SELECTION WITH GENE-CYTOPLASM INTERACTIONS. II. MAINTENANCE OF GYNODIOECY

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ABSTRACT

Gynodioecy is apparently frequently inherited through gene-cytoplasm interactions. General conditions for the protectedness of gene-cytoplasm polymorphisms for a biallelic model with two cytoplasm types were obtained previously, and these are applied to seven special cases of gene-cytoplasm interactions controlling gynodioecy and involving dominance. It is assumed that nuclear polymorphisms cannot be maintained in one cytoplasm type only. It is held that pure cytoplasmic inheritance of gynodioecy without nuclear interactions is unlikely, and it is shown that gynodioecy with gene-cytoplasm interactions is easier to establish than purely nuclear gynodioecy, for monogenic biallelic dominant or recessive inheritance. For three special cases, a resource-allocation model with simple assumptions always leads to conditions for protectedness of gynodioecy.

THERE is abundant evidence that all components of fitness, such as male and female fertility, viability and selfing rate, are affected by gene-cytoplasm interactions (see, *e.g.*, reviews by OEHLKERS 1964; EDWARDSON 1970). Nevertheless, there appear to be very few general studies of the population genetics of such interactions (GREGORIUS and ROSS 1984). Male fertility in flowering plants seems to be particularly susceptible to gene-cytoplasm interactions, and complete male sterility, caused by such interactions, is the basis of the hybrid seed industry in maize and other important crops (FRANKEL and GALUN 1977). Such male sterility occurs also in natural populations, where it gives rise, together with hermaphroditism, to the breeding system known as gynodioecy. Male sterility (femaleness) in gynodioecious species is apparently usually inherited through gene-cytoplasm interactions (ROSS 1978), and purely nuclear inheritance, or cytoplasmic inheritance with no nuclear effects, is apparently rare.

The aims of this paper are to discover conditions under which gene-cytoplasm polymorphisms for male sterility and hermaphroditism are maintained and why other forms of inheritance are apparently rare in natural gynodioecious populations.

Previous studies of gene-cytoplasm gynodioecy failed to obtain conditions that allowed a polymorphism (WATSON and CASPARI 1960; CHARLESWORTH

and GANDERS 1979) or they found them for the case of unidirectional pollen or seed migration (CASPARI, WATSON and SMITH 1966; COSTANTINO 1971). CHARLESWORTH (1981) found numerically some conditions that allowed a gene-cytoplasm polymorphism, and DELANNAY, GOUYON and VALDEYRON (1981), in addition to numerical results, found analytically equilibrium frequencies in which both the nuclear genes and the cytoplasm types were polymorphic, for the case of complete selfing among hermaphrodites.

The present study applies to gynodioecious populations the results of a previous general study of the maintenance of gene-cytoplasm polymorphisms (GREGORIUS and ROSS 1984). Purely cytoplasmically controlled polymorphisms (*i.e.*, those showing no apparent nuclear polymorphisms) are theoretically possible for gynodioecy, in contrast to the situation for hermaphroditism (GREGORIUS and ROSS 1984), since there is an inherent frequency dependency which may protect the cytoplasm that controls hermaphroditism, in a two-cytoplasm system. However, such a system seems unlikely on evolutionary grounds, since it would imply a failure of the nuclear genes to control the cytoplasmic genes, which contradicts the whole history of eukaryote development. The present study describes a model with two alleles and two cytoplasms and makes the assumption that, in the presence of the first cytoplasm alone, one of the alleles becomes fixed, whereas in the presence of the second cytoplasm alone, the other allele becomes fixed. This is because a previous study has established the conditions required for a nuclear gynodioecy (GREGORIUS, ROSS and GILLET 1982), and we now require fixation for the allele for hermaphroditism in this cytoplasm, since otherwise we obtain no new results and confound the conditions required for the maintenance of nuclear gynodioecy with those for nucleocytoplasmic gynodioecy. In addition, all of the special cases considered are dominance models, which imply (for sexual symmetry) the fixation of one allele. The allele that is fixed in the hermaphroditism cytoplasm, however, cannot be the same as the one fixed in the gynodioecy cytoplasm, as otherwise no polymorphism would be possible. The present study allows for variation in ovule or pollen fertility and in selfing rate, caused by both the nuclear genes and the cytoplasm genes, and obtains analytically conditions for the protectedness of gene-cytoplasm-determined sex polymorphisms. Another topic of interest lies in the apparently high proportion of gynodioecious species which show nucleocytoplasmic inheritance (ROSS 1978), so that in the present paper a comparison of such gynodioecy with simple nuclear inherited gynodioecy is made.

