Theoretical and Applied Genetics (2022) 135:3773–3872
https://doi.org/10.1007/s00122-022-04101-3

REVIEW

Breeding for disease resistance in soybean: a global perspective

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Received: 21 November 2021 / Accepted: 11 April 2022 / Published online: 5 July 2022

Abstract

Key message **This review provides a comprehensive atlas of QTLs, genes, and alleles conferring resistance to 28 important diseases in all major soybean production regions in the world.**

Abstract Breeding disease-resistant soybean [*Glycine max* (L.) Merr.] varieties is a common goal for soybean breeding programs to ensure the sustainability and growth of soybean production worldwide. However, due to global climate change, soybean breeders are facing strong challenges to defeat diseases. Marker-assisted selection and genomic selection have been demonstrated to be successful methods in quickly integrating vertical resistance or horizontal resistance into improved soybean varieties, where vertical resistance refers to R genes and major effect QTLs, and horizontal resistance is a combination of major and minor effect genes or QTLs. This review summarized more than 800 resistant loci/alleles and their tightly linked markers for 28 soybean diseases worldwide, caused by nematodes, oomycetes, fungi, bacteria, and viruses. The major breakthroughs in the discovery of disease resistance gene atlas of soybean were also emphasized which include: (1) identification and characterization of vertical resistance genes reside *rhg1* and *Rhg4* for soybean cyst nematode, and exploration of the underlying regulation mechanisms through copy number variation and (2) map-based cloning and characterization of *Rps11* conferring resistance to 80% isolates of *Phytophthora sojae* across the USA. In this review, we also highlight the validated QTLs in overlapping genomic regions from at least two studies and applied a consistent naming nomenclature for these QTLs. Our review provides a comprehensive summary of important resistant genes/QTLs and can be used as a toolbox for soybean improvement. Finally, the summarized genetic knowledge sheds light on future directions of accelerated soybean breeding and translational genomics studies.

Introduction

Soybean [*Glycine max* (L.) Merr.] is one of the most important crops globally. It produced 70.86% of the global supply of plant-based protein meal and 28.88% of the plant-based oil (second only to palm oil) in the 2020/2021 market year (Market View Data Base, Untied Soybean Board 2021. https://marketviewdb.centrec.com/?bi=Global_MealandOil_Consumption_Annual). Total world soybean production in 2020 was 353.5 million metric tons (Mt), and the estimated cultivated area was 127.0 million ha. While cultivated throughout the world, 96.2% of soybean production is concentrated in ten countries: Brazil (121.8 million Mt), the USA (112.5 million Mt), Argentina (48.8 million Mt), China (19.6 million Mt), India (11.2 million Mt), Paraguay (11.0 million Mt), Canada (6.4 million Mt), Russia (4.3 million Mt), Ukraine (2.8 million Mt), and Bolivia (2.8 million Mt) (FAOSTAT 2020; Fig. 1). A major constraint to soybean production is disease loss. Of more than 200 pathogens known to infect soybean, only about 35 are economically important (Hartman et al. 2016). The most prevalent diseases in major soybean production regions of the world are presented in 1. The type and severity of disease and the degree of yield and seed quality loss vary with region and year, depending on the climate and the growing season weather, cultural and disease control practices, and the genetic diversity of the pathogens and the soybean cultivars. Unfortunately, the

Communicated by Rajeev K. Varshney.

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proportion of global soybean yield loss due to diseases increased from ~ 11% in 1994 to 27% in 2006. In 1994, soybean diseases caused losses of nearly 15 million Mt (10.87% of total production), valued at more than $3 billion across the top ten soybean production countries (Wrather et al. 1997). In 1998, the world soybean yield losses due to diseases were more than 28 million Mt (18.49% of total production), more than doubled the losses in 1994 (> $6 billion) (Wrather et al. 2001). In 2006, a total of 59.9 million Mt of soybean production were reduced in the world, accounting for more than 27% of the total soybean production (220.4 million Mt) (Wrather et al. 2010).

In a recent report of soybean production losses caused by diseases in the USA and Canada from 2010 to 2014, yearly losses ranged from 10.06 to 13.92 million Mt (11.7–14.2% of total soybean production) (Allen et al. 2017). These losses are the result of many diseases caused by a range of fungi, bacteria, phytoplasmas, nematodes, and viruses. Recent meta-analyses of soybean disease losses in the USA over the last 24 years found that the greatest losses across states and years were from soybean cyst nematode (SCN) (*Heterodera glycines* Ichinohe), charcoal rot [*Macrophomina phaseolina* (Tassi) Goid], and seedling diseases (caused by several oomycetes and fungi) (Bandara et al. 2020; Roth et al. 2020). Important intermittent diseases caused by variations in the weather were Phytophthora root and stem rot (*Phytophthora sojae* Kaufmann & Gerdemann), sudden death syndrome (SDS) (*Fusarium virguliforme* O'Donnell and T. Aoki), and Sclerotinia stem rot [*Sclerotinia sclerotiorum* (Lib.)] (Roth et al. 2020). Root-knot nematode (*Meloidogyne* spp.), reniform nematode (*Rotylenchulus reniformis* Linford & Oliveira), and *Diaporthe* diseases were emerging diseases. Disease pressure appears to be increasing as greater yield losses have been observed over time (Bandara et al. 2020).

In Brazil, estimates in 1997 reported that the greatest disease losses were from stem canker (*Diaporthe aspalathi* (E. Jansen, Castl. & Crous) and *D. caulivora* (Athow & Caldwell) J.M. Santos, Vrandecic & A.J.L. Phillips), brown spot (*Septoria glycines* Hemmi), Cercospora leaf blight (CLB)/purple seed stain (PSS) [*Cercospora kikuchii* (Matsumoto & Tomoyasu) M. W. Gardner], and charcoal rot followed by soybean cyst nematode, seedling diseases, and Sclerotinia stem rot (Wrather et al. 1997). However, after soybean rust [*Phakopsora pachyrhizi* (Sydow. & Sydow.)] was introduced in Brazil in 2002, it quickly became the most suppressive soybean pathogen causing yield losses of nearly sixfold greater than CLB/PSS, the second most damaging disease in the country (Wrather et al. 2010). Soybean rust is particularly damaging in Brazil due to the year-round survival of the pathogen in production areas unlike in neighboring Argentina, where the pathogen must be re-introduced each year, therefore resulting in significantly less damage than in Brazil. The major soybean diseases in Argentina include SDS, charcoal rot, Cercospora leaf blight, brown spot, target spot [*Corynespora cassiicola* (Berk. & M.A. Curtis) C.T. Wei], and Sclerotinia stem rot. The most prevalent soybean disease in China is soybean mosaic virus (SMV). Other major diseases in China include frogeye leaf spot (*Cercospora sojina* Hara), SCN, anthracnose (*Colletotrichum* spp.), root rot (*P. sojae*, *Pythium* spp., *Fusarium* spp.), bacterial diseases, Sclerotinia stem rot, downy mildew [*Peronospora manshurica* (Naum.) Syd.], and soybean rust (Wrather et al. 1997, 2001, 2010). Prominent diseases in India include viruses, Sclerotium blight (*Sclerotium rolfsii* Sacc.), anthracnose (*Colletotrichum* spp.), and soybean rust (Wrather et al. 2010).

Russia and Ukraine are the most soybean productive countries in the world. Common soybean diseases in Russia include SCN, SMV, downy mildew, frogeye leaf spot, Phyllosticta leaf spot (*Pleosphaerulina sojicola* Miura, syn. *Phyllosticta sojicola* C. Massal.), CLB/PSS, brown spot, bacterial bustle (*Xanthomonas axonopodis* pv. *glycines*), and bacterial blight (*Pseudomonas syringae* pv. *glycinea* Coerper) (Bushnev et al. 2020; Sinegovskaya 2021). In Ukraine, SMV is a major concern which often infects together with bean yellow mosaic virus (BYMV), and Alfalfa mosaic virus (AMV) in the right-bank region (Kyrychennko et al. 2012; Mishchenko et al. 2017), while in the Forest-Steppe region, Alternaria leaf spot, downy mildew, Fusarium wilt and root rot, brown spot, and bacterial blight are the most prevalent soybean diseases (Sergiienko et al. 2021).

Africa and Australia represent geographical regions with the potential to become major soybean producers in the future (Hartman and Murithi, 2019). Africa produces about 1% of global soybean production (FAOSTAT, 2020). The major soybean diseases in Africa include soybean rust, frogeye leaf spot, red leaf blotch (*Coniothyrium glycines*), and SDS (Murithi et al. 2016; Hartman and Murithi, 2019). Australia produced 17,323 tons of soybean in 2020 (FAOSTAT 2020), and the major soybean diseases include charcoal rot, sclerotinia stem rot, Phytophthora root rot, and soybean rust (Ryley 2013).

In the future, soybean diseases may be continuously severe and difficult to manage, especially with the significant changes in the global climate (Roth et al. 2020). Since 1981, global temperatures have risen 0.18 °C per decade (www.climate.gov) and are expected to rise 6 °C by the next century (Mikhaylov et al. 2020). Temperatures and water precipitation are expected to increase in many areas (Tebaldi et al. 2006; Karl et al. 2009), but the increase in rainfall will be followed by more frequent extreme weather events as well as more frequent and severe droughts,

making the overall weather patterns less consistent and predictable (Prein et al. 2021). It is estimated that rising temperatures have hindered agricultural production gains by 21% and made the management of plant diseases increasingly challenging (Jones 2021; Ortiz-Bobea et al. 2021). In the USA, it is predicted that climate changes may reduce average soybean yields by 86–92% by 2050 (Yu et al. 2021). These climate changes may alter the types, severities, and geographical distributions of soybean diseases, especially for the intermittent diseases that are heavily influenced by environmental factors, such as Phytophthora root and stem rot, SDS, and Sclerotinia stem rot (Roth et al. 2020).

Effective soybean disease management includes cultural practices (crop rotation, tillage, clean seed, etc.), chemical applications (foliar, seed, or soil), but the most important component is the deployment of resistant cultivars (Grau et al. 2004). Resistant cultivars can carry either vertical resistance, horizontal resistance, or both. Vertical resistance is contributed by resistance genes (R genes) for specific diseases, such as SCN (*Rhg*), Phytophthora root and stem rot (*Rps*), soybean rust (*Rpp*), frogeye leaf spot (*Rcs*), bacterial blight (*Rpg*), and SMV (*Rsv* and *Rsc*). R genes have been widely deployed conferring complete resistance to some pathotypes of the pathogen. The R genes typically follow a gene-for-gene interaction with the corresponding avirulence (Avr) factors from the pathogen, and resistance occurs only when the R gene and Avr factors both exist (Whitham et al. 2016). Therefore, R genes are pathotype (race)-specific, i.e., they may confer full protection to some pathotypes of the pathogen, while they are completely susceptible to others. R genes are often non-durable, and can be quickly overcome, due to the fast shift of the pathogen populations. For instance, the *Rpp1* and *Rpp3* genes mediated resistance to soybean rust were defeated the following year after the disease first occurred in Brazil in 2001 (Garcia et al. 2008; Langenbach et al. 2016). Another example is the *Rps1k* gene which has been traditionally deployed since the 1990s, can be defeated by most of the newly emerged pathotypes of *Phytophthora sojae* (McCoy et al. 2021). Although there are some exceptions such as *Rcs3* which has provided durable resistance against all known races of frogeye leaf spot in the USA (Boerma and Phillips 1983; Mian et al. 2008), searching for novel sources of resistance genes is a vital task for the deployment of vertical resistance and sustainability of the global soybean value chain.

In contrast, horizontal resistance (sometimes called partial resistance or tolerance) is quantitative and conferred by multiple minor effect genes and/or quantitative trait loci (QTL). Unlike vertical resistance that occurs only to some specific pathogens, horizontal resistance is widely involved

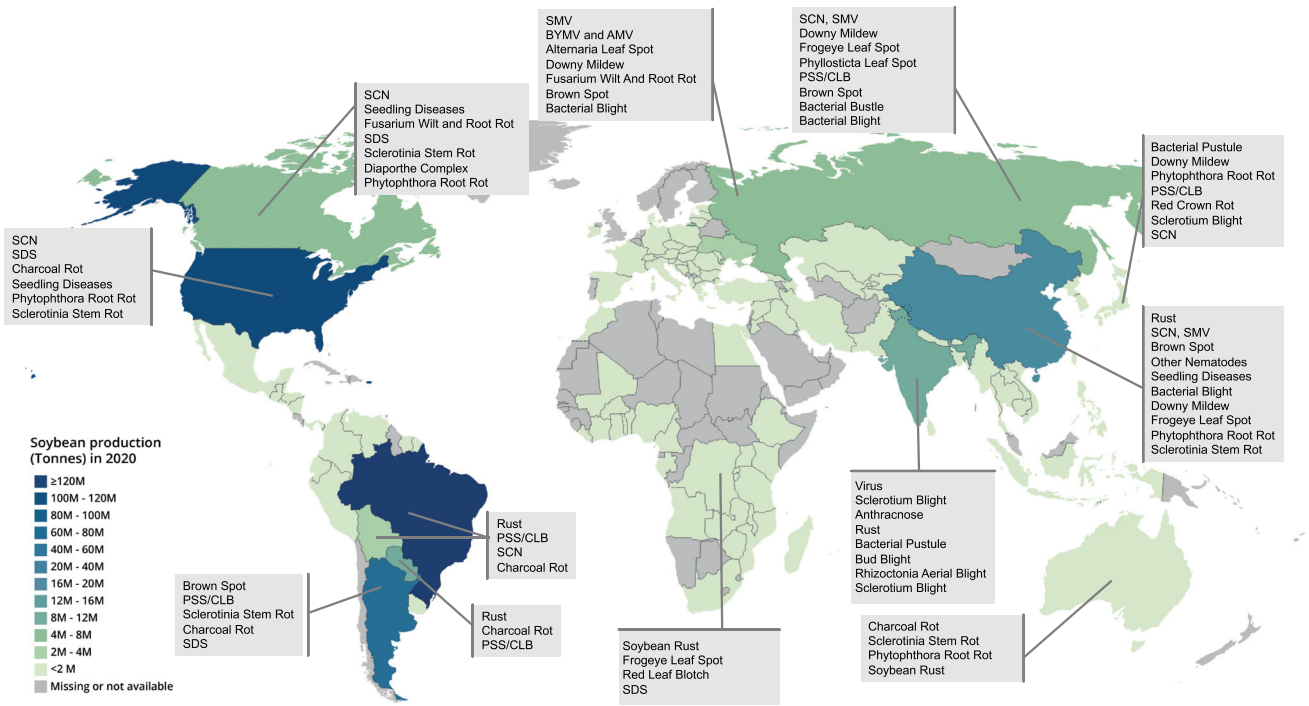

Fig. 1 Global soybean yield production in 2020 (data obtained from FAOSTAT) and major diseases in top ten soybean production countries. SCN: soybean cyst nematode; SDS: sudden death syndrome; PSS: Phomopsis seed decay; CLB: Cercospora leaf blight; SMV: soybean mosaic virus; BYMV: bean yellow mosaic virus; AMV: Alfalfa mosaic virus

in multiple soybean diseases and is known as the only type of resistance to many soybean diseases, including SDS, Sclerotinia stem rot, root-knot nematode, and most *Pythium* species. Horizontal resistance is usually considered pathotype non-specific (Dorrance et al. 2008; St. Clair 2010; Mundt 2014; Nelson et al. 2018; Karhoff et al. 2019), although some isolate specific QTLs have also been identified in soybean (Lee et al. 2014; Stasko et al. 2016; Lin et al. 2021). Therefore, horizontal resistance is considered more durable.

There is a great degree of variation in the reaction of plants to diseases, which can go from immunity to full susceptibility. In this review we consider resistance as the ability of a plant to prevent or limit disease development by means of pre-formed structures or chemicals and/or by infection-induced responses. The level of resistance will depend on various factors, including the host-pathogen interaction and the environment. Intermediate levels of resistance are often confused with tolerance. This is an ambiguous term since tolerance is usually referred as the ability of a susceptible plant to endure disease without severe losses of yield or quality. In this review we focus on the genes or QTLs that have been identified as providing different degrees of resistance to diseases.

The traditional introgression of resistance genes into resistant cultivars can take more than ten years starting from making crosses between the recurrent parents and the resistance donor parents. Fortunately, with the development of molecular marker technology, especially with the sequencing of the soybean genome and the development of low cost of high-throughput genotyping (such as the BARCSoySNP6K and BARCSoySNP50K iSelect BeadChips), breeders can make selections more efficiently and accurately (Song et al. 2013, 2020). Marker-assisted selection (MAS) has proved to be the most successful approach in the selection of R genes or major QTLs (Ribaut and Hoisington 1998). The markers used for MAS have evolved from the low-efficiency restriction fragment length polymorphism (RFLP) markers to simple sequence repeat (SSR) markers, and currently, to more efficient and cost-friendly SNP markers in modern soybean breeding programs. However, for minor effect QTLs, genomic selection (GS) has been demonstrated to outperform MAS with higher accuracy and efficiency (Bao et al. 2014; Wen et al. 2018). For example, Bao et al. (2014) genotyped 282 soybean accessions for resistance to SCN HG type 0 and discovered that GS using full marker set produced significantly more accurate predictions than MAS using two rhg1-associated DNA markers. In another study for soybean resistance to white mold (Wen et al. 2018), the GS prediction accuracy was estimated at 0.64, which was significantly higher than that of MAS (0.47–0.51), although MAS was still 24–26% higher than using random SNPs. Moreover, with the recent development of new technologies such as GWA studies, numerous SNP markers have been identified for soybean resistance against various diseases and have the potential to be deployed in the future (Wen et al. 2014; Vuong et al. 2015; Zhang et al. 2015a; Chang et al. 2016; Rincker et al. 2016a; Coser et al. 2017; Moellers et al. 2017; Lin et al. 2020). On the other hand, genome-editing technology (such as CRISPR/Cas9) allows plant breeders to fine-tune gene regulation toward the improvement of crop resistance to various diseases (Chen et al. 2019).

Due to the large volumes of literatures and the inconsistency in nomenclature, it would be difficult to communicate among researchers. Chang et al. (2018) summarized such challenges in soybean SDS disease and proposed a novel QTL nomenclature. Briefly, QTLs were manually assembled with their reported physical positions (flanking markers or nearest markers) based on the physical map of 'Williams82', and a validated QTL should be reported in at least three studies. In this review, we followed the idea of this nomenclature and extended it in most diseases for QTLs validated in at least two studies. Moreover, to distinguish QTLs from R genes (such as '*Rhg*'), a '*q*' was placed before the QTL name. For instance, *qRfv02-01* means the first (*01*) validated quantitative (*q*) resistance (*R*) to *Fusarium virguliforme* (*fv*) on Chr. 2 (*02*). Meanwhile, widely accepted resistance gene names were still maintained, especially those approved by U.S. Soybean Genetics Committee.

To assist soybean breeders to develop effective breeding strategies under the global climate change, reducing the world soybean yield loss due to diseases and ensure the continuous growth and sustainability of the global soybean production in the next decade, this review aims to: 1. provide comprehensive atlas of soybean genes and QTLs conferring resistance to 28 economically important and emerging diseases, including their donor source, genetic position, tightly linked markers, resistance spectrum, and testing methods; 2. validate high-quality QTLs across different studies based on the overlapping of their genomic positions; and 3. offer comprehensive future perspectives and breeding suggestions for disease-related pipelines. This review may also serve as a guideline and toolbox for soybean breeders around the world.

Section I. Soybean resistance to nematode diseases

Plant–parasitic nematodes are the major constraints for soybean production worldwide. Nematodes alone are responsible for a projected loss of $78 billion annually worldwide with a 10–15% average yield loss in soybean (Lima et al. 2017). The intensity of yield loss caused by parasitic nematodes are variable and typically depends on several factors including the nematode species, the nematode population density, management practices, the genetic background of soybean varieties, and soil and environmental factors (Bradley et al. 2021). In recent decades, nematode infestation has been spread in most soybean producing countries in the world including the USA, Brazil, Canada, South Africa, Japan, China, and India. Soybean cyst nematode, southern root-knot nematode, reniform, and lance nematodes are the major plant–parasitic nematodes in soybean around the world resulting in losses of as much as 100% (Wrather and Koening 2009; Kim et al. 2016; Bradley et al. 2021). The detailed information of each specific nematode and breeding efforts to enhance the levels of resistance is described below.

Soybean cyst nematode

Among plant–parasitic nematode species, soybean cyst nematode (SCN, caused by *Heterodera glycines* Ichinohe) is the most destructive sedentary and obligate parasite of soybean causing up to 30% yield loss (Mueller et al. 2016). The annual production losses caused by SCN are more than twice as much as any other diseases in North America, causing projected yearly losses of billions of dollars worldwide. In 1915, Japan reported the first occurrence of SCN, and later in 1954 it was identified in North Carolina, USA (Winstead et al. 1955; Riggs, 2004), and later in Ontario, Canada (Anderson et al. 1988). Subsequently, it spread to most soybean-producing countries causing severe yield losses worldwide. For instance, more than 3.5 million Mt of production losses caused by SCN were reported in 28 states of the USA (Koenning and Wrather 2010; Allen et al. 2017) corresponding to more than $1 billion in value (Liu et al. 2012). Later, SCN infestation was identified in Quebec province, Canada (Mimee et al. 2015) and some of the soybean cultivated provinces in China (Peng et al. 2016).

While crop damage due to SCN is devastating, the symptoms above the ground level are not every time noticeable, and infestations are typically only identified in the advanced phase of infection. At this stage, a significant amount of damage has already taken place. Symptoms include chlorosis, stunting, reduced root development, and

Table 1 Soybean diseases in major soybean production regions of the world

	Common disease name	Causal agent
Nematode diseases	Lance nematodes[a]	*Hoplolaimus* spp.
	Lesion nematodes	*Pratylenchus* spp.
	Reniform nematode[a]	*Rotylenchulus reniformis*
	Root-knot nematodes[a]	*Meloidogyne* spp.
	Soybean cyst nematode[a]	*Heterodera glycines*
Oomycete diseases	Downy mildew[a]	*Peronospora manshurica*
	Phytophthora root and stem rot[a]	*Phytophthora sojae, P. sanseomeana*
	Pythium damping off and root rot[a]	*Pythium* spp.
Fungal diseases	Alternaria leaf spot	*Alternaria* spp.
	Anthracnose	*Colletotrichum* spp.
	Brown spot	*Septoria glycines*
	Brown stem rot[a]	*Cadophora gregata*
	Cercospora leaf blight and purple seed stain[a]	*Cercospora kikuchii*
	Charcoal rot[a]	*Macrophomina phaseolina*
	Frogeye leaf spot[a]	*Cercospora sojina*
	Fusarium wilt and root rot[a]	*Fusarium* spp.
	Phomopsis seed decay[a]	*Phomopsis longicolla*
	Phyllosticta leaf spot	*Pleosphaerulina sojicola*
	Pod and stem blight	*Diaporthe phaseolorum* var. *sojae*
	Powdery mildew	*Erysiphe diffusa*
	Red leaf blotch[a]	*Coniothyrium glycines*
	Rhizoctonia damping-off and root rot[a]	*Rhizoctonia solani*
	Sclerotinia stem rot[a]	*Sclerotinia sclerotiorum*
	Sclerotium blight	*Sclerotium rolfsii*
	Seedling diseases[a]	*Fusarium* spp., *Alternaria* spp., *Pythium* spp. etc
	Soybean rust[a]	*Phakopsora pachyrhizi*
	Stem canker[a]	*Diaporthe phaseolorum* var. *caulivora; Diaporthe aspalathi*
	Sudden death syndrome[a]	*Fusarium virguliforme; F. tucumaniae; F. Brasiliense; F. crassistipitatum*
	Taproot decline[a]	*Xylaria necrophora*
	Target spot and root rot	*Corynespora cassiicola*
	Violet root and lower stem rot	*Rhizoctonia croccorum*
Bacterial diseases	Bacterial blight[a]	*Pseudomonas savastanoi* pv. *glycinea*
	Bacterial pustule[a]	*Xanthomonas axonopodis* pv. *glycines*
	Wildfire	*Pseudomonas syringae* pv. *tabaci*
Virus diseases	Alfalfa mosaic[a]	Alfalfa mosaic virus (AMV)
	Bean pod mottle[a]	Bean pod mottle virus (BPMV)
	Bean yellow mosaic	Bean yellow mosaic virus (BYMV)
	Brazilian bud blight	Tobacco streak virus (TSV)
	Cowpea mild mottle	Cowpea mild mottle virus (CMMV)
	Peanut mottle	Peanut mottle virus (PMV)
	Soybean dwarf[a]	Soybean dwarf virus (SbDV)
	Soybean mosaic[a]	Soybean mosaic virus (SMV)
	Soybean vein necrotic virus[a]	soybean vein necrotic virus (SVNV)

[a]Soybean diseases included in this review

decreased nodule formation (Niblack et al. 2006). Several traditional practices including biological, chemical, and physical methods have been attempted to control SCN infestation but were found inadequate for the management of the disease. The development and deployment of resistant cultivars along with crop rotation methods are the

Table 2 Validated genes/loci conferring resistance to soybean cyst nematode disease (caused by *Heterodera glycines*)

Locus/ Allele name	MLG (Chr.)	Tightly linked/ flanking markers	Marker position (Gmax2.0)	Resistance spectrum[a]	PVE[b] (%)	Population type (size)	Screening environment	Donor source	References
Rhg4	MLG A2 (Chr. 8)	*I* locus	6,638,879–8,684,157	Race 3	–	F6:7 (328)	Greenhouse	PI 437654	Webb et al. (1995)
		Satt632—SIUC-100–8		Race 1	7.3	F2:3 (250)		PI 438489B	Vuong et al. (2011)
		Satt632—SIUC-100–8		Race 3	13.4	F2:3 (250)		PI 438489B	
		–		Race 1	7.8	F2:3 (160)		SS97-6946	Islam et al. (2015)
				Race 3	9.2	F2:3 (160)		SS97-6946	
–	MLG J (Chr.16)	*B032*	33,522,390–37,517,727	Race 3	40.2	F2 (56)	Greenhouse	PI 209332	Concibido et al. (1994)
–	MLG A2 (Chr. 8)	*A085*	4,113,588–5,287,036		21.4				
–	MLG A2 (Chr. 8)	Satt233–Sat_040	1,621,167–6,169,649	Race 2	7	F2:3 (250)	Greenhouse	PI 567516C	Vuong et al. (2010)
qSCN18	MLG G (Chr. 18)	Satt612–Satt191 and Sat_210–Sat_403	52,048,435–57,332,1221,496,949–6,332,269	Race 1, 2, 3, 5, 14 and LY1	5.2–10.4	F2:3 (250)	Greenhouse		
qSCN10	MLG O (Chr. 10)	Sat_038–Satt592 and Sat_274–Satt592	41,364,845–47,422,630	Race 1,2,3 and LY1	7.9–21.7	F2:3 (250)	Greenhouse		
qSCN-PL10	MLG O (Chr. 10)	Marker1015215	42,660,451–42,881,952	HG type 7	7.73	F2:3 (200)	Greenhouse	Pingliang ZDD 11,047	Guo et al. (2020)
–	MLG B1 (Chr. 11)	–	–	Race 3	~ 91	F2:3 (200)	Greenhouse	Hartwig	Vierling et al. (1996)
		A006–Satt583	10,325,463–26,020,924	Race 1	16.6	F2:3 (250)	Greenhouse	PI 89772	Yue et al. (2001b)
–				Race 2	6.8				
		A006-A118	10,344,240–25,063,291	Race 5	9.5				
rhg1	MLG G (Chr. 18)	B053–Satt309	1,696,762–2,011,402	Race 1	26.6				
				Race 3	23.0				
				Race 5	10.0				
	MLG E (Chr. 15)	A135—Satt231	1–36,327,770	Race 3	15.7				
–	MLG D2 (Chr. 17)	B132–Satt372	2,703,565–6,607,864	Race 1	9.7				

