Partial male sterility and the evolution of nuclear gynodioecy in plants

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Summary

Gynodioecy, a genetic dimorphism of females and hermaphrodites, is pertinent to an understanding of the evolution of plant gender, mating and genetic variability. Classical models of nuclear gynodioecy attribute the maintenance of the dimorphism to frequency-dependent selection in which the female phenotype has a fitness advantage at low frequency owing to a doubled ovule fertility. Here, I analyse explicit genetic models of nuclear gynodioecy that expand on previous work by allowing partial male sterility in combination with either fixed or dynamically evolving mutational inbreeding depression. These models demonstrate that partial male sterility causes fitness underdominance at the mating locus, which can prevent the spread of females. However, if partial male sterility is compensated by a change in selfing rate, overdominance at the mating locus can cause the spread of females. Overdominance at introduction of the male sterility allele can be caused by high inbreeding depression and a lower selfing rate in the heterozygote, by purging of mutations by a higher selfing rate in the heterozygote, and by low inbreeding depression and a higher selfing rate in the heterozygote. These processes might be of general importance in the maintenance of mating polymorphisms in plants.

1. Introduction

The selective maintenance of a stable dimorphism of female and hermaphrodite individuals ('gynodioecy') is pertinent to an understanding of the evolution of plant gender and sexual dimorphism (Frank, 1989; Geber *et al.*, 1999), the allocation of limiting resources to sexual function (Ashman *et al.*, 2001; Delph & Carroll, 2001), outcrossing rate and mating system (Ashman, 2000; Williams *et al.*, 2000), inbreeding depression, and rate of deleterious mutation (Schultz & Ganders, 1996; Schultz, 1999). Phenotype-based models of gynodioecy have concluded that loss of male function must be compensated by a doubling of ovule success in order for females to be selectively maintained in populations of hermaphrodites (Lloyd, 1975; Charlesworth & Charlesworth, 1978). Two major sources of this compensation have been proposed: (1) the resources liberated from the defunct male structures might be redirected to increase female fertility (Darwin's 'Law of Compensation', Darwin, 1877);

and (2) the lack of male function prevents self-fertilization and the consequent production of genetically lower-quality seed progeny (Mather, 1940; Lewis, 1941; Thomson & Barrett, 1981; Lloyd, 1982).

Phenotype-based models, however, make genetic simplifying assumptions that are known to be violated in nature. Schultz (1999) examined two such assumptions: that the selective effect of self-fertilization is influenced by neither an individual's genotype at the mating locus nor the frequency of the male-sterility allele in the population. Inbreeding depression and the male-sterility locus were found to evolve in tandem and to develop associations that strongly influence the fitness of a recessive male-sterility allele. These associations can result in underdominance at the male-sterility locus at the time of introduction of the male-sterility allele. By contrast, a dominant male-sterility allele evolves in exactly the manner predicted by phenotype-based models. No under- or overdominance can occur because only two genotypes are possible in the population: homozygous hermaphrodites and heterozygous females.

Schultz (1999) did not address cases of intermediate dominance, in which male sterility and its pleiotropic

effects are partially expressed in the heterozygote. Elucidation of such cases might contribute to an understanding of sexual dimorphism in the many gynodioecious species in which male sterility is only partially expressed in some genotypes (see Discussion). The rarity of polymorphic species with partial male sterility compared with those with partial female sterility has been frequently cited as support for the outcrossing hypothesis for the evolution of sexual dimorphism in plants (e.g. Lloyd, 1982). Although the resource-compensation hypothesis makes no clear predictions about the relative fitness of the two kinds of inconstancy, the outcrossing hypothesis predicts that partial male sterility should be rare because only slight pollen fertility can lead to self-fertilization.

However, this argument is implicitly based on a phenotypic model of plant-gender evolution; a genetic model is likely to make different predictions. For example, if heterozygotes are partially male sterile, they might have a lower rate of self-fertilization (e.g. owing to smaller stamens or lower loads of self-pollen on geitonogamous pollinators). In the presence of inbreeding depression, these heterozygotes might then experience a higher seed success owing to higher-quality seed progeny. If the increase in seed success outweighs the decrease in male fertility, heterozygote superiority could cause the spread of females even when phenotypic models predict no spread. Thus, even though partial male steriles experience self-fertilization, their lower selfing rate can promote the spread of the male-sterility allele if they are heterozygous at the sterility locus.

