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# Genetic Mapping and Quantitative Trait Loci Analysis for Disease Resistance Using $F_2$ and $F_5$ Generation-based Genetic Maps Derived from 'Tifrunner' × 'GT-C20' in Peanut

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## Abstract

One mapping population derived from Tifrunner × GT-C20 has shown great potential in developing a high density genetic map and identifying quantitative trait loci (QTL) for important disease resistance, tomato spotted wilt virus (TSWV) and leaf spot (LS). Both $F_2$ and $F_5$ generation-based genetic maps were previously constructed with 318 and 239 marker loci, respectively. Higher map density could be achieved with the $F_2$ map (5.3 cM per locus) as compared to the $F_5$ (5.7 cM per locus). Quantitative trait loci analysis using multi-environment phenotyping data from $F_8$ and higher generations for disease resistance identified 54 QTL in the $F_2$ map including two QTL for thrips (12.14–19.43% phenotypic variation explained [PVE]), 15 for TSWV (4.40–34.92% PVE), and 37 for LS (6.61–27.35% PVE). Twenty-three QTL could be identified in the $F_5$ map including one QTL for thrips (5.86% PVE), nine for TSWV (5.20–14.14% PVE), and 13 for LS (5.95–21.45% PVE). Consistent QTL identified in each map have shown higher phenotypic variance than nonconsistent QTL. As expected, the number of QTL and their estimates of phenotypic variance were lower in the $F_5$ map. This is the first QTL study reporting novel QTL for thrips, TSWV, and LS in peanut (*Arachis hypogaea* L.), and therefore, future studies will be conducted to refine these QTL.

PEANUT has its global presence among growers and consumers with a total production of 37.7 million t from 24.1 million ha in 2010 (FAO, 2012). The average yield was 1564 kg ha$^{-1}$, and a wide gap exists between the genetic potential of the modern cultivars and their actual yield in the farmer's field. This gap has been heavily widened by several biotic and abiotic stress factors in the past and it may be even worse at the current scenario due to the fluctuating climatic and environmental conditions. Among the biotic stresses, early leaf spot (ELS) (caused by *Cercospora arachidicola*), late leaf spot (LLS) (caused by *Cercosporidium personatum*), and tomato spotted wilt virus (TSWV) may cause significant yield loss (Nigam et al., 2012). Tomato spotted wilt virus is generally spread by thrips and the farmers try to control TSWV indirectly with insecticide spray. In 1997 and 1998, losses due to TSWV peanut were estimated at approximately US$40 million per year for Georgia alone in the United States (Culbreath et al., 2008). Despite several chemical treatments available to control these diseases, host-plant

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Abbreviations: CIM, composite interval mapping; ELS, early leaf spot; LG, linkage group; LLS, late leaf spot; LS, leaf spot; PV, phenotypic variance; PVE, phenotypic variation explained; QTL, quantitative trait loci; RIL, recombinant inbred line; SSR, simple sequence repeat; TSWV, tomato spotted wilt virus.

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resistance is the best control mechanism, which has the advantage of being cost effective and eco-friendly.

Conventional breeding has been the major force in providing modern cultivars to the farmers. Integration of genomics tools with conventional breeding has been successful in some of the crops but peanut lagged behind in terms of genetic and genomic resources required for such approach. However, the development in genetic and genomic resources in peanut in recent years has provided the possibility for improving peanut through marker-assisted selection to lead to the more rapid development of superior cultivars using informative markers linked to desired traits. Although marker-assisted breeding has been applied on a limited scale (see Pandey et al., 2012), peanut still lacks availability of linked markers for important traits. Already marker-assisted breeding in peanut has successfully demonstrated its utility by using available limited resources in conversion of peanut cultivar Tifguard (Holbrook et al., 2008) into 'high oleic Tifguard' in 26 mo (Chu et al., 2011).

Identification of linked markers is the base to improve peanut resistance for the important diseases through marker-assisted breeding, and a mapping population derived from the cross Tifrunner × GT-C20 was developed for identification of linked markers. The parental genotypes have several contrasting traits such as Tifrunner with high level of resistance to TSWV and moderate resistance to ELS and LLS (Holbrook and Culbreath, 2007) while GT-C20 is susceptible to these diseases but has resistance to aflatoxin contamination (Liang et al., 2005). Parental screening with approximately 5000 simple sequence repeat (SSR) markers resulted in identification of 385 polymorphic loci, which were genotyped on a set of 94 individuals of the $F_2$ population. As a result, a genetic linkage map was constructed with 318 mapped loci distributed on 21 linkage groups (LGs) with genome coverage of 1674.4 cM and a marker density of 5.3 cM per locus (Wang et al., 2012). Meanwhile, this population was advanced to the $F_5$ generation and used for development of another genetic map with 239 loci distributed on 26 LGs covering a total genome distance of 1213.4 cM and average map density of 5.7 cM per locus (Qin et al., 2012). This population was then extensively phenotyped during the years for several important traits including three important diseases.

Therefore, this study reports the use of genotyping data generated at the $F_2$ and $F_5$ generation and phenotyping data generated at higher generations for identification of quantitative trait loci (QTL) for thrips, TSWV, and leaf spot (LS) including ELS and LLS in this study. The field phenotyping trials were conducted in multiple fields from 2010 to 2012. Late leaf spot was predominate pathogen in all 3 yr. Also, comparison was made for the effects of identified QTL and common genomic regions identified in the $F_2$ and $F_5$ maps.

# MATERIALS AND METHODS

## Mapping Population

A mapping population derived from the cross Tifrunner × GT-C20 was developed through single seed descent method at the Crop Protection and Management Research Unit of USDA-ARS, Tifton, GA. The female parent, Tifrunner, is a runner market-type cultivar with high level of resistance to TSWV and moderate resistance to ELS and LLS and late maturity (Holbrook and Culbreath, 2007). The male parent, GT-C20, is a Spanish-type breeding line with high susceptibility to TSWV and LS but resistance to aflatoxin contamination (Liang et al., 2005). As of now, this mapping population consists of 248 recombinant inbred lines (RILs) and has been phenotyped for several agronomic traits including disease resistance.

## Deoxyribonucleic Acid Isolation, Polymorphism, and Genotyping

Initially the total genomic DNA was extracted from young leaflets of 94 $F_2$ plants along with the parental genotypes (Tifrunner and GT-C20). Parental polymorphism screening and population genotyping were conducted with SSR markers available at University of California, Davis, CA, and Tuskegee University, Tuskegee, AL. Simultaneously, the generation advancement was done from the $F_2$ to $F_5$ generation and again the DNA was isolated from a subset of 158 $F_5$ individuals to construct a genetic map and use multiseason phenotyping data for QTL analysis. The details of polymerase chain reactions and complete genotyping and map construction were published early for the $F_2$ map (Wang et al., 2012) and $F_5$ map (Qin et al., 2012).

