Allele-specific interactions between highly-polymorphic S locus-encoded receptors and ligands control the discrimination between “self” and “non-self” pollen by the stigma in the self-incompatibility response of the Brassicaceae. The S-locus receptor kinase SRK, which is displayed at the surface of the stigma epidermis, is only bound and activated by its cognate ligand, the small S-locus cysteine-rich protein SCR, which is localized in the pollen coat. The SRK-SCR interaction triggers a poorly understood signaling cascade within the stigma epidermal cell that culminates in inhibition of pollen tube emergence or growth.

To aid in analysis of self-incompatibility, we developed an Arabidopsis thaliana transgenic self-incompatible model by transformation with functional SRK-SCR gene pairs isolated from A. lyrata or Capsella grandiflora. Our molecular genetic analysis of these transformants has elucidated several features of the recognition and response phases of self-incompatibility. In planta analysis of a large number of engineered receptor variants led to the identification of residues required for SRK function and those required for its ligand-selective activation. A reverse genetics strategy suggested that regulation of SRK catalytic activity and orchestration of the SI response in Arabidopsis do not use the regulators or signaling components that have been implicated in Brassica self-incompatibility. And a forward genetic approach uncovered unexpected linkages between inhibition of “self” pollen at the stigma surface and pistil development, suggesting that S-locus receptor-mediated signaling was co-opted from a receptor-based developmental signaling pathway.