followed by a Dominant Modifier

When the modifier converts the hermaphrodites into males, with zero ovule production, the equilibrium population consists either of predominantly males and females, at frequencies close to 0.5, or of males, females, and a substantial frequency of hermaphrodites. Some results of this kind are shown in table 3. The looser the linkage, the more the population departs from true dioecy. Some of the parameter sets shown in table 3 provide examples of the action of a linkage constraint: The modifier was eliminated when the recombination fraction was high. This is the case for all those examples in table 3 where only a low recombination fraction is shown. As expected, a linkage constraint is most likely to operate when the modifier has a

TABLE 3

PHENOTYPE FREQUENCIES IN POPULATIONS THAT HAVE REACHED EQUILIBRIUM FOR A MALE- AND A FEMALE-STERILITY GENE

OVULE OUTPUT OF MODIFIED FEMALES	RECOMBINATION FRACTION	PHENOTYPE FREQUENCIES ♂	♀	♂	Modified ♀
Male-Sterile (1st) Mutation Recessive, Female-Sterile (2d) Mutation Dominant					
.9	.01	.085	.440	.450	.025
	.1	.536	.218	.186	.060
	.5	.656	.181	.115	.048
0	.01	.087	.461	.452	0
Male-Sterile (1st) Mutation Dominant, Female-Sterile (2d) Mutation Recessive					
.9	.01	.085	.440	.450	.025
	.5	.650	.179	.121	.050
0	.01	.089	.459	.447	.004
	.5	.819	.169	.012	.001
Male-Sterile (1st) Mutation Recessive, Female-Sterile (2d) Mutation Recessive					
.9	.01	.477	.262	.221	.039
0	.01	.614	.239	.142	.004
Male-Sterile (1st) Mutation Dominant, Female-Sterile (2d) Mutation Dominant					
.9	.01	.584	.201	.159	.056
	.5	.715	.168	.081	.036

NOTE.—Ovule output of original ♀ = 1.0; pollen output of original ♀ = 1.0; ovule output of original ♀ = 1.1; pollen output of ♂ = 2.4; selfing rate = 0.7; inbreeding depression = 0.8.

strong effect in the females as well as the hermaphrodites, but the effect in the females need not be as strong as that in the hermaphrodites.

Examples of the perhaps more realistic case of a dominant modifier which partially reduces the ovule output of both females and hermaphrodites were also studied. As in the above extreme case of complete female sterility, the outcome depends on the linkage between the two loci, though a small enough effect on the females compared with the hermaphrodites may permit an unlinked modifier to become established.

With either type of modifier action, modified females are generally rare at equilibrium, so that the population comes to consist predominantly of females together with either males and hermaphrodites, or of females and two types of hermaphrodite. The simulations also show that a modifier with no effect on the females, and which satisfies condition (10), will spread to fixation, giving a population consisting of females together with either males or modified hermaphrodites, depending on the phenotypic effect of the gene; such a population would have only one segregating gene affecting sex.

In summary, modifiers converting the hermaphrodites into males, or into a more male-like form, could be selected for. In cases where a loosely linked modifier was not eliminated, we have shown that there is selection for tighter linkage between the two loci. This was done by simulating a three-locus situation, two of the loci controlling the (sub-) dioecy, as explained above, and a third locus modifying the recombination frequency between them. Even when this third locus was unlinked to the first two, in which case the effectiveness of selection on a modifier of recombination is likely to be weak, it increased in frequency, reducing the frequency of recombination between the selected loci.

