Identification of QTL Associated with Resistance to White Mold in Soybeans

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Sclerotinia is a fungal plant pathogen that infects over 500 species worldwide (Saharan and Mehta, 2008). The extend of crop losses due to this disease, the lack of genetic resistance and the difficulty of managing the disease make the pathogen subject of major research initiatives around the world. White mold in soybeans is also known as Sclerotinia Stem Rot (SSR); the causal pathogen *Sclerotinia sclerotiorum* causes significant damage in the Northern geographies of United States and Canada (Wrather et al., 1997). Chemical control is difficult to implement due to difficulty in determining timing of application. Some cultural practices have been shown to reduce disease incidence: rotation, reduced tillage (Kurle et al 2001), and wide row spacing (Mila and Yang 2008). Developing resistant varieties is the most practical way to manage the disease.

The objective of my project is to improve the ability to identify and characterize QTL associated with resistance to *Sclerotinia sclerotiorum*. Characterization and identification of quantitative trait loci will facilitate the development of novel germplasm and soybean varieties with field resistance to white mold.

Previous research has shown that resistance to white mold is partial and of quantitative nature (Kim et al, 1999). Kim and Diers (2000) studied broad-sense heritability under field conditions. Broad-sense heritability estimates for Disease Severity Index (DSI) ranged from 0.3 to 0.71 at individual environments and 0.59 across all environments. Because of the variability observed in field tests, physiological resistance to white mold is evaluated under controlled conditions. Inoculation methods using mycelium or ascospores have been tested on seedlings growing in a greenhouse environment or detached leaves in the laboratory. Mixed results have been reported of correlations between greenhouse and field evaluations which make disease resistance difficult to evaluate. Progress has been made, however by using application of mycelium culture on the apical stem or floral buds (Chen and Wang 2005 and Huynh et al 2010).

Kim and Diers (2000) evaluated Disease Severity Index (DSI) under field conditions and reported 3 QTLs on linkage groups C2 (chromosome 6), K (chromosome 9) and L (chromosome7). Arahana et al (2001) took a different approach and evaluated lesion area using the detached leaf assay in a laboratory setting; using 5 mapping populations Arahana et al (2001) reported 28 QTL on 15 different linkage groups. More recently, Guo et al. (2008), Vuong et al. (2008), and Huynh et al (2010) reported 12 QTL associated to white mold resistance by measuring stem lesion length through a combination of field and greenhouse experiments. Given the variability among previous studies there is still a need to map *Sclerotinia sclerotiorum* resistance genes in soybeans. These studies reported to date used a range between 50 (Arahana, 2001) to 134 SSR (Vuong, 2008) markers. With the development of high-throughput genotyping techniques and the availability of single nucleotide polymorphic markers (SNPs), soybean researchers have now new resources for QTL discovery in soybean (Hyten et al, 2010). In this study the Golden Gate Assay that provides a set of 1536 SNP markers will be used to genotype two mapping...
populations derived from resistant parent MN0091. Two mapping populations consisting of 165 and 100 recombinant inbred lines will be evaluated in field and greenhouse conditions. Physiological resistance will be evaluated by measuring lesion length development on main stem.

References