THE MODEL

The model is similar to that already considered (GREGORIUS and ROSS 1984). We assume a large population with two cytoplasm types S and N, together with two alleles A_1 and A_2 at the gene locus A. Both the cytoplasm types and the alleles may influence viability, male or female fertility and ovule-selfing rate. The ovule-selfing rate is the same for all hermaphrodite types within either cytoplasm type. Female and male fertilities are denoted by ϕ's and μ's, respectively, the ovule-selfing rates by σ's and the genotype frequencies by P's.

TABLE 1

The main features of the model

Trait	Genotypes					
	$N\ A_1A_1$	$N\ A_1A_2$	$N\ A_2A_2$	$S\ A_1A_1$	$S\ A_1A_2$	$S\ A_2A_2$
Ovule, pollen fertilities	ϕ_{N11}, μ_{N11}	ϕ_{N12}, μ_{N12}	ϕ_{N22}, μ_{N22}	ϕ_{S11}, μ_{S11}	ϕ_{S12}, μ_{S12}	ϕ_{S22}, μ_{S22}
Ovule-selfing rates[a]	σ_N	σ_N	σ_N	σ_S	σ_S	σ_S
Frequencies	P_{N11}	P_{N12}	P_{N22}	P_{S11}	P_{S12}	P_{S22}

[a] If a genotype is female, its selfing rate is taken as zero.

The ϕ's and μ's refer to all selection from zygote (directly after gamete fusion) to zygote and, thus, contain both viability and fertility components. The fertility component of the ϕ's may refer to both ovule and seed production, as it is assumed that all ovules are fertilized. The latter assumption is justified since we are interested in finding conditions for the establishment and maintenance of females. Hence, it is of primary interest to study situations in which females are rare, so that pollen is always abundant. If ϕ's are to refer to seed production, then the further assumption is made that the same proportion of ovules develop into seeds regardless of parent or offspring genotype, *i.e.*, that viabilities during the period of seed development are all equal. The model yields six genotypes: $N\ A_1A_1$ with female, male fertilities ϕ_{N11}, μ_{N11}, ovule-selfing rate σ_N and frequency P_{N11}; $N\ A_1A_2$ with female, male fertilities ϕ_{N12}, μ_{N12}, ovule-selfing rate σ_N and frequency P_{N12}, and so on. Female genotypes are assumed to have an ovule-selfing rate of zero, all ovules are fertilized and the amount of pollen used in selfing is assumed to be negligible. The cytoplasmic genes are transmitted by the ovules only and are always transmitted. The main features of the model are given in Table 1, and the transition equations are derived in GREGORIUS and ROSS (1984). It is assumed that when only N cytoplasm is present allele A_1 becomes fixed, whereas when only S cytoplasm is present allele A_2 becomes fixed. Therefore, genotypes $N\ A_1A_1$ and $S\ A_2A_2$ must always be hermaphrodite. If a cytoplasm contains hermaphrodite genotypes only, these are assumed to be sexually symmetrical, *i.e.*, the ratio ϕ/μ is constant for each genotype. This assumption is required in order to study the effect of femaleness restricted to a *single* cytoplasm. Otherwise, extreme forms of sexual asymmetry in the hermaphrodite cytoplasm could lead to an essentially female type in this cytoplasm also and would, thus, obscure the effect of gynodioecy expressed in one cytoplasm only. Cases in which females are expressed in both cytoplasms are studied later.

