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ARTICLE

Validation of two QTL Associated with Sclerotinia blight Resistance in Peanut

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ABSTRACT

Sclerotinia blight, caused by *Sclerotinia minor* Jagger, is a fungal disease of peanut that is widespread throughout the cooler peanut-growing regions of the U.S. and can cause yield losses up to 50%, threatening sustainable peanut production. Few cultivars with acceptable resistance have been developed due to a limited understanding of the inheritance of the trait. In this study, correlation of genotypic data collected from a recombinant inbred line (RIL) mapping population (Tamrun OL02 x PI 497429), and phenotypic data collected between 2017 and 2020 revealed two quantitative trait loci (QTL) potentially associated with Sclerotinia blight resistance. Subsequently, members from a RIL validation population (Okrun x PI 497429) were selected based on genotype and phenotyped in 2023 for resistance under heavy disease pressure. Correlation of phenotypic and genotypic data validated the two QTL, qSbl06 and qSbl15, associated with resistance to Sclerotinia blight on chromosomes 6 and 15, respectively. Although qSbl06 is minor, qSbl15 accounted for 52% of the phenotypic variation seen in the validation population. These results will be used to develop and deploy markers for qSbl15 to screen breeding populations and germplasm, as well as aid in the selection of advanced breeding lines for development of Sclerotinia-resistant cultivars.

INTRODUCTION

In the United States, *Sclerotinia minor* (*S. minor*, causal agent of Sclerotinia blight) is one of the most problematic pathogens for peanut production (Wadsworth, 1979). Sclerotinia blight was first found on peanut in Virginia and North Carolina in 1971 and 1972, respectively (Porter and Beute, 1974). Porter and Beute (1974), following the *Sclerotinia* classification of Purdy (1955), initially reported the pathogen as *S. sclerotiorum.* However, later examination by Kohn (1979) identified the small-sclerotial isolates from Virginia and North Carolina as *S. minor.* Other early reports of *S. minor* on peanut in the U.S. were from Oklahoma (Wadsworth, 1973, 1979) and Texas (Goldman *et al.*, 1995). *S. minor* has since been documented on peanut in Arkansas (Faske *et al.*, 2014).

Sclerotinia blight is primarily an issue in peanut production areas with cooler temperatures (Porter and Melouk, 1997; Dufault and Brenneman, 2023) as the optimum temperature for *S. minor* growth is 18 ˚C (Imolehin *et al.*, 1980; Smith *et al.*, 2006). Annual disease losses can approach 50% under severe conditions (Melouk *et al.*, 1992; Melouk and Backman, 1995). The primary inoculum for the disease is sclerotia, which can remain viable in soil for several years without a host (Melouk and Backman, 1995). Sclerotia can spread via infected seed, animals, water, and production machinery (Dufault and Brenneman, 2023; Melouk and Backman 1995; Melouk *et al.*, 1989). High levels of humidity are required for plant infection (Dow *et al.*, 1988). Disease

symptoms typically appear after plant canopies have reached maximum size (Phipps, 1995), creating humid, shaded microclimates favorable for sclerotial germination. Temperatures later in the growing season also favor disease development as plants approach maturity and when evening temperatures are cooler (Dow *et al.*, 1988). Visible symptoms include chlorosis of leaves followed by wilting (flagging) and stem lesions on branches close to the ground (Porter and Melouk, 1997). Infection cushions (white, fluffy mycelium) may be observed during cool and damp conditions and black sclerotia will be seen along surfaces of stem lesions. Disease lesions can progressively move toward the main stem of infected plants and eventually kill the host. Sclerotia are produced along decaying tissue of lesions and deposit in the soil where they overwinter or germinate when environmental conditions are favorable (Thiessen *et al.*, 2012).

