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RESEARCH ARTICLE

Identification of QTLs associated with Sclerotinia blight resistance in peanut (*Arachis hypogaea* L.)

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Abstract Sclerotinia blight caused by *Sclerotinia minor* (Jagger) is a significant threat to peanut production; therefore varietal improvement toward this disease is needed. To date, there have been no reported quantitative trait locus (QTL) associated with Sclerotinia blight resistance in peanut. Hence, the objective of this study was to identify QTLs for Sclerotinia blight resistance. A total of 90 $F_{2:6}$ recombinant inbred lines, derived from a released cultivar Tamrun OL07 and a breeding line Tx964117, were used as mapping population and field experiments were conducted in 2010, 2012 and 2018 at the Texas A&M AgriLife Research and Extension Center at Stephenville, Texas. A genetic map was developed using 1211 SNP markers based on double digest restriction-site associated DNA sequencing (ddRAD-Seq). In total, seven QTLs were identified, two QTLs from 2010 and five QTLs from 2018, with LOD score values of 3.2 to 7.2 and explaining 6.6–25.6% phenotypic variance. Among these QTLs, three were detected in common by WinQTLCart and R/qtl. Interestingly, one of the QTLs coincides with a previously reported peanut Leaf spot resistance QTL. The findings from this study not only provide insights into disease resistant QTLs in peanut but can also be used as potential targets for breeding programs to enhance Sclerotinia blight resistance through molecular breeding.

Keywords QTL · Disease · ddRAD-seq · Sclerotinia blight · Peanut (*Arachis hypogea* L.)

Introduction

Peanut (*Arachis hypogaea* L.) accounts for more than $1.6 billion in value in the United States and is also the third most widely grown oilseed crop in the world. Peanut seeds have high nutritional values, with high oil (40-60%) and protein (20–30%) contents (Mallikarjuna and Varshney 2014). A soil-borne fungal disease Sclerotinia blight, caused by *Sclerotinia minor*, is one of the most destructive plant pathogens worldwide. *S. minor* is capable of infecting around one hundred host plants and causes substantial damage and yield reduction of 10% to 75% of various crops such as lettuce, chicory, green bean, sunflower, and peanut (Melzer et al. 1997). *S. minor* was first identified on

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peanut in Australia in 1948, was first reported in the United States in Virginia in 1971, and the first outbreak in Texas was in 1981 (Crutcher et al. 2018; Goldman et al. 1995; Melzer et al. 1997; Phipps 1995). By 1982, Sclerotinia blight became the most important disease of peanut in Virginia (Porter and Melouk 1997) and has continued to spread across the US peanut belt. The first Sclerotinia blight in Arkansas was reported in 2014 on runner peanut (Faske et al. 2014). The strategies used to control Sclerotinia blight on peanut include fungicide spray and canopy pruning (Butzler et al. 1998; Grichar and Woodward 2016). However, both methods can only reduce the rate of disease progress. The more efficient way to mitigate the yield losses caused by this disease is development of disease resistant cultivars.

Peanut is an allotetraploid crop (AABB, 2n = 4x = 40) which hybridized naturally from two diploid wild species, *A. duranesis* (AA) and *A. ipaensis* (BB). The first simple sequence repeat-based (SSR) linkage map of cultivated peanut was developed in the last decade (Varshney et al. 2009) and the genome sequences of the two progenitor species and the cultivated peanut were released in 2016 and 2017, respectively (Bertioli et al. 2016; https://www.ncbi.nlm.nih.gov/bioproject/PRJNA419393/). In addition, recently, there have been several QTL studies for peanut improvement using high-resolution single-nucleotide polymorphisms (SNPs) (Clevenger et al. 2017, 2018; Han et al. 2018; Liang et al. 2017, 2018). However, limited progress has been achieved in peanut compared to other major crops due to its genome complexity and low DNA polymorphism rates (Mallikarjuna and Varshney 2014). QTL mapping has been used as one of the standard methods to identify QTLs in many crops, including rice, wheat and peanut (Buerstmayr et al. 2009; Gomez Selvaraj et al. 2009; McCough and Doerge 1995). Unfortunately, to our knowledge, there have been no whole genome QTL studies reported for Sclerotinia blight-related traits.