Table 2 (continued)

Locus/ Allele name	MLG (Chr.)	Tightly linked/ flanking markers	Marker position (Gmax2.0)	Resistance spectrum[a]	PVE[b] (%)	Population type (size)	Screening environment	Donor source	References
qSCN001-01	MLG M (Chr. 7)	ss107925701–ss107918678	176,243–2,483,443	Race 3	22.4	F6:13	Greenhouse	PI 438489B	Abdelmajid et al. (2014)
qSCN001-02	MLG F (Chr. 13)	ss107920816–ss107912529	1,021,174–10,404,020	Race 3	8.9		Greenhouse		
qSCN001-03	MLG E (Chr. 11)	ss107913532–ss107930960	11,523,094–15,075,151	Race 3	16.1		Greenhouse		
qSCN002-01	MLG A1 (Chr. 5)	ss107921684–ss107919814	1–2,725,084	Race 5	43.0	F6:13	Greenhouse	PI 438489B	
qSCN002-02	MLG A2 (Chr. 8)	ss107919498–ss107930668	2,487,792–3,609,192	Race 5	43.3	F6:13	Greenhouse	PI 438489B	
qSCN002-03	MLG B1 (Chr. 11b)	ss107920383–ss107922154	9,979,503–11,537,679	Race 5	38.9	F6:13	Greenhouse	PI 438489B	
–	MLG G (Chr. 18)	Satt163–Satt688	883,910–3,341,873	Race 2	14.7	F2:3 (226)	Greenhouse	PI 90763	Guo et al. (2005)
			883,910–3,341,873	Race 3	28.1	F2:3 (226)			
			883,910–3,341,873	Race 5	13.0	F2:3 (226)			
–	MLG B1 (Chr. 11)	Satt453–Satt359	32,411,307–34,173,104	Race 2	6.7	F2:3 (226)	Greenhouse	PI 90763	
				Race 5	11.2	F2:3 (226)			
–	MLG A2 (Chr. 8)	Satt400-Satt424	7,678,989–10,846,818	Race 3	17.7	F2:3 (226)			
–	MLG E (Chr. 15)	Satt573-Satt204	14,438,759–17,330,815	Race 5	12.5	F2:3 (226)			
rhg1	MLG G (Chr. 18)	Satt038-Satt309	1,295,211—2,467,798	Race 3	0.61	F7 and F8 (115)		Toyomusume	Ferdous et al. (2006)
rhg-t1	MLG B1 (Chr. 11)	Satt453-Sat_331	32,911,928–34,766,867		0.12				
rhg-t2	MLG B1 (Chr. 11)	Satt583	26,440,896–30,354,966		0.04				
–	MLG B1 (Chr. 11)	Satt583—Sat_123	27,142,236–33,060,032	Race 1	47.3	F2 (184)		PI438489B	Yue et al. (2001b)
		Satt583—Sat_123		Race 2	45.8				
		Satt168—A329		Race 3	51.5				
		Satt583—Sat_123		Race 5	34.5				
		–		Race 14	37.2				

Table 2 (continued)

Locus/ Allele name	MLG (Chr.)	Tightly linked/ flanking markers	Marker position (Gmax2.0)	Resistance spectrum[a]	PVE[b] (%)	Population type (size)	Screening environment	Donor source	References
rhg1	MLG G (Chr. 18)	Satt309–Sat_168	1,661,117–1,785,434	Race 1	26.2	F2:3	Greenhouse	Peking	Concibido et al. (1997)
				Race 3	44.8	F2:3		PI90763	
				Race 3	6.4	F5:6 (739)		Hartwig	Prabhu et al. (1999)
				Race 1	15.0	F2:3 (250)		PI 438489B	Vuong et al. (2011)
				Race 2	8.7	F2:3 (250)		PI 438489B	
				Race 3	27.9	F2:3 (250)		PI 438489B	
				Race 1	18.8	F2:3 (160)		SS97-6946	Islam et al. (2015)
				Race 3	9.5	F2:3 (160)		SS97-6946	
rhg1	MLG G (Chr. 18)	–	1,697,102–2,467,798	–	52.7	F2:3 (76)	Greenhouse	PI 90763 PI 20933 PI 88788 Peking	Concibido et al. (1997)
					51.4				
					40.0				
					28.8				
Rhg1-b	MLG G (Chr. 18)	–	1,710,006–2,011,402	TN14 (Race 2)	–	F3 (80)	Greenhouse	PI 88788	Brucker et al. (2005)
–	MLG B2 (Chr. 14)	A593_1	45,067,577–47,207,943	Race 1,3,5	14–57.7	F2:3 (200)	Greenhouse	Peking	Qiu et al. (1999)
–	MLG D2 (Chr. 17)	–	17,878,150–27,906,833	Race 14	9.5–41.1	BC3F2:3 (126)	Greenhouse	Hartwig	Schuster et al. (2001)
cqSCN10	MLG O (Chr. 10)	Satt592, Satt331, and Sat_274	41,610,215–41,958,155	Race 2	13.5	RILs (242)	Greenhouse	PI 567305	Vuong et al. (2021)
				Race 3	34.5				
				Race 5	5.6				
cqSCN11	MLG B1 (Chr. 11)	–	35,925,243–37,749,863	Race 2	3.5	RILs (242)	Greenhouse	PI 567305	
				Race 5	5.1				
cqSCN18	MLG G (Chr. 18)	–	1,010,310–2,178,121	Race 2	22.5	RILs (242)	Greenhouse	PI 567305	
				Race 3	7.9				
				Race 5	23.0				

[a]Conversion of races to HG types are: Race 1 (HG type 2.5.7), race 2 (HG type 1.2.5.7), race 3 (HG type 0), race 5 (HG type 2.5.7), race 14 (1.3.5.6.7), race 14 (HG type 1.3.6.7), race LY1 (1.2.3.4.5.6.7)

[b]Phenotypic variations explained by the molecular markers

preferably efficient practice for the management of SCN (Davis and Tylka 2000).

Breeding for SCN resistance involves the genetic mapping of QTLs/genes associated with the resistant phenotype and understanding the underlying resistance mechanism. The first *Rhg* (resistance to *H. glycines*) locus was reported around the mid-1950s (Ross and Brim 1957) which described plant introductions (PIs) 88,788 and 'Peking' (PI 548,402) as sources of SCN resistance. These two accessions were integrated into the soybean breeding programs through cycles of backcrossing. With the rapid progress in the availability of molecular markers and mapping techniques, numerous SCN-resistance loci have been reported by the soybean research community. Table 2 summarizes the main reported QTLs linked to SCN resistance. In soybean, SCN resistance trait is typically multi-genic and quantitatively inherited (Anand and Rao-Arelli 1989; Guo et al. 2005; Vuong et al. 2010, 2011). The resistance found in Peking was governed by three independent recessive genes (Caldwell et al. 1960). Since then, numerous genes/QTLs conferring SCN resistance have been mapped to date. Among these QTLs, two loci *rhg1* and *Rhg4* found on chromosomes 18 and 8, respectively, which confers resistance to SCN races 1, 2, 3, 4, and 5, have been extensively investigated (Kim et al. 2016). In diverse soybean germplasm lines, the *rhg1* locus has been constantly mapped and identified at a sub-telomeric region on the chromosome (Chr.) 18 (Kim et al. 2016). Using *rhg1*, several markers were developed, of which Satt309 (predicted at about 0.4 cM from *rhg1* locus) has been extensively applied for MAS in soybean research (Cregan et al. 1999; Silva et al. 2007). Another major QTL for SCN resistance showed a total phenotypic variation of about 9–28% to SCN HG types 2.5.7 (race 1) and 0 (race 3) and was described as *Rhg4* gene from different resistant plant accessions (Concibido et al. 2004). Meksem et al. (2001) described that *rhg1* and *Rhg4* equally demonstrated about 98% of phenotypic variation in the 'Forrest' cultivar conferring resistance to race 3 of SCN. *Rhg4* mediated resistance is largely associated with race 3 of SCN, in addition to some minor resistance against race 2 (HG types 1.2.5.7), race 1 (2.5.7), and race 14 (1.3.6.7). In Peking and PI 437654 accessions, *rhg1* and *Rhg4* loci are essential to provide complete resistance against some SCN races. QTL mapping in PI 567516C identified two SCN-resistance QTLs on chromosomes 10 and 18, which were not linked to major *rhg1* or *Rhg4* loci (Vuong et al. 2010). These QTLs conferred resistance against races 1, 2, 3, and LY1 of SCN (Young 1998). Interestingly, the QTL detected on Chr. 18 is far away from the *rhg1* locus. Another two QTLs were mapped on chromosomes 10 and 18 in PI 567,305 (Kim et al. 2016) and were showing elevated resistance to various SCN HG types, identical with the study demonstrated by Vuong et al. (2010) in PI 567516C. Therefore, these results indicated that both PI 567,305 and PI 567516C harbor novel QTLs which can provide SCN resistance. Recently, the genetic analysis of the PI 567,305 line through Infinium SoySNP6K BeadChips and genotype-by-sequencing (GBS) revealed major QTLs on chromosomes 10 and 18 (Vuong et al. 2021) conferring resistance to SCN as well as other two important nematode species such as root-knot and reniform nematodes. The unique genetic structure of PI 567,305 investigated using haplotype and copy number variation analysis suggested the presence of different resistance mechanisms from PI 88,788 or Peking-type.

In addition, three resistance loci for race 3 of SCN were detected in a GWA study of 282 soybean accessions, among which two out of these three were correlated to *rhg1* and earlier mapped, FGAM1, SCN-resistance locus whereas the third one was positioned at Chr. 18 (Zhang et al. 2017). About 8 novel QTLs for resistance to race 3 of SCN was also identified by Vuong et al. (2011). Furthermore, 13 significant SNPs for SCN resistance were also identified in 7 diverse genomic regions by Zhang et al. (2017). Out of these 13, 10 SNPs were novel, whereas the remaining 3 were linked to earlier mapped QTLs including *rhg1* and *Rhg4*. An investigation performed by Zhao et al. (2017) demonstrated the identification of 13 important SNPs (4 novels) on five chromosomes which conferred resistance to SCN race 1. Later, twelve SNPs significantly linked to SCN resistance were identified on chromosomes 7, 8, 10, and 18. Of these twelve, three were positioned close to the *rhg1* locus (Tran et al. 2019). Using these data, multiple candidate genes conferring SCN resistance have been discovered. Liu et al. (2019) described 10 genes having 27 mutations, among which three genes overlapped between the two phenotypic mutants suggesting possible involvement of these genes in nematode resistance.

The copy number of *rhg1* has been categorized into two repeat types such as high (> 6 repeats, as in PI 88,788) and low (about 3 repeats, as in Peking) (Cook et al. 2012). Yu et al. (2016) demonstrated that, in the case of *rhg1*, both gene-based polymorphism and copy number variation were significantly important for SCN resistance. It also indicated that *rhg1* resistance sources with a high copy number provided elevated resistance against SCN. Altogether it was proposed that *rhg1* locus may facilitate SCN resistance through copy number variation of numerous genes encoding amino acid transporter (AAT), a WI12 (wound-inducible) protein, and an α-soluble N-ethylmaleimide-sensitive factor (NSF) Attachment Protein (α-SNAP) (Kandoth et al. 2017; Liu et al. 2017). Furthermore, Patil et al. (2019) categorized the *rhg1-b* locus into two classes, *rhg1-b* (like lines of PI 88,788-type) and *rhg1-b1* (like lines of Cloud-type) and revealed genetic basis of broad-spectrum resistance through interactions of copy number

variation among *rhg1* and *Rhg4* genes. Liu et al. (2012) reported that the resistance at the *Rhg4* locus was provided through the serine hydroxymethyltransferase (*SHMT*) gene, whose encoding protein catalyzes the reversible conversion of tetrahydrofolate and serine to tetrahydrofolate and glycine, respectively. The two polymorphisms in the gene *GmSHMT08* positioned at the first and second exons, 389 G/C and 1165 A/T, results in modification of amino acids such as arginine vs. proline and tyrosine vs. asparagine, respectively, and further alteration of the kinetic properties (Liu et al. 2012). *GmSHMT08* encoded protein shows a multifarious role in addition to essentially being involved in the enzymatic reaction of SCN resistance (Kandoth et al. 2017). It has additional functions including structural stability, ligand binding, and interactions with other proteins (such as GmSNAP18). Kandoth et al. (2017) showed that *rhg1-a* allele is required in Forrest cultivar for SCN resistance although it does not impart any selection pressure on nematodes to shift from HG type 7. However, the nematodes were exposed to EXF67 cv. shifted to HG type 1.3.6.7 indicating the bi-genic phenomenon of resistance and necessity of *Rhg4* in Peking-type facilitating resistance.

Cook et al. (2014) showed the distribution of nonsynonymous SNPs in the *GmSNAP11* gene, its paralogous copy identified as *GmSNAP18*, with novel alleles that participated in SCN resistance, especially α-SNAP is crucial for resistance in soybean varieties derived from PI-88788. Further, Lakhssassi et al. (2017) demonstrate that the predicted protein of α-SNAP corresponds to truncated GmSNAP11 and not to GmSNAP18 (289 amino acids, aa). GmSNAP11 exists in Forrest Pecking type in two different forms such as GmSNAP11-T1 (239 aa) and GmSNAP11-T2 (244 aa). A nonsynonymous SNP known as *map-5149* tightly linked to resistance against race 3 of SCN was identified in *GmSNAP11* (Li et al. 2016a, b, c). Altogether, these results suggest the novel nature of *GmSNAP11* providing SCN resistance in soybean.

Marker-assisted selection (MAS) is an effective and routinely performed strategy to develop SCN resistant soybean lines, representing the most rapid, cost-effective, accurate, and reliable method. Shi et al. (2015) developed functional Kompetitive Allele-Specific PCR (KASP) marker assays (*GSM381* and *GSM383* at *rhg1; GSM191* at *Rhg4*) which were effectively applied for rapid and quick selection of SCN resistance, as well as identification of Peking and PI 88,788 types of resistance. Kadam et al. (2016) developed KASPar (KBioscience Competitive Allele-Specific PCR) assays from SNPs at *rhg1*, *Rhg4*, and other novel QTLs. They effectively differentiated the copy number variation at *rhg1* into three groups including (1) high resistant such as PI 88,788 type, (2) low copy resistant such as Peking type, and (3) susceptible single copy such as Williams82 type numbers. Tian et al. (2019) developed cleaved amplified polymorphic sequences (CAPS) markers using *GmSNAP11* (minor resistant to SCN) and combined with markers Rhg-389 and rhg1-2 for genotyping a panel consisting of 209 soybean accessions with variable SCN resistance.

The underlying molecular mechanisms of SCN resistance are complex and yet to be unveiled. Some studies suggested that there could be several disease-resistance proteins involved in SCN resistance, comprising Nucleotide-binding site-leucine-rich repeats (NBS-LRR), cytochrome P450s, RING domain proteins, zinc-finger domain proteins, protein kinases, transcription factors such as MYB and WRKY. Kofsky et al. (2021) studied the transcriptome of wild SCN resistant soybean (*Glycine soja*) ecotype, 'NRS100', and proposed biochemical mechanisms. This included the downregulation of the jasmonic acid (JA) signaling pathway to permit resistance response led by salicylic acid (SA) signaling-activation and polyamine synthesis which further maintains structural stability of root cell walls.

Soybean root-knot nematode

Root-knot nematodes (*Meloidogyne* spp.) are considered the most economically important and widely distributed parthenogenic plant–parasitic nematodes in the world (Trudgill and Blok 2001). Southern Root-knot nematode [SRKN, *M. incognita* (Kofold & White) Chitwood] was considered as one of the major plant–parasitic nematodes based on scientific and economic importance (Jones et al. 2013). The observed symptoms of SRKN in soybean are similar with the symptoms of abiotic stresses, including stunted growth, wilting, leaf discoloration, and deformation of the roots. The magnitude of crop losses depends on historical crop rotation and field usage, environmental parameters, initial nematode population density, soil type, and genetic background (Vieira et al. 2021).

SRKN is challenging to control due to its short life cycle and high reproductive rates (Trudgill and Blok 2001). Chemical approaches used to be an effective management option, however, most commercial nematicides and soil fumigants were banned due to toxicity to humans, animals, and environments (Abad et al. 2008). Crop rotation is especially challenging and limited since most flowering plants are hosts to SRKN. The use of genetic resistance becomes the most sustainable—economically, environmentally, and socially—alternative to efficiently control the damage caused by SRKN in soybean (Vieira et al. 2021).

Significant efforts have been taken to identify soybean accessions resistant to SRKN. Luzzi et al. (1987) screened

Table 3 Soybean loci conferring high resistance to southern root-knot nematode (caused by *Meloidogyne* spp.)

MLG (Chr.)	Locus/allele name[a]	Tightly linked/flanking markers	Marker position (Gmax2.0)	Testing methods/ Resistance spectrum	PVE[b]	Population type (size)	Donor source	References
MLG C2 (Chr. 6)	–	Satt286 and Satt365	16,200,000–19,600,000	Greenhouse test/Race *3*	–	F2:4 (35)	PI 96354	Shearin et al. (2009)
MLG M (Chr.7)	–	Satt201 and Satt590	1,301,315–2,025,244	Greenhouse test/Race *2*	62.4%	F2:3 (69)	LS 5995	Fourie et al. (2008)
MLG A2 (Chr. 8)	–	BARC-051847–11,270 and BARC-039273–07,476	22,048,168–35,856,368	Greenhouse test/Race *3*	6.4%	F8:9 (246)	PI 438489B	Xu et al. (2013)
MLG O (Chr. 10)	*qRmi10-01*	G248A-1	1,018,664–1,881,027	Greenhouse test/Race *3*	31%	F2:3 (110)	PI 96354	Tamulonis et al. (1997)
		Satt492 and Satt358	1,018,664–1,881,027	Greenhouse test/Race *3*	55.8%	F2:3 (110)	PI 96354	Li et al. (2001a)
		Satt500 and Satt358	1,018,500–1,395,790	Greenhouse test/Race *2*	31.7%	F2:3 (69)	LS 5995	Fourie et al. (2008)
		BARC-065469–11,494 and BARC-018101–02,517	1,571,105–2,067,005	Greenhouse test/Race *3*	23.6%	F8:9 (246)	PI 438489B	Xu et al. (2013)
		BARCSOYSSR-10–0090 and BARCSOYSSR-10–0105	1,470,000–1,640,000	Greenhouse test/Race *3*	50%	F5:6 (269)	PI 96354	Pham et al. (2013)
		ss715605654	1,507,123–1,519,325	Greenhouse test/Race *3*	–	PI Panel (193)	PI 96354	Passianotto et al. (2017)
MLG F (Chr. 13)	–	BARC-010501–00,676 and Sct-033	28,826,405–30,078,140	Greenhouse test/Race *3*	4.8%	F8:9 (246)	PI 438489B	Xu et al. (2013)
MLG G (Chr.18)	*qRmi18-01*	K493h-1 and Cs008D-1	47,201,155–50,158,095	Greenhouse test/Race *3*	14.4%	F2:3 (110)	PI 96354	Tamulonis et al. (1997)
		Satt012 and Satt505	47,201,155–50,158,095	Greenhouse test/Race *3*	17.7%	F2:3 (110)	PI 96354	Li et al. (2001a)
		ss715631954	47,201,155–50,158,095	Greenhouse test/Race *3*	5%	F5:6 (269)	PI 96354	Pham et al. (2013)

[a]Locus name given in this study, if the physical positions of QTLs overlap each other in at least two independent studies. *qRmi10-01* means the 1st (01) quantitative (q) resistance (R) to *M. incognita (mi)* on Chr. 10 (10)

[b]Phenotypic variations explained by the molecular markers

over 2700 soybean accessions from the USDA Soybean Germplasm Collection and found that 'Amredo', PI 96,354, PI 408,088, and PI 417,444 showed lower gall indices, fewer eggs per root system, and eggs per gram of root than the resistant check Forrest (PI 548,655) (Luzzi et al. 1987). Harris et al. (2002) screened 608 PIs from Southern China and reported that PI 594753A and PI 594775A had similar resistance levels as PI 96,354 (Harris et al. 2002). The first report on the genetic control of the resistance to SRKN indicated that reduced galling in the cultivar Forrest was controlled by a single dominant gene designated as *Rmi1* (Luzzi et al. 1994a). Hybridizations between PI 96,354 × Forrest and Forrest × PI 417,444 resulted in individual F_3 plants and F_3 populations with higher galling than Forrest, PI 96,354, and PI 417,444, implying the resistance from Forrest (*Rmi1*) differs from PI 96,354 and PI 417,444 by at least one gene (Luzzi et al. 1994b).

The first genetic mapping of resistance to SRKN (race 3) in soybean identified two QTLs on chromosomes 10 and 18, accounting for 31% and 14% of phenotypic variation, respectively (Tamulonis et al. 1997). The combination of both resistance QTLs enhanced the levels of resistance to SRKN race 3, the predominant race in the U. S. (Li et al. 2001a). An additional major QTL on Chr. 7 accounting for 62% of the phenotypic variation was reported to confer resistance to SRKN race 2, a predominant race in soybean production areas of South Africa (Fourie et al. 2008). In addition, two minor QTLs on Chr. 8 (7.4% of the phenotypic variation) and 13 (5.6% of the phenotypic variation) were reported to confer resistance to SRKN race 3 (Xu et al. 2013) (Table 3).

To better understand the mechanisms of soybean resistance to root-knot nematode, fine-mapping analyses were conducted for the major QTL on Chr. 10. Pham et al. (2013) identified three candidate genes with cell wall modification-related functions, including *Glyma.10g016600* (Extensin 1 encoding function), *Glyma.10g016700* (Extensin 2 encoding function), and *Glyma.10g017100* (Pectinesterase 1 encoding function). In another independent study, five candidate genes were identified, including *Glyma.10g017100, Glyma10g02150, Glyma.10g017200, Glyma.10g017300,* and *Glyma.10g017400*, all with pectinesterase encoding-related functions (Xu et al. 2013). Moreover, a GWA study using a panel of diverse soybean accessions narrowed down this QTL to a 12-kb region with five significant single nucleotide polymorphisms (SNPs) located within *Glyma.10g017100* accounting for 25 to 40% of phenotypic variations (Passianotto et al. 2017).

Multiple reports have shown that SRKN resistant soybean genotypes can sustain yield under variable levels of nematode infection. Yield suppression can reach as much as 97% in susceptible genotypes while resistant genotypes may show less than 1% (Herman et al. 1990). Kinloch et al. (1984) reported a negative correlation between yield and number of galls under high pressure, which translated in resistant cultivars yielding as much as 5 times greater than highly susceptible cultivars (Kinloch et al. 1984). Vieira et al. (2021) evaluated the yield performance of 202 elite soybean lines in field conditions with variable distributions of SRKN and reported resistant lines yielding on average 20% higher than susceptible lines. The presence of the major resistance allele on Chr. 10 reduced yield losses by approximately sixfold in comparison to the susceptible group (1.1% and 6.2% per 1000 SRKN second-stage juveniles in 100 cm^{-3}, respectively), which provided significant yield protection under high SRKN pressure (Vieira et al. 2021). However, because of the high concentration and wide distributions of SRKN, the limited and narrow base of genetic resistance, and lack of alternative management options, a resistance-breaking population in soybean could result in devastating yield losses (Vieira et al. 2021). Consequently, further work is needed to unveil and stack novel sources of resistance resulting in enhanced and more durable resistance in the future (Vieira et al. 2021).

