Gametophytic Self-Incompatibility Reexamined

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Summary. The conventional hypothesis of gametophytic self-incompatibility in the angiosperms involves one to four multiallelic incompatibility loci and the positive inhibition of incompatible pollen tubes. However, this concept does not accommodate recent experimental data indicating that there may be many loci. An alternative hypothesis which incorporates many loci and complementary pollen-style interactions suggests that there may be no S gene, as previously thought, and that gametophytic self-incompatibility is perhaps merely one aspect of extensive pollen-style interactions.

Self-incompatibility in fertile plants refers to those that fail to set seeds after self-pollination. This phenomenon is widely accepted as being under the control of a single multiallelic gene (*1*), the S gene. With gametophytic self-incompatibility, pollen tubes fail to complete their growth through the style if the S allele contained within the pollen is present in the style. In order to explain self-incompatibility, two mechanisms have been proposed. The first of these, the oppositional mechanism, indicates that the growth of incompatible pollen tubes is actively inhibited by specific molecules (*2*). In contrast, the complementary mechanism implies a passive system, with incompatible pollen tubes failing to activate (or obtain) nutrients, conditions, or stimuli necessary for growth (*3*, *4*).

The oppositional mechanism is widely accepted among pollen biologists (*5*), and a recent review of self-incompatibility (*6*) contained nearly 500 references to oppositional systems but only ten to complementary ones. The widespread acceptance of the oppositional mechanism is based on several observations (*5*, *6*). For example, many pollen tubes will germinate in a minimal medium, or even in moist air (*7*). Failure to germinate in the nutrient-rich stylar fluid might therefore indicate a positive opposition by an incompatible style. Furthermore, the incompatibility reaction develops more rapidly with increasing temperatures, again, perhaps indicating an active inhibition. The incompatibility reaction can be disrupted by endogenous and exogenous factors (*6*), such as aging, ionizing radiation, and high temperatures. Contrary to the requirements of one complementary proposal (*4*), it has been assumed that only a few genes were expressed in the pollen (*6*). Furthermore, specific incompatibility proteins are reported to be present in the pollen (*8*) and style (*9*); the pollen activity part of the incompatibility locus is apparently capable of mutation (*10*), and self-pollination is followed by qualitative and quantitative differences in gene activity (*6*, *11*). Finally, complementary systems seem to be rigorously excluded by the fact that, whenever the stylar genotype is heterozygous for the incompatibility locus, there will always be at least one allele to complement the allele present in the pollen (*5*). Thus it was assumed that pollen tubes cannot fail for lack of complementation. In view of these conclusions, it is only logical that most investigators have accepted the oppositional model. Nevertheless, a reexamination of studies supporting this system suggests that many of these observations could be explained in other ways.

Oppositional Foundations

The fact that pollen germinates in minimal medium seems to lose significance now that it has been learned that pollen tube growth consists of two distinct phases (*12*). The first of these phases is sustained by materials contained within the pollen grains themselves. This autonomy enables pollen to germinate and to produce a short tube either in vitro or in an incompatible style (*13*). The second growth phase, that required to reach the ovules, is dependent on nutrients provided by the style and occurs only in a compatible style. Incompatible pollen tubes, as suggested by the concept of complementation, could thus very well fail, in the second phase, for lack of appropriate stimuli.

This same biphasic pollen tube growth, with inhibition (or failure) of pollen tube growth occurring at the start of phase two, could also explain the temperature dependency of pollen tube inhibition since the shift from phase one to phase two is also dependent on temperature (*12*).

Concerning the ease with which incompatibility can be disrupted, long-term exposure to ionizing irradiation, in *Lycopersicon peruvianum*, could delay floral abscission (*6*), and thus allow slowly growing incompatible pollen tubes to reach the ovules. Subjecting styles to high-temperature treatments, a standard method of overcoming self-incompatibility, may increase the rate of secretion in styles of *Lilium longiflorum* (*14*), and the increased nutrition thus provided could allow otherwise incompatible pollen tubes to reach ovules (*6*). As for the assumption that few enzymes should be required for pollen tube growth, it is now known that large numbers of enzymes are produced, and presumably function, in the pollen of *Lycopersicum* spp. and *Cucurbita* spp. (*15–17*).