All of the special cases considered here show dominance, and in all cases we consider that dominance or recessiveness apply not only to hermaphroditism or femaleness but also to the quantities of gametes produced. For example, if gene A_1 for hermaphroditism is dominant in N cytoplasm, then the hermaphrodites $N\ A_1A_1$, $N\ A_1A_2$ are assumed to have equal numbers of ovules and pollen grains. In addition, in all special cases we consider that there are two original populations that are fixed for the hermaphrodite genotypes $N\ A_1A_1$

TABLE 2

Phenotypes for various special cases

Situation	Dominant allele in:		Genotypes					
	N cyto	*S* cyto	NA_1A_1	NA_1A_2	NA_2A_2	SA_1A_1	SA_1A_2	SA_2A_2
(a) Dom gd in *S* cyto only	A_1	A_1	H[a]	H[a]	H	F[a]	F[a]	H
(b) As previous case	A_2	A_1	H	H[a]	H[a]	F[a]	F[a]	H
(c) Rec gd in *S* cyto only	A_2	A_2	H	H[a]	H[a]	F	H[a]	H[a]
(d) As previous case	A_1	A_2	H[a]	H[a]	H	F	H[a]	H[a]
(e) Dom gd in both cytos	A_2	A_1	H	F[a]	F[a]	F[a]	F[a]	H
(f) Rec gd in both cytos	A_1	A_2	H[a]	H[a]	F	F	H[a]	H[a]
(g) Dom gd in *N* cyto, rec gd in *S* cyto	A_2	A_2	H	F[a]	F[a]	F	H[a]	H[a]

Abbreviations: Dom = dominant, rec = recessive, gd = gynodioecy, cyto = cytoplasm, H = hermaphrodite, F = female.

[a] Phenotypes corresponding to the dominant allele.

and *S* A_2A_2, respectively. The other genotypes are regarded as new types which may arise by mutation, migration or fusion of existing populations, and these new types are referred to as mutants, in contrast to the original types.

ANALYTICAL RESULTS

Case (a). Dominant gynodioecy in S *cytoplasm only*: We now consider the case of simple dominant gynodioecy, in which allele A_1 is dominant in both cytoplasms but causes male sterility (femaleness) in *S* cytoplasm only. The phenotypes and genotypes for this and the other special cases are given in Table 2. By applying the results of the previous paper (see Appendix in GREGORIUS and ROSS 1984), we find that

$$\theta_S = \max\{\phi_{S11}, \sigma_S\phi_{S22}\}/\phi_{N11},$$

where $\phi_{S11} \leq 2\phi_{S22}$ [GREGORIUS, ROSS and GILLET 1982, case (a)] because of the assumption of fixation of allele A_2 in *S* cytoplasm and where θ_S is the multiplication rate of cytoplasm *S* when its frequency is very low. Similarly, we find that

$$\theta_N = \max\{\phi_{N22}, \tfrac{1}{2}(1 + \sigma_N)\phi_{N11}\}/\phi_{S22},$$

where $\phi_{N11} > \phi_{N22}$ because of the assumption of fixation of allele A_1 in *N* cytoplasm. That such fixation follows for $\phi_{N11} > \phi_{N22}$ and sexual symmetry can be seen by applying the fitness values derived by GREGORIUS and ROSS (1981) to equation 4 in GREGORIUS (1982). Since $\theta > 1$ is the condition for protectedness, we see that *S* cytoplasm is protected, for example, if the new female genotypes *S* A_1A_1 or *S* A_1A_2 produce more ovules than the original *N* cytoplasm

hermaphrodite. This condition is similar to that found for pure cytoplasmic inheritance (LEWIS 1941), and a population consisting of genotypes $N\ A_1A_1$ and $S\ A_1A_1$ only corresponds to the purely cytoplasmic model. Increased seed set on females could occur if females were able to use for ovule production some of the resources that would otherwise be used for pollen. This, however, is not enough to guarantee the maintenance of the females, since we have not excluded the possibility of subsequent fixation of the S cytoplasm. By our basic assumptions, such fixation would also imply fixation of the A_2 allele and, thus, loss of the females. Therefore, both cytoplasms must be protected in order to protect the gynodioecy, so that $\theta_N > 1$ is also required.

We see that both θ's are sometimes dependent on the σ's, of which they are increasing functions. Thus, it is useful to consider separately those cases in which the θ's are not dependent on the σ's and those cases in which they are.