With the above type of model there are two situations where the equilibrium population consists essentially of just two forms, females and the new type of hermaphrodite. The first way that this can occur is by a modifier with no effect in females, as already noted. The second way is by means of a non-sex-limited modifier that is tightly linked to the male-sterility gene. In this situation, there are negligible frequencies of two gamete types, so that just three genotypes occur; because of the dominance of both the genes, only two phenotypes are seen. Supposing that this state has been reached, we can then ask whether further modification of the hermaphrodite, making it yet more like a male, is easier or more difficult than was the first step. We have approached this question by the same method that we used in Section III to ask whether the first modifier gene will get into the population. In the present case, the hermaphrodites are given a female fertility of less than one, as would be the case if they had already become modified in the direction of maleness; we then ask what proportional increase in pollen production would be needed, given some decrease in ovule production, for net fitness to be increased. This effect can be studied using equation (10), since the increased ovule fertility of females compared with hermaphrodites in this case corresponds with increased values of k. Some examples are given in table 2. The lower the ovule production of the hermaphrodites, the lower the compensatory increase in pollen production needed, so that it becomes easier for further changes in the same direction to be selected. It may therefore be possible for dioecy to evolve by gradual modification of the polleniferous plants, due to the successive accumulation of linked modifiers of ovule production.

Dominant Male-Sterility Gene Followed by a Recessive Modifier

Some examples of this type of situation are shown in table 3. The prediction of Section III that there can be no linkage constraint is verified by these runs. Just as in the case of a recessive male-sterility gene followed by a dominant modifier, this system can lead to equilibrium populations that are like those seen in nature, with males, females, and hermaphrodites or with two types of hermaphrodite and one common female type, the other being very rare.

With this model for the evolution of dioecy we must also consider the case with no selfing, since a dominant male-sterility factor can rise to an appreciable frequency with random mating, provided its female fertility is sufficiently high. Some runs of this sort have been carried out. The modifier can increase in frequency but does so extremely slowly. For example, when such a male-sterility gene raises the females' ovule output to 2.2, it reaches equilibrium with a female frequency of 0.083. When a

female-sterility factor which increased pollen output to 2.5 was introduced at a frequency of 0.00025, as in the other simulations, it rose to 0.00030 in 2,700 generations. When introduced at a frequency of 0.1, it reached a frequency of 0.755 within 1,000 generations, however, and the population consisted almost exclusively of males and females. Dioecy can therefore evolve in this case, but the second mutation will have a very low chance of establishment in a large population, as with most recessive genes (Haldane 1927).

Other Possible Cases

We have also simulated the two models of gynodioecy described above—i.e., recessive and dominant determination—followed by a modifier which is also recessive or dominant, as the case may be. For instance, a dominant modifier was introduced into a population segregating for a dominant male-sterility gene; the parameter values were otherwise the same as those for a recessive case that would go to an equilibrium consisting of just males and females. In this case, however, with a recombination fraction of 0.01, the modifier rose to only 0.06, instead of 0.72; dioecy did not result, but a low frequency (0.08) of males was present at equilibrium, together with a frequency of 0.17 of females, while the majority remained hermaphrodite. With an unlinked modifier, the equilibrium frequency of males became higher (0.15), the frequency of females became 0.20, and there was a frequency of 0.06 of females carrying the modifier, which would appear as somewhat sterile females. Similar results were obtained in the case of a recessive followed by a recessive.

Another case which we have considered is that of a recessive male-sterility gene followed by a gene reducing ovule production which is partially dominant rather than completely dominant or recessive. The condition described in Section III for the initial spread of a female-sterility gene must be satisfied by the heterozygote for this modifier, but the homozygotes, which we assume to be more female-sterile than the heterozygotes, need not necessarily satisfy this condition. The results from runs of this sort of case are not very different from those with full dominance; the frequencies of the different types are affected, but no qualitatively new phenomenon is seen. This type of model gives even more variability among the hermaphrodites than the models discussed earlier in this section, since now we can have distinct heterozygotes and homozygotes for the modifier as well as the original hermaphrodites. As above, there will be selection for further linked modifiers reducing the female fertility of the modified individuals.

V. DISCUSSION

In this section we review the results described earlier in the paper and try to relate them to some of the facts about the breeding systems of flowering plants.