Concerning the specific incompatibility proteins found in pollen, de Nettancourt (*6*) indicates a possible flaw in these correlations. Incompatibility genes are almost certainly associated with many closely linked genes, and the difficulty of distinguishing products of self-incompatibility genes and those of closely linked genes is formidable. This concern is heightened by reports that a large number of different substances, including esterases, peroxidases, glycoproteins, and others, are candidates for the incompatibility substance (*6*).

Support for the oppositional concept is provided by the existence of pollen-part mutations. These mutations allow self-pollen to reach ovules and are expressed only in the pollen, but have no effect on the style. In the style, the mutant continues to reject pollen carrying the nonmutant incompatibility type (*18*). However, at least in *Nicotiana* and *Petunia*, mutations to self-compatibility are sometimes associated with extrachromosomal material (*19*) although, in other cases, these

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fragments have not been found (*8*). Extrachromosomal material, whenever it is found, could complement genetic deficiencies (recognized as incompatibility alleles) and thus result in self-compatibility (*4, 6*). In these cases, pollen-part mutants are thus subject to both oppositional and complementary interpretations. Lacking such fragments, other explanations must be sought.

The production of specific proteins after self-pollination (*6, 11*) is strong support for the oppositional interpretation, even though it is not difficult to postulate that the introduction and failure of pollen tubes could certainly induce metabolic changes in stylar tissues. In view of their obvious significance, however, such studies should perhaps be undertaken with additional species.

The final objection to the complementary model—the fact that, whenever the style is heterozygous at the incompatibility locus, at least one stylar locus will complement that carried in the pollen—is discussed below.

Oppositional Shortcomings

The final test of any theory is in how well it explains empirical observations. If it succeeds, ambiguities in its conceptual foundations, such as those listed above, are relatively unimportant. It appears, however, that the classical explanation of self-incompatibility is unable to accommodate several substantive observations. For example, in multifactorial systems of self-incompatibility (*1*), Larsen (*20*) discovered that the strength of the incompatibility reaction varies quantitatively, being stronger when more stylar loci are homozygous. (The style A1A1B2B2 will reject A1B2 pollen more rapidly than would style A1A2B2B3). Such quantitative variation is unexpected since, whenever all pollen incompatibility alleles are contained in the style, the inhibition of pollen tube growth should be complete.

Furthermore, the oppositional interpretation has no provision to explain the recent finding that forced inbreeding of *Lycopersicum peruvianum*, a species that exhibits single-locus gametophytic self-incompatibility, somehow "activates new incompatibility alleles" (*21*). Inbreeding an S1S2 clone, in one example, produced an S2S2 homozygote that rejected not only S2 pollen but also S3 pollen. Backcrossing to the original S1S2 clone sometimes caused the loss of the new specificity. In a later study, moreover, inbreeding produced clones that exhibited up to five different incompatibility specificities (*22*), thus excluding the possibility that all were allelic. This latter result suggested that incompatibility loci were scattered throughout the genome and could be activated and deactivated (*21, 22*). This hypothesis fits the observations but greatly weakens the concept that incompatibility systems are highly conservative.

Other facts left unexplained by the classic interpretation include reports that the incompatibility gene is difficult to map (*6*). This is hardly expected for a single locus that has such an obvious phenotype. Also, whenever natural populations have been surveyed for incompatibility alleles, it is generally found that a high proportion of all plants carry two incompatibility alleles, and each of these is found in no other plant (*6, 23*). Population biologists were quick to realize that this was a highly unlikely situation. The death of any individual would probably reduce the pool of incompatibility alleles by two, and the growth of the population should be followed by the appearance of two new alleles for each additional plant. This prompted speculation that, in order to maintain the allelic pool reported, the incompatibility locus must exhibit an extremely high rate of mutation (*24*). However, when this possibility was tested, it became clear that mutations at the incompatibility locus are extremely rare (*10*). Since then, the issue has remained unresolved.