Previous theoretical studies by Ross and colleagues (Ross & Weir, 1976; Ross, 1978, 1980; Gregorius *et al.*, 1982) have explored the evolutionary impacts of underdominance or overdominance in the evolution of male and female sterility, but these studies were not designed to incorporate inbreeding depression as either a parametric constant or a dynamic property of multilocus mutation and partial selfing. Here, I report the results of a theoretical population-genetic model of the evolution of gynodioecy under partial dominance in which inbreeding depression is explicitly included as a fixed parameter or varying outcome of recessive mutation. In this study, I asked two questions. First, under partial recessivity of male sterility, what are the conditions for the spread of females and what are their equilibrium frequencies? Second, how are these conditions affected by dynamically evolving inbreeding depression?

2. Methods

(i) *Phenotypic model*

The phenotypic model is the classic analytical model of nuclear gynodioecy (Lewis, 1941; Lloyd, 1975;

Charlesworth & Charlesworth, 1978). This model makes the simplifying assumption of no variability in fitness within a sex morph, which allows analytical derivation of equilibrium frequencies by equating the number of successful gametes of females and hermaphrodites. These assumptions yield the oft-stated result that nuclear gynodioecy requires that ovule fertility of females is at least double the ovule fertility of hermaphrodites. Under competing selfing, the equilibrium frequency of females is given by

$$\hat{P}_{mm} = \frac{F_3 - 2(1 + r\delta)}{2(F_3 - 1 + r\delta)}, \tag{1}$$

where F_3 is the ratio of number of successful ovules of females to hermaphrodites, r is the (competing) selfing rate of hermaphrodites and δ is the population inbreeding depression. Females will spread if their equilibrium frequency is greater than zero (i.e. $F_3 > 2 - 2r\delta$). If the product $r\delta$ (the 'outcrossing advantage' of females; Schultz, 1999) exceeds 0·5 then females spread even in the absence of an allocation advantage (i.e. even if $F_3 = 1$).

(ii) *Genotypic fixed inbreeding depression model*

This is an extension of the models of Gregorius *et al.* (1982) to include a fixed inbreeding depression $\delta = 1 - w_s \div w_o$, the proportional reduction of selfed fitness (w_s) relative to outcrossed fitness (w_o). In these models, inbreeding depression is assumed to affect zygote-to-adult viability only. The model comprises a set of three standard recursion equations for each of the three sterility genotypes – male fertile (MM), male sterile (mm) and partially male sterile (Mm) – incorporating the rate of competing selfing in hermaphrodites (R_G), male and female fertility (M_G and F_G), and viability losses owing to selfing. The subscript G refers to genotype ($1 = MM$, $2 = Mm$, $3 = mm$). Inbreeding depression is assumed to be a single, fixed parameter with no differences between sterility genotypes and no evolutionary response to a changing selfing rate. Self-fertilization is assumed to have no effect on pollen fertility (i.e. pollen discounting is assumed to be zero). Because this model allows differences between genotypes within a sex morph, the number of successful gametes is not equivalent among the three genotypes at equilibrium, and the equilibrium frequencies cannot be derived from this method (Gregorius *et al.*, 1982). In this study, they were determined by iteration (see Appendix).

(iii) *Dynamic inbreeding depression model*

This model, an extension of the combinatorial model of Kondrashov (1985), combines the above genotypic recursion equations with an explicit analysis of the effects of mutation and selection on the distribution of

number of deleterious mutations in each genotype each generation. Thus, the mating polymorphism and mutational inbreeding depression evolve simultaneously and in response to each other in all three genotypes explicitly. Each genotype is assigned a Poisson probability distribution of new mutations at the zygote stage, with mean equal to the genomic mutation rate U, and relative frequencies of the genotypes at the adult stage are adjusted according to their relative viabilities. Viability at a homozygous locus is $1-s$, where s is the unconditional selection coefficient against the mutation. Viability at a heterozygous locus is $1-hs$, where h is the dominance coefficient. The mean viability of a genotype is the weighted average of $(1-s)^i (1-hs)^j$, where i and j are the number of homozygous and heterozygous mutations, and weights are the joint probabilities of i and j within the genotype. The fertilities M_G and F_G represent hard selection on the mating contribution of each genotype, which is proportional to M_G (male) and F_G (female). Genetic selection is also assumed to be hard selection, because the future mating contribution of a zygote is reduced in proportion to its genetic mortality rate, regardless of its mating genotype.

Mating alters the probability distributions of the number of mutations in both the heterozygous and the homozygous states in the three mating genotypes. The probability distribution of the number of mutations carried by a gamete is derived from the binomial probabilities of number of mutations sampled independently from the total carried by the parent (each homozygous mutation is passed on with probability 1, and each heterozygous mutation with probability 0·5). The probability distribution of the number of heterozygous mutations in an outcrossed zygote is derived from all possible gamete pairs, each weighted by its probability. Outcrossed gametes are assumed to have no homozygous mutations. The probability distribution of the number of heterozygous mutations in selfed zygotes is derived similarly from the binomial probabilities of all possible numbers of mutations sampled from the load of heterozygous mutations carried by the parent genotype. The distribution of homozygous mutations in selfed zygotes is derived from the binomial probabilities of transmission of mutations from the parental genotype (1 and 0·25 for each homozygous and heterozygous mutation).