Therefore, the major objectives of the present study were to identify QTLs associated with Sclerotinia blight resistance using the RIL population that we used in our previous QTL mapping studies (Liang et al. 2017, 2018). With advances in sequencing technology, SNP detection has become an efficient and convenient method to conduct QTL mapping. We have employed the double digest restriction-site associated DNA sequencing (ddRAD-seq) genotyping method, which has been used to produce genetic maps with high density SNP markers (Baird et al. 2008; Peterson et al. 2012). Field experiments were conducted in Stephenville, Texas, in 2010, 2012, and 2018. Major QTLs detected in this study can be used as potential targets for future molecular breeding efforts to improve Sclerotinia blight resistance in peanut.

Materials and methods

Plant materials

An $F_{2:6}$ RIL population derived from a runner type cultivar Tamrun OL07 and a runner type breeding line Tx964117 were used in this study. Tamrun OL07, a runner type released cultivar, has high yield, good percentage of total sound mature kernels (TSMK), high oleic to linoleic fatty acid ratio (O/L ratio), and has moderate resistance to tomato spotted wilt virus (TSWV) and Sclerotinia blight (Baring et al. 2006). On the other hand, Tx964117, a Texas breeding line, has high level of resistance to both early and late leafspot disease, but with average yield potential, poor percentage of TSMK, low level of resistance to TSWV, and low level of resistance to Sclerotinia blight. The same RILs population had been used in our previous study to identify QTLs for leafspot resistance, yield related, and grade related traits (Liang et al. 2017, 2018).

Experimental design

Field experiments were conducted at the Texas A&M University AgriLife Research Extension Center at Stephenville in 2010, 2012, and 2018. Stephenville is located in Central Texas, has an average of 25.5–35 °C daytime temperature and 7.9 cm precipitation per month from June to November, the growing season in this region. Plots were planted in late June and regular irrigations were performed after planting. In addition, at the end of the season plots were irrigated as needed at dusk to promote fungal growth. The Stephenville nursery is used to conduct Sclerotinia blight disease screening for the Texas A&M AgriLife peanut research program. The field experiments were performed using randomized complete block design (RCBD) with three replications. In

2010 and 2012 each plot consisted of two rows measuring 1.83 m by 3.05 m. The two parents, Tamrun OL07 and Tx964117 were replicated five times as controls in each replication in all 3 years. An extremely susceptible cultivar, Langley, was also planted in the field as a check variety. In 2018, the same design was used except one plot measuring 1.83 m by 3.05 m were planted and only disease ratings were collected.

Inoculation and disease rating

S. minor inoculum collected from the field from the previous season was cultured in a growth chamber at 18–21 °C with 80–95% of humidity and used for the inoculation in the following year. Prior to inoculation, *S. minor* was propagated on sterilized whole oats at18–21 °C with 80–95% of humidity for 21 days until the oats were covered by fungal growth. The infested oats were then ground using a Wiley mill and spread evenly over the plots. The plants were inoculated with *S. minor* approximately 90-100 days of planting as weather permitted. Following inoculation, the plots were irrigated each evening at dusk to promote fungal growth. For disease rating, the symptoms were visually scored using a 0–10 scale, where a score 0 indicates no symptom, while score 10 indicates that the plant is dead. To monitor how the disease was progressing, the plots were scored at two time points, one at approximately 115 and 130 days after planting or as conditions permitted. The second rating was used for the QTL mapping in this study.

Phenotypic data analysis

Phenotypic data across 3 years were analyzed separately. To explore the relationship of disease score rating (DSR) from different years, Pearson correlation test was performed. To examine the entry effect, an ANOVA was conducted to obtain the variance components in RStudio version 3.5.1. Broad-sense heritability was calculated for disease resistance from variance components using the following equation:

$$\boldsymbol{H}^2 = \sigma_{\boldsymbol{g}}^2 \,/\, \left(\sigma_{\boldsymbol{e}}^2 + \sigma_{\boldsymbol{g}}^2\right)$$

where σ_g^2 is the genotypic variance, and σ_e^2 is the error variance.