## Phenotyping for Disease Resistance

The entire set of RILs with 248 individuals were phenotyped for several important traits including resistance to thrips, TSWV, and LS including both ELS and LLS but LLS was the predominant disease in all 3 yr. Therefore, the general term of LS was used in this study. The field trials were conducted using randomized complete block designs with at least three replications in 2010 at Dawson and Tifton, GA, and 2011 and 2012 at Tifton, GA. Late LS was predominate pathogen in all 3 yr.

In Tifton, GA, two separate field trials were conducted at the Belflower Farm in all 3 yr. Soil type is Tifton loamy sand (fine-loamy, siliceous, thermic Plinthic Kandiudult). In each year, one experiment was planted in April to maximize potential for development of spotted wilt epidemics (Li et al., 2012) and one was planted in May to reduce potential for spotted wilt epidemics and increase the likelihood of heavy LS epidemics. Experiment plots were 6.0 m long, separated by 2.4 m alleys. Peanut seeds were planted in 91-cm-spaced twin-row plots.

Severity of TSWV was assessed using a 0 to 5 severity scale adapted from Baldessari (2008) based on visual determination of presence of symptoms and estimation

of the degree of stunting (reduction in plant height, width, or both) for symptomatic plants. Leaf spot severity was evaluated using the Florida 1 to 10 scale (Chiteka et al., 1988) in which 1 indicates no LS, 2 indicates very few lesions on the leaves and none on upper canopy, 3 indicates very few lesions on upper canopy, 4 indicates some lesions with more on upper canopy with 5% defoliation, 5 indicates noticeable lesions on upper canopy with 20% defoliation, 6 indicates numerous lesions on upper canopy with 50% significant defoliation, 7 indicates numerous lesions on upper canopy with 75% defoliation, 8 indicates upper canopy covered with lesions with 90% defoliation, 9 indicates very few leaves covered with lesions remain and some plants completely defoliated, and 10 indicates plants dead.

This population was phenotyped for thrips (TPS) for one season at Dawson (DW) in 2010 (10) (TPS_DW10) and TSWV for four seasons (E1 through E4), that is, at Dawson in 2010 (TSWV_DW10E1), at two locations of Tifton (TF) in 2010 (TSWV_TF10E2 and TSWV_TF10E3), and at Tifton in 2011 (11) (TSWV_TF11E4). This population was screened for LS for a total of 10 seasons (E1 through E10), which include screening at Dawson (DW) in 2010 (10) (LS_DW10E1 and LS_DW10E2) and at Tifton (TF) in 2010 (LS_TF10E3) and in 2011 (11) (LS_TF11E4, LS_TF11E5, LS_TF11E6, and LS_TF11E7) and three in 2012 (12) (LS_TF12E8, LS_TF12E9, and LS_TF12E10).

### Nomenclature Uniformity between Genetic Maps

The genetic maps were constructed at two different institutions using the two different generations of the same cross, that is, using 94 $F_2$ individuals at Tuskegee University and 158 $F_5$ individuals at USDA-ARS (Tifton), and published in the same year, that is, late 2012 (Wang et al., 2012) and early 2012 (Qin et al., 2012), respectively. The panel of markers screened on parental genotypes was different; hence, some differences in nomenclature used for names of markers were found. This was more frequent with the naming of unpublished markers having long identifications, for example, the markers developed through bacterial artificial chromosome -end sequencing were named with prefix "ARS" in the $F_5$ map and with prefix "GNB" in the $F_2$ map. Here we retained the names as such for all the published markers used in these two maps while few changes were made to keep size of names manageable and better viewing such as "pPGP…" and "sPGP…" were abbreviated to "seq…" to bring uniformity with recently published high dense consensus genetic maps (Gautami et al., 2012; Shirasawa et al., 2013). The purpose of all the above exercise was to bring the genetic information in uniformity, which has helped in comparison of genetic maps between each other and also with published consensus genetic map. It is important to mention that the genetic map information generated using the $F_5$ population (Qin et al., 2012) was used for construction of both the consensus genetic maps (Gautami et al., 2012; Shirasawa et al., 2013) while the $F_2$ genetic map could not be completed due to delay in screening large number of markers and genotyping.

### Reproducing Genetic Maps and Quantitative Trait Loci Analysis

The method of genetic map construction for both maps was given in detail by Qin et al. (2012) and Wang et al. (2012). Here we made the nomenclature of both the genetic maps uniform in consensus with the published consensus genetic maps (Gautami et al., 2012; Shirasawa et al., 2013) where distinct LGs have been assigned to particular genomes. MapChart 2.2 (Voorrips, 2002) was used for reproducing both the genetic maps using uniform nomenclature with the genetic map information. The genotyping information generated on both the generations ($F_2$ and $F_5$) was also used here for conducting QTL analysis using software WinQTL Cartographer, version 2.5 (Wang et al., 2007). The composite interval mapping (CIM) approach, which is based on a mixed linear model, was used for detection of QTL with LG more than 2.5. Parameters such as model 6, scanning intervals of 1.0 cM between markers, and putative QTL with a window size of 10.0 cM were used for conducting the CIM analysis.

## RESULTS

### Comparison of Both Genetic Maps with Reference Consensus Genetic Map

Upon the comparison of the corresponding LGs between these two ($F_2$ and $F_5$) maps, 19 LGs of the $F_2$ map were found identical to 20 LGs of the $F_5$ map (Supplemental Table S1). Of the total 22 LGs of the $F_2$ map and 26 LGs of the $F_5$ map, three LGs (AhIII, AhXXI, and AhXXII) and six LGs (LGT1, LGT12, LGT19, LGT22, LGT23, and LGT26) could not correspond to each other due to less number of mapped loci as well as lack of common loci, respectively. Two LGs of the $F_5$ map (LGT15 and LGT25) shared common loci with one LG (AhVIII) of the $F_2$ map. Upon comparing these two genetic maps with reference consensus genetic maps using the common marker loci, a total of 9 of the 10 LGs from A genome and 8 of the 10 LGs from B genome could be assigned. In general the co-linearity has been observed for these two maps with each other and also with the reference consensus genetic map (Fig. 1).