Reniform nematode and Lance nematode

Reniform nematode (*Rotylenchulus reniformis* Linford & Oliveira) (RN), a sedentary semi-endoparasite, first emerged in Hawaii on cowpeas [*Vigna unguiculata* (L.) Walp.] in 1931 and was identified in Georgia, USA, in 1940 (Linford and Oliveira 1940; Smith 1940; Gavilano et al. 2013). It has now become a major yield-limiting parasitic nematode species in soybean growing areas in southern and southeastern states of the USA, due to its wide range of hosts (over 300 plant species), and the ability of surviving in broad soil range and dry soil for an extended period (Herald and Thames 1982; Herald and Robinson 1990; Wrather et al. 1995; Robinson et al. 1997; Robbins et al. 1999; Koenning and Wrather 2010). The infestation on the roots of the host is initiated by the vermiform female adults, which is different from common sedentary endo-parasitic nematode genera (*Heterodera, Globodera*, and *Meloidogyne*). Female RN establish feeding sites known as syncytium and eventually become sedentary. The common name of RN refers to its kidney shape characteristics. The male RN are involved in mating but do not feed (Linford and Oliveira 1940; Gaur and Perry 1991; Ganji et al. 2013; Robbins 2013). Typical symptoms of RN infection include root decay, stunting, and foliar chlorosis (Cook et al. 1997; Kinloch 1998; Rivera and Thiessen 2020). Annual soybean yield losses of up to 33% were reported in soybean cultivars that were partially or not

Table 4 Soybean loci conferring resistance to reniform nematode (caused by *Rotylenchulus reniformis*)

MLG (Chr.)	Locus/allele name[a]	Other name	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
MLG B1 (Chr. 11)	*qRrr11-01*	GmSNP11	Sat_123 and BARC-018869–03,031	(32,194,583–33,581,636)	Partial Greenhouse assay	F8 (247)	11.3%	PI 438489B	Usovsky et al. (2021)
		–	Satt359	102.55 cM* (32,411,307)	Partial Greenhouse assay	F6 derived RILs (228)	16%	PI 437654	Ha et al. (2007)
	–	–	BARC-021459–04,106	120.13 cM* (38,902,736)	Partial Greenhouse assay	F6:9 (247)	8.9%	PI 567516C	Jiao et al. (2015)
MLG H (Chr. 12)	–	–	Satt353	8.48 cM* (1,687,387)	Partial Greenhouse assay	F5:16 (92)	10%	Hartwig (PI 437654)	Lee et al. (2016)
MLG G (Chr. 18)	–	–	Satt163	0 cM*	Partial Greenhouse assay	F5:16 (92)	13.5%	Hartwig (PI 437654)	Lee et al. (2016)
	–	–	Satt275	2.2 cM*	Partial Greenhouse assay	F5:16 (92)	10%	Hartwig (PI 437654)	Lee et al. (2016)
	qRrr18-01	GmSNP18	BARC-055551–13,421 and BARC-048275–10,534	(1,308,798–1,705,500)	Partial Greenhouse assay	F8 (247)	7.3%	PI 438489B	Usovsky et al. (2021)
		–	BARC-012237–01,756	(1,685,571)	Partial Greenhouse assay	F6:9 (247)	7.5%	PI 567516C	Jiao et al. (2015)
	–	–	Sat_168	3.9 cM* (1,706,200)	Partial Greenhouse assay	F6 derived RILs (228)	8%	PI 437654	Ha et al. (2007)
	–	–	Satt309	4.53 cM* (1,736,692)	Partial Greenhouse assay	F5:16 (92)	13.2%	Hartwig (PI 437654)	Lee et al. (2016)
MLG L (Chr. 19)	–	–	Satt513	106.37 cM* (49,223,526)	Partial Greenhouse assay	F6 derived RILs (228)	21%	PI 437654	Ha et al. (2007)

*GmComposite2003 genetic position (www.soybase.org)

[a]Locus name given in this study, if the physical positions of QTLs overlap each other in at least two independent studies. For example, *qRrr11-01* means the 1st (01) validated quantitative (q) resistance (R) to *Rotylenchulus reniformis (rr)* on Chr. 11 (11)

[b]Marker position (bp) based on the *Glycine max* genome assembly version Glyma.Wm82.a2 (Gmax2.0), only starting position is shown for SSR markers

[c]Phenotypic variations explained by the molecular markers

resistant to RN, resulting in an average loss of 28,000 Mt in southern USA in 2019 (Kim et al. 2016; Allen et al. 2020). Like other nematode pests, deployment of resistant varieties has been the most effective and economical strategy to control RN in soybean field (Kim et al. 2016).

The relationship between SCN and RN has drawn interest since they both establish syncytium as their feeding sites. Early literatures reported that there were common sources of resistances for SCN and RN (Rebois et al. 1970). Field and greenhouse screening assays were subsequently conducted, and the studies indicated that soybean cultivars that derived their resistance from PI 88,788 were resistant to SCN but susceptible to RN whereas cultivars that derived their resistance from Peking and PI 437,654 were resistant to both SCN and RN (Robbins et al. 1994a, 1994b, 1999; Robbins and Rake 1996). Greenhouse screening assays were commonly used to evaluate RN resistance for soybean. Disease screening protocol for RN was well-established by Robbins et al. (1999), in which the reproductive index (RI) was calculated based on the number of nematodes at test termination (Pf) and initial infestation density (Pi) (RI = Pf/Pi). High level of RN resistance has been reported in soybean cultivars including Peking, 'Dyer', 'Custer', Pickett', Forrest, 'Hartwig', and 'Anand' (Rebois et al. 1968; Robbins et al. 1994b; Davis et al. 1996). Lee et al. (2015) also reported RN resistance in PI 404198A, PI 438,498, PI 467,327, PI 468,915. PI 494,182, PI 507,470, PI 507,471, PI 507,476, and PI 567,516, all showing similar or less RI than the resistant check Anand.

Three QTLs conferring RN resistance in soybean have been identified on chromosomes 11, 18, and 19, respectively, from PI 437,564 (Ha et al. 2007). Other studies have reported and confirmed resistant loci on chromosomes 8 (Lee 2021), 11 (Jiao et al. 2015; Wilkes et al. 2020; Usovsky et al 2021), 12 (Lee et al. 2016), 13 (Lee 2021), 15 (Lee 2021), and 18 (Jiao et al. 2015; Lee et al. 2016; Wilkes et al. 2020; Lee 2021; Usovsky et al. 2021). Recently, Usovsky et al. (2021) discovered the pleiotropic effect of two genes [*GmSNAP18* (*rhg1-a, rhg1-b, and rhg1-b1* allele) and *GmSNAP11* (*qSCN11* locus)], conferring resistances to both SCN and RN in PI 438489B using universal soybean linkage panel (USLP 1.0) and next-generation whole-genome resequencing (WGRS) technology (Table 4).

Lance nematodes (*Hoplolaimus* spp.) (LN) are migratory ecto-endo plant–parasitic nematodes that are widespread throughout the USA (Sher 1963; Astudillo and Birchfield 1980; Yan et al. 2016). A total of seven species have been identified and reported in the southeastern USA, including *Hoplolaimus galeatus* Thorne, 1935; *H. columbus* Sher, 1963; *H. magnistylus* Robbins, 1982; *H. stephanus* Sher, 1963; *H. seinhorsti* Luc, 1958; *H. tylenchiformis* von Daday, 1905; and *H. concaudajuvencus* Golden and Minton, 1970 (Lewis and Fassuliotis 1982; Robbins 1982; Koenning et al. 1999). However, only three species (*H. columbus*, *H. galeatus*, and *H. magnistylus*) have been considered economically important lance nematodes in soybean production in the USA (Holguin et al. 2016). The outbreak of *H. columbus* was first detected in South Carolina and predominantly prevailed in South Carolina, North Carolina, and Georgia while *H. galeatus* and *H. magnistylus* were commonly reported in soybean production areas in Alabama, Arkansas, Mississippi, and Tennessee (Lewis and Fassuliotis 1982; Robbins 1982; Koenning et al. 1999). These nematodes primarily damage the structures of the epidermis and cortex in the root (Lewis and Fassuliotis 1982; Lewis, 1989) and cause root stunting/shedding, foliar chlorosis, as well as severely limiting lateral root growth under heavy infestations (Kinloch 1998; Timper 2009). Soybean yield losses from the infestation of these LN species can be as high as 70% (Mueller and Sanders 1987; Noe 1993). Although the resistance of host plants is the most effective way to control plant–parasitic nematodes, efforts to identify genetic resistance for LN have been limited. Therefore, the application of field sanitation and crop rotation with non-host crops is helpful to control LN populations and reduce LN damage in soybean production areas.

Section II. Soybean resistance to oomycete diseases

Crop germination and stand are key factors for a successful cropping season for soybean growers. During seed establishment, seedlings are subject to attack by several soilborne pathogens, resulting in lack of germination, damping-off or plant death. Poor plant stands due to diseases result in replanting and increased costs. Among the soilborne pathogens impacting soybean are the oomycetes, which include *Phytophthora*, *Pythium,* and *Phytopythium.* The impact of these soilborne diseases is not only limited to the beginning of the season, as root infections can occur at later stages, often reducing yield without significant above ground symptoms. In 2005, losses to soybean seedling diseases in the USA were estimated at 0.89 million Mt (Wrather and Koenning 2009). From 2006 to 2009, soybean yield losses due to seedling diseases have increased considerably ranking second only to soybean cyst nematode (Koenning and Wrather 2010). There are also oomycete diseases that occur in the canopy, like downy mildew caused by [*Peronospora manshurica* (Naum.) Sdy.], which under conducive conditions could affect seed quality and yield (Dunleavy 1987). Key species have been recognized as major contributors in disease development

Table 5 Soybean resistance genes/alleles (*Rps*) and validated QTL/loci conferring resistance to Phytophthora root and stem rot (caused by *P. sojae* and other *Phytophthora* spp.)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
P. sojae	MLG D1b (Chr. 2)	*RpsZS18*	–	Indelwz1, Indelwz2, Indelwz3, Indelwz4, and Indelwz5	(43,411,141,/43,423,820/ 43,425,227/ 43,429,008/ 43,434,948 a2)	Hypocotyl inoculation/ *PsRace1, PsRace3, PsRace4, PsRace5, PsUSAR2, Ps41-1, PsNKI, PsFJ2, PsFJ3, Ps6497*	F2:3 (232)	R gene	Glyma.02g245700, Glyma.02g245800, and Glyma.02g246300	Zaoshu18	Yao et al. (2010), Zhong et al. (2018b)
	MLG N (Chr. 3)	*Rps1a*	–	Satt159 (BARCSOYSSR_03_0180) and Satt009 (BARCSOYSSR_03_0226)	(3,197,998—3,932,012 a2)	Hypocotyl inoculation/Races *1, 2, 11, 13, 14, 15, 16, 17, 18, 26, 27, 31, 32, 36, 48, 50, 51, 52, 54, 55,* and isolates *PsHJL1, PsAH4, PsFJ, PsAH1, PsAM1, PsTA3, PsJS9, PsJS8, Ps52, PsJN4, pmg(1)-3, pmg(10)-1, pmg(13)-1, pmg(17)-1, OHSS03WayneBerry5, OHSS04WayneTBH62xx, OH8only,*	F2:3 (81), F2:3 (84)	R gene	–	Mukden, Harlon, Harsoy12xx L59-731 Union, L88-8470	Bernard et al. (1957), Diers et al. (1992), Weng et al. (2001), Dorrance et al. (2004), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a), Lin et al. (2013), Jang and Lee (2020)
		Rps1b	–	Satt152 (BARCSOYSSR_03_0192) and Satt530	(3,366,546—5,669,644 a2)	Hypocotyl inoculation/Races *1, 3, 4, 5, 6, 7,8, 9, 11, 13, 14, 15, 18, 21, 22, 24, 26, 27, 34, 36, 37, 40, 42, 43, 44, 46, 48, 49, 50, 51, 52, 54, 55,* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsFJ, PsAH3, PsXJ, PsJS9, Ps41-1, PsJS7, PsJS8, PsMC1, PsHY33-1, pmg(1)-3, 94–14-432(2), 94-13p-197, pmg(5)-3, 95–11-117(4), pmg(8)-3, pmg(13)-1, ISA 71D-1, OH199915.5.2.1, OH2000Wood25, OH2000Sandusky74, OHSS03HenryCo3, OHSS04WayneTBH62xx, PS03-113*	F2:3 (160–274), F2:3 (113)	R gene	–	PI 172901, Haro 13xx, L77-1863	Mueller et al. (1978), Ploper et al. (1985), Demirbas et al (2001), Dorrance et al (2004), Sugimoto et al (2012), Gunadi (2012), Zhang et al (2013a), Lin et al (2013), Jang and Lee (2020)
		Rps1c	–	Satt152 (BARCSOYSSR_03_0192) and Satt584 (BARCSOYSSR_03_0442)	(3,366,546—9,228,056 a2)	Hypocotyl inoculation / Races *1, 2, 3, 6, 7, 8, 9, 11, 13, 15, 17, 21, 23, 24, 26, 28, 29, 30, 32, 34, 36, 41, 42, 44, 48, 50, 52, 54, 55,* and isolates *PsJMS3, PsHJL1, PsAH4, PsFJ, PsAH3, PsXJ, PsBr1, PsTA3, PsJS9, Ps41-1, PsJS7, PsJS8, Ps52, Ps53, PsAH5, PsHY33-1, pmg(1)-3, 94–14-432(2), 95–11-117(4), pmg(8)-3, pmg(10)-1, pmg(13)-1, pmg(17)-1, 96-13S-106A.1, ISA 19A-1, ISA 19B-2, ButMu4S2, M1-1–1, M1-1–3, M3-3–1, M6-3–1, OH199915.5.2.1, OHSS03HenryCo3, OHSS04WayneTBH62xx, PS04-257, PS04-323*	F2:3 (95)	R gene	–	L75-3735, Williams79 L77-1727, L85-129	Mueller et al. (1978), Demirbas et al. (2001), Dorrance et al. (2004), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a), Lin et al. (2013), Jang and Lee (2020)
		Rps1d	–	Satt152 (BARCSOYSSR_03_0192)	(3,366,546 a2 and 3,465,436 a1)	Hypocotyl inoculation/Races *1, 2, 3, 4, 5, 7, 9, 11, 13, 14, 15, 16, 18, 20, 21, 22, 23,*	F2:3 (47)	R gene	–	Haro 16, PI 103091, L99-3312	Buzzell and Andrson (1992), Demirbas et al. (2001), Dorrance et al.

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
				and Sat_186 (BARCSOYSSR_03_0204)		*24, 25, 27, 28, 29, 30, 32, 34, 36, 37, 39, 45, 46, 47, 48, 49, 50, 51, 52, 53, 55,* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsJL4-1, PsJL3-2, PsFJ, PsAH3, PsAH1, PsBr1, PsTA3, PsMC1, PsHY33-1, pmg(1)-3, 94–14-432(2), pmg(5)-3, 95–11-117(4), pmg(10)-1, pmg(13)-1, 95–15-15, pmg(25)-1, 96-13S-106A.1, ISA 19A-1, ISA 19B-2, ISA 71D-1, M6-3–1, OH2000Sandusky74, OHSS03HenryCo3, OHSS03WayneBerry11, OHSS03WayneBerry5, OHSS04WayneTBH62xx, OH8only, PS03-113, PS04-323*					(2004), Sugimoto et al. (2007), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a), Lin et al. (2013), Jang and Lee (2020)
		Rps1k	–	CG1 and TC1	–	Hypocotyl inoculation/Races *1, 2, 3, 4, 5, 6, 7, 8, 9, 11, 13, 14, 15, 17, 18, 20, 21, 22, 23, 24, 26, 36, 37, 42, 43, 44, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55,* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsFJ, PsXJ, PsAH1, PsBr1, PsTA3, PsJS9, Ps41-1, PsJS7, PsJL5, PsJS8, Ps52, PsJN4, PsAH5, PsHY33-1, pmg(1)-3, 94–14-432(2), 94-13p-197, pmg(5)-3, 95–11-117(4), pmg(8)-3, pmg(10)-1, pmg(13)-1, pmg(17)-1, ISA 71–1, M1-1–1, M1-1–3, M3-3–1, M6-3–1, OH199915.5.2.1, OH2000Sandusky74, OH2000Wood25, OHSS03HenryCo3, OHSS04WayneTBH62xx, PS03-113*	F2:3 (111)	R gene	AY963292 and AY963293	Williams82, L77-1794	Bernard and Cremeens (1981), Kasuga et al. (1997), Demirbas et al. (2001), Dorrance et al. (2004), Gao et al. (2005), Bhattacharyya et al. (2005), Gao and Bhattacharyya (2008), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a), Lin et al. (2013)
		Rps7	–	Satt009 (BARCSOYSSR_03_0226) and Satt125 (BARCSOYSSR_03_0564)	(3,932,012—18,415,620 a2)	Hypocotyl inoculation/Races *10, 11, 12, 16, 18, 19, 35, 36,* and isolates *PsJL1-1, PsJM3, PsHJL1, PsAH4, PsJL4-1, PsAH1, PsBr1, PsJS9, PsJS7, M1-1–3, M3-3–1, M6-3–1*	F2:3 (81)	R gene	–	OX281, Harosoy, L93-3258	Anderson and Buzzell (1992), Lohnes and Schmitthenner (1997), Weng et al. (2001), Dorrance et al. (2004), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a), Jang and Lee (2020)
		Rps9	–	Satt631 (BARCSOYSSR_03_0162) and Sat_186 (BARCSOYSSR_03_0204)	(2,943,932 a2)	Hypocotyl inoculation/isolates *PNJ1, PNJ4, Pm2, Pm28, Pmg*	F1 (38, 35) and F2:3 (199, 259),	R gene	–	Ludou4	Wu et al. (2011a)
		RpsUN1	–	(BARCSOYSSR_03_0233 and BARCSOYSSR_03_0246)	(4,020,587—4,171,359 a2)	Hypocotyl inoculation/races *1, 3, 5, 7, 8, 10, 13, 17, 24, 28,* and isolates *ISA 19A-1* and *ISA 19B-2*	F2:3 (826)	R gene	Glyma.03g034600	PI 567139B	Lin et al. (2013), Li et al. (2016a, b, c)
		RpsYu25/ RpsYD25	–			Hypocotyl inoculation/isolates *PsMC1, Pm2, PNJ1, PNJ3,*	F2:3 (1127)	R gene	Glyma.03g034700, Glyma.03g034800	Yudou 25	Fan et al. (2009), Sun et al. (2011), Zhang