Condition (3) in part 1 of this series (GREGORIUS and ROSS 1984) shows that gynodioecy is protected independently of the σ's if

$$\phi_{S11} > \phi_{N11} \text{ and } \max\{\phi_{N22}, \tfrac{1}{2}\phi_{N11}\} > \phi_{S22}.$$

Hence, our fixation assumptions $\phi_{N22} < \phi_{N11}$ and $\phi_{S11} \leq 2\phi_{S22}$ have to be supplemented by the conditions

$$\phi_{N11} < \phi_{S11} \text{ and } \phi_{S22} < \phi_{N22}.$$

Therefore, the female mutant must have more ovules than the original hermaphrodite in the other cytoplasm, and the original hermaphrodite in S cytoplasm must have fewer ovules than the mutant in N cytoplasm. If either of these conditions do not hold, then the σ's may become relevant for protectedness of gynodioecy provided the necessary conditions for protectedness (2) of GREGORIUS and ROSS (1984) are met. For the present model, these are

$$\phi_{S22} < \phi_{N11} < \phi_{S11},$$

so that the female mutant must have more ovules than either original hermaphrodite and the original hermaphrodite in N cytoplasm more than the original hermaphrodite in S cytoplasm. Note that these conditions are necessary for protectedness, so that, if they are not fulfilled, there is no protection, regardless of the σ's. Since

$$\theta_S = \max\{\phi_{S11}, \sigma_S\phi_{S22}\}/\phi_{N11},$$

the above necessary conditions imply $\theta_S = \phi_{S11}/\phi_{N11} > 1$, so that the S cytoplasm is protected for all σ_S. Consequently, only σ_N may be relevant. To analyze this recall that

$$\theta_N = \max\{\phi_{N22}, \tfrac{1}{2}(1 + \sigma_N)\phi_{N11}\}/\phi_{S22}.$$

Hence, protectedness does not depend on σ_N if $\phi_{N22} > \phi_{S22}$. Otherwise, for $\phi_{N22} \leq \phi_{S22}$, the gynodioecy is protected for sufficiently large $\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$. Therefore,

$$\phi_{N22} \leq \phi_{S22} < \phi_{N11} < \phi_{S11}$$

is the only situation in which protectedness depends upon σ, and this requires that the hermaphrodite mutant in the N cytoplasm has the lowest number of ovules of all genotypes. The results of this and of the other special cases are summarized in Table 2.

Case (*b*): If A_2 is dominant in N cytoplasm instead of A_1, the phenotypic assignments remain the same (Table 1), but there is a difference from the previous case because now $\phi_{N12} = \phi_{N22}$ and $\mu_{N12} = \mu_{N22}$, instead of $\phi_{N12} = \phi_{N11}$ and $\mu_{N12} = \mu_{N11}$. The previous fixation conditions remain the same, so that now the gynodioecy is protected for all σ's under the same additional conditions as before, *i.e.*,

$$\phi_{N11} < \phi_{S11} \text{ and } \phi_{S22} < \phi_{N22}.$$

As before σ's may become relevant for protectedness of the gynodioecy if $\phi_{S22} < \phi_{N11} < \phi_{S11}$. From this it follows that $\theta_S > 1$, so that protectedness is independent of σ_S.

The expression for σ_N is more complicated than the previous case. Thus,

$$\theta_N = \max\{\phi_{N22}, \alpha_N\}/\phi_{S22}$$

where

$$\alpha_N = \tfrac{1}{2}\sigma_N\phi_{N11} + \tfrac{1}{2}\phi_{N22} + \tfrac{1}{2}\sqrt{\tfrac{1}{4}\phi_{N22} + \sigma_N^2\phi_{N11}(\phi_{N11} - \phi_{N22})}.$$

Since $\phi_{N11} > \phi_{N22}$, α_N and thus θ_N are increasing functions of σ_N. If in the above inequalities for σ-independent protectedness $\phi_{S22} \geq \phi_{N22}$, then $\theta_N = \phi_{N22}/\phi_{S22} \leq 1$ for $\sigma_N = 0$, and gynodioecy is not protected. For the other extreme, $\sigma_N = 1$, $\theta_N = \phi_{N11}/\phi_{S22} > 1$, and gynodioecy is protected. Thus, protectedness depends again on sufficiently large σ_N if the hermaphrodite mutant in the N cytoplasm has the lowest number of ovules.