### Quantitative Trait Loci Analysis for Biotic Stresses

The entire RILs with 248 individuals were phenotyped for thrips, TSWV, and LS in multiple fields and planting dates from 2010 to 2012 in Georgia. Late LS was predominate pathogen in all 3 yr. Therefore, the general term of LS was used in this study, including both ELS and LLS. These phenotyping data were used in combination with genotyping data based on the $F_2$ and $F_5$ generation for identification of QTL associated with each trait. A total of 77 QTL could be detected for these three diseases using both the genetic maps. Of the 77 QTL, 54 QTL (two for thrips, 15 for TSWV, and 37 for LS) were placed on the $F_2$ map (Supplemental Table S2; Fig. 2) and 23 QTL (one for thrips, nine for TSWV, and 13 for LS) on the $F_5$ map (Supplemental Table S3; Fig. 3)

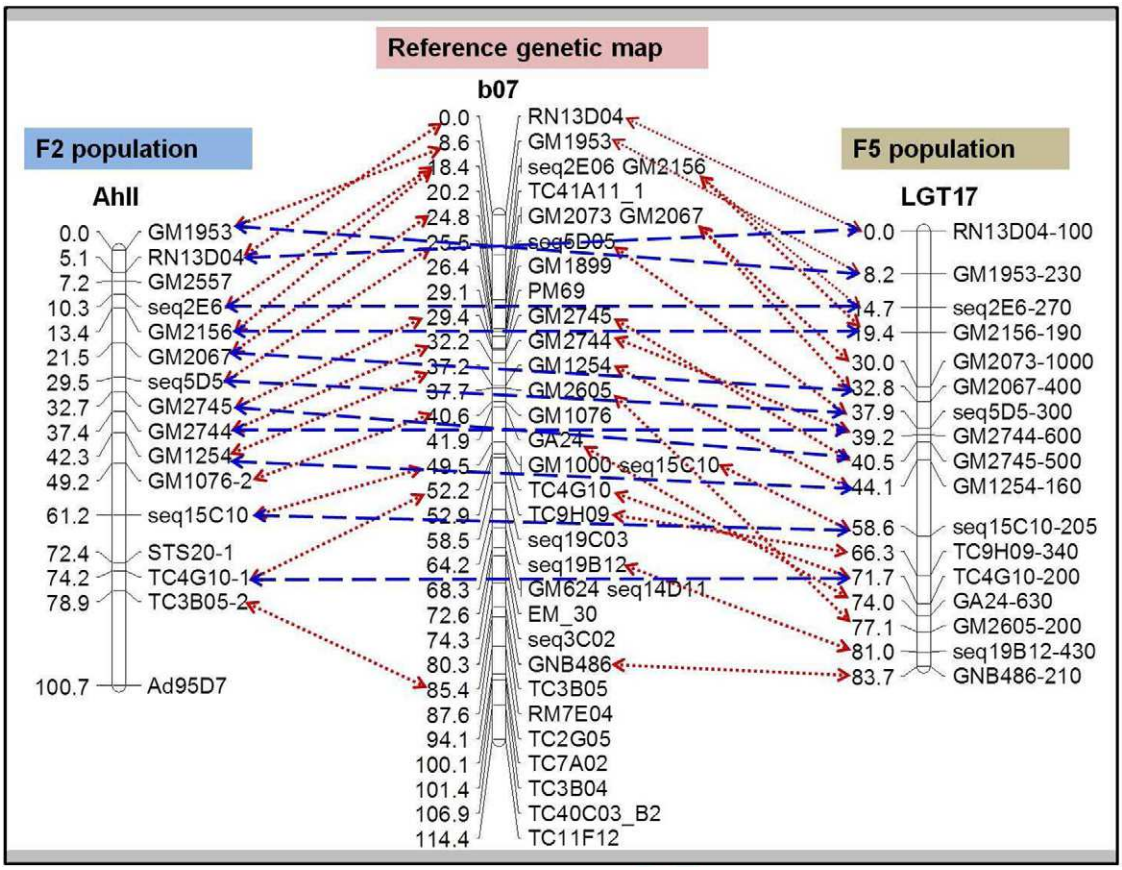


Figure 1. Co-linearity between two genetic maps of the Tifrunner × GT-C20 population and reference consensus genetic map.

with phenotypic variance (PV) range of 5.86 to 19.43% (thrips), 4.40 to 34.92% (TSWV), and 5.20 to 21.45% (LS), respectively (Table 1). The log-of-odds values ranged from 2.51 (TSWV and LS) to 5.92 (TSWV) in the $F_2$ map and 2.50 (TSWV) to 6.38 (LS) in the $F_5$ map.

## Quantitative Trait Loci Identified for Thrips

Total three QTL could be identified for thrips using genetic mapping information of both the populations and phenotyping data generated for one season at Dawson during 2010. Of the three QTL, two QTL (*qF2TPS1* and *qF2TPS2*) were detected on the $F_2$ map with PV ranging from 12.14 to 19.43% and only one QTL (*qF5TPS1*) with 5.86% PV on the $F_5$ map. Among three QTL, the *qF2TPS1* (IPAHM108-2–AHGS0347) located on AhIX and *qF2TPS2* (GM2337–TC42A02) located on AhX are the two major QTL detected for thrips with 12.14 and 19.43% PV, respectively (Table 2).

## Quantitative Trait Loci Identified for Tomato Spotted Wilt Virus

In the case of TSWV, a total of 24 QTL were detected, which include 15 QTL from the $F_2$ and nine QTL from the $F_5$ map with PV ranging from 4.40 to 34.92% and 5.20 to 14.14%, respectively (Table 1). All the 15 QTL detected in the $F_2$ map were located on eight genomic regions of six LGs (AhI, AhII, AhIX, AhX, AhXI, and AhXII) (Table 2). The same names were given to all the QTL if they were mapped with same genomic regions or marker interval. So in this case, 15 QTL were mapped on eight genomic regions as *qF2TSWV1* to *qF2TSWV8* without referring to any season (Supplemental Table S2). The three genomic regions named seq5D5 to GM2744 (*qF2TSWV3*) on AhII, TC42A02 to GM2337 (*qF2TSWV6*) on AhX, and GNB2 to AHO116 (*qF2TSWV8*) on AhXII harbored three QTL while another genomic region named IPAHM108-2 to AHGS0347 (*qF2TSWV4*) on AhIX possessed two QTL and these four genomic regions are referred as consistent QTL across two or more different environments. The PVs shown by consistent QTL were higher in general as compared to the nonconsistent QTL (which appeared in only one environment). Among four consistent QTL, *qF2TSWV3* had higher PV range (5.14–34.92%) followed by *qF2TSWV8* (6.26–21.18% PV), *qF2TSWV4* (12.92–18.11% PV), and *qF2TSWV6* (10.78–16.56% PV) (Table 2). Among nonconsistent QTL, *qF2TSWV5* had the highest PV (23.02%) followed by *qF2TSWV7* (15.75%), *qF2TSWV1* (9.41%), and *qF2TSWV2* (4.40%).