Best linear unbiased estimator (BLUE) model

Due to the insignificance of entry effect in 2012, a best linear unbiased estimation (BLUE) model was used in 2012 for obtaining a more precise DSR. The BLUE model was defined as follows:

$$\boldsymbol{y} = \boldsymbol{X}\boldsymbol{\beta} + \boldsymbol{\varepsilon}$$

where X was model matrix, the vector y was DSR observed, the vector β was estimated fixed effects, including entry effect and block effect, and ε was a vector with random effects.

DNA collection and genotyping

As previously reported in our study (Liang et al. 2017), a total of 90 RILs along with the two parents were planted in a greenhouse for DNA extraction. The DNA samples were collected from the 3 to 5-week-old peanut unexpanded leaves. A modified cetyltrimethylammonium bromide (CTAB) method was used to obtain high quality of DNA (Doyle and Doyle 1987), where 2% CTAB, 100 mM solid Tris, 700 mM NaCl, 20 mM EDTA, 0.9% sodium bisulfate, 4% polyvinylpyrrolidone (PVP-40) and 0.5% β-mercaptoethanol were used.

Genotyping was performed using the restriction site association-based method, ddRAD-seq (Peterson et al. 2012), digesting with restriction enzymes *PstI* and *MluCI*. The library preparation and sequencing were performed at the Genomic and Bioinformatics Service, Texas A&M AgriLife Research. A total of 260,445,423 raw sequencing reads were processed, 17,341 SNPs were called, and 1211 SNPs were finally selected to construct the genetic map (Liang et al. 2017).

Genetic map construction and QTL analysis

The construction of the genetic map was performed using MSTMAP online software (Wu et al. 2008) as reported in our previous study (Liang et al. 2017). Briefly, the Kosambi mapping function was used for estimating map distance based on recombinant frequency. A "no mapping distance threshold" was set at 15 cM, and "no mapping size threshold" was set at 2, as the default setting. This genetic distance was used for both QTL mapping tools.

Windows QTL cartographer 2.5 software (Wang et al. 2012) and R/qtl (Broman et al. 2003) were used to perform the QTL analysis. In Windows QTL cartographer 2.5, composite interval mapping (CIM) was performed using Kosambi map function with Ri1 cross type (recombinant inbred line, derived by selfing), and forward and backward regression method was used with F-in and F-out equal to 0.01 selection criteria. In R/qtl, analysis was performed by *cim()* function with Kosambi map function, 0.001 error probability, and other default settings. For both QTL analysis, five markers were set for background control with window size of 10 cM. A logarithm of odds (LOD) value of 3.0 was used as the significance threshold to declare the QTL. For QTLs detected in more than 1 year, they were called with LOD value above 3.0. A 1000 time-permutation test was performed in both QTL mapping software packages to obtain a more rigorous LOD threshold.

For convenience of the analysis, ten chromosomes on *A. ipaensis* (B) genome were named as chromosome eleven to twenty according to their order in the B genome. For example, chromosome 1 in the B genome is noted as chromosome 11 in this study.

Results

Disease performance across years

ANOVA revealed that there was a significant year effect across 2010, 2012, and 2018, with $P \leq 0.001$. Therefore, all phenotypic data were analyzed separately. The distribution of DSR had a high variance in 3 years. In 2010, 2012, and 2018, the DSR ranged from 0 to 6.0, 0 to 0.90, and 1.0 to 9.0, with an average of 2.6, 4.5 and 6.4, respectively, while heritability also varied from 0.29 to 0.45 (Table 1). The average DSRs for the susceptible parent Tx964117 were 2.8, 4.4, and 7.6, in 2010, 2012, and 2018, respectively. On the other hand, the moderate resistant parent Tamrun OL07 had average DSRs of 1.3, 3.4, and 7.3, in 2010, 2012, and 2018, respectively (Fig. 1). Although the resistance between the two parents was not quite distinct, the moderate resistant parent still had lower DSR than the susceptible parent Tx964117. The average yield was 1577 g per two-row plot in 2010 with a range from 590 g to 2407 g. In 2012 and 2018 only disease ratings were collected with no harvest data being collected. In addition, since there was no significant effect of the entry effect in ANOVA from the 2012 data, this phenotypic data was not used for QTL detection.