Recessive gynodioecy in S *cytoplasm only*: The only difference from the previous cases is that the allele A_1 which causes femaleness in S cytoplasm is now recessive, so that S A_1A_1 is the only female genotype. As previously, either allele A_1 or A_2 may be dominant in N cytoplasm.

Case (*c*): A_2 is dominant in N cytoplasm and, therefore, in both cytoplasms. This is intuitively the simplest case of recessive gynodioecy. Straightforward calculations show that in this case the conditions for the protectedness of gynodioecy are identical with those of case (b) (for dominant gynodioecy). Hence, recessive gynodioecy with the same allele dominant in both cytoplasms gives the same results as dominant gynodioecy with different alleles dominant in the two cytoplasms.

Case (*d*): A_1 is dominant in N cytoplasm, and A_2 is dominant in S cytoplasm. Again, when the fixation assumptions are applied to conditions (3) in part 1, it follows after some rearrangements that the gynodioecy is again protected for all σ's under the additional conditions

$$\phi_{N11} < \phi_{S11} \text{ and } \phi_{S22} < \phi_{N22}.$$

Again, the σ's may become relevant for protection if $\phi_{S22} < \phi_{N11} < \phi_{S11}$, and, therefore, protection does not depend on σ_S. We now obtain

$$\theta_N = \max\{\phi_{N22}, \tfrac{1}{2}(1 + \sigma_N)\phi_{N11}\}/\phi_{S22},$$

which is the same results as in case (a), so that σ_N becomes relevant only if $\phi_{S22} \geq \phi_{N22}$, in which case

$$\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$$

guarantees protectedness.

Case (e): Dominant gynodioecy in both cytoplasms: We now allow both cytoplasms to interact with the nucleus to produce femaleness. As always the original types $N\ A_1A_1$ and $S\ A_2A_2$ are hermaphrodite, and, thus, it follows from the dominance assumption that all of the remaining types are female. Notice that this requires A_2 dominant for femaleness in N cytoplasm and A_1 dominant for femaleness in S cytoplasm. The fixation conditions are now $\phi_{S11} \leq 2\phi_{S22}$ and $\phi_{N22} \leq 2\phi_{N11}$, so that it is only required for each cytoplasm that the female mutant does not have more than twice as many ovules as the hermaphrodite. We obtain

$$\theta_S = \max\{\phi_{S11}, \sigma_S\phi_{S22}\}/\phi_{N11},\ \theta_N = \max\{\phi_{N22}, \sigma_N\phi_{N11}\}/\phi_{S22},$$

yielding the result that gynodioecy is protected for all σ's if $\phi_{N11} < \phi_{S11}$ and $\phi_{S22} < \phi_{N22}$. Thus, each mutant female must have more ovules than the hermaphrodite in the other cytoplasm. There are now two ways in which the conditions for protectedness for all σ's are not fulfilled, namely, when $\phi_{N11} \geq \phi_{S11}$ and when $\phi_{S22} \geq \phi_{N22}$. In the former case gynodioecy is protected for all σ_N and for $\sigma_S > \phi_{N11}/\phi_{S22}$, whereas in the latter case it is protected for all σ_S and $\sigma_N > \phi_{S22}/\phi_{N11}$.

Case (f). Recessive gynodioecy in both cytoplasms: The results for this and the remaining special case are not considered in detail but are given in Table 2, together with the summaries of the other results. Since $N\ A_1A_1$ and $S\ A_2A_2$ are hermaphrodite, the present case implies that only genotypes $N\ A_2A_2$ and $S\ A_1A_1$ are female and that allele A_1 is dominant in N cytoplasm and A_2 in S cytoplasm.

Case (g). Dominant gynodioecy in N *cytoplasm, recessive gynodioecy in* S *cytoplasm*: This situation requires A_2 be dominant for femaleness in N cytoplasm and for hermaphroditism in S cytoplasm.