Androdioecy

As already mentioned, androdioecy is extremely rare. One reason for this may be that a cytoplasmic factor causing female-sterility cannot be transmitted, whereas male-sterility is often inherited in this way. The rarity or absence of androdioecy

controlled by nuclear genes is probably due to the following effects, discussed more fully by Lloyd (1975) and in Section II above. In a random mating population, a female-sterile mutant cannot be established by selection unless it more than doubles pollen output (or survival to maturity). An even greater effect is required in a partially self-fertilizing population, where the pollen which a plant contributes to the pollen pool is worth less than its ovules in terms of genes transmitted to the next generation, since a large fraction of the ovules is self-fertilized.

Gynodioecy

Gynodioecy, although not common, is found in a large number of species (Darwin 1877). As we have discussed in Section II, the conditions for the establishment of a male-sterile mutant in a partially self-fertilizing population are less severe than those for a female-sterile gene if there is inbreeding depression. The most favorable conditions are when there is a high level of self-fertilization and high inbreeding depression and the male-sterile plants have an increased ovule production due to the compensation effect of Darwin. The latter factor is not essential, but very severe inbreeding depression effects are required in its absence (see Section II for details). There is a considerable amount of data which suggests that female plants in gynodioecious populations often have higher seed production than hermaphrodites. For example, Darwin (1877, p. 302) found that the seeds from a female plant of *Thymus serpyllum* weighed about 1.79 times as much as those from a hermaphrodite. McClusker (1962, quoted by Carlquist 1966) showed that female plants of *Leucopogon melaleucoides* had a fruit set of 1.44 times that of hermaphrodites. On the other hand, these differences may reflect subsequent evolution toward dioecy (as discussed below) rather than a direct physiological consequence of male-sterility.

Neither of these figures is large enough to permit the evolution of genically determined gynodioecy if the ancestral hermaphrodite population is assumed to have been random mating. They are sufficient to promote the evolution of gynodioecy in a partially selfing species, with some inbreeding depression. The view that gynodioecy has evolved due to a combination of the two factors, compensation and avoidance of inbreeding, is further supported by the following considerations. First, there are many examples of gynodioecy in self-compatible species; for example, it is particularly common in the Labiatae, a predominantly self-compatible group (Fryxell 1957). Second, it is difficult to understand why gynodioecy is more common than androdioecy unless self-fertilization and inbreeding depression play a part. In a random mating population, androdioecy would seem more likely to evolve than gynodioecy, rather than the other way round, because a decrease in ovule production might more plausibly be thought to give a large increase in pollen output than vice versa. A different explanation for the existence of male- or female-sterility genes segregating in natural populations is heterozygote advantage of these genes (Jain 1961; Ho and Ross 1974; Ross and Weir 1975, 1976). This would be much more effective in random mating than in selfing populations, because there would have to be strong heterozygous advantage in order to maintain such polymorphism in the face of a high rate of selfing (Kimura and Ohta 1971). It is therefore not surprising that Ross and Weir (1976, p. 437), studying the evolution of dioecy with heterozygote

advantage of the male-sterility factor, conclude that "dioecy does not evolve in the present model with moderate selection differentials." Furthermore, if heterozygote advantage is an important factor, there is again no obvious explanation for the great rarity of androdioecy compared with gynodioecy; in random mating populations, where heterozygote advantage would be most effective, both systems would seem equally likely to evolve.

Finally, it is worth emphasizing that our results agree with those of Lewis (1941), Valdeyron et al. (1973), and Lloyd (1975, 1976) in suggesting that rather stringent conditions must be met if gynodioecy is to evolve, so that it is not surprising that it is uncommon.