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
				SSRYZ35, SSRYZ37, SSRYZ40, SSRYZ42, and BARCSOYSSR_03_0247	(4,150,768/4,158,266/ 4,161,570/4,204,277/ 4,205,985 a2)	*PNJ4, 6497, 7063, Pm28, Pm14, Pm31, H15, AH, PsJL1-1, PsJMS3, PsAH4, PsJL4-1, PsJL3-2, PsSX1, PsAH3, PsXJ, PsAH1, PsTA3, PsJS9, Ps41-1, PsJS7, PsJL5, PsJS8, PsJN4*			and Glyma.03g034900		et al. (2013a), Zhong et al. (2020)
		RpsYD29	–	SattWM82-50 and Satt1k4b	(3,857,715 and 4,062,474 a1)	Hypocotyl inoculation / isolates *PsHLJ5, PsHLJ3, PsJMS3, PsJL1-1, PsHLJ1, PsHLJ4, PsAH4, PsJL4-3, PsGZ2, PsJL3-2, PsSX1, PsJS4, PsJL4-1, Ps41-1, PsAH3, PsAH1, PsJS9, PsJS7, PsJS8, PsMC1, PsJN4*	F2:3 (214)	R gene	Glyma03g04030 and Glyma03g04080	Yudou 29	Zhang et al. (2013a)
		RpsHN	–	SSRSOYN-25 and SSRSOYN-44	(4,227,863–4,506,526 a1)	Hypocotyl inoculation/pathotype *HeN08-35*	F6:8 (103), F6:8 (130), F2:3 (159)	R gene	Glyma.03g04260, Glyma.03g04300, and Glyma.03g04340	Meng8206	Niu et al. (2017)
		RpsQ	–	Insert11, Insert144, SNP276	(2,997,143/2,997,106/ 3,031,924 a1)	Hypocotyl inoculation/isolates *PsJL1-2, PsGZ-2, PsFJ, PsJS7, Ps13-2, PsUSAR2, Ps13-1, PsAH, Ps13-4, PsAH4, PsHLJ5, Ps41-1, PsGS8, PsJS12, PsJMS2, PsAH1, PsSX1, PsAH5, Ps13-14, Ps13-5, PsJA08-1, Ps13-3, PsJA08-3, PsMC1, PsAH3, PsAH6, PsJL5, Ps13-12, PsJS6, PsJS10*	F2 (207), F2:3 (207)	R gene	Glyma.03g27200	Qichadou1	Li et al. (2017c)
		RpsWA[$]	–	Satt009 (BARCSOYSSR_03_0226) and T0003044871	0.9 -1.6 cM (3,919,203—4,486,048 a1)	Hypocotyl inoculation / isolates *PJ-H42, PJ-H67, R1, R4, R17, R25*	F2 (98), F7:8 (94)	R gene	–	Waseshiroge	Sugimoto et al. (2011)
		RpsWY	–	Satt631—Satt152, bin401	(4,466,230—4,502,773 a2)	Hypocotyl inoculation / isolates *Pm14, Pm28, PNJ1, P6497*	F2 (191), F7:8 (196)	R gene	–	Wayao	Cheng et al. (2017b)
		RpsHC18	–	BARCSOYSSR_03_0267 and BARCSOYSSR_03_0269	(4,152,795–4,919,481 a1)	Hypocotyl inoculation/ *PsRace1, PsRace3, PsRace4, PsRace5, PsUSAR2, Ps41-1, PsMC1, PsAH4, PsNKI, PsFJ2, PsFJ3, PsJS2*	F2:3 (177)	R gene	Glyma03G04560 and Glyma03G04590	Huachuan 18	Zhong et al. (2018a)
		RpsX (RpsQ?)	–	BARCSOYSSR_03_0161, BARCSOYSSR_03_0165, BARCSOYSSR_03_0167, and Insert144	(2,910,913–3,153,254, 2,997,106 a2)	Hypocotyl inoculation/ *PsRace1, PsRace3, PsRace4, PsRace5, PsUSAR2, Ps41-1, PsAH4, PsMC1, PsNKI, PsFJ2, PsFJ3, PsJS2, Ps6497, Ps7063*	F2:3 (137)	R gene	Glyma.03g027200	Xiu94-11	Zhong et al. (2019)
		RpsGZ	–	–	(4,003,401 and 4,370,772 a2)	Hypocotyl inoculation / *PNJ4, PNJ1*	F8:11 (228)	R gene	Glyma.03 g05300	Guizao1	Jiang et al. (2020)
		RpsDA [$]	–	–	(3,893,390–4,752,969 a2)	Hypocotyl inoculation / isolate *2457*	F5 derived (59)	66.4%	–	Daewon	Jang et al. (2020)
		RpsT1, RpsT2, RpsT3	–	BARCSOYSSR_03_0209 and BARCSOYSSR_03_0385	(3,606,810–7,614,961 a2)	Hypocotyl inoculation / isolate *Ps060626-4–1, Ps060726-2–1, Pm6, Ps74, Ps060619-1–2, Ps060619-1–1, Ps060619-1–3, Ps080626-1–7, Ps070702-4–1, Ps060710-5–1, Ps060629-*	F2:3 (105), F4:5 (165)	R gene	–	Tosan231	Matsuoka et al. (2021)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
						4–1, Ps060626-71–1, Ps060828-1–1, Ps060626-6–1, Ps080623-1–4, Ps070702-1–1, S5 Ps070726-3–4, Ps060710-3–1, Ps070621-6–1					
		Rps14	–	InDel4033 and InDel4263	(4,033,638—4,263,083 a2)	Hypocotyl inoculation / *Race 1, 2, 3, 4, 6, 7, 8, 9, 17, 25, ISA19A-1, ISA71D-1, MIN12001.01.05, MIN12004.01.01, MIN12004.03.01*, and *MIN12005.07.02*	F2 (167), F2:3 (110)	R gene	–	PI 340029	Chen et al. (2021a, b)
	MLG M (Chr. 7)	*Rps11*	–	BARCSOYSSR_07_0295	(5,523,128 a2)	Hypocotyl inoculation/Races *1, 3, 7, 10, 13, 17, 25, 28*, and isolates *ISA 19A-1, ISA71D-1, ISA 33O-8*, and 127 additional isolates (see Wang et al. 2021)	F2 (58), F2:3 (209), F4 (17,050)	R gene	a 27.7 kb NBS-LRR gene	PI 594527	Ping et al. (2016), Wang et al. (2021)
	MLG O (Chr. 10)	*RpsSu*	–	Satt358—Sat_242 (BARCSOYSSR_10_1104)	3.5–7.4 cM (1,018,481—39,392,879 a2)	Hypocotyl inoculation / isolate *Pm14*	RIL (176)	R gene	–	Su88-M21	Wu et al. (2011b)
	MLG F (Chr. 13)	*Rps3a*	–	Satt510 (BARCSOYSSR_13_1219) and Satt335 (BARCSOYSSR_13_1271)	(31,802,616—32,721,481 a2)	Hypocotyl inoculation/races *1, 2, 3, 4, 5, 8, 9, 11, 13, 14, 16, 18, 23, 25, 27, 28, 29, 31, 32, 33, 34, 35, 40, 41, 43, 44, 45, 47, 48, 49, 50, 51, 52, 54* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsJL4-1, PsJL3-2, PsFJ, PsSX1, PsXJ, PsBr1, PsAM1, Ps41-1, PsJL5, Ps52, Ps53, PsMC1, PsJN4, 2010DPH39-02–6, OH2000Wood25, OHSS03HenryCo3, OHSS03WayneBerry11, OHSS03WayneBerry5, OH25 2011, PS03-036, PS03-113, PS04-139, PS04-257, PS04-323, PTO4C2Res11*	F2:3 (89)	R gene	–	PI 171442, L83-570	Mueller et al. (1978), Dorrance et al. (2004), Gordon et al. (2007), Sugimoto et al. (2012), Gunadi (2012), Zhang et al. (2013a)
		Rps3b	–	–	–	Hypocotyl inoculation / races *1, 2, 3, 4, 5, 7, 9* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsJL4-1, PsJL3-2, PsFJ, PsSX1, PsAH3, PsAH1, PsBr1, PsAM1, PsTA3, Ps52, PsAH5, 2010DPH39-02–1, OHSS03HenryCo3, OHSS03HenryCo7, PS03-06, PS03-113*	F2:3 (160–274)	R gene	–	PI 172901, PRX146-36, L91-8347, L89-1541	Ploper et al. (1985), Dorrance et al. (2004), Gunadi (2012), Zhang et al. (2013a)
		Rps3c	–	–	–	Hypocotyl inoculation / races *1, 2, 3, 4*, and isolates *PsJL1-1, PsJMS3, PsAH4, PsJL4-1, PsJL3-2, PsFJ, PsSX1, PsAH3, PsAH1, PsBr1, PsAM1, 2010DP39-02–1, 2010DPH39-02–1, 2010W137-135–1, ButMu4S2, M1-1–2, OH2000Wood25, OHSS03WayneBerry5,*	F2 (1650, 1708, 1182, 1517, 1692, 1208, 1687, 1452)	R gene	–	PI 340046, PRX145-48, L92-7857	Athow et al. (1986), Dorrance et al. (2004), Sugimoto et al. (2012), Gunadi (2012)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
						OH25 2011, PS03-036, PS03-113, PS04-132, PS04-139, PS04-257, PS04-323, PTO4C2Res11					
		Rps8	–	Satt425 (BARCSOYSSR_13_0784) and Satt114 (BARCSOYSSR_13_1055), Sat_154 and Sat_120	(24,361,239—28,912,878 a2)	Hypocotyl inoculation/race *1, 25*, isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsJL4-1, PsJL3-2, PsFJ, PsSX1, PsAH3, PsAM1, PsJS7, Ps53, 2010DPH39-02–1, OH2000Sandusky74, OHSS03HenryCo3, OHSS03HenryCo7, OHSS03WayneBerry11, OHSS03WayneBerry5, OHSS04WayneTBH62xx, OH25 2011, PS03-036, PS03-113, PS04-132, PS04-139, PS04-257, PS04-323, PTO4C2Res11*	F2:3 (208, 202)/ F2:3 (138)	R gene	–	PI 399073	Burnham et al. (2003a), Sandhu et al. (2005), Gordon et al. (2006), Gunadi (2012), Zhang et al. (2013a), Jang et al. (2020)
		RpsSN10	–	Satt423 (BARCSOYSSR_13_0264) and Satt149 (BARCSOYSSR_13_0245)	(16,600,532—16,855,132 a2)	Hypocotyl inoculation/race *1*	F2 (124)	R gene	–	Suinong 10	Yu et al. (2010)
		RpsCD	–	–	–	Detached trifoliate leaf disease screening assay/effector *Avh180*, and isolates *Iso1005 and Iso2004*	F2:3 and RIL (142)	R gene	–	PI 408132	Davis (2017)
	MLG J (Chr. 16)	*Rps2*	–	Satt547 (BARCSOYSSR_16_1165)	(34,035,215 a2)	Hypocotyl inoculation/isolates *PsJL1-1, PsHJL1, PsJL4-1, PsJL3-2, PsFJ, PsSX1, PsAH3, PsBr1, PsTA3, PsJS9, PsJS7, PsJS8, Ps52, pmg(1)-3, 94–14-432(2), pmg(25)-1, 96-13S-106A.1, ISA 19A-1, ISA 19B-2, ISA 71D-1*	F2:3 (115)	R gene	–	CNS', L82-1449, L76-1988	Kilen et al. (1974), Polzin et al. (1994), Demirbas et al. (2001), Dorrance et al. (2004), Zhang et al. (2013a), Lin et al. (2013), Jang et al. (2020)
		RpsUN2	–	CAPS6 and SNP2	(37,239,148—37,275,206 a2)	Hypocotyl inoculation / races *1,3,4,5,10,13,24,25, 28*, and isolates *ISA 19A-1, ISA 19B-2, ISA 71D-1, ISA 33O-8*	F2:3 (826)	R gene	Glyma.16g215200 and Glyma.16g214900	PI 567139B	Lin et al. (2013), Li et al. (2016a, b, c)
	MLG D2 (Chr. 17)	*Rps10*	–	Sattwd15-28, Sattwd15-32	(30,964,476/30,983,669 a1)	Hypocotyl inoculation/isolate *PsMC1*	F2:3 (102)	R gene	Glyma17g28950 and Glyma17g28970	Wandou15	Zhang et al. (2013b)
	MLG G (Chr. 18)	*Rps4*	–	Satt191 (BARCSOYSSR_18_1750) Sat_064 (BARCSOYSSR_18_1858)	(56,333,740 a2)	Hypocotyl inoculation / isolates *PsJL1-1, PsJMS3, PsHJL1, PsJL4-1, PsJL3-2, PsSX1, PsXJ, PsAM1, Ps41-1, PsJL5, Ps53, ButMu4S2, OH2000Wood25, OHSS03WayneBerry11, OHSS03WayneBerry5, OH25 2011, PS02-014, PS03-036, PS03-113, PS04-132, PS04-139, PS04-257, PS04-323, PTO4C2Res11*	F2:3 (100)	R gene	–	L85-2352	Athow et al. (1980), Demirbas et al. (2001), Dorrance et al. (2004), Sandhu et al. (2004), Gunadi (2012), Zhang et al. (2013a)
		Rps5	–	Satt472 (BARCSOYSSR_18_1708)	(53,866,606 a2)	Hypocotyl inoculation/isolates *PsJL1-1, PsJMS3, PsHJL1, PsJL4-1, PsJL3-2, PsSX1, PsXJ, PsAM1, PsJL5, OH2000Wood25, OHSS03WayneBerry5,*	F2:3 (122)	R gene	–	L62-904, L85-3059	Buzzell and Anderson (1981), Demirbas et al. (2001), Dorrance et al. (2004), Gunadi (2012), Zhang et al. (2013a), Sahoo et al. (2017)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
						OHSS04WayneTBH62xx, PS03-036, PS03-113, PS04-139, PS04-323, PTO4C2Res11					
		Rps6	–	Satt191 (BARCSOYSSR_18_1750) and Sat_372	(54,450,956 a2)	Hypocotyl inoculation/races *1, 2, 3, 4, 10, 12, 14, 15, 16, 18, 19, 20, 21, 25, 28, 33, 34, 35, 40, 41, 42, 43, 44, 46, 47, 48, 53, 54* and isolates *PsJL1-1, PsJMS3, PsHJL1, PsAH4, PsJL4-1, PsJL3-2, PsSX1, PsXJ, Ps41-1, PsJL5, Ps53, 2010DP39-02–6, 2010W137-135–1, ButMu4S2, M1-1–3, M3-3–1, M6-3–1, OH2000Wood25, OHSS03WayneBerry11, OHSS03WayneBerry5, OH25 2011, PS02-014, PS03-036, PS03-113, PS04-139, PS04-257, PS04-323, PTO4C2Res11*	F2:3 (89)	R gene	–	Haro 62xx, L89-1581	Athow and Laviolette (1982), Demirbas et al. (2001), Dorrance et al. (2004), Gordon et al. (2007), Sugimoto et al. (2012), Gunadi 2012; Zhang et al. (2013a)
		RpsJS	–	BARCSOYSSR_18_1861, SSRG60684K, and SSRG60718K	(60,613,262—60,752,225 a1)	Hypocotyl inoculation / isolates *JS08-12, P6497, P7063, S16, S2, PNJ1, Pmg, Pm28, Pm2, HeN08-35, HLJ08-17, H15, AH, P7071, Pm31*	F2:3 (231)	R gene	–	Nannong10-1	Sun et al. (2014a)
		Rps12	–	BARCSOYSSR_18_1840 and Sat_064, 4 cM from *Rps13*	(55,962,037—56,333,703 a2)	Hypocotyl inoculation/isolates *R17, Val12-11, P7074, 1005–2.9, III5.2b, IV5.2, IV10, IV12.2a, IV13.4a, IV23.3, VI5.2b, VI12.1a, VII5, VII7, VI23.3b, S5-5, PR1, PR6, 1005–2.9 + VI23.3b + R17*	F7 derived (290)	R gene	–	PI 399036	Sahoo et al. (2017), Sahoo et al. (2021)
		Rps13	–	Sat_064, BARCSOYSSR_18_1859 and BARCSOYSSR_18_1860	(56,333,703—56,341,167 a2)	Hypocotyl inoculation / isolate *V13, R17 + Val12-11*	F8 derived (120)	R gene	–	PI 399036	Sahoo et al. (2021)
	MLG L (Chr. 19)	*RpsYB30*	–	Satt497 (BARCSOYSSR_19_0760) Satt313 (BARCSOYSSR_19_0788)	(33,865,280—34,753,167 a2)	Hypocotyl inoculation/isolates *USAR1, PsBX1, PsXJ1, PsMC1, PsZLT1, PsFJ1, PsXJ2, Ps41-1*	F4 (57)	R gene	–	Youbian30	Zhu et al. (2007)
	MLG D1a (Chr. 1)	*qRps01-01*[$]	–	BARC_2.0_Gm01_50164447 and BARC_2.0_Gm01_50295635	(50,164,447—50,295,635 a2)	Tray test / isolates *1.S.1.1*	F9:11 (316)	4.5%	–	Conrad	Stasko et al. (2016)
			–	BARC_2.0_Gm01_50206347 and BARC_2.0_Gm01_50287274	(50,206,347—50,287,274 a2)	Tray test / isolates *OH25*	F9:11 (316)	8.2%	–	Conrad	Stasko et al. (2016)
			–	BARC_2.0_Gm01_50572171 and BARC_2.0_Gm01_50797061	(50,572,171 -50,797,061 a2)	Tray test / isolates *PT2004C2.S1*	F9:11 (316)	7.6%	–	Conrad	Stasko et al. (2016)
	MLG D1b (Chr. 2)	*qRps02-01*[$]	–	Satt579 (BARCSOYSSR_02_0855) and Satt600 (BARCSOYSSR_02_1048)	(19,688,108—29,355,267 a2)	Slant board test / isolate OH25	F4:6 (66), F4:6 (79)	10.6–20.7%	–	–	Burnham et al. (2003b)
			QPRR-3	Satt579 (BARCSOYSSR_02_0855)	(19,688,108—34,875,449 a2)		F2:6 (140)	5.5–28.0%	–	–	Li et al. (2010d)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
				and Sat_089 (BARCSOYSSR_02_1152)		Field inoculation and greenhouse test (China and Canada)					
			QPRR-2	Satt005 (BARCSOYSSR_02_0998) and Satt600 (BARCSOYSSR_02_1048)	(27,699,285—29,355,267 a2)	Field inoculation and greenhouse test (China and Canada)	F2:6 (140)	11.3–22.0%	–	–	Li et al. (2010d)
	MLG N (Chr. 3)	*qRps03-01*[$]	*qHO3-1*	ss715585371	(3,607,392 a2)	Hypocotyl inoculation / isolate *OH.12108.6.3 (OH.121)*	Germplasm (429)	–	–	–	Van et al. (2020)
			C2-03–3	ss715585633	(3,691,222–3,895,958 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	4.0% IRRS[c]	–	–	Rolling et al. (2020)
			QTL 3–1	ss715585712 and ss715585728	(3,852,888 and 3,865,730 a2)	Tray test / isolates *OH121* and *C2S1*	PI lines (800)	3.2%Root rot score	–	–	Schneider et al. (2016)
		qRps03-02[$]	*Qprr3-2*	Chr03-3,904,775 and Chr03-4,404,630	(3,904,775 and 4,404,630 a2)	Hypocotyl inoculation / race *1*	F5:15 (109)	56.1%	Glyma.03G033800 and Glyma.03G033700	DongongL-28	Zhao et al. (2020)
			Rprr-3–1	–	(3,993,736 a2)	Hypocotyl inoculation / race *1*	Germplasm and cultivars (225)	33.6%	–	G	Zhao et al. (2020)
			QTL 3–2	ss715586320 and ss715586321	(4,276,534 and 4,277,380 a2)	Tray test / isolates *OH121* and *C2S1*	PI lines (800)	3.1% Root rot score	–	–	Schneider et al. (2016)
			qHO3-2	ss715586333	(4,289,618 a2)	Hypocotyl inoculation/isolate *OH.12108.6.3 (OH.121)*	Germplasm (429)	–	–	–	Van et al. (2020)
			QTL 3–3	ss715586376	(4,315,512 a2)	Tray test / isolates *OH121* and *C2S1*	PI lines (800)	3.9% Root rot score	–	–	Schneider et al. (2016)
	MLG C1 (Chr. 4)	*qRps04-01*[$]		BARC_2.0_Gm04_45977762 and BARC_2.0_Gm04_46204517	(45,977,762—46,204,517 a2)	Tray test / isolates *1.S.1.1*	F9:11 (316)	3.2%	–	Sloan	Stasko et al. (2016)
				BARC_2.0_Gm04_46096228 and BARC_2.0_Gm04_46536196	(46,096,228–46,536,196 a2)	Tray test/isolates *PT2004C2.S1*	F9:11 (316)	3.2%	–	Sloan	Stasko et al. (2016)
	MLG C2 (Chr. 6)	*qRps06-01*[$]	*qPR-6–3*	Satt520 (BARCSOYSSR_06_0386) and Satt557 (BARCSOYSSR_06_1041)	(7,023,397 a1/ 20,218,893 a2)	Modified slant board assay/ race *2*	F7:11 (176)	4.3%	–	Su88-M21	Wu et al. (2011c)
				Gm06_11776489_C_A	(11,725,151- 11,728,261 a2)	Tray test/isolate *Win371*	SoyNAM RIL (91)	17.00%	–	IA3023	Scott et al. (2019)
			QPRR-7	Satt277 (BARCSOYSSR_06_0920) and Satt365	(17,218,677 a2)	Field inoculation and greenhouse test (China and Canada)	F2:6 (140)	9.3–21.8%	–	–	Li et al. (2010d)
		qRps06-02[$]	–	BARC-062515–17,881 and BARC-040475–07,751	(21,986,774—46,596,066 a2)	Rice-based method/isolates *PT2004C2.S1, 1005–2.9, R7-2a*	F5:7 (232), F5:7 (277)	5.5% %DRL[d], 4.7% %DSA[e]	–	AR2	Abeysekara et al. (2016)
			QPRR-6	Satt489 (BARCSOYSSR_06_1129) and Satt100 (BARCSOYSSR_06_1202)	(23,848,501—31,490,622 a2)	Field inoculation and greenhouse test (China and Canada)	F2:6 (140)	5.4–21.8%	–	–	Li et al. (2010d)
			qHO6-1	ss715594028	(26,103,041 a2)	Hypocotyl inoculation / isolate *OH.12108.6.3 (OH.121)*	Germplasm (429)	–	–	–	Van et al. (2020)
	MLG O (Chr. 10)	*qRps10-01*[$]	*qPR-10–2*	Satt420 (BARCSOYSSR_10_0507) and Sat_274 (BARCSOYSSR_10_1353)	(10,091,607—43,788,256 a2)	Modified slant board assay/ race *2*	F7:11 (176)	7.7–8.3%	–	Su88-M21	Wu et al. (2011c)
			OH-10–2	ss715606258	(33,174,697—33,499,135 a2)	Layer test/isolate *OH.121*	Germplasm (478)	4.5% ISW[f]	–	–	Rolling et al. (2020)
			qHC10-1	ss715607061	(41,316,525 a2)	Hypocotyl inoculation / isolate *PT2004 C2.S1*	Germplasm (460)	–	–	–	Van et al. (2020)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
	MLG H (Chr. 12)	*qRps12-01*[$]	–	BARC-019775–04,370 and BARC-025943–05,179	(7,533,328—9,804,252 a2)	Rice-based method/isolates *PT2004C2.S1, 1005–2.9, R7-2a*	F5:7 (232), F5:7 (277)	5.7% CDSW[g]	–	AR2	Abeysekara et al. (2016)
			qHM12-1	ss715613620	(8,854,648 a2)	Hypocotyl inoculation/isolates *PT2004 C2.S1, R7-2a, 1005–2.9*	*G. soja* (520)	–	–	–	Van et al. (2020)
	MLG F (Chr. 13)	*qRps13-01*[$]	*QGP1*	Satt509 (BARCSOYSSR_11_0342) and Satt030 (BARCSOYSSR_13_0445)	(6,216,988—13,134,055 a2)	Field inoculation and greenhouse test (China and Canada)	F2:7 (112)	6.7–13.2%	–	–	Han et al. (2008)
			QPRR-1	Satt325 (BARCSOYSSR_13_0639) and Satt343 (BARCSOYSSR_13_0518)	(8,587,948—10,392,903 a2)	Field inoculation and greenhouse test (China and Canada)	F2:6 (140)	9.2–10.2%	–	–	Li et al. (2010d)
			QGP2	Satt343 (BARCSOYSSR_13_0518) and $OPG16_{600}$	(10,392,903 a2)	Field inoculation and greenhouse test (China and Canada)	F2:7 (112)	2.4–8.2%	–	–	Han et al. (2008)
		qRps13-02[$]	*OH-13–1*	ss715614516	(27,874,365—27,896,769 a2)	Layer test/isolate *OH.121*	Germplasm (478)	6.75% IRRS	–	–	Rolling et al. (2020)
			C2-13–5	ss715614543	(28,001,686—28,051,574 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	2.07% IPH[h]	–	–	Rolling et al. (2020)
			QTL-13	Chr13:28,842,184 and Chr13:30,776,191	(28,842,184–30,776,191 a2)	Hydroponic assay/mixed inoculum (pathotypes 1a, 1b, 1c, 1d, 1 k, 3a, 6, and 7)	F5:6 (147)	17.6% CDW[i]	Glyma.13G190400	PI 449459	de Ronne et al. (2020)
			QTL-13	BARCSOYSSR_13_1103	51-52 cM (29,647,017 a2)	Tray test and layer test/isolates *C.2.S.1, OH25, OH7, 1.S.1.1, OH30*	F7:8 (305)	8.7%-16.1%	–	PI 398841	Lee et al. (2013a), Lee et al. (2014)
			qHC13-2	ss715614840	(29,698,315 a2)	Hypocotyl inoculation/isolate *PT2004 C2.S1*	Germplasm (460)	–	–	–	Van et al. (2020)
			OH-13–2	ss715614895	(29,971,253—30,065,880 a2)	Layer test/isolate *OH.121*	Germplasm (478)	2.6% IRW[j]	–	–	Rolling et al. (2020)
			OH-13–3	ss715614914	(30,086,805—30,144,416 a2)	Layer test/isolate *OH.121*	Germplasm (478)	8.0% IRW	–	–	Rolling et al. (2020)
				Gm13_29043806_T_C	(30,125,163- 30,154,255 a2)	Tray test/isolate *Win371*	SoyNAM RIL (122)	42.2%	–	HS6-3976	Scott et al. (2019)
			OH-13–4	ss715614952	(30,291,675—30,301,385 a2)	Layer test/isolate *OH.121*	Germplasm (478)	2.6% IRRS, 2.3% IRW	–	–	Rolling et al. (2020)
			C2-13–6	ss715614993	(30,502,735—30,618,405 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	3.5% IRW	–	–	Rolling et al. (2020)
			qHM13-1	ss715615005	(30,628,076 a2)	Hypocotyl inoculation/isolates *PT2004 C2.S1, R7-2a, 1005–2.9*	Germplasm (448)	–	–	–	Van et al. (2020)
			C2-13–7	ss715615007	(30,646,059—30,654,291 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	11.2% IRW	–	–	Rolling et al. (2020)
			OH-13–5	ss715615020	(30,667,266—30,700,217 a2)	Layer test/isolate *OH.121*	Germplasm (478)	3.1% ΔRW[k]	–	–	Rolling et al. (2020)
				Sct_033 (BARCSOYSSR_13_1230)	20.6—31.7 cM (30,739,608 a2)	Tray test/isolate *1.S.1.1*	F4:6 (375)	5.4%	–	–	Wang et al. (2010)
				Sct_033	34 cM (30,739,608 a1)	Hypocotyl inoculation/isolate *OH17*, race 2	F7:8 and F7:9 (188)	20.1–35.8%	–	PI 408105A	Nguyen et al. (2012)
			QTL 13–1	ss715615031	(30,766,058 a2)	Tray test/isolates *OH121* and *C2S1*	PI lines (800)	2.5% Root weight	–	–	Schneider et al. (2016)
			–	SNP 35,123,596	(35,123,596 a1)	Slant board assay/isolate *P7076*	Germplasm (279)	–	Glyma13g32980, Glyma13g33900, Glyma13g33512	–	Li et al. (2016a, b, c)
				Gm13_39560450_G_A		Tray test/isolate *Win371*		7.2%	–	HS6-3976	Scott et al. (2019)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
					(40,233,656- 42,919,730 a2)		SoyNAM RIL (122)				
			–	php2385	44-50 cM	Tray test/isolate C2S1	F10 and F11 (298)	7%	–	V71-370	Tucker et al. (2010)
			QTL-13	–	52-54 cM	Tray test and layer test/isolates *C.2.S.1, OH25, OH7, 1.S.1.1, OH30*	F7:8 (305)	13.2%-15.1%	–	PI 398841	Lee et al. (2014)
			QTL-13	BARC-051883–11,286 to BARC-042715–08,379	49-57 cM (24,848,378—44,053,323 a1)	Tray test/isolate *OH25*	F7:8 (157)	8.6%	–	PI 407861A	Lee et al. (2013b)
	MLG E (Chr. 15)	*qRps15-01*[$]	*QTL-15*	BARC-055329–13,210 to BARCSOYSSR_15_0160	16-19 cM (2,740,854–3,563,138 a1)	Tray test/isolate *OH25*	F7:8 (157)	7.2%	–	OX20-8	Lee et al. (2013b)
			C2-15–1	ss715621545	(2,952,387—3,182,673 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	1.0% IRRS	–	–	Rolling et al. (2020)
		qRps15-02[$]	*qPR-15–1*	Satt651 (BARCSOYSSR_15_0306) and Satt598 (BARCSOYSSR_15_0645)	(6,823,519—13,653,981 a2)	Modified slant board assay/ race *2*	F7:11 (176)	14.0–15.9%	–	Su88-M21	Wu et al. (2011c)
			–	Gm15_11496274	(11,496,274 a1)	Layer test/isolate ? (vir 1d, 2, 3b, 3c, 4, 5, 6, 7)	Cultivars (169)	11.7%	Glyma15g15030	–	Ludke et al. (2019)
	MLG J (Chr. 16)	*qRps16-01*[$]	*16–2*	BARC_2.0_Gm16_3124736 and BARC_2.0_Gm16_3362395	(3,124,736–3,362,395 a2)	Tray test/isolates *1.S.1.1,*	F9:11 (316)	5.4%	–	Sloan	Stasko et al. (2016)
			16–2	BARC_2.0_Gm16_3124736 and BARC_2.0_Gm16_3362395	(3,124,736–3,362,395 a2)	Tray test/isolates *PT2004C2.S1*	F9:11 (316)	3.5%	–	Sloan	Stasko et al. (2016)
	MLG D2 (Chr. 17)	*qRps17-01*[$]	*C2-17–1*	ss715626781	(33,515,060—33,574,931 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	4.5% ΔRW	–	–	Rolling et al. (2020)
			qHC17-1	ss715626781	(33,549,403 a2)	Hypocotyl inoculation/isolate *PT2004 C2.S1*	Germplasm (460)	–	–	–	Van et al. (2020)
		qRps17-02[$]	*C2-17–2*	ss715627019	(36,411,792–36,398,362 a2)	Layer test/isolate *C2.S1*	Germplasm (495)	2.4% IRW	–	–	Rolling et al. (2020)
			–	Satt301-190	(36,718,722 a2)	Slant board assay/race *2*	China mini core collection (175)	5.2%	–	ZDD10252	Sun et al. (2014a, b)
	MLG G (Chr. 18)	*qRps18-01*[$]	*QDRL-18*	BARC-020839–03,962, BARC025777-05,064, and BARC-047665–10,370	13-16 cM (981,868/ 2,843,515 a2)	Tray test, layer test, and field test (OH, US)/isolates *OH7, OH7-8, OH25, OH12108, C2.S1, OH2010.739, OH2010.001*	F7:8 (367), F7:8 (338)	20.4%, 24.7%	–	PI 427105B and PI 427106	Lee et al. (2014), Karhoff et al. (2019)
			OH-18–1	ss715629906	(2,128,180–2,155,661 a2)	Layer test/isolate *OH.121*	Germplasm (478)	7.2% ISW	–	–	Rolling et al. (2020)
		qRps18-02[$]	–	BARC_2.0_Gm18_56710850 and BARC_2.0_Gm18_56766936	(56,710,850–56,766,936 a2)	Tray test/isolates *1.S.1.1*	F9:11 (316)	5.3%	–	Conrad	Stasko et al. (2016)
			–	BARC_2.0_Gm18_56710850 and BARC_2.0_Gm18_56876857	(56,710,850–56,876,857 a2)	Tray test/isolates *OH25*	F9:11 (316)	13.6%	–	Conrad	Stasko et al. (2016)
	MLG L (Chr. 19)	*qRps19-01*[$]	*19–1*	BARC-047496–12,943 and BARC_2.0_Gm19_46116996	(42,821,735 a1/ 46,116,996 a2)	Tray test/isolates *PT2004C2.S1*	F9:11 (316)	4.6%	–	Conrad	Stasko et al. (2016)
			19–1	BARCSOYSSR_19_1243 and BARCSOYSSR_19_1286	(43,533,689–44,370,710 a2)	Tray test/isolates *1.S.1.1,*	F9:11 (316)	3.1%	–	Conrad	Stasko et al. (2016)
			19–1	BARCSOYSSR_19_1286 and BARC_2.0_Gm19_46116996	(44,370,710–46,116,996 a2)	Tray test/isolates *OH25*	F9:11 (316)	9.1%	–	Conrad	Stasko et al. (2016)
		qRps19-02[$]	*19–2*	BARCSOYSSR_19_1452 and Glyma.19G226100	(47,528,116–47,787,869 a2)	Tray test/isolates *PT2004C2.S1*	F9:11 (316)	4.1%	–	Conrad	Stasko et al. (2016)

Table 5 (continued)

Causal agent	MLG (Chr.)	Locus/ allele name	Other name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Candidate genes	Donor source/ allele	References
			19–2	BARCSOYSSR_19_1452 and Glyma.19G226100	(47,528,116–47,787,869 a2)	Tray test/isolates *OH25*	F9:11 (316)	7.8%	–	Conrad	Stasko et al. (2016)
		qRps19-03[$]	*19–3*	BARC_2.0_Gm19_50305134 and BARC-014385–01,342	(50,305,134 a2/ 50,222,676 a1)	Tray test / isolates *OH25*	F9:11 (316)	6.6%	–	Conrad	Stasko et al. (2016)
			QTL 19–3	ss715636056, ss715636059, ss715636064, ss715636073, ss715636076, ss715636077, ss715636083, ss715636084	(50,544,363–50,681,263 a2)	Tray test/isolates *OH121* and *C2S1*	PI lines (800)	2.5% Root rot score	–	–	Schneider et al. (2016)
			QTL-19	Chr19:50,040,258 and Chr19:50,556,102	(50,040,258–50,556,102 a2)	Hydroponic assay/mixed inoculum (pathotypes 1a, 1b, 1c, 1d, 1 k, 3a, 6, and 7)	F5:6 (147)	13.1% CDW	Glyma.19G262700	PI 449459	de Ronne et al. (2020)
P. sansomeana	MLG A1 (Chr.5)	–	*qPsan5.1*	Gm05_32565157_T_C and Gm05_32327497_T_C	54.71 cM (32,832,462–32,594,828 a2)	Modified layer test / *MPS17-22, V-NESO2 5–45, V-KSSO2 3–6, MPS17-24*(in combination with *qPsan16.1*), *C-NESO2 5–12*(in combination with *qPsan16.1*)	F4:5 (218)	6%	–	E13390	Lin et al. (2021)
	MLG J (Chr.16)	–	*qPsan16.1*	Gm16_35700223_G_T and Gm16_35933600_A_G/ Gm16_35816475_T_C	39.01 cM (36,203,537–36,436,443 a2)	Modified layer test / *MPS17-22, V-NESO2 5–45, V-KSSO2 3–6, MPS17-24*(in combination with *qPsan5.1*), *C-NESO2 5–12* (in combination with *qPsan5.1*), *C-IASO2 6–15, and MICO3-28*	F4:5 (218)	5.5%	–	E13901	Lin et al. (2021)

[$]Locus name given in this study

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[b]Phenotypic variations explained by the molecular markers

[c]IRRS: inoculated root rot score

[d]%DRL: percentage of diseased root length

[e]%DSA: percentage of diseased root surface area

[f]ISW: Inoculated shoot weight

[g]CDSW: Corrected dry shoot weight

[h]IPH: inoculated plant height

[i]CDW: corrected dry weight

[j]IRW: inoculated shoot weight

[k]ΔRW: change in root weight

and most breeding efforts have focused on minimizing impacts by *Phytophthora* and *Pythium* (Dorrance et al. 2009; Rupe et al. 2011). Recent efforts have expanded the knowledge of oomycete species causing disease on soybean, but the range of this potential species varies with the locations (Rojas et al. 2017), and among those, some species are considered emerging such as *Phytophthora sansomeana* E.M. Hansen & Reeser (McCoy et al. 2018).

