Mapping and Selection of Bacterial Spot Resistance in Complex Populations

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Bacterial spot of tomato is caused by at least four species of bacteria in the genus Xanthomonas. Physiological races of the pathogen exist, and developing resistant varieties will require pyramiding resistance from multiple accessions. Sources of resistance include the S. lycopersicum breeding lines from Gilbert's program, Hawaii 7998 and Hawaii 7981; the cherry accession PI114490 and the S. pimpinellifolium accessions PI 128216. Inbred backcross (IBC) populations have been developed for each of these sources, and QTL analysis has identified linkage to major loci contributing to both hypersensitive response (HR) and field resistance. Rx-3 from Hawaii 7998 is localized to chromosome 5; Rx-4 from PI 128216 is localized to chromosome 11; and major QTL from Ha 7998 and PI114490 are also localized to chromosome 11. Advanced lines containing resistance from at least three distinct sources were crossed and the hybrids were again inter-mated to produce a complex segregating population. Resistant and susceptible extremes were selected from nurseries inoculated with T1 (Fremont, OH) or T3 (Wooster, OH) in 2007. For each trial, over 1,100 individual plants were evaluated and selection for the best and worst 5% was imposed. Progeny from these selections were evaluated as plots in replicated trials in 2008. For the T1 evaluation, plants rated as resistant in 2007 produced plots with an average disease rating of 4.02 in 2008 (the best resistant parent was rated 3.5), while plants rated as susceptible produced plots with an average rating of 5.16 (LSD 0.5 = 0.39). The narrow sense heritability was estimated to be 0.32 and realized gain under selection was 13%. We investigated four statistical models for association mapping: single-point analysis; single-point analysis corrected for population structure; haplotype analysis; and haplotype analysis corrected for population structure. Markers that were linked to specific resistance in biparental populations were often in linkage equilibrium (unlinked) in the complex population. Thus a given marker allele state might be associated with resistance in one parent, but susceptibility in others. Based on previous knowledge of relative map position for Rx-3, single marker analysis was ineffective at localizing resistance. Haplotype analysis markedly improved our ability to localize QTL, and correcting for population structure reduced the detection of spurious marker-trait linkage. Application of MAS to complex populations will be dependent on identifying markers that are in LD within the population. The rate-limiting step for application to elite populations will therefore be the identification of a sufficient number of markers to establish tight linkage. Haplotype analysis and accounting for population structure will improve association analysis in populations with complex parentage. Population structure can be estimated from pedigree data or unlinked markers, and a breeding value can be estimated for individuals based on both marker data and pedigree. Although these approaches appear promising, marker coverage is insufficient to replace phenotypic selection in early generations.