DISCUSSION

General results: A comparison of the situation in which females may occur in one cytoplasm only [first group of special cases (a) to (d)] with that in which they may occur in both cytoplasms (second group) shows that the fixation and protectedness conditions are easier to fulfill in the second group. In the first group protectedness for all σ's (under the fixation conditions) requires

$$\phi_{S22} < \phi_{N22} < \phi_{N11} < \phi_{S11} \leq 2\phi_{S22}$$

for all dominance relations. The requirement that the female genotype $S\ A_1A_1$ should have more ovules than any hermaphrodite is perhaps not difficult to fulfill, since females may have additional resources for ovule production made available through the absence of a requirement to produce pollen. It seems less likely, however, that the normal hermaphrodite in S cytoplasm should have

fewer ovules than the mutant hermaphrodite in N cytoplasm, *i.e.*, that $\phi_{S22} < \phi_{N22}$. This inequality $\phi_{S22} < \phi_{N22}$ is required for the second group of special cases also (*i.e.*, where females may occur in both cytoplasms). However, in this group the mutant type $N\ A_2A_2$ is always female and may have more ovules than a hermaphrodite for the reason already given. When protectedness depends upon σ's (last column, Table 3) the group I models show protection for all σ_S and sufficient σ_N. It may be sufficient that $\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$, but this requires high σ_N if the two original hermaphrodite genotypes do not differ greatly in ovule fertility, *e.g.*, $\sigma_N > 0.8$ if ϕ_{S22} is 0.9 as great as ϕ_{N11}. The other models in group I require a more complex formula for σ_N (GREGORIUS and ROSS 1984) and show, *e.g.*, for $\phi_{N22} = 0.8$, $\phi_{S22} = 0.9$, $\phi_{N11} = 1$, that σ_N must have a minimum value of between 0.5 and 0.6. The group II models also seem to require high σ's (for similar ϕ_{S22}, ϕ_{N11}). However, ϕ's need not always be similar, as may be seen, for example, by comparing the self-incompatible race c1 of *Leavenworthia crassa* with the probably much more inbreeding race c15 [ϕ's equal 2762 and 799, respectively, (LLOYD 1965, Tables 3 and 6)]. Notice that for group I models the female must have the greatest ovule fertility of all genotypes for protection to occur, but that for group II models are female is required to have the greatest ovule fertility, whereas the other may have much less. For example, protection for all σ's may occur in group II models when

$$\phi_{S22} < \phi_{N22} < \phi_{N11} < \phi_{S11} \leq 2\phi_{S22},$$

and it may depend upon σ's when

$$\phi_{N22} \leq \phi_{S22} < \phi_{N11} < \phi_{S11} \leq 2\phi_{S22},$$

where ϕ_{N22} and ϕ_{S11} are the ovule fertilities of the female genotypes. Thus, in the latter case one female genotype must have the lowest and the other the highest ovule fertility of all genotypes. This requirement that females with one cytoplasm type have a low seed fertility, whereas those with the other cytoplasm have the highest seed fertility of all genotypes, seems to be met in *Plantago lanceolata*. In this self-incompatible gynodioecious species there are two morphologically and cytoplasmically distinct types of female (MS1 and MS2), which are usually found together in natural populations (VAN DAMME and VAN DELDEN 1982). Inheritance of gynodioecy is through interaction of several nuclear genes with the two cytoplasm types (VAN DAMME 1983a), and MS1 produces more seed than hermaphrodites, whereas MS2 produces similar amounts (VAN DAMME 1983b). However, it is not known whether the two female types have a nuclear gene in common, in this species.

Several other results seem worth emphasizing. A female with a seed fertility too low to allow a polymorphism in the group I models may become established in the group II models. This is another example of the importance of the mode of inheritance on the maintenance of sex polymorphisms (GREGORIUS, ROSS and GILLET 1982). For all models, if females are protected for $\sigma = 0$, then they are protected for all σ's. For all models, if a cytoplasm is at a disadvantage, it must have a sufficiently great σ value, if the polymorphism is to be protected.