Table 1 The average disease score (DSR), standard deviation (SD), coefficient of variance (CV) and broad-sense heritability (H^2) for Sclerotinia disease resistance in the RIL mapping population

Years	Range	Mean	SD	CV (%)	H^2
2010	0–6.0	2.6	1.54	58.6	0.31
2012	0–9.0	4.5	2.33	52.1	–[a]
October 2018	1.0–5.0	2.4	0.86	35.3	0.29
November 2018	1.0–9.0	6.4	1.90	29.6	0.45

[a]The entry effect in 2012 was not significant; therefore, the heritability was not shown

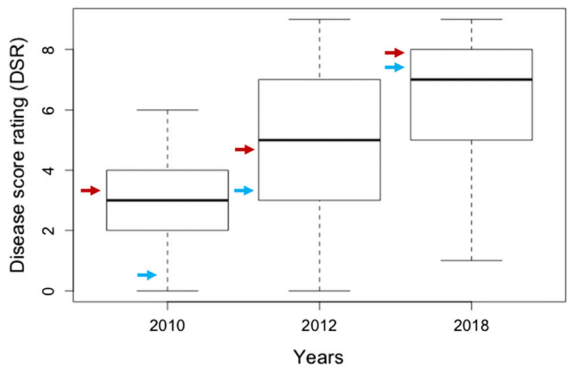

Fig. 1 The boxplots of disease score rating (DSR) of the RIL population in 2010, 2012, and 2018. The red arrow indicates DSR of the susceptible parent Tx964117, and the blue arrow indicates DSR of the moderate resistant parent Tamrun OL07

The Pearson correlation coefficients showed significant positive correlation in all 3 years (Fig. 2). We further examined the correlation between the first disease rating and the second rating in 2018. With one month apart, disease rating from the two time points had a significant correlation of 0.66. The DSR in the first rating had a mean of 2.4 with a range of 1.0 to 5.0, while the second rating had a mean DSR of 6.4 with a range of 1.0 to 9.0 (Table 1). This suggests that the disease progressed significantly in a short period of time.

