THE POPULATION GENETICS OF GYNODIOECY WITH CYTOPLASMIC-GENIC MALE-STERILITY

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SUMMARY

Two models of gynodioecy controlled by cytoplasmic factors and nuclear genes are studied. In one model, there is a male-sterility determining cytoplasm which produces its effect only in the absence of a restorer allele at a nuclear locus. In the other model, either the cytoplasmic factor or the nuclear gene can produce male-sterility independently of the other. Neither model permits stable coexistence of the two types of factor in a population.

1. INTRODUCTION

GYNODIOECIOUS angiosperm populations consist of separate hermaphroditic and male-sterile (female) individuals. Male-sterility may be controlled by nuclear genes (Lloyd, 1974), cytoplasmically, or by the interaction of cytoplasm and nuclear genes (Grun, 1976). In cases of cytoplasmic-genic male-sterility, plants with male-sterile cytoplasm are phenotypically hermaphrodite if they have an active fertility-restoring allele at the nuclear gene locus, and are pistillate if they do not. This type of system is well known and important in crop plants (Duvick, 1965; Maan and Lücken, 1972) and has recently been demonstrated in a natural gynodioecious population (Ganders, 1978).

With either genic or cytoplasmic-genic inheritance of male-sterility, hermaphrodites leave descendants through both pollen and ovules, while male-steriles contribute descendants only through ovules. This is equivalent to selection against male-sterility, and male-steriles can be maintained in populations only if there is some counteracting selection for male-sterility. Lloyd (1974, 1975) has shown that male-steriles can be maintained in populations if their obligatorily outcrossed progeny are more fit than the inbred progeny from self-fertilised hermaphrodites. He derived equations for the equilibrium frequency of females as a function of the selfing rate of hermaphrodites (s) and the relative survival rate of the progeny of self-fertilisation ($1 - \delta$), for the case of male-sterility determined by dominant or recessive nuclear genes, and for cytoplasmic male-sterility.

The purpose of the present paper is to study models involving both nuclear genes and cytoplasmic factors. The first model assumes cytoplasmic male-sterility with restorer genes, as outlined above. We shall show that if any male-sterile genotypes are maintained, the population will also be fixed for the male-sterile cytoplasm. Previous studies of the population genetics of this type of system have assumed that progeny from self-fertilised and non-selfed ovules are of equal viability ($\delta = 0$), and that the ovule output of the male-sterile and hermaphrodite plants are the same. These studies showed

a strong disadvantage to a sterility allele in these circumstances (Watson and Caspari, 1960), even in the face of considerable mutation pressure or pollen flow (Caspari *et al.*, 1966; Costantino, 1971).

We have also studied a model of male-sterility caused independently by either a nuclear gene or a cytoplasmic factor.

2. THE MODELS

(i) *Cytoplasmic male-sterility with a restorer gene*

The first model assumes that there are two types of cytoplasm, (F) and (S). Cytoplasm (S) permits sterility of certain genotypes (*e.g.* *FF* and *Ff* are female and *ff* hermaphrodite, in the case of a recessive restorer gene). All genotypes are hermaphrodite if they have the (F) cytoplasm. If restoration of fertility by the restorer gene is complete, there will be just two phenotypes, females and hermaphrodites. Let the selfing rate of hermaphrodites be s , and the inbreeding depression be δ . The females cannot self-fertilise.

Let the frequencies of *FF*, *Ff* and *ff* with (F) cytoplasm be h_1 , h_2 and h_3 , respectively. Let the frequency of plants with (F) cytoplasm be P ($P = h_1 + h_2 + h_3$). The frequencies of these forms in the next generation, assuming that the ovule output of females equals that of hermaphrodites, are:

$$h'_1 = \{s(1-\delta)(h_1 + h_2/4) + p(1-s)(h_1 + h_2/2)\}/X$$

$$h'_2 = \{s(1-\delta)h_2/2 + (1-s)(h_1[1-p] + h_2/2 + h_3p)\}/X$$

$$h'_3 = \{s(1-\delta)(h_2/4 + h_3) + (1-s)(1-p)(h_2/2 + h_3)\}/X$$

where p is the frequency of *F* in the pollen, and X is a normalising factor. Therefore

$$\begin{aligned} P' &= P[s(1-\delta) + 1-s]/X \\ &= P(1-s\delta)/X. \end{aligned} \quad (1)$$

X takes into account the total number of viable seeds produced by all sex forms, and is therefore given by

$$\begin{aligned} X &= H(1-s\delta) + 1-H \\ &= 1-Hs\delta \end{aligned}$$

where H is the frequency of hermaphrodites in the population. We thus have

$$\frac{P'}{P} = \frac{1-s\delta}{1-Hs\delta} \quad (2)$$

This defines the conditions for the (F) cytoplasm to spread. The ratio P'/P exceeds 1 only if $H > 1$, which is impossible. Therefore the frequency of (F) cytoplasm must always decrease, if females are present, whatever the mode of inheritance of male-sterility in (S).