TABLE 3

Summary of results

Situation	Dominant allele in:		Fixation conditions	Conditions for protectedness of gynodioecy		
				Necessary for protectedness	Protected for all σ's	Protectedness depends on σ's
	N cyto	S cyto				
(a) Dom gd in S cyto only	A_1	A_1	$\phi_{N22} < \phi_{N11}$, $\phi_{S11} \leq 2\phi_{S22}$	$\phi_{S22} < \phi_{N11} < \phi_{S11}$	$\phi_{N11} < \phi_{S11}$, $\phi_{S22} < \phi_{N22}$	$\phi_{S22} \geq \phi_{N22}$, $\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$, all σ_S
(b) As previous case	A_2	A_1	As previous case	As previous case	As previous case	$\phi_{S22} \geq \phi_{N22}$, sufficient σ_N, all σ_S
(c) Rec gd in S cyto only	A_2	A_2	As previous case	As previous case	As previous case	As previous case
(d) As previous case	A_1	A_2	As previous case	As previous case	As previous case	$\phi_{S22} \geq \phi_{N22}$, $\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$, all σ_S
(e) Dom gd in both cytos	A_2	A_1	$\phi_{N22} \leq 2\phi_{N11}$, $\phi_{S11} \leq 2\phi_{S22}$	$\text{Max}\{\phi_{N11}, \phi_{N22}\} > \phi_{S22}$, $\text{Max}\{\phi_{S11}, \phi_{S22}\} > \phi_{N11}$	As previous case	(1) $\phi_{S22} \geq \phi_{N22}$, $\sigma_N > \phi_{S22}/\phi_{N11}$, all σ_S (2) $\phi_{N11} \geq \phi_{S11}$, $\sigma_S > \phi_{N11}/\phi_{S22}$, all σ_N
(f) Rec gd in both cytos	A_1	A_2	As previous case	As previous case	As previous case	(1) $\phi_{S22} \geq \phi_{N22}$, $\sigma_N > 2\phi_{S22}/\phi_{N11} - 1$, all σ_S (2) $\phi_{N11} \geq \phi_{S11}$, $\sigma_S > 2\phi_{N11}/\phi_{S22} - 1$, all σ_N
(g) Dom gd in N cyto, rec gd in S cyto	A_2	A_2	As previous case	As previous case	As previous case	(1) $\phi_{S22} \geq \phi_{N22}$, $\sigma_N > \phi_{S22}/\phi_{N11}$, all σ_S (2) $\phi_{N11} \geq \phi_{S11}$, $\sigma_S > 2\phi_{N11}/\phi_{S22} - 1$, all σ_N

Abbreviations as for Table 1.

The present results seem to throw light on other experimental results, in addition to those for *P. lanceolata* already considered. In *Armeria maritima*, European populations are self-incompatible hermaphrodites with showy flowers, but during migration by long-distance dispersal through Arctic regions to North America, this species became self-compatible, with less showy flowers. In California, however, this species has showy flowers and is gynodioecious (BAKER 1967), so that this could be a case in which protectedness of gynodioecy depended upon sufficiently high σ's and adequate pollination.

Is gynodioecy ever an outbreeding mechanism? Gynodioecy may be an outbreeding mechanism since by definition it introduces an additional amount of outbreeding into a population if it hitherto consisted of partially selfing hermaphrodites. Moreover, gynodioecy also reinforces negative assortative mating by excluding matings among females and inducing a greater proportion of hermaphrodites to mate with the other rather than with their own type. However, several gynodioecious species are self-incompatible, so that the gynodioecy cannot be an outbreeding mechanism in such species (BAKER 1963; ROSS 1970; HOROVITZ and GALIL 1972), although negative assortative mating is reinforced here also. Under the present model it is clear that females may become established whether they increase the degree of outcrossing or not. This last conclusion is supported by the situation in *Plantago maritima* from Europe and closely related species or varieties in North America. European types are self-incompatible and at least sometimes gynodioecious, whereas North American types are self-compatible but gynodioecy has apparently not been found (GREGOR 1939; ROSS 1970). Thus, there seems to be an association between self-incompatibility and gynodioecy in this species.