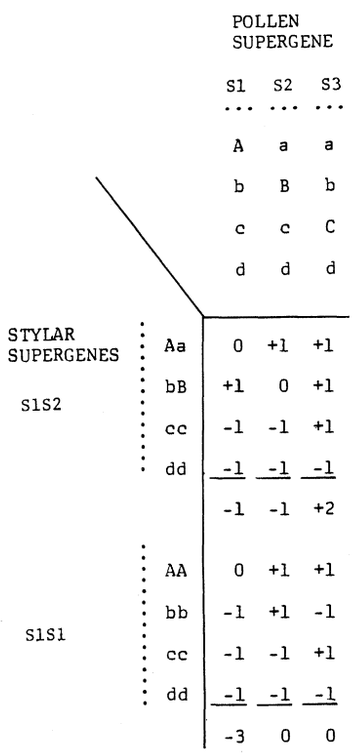

Fig. 1. Possible interactions between pollen and stylar genotypes. The pollen supergene either does or does not encounter a matching supergene within the style. The style may be either heterozygous or homozygous for that supergene.

A final observation that is not explainable within the oppositional interpretation is the report that androgenic haploids of self-incompatible *Lolium perenne*, after chromosome doubling, show a high frequency of self-compatible individuals (*25*). Because the S alleles are known to function perfectly well in the homozygous condition (*6*), the loss of self-incompatibility in dihaploids is unexpected.

Since the oppositional interpretation leaves several observations unexplained, the ambiguities in its foundations become more substantive, and thus we have attempted to suggest an alternative concept, presented below.

The Heterosis Model

This alternative was designed to incorporate the fact that five observations, which have not otherwise been explained, seem to have one aspect in common; each of them suggests, as will be shown below, the expression of numerous deleterious recessives, made homozygous by inbreeding. We have thus considered that incompatibility could be a prezygotic expression of genetic load mediated through extensive (*26, 27*), but as yet largely unexplored, pollen-style interactions.

The model, called the heterosis model of self-incompatibility, is based on the assumption that if pollen and style carry dissimilar alleles, there will be heterotic interactions between them and pollen tube growth rate will increase. If the style is homozygous for a deleterious recessive allele and the pollen carries the same allele, pollen tube growth rate will be reduced. The actual growth rate of the pollen tube will be the sum of all pollen-style interactions. Somewhat surprisingly, the result seems to accommodate the data used in arriving at it, and also many of the inexplicable aspects of gametophytic self-incompatibility listed above.

An introduction to this interpretation is facilitated if it is assumed, temporarily, that incompatibility loci (S, Z, and so forth) are actually supergenes, that is, groups of closely linked loci. It is necessary to assume also that each supergene contains one dominant and several deleterious recessive genes. Thus the incompatibility supergene S1 could be represented as Abcd and S2 as aBcd. If we further assume that pollen-style interactions are determined by the genotypes of

the two participants, then it is possible to construct a quantitative expression both for these interactions and also for pollen tube growth rates. For example, let us assume that, if pollen grain carries a recessive allele (*a*) and style is either heterozygous or homozygous for the corresponding dominant *A*, there will be a heterotic interaction between them. This heterosis can be expressed by assigning to such an interaction an arbitrary value of +1 (Fig. 1, pollen supergene S2). The same heterotic interaction would result when the style is homozygous recessive *aa* and the pollen carries the dominant allele *A*. If the style is homozygous recessive for the locus *c*, for example, and the pollen carries the same recessive allele, *c*, the deleterious qualities of the recessive allele are expressed, and pollen tube growth rate is reduced. We express this reduction by assigning, to such a combination, a value of −1. If both pollen and style carry the dominant allele *A*, there is neither a heterotic nor a detrimental effect on pollen tube growth rate, and we assign to this effect a value of zero (*28*).