It seems likely that gynodioecy is more often due to recessive male-sterility genes than to dominant ones, though few cases of single-gene inheritance in gynodioecy are known. Baker (1966, and personal communication; Jain et al. 1978) found a case of dominant inheritance of male-sterility in gynodioecious populations of *Limnanthes douglasii*. Arroyo and Raven (1975) report both recessive and dominant inheritance in gynodioecious Fuchsias. Lloyd (1974*a*) quotes other examples of both modes of inheritance which suggest a preponderance of recessive cases. It is not surprising that so few cases of single-gene inheritance are known in gynodioecious species since they may readily evolve sub-dioecy, whereas this is not true when the gynodioecy is cytoplasmically determined. Perhaps subdioecious species would provide better material for the study of the genetics of male-sterility in natural populations. Lloyd (1974*a*, p. 32) suggests that recessive male-sterility is more likely to be found because it confers "the ability ... to regenerate sexual dimorphism when females are lost." A simpler explanation is that male-sterility represents a loss of function, and most genes of this sort are recessive (Wright 1934). If the male-sterility in gynodioecious populations is most often recessive, this provides support for the idea that inbreeding is an important factor, since recessive mutations have little chance of being established by selection in a large random mating population.

Dioecy

In Sections III and IV we have described models for the evolution of gynodioecy into full dioecy or subdioecy. The simplest path is when gynodioecy is followed by a mutation causing complete female sterility. If male-sterility is recessive, this mutation must be dominant for full dioecy to evolve, and this will result in male heterogamety. Conversely, if male-sterility is dominant, dioecy will not evolve unless the second mutation is recessive, and once dioecy has evolved there will be female heterogamety. Since, as discussed above, recessive male-sterility is probably more common than dominant, it is not surprising that male heterogamety is considerably commoner than female heterogamety. The latter has been found in three groups: *Fragaria* (Westergaard 1958), *Potentilla fruticosa* (Grewal and Ellis 1972), and *Cotula* (Lloyd 1974*a*). Twenty-three firmly established cases where the male is the heterozygous sex are listed by Westergaard (1958). Full dioecy will result on this model when the two genes are tightly linked (table 3). With male heterogamety, the "*Y*" chromosome will have a dominant female-suppressor, and the "*X*" will carry a recessive male-suppressor. This type of genetic control of sex determination is essentially that found

in *Silene dioica* (*Melandrium*), reviewed by Westergaard (1958), although some other genes affecting sex are also present. Westergaard also discusses genetical work with several other species and shows that they conform to the same basic model—in some cases (*Ecballium*) exactly as above, while in others (*Carica papaya*) there are other genes which affect sex expression. Data on *Vitis* also fit this scheme (Lewis 1942). Westergaard regards the *Ecballium* system as most primitive, which seems plausible since there are dioecious and monoecious races in this group, suggesting that dioecy is of recent origin. If the two genes in our models are not closely linked, the equilibrium populations contain males, females and hermaphrodites, as shown in table 3. This sort of situation is known to occur in a number of species (Fryxell 1957; Westergaard 1958) and is possible with either male or female heterogamety. It would be interesting to know more about the genetics of sex in such cases, in particular the linkage relations of the genes involved. The model of the evolution of dioecy proposed by Ross and Weir (1976), which involves heterozygote advantage of the male-sterility factor, leads to equilibria with males, females, and hermaphrodites, even with complete linkage; this is not possible with our model.

We have shown in Sections III and IV that modifiers causing relatively slight reduction of female fertility can be established in gynodioecious populations and that once this has happened the selection pressure in favor of increased maleness of the hermaphrodites is intensified. Full dioecy, of the type discussed above, can only evolve given genes with the right dominance and linkage relations. Our model therefore predicts that intermediate stages between gynodioecy and full dioecy should be fairly common. Populations segregating for one or more modifiers of this sort will show great variability in the female fertility of the hermaphrodites, with the more extreme modified hermaphrodites behaving as functional males. The average ovule production of hermaphrodites in such populations will be much less than that of females. Darwin (1877, p. 292) was struck by this type of situation in the spindle-tree *Euonymus europaeus*, which he described "as showing how gradually an hermaphrodite plant may be converted into a dioecious one." Later work has shown that there is a virtual continuum of stages between gynodioecy and dioecy, e.g., in several species of the Hawaiian flora (Carlquist 1966) and in *Fuchsia* (Arroyo and Raven 1975). In *Pimelea*, Burrows (1960) has shown that species vary from those with a slight difference in ovule fertility between females and hermaphrodites at one extreme through ones with a greater difference, to virtually complete dioecy at the other. The frequencies of female plants vary concomitantly, from around 15% up to 50%. In all these cases of subdioecy, the females are relatively constant in their sex expression, and it is the polleniferous plants (hermaphrodites and males) which are variable. Westergaard (1958) comments on the fact that, even in cases of female heterogamety, the males are more variable than the females. This is quite consistent with our model.