Mutational parameters used in this model were $U=0·02$ to 1, $s=0·1$ to 0·95 and $h=0·02$ to 0·35. Because the results of this model were substantially different from those of the genotypic fixed inbreeding depression model only for $U>0·5$ and $h<0·1$ (higher mutation rates to highly recessive mutations), the behaviour of this model is reported only for $U=1$, $h=0·02$ and $s=0·95$ or 0·7. Results, however, were qualitatively identical for $s>0·5$, the lower limit of computational speed. Although these parameters

represent a higher mutation rate to highly recessive alleles than supported by studies of bacteria, invertebrates and *Arabidopsis* (Drake *et al.*, 1998; Lynch *et al.*, 1999), it is indirectly supported by studies of long-lived trees with high rates of mitotic mutation (Klekowski, 1988; Klekowski & Godfrey, 1989; Klekowski, 1998), and allows a coexistence of high inbreeding depression and substantial selfing rate, as observed in several gynodioecious species and trees regardless of mating system. A coexistence of high selfing rate and inbreeding depression is a central requirement for the evolution of gynodioecy owing to a female outcrossing advantage (Schultz, 1999), and for the evolution of self-incompatibility in plants in classical models (Charlesworth & Charlesworth, 1979). Alternative mechanisms of inbreeding depression (e.g. overdominance or stabilizing selection on a quantitative trait) were not considered in this model.

3. Results

(i) *Conditions for female spread, fixed inbreeding depression model*

The fixed inbreeding depression model predicts the spread of females when the number of successful gametes of the heterozygote exceeds those of the homozygote *MM* (see Appendix). Setting the expression for the heterozygote equal to that of the homozygous hermaphrodite yields $C=1$, where

$$C = \frac{R_2 F_2 (2\delta - 1)}{M_2(1 - R_1) - 2 + 2R_1\delta + F_2}. \tag{2}$$

The inequality implies that females spread when $C>1$ and $\delta<0·5$, or when $C<1$ and $\delta>0·5$. This equation gave results similar to the dynamic inbreeding depression model only when $R_2<R_1$ or when inbreeding depression was low ($\delta<$ about 0·2).

(ii) *Partial male sterility, no reduction in selfing rate*

If partial male sterility in the heterozygote was uncompensated then the male-sterility locus was underdominant and females failed to spread without an increase in fertility well above their threshold for spread according to the phenotypic model, according to both the fixed and dynamic inbreeding depression models (Table 1).

The threshold for female spread could be greatly elevated as a result of differential purging of deleterious mutations in the three mating genotypes. Because the reduction in selfing rate was expressed only in completely male-sterile individuals, hermaphrodites remained only partially outcrossing, which shielded deleterious mutations in a subset of progeny. Heterozygous hermaphrodites on average contained more

Table 1. *Minimum female fertility for the spread of females under partial male sterility* $(M_2 < 1)$ *in the heterozygote. In these models,* $R_1 = R_2 = 0.5$ *and* $F_2 = 1.0$. *In the dynamic model,* $U = 1$ *and* $h = 0.02$. *If linear fertility compensation for male sterility was allowed in the heterozygote, the minimum* F_3 *for female spread was near 2.0 for both the dynamic and fixed inbreeding depression models*

| | Minimum F_3 for female spread | | |
| | Dynamic δ | Fixed $\delta = 1$ | |
M_2	$s = 0.95$	$s = 0.7$	
1·0	7·4	6·7	1·1
0·9	19	23	2·3
0·7	30	40	4·1
0·5	34	49	6·3
0·3	37	55	9·9
0·1	39	59	21·5

mutations and thus produced progeny of lower fitness as detailed in a previous study (Schultz, 1999). Thus underdominance was enhanced by differential purging. As a result, the threshold ovule fertility for female spread could be 20–50 times that of the hermaphrodites, an order of magnitude higher than the threshold according to the phenotypic model, which does not take into account variation in male fertility among hermaphrodites.

If partial male sterility was compensated by a linear increase in female fertility then both sources of underdominance were eliminated and females spread in accordance to the phenotypic model (result not shown).