Similarly, all the nine QTL (5.20–14.14% PV) identified in the $F_5$ map were located on seven genomic regions on seven different LGs named LGT1 (TC3H02-410–seq14A7-300), LGT6 (TC11A02-300–GNB523-500), LGT7 (GNB519-205–GM1076-460), LGT9 (AC3C07-70–RN35H04-1500), LGT11 (GNB619-340–GM2607-90), LGT12 (seq14G03-500–GM2808-400), and LGT25

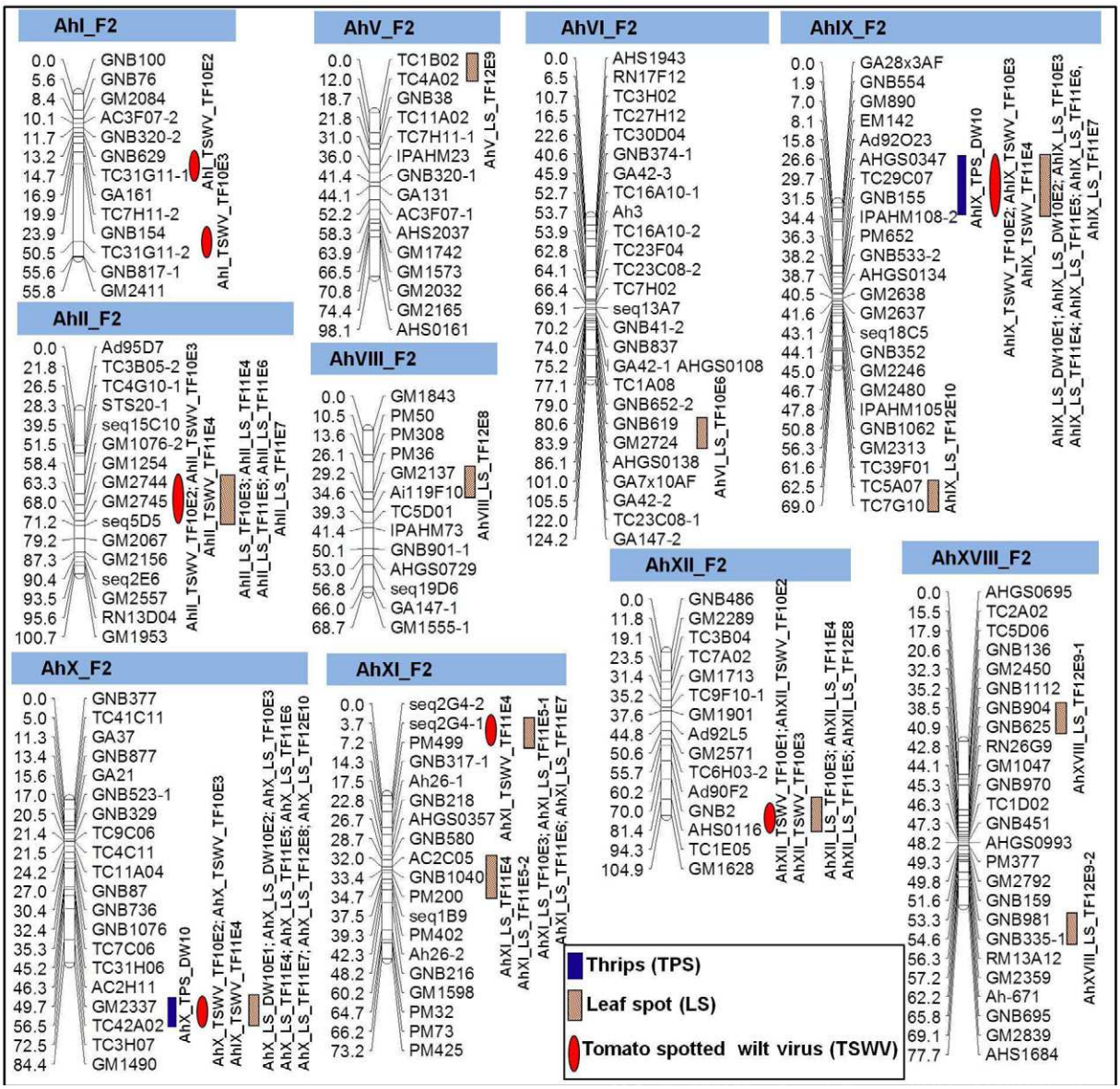


Figure 2. Quantitative trait loci (QTL) locations for thrips (TPS), tomato spotted wilt virus (TSWV), and leaf spot (LS) on the $F_2$ generation-based genetic map of the Tifrunner × GT-C20 population. The original genetic linkage map was given in detail by Wang et al. (2012). The original linkage group names were used with addition of underline and $F_2$ such as AhI_F2 as linkage group 1 of $F_2$ map. The phenotype data nomenclature was named in combination of the location such as Dawson (DW) and Tifton (TF), the year of 2010 (10), 2011(11), or 2012 (12), the environment (E), and the replication number. The QTL were named in combination such as AhI_TSWV_TF10E2 designed as the linkage group number (AhI), underline, disease name (TSWV), underline, and the season of the phenotype data collected (TF10E2).

(IPAHM167-130–GM1555-1000). These genomic regions were named as *qF5TSWV1* to *qF5TSWV7*, respectively (Supplemental Table S3). Two genomic regions, that is, *qF5TSWV4* (AC3C07-70–RN35H04-1500) and *qF5TSWV7* (IPAHM167-130–GM1555-1000), were consistent as both harbored two QTL for TSWV, which were located on LGT9 and LGT25 with PV range of 11.45 to 14.14% and 7.25 to 7.62%, respectively (Table 3). Among the five nonconsistent QTL, *qF5TSWV5* had high PV (10.80%) followed by *qF5TSWV6* (10.64%), *qF5TSWV1* (9.31%), *qF5TSWV2* (7.71%), and *qF5TSWV3* (5.20%).

Of the 15 QTL detected in the $F_2$ map, 11 QTL were contributed by Tifrunner while four QTL were contributed by GT-C20 with additive effects, ranging from −0.443 (*qF2TSWV8*) to −1.250 (*qF2TSWV6*) and 0.797 (*qF2TSWV7*) to 1.347 (*qF2TSWV4*), respectively (Supplemental Table S2). Similarly in the case of the $F_5$ map, five QTL were contributed by Tifrunner while four QTL were contributed by GT-C20 with additive effects, ranged from −0.235 (*qF5TSWV3*) to −3.860 (*qF5TSWV1*) and 0.332 (*qF5TSWV6*) to 0.401 (*qF5TSWV4*), respectively (Supplemental Table S3).