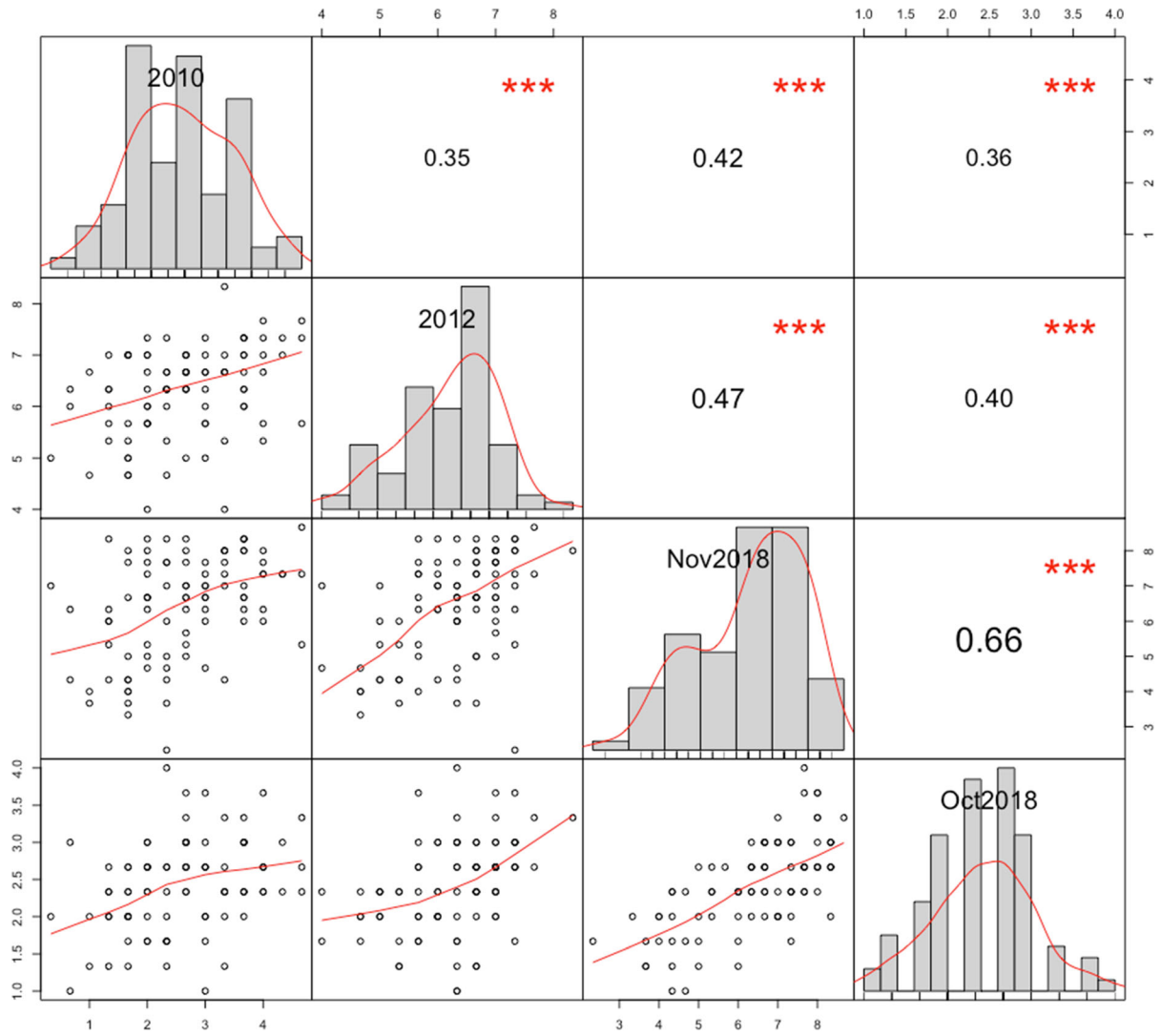

Fig. 2 Phenotypic performance and Pearson correlation coefficients among disease score rating (DSR) from different years. Nov2018 indicated the second disease rating in 2018, and Oct2018 indicated the first rating in 2018."***" for p value < 0.0001 according to Pearson correlation test

Genetic map and QTL analysis

A total of eight QTLs for Sclerotinia blight resistance were detected across 3 years (Table 2). Among these QTLs, three of them were detected by both software packages, WinQTLCart and R/qtl. And two QTLs were detected in more than 1 year. In 2010, two QTLs were detected by both WinQTLCart and R/qtl, i.e. *qS4* and *qS14*, with LOD values of 4.4 and 4.7, and R^2 of 14.5% and 17.3% from WinQTLCart; and LOD values of 4.4 and 5.3, and R^2 of 6.6% and 11.6% from R/qtl (Table 2). *qS4* was derived from the elite cultivar Tamrun OL07; on the other hand, the susceptible breeding line Tx964117 was the donor of *qS14*. There were two QTLs detected in 2012, *qS8.1* and *qS14*. Notably, the two QTLs identified in 2012 were also detected in other years. *qS14* was detected in both 2010 and 2012 and by both QTL detecting software; while *qS8.1* was detected in 2012 by R/qtl, and detected in 2018 by WinQTLCart. Six QTLs were identified in 2018; four of them were detected by WinQTLCart only, i.e. *qS1, qS2, qS8.1* and *qS20.1*, one QTL was only identified by R/qtl (*qS20.2*), and another one was detected by both WinQTLCart and

Table 2 QTLs identified for disease score rating (DSR) of Sclerotinia blight in the RIL mapping population in 2010, 2012, and 2018

