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Recognition of "self" can be deadly: receptor-ligand interactions and signalling networks that trigger programmed cell death in pollen

Professor Noni Franklin-Tong
School of Biosciences, University of Birmingham, UK

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場所：東京大学理学部２号館223号室
連絡先：福田裕穂（fukuda@biol.s.u-tokyo.ac.jp/03-5841-4461）

Self-incompatibility (SI) is an important mechanism used by many higher plant species to prevent inbreeding. It is controlled by a multi-allelic *S* locus that allows discrimination between "self" (incompatible) pollen from "non-self" (compatible) pollen, which is allowed to fertilize the plant by interaction of pollen and pistil *S* locus components. In *Papaver rhoeas*, the pistil *S* determinant (recently renamed as **PrsS, Papaver rhoeas stigma S**) is a small novel protein that acts as a signalling ligand. It interacts with its cognate pollen *S*-determinant (**Papaver rhoeas pollen S**), PrpS. *PrpS* is specifically expressed in pollen, is linked to the pistil *S* gene, and displays the high polymorphism expected of an *S* locus determinant. It encodes a novel ~20 kDa transmembrane protein with no homology to proteins in existing databases. PrpS was recently shown to be functionally involved in SI. Identification of *PrpS* as the *Papaver* pollen *S*-determinant strongly supports the hypothesis that *Papaver* SI is triggered by a receptor-ligand interaction. This was originally based on the finding that when PrsS interacts with incompatible pollen, it triggers increases in cytosolic free Ca^{2+}. We have recently begun to use electrophysiology to characterize the currents activated by SI, and speculate that PrpS may function as a channel or pore protein.

Ultimately, and probably the major target for SI signals is initiation of programmed cell death (PCD) involving several caspase-like activities in incompatible pollen. This provides a very neat way to get rid of unwanted "self" pollen and prevent self-fertilization. I will talk about the Ca^{2+}-dependent signalling network, and its targets and how we currently think it is integrated. The actin cytoskeleton seems to be centrally involved in integrating or signalling to PCD and I will discuss new data from our SI system relating to this, suggesting polymerization of actin may be important; analysis of actin-binding proteins that mediate this stage may provide clues to the story. I will also present unpublished data on reactive oxygen species (ROS) and nitric oxide (NO) signalling triggered by the SI response. Recent work has shown that SI-induced caspase-like proteins, such as DEVDase, have a very narrow acidic pH optimum, which suggests SI might trigger dramatic changes in the cytosolic pH. We have investigated whether acidification of the cytosol may be due to disruption of organelles, and I will present data showing that the vacuolar compartments of SI-induced pollen tubes undergo reorganization and disintegrate. This SI-induced disruption could potentially generate the optimal acidic pH for caspase-like activities which results in PCD in incompatible pollen tubes.

References

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