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An Insight into Physiological and Molecular Basis of Sporophytic Self Incompatibility in Cole Crops

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Abstract

Self-incompatibility (SI) is genetically controlled, physiological hindrance to self-fruitfulness, and is probably the most important way to enforce out crossing. In all the members of Brassicaceae family including Cole crops, homomorphic sporophytic self-incompatibility (SSI) system exists, associated with trinucleate pollen and inhibition of self-pollen germination on 'dry' stigmatic surface (Bateman 1952, 1955). The sporophytic system of self-incompatibility is a widespread genetic phenomenon, in Brassica species, self-incompatibility has been mapped genetically to a single chromosomal location. In this region, several closely linked genes have been identified and this event is of commercial importance in hybrid breeding of Brassicaceae crops and is controlled by single S locus with multiple S haplotypes (Sehgal *et al*., 2018). The molecular genetic studies of Brassica 'S' locus has revealed the presence of three tightly linked loci viz. S-receptor kinase (SRK), S-locus cysteine-rich protein/S-locus protein 11 (SCR/SP11), and S-locus glycoprotein (SLG). On self-pollination, the allele-specific ligand–receptor interaction activates signal transduction in stigma papilla cells and leads to rejection of pollen tube on stigmatic surface. In addition, arm-repeat-containing protein 1 (ARC1), M-locus protein kinase (MLPK), kinase-associated protein phosphatase (KAPP) play key role in Self-incompatibility signalling pathway.

Keywords: Self incompatibility, Sporophytic, Haplotypes, S-receptor kinase (SRK)

Introduction

Self-incompatibility refers to the genetic phenomenon of inability of plants having functional gametes to set seeds when either self-pollinated or crossed with some of their genetic relatives. That is, the inability of pollen to fertilize the stigma of the same flower hence, promotes outcrossing. Self-incompatibility was earliest reported by Koelreuter in *Verbascum phoeniceum* (1764) and pioneer discussion on SI was done by Darwin (1877) and the term was given by Stout (1917). Bateman (1952) provided explanation on SI in *Brassica campestris* and *Raphanus sativus*.

The presence of self- incompatibility allows natural hybridization between two dissimilar lines without the need for emasculation. On the other hand, unlike male sterility, normal and functional pollen grains are produced in the self-incompatible plants, which amply attract the pollinators so that

natural hybridization is accomplished successfully. Being a natural method, self-incompatibility has no adverse side effects, such as those often found with cytoplasmic or chemically induced sterility. However, it is often less than perfect. The possibility of using self-incompatibility to produce hybrids was suggested many years ago by Pearson (1932). However, in the recent years, use of self-incompatible lines has become a quality practice for the production of commercial hybrid seed in many cole crops. Among the cole vegetables like cabbage, cauliflower, broccoli etc., sporophytic self-incompatibility mechanism is being tapped for the commercial hybrid seed production in several locations including India.

Classification of Self incompatibility

Lewis (1954) has given two types

1. Heteromorphic self-incompatibility:

   In this case, incompatibility is owing to variation in the flower morphology

   It has two types:

   • Distyly, it's found in Primula

   • Tristyly, it's found in Lythracea

2. Homomorphic self-incompatibility:

Self-incompatibility is because of genotype of the plant or genotype of the pollen.

Mainly two types are there

• Gametophytic self-incompatibility, which was given by East and Mangeldorf (1925 in Tobacco)

• Sporophytic self-incompatibility explained by Hughes and Babcock (1950 in Crepis foetida)

In homomorphic system, the mechanism is based on protein-protein interactions, and are controlled by a single locus termed S, with multiple alleles and also it is said to be the majorly found self-incompatibility.

Physiological basis of Sporophytic Self-incompatibility

Brassica oleraceae exhibits pollen grains with an outer lipidic coating superficial layer (CSL), below which, the tryphine exists. Stigma is papillate and covered with a pellicle or sheath and also cuticle (/wax). When compatible pollen falls on the stigma, pollen CSL fuses with papillae pellicle within a fraction of second, followed by the tryphine flow out and later resulting in a gel formation with adjacent pellicle. Finally, the pollen hydrates, germinates, releasing cutinase leading to pollen tube development. If in case, an incompatible pollen falls on the stigma, SI reaction is soon triggered, CSL does not fuse with the pellicle cells and tryphine do not form gel completely affecting the hydration of the pollen grain. In any case, SI reaction is accompanied by callose deposition in the papillae which is synthesised by the cytoplasmic cells of the stigmatic cells.

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Cole crops	Level of SI
Kale and round headed cabbage	High level of SI (Taylor and Anderson,1965)
Broccoli	Comparatively low level of SI
Early summer cauliflower	Very low level of SI (Watts1965)

(Sehgal et al., 2018)

Molecular Mechanism of Self Incompatibility in Cole Crops (SSI)

Self-incompatibility operating in the Brassicaceae is primarily controlled by multi-allelic single locus S. The structure of the locus is complex with at least three important genes viz., S locus glycoprotein (SLG), S locus receptor kinase (SRK) (Stein et al.,1995) and S locus cysteine- rich protein (SCR) (Suzuki et al.,1999; Takayama et al., 2000).