Nucleocytoplasmic vs. *nuclear gynodioecy*: Most gynodioecious species appear to show gene-cytoplasmic inheritance (ROSS 1978), so that the question arises why this should be so and why should simple monogenic inheritance be apparently rare? Does gene-cytoplasm gynodioecy have an advantage over nuclear gynodioecy? This question has two aspects. First, if gynodioecy is maintained by interaction between nucleotypes and plasmatypes in one population, and by nuclear effects alone in a second population, which system would prevail if they were combined in a single population? Second, for the case of dominance, if females have available two ways of becoming established in a bisexual population, namely, via a new plasmatype with nuclear interaction or via a new nuclear gene, which of the strategies requires a smaller increase in female reproductive effort and is, therefore, more likely to occur? In this paper we have answered the second aspect of the question by showing for a single gene locus with dominance that gene-cytoplasm gynodioecy is considerably more successful than nuclear gynodioecy (compare the results of GREGORIUS, ROSS and GILLET 1983). However, both nuclear (ROSS and WEIR 1975) and gene-cytoplasmic (M. D. ROSS, unpublished results) gynodioecy can be established for loci that show overdominance for fertility, so that the possibility remains that gene-cytoplasm gynodioecy is more frequent than genic gynodioecy because the cytoplasm prevents the further evolution of gynodioecy toward dioecy (ROSS 1978, 1982).

Resource allocation: We now apply a model of resource allocation (ROSS and GREGORIUS 1983) to the present results. The model assumes that every genotype is equally viable and has equal reproductive resources, which it fully uses and which may be variously distributed between male and female sex functions. The model cannot, therefore, not be applied to the group I models of the present paper, as these allow different reproductive resources between hermaphrodite types within a cytoplasm type. For group II models, we designate R_{N11} as the proportion of the total reproductive resources that are devoted to ovules or seeds for genotype $N\ A_1A_1$, and so on for the other genotypes, where total resources equal one for all genotypes. Thus, $R_{N22} = R_{S11} = 1$ for the female genotypes, and the fixation conditions require that $R_{N11}, R_{S22} \geq \frac{1}{2}$, *i.e.*, that the hermaphrodities must devote at least one-half of their reproductive resources to ovules or seeds. The necessary conditions for protectedness are always fullfilled, since $\max\{R_{N11}, R_{N22}\}$ and $\max\{R_{S11}, R_{S22}\}$ always equal 1, and are by definition greater than R_{S22}, R_{N11}, which are R values for hermaphrodites and must, therefore, be less than 1. Since $R_{S22}, R_{N11} < 1$, the polymorphism is protected for all selfing rates. These results may be further illustrated by using the fixation assumptions that in a population containing N cytoplasm only genotype $N\ A_1A_1$ would become fixed and analogously that $S\ A_2A_2$ would become fixed in S cytoplasm. We may, therefore, assign to these genotypes the optimal resource allocations of $\frac{1}{2}(1 + \sigma_N)$ and $\frac{1}{2}(1 + \sigma_S)$, respectively (ROSS and GREGORIUS 1983). For this situation the fixation conditions are always fulfilled, so that a polymorphism is always protected.

Fixation assumptions: The fixation assumptions, that the hermaphrodite homozygote A_1A_1 would become fixed if N cytoplasm only was present and that A_2A_2 would become fixed if S cytoplasm only was present, have been justified theoretically. Experimental grounds for the fixation assumptions can be found in the voluminous literature on nucleocytoplasmic male sterility (see *e.g.*, reviews by EDWARDSON 1970; MICHAELIS 1954; OEHLKERS 1964). For example, numerous hybridization experiments among races and species of Epilobium have shown that, when the nucleus of one species or race is put together with the cytoplasm of another, by hybridization and repeated backcrossing to the male parent, then the later generations frequently show male sterility but remain female fertile (MICHAELIS 1954). These results may be interpreted as showing that the nucleus and cytoplasm of each original strain are mutually adapted for male fertility, allowing us to regard the male fertility genes within each race as fixed. Since cytoplasmic male-sterility mutations are apparently rather infrequent (JAIN 1959), these results suggest that gynodioecy may often have arisen through intra- or interspecific hybridization.

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