(iii) Partial male sterility, reduction in selfing rate

If partial male sterility in the heterozygote was compensated by a reduction in selfing rate, then a high inbreeding depression (> 0.5) resulted in overdominance at the sterility locus and the spread of females, even if females had no fitness advantage according to the phenotypic model (Table 2, Fig. 1). Indeed, females could spread under these conditions even if the female phenotype had zero fertility, simply due to overdominance (Table 2, last row). Females spread as a result of an outcrossing benefit to the heterozygote, even in a population with a low mean selfing rate. Equilibrium female frequencies were positively related to the inbreeding depression, the reduction in selfing rate and fertilities of both sex functions in the heterozygote and the female. Overdominance occurred typically at both introduction of the male-sterility allele and equilibrium (Table 2). If the heterozygote was completely male fertile then females spread if the heterozygote selfing rate was below that of the

homozygous hermaphrodite, in the presence of inbreeding depression > 0.5 (Fig. 1). If the heterozygote was reduced in male fertility, a lower selfing rate required a higher inbreeding depression for the spread of females (Fig. 1). The results for a reduction in the heterozygote selfing rate of more than 0.1 were virtually identical in the fixed and dynamic inbreeding depression models (comparison not shown).

(iv) Partial male sterility, increase in selfing rate

If partial male sterility was accompanied by an increase in selfing rate then a high inbreeding depression (> 0.5) resulted in underdominance at the mating locus that could prevent the spread of females in the absence of very large increases in female fertility (Fig. 2). This underdominance, however, could be overcome by differential purging at the mating locus (Fig. 2). For very high selfing rates in the heterozygote, purging of mutations in their progeny produced females with substantially fewer mutations than hermaphrodites. This allowed the spread of the male-sterility allele even if females and hermaphrodites had nearly equivalent female fertilities.

Conversely, if population inbreeding depression was low (< 0.5) then an increase in the selfing rate in the heterozygote promoted the spread of females (Eqn 2, Fig. 1). Overdominance in this case was caused by the automatic transmission advantage of selfing (Fisher, 1941). For example, in the limiting case of no inbreeding depression, for each successful outcrossed ovule of a completely outcrossing hermaphrodite, a completely selfing hermaphrodite produced a successful selfed pollen grain and a successful ovule. This advantage in general depends on the level of pollen discounting, assumed to be zero in this study.

4. Discussion

The classical analytic theory for the maintenance of nuclear gynodioecy is based on frequency-dependent selection through the male function of hermaphrodites (Lloyd, 1975). In this phenotypic theory, females spread only if their female fertility is more than double that of hermaphrodites. The equilibrium sex ratio is maintained by a positive correlation between female frequency and hermaphrodite pollen success.

An additional or alternative mechanism for the initial spread of females and the maintenance of gynodioecy is fitness overdominance at the male-sterility locus (Ross & Weir, 1976; Ross, 1978, 1980; Gregorius *et al.*, 1982). This study demonstrates a simple mechanism whereby overdominance in fitness can be produced in any gynodioecious species in which hermaphrodites are self-compatible and experience inbreeding depression. If male sterility is a single-locus, nuclear trait and heterozygotes experience a partial

Table 2. *Equilibrium female frequencies and gamete success, under partial male-sterility and reduced selfing rate in the heterozygote. In all cases,* $R_1 = 0.7$ *and* $M_2 = 0.8$. *Predicted* mm *frequency is based on the phenotypic model for which the homozygote and heterozygote hermaphrodites are pooled to calculate an overall mean hermaphrodite selfing rate and female fertility. Successful gametes at introduction and equilibrium are expressed relative to the homozygous hermaphrodite (which is set to 1)*

Parameters				Equilibrium frequencies			Predicted mm frequency	Mean R of hermaphrodites	Relative female fertility of females	Successful gametes at introduction		Successful gametes at equilibrium	
F_2	F_3	R_2	δ	MM	Mm	mm				Mm	mm	Mm	mm
1·00	1·0	0·0	0·9	0·405	0·479	0·116	0·00	0·32	1·00	1·67	1·35	1·28	0·74
1·00	1·0	0·1	0·9	0·406	0·478	0·116	0·00	0·38	1·00	1·57	1·35	1·24	0·78
1·00	1·0	0·3	0·9	0·410	0·474	0·116	0·00	0·49	1·00	1·35	1·35	1·15	0·86
1·00	1·0	0·5	0·9	0·418	0·467	0·115	0·07	0·59	1·00	1·13	1·35	1·04	0·96
1·00	1·0	0·7	0·9	1·000	0·000	0·000	0·21	0·70	1·00	0·92	1·35	–	–
1·00	1·0	0·0	0·8	0·490	0·430	0·090	0·00	0·37	1·00	1·41	1·13	1·19	0·73
1·00	1·0	0·1	0·8	0·500	0·420	0·080	0·00	0·42	1·00	1·34	1·13	1·16	0·76
1·00	1·0	0·3	0·8	0·520	0·400	0·080	0·00	0·53	1·00	1·20	1·13	1·10	0·84
1·00	1·0	0·5	0·8	0·600	0·340	0·060	0·00	0·63	1·00	1·07	1·13	1·02	0·95
1·00	1·0	0·7	0·8	1·000	0·000	0·000	0·11	0·70	1·00	0·93	1·13	–	–
1·04	1·2	0·0	0·8	0·434	0·461	0·106	0·00	0·34	1·18	1·45	1·36	1·18	0·80
1·04	1·2	0·1	0·8	0·434	0·459	0·107	0·00	0·39	1·18	1·38	1·36	1·16	0·83
1·04	1·2	0·3	0·8	0·433	0·457	0·110	0·00	0·49	1·18	1·24	1·36	1·09	0·91
1·04	1·2	0·5	0·8	0·426	0·457	0·117	0·10	0·60	1·18	1·10	1·36	1·02	0·98
1·04	1·2	0·7	0·8	0·391	0·472	0·137	0·20	0·70	1·17	0·96	1·36	0·92	1·05
1·00	0·0	0·0	1·0	0·507	0·420	0·073	0·00	0·38	0·00	2·07	0·00	1·58	0·00