Sex chromosomes.—In those cases with a male-determining *Y* which have been investigated genetically, the sex genes are all on the same chromosome and behave as if completely linked (Westergaard 1958). Previous papers on the evolution of dioecy have concentrated on deriving conditions under which only males and females will be present. Complete linkage is of course such a condition (Lewis 1942; Ross and Weir 1976), and this has been regarded as an explanation of the genetic findings. It

does not, however, constitute an explanation of the evolution of these genetic systems since it specifies neither the path by which the present state has been reached nor the causal factors involved. Our model provides a possible evolutionary explanation, based on consideration of the conditions (including strength of linkage) for the spread of a gene reducing female fertility, when introduced into a population segregating for a recessive male-sterility gene. If sufficiently tightly linked to the male-sterility locus, such a gene may increase in frequency even when it would be eliminated if unlinked. Such a "linkage constraint" often exists even when the effect on the females is much less (in terms of percentage loss of ovule production) than that on the hermaphrodites. This effect therefore provides one major reason for the existence of sex chromosomes. Bodmer and Parsons (1962) first analyzed the conditions for the operation of this type of effect, in random mating populations subjected to epistatic selection. We have proposed it as an explanation for the supergenes of Batesian mimicry (Charlesworth and Charlesworth 1975). It may well be generally important in the evolution of polymorphisms involving linked genes.

Once a linked modifier of female fertility has been established, there is selection pressure in favor of the reduction of crossing-over between it and the male-sterility locus. In *Silene dioica*, crossing-over between the *X* and *Y* chromosomes is confined to the region outside the sex genes, presumably as a result of this process (Westergaard 1958). In the case of female heterogamety, where the original male-sterility gene must be dominant, no linkage constraint can operate, and so selection for reduced recombination is the only factor operating. One might therefore expect full dioecy to be difficult to establish in this case.

If the operation of a linkage constraint is the selective basis of the linkage of the sex-determining genes, we would expect dioecious species to have the same chromosome numbers as hermaphrodite or gynodioecious relatives, whereas if the genes have to be brought together from separate chromosomes, one might well expect centric fusions to be involved, with a consequent lowering of chromosome number. Although the possibility exists that chromosome changes may have occurred since the establishment of dioecy, it is perhaps significant that dioecious species often have the same chromosome numbers as their relatives. For example, the dioecious species of *Silene* (*dioica*, *alba*, and *otites*) have $2n = 24$, and so do 10 other British species whose chromosome numbers are given by Clapham, Tutin, and Warburg (1962). Similarly the dioecious *Ribes alpinum* has $2n = 16$, the same number as the four other British species of *Ribes*. The only other comparisons of this sort that can be made, using the British flora, are inconclusive, because the chromosome numbers of the groups are very variable. In *Rumex*, the hermaphrodite species all have haploid numbers that are multiples of 10, and so does *R. scutatus*, which is described as "polygamous." The dioecious *R. acetosa* and *R. acetosella*, however, have $2n = 14$ or multiples of 14, with an extra *Y* chromosome in *R. acetosa* males. This might be a case of reduction of chromosome number during the evolution of dioecy. According to Smith (1967), a similar distinction between dioecious and bisexual species holds good throughout the *Acetosa* subgenus. In *R. hastatulus*, which is also dioecious, the chromosome number has been reduced even further (Smith 1969). In Texas $2n = 10$, with an XY sex chromosome pair; in North Carolina, $n = 4$ in females, and in males

there are three pairs of autosomes and XY_1Y_2. This seems to have resulted from a centric fusion between the major arm of the Texas third chromosome and the acrocentric X, followed by a translocation between the two Y chromosomes. The intermediate stage is found in material from South Illinois–Missouri. Whether these translocations have any importance in relation to the sex-determining genes is, of course, not known, but it may be significant that in this species the Y chromosome carries a male-promoting factor (Smith 1963), whereas the other *Acetosa* species studied have an X/autosome balance system. It is possible that the X/autosome mechanism has arisen secondarily from the male-determining Y system considered in this paper. Our model obviously cannot explain its origin directly.