Once a field is infested with *S. minor,* disease management options for Sclerotinia blight are limited. Cultural practices such as crop rotation may not be economically feasible for growers due to long-lived sclerotia and the extensive host range of the pathogen (Goldman *et al.*, 1995; Melzer *et al.*, 1997; Hollowell *et al.*, 2003). Methods to manage plant canopy microclimates are ineffective or may be impractical for growers. Reducing seeding rates to create more open canopies does not appear to significantly reduce disease incidence or yields (Phipps, 1987, 1995; Maas *et al.*, 2006). Efforts should be made to minimize unnecessary damage to peanut vines (Porter and Powell, 1978) and to avoid excessive irrigation (Porter and Melouk, 1997). Fungicides are available for controlling Sclerotinia blight (Porter and Melouk, 1997), but application is expensive. Moreover, up to three applications per season may be necessary to maintain a healthy crop in years highly favorable for disease development (Jordan *et al.*, 2007).

Host plant resistance offers the most sustainable means of disease control and as with most diseases, planting resistant cultivars is the most economical approach for managing *S. minor.* Developing cultivars with resistance to Sclerotinia blight is difficult because the inheritance of the trait is quantitative (Wildman *et al.*, 1992) and complex (Corwin, 2017). It is likely that host resistance is controlled by hundreds of defense genes because *S. minor* is a necrotrophic pathogen (Glazebrook, 2005; Liang *et al.*, 2021). In contrast to Mendelian traits that are controlled by a single gene, quantitative traits are controlled by multiple genes with individual effects that are cumulative and often subject to variable environmental conditions. These traits make it difficult for breeders to identify the contributions of each gene to the overall phenotype. Breeders have been able to track quantitative inheritance by the identification and development of markers associated with QTLs in RIL mapping populations. RIL populations designed for mapping disease resistance are traditionally developed by crossing a resistant parent with a susceptible parent, followed by repeated selfing past the F_2 generation. In peanut, selfing to the F_6 generation is advised to ensure all possible gene combinations are represented in the resulting population.

Cultivated peanut is tetraploid ($2n$=$4x$=40), likely derived from a cross between the wild diploid species of *A. duranensis* and *A. ipaënsis*, followed by spontaneous chromosome duplication (Kochert *et al.*, 1996; Seijo *et al.*, 2004; Bertioli *et al.*, 2016; Chen *et al.*, 2019). The tetraploid nature of peanut and the complexity of the genome are factors that contribute to bottlenecks in genetic improvement. The lack of available genetic diversity in cultivated peanut is a limitation because only genes from within the cultivated species or several closely related wild species can be utilized. Some progress has been made in locating genomic regions responsible for *S. minor* resistance in peanut. Chenault *et al.* (2008) examined the genetic diversity among a set of well-phenotyped cultivars with pairs of simple sequence repeat (SSR) markers reported by Ferguson *et al.* (2004). That work identified a specific marker associated with Sclerotinia bight resistance, located on chromosome 7. Validated by the work of Bennett *et al.* (2018), the marker was used in later work to screen germplasm and identify new possible sources of resistance (Bennett *et al.*, 2018; Chenault *et al.*, 2010; Chamberlin, 2014; Chamberlin and Puppala, 2018; Chamberlin *et al.*, 2018, 2020).

Through screening of germplasm, sources of resistance to Sclerotinia blight have been identified (Damicone *et al.*, 2010; Chamberlin, 2014; Bennett *et al.*, 2018; Chamberlin *et al.*, 2018 and Chamberlin *et al.,* 2020; Dura *et al.*, 2020) and some have been incorporated into breeding programs to enable progress in developing cultivars with some level of resistance. Lariat, a cultivar recently released for Southwest production (Chamberlin *et al.*, 2018) was developed from a cross with the *Sclerotinia*-resistant accession PI 497429 from the U.S. core collection (Holbrook *et al.*, 1993; Damicone *et al.*, 2010) and requires no fungicide application to control the disease. The landrace PI 497429, collected from Bolivia in 1983, has a bunch/spreading growth habit, conventional oil chemistry, and variegated seed coat color (striped) (NPGS). The objective of this study was to map the QTL responsible for the strong Sclerotinia blight resistance conferred by PI 497429.