loss of male fertility owing to reduction of pollen production or stamen size then heterozygotes might also experience a reduction in selfing rate. A lower selfing rate could be caused by a lower load of self-pollen carried by animal pollinators during interflower visits (Schoen *et al.*, 1996) or greater anther–stigma separation in time or space (Holtsford & Ellstrand, 1992; Karron *et al.*, 1997; Motten & Stone, 2000).

Whatever the ecological cause of lower selfing in the heterozygote, in the presence of an inbreeding depression of more than 0·5, it can overcompensate for a moderate loss in male fertility. If so, the number of successful gametes produced by the heterozygote exceeds those of the homozygous hermaphrodite and perhaps those of females also. The loss in successful outcross pollen in the heterozygote is then compensated by a gain in outcrossed ovules. This compensation maintains a dimorphism of females and hermaphrodites regardless of the presence or absence of any phenotypic advantage of male sterility, and even in a population with a low selfing rate. Females might be maintained at frequencies well above those predicted by the phenotypic model. This discrepancy occurs because some of the advantages of the male sterility allele are expressed by the heterozygous hermaphrodite, which therefore increases the mean 'phenotypic' performance of hermaphrodites relative to females, leading to artificially lower equilibrium frequencies predicted for females.

However, if partially male-sterile heterozygotes are not compensated by a reduction in selfing rate then the sterility locus becomes underdominant and females can fail to spread without a sizable fertility advantage. Underdominance arises from both the reduced male fertility and the greater number of deleterious mutations carried by the heterozygote, which selfs at the same rate as the homozygous hermaphrodite but is produced more often by outcrossing. By contrast, if the heterozygote has an increased selfing rate then its progeny become more purged of mutations, reducing the underdominance and the threshold fertility advantage necessary for the spread of females. Although an increase in selfing rate seems to be less likely than a decrease in a partial male sterile, an increase might occur if a reduction in stamen size also reduces the anther–stigma separation within a flower.

Because the ranges of values of inbreeding depression and selfing rate that allow the selective maintenance of females owing to an 'outcrossing advantage' are wider in these models than allowed by the phenotypic model, these results can explain the maintenance of nuclear gynodioecy for cases in which the phenotypic model predicts an absence of females at equilibrium. However, these models do not alter the theoretical asymmetry between gynodioecy and androdioecy (Lloyd, 1975, 1982), because partial female sterility is unlikely to result in a change in an individual's selfing rate.