Barlow and Wiens (1976) report a similar but more extreme case in an African mistletoe, *Viscum fischeri*, in which the males are heterogametic for a set of multiple sex chromosomes ($5Y/4X$) that produce a multivalent chain of nine chromosomes at meiosis in addition to seven autosomal bivalents. Translocation heterozygosity is common in the dioecious African mistletoes but is unknown in the monoecious species and in the other dioecious species of *Viscum* that have been studied, in which no sex chromosomes could be identified. This suggests that translocations might be involved in the evolution of dioecy, though there is no reason to attribute the whole development of this complex translocation system to this factor alone.

The roles of compensation and inbreeding depression.—The models developed in this paper seem to agree in many respects with the results of observations on dioecious species. However, we have had to make a number of assumptions which do not have direct observational support. In the first place, we have assumed that a mutation abolishing reproduction either as a male or as a female will to some extent increase the output of the other type of gamete, as originally suggested by Darwin. Although gynodioecy can evolve without any such compensation provided that selfing and inbreeding depression are of sufficient magnitude, a female-sterility gene will always be eliminated unless pollen production is sufficiently raised, even in a gynodioecious population (Section III). In a gynodioecious population, the presence of male-sterile individuals means that investment in pollen by male-fertiles gives a higher genetical return than in a completely hermaphrodite population under similar conditions. This is because the genetic contributions of females and hermaphrodites are equal in an equilibrium gynodioecious population (Section II), so that hermaphrodites must pass on more of their genes through pollen than they would in the absence of females. Provided that the females have a sufficiently greater ovule output than the hermaphrodites, a less-than-proportional increase in pollen production may therefore be enough to compensate for a loss in ovule output (Section III). In view of the necessity of assuming that compensation will occur, it would be interesting to know what effects *mutants* causing male or female sterility have on the fertility that remains. Ross (1977) has reviewed the available data, but unfortunately this is mostly only qualitative, and quantitative data are scarce. In a study of cytoplasmic male-sterility in cotton, Rosales and Davis (1976) found an increased seed production by females, provided adequate pollinator visits were ensured.

A second assumption which we have made is that inbreeding depression can be severe in a partially selfing species. Most experimental estimates of inbreeding depression have been made using outbred species. It would be interesting to have

data for inbreeders since, as discussed earlier, gynodioecy seems most likely to have evolved from such species. The only data known to us that bear on this point, although indirectly, are those of Marshall and Allard (1970). These authors found excess of heterozygotes at allozyme loci in *Avena barbata*, a largely selfing species. This is most simply interpreted as a consequence of homozygote disadvantage at loci linked to the allozyme loci themselves and thus provides evidence that the products of selfing are at a disadvantage compared with outbred individuals. Lloyd (1972*b*) showed that several monoecious species of *Cotula* (a genus in which there are also several dioecious species) are self-fertile. He also found that the percentage of seeds germinating was about 30% lower for selfed than for outcrossed progeny. The natural rate of selfing is, however, not known for these plants, nor for most hermaphrodite or monoecious relatives of dioecious species. Comparative data of this sort would be valuable. At present all that can be said is that many dioecious species have a low frequency of hermaphrodites, and these can readily be selfed in genetical studies (Westergaard 1958). Furthermore, self-fertile hermaphrodite strains have been obtained by selection in a number of dioecious crop plants—e.g., strawberries (Darwin 1877), the grape vine, cannabis, and spinach (Westergaard 1958)—and in garden plants that are grown for their berries (Holly, Celastrus). This shows that they cannot have a strong self-incompatibility system.