Phytophthora root and stem rot

Phytophthora root and stem rot (PRSR) of soybean is one of the most prevalent and widely distributed soybean diseases, causing reduced yield and worldwide losses of 2.3 million Mt per year (Erwin and Ribeiro 1996; Koenning and Wrather 2010; Allen et al. 2017). *Phytophthora sojae* Kaufmann & Gerdemann, the main causal agent of this disease, was initially reported in the mid-1950s in the Midwest region of the USA (Kaufmann and Gerdemann 1958) and has since become a major concern for soybean production causing annual losses of approximately 1.2 million Mt in the USA (Wrather et al. 2010). *P. sojae* is an oomycete pathogen that survives in the soil as oospores. Under optimal conditions, oospores germinate and infect seeds and roots causing seed rot and damping-off of seedlings. *P. sojae* may also cause root and stem rot that results in wilting and plant death. While the typical brown to purple water-soaked lesions on the stem appear mid-late season on infected plants, early-season infection may also result in an uneven plant stand and possibly need of replanting (Bienapfl et al. 2011; Dorrance et al. 2016).

Screening of *P. sojae* for race identification and soybean line resistance has been based on the use of hypocotyl inoculations (Dorrance et al. 2008; Stewart and Robertson 2012; Lin et al. 2014). For *P. sojae*, *Rps 1a*, *1b*, *1c*, *1 k*, *3a*, *3b*, *3c*, *4*, *6*, *7*, or *8* are part of the set of differentials, and recent surveys have tested isolates identifying emerging races. Of those, *Rps1a-1 k*, *Rps3a*, *Rps6* and *Rps8* are deployed through resistant cultivars. However, there are reports of resistance breakdown of *Rps1* in soybean-producing states in the Midwest of USA (Dorrance et al. 2016; Matthiesen et al. 2021; McCoy et al. 2021). In lower frequency, *Rps3a* and *Rps6* were also defeated by some isolates in the Midwest. Since not all identified resistance genes have been deployed, it is important to monitor races for future breeding efforts as some of the remaining resistance genes have also been overcome by a few field isolates (Dorrance et al. 2016; McCoy et al. 2021).

Fortunately, novel *Rps* genes or alleles have been identified conferring broad-spectrum resistance to *P. sojae* races. To date, more than 40 *Rps* genes or alleles have been reported worldwide (Table 5). Intriguingly, the *Rps* genes/alleles were not evenly distributed but were clustered on some specific chromosomes. For instance, more than half of the *Rps* genes/alleles (*Rps1a-1 k*, *Rps7*, *Rps9*, *RpsUN1*, *RpsYD25*, *RpsYD29*, *RpsHN*, *RpsQ*, *RpsWA*, *RpsWY*, *RpsHC18*, *RpsX*, *RpsGZ*, *RpsDA*, *RpsT1*, *RpsT2*, *RpsT3*, and *Rps14*) were mapped in a nucleotide-binding site-leucine-rich repeat (NBS-LRR) gene enriched region on Chr. 3; Six *Rps* genes/alleles were located on chromosomes 13 (*Rps3a*, *Rps3b*, *Rps3c*, *Rps8*, *RpsSN10* and *RpsCD*) and 18 (*Rps4*, *Rps5*, *Rps6*, *RpsJS*, *Rps12*, and *Rps13*), respectively. The rest of *Rps* genes were located at chromosomes 2 (*RpsZS18*), 7 (*Rps11*), 10 (*RpsSu*), 16 (*Rps2* and *RpsUN2*), 17 (*Rps10*), and 19 (*RpsYB30*) (Table 5).

Fine mapping studies toward map-based cloning of *Rps* genes have also been reported. The first cloned *Rps* gene is the *Rps1k* from Williams82, from which three highly similar coiled coil (CC)-NBS-LRR genes were identified and verified through transgenic progenies (Gao et al. 2005). Unfortunately, none of these genes can be identified in any versions/sources of the Williams82 genome assemblies including unassembled contigs (Wang et al 2021). In another study, *RpsUN1* and *RpsUN2* were further narrowed to a 151 kb and 36 kb genomic regions using 826 F2:3 families. Expression analyses via reverse-transcription (RT)-PCR and RNA-seq suggested that *Glyma.03g034600* and *Glyma.16g215200*/*Glyma.16g214900* were high-confidence candidate genes for *RpsUN1* and *RpsUN2*, respectively (Li et al. 2016a, b, c). Most recently, a map-based cloning study revealed that the *Rps11* gene encoded a 27.7 kb NBS-LRR gene, and is derived from rounds of unequal recombination events, which resulted in promoter fusion and LRR expansion that contributed to the broad-spectrum resistance (Wang et al. 2021). More importantly, *Rps11* alone can defeat 127 isolates (80% of all tested isolates) widely distributed across the USA (Ping et al. 2016; Wang et al. 2021). It is expected that commercial soybean varieties carrying the *Rps11* gene will soon be available in the market.

In *Phytophthora* studies, pathogen inoculation methods to assess populations could also influence the outcome; For instance, hypocotyl inoculation has been a standard method to detect vertical resistance and is a premier step to exclude the influence of potential R genes before detecting horizontal resistance (Dorrance et al. 2018). On the other hand, the most commonly used methods to detect horizontal resistance to *P. sojae* are layer test and tray test which were based on colonized substrate to deliver the pathogen to the plant tissue (Dorrance et al. 2008; Wang et al. 2012). More recently, a hydroponic assay was developed that can detect both vertical and horizontal resistance through infection of soybean root system with zoospores (Lebreton et al. 2018). Different phenotypic traits can be collected including lesion size, root mass, shoot biomass, root scores, and

Table 6 Soybean loci associated with resistance to Pythium damping-off and root rot (caused by *Pythium* spp.)

Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
Pythium aphanidermatum	MLG (Chr. 4)	–	ss715589319	49.00–51.00 cM (7,868,252 a2)	Seed plate assay and an infested vermiculite assay/isolate *64*	F2:6 (84)	8.3–13.8%	Archer	Urrea et al. (2017)
	MLG (Chr. 7)	–	ss715598762	121.20 cM (8,151,504 a2)	Seed plate assay and an infested vermiculite assay/isolate *64*	F2:6 (84)	4.5–13.9%	Archer	Urrea et al. (2017)
	MLG F (Chr. 13)	*Rpa1*	Satt510 and Satt114	(28,912,864–31,802,559 a2)	Hypocotyl inoculation/isolate *64*	F2:4 (86)	R gene	Archer	Rosso et al. (2008)
Pythium irregulare	MLG D1a (Chr. 1)	-	Satt515	(37,027,518 a2)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	14.0% Weight, 17.7% Root rot	PI 424354	Ellis et al. (2013b)
		–	Gm01_49641478_A_G	(50,295,199–50,583,510 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	6.9%	LG00-3372	Scott et al. (2019)
		–	Gm01_52253980_C_T	(53,141,084 a2)	Modified seed rot assay	F9 (307)	6.6%SRS[c], 7.4%ROTS[d]	–	Clevinger et al. (2021)
	MLG D1b (Chr. 2)	–	Gm02_5035934_C_A	(5,054,610–5,321,601 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	9.6%	LD02-9050	Scott et al. (2019)
		–	Gm02_6529620_G_A	(6,572,325–7,031,201 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	7.8%	LD02-9050	Scott et al. (2019)
	MLG N (Chr. 3)	–	–	(31,912,038–33,553,037 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	12.3%	LG00-3372	Scott et al. (2019)
		–	Gm03_45516951_G_A	(43,485,660–43,849,572 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	6.9%	IA3023	Scott et al. (2019)
	MLG C1 (Chr. 4)	–	–	(5,314,249–5,903,949 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	20.0%	IA3023	Scott et al. (2019)
		–	Gm04_5837752_G_A	(5,314,249–6,972,200 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	12.2%	IA3023	Scott et al. (2019)
	MLG A1 (Chr. 5)	–	Gm05_389226_T_C	(2,220,637 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	12.2%	IA3023	Scott et al. (2019)
		–	BARC_050697_09840	(31,837,726 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	6.0%	PI 424354	Ellis et al. (2013b)
		–	Gm05_40791973_A_G	(39,546,900 a2)	Modified seed rot assay	F9 (307)	5.5%SRS, 4.8%ROTS	–	Clevinger et al. (2021)
	MLG C2 (Chr. 6)	–	BARC_013837_01254	(14,247,105 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	15.4%Weight, 14.9% Root rot	PI 424354	Ellis et al. (2013b)
		–	Gm06_31863080_C_T	(32,783,474 a2)	Modified seed rot assay	F7 (198)	26.6%SRS, 6.1%ROTS	–	Clevinger et al. (2021)
	MLG A2 (Chr. 8)	–	BARC_032503_08989	(7,774,531 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (127)	12.6% Weight	PI 424354	Ellis et al. (2013b)
		–	Gm08_8695745_A_C	(8,725,772 a2)	Modified seed rot assay	F9 (307)	16.7%SRS, 24.1%ROTS	–	Clevinger et al. (2021)
		–	BARC_041561_08032	(12,458,945 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (127)	10.3% Root rot	PI 424354	Ellis et al. (2013b)
		–	BARC_021577_04150	(18,143,273 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	8.8% Weight, 7.0% Root rot	PI 424354	Ellis et al. (2013b)
		–	BARC_018101_02517	(1,571,105 a1)		F2:3 (127)	14.7% Weight		

Table 6 (continued)

Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
	MLG O (Chr. 10)				Greenhouse test/isolates *Br2-3–5, Cler1-4–1*			PI 424354	Ellis et al. (2013b)
		–	Gm10_43821942_T_C	(44,218,338 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	10.0–10.9%	LG00-3372	Scott et al. (2019)
	MLG B1 (Chr. 11)	–	BARC_053481_11881	(3,675,507 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (127)	4.4% Weight	PI 424354	Ellis et al. (2013b)
		–	Gm11_15558504_T_C	(25,067,208 a2)	Modified seed rot assay	F7 (198)	7.9%SRS	–	Clevinger et al. (2021)
		qRRW11	Gm11_36581897_A_G	(32,115,772 a2)	Greenhouse test/isolate *CMISO2-5–14*	F4:7 (79)	15.4%RRW[e]	E09088	Lin et al. (2018)
		–	Gm11_38289103_C_T	(34,177,149–34,296,488 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	10.2%	LD02-9050	Scott et al. (2019)
	MLG F (Chr. 13)	–	BARC_900926_00961	(685,173 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (127)	8.2% Weight, 17.6% Root rot	PI 424354	Ellis et al. 92013b)
		–	BARC_062009_17616	(19,330,554 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	7.1% Weight, 6.3% Root rot	PI 424354	Ellis et al. (2013b)
		–	–	(22,901,190–25,230,180 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	12.4%	IA3023	Scott et al. (2019)
	MLG B2 (Chr. 14)	–	BARC_2.0_Gm14_2013931	(2,013,931 a2)	Greenhouse test/isolate *Brown 2–3-5*	F9:11 (316)	6.6%	Sloan	Stasko et al. (2016)
		–	BARC_065411_19443	(2,250,656 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	7.4% Root rot	PI 424354	Ellis et al. (2013b)
		–	BARC_015539_02002	(5,429,258 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	8.3% Weight	PI 424354	Ellis et al. (2013b)
	MLG J (Chr. 16)	–	Gm16_2780183_T_C	(1,034,335–3,225,680 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	8.3%	LG00-3372	Scott et al. (2019)
		–	Gm16_27322120_C_T	(28,348,383 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	12.4%	LD02-9050	Scott et al. (2019)
	MLG D2 (Chr. 17)	–	–	(4,610,230–6,517,544 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	11.0%	IA3023	Scott et al. (2019)
		–	–	(6,517,544–7,100,289 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (91)	13.6%	IA3023	Scott et al. (2019)
	MLG G (Chr. 18)	–	–	(9,205,527–10,045,551 a2)	Tray test/isolate *Br2-3–5*	SoyNAM RIL (116)	9.3%	LG00-3372	Scott et al. (2019)
	MLG L (Chr. 19)	*19–2*	BARC_2.0_Gm19_47784141	(47,784,141 a2)	Greenhouse test/isolate *Brown 2–3-5*	F9:11 (316)	5.5%	Sloan	Stasko et al. (2016)
	MLG I (Chr. 20)	*qRRW20*	Gm20_1348454_T_G	(1,344,091 a2)	Greenhouse test/isolate *CMISO2-5–14*	F4:7 (113)	12.7%-13.3% RRW	E05226-T	Lin et al. (2018)
		–	BARC_052017_11314	(2,109,173 a1)	Greenhouse test/isolates *Br2-3–5, Cler1-4–1*	F2:3 (192)	8.3% Weight, 6.0% Root rot	PI 424354	Ellis et al. (2013b)
Pythium oopapillum	MLG D1b (Chr. 2)	–	Gm02_47515175_G_A	(44,427,664 a2)	Modified seed rot assay	F8 (137)	8.9%SRS, 10.7%ROTS	–	Clevinger et al. (2021)
	MLG J (Chr. 16)	–	Gm16_6496577_A_C	(6,643,454 a2)	Modified seed rot assay	F7 (169)	8.6%SRS, 6.9%ROTS	–	Clevinger et al. (2021)

Table 6 (continued)

Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
Pythium sylvaticum	MLG D1a (Chr. 1)	–	Gm01_52253980_C_T	(53,141,084 a2)	Modified seed rot assay	F9 (307)	5.7%ROTS	–	Clevinger et al. (2021)
	MLG C2 (Chr. 6)	–	Gm06_31863080_C_T	(32,783,474 a2)	Modified seed rot assay	F7 (198)	26.9%SRS, 26.2%ROTS	–	Clevinger et al. (2021)
	MLG A2 (Chr. 8)	–	Gm08_8695745_A_C	(8,725,772 a2)	Modified seed rot assay	F9 (307)	4.9%SRS, 21.4%ROTS	–	Clevinger et al. (2021)
	MLG O (Chr. 10)	–	Gm10_43004105_A_C	(43,489,645 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	9.8%RRW	–	Lin et al. (2020)
		q10.1	Gm10_42975806_T_C	(43,517,944 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	9.8%RRW	–	Lin et al. (2020)
			Gm10_42965189_G_T	(43,528,561 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	9.8%RRW	–	Lin et al. (2020)
			Gm10_42963703_G_A and Gm10_43178809_G_T	109.21 cM (43,530,047–43,757,485 a2)	Greenhouse test/isolate *CMISO2 2–30*	F4:7 (113)	11.2%RRW	E05226-T	Lin et al. (2020)
		q10.2	Gm10_44563220_A_G and Gm10_44744804_A_C	123.61 cM (45,141,190–45,322,752 a2)	Greenhouse test/isolate *CMISO2 2–30*	F4:7 (113)	13.7%RRW	E05226-T	Lin et al. (2020)
		–	Gm10_48769298_A_G	(49,366,501 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	10.2%RRW	–	Lin et al. (2020)
	MLG B2 (Chr. 14)	–	Gm14_4770786_C_T	(4,856,342 a2)	Modified seed rot assay	F7 (198)	12.2%SRS, 13.1%ROTS	–	Clevinger et al. (2021)
	MLG G (Chr. 18)	*q18.1*	Gm18_6584445_C_T Gm18_6636054_G_T	8.51 cM (6,609,920—6,661,172a2)	Greenhouse test/isolate *CMISO2 2–30*	F4:7 (113)	9.5%RRW	E05226-T	Lin et al. (2020)
		q18.2	Gm18_7898429_A_C	(7,920,476 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	9.3%RRW	–	Lin et al. (2020)
			Gm18_7895324_G_A and Gm18_8851746_A_G	68.41 cM (7,917,371—8,886,122 a2)	Greenhouse test/isolate *CMISO2 2–30*	F4:7 (80)	11.3%RRW	E09088	Lin et al. (2020)
		–	Gm18_51777072_A_G	(47,496,041 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	9.6%RRW	–	Lin et al. (2020)
		–	Gm18_57517100_C_T	(53,247,366 a2)	Modified seed rot assay	F8 (137)	12.0%SRS, 10.9%ROTS	–	Clevinger et al. (2021)
	MLG I (Chr. 20)	–	Gm20_2245263_G_A	(2,239,157 a2)	Greenhouse test/isolate *CMISO2 2–30*	Germplasm (115), improved lines (99)	8.1%RRW	–	Lin et al. (2020)
		q20.1	Gm20_36002148_T_C and Gm20_36095037_G_A	13.91 cM	Greenhouse test/isolate *CMISO2 2–30*	F4:7 (80)	16.5%RRW	E05226-T	Lin et al. (2020)

Table 6 (continued)

Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
				(37,097,315—37,190,252 a2)					
Pythium torulosum	MLG M (Chr. 7)	–	Gm07_16031010_C_T	(16,121,771 a2)	Modified seed rot assay	F7 (169)	7.9%SRS	–	Clevinger et al. (2021)
	MLG A2 (Chr. 8)	–	Gm08_8695745_A_C	(8,725,711 a2)	Modified seed rot assay	F9 (307)	66.6%SRS	–	Clevinger et al. (2021)
Pythium ultimum var. *sporangiiferum*	MLG N (Chr. 3)	–	Gm03_140242_G_A	(8,046–172,048 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (123)	9.7%	IA3023	Scott et al. (2019)
		–	Gm03_511376_C_T	(425,209–587,640 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (123)	15.9%	IA3023	Scott et al. (2019)
		–	–	(425,209–510,431 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (123)	10.1%	IA3023	Scott et al. (2019)
	MLG A1 (Chr. 5)	–	Gm05_41540078_C_A	(38,639,960–38,838,119 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (122)	10.2%	IA3023	Scott et al. (2019)
	MLG B1 (Chr. 11)	–	Gm11_36517294_T_C	(32,006,970 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (122)	9.2%	IA3023	Scott et al. (2019)
	MLG D2 (Chr. 17)	–	–	(37,799,552–38,965,056 a2)	Tray test/isolate *Will1.6.7*	SoyNAM RIL (122)	9.8%	HS6-3976	Scott et al. (2019)
Pythium ultimum var. *ultimum*	MLG D1a (Chr. 1)	–	–	(8,388,480–10,556,016 a2)	Tray test/isolate *Miami1-3–7*	SoyNAM RIL (94)	12.1%	IA3023	Scott et al. (2019)
	MLG D1b (Chr. 2)	-	-	(13,877,996–14,102,777 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (122)	9.2%	HS6-3976	Scott et al. (2019)
		-	Gm02_13904897_A_G	(13,877,996–14,206,854 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (122)	12.8%	HS6-3976	Scott et al. (2019)
	MLG N (Chr. 3)	-	Gm03_588585_C_T	(510,431–853,885 a2)	Tray test/isolate *Miami1-3–7*	SoyNAM RIL (94)	16.8%	IA3023	Scott et al. (2019)
	MLG A1 (Chr. 5)	-	Gm05_41903142_C_T	(38,388,163–38,446,748 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (75)	18.5%	IA3023	Scott et al. (2019)
	MLG C2 (Chr. 6)	–	BARC-055889–13,824 and BARC-064115–18,558	(17,236,088–17,966,360 a2)	Greenhouse cup assay/isolate *Miami 1–3-7*	F7:8 (247)	13.9–11.4% PS [f], 8.5–12.2%RW [g], 11.8–12.6%RRS [h]	Magellan	Klepadlo et al. (2019)
		–	bin90–bin91	(17,234,790–17,585,393 a2)	Greenhouse cup assay/isolate *Miami 1–3-7*	F7:8 (247)	8.8%PS, 7.5–8.4%, 7.8–8.8%RRS	Magellan	Klepadlo et al. (2019)
	MLG M (Chr. 7)	–	Gm07_36955973_T_C	(30,099,305–36,907,555 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (75)	16.3%	S06-13,640	Scott et al. (2019)
	MLG A2 (Chr. 8)	–	bin36–bin37	(8,767,341–9,027,146 a2)	Greenhouse cup assay/isolate *Miami 1–3-7*	F7:8 (247)	8.8–12.3%PS, 11.4–16.8%RW, 7.8–11.4%RRS	Magellan	Klepadlo et al. (2019)
		–	BARC-010097–00,518 and Satt187	(8,937,354–9,192,645 a2)	Greenhouse cup assay/isolate *Miami 1–3-7*	F7:8 (247)	7.3% PS, 11.6% RW, 6.4% RRS	Magellan	Klepadlo et al. (2019)
		–	BARC-010097–00,518 and BARC 050,171–09,440	(8,937,354–9,457,315 a2)	Greenhouse cup assay/isolate *Miami 1–3-7*	F7:8 (247)	10.1% PS, 11.6% RW, 10.5% RRS	Magellan	Klepadlo et al. (2019)
		–	–		Tray test/isolate *Miami1-3–7*	SoyNAM RIL (94)	17.2%	IA3023	

Table 6 (continued)

Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
	MLG F (Chr. 13)	–	Gm13_40441579_G_T	(25,230,180–26,955,004 a2) (40,935,278–41,953,362 a2)	Tray test/isolate *Miami1-3–7*	SoyNAM RIL (94)	12.7%	IA3023	Scott et al. (2019) Scott et al. (2019)
	MLG D2 (Chr. 17)	–	–	(4,949,843–6,517,544 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (75)	24.4%	IA3023	Scott et al. (2019)
		–	Gm17_41060022_G_A	(40,457,644–40,876,232 a2)	Tray test/isolate *Miami1-3–7, N201.2.2*	SoyNAM RIL (122)	12.2%	HS6-3976	Scott et al. (2019)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[b]Phenotypic variations explained by the molecular markers

[c]SRS: seed rot severity

[d]ROTS: percent rotted seeds in inoculated plates

[e]RRW: ratio of fresh root weight

[f]PS: plant stand

[g]RW: fresh root weight

[h]RRS: root rot score

corrected dry weight (CDW) (Dorrance et al. 2008; de Ronne et al. 2020, 2021). Twenty-one validated QTLs were stably identified in at least two independent studies (Table 5). These QTLs were distributed on 13 soybean chromosomes and may be of high priority to develop soybean varieties with horizontal resistance against *P. sojae*. Notably, *qRps18-01* (formerly named *QDRL-18* or *OH-18–1*), a major QTL conferring more than 20% of horizontal resistance (Lee et al. 2014; Karhoff et al. 2019; Rolling et al. 2020), as well as other newly identified QTLs, are being integrated into future soybean varieties through collaborated efforts. Moreover, more than 130 additional QTLs were also reported which provided diverse options for soybean breeders (Supplementary Table 1).

With respect to other *Phytophthora* species, *P. sansomeana* E.M. Hansen & Reeser is an emergent pathogen in soybean-producing areas and causes root rot diseases. Lin et al. (2021) identified and validated two QTLs that contributed horizontal resistance to this pathogen from improved soybean varieties developed at the Michigan State University soybean breeding program (Table 5). Marker-assisted resistance spectrum analysis indicated five patterns of interactions between QTLs and *P. sansomeana* isolates. The validated QTLs can be efficiently integrated into future soybean varieties using MAS with low linkage drag of undesirable agronomic traits, since both donor parents are improved soybean varieties.

Pythium damping-off and root rot

The genus *Pythium* is typically linked with early-season diseases, such as seedling root rot and damping-off, and multiple species have been implicated (Zhang et al. 1998; Zhang and Yang. 2000). Among the most damaging species, *P. aphanidermatum*, *P. ultimum*, *P. irregulare*, and *P. sylvaticum* have been used to screen potential sources of resistance for breeding efforts to reduce the impact of these pathogens (Ellis et al. 2013b; Scott et al. 2019; Lin et al. 2020; Clevinger et al. 2021). Horizontal resistance is currently the only type of resistance identified for most *Pythium* species, except *Rpa1*, which was identified from cv. 'Archer' as a single dominant resistance gene against *P. aphanidermatum* (Table 6) (Cianzio et al. 1991; Kirkpatrick et al. 2006; Bates et al. 2008; Rosso et al. 2008). The *Rpa1* gene is located on Chr. 13 (molecular linkage group F, MLG F), 10.6 cM and 26.6 cM from the SSR markers Satt510 and Satt114, respectively (Rosso et al. 2008). In addition to *Rpa1*, two QTLs were identified for *P. aphanidermatum* from Archer, which were located on chromosomes 4 and 7, and accounting for 8.29–13.85% and 4.5–13.85% of phenotypic variations, respectively (Urrea et al. 2017). Moreover, Archer also confers

resistance to seed rot and root discoloration caused by *P. ultimum* and other species of *Pythium* including *Phytopythium. vexans* (formerly *Pythium vexans*), *P. irregulare*, and hyphal swelling (HS) group (Bates et al. 2004, 2008; Kirkpatrick et al. 2006; Rupe et al. 2011), yet the genes/QTLs conferring those resistances in Archer are unclear.

Horizontal resistance was also identified for other *Pythium* spp. Lin et al. (2020) identified and validated two QTLs for *P. sylvaticum* using QTL mapping and GWA methods. The two QTLs were located on chromosomes 10 and 18 and explained 9.8–11.2% and 9.3–11.3% of phenotypic variations, respectively. Remarkably, pleiotropic QTLs have been frequently identified for resistance to several *Pythium* species or varieties. For example, Scott et al. (2019) identified one QTL on Chr. 3 for resistance to *P. ultimum* var. *ultimum* and *P.ultimum* var. *sporangiiferum*, and other two QTLs (on chromosomes 13 and 17, respectively) that both confer resistance to *P. irregulare* and *P. ultimum* var. *ultimum*. In a more recent study, a major QTL was identified (nearest marker Gm08_8695745_A_C) conferring resistance to *P. irregulare* (16.7–24.1% of phenotypic variations), *P. sylvaticum* (4.9–21.4%), and *P. torulosum* (66.6%), and another large effect QTL (nearest marker Gm06_31863080_C_T) for resistance to *P. sylvaticum* (26.2–26.9%) and *P. irregulare* (6.1–26.6%) (Clevinger et al. 2021). In the future, these validated and pleiotropic QTLs will be of high priority in MAS to develop soybean varieties with tolerance to different *Pythium* pathogens.