The heart of the heterosis model is our assumption that the pollen tube growth rate will be proportional to the sum of all such pollen-style interactions. Incompatible pollinations are due, not to specific inhibitory molecules, but rather to pollen tube growth being too slow to allow fertilization before floral abscission.

If there are four loci within each incompatibility supergene, there will be four separate interactions as pollen tubes penetrate the style (Fig. 1). With S1 pollen in an S1S2 style, the sum of these four interactions is −1. Alternatively, if the incompatibility supergene carried by the pollen, for example, S3, does not match either supergene carried by the style, then the sum total of interactions is +2. If a style is homozygous for an incompatibility supergene, a matching pollen genotype scores −3 while a "nonmatch" scores zero.

The relative outcome of interactions is unchanged by increasing the numbers of loci in each supergene, although point totals are more negative (Fig. 2). Furthermore, the heterosis interpretation allows the number of incompatibility supergene loci to be increased to two, thus mimicking the S and Z incompatibility loci of the Gramineae (*29*), to four as is the case with *Beta vulgaris* (*20*) and *Ranunculus acris* (*30*), or to any number.

A four-supergene system (Fig. 3) explains Larsen's report (*20*) that self-incompatibility becomes weaker when large numbers of incompatibility loci are heterozygous. For each of the combinations underlined by dots, two pollen supergenes are matched by supergenes within the stylar complement and two are not. Because increased stylar heterozygosity raises the number of heterotic pollen-style interactions, the values for the underlined combinations are directly proportional to levels of stylar heterozygosity. If, for example, a score of −7 or higher allows fertilization to occur before floral abscission, sufficient heterozygosity should allow, as reported (*19*), self-compatibility (Fig. 3).

With the complementation interpretation of gametophytic self-incompatibility, inbreeding can indeed activate new "incompatibility alleles" (*6, 21, 22*). For example, in Fig. 4, let us assume that a score of −8 or lower results in failure of the pollen tubes to reach ovules before abscission and that we have a population within which the incompatibility supergenes P, Q, and R are monomorphic (that is, exhibit only a single configura-

Pollen Genotypes / Stylar Genotypes	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10
	A	a	a	a	a	a	a	a	a	a
	b	B	b	b	b	b	b	b	b	b
	c	c	C	c	c	c	c	c	c	c
	d	d	d	D	d	d	d	d	d	d
	e	e	e	e	E	e	e	e	e	e
	f	f	f	f	f	F	f	f	f	f
	g	g	g	g	g	g	G	g	g	g
	h	h	h	h	h	h	h	H	h	h
	i	i	i	i	i	i	i	i	I	i
	j	j	j	j	j	j	j	j	j	J

Stylar Genotypes	S1	S2
A	a	0 + + + + + + + + +
b	B	+ 0 + + + + + + + +
c	c	- - + - - - - - - -
d	d	- - - + - - - - - -
e	e	- - - - + - - - - -
f	f	- - - - - + - - - -
g	g	- - - - - - + - - -
h	h	- - - - - - - + - -
i	i	- - - - - - - - + -
j	j	- - - - - - - - - +
		-7 -7 -4 -4 -4 -4 -4 -4 -4 -4

Fig. 2 (left). Model of an incompatibility supergene exhibiting ten allelic variants. A score of −4 will lead to seed set while −7 will not.