Finally, we should point out that we do not suggest that the model described in this paper is the only way in which dioecy can evolve. Dioecy has probably evolved from monoecy on several occasions (Lewis 1942), for example, in *Mercurialis*, *Ecballium*, and others (Westergaard 1958), *Cotula* (Lloyd 1972*a*), and *Viscum* (Barlow and Wiens 1976). Lloyd (personal communication) has pointed out that no case of gynodioecy is known in which the polleniferous plants have separate male and female flowers and that this suggests that dioecy has evolved from monoecy via some other intermediate stage than gynodioecy. Now there is one obvious difference between hermaphrodite and monoecious plants with respect to the fate of genes causing a slight reduction in male fertility. In a hermaphrodite flower, a small reduction in pollen production would be unlikely to reduce greatly the proportion of selfed ovules and so protect against inbreeding depression; moreover, such a mutant type would have fewer outcrossed progeny. These two factors can make a small reduction in pollen output selectively disadvantageous, even in conditions which enable a gene causing complete male-sterility to be selected. In a monoecious species, however, it seems much more likely that a reduction in pollen output by a plant might reduce the chance of self-fertilization of its female flowers sufficiently to outweigh the disadvantage of a reduction in outcrossing potential. It can be shown by an argument similar to that in Section III that once a gene reducing pollen fertility has been established in a population, further steps in the same direction are more difficult since the increased female character of the population makes pollen more genetically valuable than before (Charlesworth and Charlesworth 1978). The new type of population would, however, be more likely to acquire factors reducing female fertility. Thus a monoecious species might incorporate in alternation genes slightly affecting male and female fertility, finally becoming dioecious. This accords with Lloyd's (1972*a*) findings in *Cotula*, where monoecy seems to have evolved into subdioecy and dioecy. There are also a number of cases in which dioecy seems to

have evolved from heterostyly. Baker (1959) describes a case in *Mussaenda*, and Ornduff (1966) gives a possible instance in *Nymphoides*. Opler et al. (1975) have studied another case in *Cordia*. The selective causes of this type of change are scarcely understood at all. Darwin (1877) also discusses several cases where this may have happened, but no further information seems to have been obtained about these species.

SUMMARY

A model for the evolution of gynodioecy from the hermaphrodite or monoecious condition is described, taking into account the effects of partial selfing and inbreeding depression. It is shown that a mutant causing male-sterility can be selected even if the female plants have the same ovule output as the hermaphrodites, but that the conditions for this are very stringent: The product of the selfing rate and the inbreeding depression must exceed one-half. If the females have an increased ovule output, gynodioecy can evolve with lower values of the selfing and inbreeding depression parameters. Expressions for the equilibrium frequency of females and of the male-sterility gene in both the dominant and the recessive case are given.

By a similar technique, conditions for the evolution of androdioecy are derived. In a selfing population, these conditions are much less easily satisfied than those for gynodioecy, though in a randomly mating population the conditions are similar: If ovule production is abolished, pollen production must be more than doubled, or vice versa. Since androdioecy is known to be a very rare condition, it seems likely that avoidance of selfing has played a role in the evolution of gynodioecy.

Using the equilibria derived for gynodioecy, the conditions for the evolution of subdioecy or dioecy, by means of a partial or total female-sterility mutation, are studied. In contrast to the situation in a hermaphrodite population, a female-sterility gene can be selected in a gynodioecious population if it confers a moderate increase in pollen output; some increase in pollen output is essential. The fate of such a female-sterility gene also depends on its linkage with the male-sterility gene. If this is recessive, and the female-sterility gene is dominant and has an effect in the females as well as the hermaphrodite individuals, then the second mutation will usually be eliminated unless it occurs at a locus tightly linked to the first gene. In other cases there is no such "linkage constraint," though in all situations there may be selection for tighter linkage between the loci; this will result in an initially subdioecious population becoming more fully dioecious.