MATERIALS AND METHODS

Plant Materials

Two RIL populations were developed to map and validate the resistance conferred by PI 497429. The $F_{2:6}$mapping population (RIL5) developed was derived from a 2005 cross between the susceptible cultivar Tamrun OL02 (Simpson *et al.*, 2006) and the resistant PI 497429 (Damicone *et al.*, 2010). The $F_{2:6}$validation population (RIL10) resulted from a 2005 cross between the susceptible cultivar Okrun (Banks *et al.*, 1989) and the resistant PI 497429 (Damicone *et al.*, 2010). The average incidence of Sclerotinia blight for the parents of the populations has been reported as 62% for Tamrun OL02, 66% for Okrun, and 4% for PI 497429 (Chenault *et al.*, 2008). Populations were developed via traditional methods resulting in 288 members of the mapping population and 252 members of the validation population.

Phenotyping

The RIL 5 population (mapping), along with susceptible checks Flavor Runner 458 and resistant check Lariat, was phenotyped for Sclerotinia blight resistance in plots at the Oklahoma Agricultural Experiment Station (OAES) Caddo Research Station near Ft. Cobb, Oklahoma, in 2017, 2018, and 2020.

Selected members of the RIL10 population (validation) were phenotyped along with resistant and susceptible checks (PI 497429 and Okrun, respectively) in 2023. Plots were arranged in a randomized complete block design with three replications. All plots were inoculated each year with 0.25 g of *S. minor* sclerotia cultured on potatoes (Patterson and Grogan, 1988). Disease readings were taken each year when plants were at R7 (beginning maturity) and R8 (harvest maturity) (Boote, 1982). Disease incidence was measured by counting the number of 6-in sections within each plot displaying symptoms of Sclerotinia blight, such as wilting and shredded stems (Melouk and Backman,1995; Porter and Melouk, 1997). Mean disease incidence was estimated using PROC GLIMMIX in SAS version 9.4 (SAS Institute, 2023).

Genotyping

DNA was extracted from all members of both populations using a modified CTAB method (Aboul-Maaty and Oraby, 2019). DNA was normalized to 20ng/µL and sequenced using the Twist 96-plex protocol via Illumina NovaSeq S4. Demultiplexed paired fastq files were analyzed via the Khufu bioinformatics platform (HudsonAlpha Institute for Biotechnology). Bulks of resistant and susceptible lines were assayed for allele frequency differences genome wide. Significant peaks were identified by comparisons against a null model, and peaks were extracted as potential QTL. Paired-end fastq files were processed and mapped to Tifrunner gnm2.ann2 (Bertioli *et al.*, 2019) using KhufuCore (https://www.hudsonalpha.org/khufudata/). QTLs were mapped using a modified version of QTLseq (Korani, *et al.*, 2021). QTLs were visualized using the Khufu visualization tool, khufu_var3. (https://w-korani.shinyapps.io/khufu_var3/). Confidence Interval limit was calculated using QTLseqer (https://github.com/bmansfeld/QTLseqr).

RESULTS AND DISCUSSION

Less Sclerotinia blight was observed in 2017 and 2018 than in 2020 (Figure 1). Mean disease incidence in 2017 and 2018 ranged from less than 1% to 39% and 27%, respectively. The 2020 season, with below-average temperatures for September and October, was highly favorable for Sclerotinia blight, enabling better separation of the mapping population's disease response (Figure 1). Average disease incidence across all years among members of the mapping population ranged from 0-89% (Figure 1), and the susceptible (Flavor Runner 458; Beasley and Baldwin, 2009) and resistant (Lariat; Chamberlin et al, 2018) checks averaged 41% and 25%, respectively.