Downy mildew

Soybean downy mildew, caused by *Peronospora manshurica* (Naum.) Sdy., is a common leaf disease throughout the world (Lim et al. 1989). Although severe yield loss is rarely reported, soybean downy mildew can reduce the size and quality of soybean seeds (Palmer et al. 2004; Taguchi-Shiobara et al. 2019). Three resistance genes, *Rpm1*, *Rpm2*, and *Rpmx,* have been reported from soybean varieties 'Kanrich', 'Fayette', and PI 88,788, and 'AGS129', respectively, although the genetic and physical location of the resistance genes remain unclear (Geeseman et al. 1950ab, Bernard and Cremeens 1971; Lim et al. 1984; Lim 1989; Chowdhury et al. 2002). Recently, quantitative resistance to soybean downy mildew was first reported in Japan (Taguchi-Shiobara et al. 2019). Remarkably, *QRpm3-1* and *QRpm7-1* were identified and confirmed in several mapping populations across multiple years, each explaining 18–72% and 28–91% of the observed phenotypes (Table 7).

Section III. Soybean resistance to fungal diseases

Sudden death syndrome and Fusarium wilt and root rot

In the USA, Sudden Death Syndrome (SDS) was initially detected in the State of Arkansas in 1971 (Rupe and Weidemann 1986; Rupe 1989) and has since spread to the majority of soybean producing states (Hartman et al. 2016). In recent years, SDS has been detected in South Dakota (Tande et al. 2014), New York (Cummings et al. 2018), and North Dakota (Nelson et al. 2018). In Brazil, it was first observed in 1981/82 in the State of Minas Gerais (Nakajima et al. 1996). It received the name of red root rot (PVR), as it is still known in that country. This important disease also occurs in Argentina (Ploper 1993), Canada (Anderson and Tenuta 1998), Bolivia (Yorinori 1999), Paraguay (Yorinori 2002), and Uruguay (Ploper et al. 2003).

The major causal agent of SDS identified in the USA is the fungus *Fusarium virguliforme* O'Donnell and T. Aoki (formerly *F. solani* (Mart.) Sacc. f. sp. *glycines*) (Aoki et al. 2003), although a recent study reported that *F. Brasiliense* also causes SDS in the USA (Wang et al. 2019). SDS and *F. virguliforme* were also reported in Malaysia (Chehri et al. 2014) and South Africa (Tewoldemedhin et al. 2014). In Brazil, four fungi have been reported to cause SDS, including *F. virguliforme*, *F. brasiliense*, *F. crassistipitatum*, and *F. tucumaniae*. In addition, *F. brasiliense, F. crassistipitatum, and F. tucumaniae* have been reported to cause SDS in other countries in South America (Aoki et al. 2003, 2005, 2012).

Significant yield losses can occur due to SDS (Aoki et al. 2003). SDS favors cool and wet environment. The symptoms of SDS can be observed on the roots and the aboveground foliage. The fungus initiates its infestation by colonizing the soybean roots, causing root rot and necrosis, which leads to the loss of root mass and root nodules. The fungus may sporulate on the roots producing clusters of conidia that appear to be blue. The aboveground symptom of SDS is caused by the translocation of phytotoxin, the symptoms include interveinal chlorosis and necrosis; leaf abscission at the top of the petiole rather than the base; and eventually, early plant death. Foliage symptoms are generally observed in the later reproductive stages after flowering but may develop earlier (Roy et al. 1997; Aoki et al. 2003; Hartman et al. 2016; Chang et al. 2018).

Cultural practices and planting resistant varieties are the most common methods used to manage SDS (Wrather et al. 1995; Luckew et al. 2012). The soybean community has devoted substantial effort to identifying QTLs that underlie

Table 7 Soybean loci associated with resistance to downy mildew (caused by *Peronospora manshurica*)

MLG (Chr.)	Locus name	Tightly linked/ flanking markers	Marker position	Testing methods/ Resistance spectrum	Population type/size	PVE[a]	Donor source	References
–	*Rpm1*	–	–	Greenhouse test/ races *1–32*		R gene	Kanrich	Geesman et al. (1950a b), Bernard and Cremeens (1971), Lim et al. (1984)
–	*Rpm2*	–	–	Greenhouse test/ races *2, 33*	F2/98–242, F3/55–106	R gene	Fayette and PI 88788	Lim et al. (1984), Lim (1989)
–	*Rpmx*	OPH-02_{1250} and OPP-10_{831}	–	Field test (Thailand)	F2/102	R gene	AGS129	Chowdhury et al. (2002)
MLG D1b (Chr. 2)	*QRpm2-1*	WGSP02_0160-WGSP02_0170	Around 50 Mb	Field test (Japan)	F6 and F7(112)	8–10%	Harosoy	Taguchi-Shiobara et al. (2019)
MLG N (Chr. 3)	*QRpm3-1*	WGSP03_0040-WGSP03_0070	5-30 Mb	Field test (Japan)	F6 and F7(155), F5 and F6(190), F6 and F7(112)	18–72%	Fukuibuki, Kinusayaka, Harosoy	Taguchi-Shiobara et al. (2019)
MLG C1 (Chr. 4)	*QRpm4-1*	WGSP04_0120-WGSP04_0140	–	Field test (Japan)	F6 and F7(155)	4%	Satonohohoemi	Taguchi-Shiobara et al. (2019)
MLG C2 (Chr. 6)	*QRpm6-1*	WGSP06_0200-WGSP06_0210	–	Field test (Japan)	F6 and F7(155)	8%	Fukuibuki	Taguchi-Shiobara et al. (2019)
MLG M (Chr. 7)	*QRpm7-1*	WGSP07_0060-WGSP07_0070	Around 5 Mb	Field test (Japan)	F5 and F6 (189), F9 and F10(231), (F5 and F6(190)	6–91%	Tachinagaha, Suzumaru, COL/Akita2009/TARC/1	Taguchi-Shiobara et al. (2019)
	QRpm7-2	WGSP07_0080-WGSP07_0130	–	Field test (Japan)	F9 and F10(231)	47–91%	Suzumaru	Taguchi-Shiobara et al. (2019)
MLG A2 (Chr. 8)	*QRpm8-1*	WGSP08_0110-WGSP08_0130	Around 20 Mb	Field test (Japan)	F6 and F7(155)	13–24%	Fukuibuki	Taguchi-Shiobara et al. (2019)
MLG B1 (Chr. 11)	*QRpm11-1*	WGSP11_0100-WGSP11_0120	–	Field test (Japan)	F9 and F10(231)	4%	Suzumaru	Taguchi-Shiobara et al. (2019)
MLG H (Chr. 12)	*QRpm12-1*	WGSP12_0120-WGSP12_0130	Around 35 Mb	Field test (Japan)	F6 and F7(112)	6–8%	Harosoy	Taguchi-Shiobara et al. (2019)
MLG F (Chr. 13)	*QRpm13-1*	WGSP13_0080-WGSP13_0120	–	Field test (Japan)	F5 and F6 (189)	3%	Tachinagaha	Taguchi-Shiobara et al. (2019)
MLG B2 (Chr. 14)	*QRpm14-1*	WGSP14_0050-WGSP14_0060	–	Field test (Japan)	F5 and F6 (189)	4%	Tachinagaha	Taguchi-Shiobara et al. (2019)
MLG E (Chr. 15)	*QRpm15-1*	WGSP15_0130-WGSP15_0140	–	Field test (Japan)	F5 and F6(190)	3%	Kinusayaka	Taguchi-Shiobara et al. (2019)

Table 7 (continued)

MLG (Chr.)	Locus name	Tightly linked/ flanking markers	Marker position	Testing methods/ Resistance spectrum	Population type/size	PVE[a]	Donor source	References
MLG J (Chr. 16)	*QRpm16-1*	WGSP16_0090-WGSP16_0100	–	Field test (Japan)	F5 and F6(190)	3%	COL/Akita2009/TARC/1	Taguchi-Shiobara et al. (2019)
MLG G (Chr. 18)	*QRpm18-1*	WGSP18_0150-WGSP18_0160	Around 50-60 Mb	Field test (Japan)	F5 and F6 (189)	11–16%	Tachinagaha	Taguchi-Shiobara et al. (2019)
MLG L (Chr. 19)	*QRpm19-1*	WGSP19_0150-WGSP19_0170	–	Field test (Japan)	F6 and F7(155)	7%	Fukuibuki	Taguchi-Shiobara et al. (2019)
MLG I (Chr. 20)	*QRpm20-1*	WGSP20_0100-WGSP20_0130	–	Field test (Japan)	F5 and F6 (189)	4%	Tachinagaha	Taguchi-Shiobara et al. (2019)
	QRpm20-2	WGSP20_0090-WGSP20_0100	–	Field test (Japan)	F6 and F7(112)	5%	Harosoy	Taguchi-Shiobara et al. (2019)

[a]Phenotypic variations explained by the molecular markers

SDS resistance. To date, more than 200 resistance-associated markers have been identified (Table 8 and Supplementary Table 2). After mapping a resistance locus, it is important to confirm and incorporate it into multiple genetic backgrounds to determine whether it will maintain its effect and be useful in a breeding program. Based on the classification of Chang et al.(2018) as well as the studies thereafter, twenty-five confirmed QTLs have been identified from at least two independent studies (Table 8), including a single locus on chromosomes 2, 4, 5, 8, 9, 14, 16, and 19, two on chromosomes 3, 13, 15 and 17, and three on chromosomes 6, 18, and 20. Most of these loci were confirmed in at least one field study, except *qRfv06-03*, which was confirmed in three greenhouse studies (Abdelmajid et al. 2012; Bao et al. 2015; Luckew et al. 2017), and *qRfv20-03*, which was validated in a greenhouse study and a growth chamber study (Swaminathan et al. 2016; de Farias Neto et al. 2007). Notably, *qRfv05-01* confers resistance to both *F. virguliformes* and *F. tucumaniae*, a causal agent of SDS in South America. Ninety additional loci were also reported and may need confirmation in future studies (Supplementary Table 2). The confirmed QTLs can be pyramided into elite cultivars with high confidence for durable resistance. There are no reports on genetic mechanisms of the genes but, stacking the two distinct SDS resistance mechanisms, resistance to root rot and leaf scorch is the better strategy to increase resistance (Wang et al. 2016).

In addition to SDS, other *Fusarium* spp. pathogens (such as *F. redolens*, *F. proliferatum*, *F. oxysporum*, *F. equiseti*, *F. acuminatum*, *F. moniliforme*, *F. graminearum*, *F. semitectum*, *F. chlamydosporum*, *F. compactum*, *F. merimoides*, *F. roseum*, *F. tricinctum, F. avenaceum,* and *F. sporotrichioides*) can also infect soybean, causing wilt, damping-off, and root rot (Arias et al. 2013). Of these *Fusarium* spp., *F. graminearum* was highly aggressive (root rot severity > 90%), causing seed rot and seedling damping-off in South America, Canada, and the USA (Pioli et al. 2004; Broders et al. 2007; Xue et al. 2007; Ellis et al. 2013a; Arias et al. 2013; Cheng et al. 2017a). Horizontal resistance is the only type of resistance so far identified for *F. graminearum*. Since the first report of five QTLs from 'Conrad' and 'Sloan', a total of thirty QTLs have been identified, accounting for 3.1–40.2% of phenotypic variations on 13 soybean chromosomes (Table 9). Based on the physical locations of the tightly linked or flanking markers, five loci can be validated from two or more QTL mapping or GWA studies, including *qRfg08-01* (17.2–47.4 Mb) and *qRfg08-02* (4.0–9.2 Mb) on Chr. 8, *qRfg13-01* (11.1–39.3 Mb) on Chr. 13, and *qRfg19-01* (47.5–47.8 Mb) and *qRfg19-02* (9.2–41.3 Mb) on Chr. 19 (Table 9). These QTLs can be of higher interest to develop resistant soybean varieties against *F. graminearum.*

Table 8 Validated loci associated with resistance to soybean sudden death syndrome (SDS) disease (caused by *Fusarium virguliformes*)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
qRfv02-01	MLG D1b (Chr. 2)	*SDS13-5, qFDS003-02*	ss107920774—ss107912689	30.0–36.0 cM	Greenhouse test/ isolate *Mont1*	F6:13 (50)	5.2% FDS[d]	PI 438489B	Abdelmajid et al. (2012)
		–	ss244884978	(49,773,810 a1)	Field test (MI, US)	Advanced breeding lines (300)	6.4% DI[e]	–	Wen et al. (2014)
		SDS15-4	BARC-041581–08,046—BARC-046084–10,230	93.341–02.59 cM (41,337,886–43,414,601 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	8.4% Stem cut	LS94-3207	Swaminathan et al. (2016)
qRfv03-01	MLG N (Chr. 3)	*SDS2-7, SDS QTL 1 N, qRfs6*	$OC01_{650}$	–	Field test (IL, US)	F5:11 (100)	16% DI	Forrest	Chang et al. (1996)
		SDS2-8, SDS QTL 1 N	$OF04_{1600}$	–	Field test (IL, US)	F5:11 (100)	10% DI	Forrest	Chang et al. (1996), Chang et al. (2018)
		SDS1-2, SDS1-4	$OC01_{650}$	–	Field test (IL, US)	F5:9 (100)	30% DI	Forrest	Hnetkovsky et al. (1996), Chang et al. (2018)
		–	*ss715586494_C_T*	(44,251,912 a2)	Greenhouse test	Germplasm (214)	9% DAI29[f]	–	Zhang et al. (2015a), Chang et al. (2018)
qRfv03-02	MLG N (Chr. 3)	*SDS13-10, qRRS001-01*	ss107912585—ss107920575	38.3–42.6 cM	Greenhouse test/ isolate *Mont1*	F6:13 (50)	9.9% RRS[g]	PI 438489B	Abdelmajid et al. (2012), Chang et al. (2018)
		qDX004	ss245025977—ss245026227	15.90–16.10 cM	Field test (IL, US)	F5:7 (94)	0.1% DX [h]	–	Anderson et al. (2015), Chang et al. (2018)
		SDS14-2, qDX003	ss245026358—ss245025977	15.70–15.90 cM	Field test (IL, US)	F5:7 (94)	0.8% DX	–	Anderson et al. (2015), Chang et al. (2018)
		SDS8-3, qRfs6	Satt080	–	Field test (IL, US)	F6 derived (90); F2:3 (321)	15.6% DI	Pyramid	Njiti et al. (2002), Luckew et al. (2013), Chang et al. (2018)
		Di1, qRfs6	Satt080—Satt387	51.61 cM	Field test (IL, US)	RIL (94); F2:3 (321)	15.9% DI	Forrest	Kassem et al. (2006), Luckew et al. (2013), Chang et al. (2018)
		ds2, qRfs6	Satt080—Satt387	51.61 cM	Field test (IL, US)	RIL (94); F2:3 (321)	14.2% DS[i]	Forrest	Kassem et al. (2006), Luckew

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
									et al. (2013), Chang et al. (2018)
		dx1, qRfs6	Satt080—Satt387	49.61 cM	Field test (IL, US)	RIL (94); F2:3 (321)	17.3% DX	Forrest	Kassem et al. (2006), Luckew et al. (2013), Chang et al. (2018)
		di8, QRfs7	Satt080—Satt387	51.61 cM	Field test (IL, US)	RIL (100)	15.9%		Abdelmajid et al. (2007), Chang et al. (2018)
		-	Satt387	(34,554,705 a2)	Field test (IL, US)	F6 derived (90)	10.2% DI	Pyramid	Njiti et al. (2002)
qRfv04-01	MLG C1 (Chr. 4)	*SDS13-15, qFDS004-03*	ss107924445—ss107918378	57.3–83.9 cM	Greenhouse test/ isolate *Mont1*	F6:13 (50)	4.8%	PI 438489B	Abdelmajid et al. (2012)
		SDS9-3	A063_1	–	Greenhouse test/ Strain ST-90	F7:14 (284)	5% DS	Noir1'	Njiti and Lightfoot (2006)
		SDS disease incidence 20–1, SDS disease index 20–1, qSDS-4	ss245526764—ss245561373	48.56–83.86 cM (43.8 Mb–47.3 Mb a1)	Field test (MI, US)	F4 derived (129)	3.7–5.3% DI, DX	GD2422	Tan et al. (2018)
		qPY4-1	ss245560843—ss245567348	(47.29 Mb48.08 Mb a2)	Field test (MI, US)	F4 derived (153)	6.36% plot yield	E07080	Tan et al. (2019)
qRfv05-01	MLG A1 (Chr. 5)	*SDS14-4, qDX005*	ss245747167-ss245786667	9.20–10.00 cM/ 8.50–11.70 cM	Field test (IL, US)	F5:7 (94)	0.01–0.04% DX	-	Anderson et al. (2015)
		SDS15-8	BARC-059081–15,595 to BARC-065229–19,273	57.79–78.44 cM (35,367,094–37,815,203 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	7.0% Root feeding	LS94-3207	Swaminathan et al. (2016)
		RSDS3	Satt545	(36,463,225 a2)	Greenhouse test/ *F. tucumaniae* sp. nov. MJ161	F8 derived (156)	9.3% DX	Moshidou Gong 503	Yamanaka et al. (2006)
qRfv06-01	MLG C2 (Chr. 6)	*di5, qRfs4*	Satt371	4.4 cM (49,760,138 a2)	Field test (IL, US)	RIL (100); F2:3 (321)	12.1% DI	Essex	Abdelmajid et al. (2007), Luckew et al. (2013), Chang et al. (2018)
		SDS14-6, qDX007	ss246087580-ss246092064	7.20–7.50 cM	Field test (IL, US)	F5:7 (94)	0.6% DX	–	

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
									Anderson et al. (2015), Chang et al. (2018)
		qDX008	ss246091245-ss246092064	7.20–7.30 cM	Field test (IL, US)	F5:7 (94)	0.5% DX	–	Anderson et al. (2015), Chang et al. (2018)
		SDS2-5, SDS QTL 1C2	$OO05_{250}$	–	Field test (IL, US)	F5:11 (100)	13% DI	Essex	Chang et al. (1996), Chang et al. (2018)
		SDS2-6, SDS QTL 1C2	K455D-1	–	Field test (IL, US)	F5:11 (100)	16% DI	Essex	Chang et al. (1996), Chang et al. (2018)
		SDS1-1	$OO05_{250}$	–	Field test (IL, US)	F5:9 (100)	26% DI	Essex	Hnetkovsky et al. (1996), Chang et al. (2018)
		SDS7-5, qRfs4	Satt371	(49,760,138 a2)	Field test (IL, US)	F5:13 (100); F2:3 (321)	12.0% DI	Essex	Iqbal. et al. (2001), Luckew et al. (2013), Chang et al. (2018)
		SDS8-2	Satt307	–	Field test (IL, US)	F6 derived (90)	13.6% DI	Douglas	Njiti et al. (2002), Chang et al. (2018)
		SDS16-5	BARC-010457–00,640—BARC-025767–05,060	121.26–126.23 cM (45,851,263–48,305,238 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	8.6% Root feeding	A95-684043	Swaminathan et al. (2016), Chang et al. (2018)
		SDS14-5, qDX006	ss246091245—ss246092064	7.20–7.30 cM	Field test (IL, US)	F5:7 (94)	0.9% DX	–	Anderson et al. (2015), Chang et al. (2018)
		SDS4-2	K455	–	Field test (IL, US)	F5:13 (100)	2%—9% DX, IS	Essex'	Njiti et al. (1998)
		–	ss246038868	–	Field test (MI, US)	Advanced breeding lines (300)	5.7% DX	–	Wen et al. (2014)
		qSDS6-6	ss246084690—ss246086447	(46.11–46.25 a2)	Field test (MI, US)	F4 derived (153)	8.8%	E07080	Tan et al. (2019)
		qSDS6-7	ss246098726—ss246102570	(47.27–57.59 a2)	Field test (MI, US)	F4 derived (153)	10.8%	E07080	Tan et al. (2019)
qRfv06-02	MLG C2 (Chr. 6)	*ds6, QRfs5*	Satt489—Satt286	99.21 cM (16,221,044–23,848,501 a2)	Field test (IL, US)	RIL (100)	12.1% DI	Essex	Abdelmajid et al. (2007), Chang et al. (2018)
		ds1	Satt489—Satt286			RIL (94)	15.4% DS	Essex	

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
				99.21 cM (16,221,044–23,848,501 a2)	Field test (IL, US)				Kassem et al. (2006), Chang et al. (2018)
		SDS11-1, cqRfs4	Satt277, Satt079	(17,218,677, 44,503,658 a2)	Field test (IL, US)	F5:14 (92)	8.2%-24.1% DX	Flyer	Kazi et al. (2008), Chang et al. (2018)
		SDS16-6	BARC-021735–04,194—BARC-062515–17,881	97.83–121.26 cM (16,029,425–46,596,066 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	12% Root feeding	A95-684043	Swaminathan et al. (2016), Chang et al. (2018)
		qSDS6-3	ss245888974—ss245909007	(15.03–16.81 Mb a2)	Field test (MI, US)	F4 derived (153)	7.0–9.5%	E07080	Tan et al. (2019)
		qSDS6-4	ss245925990—ss246010254	(18.63–39.82 Mb a2)	Field test (MI, US)	F4 derived (153)	9.8%	E07080	Tan et al. (2019)
		qSDS6-5	ss246041195—ss246068439	(43.16–44.82 Mb a2)	Field test (MI, US)	F4 derived (153)	11.1%	E07080	Tan et al. (2019)
		qPY6-1	ss245879277—ss245882767	(14.05–14.42 Mb a2)	Field test (MI, US)	F4 derived (153)	4.8% plot yield	E07080	Tan et al. (2019)
		SDS13-6, qFDS003-03, qFDS004-04	ss107929602—ss107925487 ss107930961—ss107912561	32.8–39.2 cM/34.5–39.8 cM	Greenhouse test/ isolate *Mont1*	F6:13 (50)	3.2–4.7% FDS	PI 438489B	Abdelmajid et al. (2012)
qRfv06-03	MLG C2 (Chr. 6)	*SDS13-14, qFDS003-05, qFDS004-03*	ss107917031—ss107912977	16.9–32.8 cM (12,273,659–17,424,199 a1)	Greenhouse test/ isolate *Mont1*	F6:13 (50)	2.1–2.4%	PI 438489B	Abdelmajid et al. (2012)
		cqRfs4	BARC-028177–05,786	(13,551,218 a1)	Greenhouse test/ isolate *Somerset #1A*	Ancestral lines, advanced breeding lines, cultivars, and landraces (282)	–	–	Bao et al. (2015)
		Fusarium root rot 1–1	Gm06_13621986_A_C to Gm06_15571070_T_C	(13,621,986–15,571,070 a1)	Greenhouse test/ isolate *LL0009* (NE305)	F2:3 (200)	18.0% Root rot severity	MN1606SP	Luckew et al. (2017)
qRfv08-01	MLG A2 (Chr. 8)	*SDS13-13, qFDS003-06*	ss107915722—ss107918074	15.0–28.0 cM (4,646,825–9,489,665 a1)	Greenhouse test/ isolate *Mont1*	F6:13 (50)	17.40%	PI 438489B	Abdelmajid et al. (2012)
		SDS15-2	BARC-031701–07,215—BARC-016685–03,321	14.99–51.86 cM (3,060,343–8,204,715 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	8.4% Stem cut	A95-684043	Swaminathan et al. (2016)
		SDS15-3					5.8% Stem cut		

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
			BARC-016685–03,321—BARC-038631–07,266	51.86–58.43 cM (8,204,715–10,215,938 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)		A95-684043	Swaminathan et al. (2016)
		SDS disease incidence 20–2, qSDS-8	ss246481149—ss246509551	70.15–84.97 cM (7.8–10.8 Mb a1)	Field test (MI, US)	F4 derived (129)	5.2–8.5% DI	LD01-5907	Tan et al. (2018)
qRfv09-01sss	MLG K (Chr. 9)	*SDS14-7, qDX009*	ss246870684—ss246865400	0.40–0.50 cM	Field test (IL, US)	F5:7 (94)	0.5% DX	–	Anderson et al. (2015), Chang et al. (2018)
		SDS16-1	BARC-056323–14,257—BARC-010353–00,615	45.74–50.93 cM (22,251,525–38,869,688 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	13% Stem cut	LS98-0582	Swaminathan et al. (2016), Chang et al. (2018)
		qRfs18	Satt381	(12,849,836 a2)	–	–	–	–	Lightfoot et al. (2015), Chang et al. (2018)
		qSDS9-1	ss246827311—ss246949164	(7.00–34.27 Mb a2)	Field test (MI, US)	F4 derived (153)	9.0–11.7%	U01-390489	Tan et al. (2019)
		SDS18-3, SDS-3	BARC-058901–15,494—BARC-050815–09,887; Satt552–Satg002	(11,775,727–33,502,306 a2)	Growth chamber/ isolates *Clinton 1B* and *Scott F2II 1a*	F7:8 (200)	4.6%	LS94-3207	Swaminathan et al. (2018)
qRfv13-01	MLG F (Chr. 13)	*dx1, QRfs9*	Satt510	27.3 cM (31,802,559 a2)	Field test (IL, US)	RIL (100)	10.2% DX	Forrest	Abdelmajid et al. (2007), Chang et al. (2018)
		SDS disease incidence 21–1	Gm13_26749514_T_C—Gm13_42027425_G_T	(27,943,258–43,467,121 a2)	Greenhouse test/ isolate *LL0009* (NE305)	F2:3 (200)	11.4% DI	Spencer	Luckew et al. (2017), Chang et al. (2018)
		SDS15-1	BARC-065495–19,507—BARC-030899–06,963	72.97–78.05 cM (29,074,011–30,510,485 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	16% Stem cut	A95-684043	Swaminathan et al. (2016), Chang et al. (2018)
		SDS16-8	BARC-010501–00,676—BARC-042515–08,280	74.12–78.05 cM (29,598,124–30,174,729 a1)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	12% Root feeding	LS98-0582	Swaminathan et al. (2016), Chang et al. (2018)
		–	ss248117124	(34,867,303 a2)	Field test (MI, US)	Advanced breeding lines (300)	5.7% DX	–	Wen et al. (2014), Chang et al. (2018)
		–	ss715614656_G_A	(28,548,247 a2)	Greenhouse test	Germplasm (214)	6% DAI20	–	