The empirical relevance of these results hinges on the occurrence of partial male sterility in natural populations of gynodioecious species. Although no study has apparently been published on variation in selfing
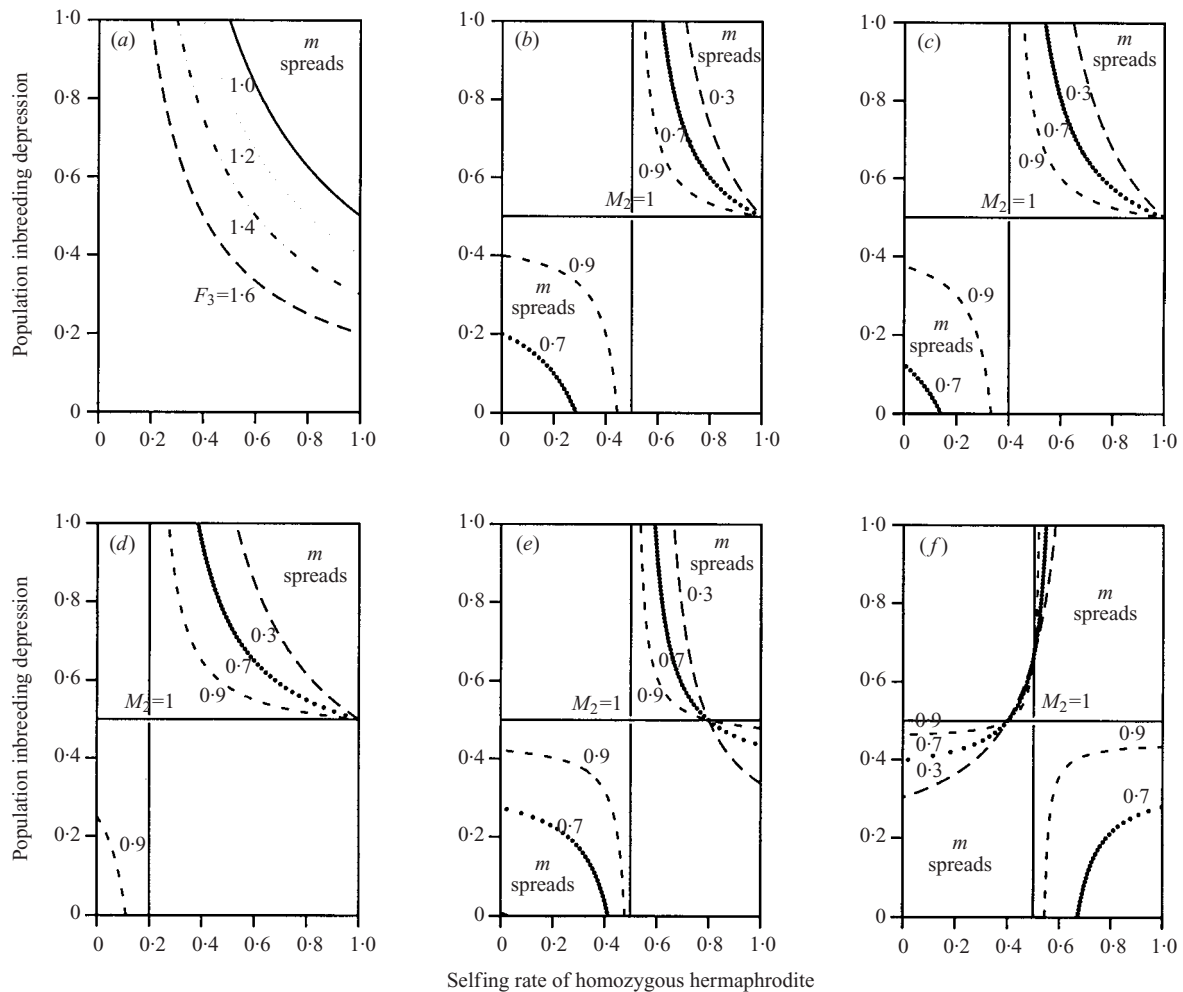
Fig. 1. Parameter values for the spread of a male-sterility allele owing to heterozygote superiority. Regions indicated are the joint conditional values of population inbreeding depression δ and selfing rate of homozygous hermaphrodite R_1 beyond which females spread in the population. (*a*) Phenotypic model (Eqn 1). (*b–f*) Genetic model with the male fertility of heterozygote M_2 as indicated, and conditions for female spread owing to overdominance at the sterility locus caused by differences among hermaphrodites in selfing rate and male fertility (Eqn 2). In all cases shown, females spread if selfing rate and inbreeding depression are either both high or both low. (*b*) Selfing rate of heterozygote $=0.5$, no female or heterozygote allocational advantage. (*c*) Selfing rate of heterozygote $=0.4$, no allocational advantage. (*d*) Selfing rate of heterozygote $=0.2$, no allocational advantage. (*e*) Selfing rate of heterozygote $=0.5$, female allocational advantage $=0.2$ and heterozygote allocational advantage proportional to loss of male fertility. (*f*) Selfing rate of heterozygote $=0.5$, female allocational advantage $=0.6$ and heterozygote allocational advantage proportional to loss of male fertility.

rates within self-compatible hermaphrodites of gyno-dioecious species, many examples are known of partial reduction of male fertility in such species. In *Stellaria longipes*, some cultivated hermaphrodites produce fewer than ten well-developed stamens (Philipp, 1980). In *Salvia nemorosa*, some hermaphrodites have reduced pollen fertility, especially late in the flowering season (Linnert, 1958). In several species of *Geranium*, hermaphrodites consist of fully male-fertile and partially male-fertile individuals (Putrament, 1962; Agren & Willson, 1991). In *Geranium maculatum*, partial male steriles occurred in frequencies from 0·2 to 0·27 in several study populations. Pollen fertility in hermaphrodites followed an exponential distribution, with most individuals having high fertility (60–90 %) and very few individuals having low fertility (positive

