White mold (Sclerotinia sclerotiorum) is a serious disease of common bean (Phaseolus vulgaris). In snap beans, not only does it reduce yield and quality, but harvest lots with greater than 3% incidence of moldy pods are rejected at the cannery. One of the best sources of resistance to white mold is P. coccineus (runner bean). We first screened the P. coccineus USDA-NPGS plant introduction collection to identify accessions with highest levels of resistance (Gilmore et al., 2002). The resistant accession PI255956 was crossed to susceptible ‘Wolven Pole’ to create an F2 mapping population. We characterized the population phenotypically for white mold resistance using the straw test, and mapped molecular markers and QTL associated with resistance. Subsequently, we crossed PI255956 to Oregon 91G bush blue lake green bean. The F1 was backcrossed twice to 91G using the backcross-inbred (BCIB) method to create a BC2F4:6 population that was characterized phenotypically and mapped (Haggard & Myers, 2006). We summarize our findings from these mapping efforts.

Wolven Pole/PI255956 population: This population of 188 individuals was tested with an eight day and five week straw test. A five week reading of the straw test was implemented because of the resistance levels observed in P. coccineus. Random amplified polymorphic DNAs (RAPDs), simple sequence repeats (SSRs), and amplified fragment length polymorphisms (AFLPs) were used to create a map. Two-hundred fifteen markers were placed in thirteen linkage groups and spanned a total distance of 797 cM (LOD 4, 30 cM maximum distance between linked markers). We estimate that the P. coccineus map covers approximately 65% of the genome.

Figure 1. Linkage fragment from P. coccineus map with a pair of QTL that explains 89.6% of the phenotypic variation for white mold resistance in a five week straw test.

Linkage Group C

<table>
<thead>
<tr>
<th>Distance (cM)</th>
<th>Markers</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>OPQ-111750</td>
</tr>
<tr>
<td>8.4</td>
<td>OPQ-111700</td>
</tr>
<tr>
<td>20.3</td>
<td>E32M59-418</td>
</tr>
</tbody>
</table>

Single factor analysis revealed highly significant association with nine marker loci on four linkage groups. With composite interval mapping, four QTL were placed on this map. The two QTL related to the five week straw test explained 89.6% of the phenotypic variation (Fig. 1). Two other QTL, associated with the eight-day straw test results, explained 13.8% of the phenotypic variation.

Previously, we used interval mapping to place five QTL for white mold resistance on five separate linkage groups (Gilmore & Myers, 2004). The only QTL that matches the revised map is that placed on LG C. The previous map was based on ninety-four RAPD markers and 11 SSR markers mapped in 94 progeny, which may explain the difference in number of magnitude of QTL.

OR 91G/PI255956 BC-Inbred population: One-hundred fifteen BC2F4 lines were genotyped using AFLPs and SSR markers. Corresponding BC2F5 progeny were evaluated for resistance to white mold in a straw test repeated three times, and for oxalate tolerance in a laboratory test. BC2F6
lines were then tested for resistance under field conditions. Of 172 SSR primer pairs, 98 were polymorphic between parents. Of those, 77 were scorable in the progeny, and two revealed single introgressions. The remaining 21 SSRs were either monomorphic between progeny, or would not amplify in the progeny and were discarded. The single pair of AFLP primers amplified 56 scorable segregating fragments. The linkage map consisted of 11 linkage groups that correspond to 9 of the 11 core map linkage groups based on known SSR marker locations, and a single LG with no anchoring loci. The 11 LGs included 59 loci, covering a total genome length of 140 cM, or approximately 12% of the estimated length of the common bean genome.

Chi-square tests revealed significant divergence from the expected Mendelian segregation ratios at most loci. The only linked SSR marker that fit the expected ratio was BMd-52 on LG 09. Of the unlinked SSR markers, only PVag004 and PVat007 fit the expected ratio. While the homozygous recurrent parental marker class was represented at the expected rate over most loci, the heterozygotic marker class was overrepresented and the homozygous donor parental marker class underrepresented.

Single factor analysis of variance identified 29 marker loci contributing to response in at least one phenotypic test. One QTL conditioning 6% of the phenotypic variance for field resistance was identified by composite interval mapping on LG 09, anchored to the consensus linkage group b09 by SSR loci. Several SSR markers polymorphic between parents failed to segregate in the progeny, particularly those corresponding to bean core map linkage groups b01, b04, and b05.

Fourteen BCIB interspecific lines with snap bean characteristics (762/2-6, 811/43-4, 826/48-3, 828/48-5, 836/3-15, 840/4-6, 853/6-9, 856/7-2, 861/13-14, 880/11-1, 891/15-2, 897/18-1, 903/20-2, and 904/20-3) have shown white mold resistance similar to G122, NY6020, and Ex Rico over two field seasons.

**CONCLUSIONS**

Our initial mapping effort in *P. coccineus* led us to believe that several QTL with small effect conditioned white mold resistance in this species. As markers were added to the map, we discovered a pair of major QTL for resistance with additional minor QTL. The markers associated with this fragment did not map in the interspecific BCIB population, which suggests that this fragment was not transferred. Several entire linkage groups and many regions of other linkage groups were not represented in the BCIB population. This may represent an interspecies hybrid incompatibility barrier to recombination and transfer of genes from *P. coccineus* into *P. vulgaris*. We will test this hypothesis in another BCIB interspecific population that is currently under development.

**REFERENCES**