## Quantitative Trait Loci Identified for Leaf Spot

Quantitative trait loci analyses for 10 different phenotyping data of LS led to identification of a total of 50 QTL, which

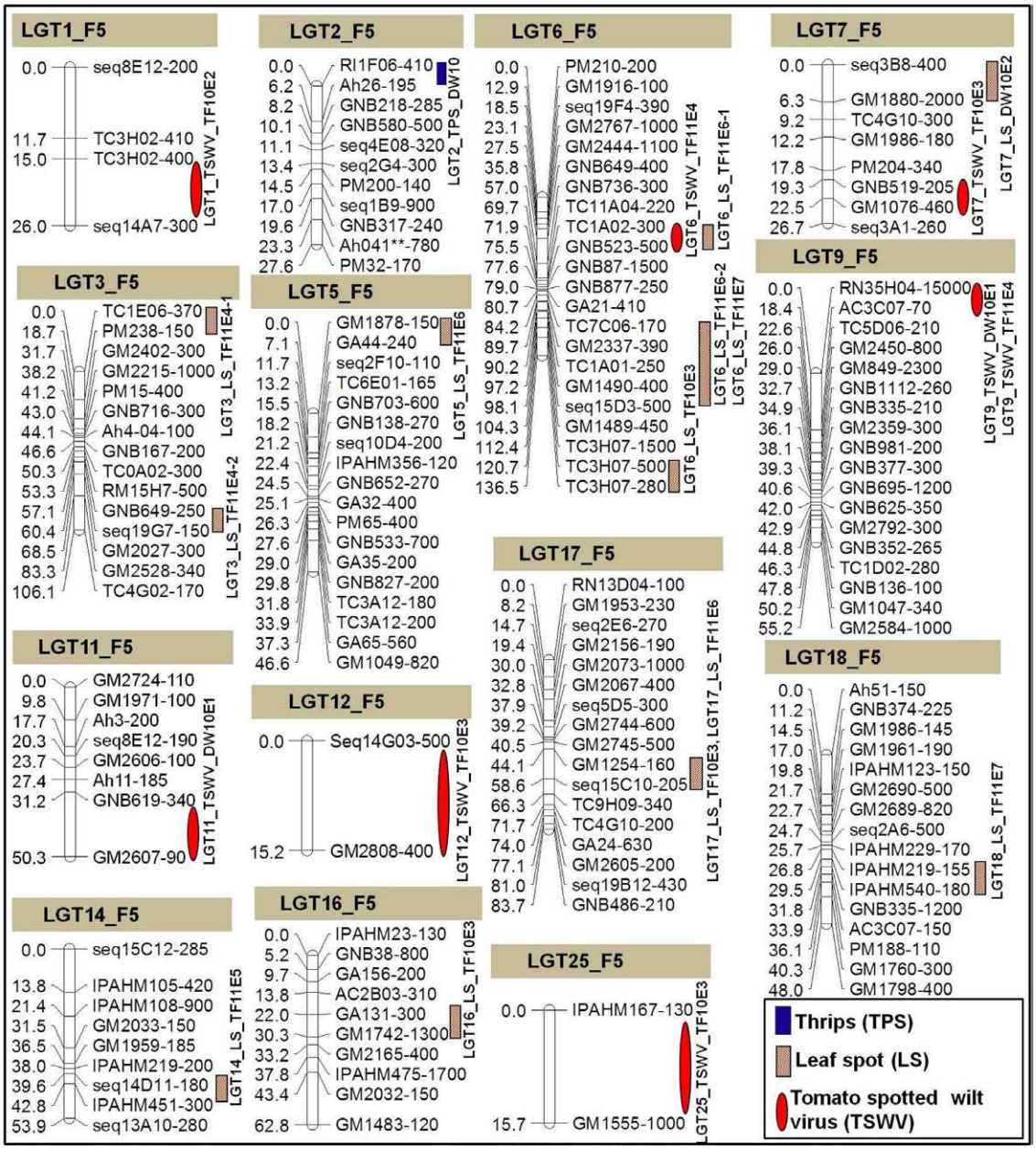


Figure 3. Quantitative trait loci (QTL) locations for thrips (TPS), tomato spotted wilt virus (TSWV), and leaf spot (LS) on the $F_5$ generation-based genetic map of the Tifrunner × GT-C20 population. The original genetic linkage map was given in detail by Qin et al. (2012). The original linkage group names were used with addition of underline and $F_5$ such as LGT1_F5 as linkage group 1 of $F_5$ map. The phenotype data nomenclature was named in combination of the location such as Dawson (DW) and Tifton (TF), the year of 2010 (10), 2011(11), or 2012 (12), the environment (E), and the replication number. The QTL were named in combination such as LGT1_TSWV_TF10E2 designed as the linkage group number (LGT1), underline, disease name (TSWV), underline, and the season of the phenotype data collected (TF10E2).

include 37 QTL for the $F_2$ map and 13 QTL for the $F_5$ map with PV ranging from 6.61 to 27.35% and 5.95 to 21.45%, respectively (Table 1). All the 37 QTL detected in the $F_2$ map were located on 12 genomic regions of nine LGs (AhII, AhV, AhVI, AhVIII, AhIX, AhX, AhXI, AhXII, and AhXVIII). The same names were given to the QTL if they are mapped with same genomic regions or marker interval. Therefore, 37 QTL mapped on 12 genomic regions on the $F_2$ map were named as *qF2LS1* to *qF2LS12* without referring to any season (Supplemental Table S2).

The seven genomic regions, namely GM2744 to seq5D5 (*qF2LS1*) on AhII, IPAHM108-2 to AHGS0347 (*qF2LS5*), TC5A07 to TC7G10 (*qF2LS6*), and TC42A02 to GM2337 (*qF2LS7*) on AhIX, seq2G4 to PM499 (*qF2LS8*) and PM200 to AC2C05 (*qF2LS9*) on AhXI, and GNB2 to AHO116 (*qF2LS10*), harbored five, four, four, nine, four, two, and four QTL, respectively; therefore, these seven genomic regions are referred as consistent QTL (Table 2). The PVs explained by the consistent QTL for LS were higher in general as compared to the nonconsistent QTL.

Among seven consistent QTL, three consistent QTL, namely *qF2LS5*, *qF2LS6*, and *qF2LS7*, contributed more or less equally as their PV ranged from 11.27 to 24.45%, 10.8 to 24.19%, and 13.48 to 24.85%, respectively, followed by *qF2LS10* (15.30–21.19%), *qF2LS8* (6.61–18.97%), *qF2LS1* (7.80–13.11%), and *qF2LS9* (10.29–11.51%) (Table 2). Similarly, among the five nonconsistent QTL, *qF2LS11* had the highest PV (27.35%) followed by *qF2LS3* (12.56%), *qF2LS12* (11.59%), *qF2LS2* (8.22%), and *qF2LS4* (8.11%).