Year	QTL	Chr.	Peak (cM)	Closest marker	Good Allele[a]	WinQTLCart.			R/qtl		
						LOD	Add.[b]	R^b (%)	LOD	Add.	R^b(%)
2010	*qS4*	4	431	Aradu.A04_121130432	A	4.4*	0.4	14.5	4.4	0.27	6.6
	qS14	14	82.2	Araip.B04_132742316	B	4.7*	0.43	17.3	5.3	0.36	11.6
2012	*qS8.1*	8	166.4	Aradu.A08_4286171	B				4.0	0.21	6.6
	qS14	14	82.2	Araip.B04_132742316	B	3.2	0.28	12.1	4.1	0.26	10.8
2018	*qS1*	1	200.9	Aradu.A01_3241880	A	3.7	0.78	12.0			
	qS2	2	39.0	Aradu.A02_89841822	B	3.4	1.33	9.2			
	qS8.1	8	166.4	Aradu.A08_4286171	B	3.9	0.67	13.0			
	qS8.2	8	174.9	Aradu.A08_4286139	B	5.0	0.79	15.9	7.2*	0.71	18.3
	qS20.1	20	32.3	Araip.B10_119489004	B	3.8	0.89	25.6			
	qS20.2	20	53	Araip.B10_57358933	A				3.2	0.58	11.0

[a]Allele A indicates the allele was from Tamrun OL07, while allele B was from Tx964117. Good allele means the allele reduced DSR.
[b] "Add." indicates additive effect of QTLs. "*" for p-value < 0.05 according to 1000 times permutation threshold

R/qtl (*qS8.2*). There were QTLs located on chromosome 8 as well as chromosome 20 in a very close distance, however, they were identified as two QTLs due the non-overlapped QTL interval and the opposite allelic effect (*qS20.2*). *qS1* and *qS20.2* were from the elite parent Tamrun OL07, with LOD values of 3.7 and 3.2, and R^2 of 12.0% and 11.0%, respectively. On the other hand, *qS2, qS8.1,* and *qS20.1* were from the susceptible parent Tx964117, with LOD values of 3.4, 3.9, and 3.8, and R^2 of 9.2%, 13%, and 25.6%, respectively. Interestingly, the DSR decreased allele of the largest QTL *qS8.2* also came from Tx964117, with LOD values of 5.0 and 7.2, and R-square of 15.9% and 18.3%, from WinQTLCart and R/qtl, respectively.

Discussion

Phenotype variation

As shown in Table 1, the phenotypic variability in our experiments was relatively high, especially in 2010 and 2012. This may be due to the experimental conditions in Stephenville. These experiments were conducted under field conditions instead of a well-controlled greenhouse. Therefore, the environmental effects such as temperature, humidity, and rainfall had a large effect on plant growth and disease establishment and progress. For example, in 2018, there was an extended cool, rainy period that accelerated the disease progression; on the other hand, 2010 was a dry year with few precipitations during the growing season that restrained the overspread of *S. minor*. We also noticed that the possibility of new race(s) existed in the field in 2018. The disease performance showed that both resistant and susceptible parents showed a susceptible response in 2018 (Fig. 1). A further pathogen genomics study is required to validate the existence of the new race(s). Moreover, box plots of the three different experiments demonstrated that the position of the plots in the experimental field also had a significant effect on disease severity. Among the three blocks used in the experiment, block #3 always had the lowest DSR, whereas block #2 always had the highest DSR (Fig. 3). This phenomenon is consistent with the results from ANOVA that block effects were significant in all 3 years. Overall, these factors might partially explain the high phenotypic variability in this study and a rather low correlation coefficient value between the first and second disease ratings in 2018.

Our previous study compared the yield between an ideal peanut production environment (without disease) and a Sclerotinia blight screening field (Liang et al. 2018). The results showed that Sclerotinia blight could cause a 45% yield loss, and the amount of yield loss highly depends on the disease severity. Although the yield difference could also be due to other environmental effects between the two locations, the major factor that caused the yield loss was the damage

to the plant. Based on the results from our experiments in 2012 and 2018, Sclerotinia blight has caused nearly total yield loss by the death of nearly all the plants. A huge loss of peanut production due to Sclerotinia blight has been previously reported (Chenault et al. 2006; Porter and Melouk 1997).