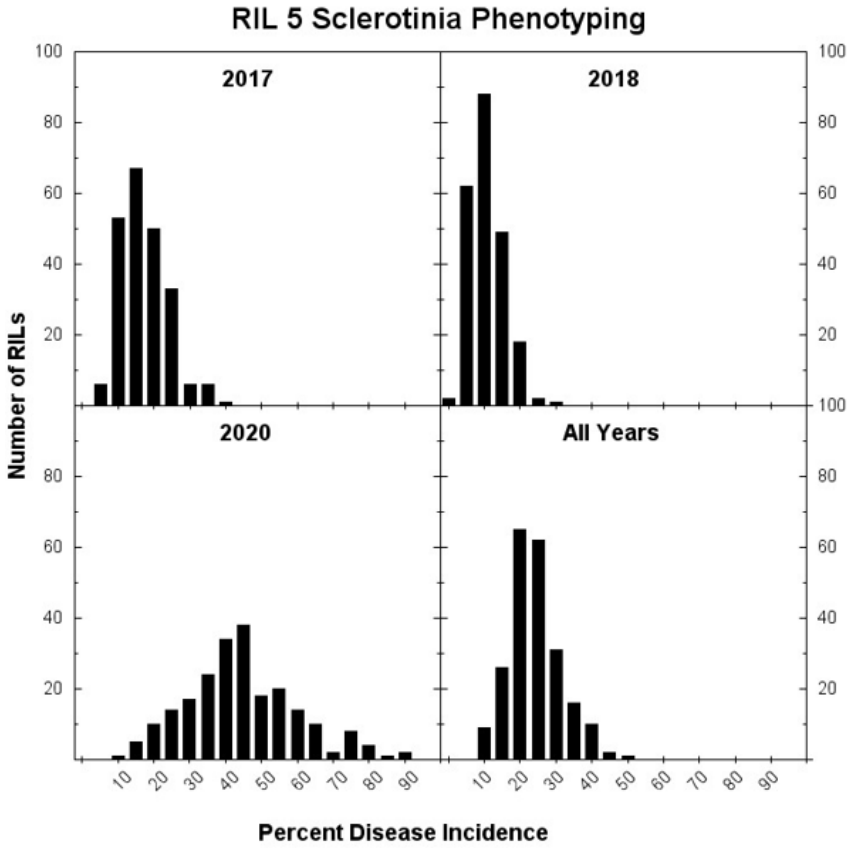

Figure 1. Frequency distribution of Sclerotinia blight incidence in the RIL 5 mapping population in 2017, 2018, and 2020.

Correlation of genotypic and phenotypic data for the mapping population and Khufu™ platform analysis identified two QTLs (Figure 2) associated with Sclerotinia blight resistance: a minor QTL on chromosome 6 (107.5M to 113.5M) and a major QTL on chromosome 15 (41M to 151M). Eight and seven lines were used to generate a resistant

and a susceptible bulk, respectively. Khufu generated QTLseq data for 406,710 SNPs. The data were filtered for a minor allele frequency of 0.25, and only SNPs that showed a minimum depth of 5X in both bulks were used in the final analysis. A sliding window of 75 SNPs was used to reduce the noise. Clear peaks over the confidence interval limit were identified as QTL candidates. To validate the effect of the major QTL on chr15, members of the validation population RIL10 were selected based on genotype (presence or absence of the chr15 QTL) and phenotyped for resistance in 2023.