The 13 QTL (5.95–21.45% PV) identified on the $F_5$ map were located on 11 genomic regions of eight different LGs, LGT3, LGT5, LGT6, LGT7, LGT14, LGT16, LGT17, and LGT18 (Supplemental Table S3). These genomic regions were named as *qF5LS1* to *qF5LS11*, respectively. Two genomic regions, that is, *qF5LS5* (TC7C06-170–seq15D3-500) and *qF5LS10* (GM1254-160–seq15C10-205), were consistent as both harbored two QTL for LS, which were located on LGT6 and LGT10 with PV range of 7.61 to 11.20% and 7.50 to 9.08%, respectively. Among the nine nonconsistent QTL, *qF5LS1* had a PV of 21.45% while the remaining eight QTL (*qF5LLS2*, *qF5LS3*, *qF5LS4*, *qF5LS6*, *qF5LS7*, *qF5LS8*, *qF5LS9*, and *qF5LS11*) had a low PV range of 5.95 (*qF5LS8*) to 8.98% (*qF5LS3*) (Table 3).

**Table 1. Summary on quantitative trait loci (QTL) analysis based on the $F_2$ and $F_5$ population for disease resistance.**

| Traits | No. of QTL identified | LOD† value range | Phenotypic variance explained | Additive effect (a0) range |
|---|---|---|---|---|
| | | | % | |
| Based on the $F_2$ population | | | | |
| Thrips | 2 | 2.69–3.27 | 12.14–19.43 | 0.482–0.608 |
| Tomato spotted wilt virus | 15 | 2.51–5.92 | 4.40–34.92 | 1.347–0.526 |
| Leaf spot | 37 | 2.51–5.68 | 6.61–27.35 | 4.629–0.720 |
| Based on the $F_5$ population | | | | |
| Thrips | 1 | 2.51 | 5.86 | 0.0518 |
| Tomato spotted wilt virus | 9 | 2.50–4.61 | 5.20–14.14 | 0.400–0.249 |
| Leaf spot | 13 | 2.51–6.38 | 5.95–21.45 | 0.273–0.174 |

†LOD, log-of-odds.

Of the 37 QTL detected for LS in the $F_2$ map, 20 QTL were contributed by Tifrunner while 17 QTL were contributed by GT-C20 with additive effects, ranging from −0.861 (*qF2LS1* at season TF11E4 [Tifton in 2011 in season E4]) to −2.921 (*qF2LS10* at season TF12E8 [Tifton in 2012 in season E8]) and 0.720 (*qF2LS8* at

**Table 2. Consistent quantitative trait loci (QTL) detected for thrips, tomato spotted wilt virus, and leaf spot in the $F_2$ Tifrunner × GT-C20 population.**

| Traits | Linkage group | Marker interval | Phenotype data† | LOD‡ value | Phenotypic variance explained | Additive effect |
|---|---|---|---|---|---|---|
| | | | | | % | |
| Thrips | | | | | | |
| *qF2TPS1* | AhIX | IPAHM108-2–AHGS0347 | DW10 | 2.69 | 12.14 | 0.482 |
| *qF2TPS2* | AhX | GM2337–TC42A02 | DW10 | 3.27 | 19.43 | −0.608 |
| Tomato spotted wilt virus | | | | | | |
| *qF2TSWV1* | AhI | GNB629–TC31G11 | TF10E2 | 2.62 | 9.41 | −1.153 |
| *qF2TSWV2* | AhI | GA161–GNB154 | TF10E3 | 2.51 | 4.40 | −0.685 |
| *qF2TSWV3* | AhII | seq5D5–GM2744 | TF10E2, TF10E3, and TF11E4 | 2.79–5.92 | 5.14–34.92 | (–) 3.539 to (–) 0.526 |
| *qF2TSWV4* | AhIX | IPAHM108-2–AHGS0347 | TF10E2 and TF10E3 | 3.99–4.84 | 12.92–18.11 | 1.024 to 1.347 |
| *qF2TSWV5* | AhIX | TC5A07–TC7G10 | TF11E4 | 4.42 | 23.02 | 1.120 |
| *qF2TSWV6* | AhX | TC42A02–GM2337 | TF10E2, TF10E3, and TF11E4 | 3.01–3.28 | 10.78–16.56 | (–) 1.250 to (–) 0.743 |
| *qF2TSWV7* | AhXI | seq2G4–PM499 | TF11E4 | 2.93 | 15.75 | 0.797 |
| *qTSWV8* | AhXII | GNB2–AHO116 | DW10E1, TF10E2, and TF10E3 | 2.61–4.16 | 6.26–21.18 | (–) 1.374 to (–) 0.443 |
| Leaf spot | | | | | | |
| *qF2LS1* | AhII | GM2744–seq5D5 | TF10E3, TF11E4, TF11E5, TF11E6, and TF11E7 | 2.69–3.59 | 7.80–13.11 | (–) 1.422 to (–) 0.861 |
| *qF2LS2* | AhV | TC1B02-TC4A02 | TF12E9 | 2.54 | 8.22 | 1.399 |
| *qF2LS3* | AhVI | GM2724–GNB619 | TF11E6 | 2.68 | 12.56 | 1.064 |
| *qF2LS4* | AhVIII | PM36–GM2137 | TF12E9 | 2.78 | 8.11 | −1.771 |
| *qF2LS5* | AhIX | IPAHM108-2–AHGS0347 | DW10E1, DW10E2, TF11E6, and TF11E7 | 2.51–5.68 | 11.27–24.45 | 1.188 to 2.262 |
| *qF2LS6* | AhIX | TC5A07–TC7G10 | TF10E3, TF11E4, TF11E5, and TF12E10 | 3.33–5.01 | 10.8–24.19 | 1.253 to 1.834 |
| *qF2LS7* | AhX | TC42A02–GM2337 | DW10E1, DW10E2, TF10E3 TF11E4, TF11E5, TF11E6, TF11E7, TF12E8, and TF12E10 | 2.51–4.82 | 13.48–24.85 | (–) 2.519 to (–) 0.978 |
| *qF2LS8* | AhXI | seq2G4–PM499 | TF10E3, TF11E5, TF11E7, and TF12E8 | 2.55–3.52 | 6.61–18.97 | 0.720 to 1.399 |
| *qF2LS9* | AhXI | PM200–AC2C05 | TF11E4 and TF11E5 | 2.51–2.70 | 10.29–11.51 | 0.738 to 1.347 |
| *qF2LS10* | AhXII | GNB2–AHO116 | TF10E3, TF11E4, TF11E5, and TF12E8 | 2.65–2.90 | 15.30–21.19 | (–) 1.208 to −2.921 |
| *qF2LS11* | AhXVIII | GNB904–GNB625 | TF12E9 | 3.54 | 27.35 | 4.629 |
| *qF2LS12* | AhXVIII | GNB159–GNB335 | TF12E9 | 3.11 | 11.59 | −2.497 |

†The phenotype data nomenclature was named in combination of the location such as Dawson (DW) and Tifton (TF), the year of 2010 (10), 2011(11), or 2012 (12), the environment (E), and the replication number.

