Identification of SNP Markers Associated with Resistance to Aphanomyces Root Rot Race 2

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Aphanomyces root rot (ARR), caused by the oomycete *Aphanomyces euteiches*, is one of the most important diseases of alfalfa in the U.S. Infected seedlings are stunted with chlorotic cotyledons that become necrotic as the disease progresses. Infected roots and hypocotyls first appear gray and water-soaked, then turn yellowish-brown. In established plants, the pathogen causes a sublethal rot of fibrous and lateral roots in wet soil conditions, reducing nodulation, and leading to yield loss and reduced stand life. Two races of the pathogen are distinguished by differential cultivars; WAPH-1 with resistance to race 1 and WAPH-5 with resistance to race 1 and race 2. Although most commercial cultivars in fall dormancy classes 2 to 5 are highly resistant or resistant to race 1, fewer have resistance to race 2, the predominant race in North America. Race-specific resistance involves a hypersensitive response of individual epidermal or cortical cells upon zoospore penetration and is highly heritable, suggesting that resistance is conditioned by a small number of genes, most likely of the NBS-LRR resistance genes. Resistance to race 2 in commercial cultivars was selected from improved plant materials from breeding programs, while resistance in WAPH-5 was derived from PI 468018, PI 439006, and PI 464781, which were identified as having low levels of resistance to *A. euteiches* race 2. Resistant and susceptible plants were identified from the commercial cultivar 53V52 and the check cultivar WAPH-5 and were used as parents to produce F1 mapping populations. Genotyping of the 53V52 derived line was done using genotyping-by-sequencing and with 3,000 DaRTag SNP markers while genotyping of the WAPH-5 derived line was done with only DaRTag markers. Plants were phenotyped with one or more race 2 strains. Markers significantly associated with resistance was identified on chromosome 2 in the 53V52 line and resistance in the WAPH-5 line on chromosome 5. A principal component analysis of the SNP markers in the WAPH-5 derived F1 population indicates that the resistance trait was derived from a small number of parents. Transcript profiling was done to gain a better understanding of the compatible and incompatible interactions and mapped to the QTLs for race 2 resistance to identify candidate genes. Markers associated with race 2 resistance from WAPH-5 can be used to identify the resistance QTL in diverse germplasm and to follow introgression of the trait into elite parents to increase resistance to the disease.