All the above has assumed that the ovule output of females is the same as that of hermaphrodites, but this is unlikely to be so. Suppose that the ovule output of females is $1+k_z$ when the frequency of females in the population is z . This takes into account two factors, first that the females might have an intrinsically higher ovule output than hermaphrodites (when females are rare), and second that the chance of non-selfed ovules getting fertilised will

be likely to decrease as females become commoner. This second factor will affect all ovules of female plants, and all non-selfed ovules of hermaphrodites. Both factors can be incorporated by changing the normalising factor in equation (1) to

$$X = 1 + k_z(1 - H) - Hs\delta$$

in which k_z expresses the relative excess in ovule production of females over that of hermaphrodites, when females are present in frequency $\bar{z}$. Most models have assumed that all ovules of hermaphrodites get fertilised, either by selfing or outcrossing (Lloyd, 1974); this assumption permits s in the above expression to be a constant. The more general case, in which non-selfed ovules of hermaphrodites may remain unfertilised if females get too common, can also be expressed as above, remembering that s now becomes an increasing function of $\bar{z}$.

With this model, we therefore have

$$\frac{P'}{P} = \frac{1 - s\delta}{1 - Hs\delta + k_z(1 - H)} \quad (3)$$

which leads to the condition for the increase of (F) cytoplasm:

$$k_z < -s\delta.$$

If females are maintained in the population at all, this condition cannot be satisfied. The reason for this is as follows. Consider the fitnesses of females and hermaphrodites. If females are increasing in frequency, or are maintained at equilibrium, their fitness must be at least equal to that of the hermaphrodites. The hermaphrodites' fitness is made up of a component due to reproduction as male, and a component due to seed production (Lloyd, 1977; Charlesworth and Charlesworth, 1978), whereas females have zero pollen fitness. Therefore the ovule fitness of females must exceed that of hermaphrodites, unless females are decreasing in frequency. The relative ovule fitnesses of females and hermaphrodites when females are present at frequency $\bar{z}$ are $1 + k_z$ and $1 - s\delta$, respectively. Therefore $k_z > -s\delta$. This completes the proof that (F) cytoplasm will be eliminated from an (S) population in which a male-sterility allele and a restorer allele are maintained.

(ii) *Male-sterility gene plus male-sterility cytoplasm*

In this model, we assume that cytoplasm (S) causes male-sterility without the involvement of any nuclear genes. Also, plants with cytoplasm (F) can be rendered male-sterile by a nuclear gene which can be dominant or recessive. We shall assume first that the two causes both produce exactly the same female phenotype. Suppose that the sterility gene is at equilibrium. If cytoplasm (S) is introduced at frequency Q ($= 1 - P$), it is easy to show that its frequency in the next generation is

$$Q' = \frac{Q(1 + k_z)}{1 + k_z(1 - H) - Hs\delta} \quad (4)$$

using the same notation as in the previous model. Since $1 + k_z$ is necessarily larger than the denominator in this expression, the cytoplasmic factor can spread.

Equation (4) shows that a cytoplasmic factor causing male-sterility will

spread, and the fertility of females will continue to fall, until $k_Z = -s\delta$. This is equivalent to the result of Lloyd (1974) for cytoplasmically inherited male-sterility. Once cytoplasm (S) begins to spread, the fitness of females will therefore be reduced, relative to that of hermaphrodites. Within the portion of the population with (F) cytoplasm, females will thus be at a disadvantage. Also, when the (S) cytoplasm is at equilibrium, the genic male-sterility factor cannot spread. It will therefore be eliminated from a population in which the (S) cytoplasm is maintained, so that finally male-sterility will become wholly cytoplasmically determined.

Now suppose that the cytoplasmic factor (S) is associated with some disadvantage, such as lower viability or ovule output. The condition for such a factor to spread is given by equation (4), replacing the $(1+k_Z)$ in the numerators with $(1+k_Z)(1-c)$ where c is the reduction in viability of plants carrying the (S) cytoplasm, compared with (F) plants. Combining this modified equation (4) with the expression for the equilibrium frequency of females determined by a nuclear gene (Charlesworth and Charlesworth, 1978)

$$\hat{Z} = (k_Z + 2s\delta - 1) / [2(k_Z + s\delta)],$$

we obtain the condition for (S) to spread into a population at equilibrium for genically-determined females:

$$1 - c > \frac{1}{2}.$$

At equilibrium for (S) in the absence of male-sterility genes, we will have $(1-c)(1+k_Z) = 1-s\delta$. The condition for genically determined females to spread in such a population can be found by considering only the (F) portion of the population, since this is a closed system, with no contribution from the (S) plants. This condition is thus just the condition for a nuclear male-sterility factor to spread (Charlesworth and Charlesworth, 1978):

$$1 + k_Z > 2(1 - s\delta).$$

But, from the formula given above for the equilibrium of the (S) cytoplasm, we have

$$1 + k_Z = \frac{1 - s\delta}{1 - c}.$$

This yields the condition for the nuclear male-sterility gene to spread, $1 - c < \frac{1}{2}$, which cannot be satisfied if the condition for the sterility cytoplasm to spread into a population at equilibrium for the gene can be satisfied. Therefore, either the gene or the cytoplasmic factor must be eliminated, unless $c = \frac{1}{2}$, exactly, and the population already contains both factors.