These results agree with some of the facts known about the evolution of dioecy in plants. First, since gynodioecy is more often controlled by a recessive than a dominant gene, male heterogamety should be commoner than female, as is observed. Second, subdioecy should be common, since full dioecy requires not only the correct phenotypic effects of the two genes but also complementary dominance relations and tight linkage; subdioecy is indeed known in many species. The equilibria reached by our model have only one type of female in appreciable frequency, whereas the polleniferous individuals may fall into several genotypic classes; it is often observed that in subdioecious species the males are more variable than the females, regardless of which is the heterogametic sex. Finally, the equilibria generated by our model agree closely with the results of genetical studies of those dioecious species with

male-determining Y chromosomes that have been investigated, in which both male- and female-sterility factors have been found, showing complementary dominance relations and no crossing-over between the loci, so that just two gamete types exist. Such a situation can be explained by the operation of the linkage constraint, which ensures that only linked mutations become established and does not require that unlinked genes have been brought together. This is consistent with the fact that dioecious species often have the same chromosome numbers as their bisexual relatives.

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APPENDIX

Analysis of the Conditions for the Spread of a Dominant Female-Sterility Mutation in a Gynodioecious Population

Let B_1 be the original allele at the female-sterility locus and B_2 be a dominant allele which renders its carriers female-sterile. The equilibrium frequencies at the male-sterility locus are $\hat{X}(A_1A_1)$, $\hat{Y}(A_1A_2)$, and $\hat{Z}(A_2A_2)$, where $\hat{Y}$ and $\hat{Z}$ are given by equations (7) and (5), respectively, and $\hat{X} = 1 - \hat{Y} - \hat{Z}$. B_2 is assumed to be introduced into this population at a low frequency. Since $B_2 - A_2/A_2$ is totally sterile, the only genotypes which we need to consider are B_2A_1/B_1A_1, B_2A_1/B_1A_2, and B_2A_2/B_1A_1. Let the frequencies of these genotypes among the adults of a given generation be ε_1, ε_2, and ε_3, respectively. Ignoring second-order terms in the ε's, we obtain the following recurrence relations:

$$\begin{aligned} \varepsilon'_1 &= a[\varepsilon_1 + (1 - R)\varepsilon_2 + R\varepsilon_3] \\ \varepsilon'_2 &= b[\varepsilon_1 + (1 - R)\varepsilon_2 + R\varepsilon_3] \\ \varepsilon'_3 &= a[R\varepsilon_2 + (1 - R)\varepsilon_3], \end{aligned} \tag{A1}$$

where $a = (\hat{X} + \frac{1}{2}\hat{Y})(1 - s)(1 + K)/(1 - \hat{Z})(1 + k)$,

$$b = [\tfrac{1}{2}\hat{Y}(1 - s) + \hat{Z}(1 + k)](1 + K)/(1 - \hat{Z})(1 + k).$$

The characteristic equation for this system is:

$$\lambda\{\lambda^2 - \lambda[2a + b - R(a + b)] + a[(a + b) - R(a + 2b)]\} = 0. \tag{A2}$$

Equation (A2) has one root of zero and the other two given by the zeros of the quadratic in braces. Since the matrix for the linear transformation (A1) is nonnegative, the largest eigenvalue is real and positive and must therefore be the larger zero of the quadratic. By the rule for the differentiation of an implicit function, the derivative of the largest eigenvalue, λ_0, with respect to R is:

$$\frac{d\lambda_0}{dR} = \frac{a(a + 2b) - \lambda_0(a + b)}{2\lambda_0 - [2a + b - R(a + b)]}. \tag{A3}$$

At $R = 0$, equation (A2) shows that $\lambda_0 = a + b$ and (A3) that $d\lambda_0/dR = -b$. Together with the fact that (A3) implies that $d^2\lambda_0/dR^2$ has the same sign as $d\lambda_0/dR$ at each point, this proves that λ_0 is a concave, decreasing function of R.

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