

Mapping of Early Blight Resistance QTLs in a RIL Population of Tomato

Hamid Ashrafi*, Guoyang Lin and Majid R. Foolad

Department of Horticulture and the Intercollege Graduate Degree Program in Genetics, The Pennsylvania State University, University Park, PA

Early blight (EB), caused by *Alternaria solani* and *A. tomatophila*, is one of the most common and destructive diseases of the cultivated tomato (*Solanum lycopersicum*) in areas of heavy dew, frequent rainfall, and high relative humidity. In the U.S. the disease can be very severe in the Midwestern, eastern and northeastern regions. EB is associated with physiological maturity of the plant; older, senescing leaves are more susceptible than young, immature leaves, and a heavy fruit set enhances the disease. No genetic source with strong resistance to EB is known in the cultivated species. At present, sanitation, long crop rotation, and routine application of fungicides are the most common disease control measures. Sources of genetic resistance to EB were previously identified within the green-fruited species *Solanum habrochaites* (formerly *Lycopersicon hirsutum*) and several breeding lines with measurable level of resistance were developed at the NC State Tomato Breeding Program (RG Gardner). The NC lines can tolerate an extended fungicide spray interval, however, they are often late maturing. We have identified sources of EB resistance within the red-fruited species *S. pimpinellifolium*. While we have used these accessions in our traditional breeding program at Penn State to develop high-yielding, early-maturing tomatoes with improved EB resistance, we also have identified and mapped resistance QTLs in different *S. lycopersicum* · *S. pimpinellifolium* populations to facilitate marker-assisted breeding. Here we report on development and use of a new RIL population (n = 172 lines) for mapping EB resistance QTLs. A genetic linkage map of the population was developed with ~275 molecular markers, which was used to identify QTLs based on a 4-year field study (F₇ - F₁₀). Five QTLs were identified with individual phenotypic effects ranging from 6.0% to 26%, of which three QTLs on chromosomes 5, 6 and 9 were consistent across 3-4 years/ generations. Co-localizations of QTLs with candidate ESTs such as *Mi-1*, ethylene response factor-5, lipoxygenase B were observed.