Pollen Genotypes / Stylar Genotypes	P1 Q1 R1 S1	P1 Q1 R1 S2	P1 Q1 R2 S2	P1 Q2 R2 S2	P2 Q2 R2 S2	P2 Q2 R2 S3	P2 Q2 R3 S3	P2 Q3 R3 S3	P3 Q3 R3 S3
P1P1Q1Q1R1R1S1S1	-12	-9	-6	-3	0	0	0	0	0
P1P1Q1Q1R1R1S1S2	-10	-10	-7	-4	-1	+2	+2	+2	+2
P1P1Q1Q1R1R2S1S2	-8	-8	-8	-5	-2	+1	+4	+4	+4
P1P1Q1Q2R1R2S1S2	-6	-6	-6	-6	-3	0	+3	+6	+6
P1P2Q1Q2R1R2S1S2	-4	-4	-4	-4	-4	-1	+2	+5	+8

Fig. 3 (right). Relative pollen tube growth rates among representative crosses in a population containing four incompatibility supergenes—P, Q, R, and S. Each supergene contains four loci, thus producing four pollen-style interactions. Each value shown is therefore the sum of 16 separate interactions. (See Fig. 1 for these values.) Combinations underlined by dots are those in which two supergenes of pollen and style match and two do not.

tion). A fourth supergene, S, is rich in allelic variants, (S1, S2, . . . S*n*) (*31*). Forced selfing of a plant heterozygous at the S supergene, for example, S1S2, in such a population (see stylar genotype 2 in Fig. 4) would produce some progeny that are homozygous also at the S supergene (see stylar genotype 1). These S homozygotes would reject all pollen from the population, irrespective of which S allele the pollen carried. This would be interpreted as the activation of several new incompatibility loci (*22*). Backcrossing to the original parent (stylar genotype 2) would, in half the cases, restore heterozygosity to the S supergene and the new specificities would "revert" to the original specificities (S1S2), rejecting only pollen that carried the S1 or the S2 variant.

Our model also gives an indication of why the incompatibility locus has been difficult to map. If investigators have worked under the assumption that there was only a single incompatibility locus, when in reality there were many, their analyses would certainly have been extremely difficult to complete.

The concept of many incompatibility supergenes, instead of the one implied by the conventional model, may also explain the unexpectedly great number of incompatibility alleles found to exist in natural populations. This point is better understood when we consider the method by which the incompatibility alleles in a natural population have been surveyed. Each plant tested (that is, plant X1, X2, . . . X*n*) is crossed to a standard line that is homozygous for one incompatibility allele, for example, S1S1. The progeny from different crosses (S1S3 and S1S4 from S1S1 × X1, S1S5 and S1S6 from S1S1 × X2, and so on) are then interbred, and if they are interfertile their original parents, X1, X2, . . . X*n*, must have carried different incompatibility alleles. Such studies generally show that nearly all progeny are interfertile, and this is logically interpreted as indicating that most plants in natural populations carry incompatibility alleles that are present only in that plant. The heterosis interpretation, however, suggests that, in crossing X1, X2, . . . X*n* to the S1S1 standard, enough heterogeneity is generated between the progeny and enough heterozygosity within them so that nearly all pollen-style combinations are highly complementary. The progeny are, accordingly, all interfertile. This interpretation thus suggests that, in natural populations, incompatibility is not under the control of a single locus, which exists as an inexplicably large number of low-frequency alleles. Instead, it may involve many loci, each comprising a small number of alleles.

Stylar Genotypes \ Pollen Genotypes	P1 Q1 R1 S1	P1 Q1 R1 S2	P1 Q1 R1 S3	P1 Q1 R1 S4
P1P1Q1Q1R1R1S1S1	−12	−9	−9	−9
P1P1Q1Q1R1R1S1S2	−10	−10	−7	−7
P1P1Q1Q1R1R1S1S3	−10	−7	−10	−7
P1P1Q1Q1R1R1S1S4	−10	−7	−7	−10

Fig. 4. Possible mechanism for increasing the number of pollen types rejected. If a value of −8 or less confers incompatibility on a combination, then selfing stylar genotype 2 (to obtain plants of stylar genotype 1) will result in some plants (genotype 1) that reject pollen genotypes 1 to 4 even though pollen genotypes 2 to 4 do not match the stylar genotype.