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
									Zhang et al. (2015a, b), Chang et al. (2018)
qRfv13-02	MLG F (Chr. 13)	–	SGM13_13250813		Growth chamber	F2 lines (135)	12.6% foliar necrosis	PI 243518	Chang et al. (2020)
		ds1, QRfs8	Satt160—Satt252	(16,454,986–17,875,691 a2)	Field test (IL, US)	RIL (100); F2:3 (321)	16.9% DS, 11.20% DX	Forrest	Abdelmajid et al. (2007), Luckew et al. (2013) Chang et al. (2018)
		ds3, qRfs12	Satt160—Satt252	(16,454,986–17,875,691 a2)	Field test (IL, US)	RIL (94); F2:3 (321)	6.3% DS	Essex	Kassem et al. (2006), Luckew et al. (2013), Chang et al. (2018)
		–	Gm13-4,584,015	(17,285,679 a2)	Field test (MI, US)	Cultivars (392)	7.2% DI	–	Wen et al. (2014)
		SDS17-2, SDS-5	BARC-900926–00,961—BARC-041237–07,944; Sat_298–Satt423	(16,825,744–21,179,508 a2)	Growth chamber/ isolates *Clinton 1B* and *Scott F2II 1a*	F7:8 (200)	9%	LS98-0582	Swaminathan et al. (2018)
		–	Sat_039—Satt160	–	Field test (IL, US)	F5:14 (92)	20% DS, 19% DX	Forrest	Yuan et al. (2012)
qRfv14-01	MLG B2 (Chr. 14)	*SDS14-10, qDX012*	ss248293401—ss248275088	1.40–4.10 cM	Field test (IL, US)	F5:7 (94)	0.03% DX	–	Anderson et al. (2015)
		qDX013	ss248293401—ss248275088	12.90–18.20 cM	Field test (IL, US)	F5:7 (94)	0.03% DX	–	Anderson et al. (2015)
		SDS14-11, qDX014	ss248293401—ss248275088	10.30–13.00 cM	Field test (IL, US)	F5:7 (94)	6.4% DX	–	Anderson et al. (2015)
		SDS disease index 21–1	Gm14_7195140_G_A—Gm14_27937142_C_T	(7,302,532–32,105,943 a2)	Greenhouse test/ isolate *LL0009* (NE305)	F2:3 (200)	34.4% DX	MN1606SP	Luckew et al. (2017)
		–	ss715617333_C_T	(9,890,873 a2)	Growth chamber/ isolates *Mont-1, Scott F2I1 1a* and *Clinton 1B*	PI lines (254)	8.6%	–	Swaminathan et al. (2019)
qRfv15-01	MLG E (Chr. 15)	*qDX016*	ss248604753—ss248616287	1.40–2.70 cM	Field test (IL, US)	F5:7 (94)	0.6% DX	–	Anderson et al. (2015)
		SDS disease incidence 21–2	Gm15_9733870_T_C—Gm15_15746095_G_A	(9,887,588–15,771,590 a2)		F2:3 (200)	9.7% DI	MN1606SP	Luckew et al. (2017)

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
					Greenhouse test/ isolate *LL0009* (NE305)/				
qRfv15-02	MLG E (Chr. 15)	*qDX015*	ss248604753—ss248616287	1.50–3.00 cM	Field test (IL, US)	F5:7 (94)	0.05% DX	–	Anderson et al. (2015)
		SDS disease incidence 21–2	Gm15_13651090_G_A—Gm15_47871831_A_C	(13,666,730–48,664,536 a2)	Greenhouse test/ isolate *LL0009* (NE305)/	F2:3 (200)	13.0% DX	MN1606SP	Luckew et al. (2017)
		–	ss248698930	(20,239,752 a1)	Field test (MI, US)	Cultivars (392)	7.7% DX	–	Wen et al. (2014)
qRfv16-01	MLG J (Chr. 16)	*SDS15-7*	BARC-016775–02,320—BARC-014745–01,638	27.99–38.70 cM (4,273,014–7,026,287 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	5.2% Stem cut	LS94-3207	Swaminathan et al. (2016), Chang et al. (2018)
		SDS14-12, qDX017	ss248983974—ss248977568	11.50–14.00 cM	Field test (IL, US)	F5:7 (94)	0.9% DX	–	Anderson et al. (2015), Chang et al. (2018)
		–	Satt285a—Satt132	(2,827,903–9,934,104 a2)	Field test (IL, US)	F5:14 (92)	39% DI	–	Yuan et al. (2012), Chang et al. (2018)
qRfv17-01	MLG D2 (Chr. 17)	*SDS11-2, cqRfs11, qRfs7s*	Satt574, Sat_001	(31,915,278 a1, 36,455,269 a2)	Field test (IL, US)	F5:14 (92); F2:3 (321)	10.2–25.2% IS	Flyer	Kazi et al. (2008), Luckew et al. (2013), Chang et al. (2018)
		–	BARC-059487–15,840	(34,725,321 a2)	Greenhouse test/ isolate *Somerset #1A*	Ancestral lines, advanced breeding lines, cultivars, and landraces (282)	–	–	Bao et al. (2015), Chang et al. (2018)
		–	BARC-061049–17,016	(35,707,915 a2)	Greenhouse test/ isolate *Somerset #1A*	Ancestral lines, advanced breeding lines, cultivars, and landraces (282)	–	–	Bao et al. (2015) Chang et al. (2018)
		–	Sat_001	(36,455,269 a2)	–				Lightfoot et al. (2015), Chang et al. (2018)
qRfv17-02	MLG D2 (Chr. 17)	-	BARC-051665–11,191	(14,613,850 a2)	Greenhouse test/ isolate *Somerset #1A*	Ancestral lines, advanced breeding lines,	–	–	Bao et al. (2015)

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
						cultivars, and landraces (282)			
		SDS19-2	Satt389	(13,771,699 a2)	Field tests (IA/ MI/IL, US)	F5 derived (91)	17.9% DX	Ripley	Brzostowski et al. (2018)
		qRfs7	Satt222—Satt389	–	Greenhouse test/ isolate *FSG-1*	F4 derived (96)	11%	PI 567374	de Farias Neto et al. (2007)
qRfv18-01	MLG (Chr. 18)	*SDS4-3, SDS6-1, rfs1*	Bng122D/Bng122_1	–	Field test (IL, US)	F5:13 (100)	16–18% DX, 38–73% IS [j]	Forrest	Njiti et al. (1998), Meksem et al. (1999) Chang et al. (2018)
		G-QTL-3	Satt130, Satt356, Satt570	(4,639,971/3,172,879 a2)	Field test (IL, US)	RIL (100)	11.9–19.2% DI, 8.6–13.9%	Forrest	Abdelmajid et al. (2007), Chang et al. (2018)
		G-QTL-2, Rfs2	Satt309, Satt594, Satt217, OI03-P4	(1,736,832/22,375,695/ 4,713,265 a2)	Field test (IL, US)	RIL (100); F2:3 (321)	12.5–17.7% DI, 10.1–13.3% DX	Forrest	Abdelmajid et al. (2007), Luckew et al. (2013), Chang et al. (2018)
		SDS2-1, SDS3-1, SDS QTL 1G	$OG13_{490}$	–	Field test (IL, US)	F5:11 (100)	17% DI, 10% DS	Forrest	Chang et al. (1996), Chang et al. (2018)
		SDS2-2, SDS3-2, SDS QTL 1G	$OI03_{450}$	–	Field test (IL, US)	F5:11 (100)	20% DI, 12% DS	Forrest	Chang et al. (1996), Chang et al. (2018)
		rfs1	$OI03_{450}$	–	Field test (IL, US)	F5 derived (199)	4–38% DX, 39–47% IS	Forrest	Meksem et al. (1999), Chang et al. (2018)
		SDS7-1	Satt214	–	Field test (IL, US)	F5:13 (100)	24.1% DI	Forrest	Iqbal. et al. (2001), Chang et al. (2018)
		SDS4-1, SDS4-3	$OI03_{512}$	–	Field test (IL, US)	F:13 (100)	21–47% DX, IS	Forrest'	Njiti et al. (1998), Chang et al. (2018)
		SDS7-3	Satt570	(3,172,879 a2)	Field test (IL, US)	F5:13 (100)	19.2% DI	Forrest	Iqbal. et al. (2001), Chang et al. (2018)
		SDS7-2, Rfs2	Satt309	(1,736,692 a2)	Field test (IL, US)	F5:13 (100); F2:3 (321)	16.3% DI	Forrest	Iqbal. et al. (2001), Luckew et al. (2013) Chang et al. (2018)
		SDS11-3, cqRfs1	Satt038_2	(1,344,090 a2)		F5:14 (92)	28.1% IS	Hartwig	

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
					Field test (IL, US)				Kazi et al. (2008), Chang et al. (2018)
		SDS11-4, cqRfs13	Satt130	(4,639,971 a2)	Field test (IL, US)	F5:14 (92)	12.9% DX	Hartwig	Kazi et al. (2008), Chang et al. (2018)
		SDS6-2, Rfs2	Satt309	(1,736,692 a2)	Field test (IL, US)	F5:13 (100); F2:3 (321)	14–63%, 8%-9% IS	Forrest	Meksem et al. (1999), Luckew et al. (2013)
		-	Satt309	(1,736,692 a2)	Field test (IL, US)	F5:14 (92)	18% DI, 12% DS, 14% DX	Forrest	Yuan et al. (2012), Chang et al. (2018)
		SDS8-1, qRfs3	Satt163	–	Field test (IL, US)	F6 derived (90); F2:3 (321)	16.0% DI	Pyramid	Njiti et al. (2002), Luckew et al. (2013), Chang et al. (2018)
		Rfs2	Satt309	(1,736,692 a2)	Field test (IL, US)	F6 derived (90); F2:3 (321)	8.5% DI	Pyramid	Njiti et al. (2002), Luckew et al. (2013) Chang et al. (2018)
		SDS5-1, Rfs1	Satt038	(1,344,090 a2)	Field test (IL, US)	F5 derived (94)	0.5% R6, 28% R8 IS	Hartwig	Prabhu et al. (1999), Chang et al. (2018)
		Rfs2	GmRLK18-1 (Glyma18g02680)	(1,711,924—1,714,468 a1)	Greenhouse test/ strain *Mont-1*	-	-	-	Srour et al. (2012), Chang et al. (2018)
		qRfs2	Satt309, TMD, SIUC-Sat1	(1,736,692 a2)	Field test (IL, US)	F5 derived (100); F2:3 (321)	-	Forrest	Triwitayakorn et al. (2005), Luckew et al. (2013), Chang et al. (2018)
		–	ss249511029	(1,611,921 a1)	Field test (MI, US)	Advanced breeding lines (300)	9.3% DX	-	Wen et al. (2014), Chang et al. (2018)
		–	Gm18-1,709,751	(1,709,751 a1)	Field test (MI, US)	Cultivars (392)	10.6% DX	–	Wen et al. (2014)
		–	ss249517154	(2,113,196 a1)	Field test (MI, US)	Advanced breeding lines (300)	8.3% DI	–	Wen et al. (2014)
		G-QTL-1	Satt214, Satt275	1,239,847 (a2)	Field test (IL, US)	RIL (100)	11.4–24.2% DI, 23.0% DS,	Forrest	Abdelmajid et al. (2007), Chang et al. (2018)

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
							10.0–25.5% DX		
		dx2	Satt214—Satt275	1,239,847 (a2)	Field test (IL, US)	RIL (94)	6.92% DX	Essex	Kassem et al. (2006)
		di2	Satt214—Satt275	1,239,847 (a2)	Field test (IL, US)	RIL (94)	7.45% DI	Essex	Kassem et al. (2006)
		ds5	ACC230—Satt214	103.7–110.5 cM	Field test (IL, US)	RIL (94)	17.30% DS	Essex	Kassem et al. (2006)
		–	Satt038	(1,344,090 a2)	Field test (IL, US)	F6 derived (90)	12.3% DI	Pyramid	Njiti et al. (2002)
qRfv18-02	MLG G (Chr. 18)	*SDS2-4, SDS3-3, SDS QTL 2G*	$OE02_{1000}$	–	Field test (IL, US)	F5:11 (100)	10% DI, 12% DS	Forrest	Chang et al. (1996), Chang et al. (2018)
		SDS7-4	$OEO2_{1000}$	–	Field test (IL, US)	F5:13 (100)	12.6% DI	Forrest	Iqbal. et al. (2001), Chang et al. (2018)
		SDS13-9, SDS13-12, qFDS004-02, qRRS001-03	ss4969823—ss107924619	24.4–28.1 cM (1,710,074–61,041,603 a1)	Greenhouse test/ isolate *Mont1*	F6:13 (50)	8.8% FDS, 2.3–33.3% RRS	PI 438489B	Abdelmajid et al. (2012), Chang et al. (2018)
		SDS2-3, SDS3-4, SDS QTL 2G	$OE04_{450}$	–	Field test (IL, US)	F5:11 (100)	16% DI, 20% DS	Forrest	Chang et al. (1996), Chang et al. (2018)
		G-QTL-4	Satt010, Satt324, $OEO2_{1000}$	(5,927,346 a2)	Field test (IL, US)	RIL (100)	11.5–24.3% DI, 23.3% DS, 10.4–28.4% DX	Forrest	Abdelmajid et al. (2007), Chang et al. (2018)
		-	Satt324—Satt594	(5,927,346–22,375,830 a2)	Field test (IL, US)	F5:14 (92)	35% DI	Forrest	Yuan et al. (2012)
qRfv18-03	MLG G (Chr. 18)	*SDS14-2*	BARC-024251–04,812	(59,472,567 a1)	Greenhouse test/ isolate *Somerset #1A*	Ancestral lines, advanced breeding lines, cultivars, and landraces (282)	–	–	Bao et al. (2015)
		qSDS18-1	ss249931277—ss249984976	(58.29–61.89 Mb a2)	Field test (MI, US)	F4 derived (153)	7.9–15.8%	U01-390489	Tan et al. (2019)
		qPY18-1	ss249931277—ss249984976	(58.29–61.89 Mb a2)	Field test (MI, US)	F4 derived (153)	26.71% plot yield	E07080	Tan et al. (2019)
		qFvC18-1	ss249931277—ss249953873	(58.29–59.83 Mb a2)	Field test (MI, US)	F4 derived (153)	8.42% pathogen	U01-390489	Tan et al. (2019)

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
							content in root		
		qRDW18-1	ss249942583—ss249953873	(59.07–59.82 Mb a2)	Field test (MI, US)	F4 derived (153)	17.9–20.9% root dry weight	E07080	Tan et al. (2019)
qRfv19-01	MLG L (Chr. 19)	*SDS9-1*	Sat_099	(43,727,029 a2)	Greenhouse test/ Strain *ST-90*	F7:14 (284)	7% DS	Minsoy'	Njiti and Lightfoot (2006)
		SDS18-2, SDS-2	BARC-047496–12,943—BARC-029419–06,181; Satt678–Satt664	(43,023,466–46,730,376 a2)	Growth chamber/ isolates *Clinton 1B* and *Scott F2II 1a*	F7:8 (200)	16%	LS94-3207	Swaminathan et al. (2018)
		SDS13-3, qFDS002-03	ss107913933—ss107929955	42.0–49.9 cM (41,343,324—47,114,567 a1)	Greenhouse test/ isolate *Mont1*	F6:13 (50)	6–17.7% FDS	PI 438489B	Abdelmajid et al. (2012)
		SDS14-13, qDX018	ss250232030—ss250233870	0.0–0.70 cM	Field test (IL, US)	F5:7 (94)	0.01% DX	-	Anderson et al. (2015)
		-	Satt166—Satt448	(42,119,600–42,616,473 a2)	Field test (IL, US)	F5 derived (91)	14%	Ripley	de Farias Neto et al. (2007)
qRfv20-01	MLG I (Chr. 20)	*qRfs5*	Satt354, Satt270	(35,362,576 a2)	Greenhouse test/ isolates *Clinton1b* and *Scott*	F2:3 (321)	8.2–11.5% DI, 12.8% DS, 12.4% DX	Essex	Luckew et al. (2013)
		SDS7-6, qRfs5	Satt354	–	Field test (IL, US)	F5:13 (100); F2:3 (321)	11.5% DI	Essex	Iqbal. et al. (2001), Luckew et al. (2013)
		SDS15-9	BARC-020245–04,514—BARC-038869–07,364	50.11–63.33 cM (35,312,272—37,595,955 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	6.3% Root feeding	LS94-3207	Swaminathan et al. (2016)
qRfv20-02	MLG I (Chr. 20)	*SDS16-4*	BARC-052017–11,314—BARC-057793–14,926	22.84–35.34 cM (2,103,067 -28,472,273 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	15% Root feeding	LS98-0582	Swaminathan et al. (2016)
		qSDS20-1	ss250304625—ss250327854	(1.06–3.76 Mb a1)	Field test (MI, US)	F4 derived (153)	9.8%	E07080	Tan et al. (2019)
		SDS disease index 21–3	Gm20_2954372_G_T—Gm20_30048849_G_T	(2,947,656—31,195,048 a2)	Greenhouse test/ isolate *LL0009* (NE305)	F2:3 (200)	20.0% DX	Spencer	Luckew et al. (2017)
		SDS18-1, SDS-1	BARC-054889–12,193—BARC-041129–07,912; Satt700–Satt496	(12,117,394—33,590,931 a2)	Growth chamber/ isolates *Clinton*	F7:8 (200)	11%	A95-684043	Swaminathan et al. (2018)

Table 8 (continued)

Locus name[a]	MLG (Chr.)	Other names	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source	References
					1B and *Scott F2II 1a*				
		SDS17-1, SDS-4	BARC-057793–14,926, Satt127—Sat_268	(12,169,135–35,176,184 a1)	Growth chamber/ isolates *Clinton 1B* and *Scott F2II 1a*	F7:8 (200)	7.6%	LS98-0582	Swaminathan et al. (2018)
qRfv20-3	MLG I (Chr. 20)	-	Sat_299	(43,634,534 a2)	Greenhouse test/ isolate *FSG-1*	F4 derived (96)	11%	PI 567374	de Farias Neto et al. (2007)
		SDS15-5	BARC-038869–07,364—BARC-059937–16,229	63.33–113.76 cM (37,595,955—45,134,969 a2)	Growth chamber/ isolates *Clinton 1B, Scott F2II 1a* and *Scott B2*	F7 derived RIL (200)	5.9% Stem cut	LS94-3207	Swaminathan et al. (2016)

[a]Locus name given in this study, if the physical positions of QTLs overlap each other in at least two independent studies. For example, *qRfv02-01* means the 1st (01) validated quantitative (q) resistance (R) to *Fusarium virguliforme (fv)* on Chr. 2 (02)

[b]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[c]Phenotypic variations explained by the molecular markers

[d]FDS: foliar disease severity

[e]DI: disease incidence

[f]DAI: days after inoculation

[g]RRS: root rot severity

[h]DX: disease index

[i]DS: disease severity

[j]IS: infection severity (root)

Table 9 Soybean loci conferring resistance to *Fusarium graminearum*

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source/ allele	References
MLG D1b (Chr. 2)	–	–	rs33907639	(33,907,639 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	14%	Allele C	Zhang et al. (2019)
	–	*qFG-4*	Satt600–Satt611	(28,903,021–29,355,267 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	7.7%	Conrad	Zhang et al. (2020)
MLG C1 (Chr. 4)	–	–	rs52044814	(52,044,814 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	14%	Allele T	Zhang et al. (2019)
	–	–	rs658576	(658,576 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	6%	Allele T	Zhang et al. (2019)
MLG A1 (Chr. 5)	–	–	rs29240006	(29,240,006 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	14%	Allele C	Zhang et al. (2019)
	–	–	rs5676224	(5,676,224 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	14%	Allele A	Zhang et al. (2019)
MLG C2 (Chr. 6)	–	–	ss715593740–ss715593784	(17,401,316–18,230,296) a2)	Rolled towel assay/isolate *Fay11*	F7:10 (184)	8.1%	PI 567301B	Acharya et al. (2015)
	–	*qRfg_Gm06*	BARC-042161–08,193	(19,857,954–20,280,838 a2)	Rolled towel assay/isolate *Fay11*	F6:7 (241)	40.2%	PI 567516C	Cheng et al. (2017a)
	–	–	rs9479021	(9,479,021 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	15%	Allele G	Zhang et al. (2019)
	–	*qFG-1*	Satt134–Satt365	111.68–112.83 cM*	Rolled towel assay/isolate *L09*	F2:14 (140)	5.4%	Hefeng25	Zhang et al. (2020)
MLG M (Chr. 7)	–	–	rs42503759	(42,503,759 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	13%	Allele C	Zhang et al. (2019)
MLG A2 (Chr. 8)	*qRfg08-01*	–	BARC_051847_11270	0.0–10.8 cM (35,856,368 a1)	Rolled towel assay/isolate *Fay11*	F6:8 (262)	9.2%	Conrad	Ellis et al. (2013a)
		qFG-3	Satt233 - Satt538	(17,232,172–47,395,378 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	10.9%	Hefeng25	Zhang et al. (2020)

Table 9 (continued)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source/ allele	References
	qRfg08-02	–	Sat_157–ss715602786	(8,353,754—8,657,875 a2)	Rolled towel assay/isolate *Fay11*	F7:10 (184)	38.5%	PI 567301B	Acharya et al. (2015), Million et al. (2019)
		qFG-2	Sat_215 - Sat_406	(3,993,698—9,204,446 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	24.5%	Hefeng25	Zhang et al. (2020)
MLG O (Chr. 10)	–	–	rs13411695	(13,411,695 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	12%	Allele G	Zhang et al. (2019)
MLG F (Chr. 13)	–	–	FLOWER_COLOR W1/w1 locus	21.0–23.8 cM	Rolled towel assay/isolate *Fay11*	F6:8 (262)	5.1%	Conrad	Ellis et al. (2013a)
	qRfg13-01	–	BARC_2.0_Gm13_16926707	(16,926,707 a2)	Rolled towel assay/isolate *Fay11*	F9:11 (316)	3.1%	Conrad	Stasko et al (2016)
		qFG-5	Satt554–Sat_387	(11,101,819–39,252,658 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	11.4%	Conrad	Zhang et al. (2020)
MLG B2 (Chr. 14)	–	–	BARC_2.0_Gm14_2523881	(2,523,881 a2)	Rolled towel assay/isolate *Fay11*	F9:11 (316)	4.8%	Sloan	Stasko et al (2016)
MLG E (Chr. 15)	–	–	BARC_025663_049888	0.0–19.0 cM	Rolled towel assay/isolate *Fay11*	F6:8 (262)	7.2%	Conrad	Ellis et al. (2013a)
MLG J (Chr. 16)	–	–	Satt693	12.2–21.9 cM (6,325,509 a2)	Rolled towel assay/isolate *Fay11*	F6:8 (262)	5.2%	Conrad	Ellis et al. (2013a)
	–	*qFG-6*	Satt380–Satt183	(25,456,677–26,823,650 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	5.0%	Conrad	Zhang et al. (2020)
MLG D2 (Chr. 17)	–	–	rs21473423	(21,473,423 a2)	Rolled towel assay /isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	13%	Allele G	Zhang et al. (2019)
	–	–	rs7363671	(7,363,671 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	15%	Allele G	Zhang et al. (2019)
MLG L (Chr. 19)	*qRfg19-01*	–	BARCSOYSSR_19_1452	31.7–40.3 cM (47,528,116 a2)	Rolled towel assay/isolate *Fay11*	F6:8 (262)	3.6%	Sloan	Ellis et al. (2013a)

Table 9 (continued)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor source/ allele	References
		QTL 19–2	BARC_2.0_Gm19_47784141	(47,784,141 a2)	Rolled towel assay/isolate *Fay11*	F9:11 (316)	8.6%	Sloan	Stasko et al (2016)
	qRfg19-02	–	rs38240023	(38,240,023 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	13%	Allele G	Zhang et al. (2019)
		qFG-7	Sct_010–Satt652	(9,175,726–41,380,304 a2)	Rolled towel assay/isolate *L09*	F2:14 (140)	8.7%	Conrad	Zhang et al. (2020)
	–	–	rs42918129	(42,918,129 a2)	Rolled towel assay/isolate *L09*	Landraces and elite cultivars from the Chinese National Soybean GeneBank(314)	13%	Allele C	Zhang et al. (2019)

[a]Locus name given in this study, if the physical positions of QTLs overlap each other in at least two independent studies. For example, *Rfg08-02* means the 2nd (02) validated quantitative (*q*) resistance (R) to *Fusarium graminearum (fg)* on Chr. 8

[b]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[c]Phenotypic variations explained by the molecular markers

Table 10 Soybean loci conferring resistance to stem canker (caused by *Diaporthe aspalathi* and *D. caulivora*) and Phomopsis seed decay (caused by *D. longicolla*)

Causal agent	Locus name	MLG (Chr.)	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
Diaporthe aspalathi	*Rdc1* (*Rdm1*)	–	–	–	Field test	F3 (40)	–	Tracy-M	Kilen and Hartwig (1987)
	Rdc2 (*Rdm2*)	–	–	–	Field test	F3 (40)	–	Tracy-M	Kilen and Hartwig (1987)
	Rdc3 (*Rdm3*)	–	–	–	Greenhouse test	F2 (200–600)	–	Crockett and Dowling	Bowers et al. (1993)
	Rdc4 (*Rdm4*)	MLG A2 (Chr. 8)	–	–	Greenhouse test	F2 (200–600)	–	Hutcheson and Dowling	Bowers et al. (1993), Tyler (1996)
	Rdm5	MLG A2 (Chr. 8)	–	12.4 cM	Greenhouse test	F2:3 (105)	–	Hutcheson	Chiesa et al. (2009)
	Rdm_{MJ19RR}	MLG C2 (Chr. 6)	Satt433	13.3 cM (47,516,523)	Greenhouse test	F2 (147)	–	MJ19RR	Gilli et al. (2020)
	GBSRdm370	MLG B2 (Chr. 14)	–	(1,744,370)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm556	MLG B2 (Chr. 14)	–	(1,725,556)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm287	MLG B2 (Chr. 14)	–	(1,710,287)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm224	MLG B2 (Chr. 14)	–	(1,986,224)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm562	MLG B2 (Chr. 14)	–	(1,740,562)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm793	MLG B2 (Chr. 14)	–	(1,768,793)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm339	MLG B2 (Chr. 14)	–	(1,921,339)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm374	MLG B2 (Chr. 14)	–	(1,921,374)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm219	MLG B2 (Chr. 14)	–	(1,795,219)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm204	MLG B2 (Chr. 14)	–	(1,751,204)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm516	MLG B2 (Chr. 14)	–	(1,612,516)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm964	MLG B2 (Chr. 14)	–	(1,850,964)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm114	MLG B2 (Chr. 14)	–	(1,851,114)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)

Table 10 continued

Causal agent	Locus name	MLG (Chr.)	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
	GBSRdm450	MLG B2 (Chr. 14)	–	(1,612,450)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm397	MLG B2 (Chr. 14)	–	(1,612,397)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm518	MLG B2 (Chr. 14)	–	(1,744,518)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm120	MLG B2 (Chr. 14)	–	(1,741,120)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm712	MLG B2 (Chr. 14)	–	(1,581,712)	Greenhouse test	Accessions	–	–	Dos Santos et al. (2019)
	GBSRdm875	MLG B2 (Chr. 14)	–	(1,581,875)	Greenhouse teste	Accessions	–	–	Dos Santos et al. (2019)
Diaporthe longicolla	*PSD 6–1*	MLG C2 (Chr. 6)	Satt100—Satt460	110.8 cM (31,490,622–44,049,891)	Greenhouse test	F8 (124)	46.3%	Taekwangkong	Sun et al. (2013)
	PSD-10–2	MLG O (Chr. 10)	Sat_038—Satt243	85.8 cM (46,052,103–46,657,863)	Greenhouse test	F8 (124)	14.1%	SS2-2	Sun et al. (2013)
	–	MLG F (Chr. 13)	Sat_317 and Sat_120	5.9–12.7 cM (32,196,800)	Field test	F2 (140)	–	MO/PSD-0259	Roy and Abney (1988), Ploper et al. (1992), Minor et al. (1993), Zimmerman and Minor (1993), Jackson et al. (2004)
	–	MLG B2 (Chr. 14)	Sat_177 and Sat_342	4.3–15.8 cM (971,657–2,956,930)	Field test	F2 (140)	–	PI 80837	Roy and Abney (1988), Ploper et al. (1992), Minor et al. (1993), Zimmerman and Minor (1993), Jackson (2004)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax2.0*, only starting position is shown for SSR markers

[b]Phenotypic variations explained bsy the molecular markers

Stem canker/Phomopsis seed decay

The *Diaporthe/Phomopsis* complex, the genus *Diaporthe* Nitschke (asexual morph *Phomopsis*) (Sacc.) comprises several species of fungi causing important diseases in soybean: northern and southern stem canker, *Diaporthe* seed decay, and pod and stem blight (Santos et al. 2011). This complex is dispersed worldwide resulting in greater yield losses in soybean than any other single fungal pathogen (Sinclair 1993). Phomopsis seed decay (PSD) is mainly caused by *Phomopsis longicolla* (*D. longicolla*), while soybean stem canker (SSC) is primarily caused by two different species, *D. aspalathi* (E. Jansen, Castl. & Crous) (syn. *Diaporthe phaseolorum* var. *meridionalis*) and *D. caulivora* (Athow & Caldwell) J.M. Santos, Vrandecic & A.J.L. Phillips (syn. *Diaporthe phaseolorum* var. *caulivora*) (Fernández et al. 1999; Pioli et al. 2003; Santos et al. 2011; Udayanga et al. 2015) and *D. sojae* is the cause of pod and stem blight (Udayanga et al. 2015). Recently, *D. gulyae, D. bacilloides,* and *D. ueckerae* have also been associated with soybean diseases (Mathew et al. 2018; Petrović et al. 2021).