‡LOD, log-of-odds.

**Table 3. Summary of quantitative trait loci (QTL) detected for thrips, tomato spotted wilt virus, and leaf spot in the $F_5$ the Tifrunner × GT-C20 population.**

| Traits | Linkage group | Marker interval | Phenotype data† | LOD‡ value | Phenotypic variance explained | Additive effect |
|---|---|---|---|---|---|---|
| | | | | | % | |
| Thrips | | | | | | |
| *qF5TPS1* | LGT2 | RI1F06-410–Ah26-195 | DW10 | 2.51 | 5.86 | 0.0518 |
| Tomato spotted wilt virus disease | | | | | | |
| *qF5TSWV1* | LGT1 | TC3H02-410–seq14A7-300 | TF10E2 | 3.56 | 9.31 | −0.3860 |
| *qF5TSWV2* | LGT6 | TC11A02-300–GNB523-500 | TF11E4 | 3.50 | 7.71 | −0.2486 |
| *qF5TSWV3* | LGT7 | GNB519-205–GM1076-460 | TF10E3 | 2.50 | 5.20 | −0.2357 |
| *qF5TSWV4* | LGT9 | AC3C07-70–RN35H04-1500 | DW10E1 and TF11E4 | 3.90–4.61 | 11.45–14.14 | 0.335 to 0.401 |
| *qF5TSWV5* | LGT11 | GNB619-340–GM2607-90 | DW10E1 | 3.50 | 10.80 | 0.3453 |
| *qF5TSWV6* | LGT12 | seq14G03-500–GM2808-400 | TF10E3 | 3.40 | 10.64 | 0.3318 |
| qF5TSWV7 | LGT25 | IPAHM167-130–GM1555-1000 | TF10E2 and TF10E3 | 2.52–2.60 | 7.25–7.62 | (–) 0.347 to (–) 0.274 |
| Leaf spot disease | | | | | | |
| *qF5LS1* | LGT3 | TC1E06-370–PM238-150 | TF11E4 | 6.38 | 21.45 | −0.2430 |
| *qF5LS2* | LGT3 | seq19G7-150–GNB649-250 | TF11E4 | 2.65 | 6.10 | 0.1311 |
| *qF5LS3* | LGT5 | GM1878–GM637-240 | TF11E6 | 3.25 | 8.98 | −0.1835 |
| *qF5LS4* | LGT6 | TC11A02-300–GNB523-500 | TF11E6 | 3.35 | 8.02 | −0.1739 |
| *qF5LS5* | LGT6 | TC7C06-170–seq15D3-500 | TF11E6 and TF11E7 | 2.94–3.41 | 7.61–11.20 | (–) 0.169 to (–) 0.199 |
| *qF5LS6* | LGT6 | TC3H07-500–TC3H07-280 | TF10E3 | 2.53 | 8.15 | −0.1939 |
| *qF5LS7* | LGT7 | seq3B8-400–GM1880-2000 | DW10E2 | 2.69 | 7.35 | 0.2733 |
| *qF5LS8* | LGT14 | seq14D11-180–IPAHM451-300 | TF11E5 | 2.65 | 5.95 | 0.2218 |
| *qF5LS9* | LGT16 | GM678-300–GM1742-1300 | TF10E3 | 2.74 | 7.04 | −0.1818 |
| *qF5LS10* | LGT17 | GM1254-160–seq15C10-205 | TF10E3 and TF11E6 | 2.51–2.95 | 7.5–9.08 | 0.172 to 0.212 |
| *qF5LS11* | LGT18 | IPAHM229-170–IPAHM219-155 | TF11E7 | 3.70 | 8.71 | 0.1762 |

†The phenotype data nomenclature was named in combination of the location such as Dawson (DW) and Tifton (TF), the year of 2010 (10), 2011(11), or 2012 (12), the environment (E), and the replication number.

‡LOD, log-of-odds.

season TF11E6 [Tifton in 2011 in season E6]) to 4.629 (*qF2LS11* at season TF12E9 [Tifton in 2012 in season E9]), respectively (Supplemental Table S2). Similarly in the $F_5$ map, of the 13 QTL detected for LS, five QTL were contributed by Tifrunner while six QTL were contributed by GT-C20 with additive effects, ranging from −0.1739 (*qF5LS4* at season TF11E6) to −2.430 (*qF5LS1* at season TF11E4) and 0.1311 (*qF5LS2* at season TF11E4) to 0.2733 (*qF5LS7* at season DW10E2 [Dawson in 2010 in season E2]), respectively (Supplemental Table S3).

## Common Quantitative Trait Loci Identified Among the Traits

Two common regions were identified in the $F_2$ map for all the three diseases. The first common genomic region (GM2337–TC42A02) was located on AhX, which harbored one QTL for thrips (*qF2TPS2*), three QTL for TSWV (*qF2TSWV6* for three seasons), and nine QTL for LS (*qF2LS7* for 9 of the total 10 seasons). This genomic region is contributing 19.43% PV for thrips, 10.78 to 16.56% PV for TSWV, and 13.48 to 24.85% PV for LS. In all the three traits, the phenotypic contribution came from the resistant parent, Tifrunner. The second common region (IPAHM108-2–AHGS0347) located on AhIX harbored one QTL for thrips (*qF2TPS1*), two QTL for TSWV (*qF2TSWV4* for two seasons), and four QTL for LS (*qF2LS5* for four of the total 10 seasons). This genomic region is contributing 12.14% PV for thrips, 12.92 to 18.11% PV for TSWV, and 11.27 to 24.45% PV for LS. Interestingly, for all the three diseases, the phenotypic contribution came from the susceptible parent, GT-C20, for this second common region.

Furthermore, other four genomic regions harbored QTL for both TSWV and LS. These four genomic regions are located on four different LGs of the $F_2$ map, that is, on AhII (GM2744–seq5D5), AhIX (TC5A07–TC7G10), AhXI (seq2G4–PM499), and AhXII (GNB2–AHO116). The first genomic region (GM2744–seq5D5) harbored three QTL for TSWV (5.14–34.92% PV) and five QTL for LS (7.80–13.11% PV) with the contribution from the resistant parent, Tifrunner. Similarly, the second genomic region (TC5A07–TC7G10) harbored a single QTL for TSWV (23.02% PV) and four QTL for LS (10.08–24.19% PV) with the contribution coming from the susceptible parent, GT-C20. The third genomic region (seq2G4–PM499) harbored a single QTL for TSWV (15.75% PV) and four QTL for LS (6.61–18.97% PV) contributed by the susceptible parent, GT-C20. The fourth genomic region (GNB2–AHO116) harbored three QTL for TSWV (6.26–21.18% PV) and four QTL for LS (15.30–21.19% PV) contributed by the resistant parent, Tifrunner.