but near zero). Individuals varied slightly annually in their pollen fertility. Intermediate male sterility has been documented in several cytotypes of gyno-dioecious *Plantago* species (Vandamme & Vandelden, 1982; Vandamme, 1984; Koelewijn & Van Damme, 1995), contributing to the suggestion by some authors that overdominance might contribute to gynodioecy in *Plantago lanceolata* (Vandamme, 1984). In gyno-dioecious *Saxifraga granulata*, frequencies of partial male steriles averaged close to 4 % in three populations (Stevens & Richards, 1985). In *Thymus vulgaris*, partial male steriles occur with corolla size also intermediate between females and full hermaphrodites (Assouad & Valdeyron, 1975; Thompson *et al.*, 2002). In *Glechoma hederacea*, 10 % of individuals at 31 sites were partially male sterile, with both female and hermaphrodite
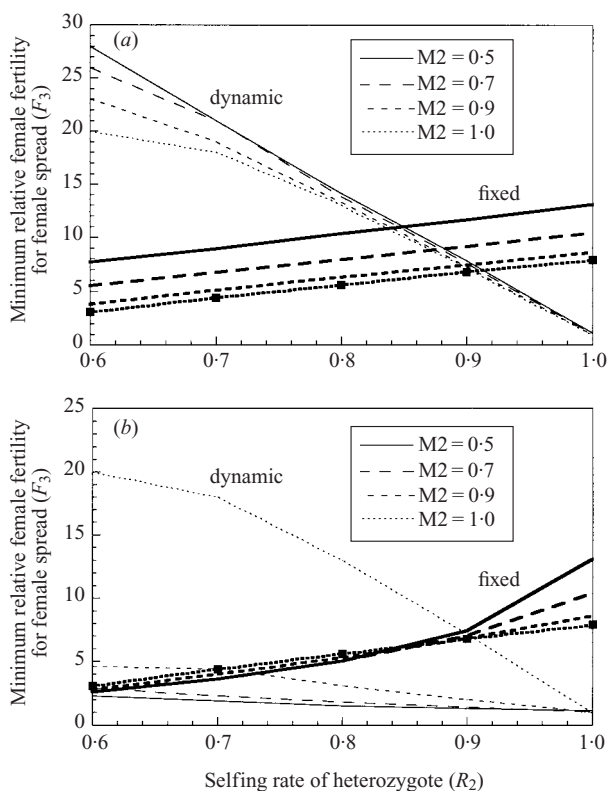
Fig. 2. Conditions for female spread under increased selfing rate in partially male-sterile heterozygotes. In all cases, $R_1 = 0.5$. In the dynamic inbreeding depression model, $U = 1$, $s = 0.95$ and $h = 0.02$. Inbreeding depression was set to 1 in the fixed inbreeding depression model. (*a*) No compensatory increase in F_2. (*b*) Linear compensatory increase in F_2 in response to reduced M_2.

flowers (Widen, 1992; Widen & Widen, 1999); partial male steriles exhibited a lower siring success. In *Silene acaulis* on Pennsylvania Mountain in Colorado, the frequency of individuals producing both perfect and pistillate flowers was 35 % in the study populations (Shykoff, 1988). In all these species, variation in male function is visually obvious in the morphology of the androecium. Purely physiological variation in pollen success is more difficult to demonstrate and might occur in many other species in which no morphological differences are evident.

The full mechanisms of genetic control of male sterility are not known for most of these species and, in some, variation in the expression of partial male sterility might be entirely environmental (as has been shown in populations of *Silene acaulis* (Delph, unpublished) and perhaps *Glechoma hederacea*). Nevertheless, partial male sterility, regardless of its (environmentally mediated) magnitude, might occur only in individuals carrying alleles that also cause full male sterility. If the alleles responsible for complete male sterility are also responsible for partial male sterility, regardless of how many loci are involved, then their overall fitness and rate of spread will depend on their consequences in both types of individuals. The

consequences to both male and female fertility will need to be evaluated within a full genetic model to determine the selective causes of maintenance of the sexual dimorphism in these species. However, it is not currently known whether the current results about differential purging would be relevant to cases in which male fertility is a quantitative trait, because theoretical models have yet to analyse this phenomenon in populations with quantitative genetic variation in the rate of self-fertilization.

