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## Genetic mapping of QTL conditioning resistance to soybean cyst nematode in PI464925B

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Soybean cyst nematode (SCN) (Heterodera glycines Ichinohe) is estimated to cause the greatest yield losses to soybean [Glycine max (L.) Merr.] of any pest worldwide. It has been determined that host plant resistance is the most cost-effective and environmentally conscious method of controlling SCN. Phenotypic resistance appears to be quantitative and few cultivars exhibit resistance to one or more races of SCN. Identification of genetically resistant lines will be needed to compensate for various environmental SCN populations. Plant introductions (PIs) from the USDA Soybean Germplasm Collection have been screened for resistance to SCN and relatively few sources have been identified as new sources of SCN resistance. A wild soybean PI464925B (Glycine soja Siebold & Zucc.) is a soybean plant introduction from China shown to have resistance to SCN race 3. In this study, PI464925B was crossed with the SCN susceptible cultivar 'Hutcheson' to generate  $F_1$  hybrids. One hundred twenty-two  $F_2$  derived  $F_3$  progenies were evaluated for reaction to SCN race 3 in a thermo-regulated waterbath (27±1 °C) in the greenhouse at the University of Missouri for reaction to SCN race 3. DNA from leaf tissue of the parents and progeny was extracted and one hundred seventeen of the progenies were used for construction of linkage maps and location of the QTL(quantitative trait loci) by using SSR(simple sequence repeats) markers. Multiplex PCR was performed using fluorescent labeled primers with subsequent analysis on an ABI 3100 DNA sequencer to increase high-throughput of genetic mapping. Genemapper (v3.5) was used for automatic allele sizing and genotyping. Parental testing showed 201 polymorphic SSR markers (56%), providing an average genomic coverage of 12 cM between two markers. Among them, genotypic data from 113 labeled SSR markers on the  $F_3$  progeny were collected to analyze association with the SCN response. QTL locations and genetic contribution of the favored alleles will be discussed.