3. DISCUSSION

The first model studied above leads to the conclusion that if the male-sterility (non-restorer) allele is maintained in the population, the male-sterile cytoplasm will become fixed. It is not possible with this model to maintain an equilibrium with both (F) and (S) cytoplasm and a restorer gene unfixed. If the male-sterility allele is eliminated, however, the (S) cytoplasm can remain in the population, but this is a neutral equilibrium, as under the assumptions of the model the different types of cytoplasm have no effect on the phenotype, in the absence of the sterility allele.

These conclusions have been confirmed by computer calculations

starting from a range of initial states of the population. In general, when moderate frequencies of male-sterile individuals were maintained at the equilibrium, the equilibrium was reached rapidly (in well under 200 generations when the starting populations contained all the genotypes at frequencies of 1 per cent or more). Equilibrium was reached very slowly, however, in cases where the equilibrium frequency of male-sterility was very small.

With the second model studied, with independent nuclear and cytoplasmic factors each able to produce male-sterility, the same conclusion is reached: once again, it is not possible to maintain a doubly polymorphic population.

These results suggest that gynodioecy caused by cytoplasmic male sterility factors with restorer genes should be rare in natural populations. Once the male-sterile cytoplasm became fixed, the male-sterility would appear to be genically controlled. The only way to demonstrate the cytoplasmic component would be by crossing to plants with male-fertile cytoplasm. Such plants would occur only in hermaphroditic populations or in populations that had not yet reached equilibrium. However, cytoplasmic-genic inheritance of male sterility has been documented in a natural population of *Nemophila menziesii* var. *intermedia*, a normally hermaphroditic taxon (Ganders, 1978). Gynodioecy has almost certainly been established recently in this population, so there may not yet have been enough time for the male-sterility cytoplasm to reach fixation. Alternatively, male-fertile cytoplasm could be periodically reintroduced by seeds from neighbouring hermaphrodite populations.

Other examples may not be so easy to explain. Some involvement of cytoplasmic factors in the male-sterility of natural or primitively cultivated gynodioecious species has been repeatedly suggested (*e.g.* Correns, 1928; Simmonds, 1971; Horowitz and Galil, 1972; Kheyr-Pour, 1975). In none of these cases has the genetics been fully worked out, but suggestions of complex systems of inheritance, involving nuclear genes as well as cytoplasmic factors, have been made for most of them. From the present results, it would be surprising if either of the two simple genic-cytoplasmic systems studied above was involved in the genetic control of male-sterility within any single population.

A final point concerns gynodioecy in self-incompatible species. It is well known that a cytoplasmic male-sterility factor can spread into a population of hermaphrodite plants, even if these are self-incompatible, provided that the ovule output of the females is at least somewhat increased (Lewis, 1941). In a self-compatible species, selection for the females is negatively frequency dependent; the quantity of pollen decreases as the frequency of females increases, and there comes a time when the ability of the hermaphrodites to self-fertilise gives them equal fitness with the females, and an equilibrium is established. It has been pointed out to us by J. Maynard Smith that this does not occur in a self-incompatible species, in which the hermaphrodites are as dependent as the females on pollen from other plants, and would presumably get the same fraction of their ovules fertilised as the females, unless pollinators detect and avoid female plants. Thus any advantage in terms of ovule number will be maintained however frequent females become: they could even go to fixation, and the population would die out. This means that we should not expect to find cytoplasmic male-sterility together

with self-incompatibility. A few cases of male-sterility with self-incompatibility have been reported (Horowitz and Galil, 1972; Young, 1972; Connor, 1973; Ross, 1973), and the genetics of the first of these, though not fully worked out, was reported to be partly cytoplasmic. This case would seem to merit further study. The second case has not been studied genetically, while the third, *Cortaderia selloana*, has equal frequencies of males and females, and might therefore be functionally dioecious. Although the genetics of these cases is not known, none of them shows a frequency of females above 50 per cent, which would strongly suggest cytoplasmic inheritance (though lower frequencies do not, of course, rule it out). The only other possible case of which we are aware is *Plantago lanceolata*, in which some Californian populations have more than 50 per cent females (H. G. Baker, pers. comm.), and which is self-incompatible (Ross, 1973), at least in some regions.

In conclusion, it appears that cytoplasmically inherited gynodioecy presents a number of problems which have not yet been solved.

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