In principle, the results of these models could apply equally within the male-sterility locus in nuclear gynodioecy and within the fertility-restorer locus in nucleocytoplasmic gynodioecy. Partial fertility restoration in male-sterility cytotypes has been observed in *Plantago* (Vandamme & Vandelden, 1982), maize (Tracy *et al.*, 1991; Wise *et al.*, 1999), quinoa (Ward & Johnson, 1993), sorghum (Senthil *et al.*, 1994) and lesquerella (Dierig *et al.*, 2001). In maize, late-breaking partial fertility restoration is well known and economically useful (Tracy *et al.*, 1991). In some cases, the degree of restoration appears to be related to the concentration of nuclear-coded enzymes similar to mitochondrial enzymes involved in energy catabolism, such as aldehyde dehydrogenases (Wise *et al.*, 1999). Partial restoration of cms cytotypes in nature would, in theory, create the same kinds of fitness differentials among restorer genotypes within a particular sterile cytotype as described here within a nuclear male-sterility locus. These results are thus of heuristic value in understanding the evolutionary dynamics of cms in these species. However, additional phenomena not addressed here, such as costs of restoration and the presence of multiple cytotypes, will undoubtedly complicate these dynamics.

In conclusion, this study found that fitness overdominance or underdominance at a mating locus can be caused simply by genotypic differences in the selfing rate in a self-compatible species. No phenetic overdominance or underdominance in any trait is necessary to produce these effects. These phenomena are shown to influence crucially the evolution of sexual function in a plant population. Partial nuclear male sterility in itself causes underdominance that inhibits the spread of the male-sterility allele in populations of hermaphrodites. However, if compensated by a change in the rate of self-fertilization, partial male steriles may outperform male-fertile individuals and promote the spread of male sterility, establishing a gynodioecious mating system in the absence of a phenotypic twofold superiority of females at introduction. Gynodioecy can be established in this way if the heterozygote has a lower selfing rate (and inbreeding depression is high) or a lower selfing rate (if inbreeding depression is low or if differential purging occurs). Resulting equilibrium female frequencies can greatly exceed those predicted purely from a phenotypic comparison of female and

hermaphrodite performance. These findings indicate that a complete accounting of the selective causes of spread of an allele causing outcrossing might be difficult in natural populations even under single-locus nuclear control of the trait. Comparisons of both male and female fitness components in all three genotypes might be necessary, especially in populations exhibiting morphological evidence of partial male sterility.

Appendix

Under the fixed inbreeding depression model, the numbers of successful gametes produced by genotypes MM, Mm and mm are expressed as:

$$w_{MM} = \frac{M_1[(1-Mm-mm)F_1(1-R_1)+Mm\,F_2(1-R_2)+F_3\,mm]}{M_1(1-Mm-mm)+M_2\,Mm} + (1-R_1\delta)F_1+R_1F_1(1-\delta);$$

$$w_{Mm} = \frac{M_2[(1-Mm-mm)F_1(1-R_1)+Mm\,F_2(1-R_2)+F_3\,mm]}{M_1(1-Mm-mm)+M_2\,Mm} + (1-R_2\delta)F_2+R_2F_2(1-\delta);$$

$$w_{mm} = F_3,$$

where MM, Mm and mm are the frequencies of the three genotypes.

The recursion equations for the three genotypes are as follows:

$$\begin{aligned} MM' = \{ &MM\,F_1[MM\,M_1\div(MM\,M_1+Mm\,M_2)](1-R_1)+MM\,F_1[Mm\,M_2\div(MM\,M_1+Mm\,M_2)](1-R_1)\div2 \\ &+Mm\,F_2[Mm\,M_2\div(MM\,M_1+Mm\,M_2)](1-R_2)\div4+Mm\,F_2[MM\,M_1\div(MM\,M_1+Mm\,M_2)](1-R_2)\div2 \\ &+MM\,F_1R_1(1-\delta)+Mm\,F_2R_2(1-\delta)\div4\}\div K \end{aligned}$$

$$\begin{aligned} Mm' = \{ &MM\,F_1[Mm\,M_2\div(MM\,M_1+Mm\,M_2)](1-R_1)\div2+Mm\,F_2[MM\,M_1\div(MM\,M_1+Mm\,M_2)](1-R_2)\div2 \\ &+Mm\,F_2[Mm\,M_2\div(MM\,M_1+Mm\,M_2)](1-R_2)\div2+mm\,F_3[Mm\,M_2\div(MM\,M_1+Mm\,M_2)]\div2 \\ &+mm\,F_3[MM\,M_1\div(MM\,M_1+Mm\,M_2)]+Mm\,F_2R_2(1-\delta)\div2\}\div K \end{aligned}$$

$$\begin{aligned} mm' = \{ &mm\,F_3[Mm\,M_2\div(MM\,M_1+Mm\,M_2)]\div2+Mm\,F_2[Mm\,M_2\div(MM\,M_1+Mm\,M_2)](1-R_2)\div4 \\ &+Mm\,F_2R_2(1-\delta)\div4\}\div K \end{aligned}$$

$$K=MM\,F_1(1-R_1\delta)+Mm\,F_2(1-R_2\delta)+mm\,F_3$$

where the prime indicates the subsequent generation.

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