The heterosis model also suggests why androgenically produced haploids of self-incompatible plants may be self-compatible. It suggests that self-incompatibility is merely one manifestation of genetic load and, in haploids, only those rare individuals that happen to be largely free of deleterious recessives survive. When these rare individual haploids undergo chromosome doubling, they give rise to diploids that carry few such recessives. According to the heterosis interpretation of self-incompatibility, these rare individuals should be self-compatible.

Finally, this interpretation indicates why the complementation model could function even when the style is heterozygous for incompatibility alleles (*5*). Self-pollination allows fewer complementations than does cross-pollination. Thus, although pollen tube growth rates are indeed increased by stylar heterozygosity, predicted growth rates for self-pollen tubes will, nevertheless, be slower than will those for nonself-pollen tubes (Fig. 1).

Nature of the Incompatibility Supergene

In the above discussion, we explained our model of pollen-style interactions and self-incompatibility by assuming the existence of a number of supergenes, each somewhat analogous to the usual concept of S genes. In reality, there is at present no information available on either how many genetic factors are involved in gametophytic self-incompatibility or what their linkage relationships might be. Perhaps, instead of being linked to each other as supergenes, these loci are randomly scattered throughout the entire genome. In other cases, selection for increased linkage between loci having major effects on pollen-style interactions could produce something like the conventionally envisioned S genes. However, the advantages, if any, of increased linkage between such loci have not yet been fully explored.

In some cases, it may be appropriate to abandon the concept of incompatibility genes altogether and, instead, describe some angiosperm species which are unable to set seeds after self-pollination, not as being self-incompatible, but rather as possessing too many deleterious recessive alleles to allow self-fertility.

The development of the heterosis model fits what seems to be a well-established pattern. Although self-incompatibility has been reported in ferns, it was shown to be a simple case of genetic load (*32*), that is, outbreeding produced more viable offspring than inbreeding did. Similarly, indications of self-incompatibility within the gymnosperms were later shown to be the expression of deleterious recessives (*33*). With the proposed interpretation of gametophytic self-incompatibility in the angiosperms, a similar conclusion is reached. The major difference here is that, in the angiosperms, the selection is prezygotic, whereas with the ferns and gymnosperms it is postzygotic. Presumably, the difference is due to the presence of the angiosperm style, a structure that greatly enhances opportunities for pollen tube competition and selection (*34*). However, even within the angiosperms, postzygotic expression of incompatibility has been reported (*35, 36*).

Not least among the implications of the heterosis model is the support it provides for the possibility, first suggested by Larsen (*20*), that self-incompatibility is not a system of ancient origin. Instead it is perhaps far more dynamic, able to vary from self-fertility to self-incompatibility.

The significance of the proposed interpretation goes beyond self-incompatibility. For example, if there are heterotic interactions between pollen tubes and stylar tissues, they could include also loci that are not specifically involved in self-incompatibility. This possibility takes on a special significance since as many as 60 percent of the structural genes that are expressed in the sporophyte of *Lycopersicum esculentum* are expressed also in the pollen (*16*). If a substantial fraction of this 60 percent is subjected to the heterosis-like pollen-

style interactions that our model requires, then, given pollen competition, these interactions could maintain heterozygosity at levels higher than those predicted on the basis of inbreeding coefficients (*37*). Such a selective mechanism would also help to explain both the vigor of plants produced under conditions of intense pollen competition and the relatively great vigor exhibited by the angiosperms (*38*).

References and Notes

1. There are examples of gametophytic self-incompatibility that involve four or more incompatibility loci [see (*20, 29, 30*)], all multiallelic, and, in these cases, incompatibility results only when all pollen incompatibility alleles are included in the stylar complement. In most cases, however, it is believed that only a single locus is involved.
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