QTLs identified in this study

A total of eight QTLs associated with Sclerotinia blight resistance were detected in this study (Table 2). Three QTLs on chromosome A4, B14, and A8 (i.e., *qS4*, *qS14*, and *qS8.2*) had very high LOD scores, passed threshold after permutation, and also were detected by both WinQTLCart and R/qtl. Two of three large QTLs were derived from the low tolerance parent Tx964117. Interestingly, two QTLs were detected in multiple years were also derived from Tx964117, including *qS14,* which was identified in 2010 and 2012, and *qS8.1*, which was identified in both 2012 and 2018. One possible reason could be that there were some other race(s) existed in the field that Tx964117 has better tolerance instead. The moderate tolerance parent Tamrun OL07 was bred and evaluated for the Sclerotinia blight resistance for more than a decade (Baring et al. 2006); therefore, along with the selection of pathogen and the existence of new race(s), the resistance may not as good as before. This factor could have significant impacts on phenotypic variability and may reduce the power of QTL detection. On the other hand, this phenomenon could also due to the interaction with the genetic background, which is not uncommon in QTL studies for various traits in different crops. For example, a large QTL for increased yield in rice under drought conditions derived from the susceptible parent has been previously reported (Bernier et al. 2007). This QTL has been transferred through marker-assisted selection to some elite varieties (Dixit et al. 2017; Henry et al. 2014). This may partially explain why many progenies had better disease resistance than the donor parent. However, this hypothesis also requires further investigation.

To our knowledge, only one molecular marker has been reported associated with Sclerotinia blight resistance in peanut thus far (Chenault et al. 2008). The sequences of this SSR marker pair are pPGPseq 2E6R (5'-CCTGGGCTGGGGTATTATTT-3') and pPGPseq 2E6L (5'-TACAGCATTGCCTTCTGGTG-3'). This marker has been used in several studies, such as the evaluation of the U.S. peanut mini core collection (Chenault et al. 2009). New sources for Sclerotinia resistance have also been identified (Bennett et al. 2018; Yol et al. 2014). A BLAST search has been performed to identify the position of this SSR marker using the full length of pPGPseq 2E6 sequence (Ferguson et al. 2004). The sequence has a hit on chromosome A07 of cultivar peanut Shitouqi with 98% identities and 83% coverage. Therefore, all eight QTLs identified in this study are novel QTLs.

Interestingly, a previous reported peanut leaf spot disease resistance QTL *qLS14.1* had the same marker peak as *qS14*, which was detected in this study (Liang et al. 2017). The beneficial allele of both *qS14* and *qLS14.1* are from Tx964117. Leaf spot is a foliar fungal disease caused by *Cercospora arachidicola* (early leafspot) or *Cercosporidium persoatum* (late leafspot); therefore, *qS14/qLS14.1* could be a good target QTL for future marker-assisted breeding programs. Previously, a peanut transgenic study indicated that transgenic peanut lines with antifungal genes, such as rice chitinase and alfalfa glucanase, had partial resistance to Sclerotinia blight (Chenault et al. 2005). Other than fungal resistant genes, a previous study also showed that transferring barley oxalate oxidase may also improve the resistance of peanut to Sclerotinia blight (Chenault et al. 2005; Hu et al. 2015).

Sclerotinia blight had appeared in the United States since 1971 and was first identified in Texas in 1981 (Crutcher et al. 2018; Goldman et al. 1995). However, no disease resistance QTLs have been identified in the past decade. Most of the current disease resistant studies have focused on large-effect QTLs and mainly were qualitative loci; on the contrary, quantitative resistance is less known (Corwin and Kliebenstein 2017). Many large-effect R genes correspond to their specific pathogen and activate a rapid defense response such as programmed cell death, an effective defense mechanism against biotrophic pathogens. However, the gene-for-gene response would not be observed in the interaction with necrotrophic pathogens such as *S. minor* (Glazebrook 2005), since necrotrophic pathogens indeed benefit from host cell death. The defense mechanisms against necrotrophs are usually controlled by hundreds of causal genes, such as the strengthening of the cell wall, biosynthesis of defense compounds, and the interaction between different mechanisms such as the JA signaling

pathway and camalexin production (Corwin and Kliebenstein 2017; Glazebrook 2005). These might explain why there were no QTL studies of Sclerotinia blight being previously reported. Our study provides the first QTL identification for Sclerotinia blight resistance using high-resolution SNPs markers. Some of these QTLs can be used as potential targets for varietal improvement.

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Compliance with ethical standards

The authors declare that they have no conflict of interest.

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