

Wed, 29 Mar 2017 | 5pm | DBS Conference Room 1

Hosted by A/P Loh Chiang Shiong

# Natural Variation in the Plant Immune System and its Adaptive Value



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#### **Suggested readings**

1. 1001 Genomes Consortium. 1,135 Genomes Reveal the Global Pattern of Polymorphism in *Arabidopsis thaliana*. *Cell*, 2016. 166(2): p. 481-91.
2. Bomblies, K. and D. Weigel, Hybrid necrosis: autoimmunity as a potential gene flow barrier in plant species. *Nat Rev Genet*, 2007. 8(5): p. 382-93.
3. Chae, E., et al., Species-wide genetic incompatibility analysis identifies immune genes as hot spots of deleterious epistasis. *Cell*, 2014. 159(6): p. 1341-51.
4. Chae, E., D.T. Tran, and D. Weigel, Cooperation and Conflict in the Plant Immune System. *PLoS Pathog*, 2016. 12(3): p. e1005452.

Individuals of a species respond differently to environmental stresses and the genetic makeup is largely responsible for the variation in responses. Now is the most exciting time to investigate the phenotypic variation in complex traits, as the advent of new sequencing technologies made tremendous genomic information available in a given species [1]. Genome sequencing projects revealed that genetic variability in the plant immune system is exceptional, reflecting complex defense strategies that plants employ to fend off myriad pathogens. The extreme variation sometimes makes a fatal outcome. Hybrid necrosis is the best-known example of genetic incompatibility in plants, in which autoimmune responses are triggered by deleterious interactions of independently evolved immune alleles [2]. My postdoctoral research exploited genetic and genomic tools available in the model plant species *Arabidopsis thaliana* to systematically investigate intraspecific hybrid necrosis. The species-wide work identified several incompatibility hot spots in the genome, often in regions densely populated by NLR immune receptor genes with high variability in the populations [3]. A particularly dangerous locus is a highly variable cluster of NLR genes, DANGEROUS MIX2 (DM2), which causes multiple, independent incompatibilities with genes that encode a range of biochemical functions, including other NLRs. Our findings suggest that deleterious interactions of immune components at the front lines of host-pathogen co-evolution limit the combinations of favorable disease resistance alleles accessible to plant genomes.

This systematic work provides a unique platform to further investigate molecular mechanisms of immune receptor activation and to dissect tradeoffs between immunity and growth in plants [4]. In my talk at NUS, I will address topics on how studies of genetic incompatibility contribute to mechanistic understanding of adaptation, how natural variation can be exploited to understand trait evolution, and how the current knowledge on G x G can be translated to plant breeding.