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MOLECULAR DISSECTION OF DISEASE RESISTANCE

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Abstract Only

Bacterial blight of cotton, incited by the pathogen Xanthomonas campestris pv. malvacearum (Smith) Dye (Xcm), is a classical example of a plant-pathogen relationship in an important crop. A 70-year history of research has led to our current understanding of both plant resistance of cotton and pathogenicity of the bacterium. Several unique features of the cotton-Xcm system distinguish it from other systems for studying plant-pathogen interactions. Cotton is an allotetraploid comprised of two genomes that form strictly bivalents at meiosis. Polyploid cotton arose about 1-2 million years ago as a result of interspecific hybridization between an Old World A-genome diploid cotton and a New World D-genome diploid cotton. Currently, 19 races of Xcm pathogenic to cotton are recognized in the United States. Twenty-two genes conferring resistance to Xcm have been reported, with most being described as partially to completely dominant. New molecular approaches empower us to study complex plant-pathogen interactions in crops, at a new level of resolution. Analysis of the subgenomic (A vs. D) distribution of genes conferring resistance to Xcm in tetraploid AD-genome cottons provides an interesting system for studying the impact of allopolyploid formation on host-pathogen interactions. Among wild relatives of cotton, A-genome cotton shows near-immunity to Xcm which was deemed sufficiently valuable to begin introgression into cultivated cotton. The Xcm immunity of A-genome cottons and the observation that new virulent strains appear to have arisen in Africa is consistent with the probable Old World origin of the pathogen. Although D-genome diploids show varying degrees of resistance, none show “immunity” (Knight 1948), and they have not been an important source of resistance genes for the improvement of cultivated cottons. Structural genomics has revealed an interesting trend regarding the chromosomal locations and subgenomic distribution of cotton R-genes that confer resistance to Xcm. Genetic mapping generally corroborated classical predictions regarding the number and dosage effects of genes (B2, B3, and B12) that confer Xcm resistance. Only the "b6 phenotype" conferring resistance to Xcm Races 7 and 18 exhibited numerous inconsistencies. Among the seven resistance genes derived from tetraploid cottons, six (86%) mapped to D-subgenome chromosomes. If each subgenome were equally likely to evolve new R-gene alleles -- “1 or fewer successes in 7 trials, where p=q=0.5” -- yields a likelihood of only 0.063. This suggests the D-subgenome of tetraploid cotton has a higher propensity to give rise to new R-gene alleles and may suggest that polyploid formation has offered novel avenues for phenotypic response to selection. Possible explanations of this bias include biogeographic factors, differences in evolutionary rates between subgenomes, gene conversion or other intergenomic exchanges that escaped detection by genetic mapping, or other fact