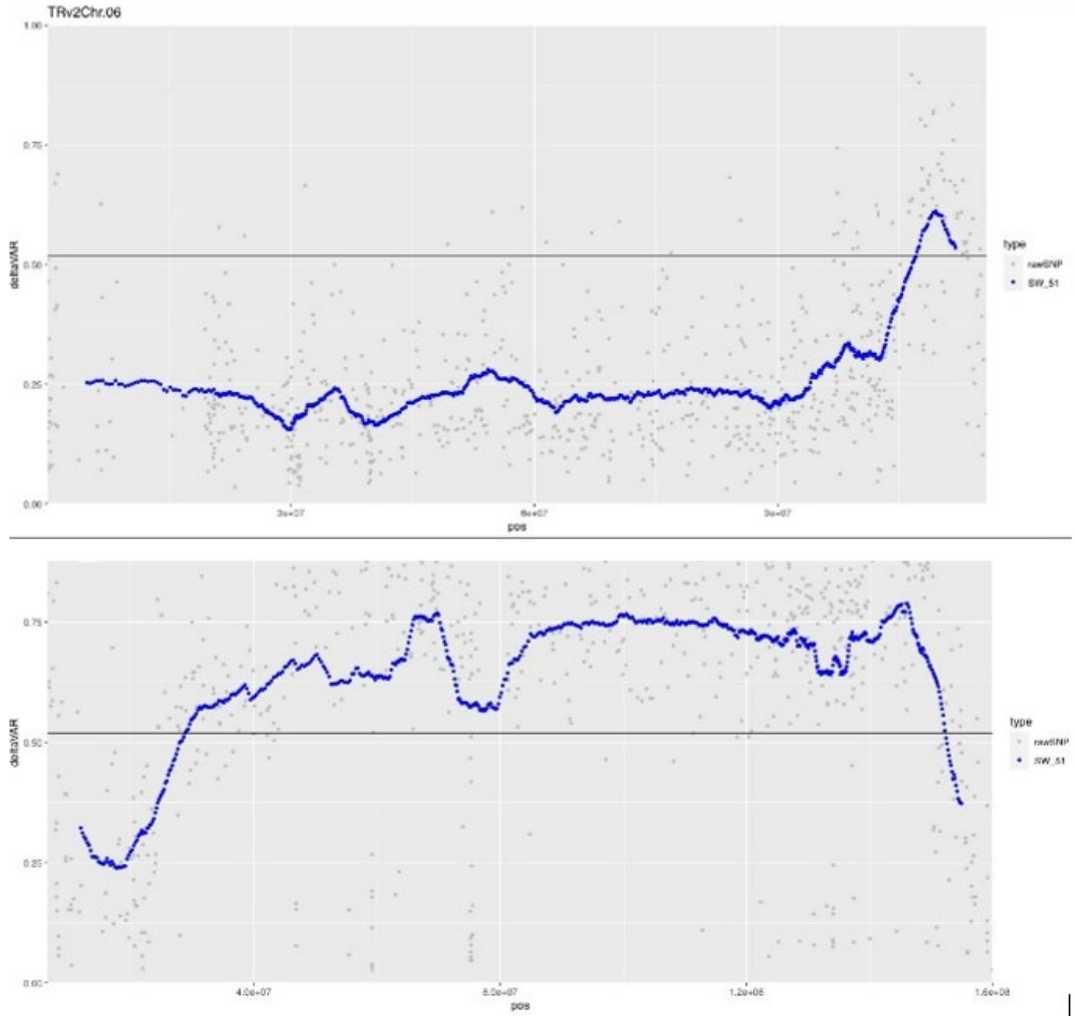

Figure 2. Identified QTL on chromosomes 6 (A) and 15 (B) consistent with resistance to Sclerotinia blight.

Average disease incidence among 21 members of RIL10 ranged from 7% to 67%, where the incidence in the susceptible check Okrun was 76% and the resistant check PI 497429 had an incidence of 6% (Figure 3). Lines that carried the chr15 QTL ranged from 7.1 to 16.2% disease incidence, while those where the QTL was absent had an incidence of 17.5% to 66.7%. The QTL on chr 15 accounted for 52% of the phenotypic variation seen in the validation population. The effect of the chr15 QTL is shown in Figure 4).

This QTL associated with Sclerotinia blight resistance can be used effectively to select for lines with less than 16% disease incidence in materials with PI 497429 in their lineage. The resistance QTL on chromosome 15 is large (~110 Mb). Using the Basic Local Alignment Search Tool (BLAST) to search GenBank databases, 3,211 genes within the region. More research is needed to narrow the region within the chr15 QTL that controls resistance so that Kompetitive Allele-Specific PCR (KASP) markers for selection can be developed.

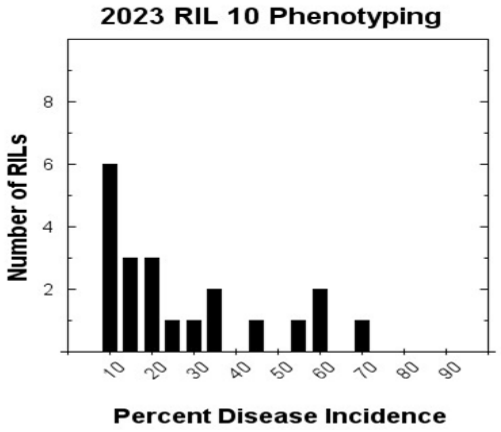

Figure 3. Frequency distribution of Sclerotinia blight incidence in RIL 10 validation lines in 2023.

