Inhibiting Infection by Xcm

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Cotton serves as a significant source of fiber, feed, foodstuff and oil products. Its production is hindered by various biotic and abiotic stresses. The complex genome and tetraploid nature of this important crop, as well as limited molecular and genetic tools, make it difficult to understand the molecular mechanisms underlying cotton disease resistance. My laboratory has been developing molecular tools and genetic resources for gene discovery and manipulation at genome-wide level in cotton. We have recently developed a protoplast-based transient transfection assay for gain-of-function and Agrobacterium-mediated virus-induced gene silencing (VIGS) assay for loss-of-function studies in cotton. These assays allow us to rapidly identify novel gene functions and decipher their roles in signaling pathways. The highly efficient VIGS assays work in both diploid and tetraploid cottons and various commercial cultivars. Construction a high-coverage cotton VIGS library enables us to deploy functional genomic approach to understand cotton gene functions at a large scale within a short time period. Protoplasts can be used as a vehicle to transiently express and systemically characterize the candidate gene functions. The identified novel genes will provide valuable resources for cotton improvement with particular interest in disease resistance.

Bacterial blight of cotton (BBC), caused by *Xanthomonas campestris* pv. *malvacearum* (*Xcm*), is among the most devastating diseases of cotton worldwide. *Xcm*, like many Gram-negative bacteria, injects a battery of effector proteins through the type III secretion system into host cells to promote pathogenesis. In non-resistant genotypes, pathogen effector proteins often function as pathogenicity (Pth) factors recognized by host susceptibility (*S*) genes important for disease. Importantly, all known *pth* genes of *Xcm* encode transcription activator-like (TAL) effector proteins. Recent advances revealed that TAL effectors modulate transcription of host genes by directly and specifically binding to their promoters. In most studied cases, virulence function of TAL effector results from transcriptional activation of a corresponding *S* gene. That is, host “recognition” of the pathogen occurs at the *S* gene promoter. The decoding of host DNA recognition by TAL effectors has made it possible to computationally predict TAL effector binding elements (EBEs) in a host genome and identify candidate *S* genes. By combining genome-wide expression profiling, code-assisted bioinformatics prediction and protoplast and VIGS-based functional genomic approach, we aim to identify the cotton genes mediating BBC disease. We also aim to control cotton disease to *Xcm* infection by targeted genome editing of *Xcm* recognition site in the key susceptibility genes mediating BBC. This effort will provide a proof-of-concept biological means to control cotton disease to *Xcm* infections.