Female determinants

The below mentioned are the female determinant proteins encoded by the S locus specifying SI with respect to stigma.

1. S locus glycoprotein (SLG): Was the first gene to be recognised as a female determinant in the S locus encoding a glycoprotein secreted into stigmatic papillar cell wall. It works as a co-receptor of male determinant and significant for SRK stabilization, sharing about 90% similarity with SRK. 2. S locus receptor kinase (SRK): Is an indispensable protein for SI reaction, plasma membrane anchored signalling receptor which encompasses three domains namely, an extra cellular S domain which is the centre for recognition of pollen ligand, Transmembrane domain and an intracellular kinase domain which acts as a signal transduction in stigma cells (Sehgal et al.,2018).

Male determinants

These proteins are encoded by the S locus specifying SI with respect to pollen. These genes are generally accumulated at the tapetum cell wall when the pollens get matured. SCR or SP 11 is the male determinant synthesised on anther tapetum (*Brassica rapa*) as S 9 haplotype specific gene and named as SP 11 (Suzuki et al.,1999). Upon pollination, SP 11 penetrates the papilla cell wall and binds SRK in an S haplotype- specific manner hence the autophosphorylation of SRK, triggering a signalling cascade that results in the rejection of self-pollen (Takayama et al., 2003).

Molecular mechanism of sporophytic self-incompatibility

Self-pollen recognition and rejection are the major steps involved in the mechanism of self-incompatibility. Expression of the above-mentioned key proteins is tightly regulated, being undetectable in small flower buds and reaching maximal levels just before flower opening. During anthesis itself, the discrimination between self and cross pollen occurs. In the stigma, the SRK protein appears to form dimers or oligomers with the two proteins that resemble thioredoxin-H (THL1 and

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THL2). In vitro, these two proteins bind with the kinase domain of SRK without regard to phosphorylation, and it has been shown that this association prevents SRK oligomers from becoming autophosphorylated. As a result, it is anticipated that the interaction with THL1 will keep SRK in a "inactive" state in vivo. When pollen grains or isolated pollen coatings of the same S haplotype are present, the SRK complex changes quickly into a "activated" form in vivo thanks to a haplotype-specific interaction between SCR and the receptor domain of the protein. The dissociation of THL1 and THL2 from the domain is observed to occur immediately as a result of this interaction, but it still has to be proven in vivo. This contact causes the rapid autophosphorylation of serine and threonine residues in the kinase domain of SRK. The kinase domain of this activated form of SRK is then projected to interact with particular cytosolic proteins that target the incompatible pollen grain for rejection, leading to the initiation of a signalling cascade within papilla cells.

On the Brassica stigma, incompatible pollen is effectively rejected; a single papilla cell will permit the development of a compatible pollen grain while rejecting an incompatible grain that is unquestionably adjacent to it. Because the differences in development (hydration, germination, and stigma penetration) between compatible and incompatible pollen grains are visible within 10–20 minutes of pollination, the 'rejection' process likewise happens extremely quickly. 'Rejection' at this time period is also reversible since incompatible pollen grains can be 'resurrected' by being transferred to a stigma with a compatible stigma. Any theories on the mechanism of self-pollen rejection must take into account these physiological observations. A protein called ARC1 that is particular to the stigma and has the Armadillo repeat motif is a leading contender for the role of the cytosolic signalling cascade's initiator that controls self-pollen rejection. The kinase domain of SRK interacts strongly and specifically with ARC1 in a phosphorylation-dependent manner and antisense loss-of-function experiments linked reduced ARC1 expression profiles to a diminished capacity of stigmas to effectively reject incompatible pollen.

The pollen cell wall contains a lot of PCPs, which come into contact with the stigma cell wall's abundant SLG and SLR1 proteins. SLG, which is freely diffusible within the cell wall, could bind PCR at the interface of the papillar cell wall and pollen for presentation at the membrane, acting as an extracellular regulator of ligand access to the signalling receptor. Perhaps the PCR protein is bound to the extracellular amino acid residues of SRK. According to the conventional theory, SRK dimerization is what triggers the SI response (Nasrallah et al., 1994). The enzymatic characteristics of recombinant SRK were examined by Giranton et al. (1999), who hypothesized that, rather than ligand-dependent dimerization of SRK molecules, signal transmission during the SI response is mediated by alteration of an existing SRK oligomeric complex.

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Conclusion

With the progress in advanced molecular approaches, it is possible to elucidate the multiple complex mechanisms involved in SI response and understanding multiple Brassica SI signalling pathways so that the SI lines can be best exploited for commercial hybrid seed production in Brassica species.

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