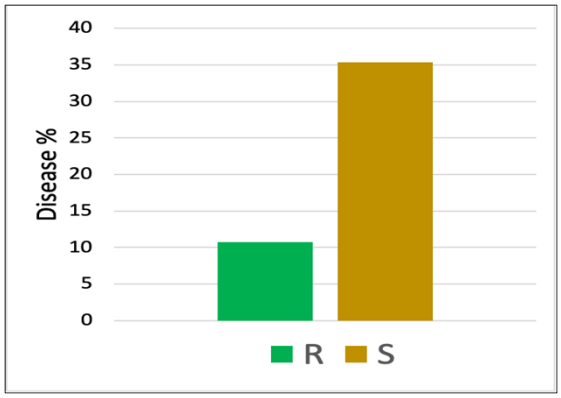

Figure 4. Effect of the chr15 QTL on disease incidence. R=chr15 QTL present (resistant); S=chr15 QTL absent (susceptible).

To date, a limited number of QTLs associated with resistance to Sclerotinia blight have been found (Liang *et al.*, 2021; Rosso *et al.*, 2023). Liang *et al.* (2021) developed and characterized a RIL population from a Tamrun OL07 (Baring *et al.*, 2006; moderately resistant) by TX964117 (susceptible) cross and identified eight novel QTLs associated with resistance, located on chromosomes 4, 8, and 14. Of the 8 QTLs identified, three were attributed to the moderately resistant parent Tamrun OL07 and were responsible for 6.6 - 14.5% of the phenotypic variance observed. Interestingly, five of the QTLs identified (responsible for 6.6-25.6% of the phenotypic variance) were attributed to the susceptible parent TX964117. The authors posed high phenotypic variability across years due to weather and pathogen strain evolution as possible explanations for their results. Another possibility is that Tamrun OL07 is a moderately resistant cultivar (averaging 28% incidence in fields where susceptible cultivars averaged 62% disease) that could display a susceptible phenotype under high disease pressure. Choosing two parents with strongly opposite phenotypes is crucial for developing RIL populations for gene mapping because it ensures there is genetic variation that can be tracked and correlated with the trait of interest. The resistant RIL parent used in this study (PI 497429) is highly resistant to Sclerotinia blight that averages 6% disease in fields where the susceptible parent averaged 76% incidence.

Rosso *et al.* (2023) examined an interspefic RIL population with introgressed regions from three diploid wild species. In that study, individuals resulting from a cross between a resistant artificial amphidiploid JS 1806 and susceptible experimental line JS 17304-7 B (de Blas *et al.*, 2019; de Blas *et al.,* 2021) were genotyped and evaluated for Sclerotinia blight resistance. Their synthetic amphidiploid derived from a cross between *A. cardenasii* (Krapov. & W.C. Greg (KSSc 36,015)) × *A. correntina* ((Burkart) Krapov. & W.C. Greg (K 11905)) × *A. batizocoi* (Krapov. & W.C. Greg (K 9484)) with subsequent duplication of chromosomes. From this work, two large QTLs located on chromosomes 4 and 14 were identified as associated with resistance to *S. minor* and were responsible for 29% and 22% observed phenotypic variance, respectively. Content analysis of the two QTLs showed similarity to defense response genes in other crops. These results support the work of Liang *et al.* (2021) showing that chromosomes 4 and 14 play important roles in Sclerotinia blight resistance. In further work, the same group expanded this work to design KASP markers to select for resistance utilizing single nucleotide polymorphisms (SNPs) found within the QTLs on chromosomes 2 and 8 (Bressano *et al.*, 2025). These markers were used to screen breeding lines and select those with resistant alleles for planting the following season. Selection efficiency of the markers was reported at 92% for identifying Sclerotinia blight resistant materials.

None of the QTLs previously identified as associated with Sclerotinia blight resistance are the same as those identified by this work. The identification of numerous QTLs associated with resistance, including those identified in this study, underscores the complexity of the quantitative inheritance of the resistance trait. The resistance QTL on chromosome 15 is large (~110 Mb), making development of Kompetitive Allele-Specific PCR (KASP) markers for selection and the identification of a candidate gene(s) for resistance difficult. However, using the Khufu genotyping platform which utilizes low-coverage whole genome sequencing and SNP calling, we can easily select for this QTL and reduce the number of breeding lines descended from crosses with PI 497429 with potential for Sclerotinia blight resistance to be tested in the field.

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