In contrast to the $F_2$ map, there was no common QTL for all three traits in the $F_5$ map. There was only one

common genomic region located on LGT6 (TC11A02-300–GNB523-500) harboring one QTL for TSWV (*qF5TSWV2*) with 7.71% PV and one for LS (*qF5LS4*) with 8.02% PV.

### Common Quantitative Trait Loci Identified Between Two Maps

There was one QTL controlling LS in the $F_2$ map (AhXVIII) and one QTL controlling TSWV in the $F_5$ map (LGT7) flanked by same markers, that is, GNB159 to GNB335. In the other case, even though the flanking markers were not same, the QTL were found on the same LG. Such QTL have been observed between corresponding LGs of both genetic maps, for example between AhII and LGT17, AhV and LGT16, AhVI and LGT11, and AhX and LGT6.

## DISCUSSION

Due to the increased uniformity in marker nomenclature, the corresponding LGs between these two maps have been identified. Furthermore, a total of 9 of the 10 LGs from A genome and 8 of the 10 LGs from B genome could be assigned after comparing these two genetic maps with the reference consensus genetic maps using the common marker loci (Gautami et al., 2012). In general, a good co-linearity has been observed for these two genetic maps and with the reference consensus genetic map (Fig. 1). This population has shown great potential not only for genetic mapping but also for identification of QTL to several economically important traits such as morphological descriptors, oil quality, and disease resistance. Here, a successful attempt was made to make use of both the genetic maps and the identified QTL for the three resistance traits to thrips, TSWV, and LS.

A RIL population is a set of genotypes of highly inbred $F_2$ lines. Recombinant inbred lines approach complete homozygosity for all loci as the number of generations of inbreeding approaches infinity. In practice, the convention is to use six to eight generations of inbreeding, resulting in approximately 99.84 to 99.96% homozygosity, respectively. A major advantage of RILs is that the descendents of any one RIL are genetically identical and hence "immortal," allowing RILs to be marker genotyped once and phenotyped repeatedly in multiple labs and experiments (Elnaccash and Tonsor, 2010). It is well understood that RIL-based QTL analysis is more reliable than the $F_2$–based mapping populations for identification of QTL. The majority of the studies showed identification of large number of QTL with overestimated phenotypic effect. However, none of the studies was conducted at both the stages ($F_2$ and RIL) using the same population and therefore this study was focused on using genotyping data generated at the $F_2$ and $F_5$ generation and phenotyping data generated at the $F_8$ generation onward on the same population. Phenotyping data generated on this population after the $F_8$ generation was used for both the genetic maps to identify QTL for the three traits, thrips, TSWV, and LS. Therefore, a total of 77 QTL were identified in these two maps, 54 QTL in the $F_2$ map (Fig. 2) and 23 QTL in the $F_5$ map (Fig. 3) with PV up to 19.43 (thrips), 34.92 (TSWV), and 21.45% (LS), respectively.

We should therefore expect that the $F_2$ and the RIL populations might show high PV and this effect will be exaggerated in the RIL compared to the $F_2$ because all individuals are homozygous at virtually all loci, and the large sample size in the RIL reduces the variance of the mean and transgressive segregation and homozygosity increase the mean's variance (Beavis, 1998). As expected, the PV explained by QTL detected in the $F_2$ map showed relatively higher PV as compared to the $F_5$ map. Occurrence of more QTL with relatively higher estimation of phenotypic effect in the $F_2$ map than the $F_5$ map was due to presence of higher level of heterozygosity in the $F_2$ generation. Nevertheless, this study has provided comparative QTL analysis using genotyping data generated at the $F_2$ and $F_5$ generation on the same population and confirms the assumption established based on studies on different populations. Because of above two technical deficiencies (higher number of QTL and high estimation of PV) of using the $F_2$ population for conducting QTL analysis, earlier studies support the use of RIL populations such as double haploids and RILs. These RIL populations have additional advantage of being useful for phenotyping the population for multiple seasons and locations to identify consistent (across seasons) and stable (across locations) QTL.

It was interesting to note that not only alleles of the resistant parent have contributed towards the total PV but the susceptible parent also made significant contribution through favorable alleles. For thrips no study so far has been conducted while for TSWV, earlier using the same population, Qin et al. (2012) reported one QTL with 12.9% PV (*qtswv1*). Besides the above QTL, no other QTL for TSWV has been reported so far in peanut. Therefore, all the QTL identified in current study for thrips and TSWV are novel in nature and are of great importance for further study and their deployment in molecular breeding.

The highest PV explained by any QTL for LS was 27.35% (*qF2LS11*) in the present study while earlier QTL analysis using extensive phenotyping data on two RIL populations (TAG 24 × GPBD 4 and TG 26 × GPBD 4) for seven to eight seasons and genotyping data (207 marker loci each) resulted in identification of a total of 28 QTL for LLS (10.1 to 67.8% PV) (Khedikar et al., 2010; Sujay et al., 2012). These QTL include a major QTL for LLS with up to 62.34% PV flanked by GM1573 or GM1009 and seq8D09.

Plants possess a strong immune system and defense mechanism to prevent themselves from the pathogens. Therefore, common genomic regions controlling more than one disease may be even more important to improve plant resilience. Considering the above hypothesis, two common genomic regions (GM2337–TC42A02 and IPAHM108-2–AHGS0347) were identified in the $F_2$ map for all the three diseases while four common genomic regions (GM2744–seq5D5, TC5A07–TC7G10, seq2G4–PM499, and GNB2–AHO116) in the $F_2$ map and one common genomic region (TC11A02-300–GNB523-500)

in the $F_5$ map were identified for LS and TSWV. The presence of common QTL has also been reported by Sujay et al. (2012) wherein three genomic regions harbored QTL from two populations for both leaf rust and LLS. Therefore, these common genomic regions may harbor genes that play a major role in plant defense against several pathogens and hence can be used for improving resistance for more than one disease through increasing resistance.

In summary, through screening more than 5000 markers, genetic maps up to 329 marker loci have been developed. High DNA polymorphism and high phenotypic variability between parental genotypes have made the Tifrunner × GT-C20 population a very good genetic material for identification of linked markers through QTL analysis to thrips, TSWV, and LS. Common genomic regions controlling more than one disease has also been identified with significant contribution towards disease resistance. Thus, this population has shown great potential for dense genetic mapping and identification of QTL controlling several disease and agronomic traits in peanut. In addition it was evident that the number of QTL and the estimates of PV were reduced in the $F_5$ map. The identified QTL, consistent or not, will be studied further through fine mapping for potential use in breeding for genetic improvement of disease resistance in peanut.

## Supplemental Information Available

Supplemental material is available at http://www.crops.org/publications/tpg.


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