Northern stem canker (caused by *D. caulivora*) was first observed in the late 1940s in the northern USA (Athow and Caldwell 1954) and resulted in severe yield losses in the mid-1950s. Hildebrand (1956) developed a greenhouse assay for stem canker which involved growing the fungus on sterilized wooden toothpicks and inserting the toothpicks into the soybean stems. Susceptible cultivars develop a canker and die, while resistant cultivars do not develop a canker symptom. Hildebrand noted that seedlings of 'Hawkeye' and 'Blackhawk' appeared resistant when inoculated, became susceptible at mid-stage, and then grew increasingly resistant as the plants matured. In the late 1990s, northern stem canker emerged as an important disease in the northern USA and Ontario, Canada (Wrather et al. 2003a). Thickett et al. (2007) developed a cut stem assay by placing inoculum on the cut surface of seedling stems which were severed above the unifoliate leaves. After two weeks, the length of the lesions was longer on the susceptible cultivars, and results agreed with field observations. To date, little has been done to elucidate the genetic resistance to *D. caulivora*.

Southern stem canker (caused by *D. aspalathi*) was first reported in the 1970s causing an estimated loss of $37 million in 1983 (Backman et al. 1985; Weaver et al. 1988). Initially identified as *D. phaseolorum* var. *caulivora*, southern isolates were noticeably different from northern isolates in culture (McGee and Biddle 1987). The name of the fungus was changed to *D. phaseolorum* var. *meridionalis* and is now *D. aspalathi* (Rensburg et al. 2006; Santos et al. 2011). Southern stem canker begins as a canker on the lower stem during mid-reproductive development (Weaver et al. 1988; Rupe 2016). The canker grows on one side of the stem but does not girdle the stem producing a toxin that results in distinctive foliar symptoms before prematurely killing the plant. Consistent cultivar reactions to southern stem canker were observed in the field, but the occurrence of the disease varied from year to year. Keeling (1985) reported that cultivar responses to inoculating 10-day-old seedlings with infested toothpicks were in good agreement with field ratings. The toothpick inoculation method was later used on 60-day-old field plants and compared to inoculating the plant with ascospores. Both methods consistently produced stem canker symptoms and were able to identify cultivar responses from very susceptible to very resistant (Keeling 1988). Single dominant resistance genes to southern stem canker were reported from the cultivar 'Tracy-M', *Rdc1* and *Rdc2* (later renamed *Rdm1* and *Rdm2*, respectively) (Kilen and Hartwig 1987), in 'Crockett', *Rdc3* (later renamed *Rdm3*), and in 'Dowling', *Rdc4* (later renamed *Rdm4*) (Bowers et al. 1993) (Table 10). *Rdc4* was also found in the cultivar 'Hutcheson' (Tyler 1996). Initially, all these genes appeared to be equally effective against all isolates of *D. aspalathi* (Keeling, 1988), but a report from Argentina isolates of *D. aspalathi* were found virulent on one or more of each of these genes (Pioli et al. 2003). Interestingly, they found a number of isolates of *D. aspalathi* that were virulent on lines with *Rdc1* and lines with *Rdc2* but were avirulent on Tracy-M which has both *Rdc1* and *Rdc2*. Moderate levels of resistance to southern stem canker have been reported from the field and greenhouse inoculations, but the genetic nature of that resistance has not been explored.

Phomopsis seed decay

Phomopsis seed decay (PSD) of soybean is the major cause of poor seed quality and significant yield loss in most soybean-growing regions (Sinclair, 1993). PSD is favored by hot and humid environmental conditions and is usually worse with early maturing cultivars planted early in the season. Severe symptoms are shriveled, elongated, or cracked, chalky appearance, but seed infection is usually symptomless. These symptomless infections can result in pre- and post-emergence damping-off (Sinclair 1993; Kulik and Sinclair 1999; Koenning 2010). Resistance to PSD has been reported in PI 82,264 (Walters and Caviness 1973), PI 181,550 (Athow 1987), the cultivar 'Delmar' (Crittenden and Cole 1967; Brown et al. 1987), PI 200,501, and 'Arksoy' (Ross 1986), and in PI 80,837, PI 417,479, and PI 360,841 (Brown et al. 1987) (Table 10). PI 417,479 was reported to have two dominant genes for resistance to PSD,

Table 11 Validated soybean loci associated with quantitative resistance to Sclerotinia stem rot (caused by *Sclerotinia sclerotiorum*)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/ flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor	References
MLG D1a (Chr. 1)	*qRss01-01*	–	–	(5,594,597 a2)	Cotton pad method/strain *NB-5*	Canada breeding lines (127)	32%	Allele T	Boudhrioua et al. (2020)
		–	–	(5,594,597 a2)	Cotton pad method/strain *NB-5*	F6:8 (47)	–	Maple Donovan	Boudhrioua et al. (2020)
	qRss01-02	–	S1_36,783,951, S1_36,497,505, S1_35,474,053, S1_35,045,463, S1_36,006,734, S1_36,045,483, S1_35,152,187	(35,045,463—36,783,951 a1)	Greenhouse test/ isolate *Jatai*	Brazil breeding lines (275)	2.3–4.0%	–	Wei et al. (2017)
		Qswm1-1	BARCSOYSSR01_0884, BARCSOYSSR01_1102	89.21–91.19 cM (35,860,562–43,694,885 a2)	Greenhouse test	F5:10 (128)	7.9%	–	Zhao et al. (2015)
MLG C1 (Chr. 4)	*qRss04-01*	*Locus3*	ss715587925 and ss715588278	(42,372,944–46,104,694 a2)	Field test (MI, US), greenhouse test/ isolate *105HT*	Improved lines (962)	5.5–5.6%	–	Wen et al. (2018)
		–	Gm04:43,486,259	(43,486,259 a2)	Cut stem method	China accessions (185)	8%	Allele T	Jing et al. (2021)
MLG C2 (Chr. 6)	*qRss06-01*	*Sclero 7–1*	Sat_238 and Satt708	(40,461,941—43,990,660 a2)	Field inoculation (QC, Canada)/ strain *NB-5*	F4 derived RILs (180)	18.9–23.6%	Maple Donovan	Huynh et al. (2010)
		–	Gm06:43,719,111	(43,719,111 a2)	Cut stem method	China accessions (185)	8–9%	Allele A	Jing et al. (2021)
MLG A2 (Chr. 8)	*qRss08-01*	–	Sat_129, Satt329 (Patent)	(14,660,743–21,117,799 a2)	Greenhouse	–	12.0%	P^S	Han et al. (2007)
		Sclero 10–2	Sat_199	70.95 cM (15,071,890 a2)	Field inoculation (IA and WI, US) and greenhouse test	PI (66), breeding lines (35), F4:6 (392)	15.8%	–	Kandel et al. (2018)
		Qsp-3	Satt525 and Satt233	(17,010,941–17,232,172 a2)	Greenhouse test	F5:6 (149)	8.0%	MapleArrow	Li et al. (2010e)
		QTL3, Sclero 2–3	Satt233	(17,232,172 a2)	Detached leaf method/isolate *143*	F5 (100)	4–10%	Corsoy79	Arahana et al. (2001)
		Sclero 9–1	Satt233-Satt327	(17,232,172–20,468,620 a2)	Field inoculation (MI, US) and		10.4%	IA2053	Guo et al. (2008)

Table 11 (continued)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/ flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor	References
					greenhouse inoculation/ *isolate HT105*	F2:3 (94), F2:4 (94), F2:5 (94)			
		–	ss715599948	(17,490,619 a2)	Field inoculation (IA, US)	USDA germplasm collection (474)	–	–	Moellers et al. (2017)
		Qswm8-1	BARCSOYSSR08_1160, BARCSOYSSR08_1127	55.89–59.74 cM (20,468,640–21,633,276 a2)	Greenhouse test	F5:10 (128)	11.3%	–	Zhao et al. (2015)
MLG K (Chr. 9)	*qRss09-01*	*QTL19, Sclero 2–16; Sclero 3–11; Sclero 4–7; Sclero 5–10*	Satt273	(38,799,271 a2)	Detached leaf method/isolate *143*	F5 (400)	4–10%	Corsoy79, Dassel, S19-90, Williams82	Arahana et al. (2001)
		Sclero 8–3	Satt273	(38,799,271 a2)	Greenhouse test/ isolate *105HT*	F4:5 (155)	5.5%	PI 194639	Vuong et al. (2008)
MLG O (Chr. 10)	*qRss10-01*	*QTL26, Sclero 2–22; Sclero 3–17; Sclero 5–14; Sclero 6–11*	Satt478	(39,108,281 a2)	Detached leaf method/isolate *143*	F5 (400)	4–10%	Corsoy79, Dassel, S19-90, Vinton81	Arahana et al. (2001)
		Sclero 10–3	Satt478	66.01 cM (39,108,281a2)	Field inoculation (IA and WI, US) and greenhouse test	PI (66), breeding lines (35), F4:6 (392)	2.5%	–	Kandel et al. (2018)
	qRss10-02	*Sclero 10–5*	Satt243	107.30 cM (46,657,863 a2)	Field inoculation (IA and WI, US) and greenhouse test	PI (66), breeding lines (35), F4:6 (392)	2.0%	–	Kandel et al. (2018)
		–	ss715607699	(47,626,066 a2)	Greenhouse test	USDA germplasm collection (474)	–	–	Moellers et al. (2017)
		QTL28, Sclero 2–24; Sclero 3–19; Sclero 4–11; Sclero 5–16; Sclero 6–13	Satt243, Sat_108, and Sat_109	(46,657,863–48,199,089 a2)	Detached leaf method/isolate *143*	F5 (500)	4–10%	Corsoy79, Dassel, S19-90, Vinton81, Williams82	Arahana et al. (2001)
MLG H (Chr. 12)	*qRss12-01*	*qLLS12-1*	Block2877-Block2897	1.90 cM (35,766,547–38,843,925 a2)	Greenhouse test	F5:20 (149)	5.2%	Maple Arrow	Zou et al. (2021)
		–	Gm12:36,426,007	(36,426,007 a2)			7.0–8.8%	Allele C	

Table 11 (continued)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/ flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor	References
					Detached leaf method	China landraces (38), elite cultivars (147)			Sun et al. (2020)
MLG F (Chr. 13)	*qRss13-01*	*Qswm13-1*	BARCSOYSSR13_0114-BARCSOYSSR13_0197	124.11–127.86 cM (2,195,422–3,999,858 a1)	Greenhouse test	F5:10 (128)	23.3–23.6%	–	Zhao et al. (2015)
		–	rs3296245	(3,296,245 a1)	Greenhouse test	China core germplasm collection (330)	9.5–22.6%	Allele A	Zhao et al. (2015)
		–	rs3296576	(3,296,576 a1)	Greenhouse test	China core germplasm collection (330)	9.5–23.6%	Allele C	Zhao et al. (2015)
		–	rs3446126	(3,446,126 a1)	Greenhouse test	China core germplasm collection (330)	7.0–17.2%	Allele G	Zhao et al. (2015)
	qRss13-02	*qLLS13-1*	Block2994-Block2997	174.00 cM (17,233,137–17,548,123 a2)	Greenhouse test	F5:20 (149)	5.3%	Maple Arrow	Zou et al. (2021)
		qDRS13-1	Block2996-Block2997	174.00 cM (17,417,745–17,548,123 a2)	Greenhouse test	F5:20 (149)	21.1%	Maple Arrow	Zou et al. (2021)
		–	–	(17,472,342 a2)	Greenhouse test	CNSGB germplasm (261)	–	Allele G	Zou et al. (2021)
MLG E (Chr. 15)	*qRss15-01*	–	–	(13,339,206–13,929,317 a1)	Greenhouse test/ strain *NB-5*	Breeding lines (130)	14.5%	–	Bastien et al. (2014)
		–	–	(13,339,206–13,929,317 a1)	Greenhouse test/ strain *NB-5*	F4:6 (48)	–	PR918827	Bastien et al. (2014)
		–	–	(13,665,369 a2)	Cotton pad method/strain *NB-5*	Canada breeding lines (127)	15%	Allele A	Boudhrioua et al. (2020)
MLG D2	*qRss17-01*	*QTL10, Sclero 3–6*	Satt154	(9,576,644 a2)	Detached leaf method/isolate *143*	F5 (200)	4–10%	Williams82	Arahana et al. (2001)

Table 11 (continued)

MLG (Chr.)	Locus name[a]	Other name	Tightly linked/ flanking markers	Marker position cM (bp)[b]	Testing methods/ Resistance spectrum	Population type (size)	PVE[c]	Donor	References
(Chr. 17)		*Sclero 10–8*	Satt154	46.76 cM (9,576,644 a2)	Field inoculation (IA and WI, US) and greenhouse test	PI (66), breeding lines (35), F4:6 (392)	4.0%	–	Kandel et al. (2018)
MLG L (Chr. 19)	*qRss19-01*	–	Satt523, SLS2C.F20 (Patent)	(7,127,430 a2)	Greenhouse test	–	16.0%	–	Han et al. (2007)
		Sclero 10–10	Satt523	25.56 cM (7,127,430 a2)	Field inoculation (IA and WI, US) and greenhouse test	PI (66), breeding lines (35), F4:6 (392)	8.5%	–	Kandel et al. (2018)

[a]Locus name given in this study, if the physical positions of QTLs overlap each other in at least two independent studies. For example, *qRss10-02* means the 2nd (02) validated quantitative (q) resistance (R) to *Sclerotinia sclerotiorum* (*ss*) on Chr. 10

[b]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[c]Phenotypic variations explained by the molecular markers

one located on linkage group F and one on linkage group H (Zimmerman and Minor 1993). The PSD resistant line, 'MO/PSD-0259' was developed from PI 417,479 (Elmore et al. 1998; Minor et al. 1993). MO/PSD-0259 was used to develop two PSD resistant lines, 'SS 93–6012' and 'SS 93–6181' (Wrather et al. 2003b). The resistance in PI 80,837 was determined to be conferred by a single dominant gene that is different from the one in MO/PSD-0259 (Jackson et al. 2005). A genetic study using a greenhouse inoculation method with progenies derived from a cross between the resistant cultivar 'Taekwangkong' and the susceptible cultivar 'SS2-2' reported two QTLs associated with PSD resistance which were tightly linked with genes for maturity (Sun et al. 2013). Many PIs in maturity groups III, IV, and V were identified as resistant to PSD across three states (Li et al. 2010a). Resistance to PSD was identified in six commercial cultivars in inoculated and non-inoculated tests (Li et al. 2017a). In a study evaluating the response of PIs to purple seed stain (PSS), nine PIs with resistance to PSS were also resistant to PSD (Li et al. 2019). PI 80,837 also has resistance to both PSS and PSD (Jackson et al. 2005, 2006). A cut stem seedling assay similar to that described for inoculations with *D. caulivora* by Thickett et al. (2007) was used with *D. longicolla* (Li 2018). This method gave similar results as field tests. A draft genome sequence for *D. longicolla* has been published (Li et al. 2015a, 2017a), and the glycoside hydrolase subnetwork appears to be important in pathogeneses (Li et al. 2018).

Numerous management practices can be applied to control PSD, including deep tillage, crop rotation with non-legume crops, treating seeds with fungicides, and applying fungicides during pod-fill. To date, the most effective management option is the use of resistant cultivars (Park 1991; Roy et al. 1994; Jackson et al. 2005; Pathan et al. 2009; Mengistu et al. 2010). A report by Sun et al. (2013) identified two QTLs for PSD resistance associated with days to maturity in soybean (Table 10). This was an important discovery because early maturing soybean genotypes are often highly susceptible to PSD due to the weather conditions during pod and seed development. Several screening methods have been used to identify sources of resistance, including those mentioned above for stem canker, seed plate assay (Li et al. 2011), and cut-stem inoculation method.

Sclerotinia stem rot

Sclerotinia stem rot (or white mold), caused by *Sclerotinia sclerotiorum* (Lib.), can cause significant yield losses in soybean and overall reduction of seed quality in North Central USA and northeastern China under conducive cool

Table 12 Soybean loci conferring resistance to soybean rust (caused by *Phakopsora pachyrhizi*)

MLG (Chr.)	Locus/ allele name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
MLG N (Chr. 03)	*Rpp5*	Sat_275 and Sat_280	40.81–43.45 cM (29,862,641–32,670,432 a2)	Growth chamber/isolate *BRSMS Bacuri*	F2:3 (173)	R gene	PI 200456	Garcia et al. (2008
		Sat_275 and Sat_280	40.81–43.45 cM (29,862,641–32,670,432 a2)	Growth chamber/isolate *BRSMS Bacuri*	F2:3 (177)	R gene	PI 200526	Garcia et al. 2008
		Sat_275 and Sat_280	40.81–43.45 cM (29,862,641–32,670,432 a2)	Growth chamber/isolate *BRSMS Bacuri*	F2:3 (174)	R gene	PI 471904	Garcia et al. (2008)
MLG C2 (Chr. 06)	*Rpp3*	Sat_263 and Sat_238	118.67–117.45 cM (44,738,585–43,990,660 a2)	Growth chamber/isolate *Japanese T1-2*	F2 (86)	70%	PI 416764	Hossain et al. (2015)
		Satt460 and Sat_263	117.76–118.67 cM (44,049,891–44,738,585 a2)	Isolates *AL04-1 (USA), AU79-1 (Australia), BZ01-1 (Brazil), HW94-1 (USA), IN73-1 (India), LA04-1 (USA), PG01-2 (Paraguay), SA01-1 (South Africa), TW72-1 (Taiwan) AND TW80-2 (Taiwan)*	F2:3 (110)	R gene	PI 462312	Hyten et al. (2009)
		Satt079 and Satt307	117.87–121.26 cM (44,503,658 – 46,820,673 a2)	Local isolate (Brzil)	F2:4 (116)	R gene	FT-2 (Brazil)	Brogin (2005)
		Satt 460 and Staga001	117,76–119.84 cM (44,049,891–45,427,175 a2)	Field test/local field isolates	F2:3 (91 and 68)	R gene	PI 567099A	Ray et al. (2011)
		Satt460 and Satt307	117.76–121.26 cM (44,049,891–46,820,673 a2)	Field and Greenhouse test/local field isolate	F6:7 RILs (117)	15–14%	Hyuuga	Monteros et al. (2007)
	–	BARC-023517–05,442 and BARC-040475–07,751	(21,986,774–28,804,685 a2)	Field inoculation (Ha Noi, Vietnam)	F6:7 (250)	11.7%	DT2000 (PI 635999)	Vuong et al. (2016)
	–	BARC-040475–07,751 and BARC-051071–10,973	(21,986,774–22,185,687 a2)	Field tests (FL, US)	F6:7 (250)	8.6%	DT2000 (PI 635999)	Vuong et al. (2016)
	–	Sat_312 and BARC-203517–05,442	(27,940,542–36,131,665 a2)	Field tests (FL, US)	F6:7 (250)	8.4%	DT2000' (PI 635999)	Vuong et al. (2016)
MLG A2		Satt409 and Satt429	145.57–162.02 cM	Greenhouse test/local isolate (Georgia, USA)	F6 (240)	10%	Benning (PI 595645)	Harris et al. (2015)

Table 12 (continued)

MLG (Chr.)	Locus/ allele name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
(Chr. 08)	*QTL Asian Soybean Rust 2–1*		(45,106,638—47,217,842 a2)					
MLG K (Chr. 09)	*QTL Asian Soybean Rust 2–2*	Satt326 and Sat_363	49,52–50,58 cM (29,967,163–36,143,707 a2)	Greenhouse test/local isolate (Georgia, USA)	F6 (240)	5%	PI 416937	Harris et al. (2015)
MLG F (Chr. 13)	*QTL Asian Soybean Rust 2–3*	Satt490 and Satt554	97,97–111,88 cM (36,699,189–39,252,658 a2)	Greenhouse test/local isolate (Georgia, USA)	F6 (240)	9%	Benning	Harris et al. (2015)
MLG E (Chr. 15)	*QTL Asian Soybean Rust 2–4*	Sat_124 and Satt369	15,86–56,27 cM (11,099,721 a1–49,011,265 a2)	Greenhouse test/local isolate (Georgia, USA)	F6 (240)	17%	PI 416937	Harris et al. (2015)
MLG J (Chr. 16)	*Rpp2*	Satt215 and Sat_361	44.08–44.49 cM (28,944,536–30,478,500 a2)	Growth chamber/isolate *BRSMS Bacuri*	F2:3 (174)	R gene	PI 224270	Garcia et al. (2008)
		Satt620 and Sat_366	52.84 cM (29,205,413–30.404.629 a2)	Growth chamber/isolate *E1-4–12*	F2 (143)	70%	Iyodaizu	Yamanaka et al. (2015a)
MLG G (Chr. 18)	*Rpp1*	Sct_187 and Sat_064	107.11–108.69 cM (60,463,057 a1—56,333,703 a2)	Greenhouse test/isolate *India 73–1*	BC6 F2:3 (126)	R gene	PI 200492	Hyten et al. (2007)
		–	–	Field tests (USA)	Germplasm (576)	R gene	PI 547875 (L85-2378)	Walker et al. (2011)
		Satt191 and Sat_117	96.57–100 cM (58,722,811–58,879,539 a1)	Greenhouse test/local isolates (Brazil)	F2 (160)	R gene	PI 594760B	Garcia et al. (2011)
		Satt191 and Sat_064	96.57–108.69 cM (54,450,956–56,333,703 a2)	Growth chamber/isolate *Japanese T1-2*	F2 (90)	50%	Xiao Jing Huang	Yamanaka et al. (2015a)
		Sct_187 and Sat_064	107.11–108.69 cM (60,463,057 a1–56,333,703 a2)	Growth chamber/isolate *E1-4–12*	F2 (120)	65%	PI 594177 (Himeshirazu)	Yamanaka et al. (2015a)
		Sat_064 and SSR66	108.69 cM (56,333,703 a2)	Growth chamber/isolate *Japanese T1-2*	F2 (117)	60%	PI 587905	Hossain et al. (2015)
		Sat_064	108.69 cM (56,333,703 a2)	Growth chamber/isolate *Japanese T1-2*	F2 (82)	56%	PI 594767A	Hossain et al. (2015)

Table 12 (continued)

MLG (Chr.)	Locus/ allele name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
	Rpp1? Rpp1-b?	Satt191 and Sat_064	96.57–108.69 cM (54,450,956–56,333,703 a2)	Field test/isolates *TW72-1 (Taiwan), ZM01-1 (Zimbabwe), IN73-1 India), HW94-1 (Hawai, USA), HW98-1 (Hawai, USA), AU79-1 (Australia), LA04-1 (Lousiana, USA), AL04-3 (Alabama, USA)*	F2:3 (186)	R gene	PI 587886	Ray et al. (2009)
		Satt191 and Sat_372	96.57–107.75 cM (54,450,956 a2)	*Field test/isolates TW72-1 (Taiwan), ZM01-1 (Zimbabwe), IN73-1 India), HW94-1 (Hawai, USA), HW98-1 (Hawai, USA), AU79-1 (Australia), LA04-1 (Lousiana, USA), AL04-3 (Alabama, USA)*	F2:3 (164)	R gene	PI 587880A	Ray et al. (2009)
	Rpp1-b?	Sat_064 and AF162283	108.69–87.94 cM (56,333,703 a2–57,436,765 a1)	Growth chamber/isolate *E1-4–12*	F2 (106)	65–67%	PI 587855	Yamanaka et al. (2016)
	rpp1	Sat_117 and Sct_187	100–107.11 cM (58,879,539—60,463,057 a1)	Greenhouse test/local isolates (Brazil)	F2 (105)	R gene	PI 594760B	Garcia et al. (2011)
	Rpp1-b	Sat_064 Between: BARC-010495–00,656 BARC-014379–01,337	108.69 cM (56,333,703 a2)	Greenhouse test/isolate *ZM01-1*	F3:4 (98)	70%	PI 594538A	Chakraborty et al. (2009)
	Rpp4	Satt288 and Satt191	76.76–96.57 cM (51,127,425–54,450,956 a2)	Growth chamber/isolate *BRSMS Bacuri*	F2:3 (175)	R gene	PI 459025	Garcia et al. (2008)
		Satt288 and AF162283	76.76–87.94 cM (51,127,425 a2–57,436,765 a1)	Field collection	F2:3 (80)		PI 459025	Silva et al. (2008)
	QTL Asian Soybean Rust 1–1	SSR50 and SSR1859	(60,518,978–60,613,084 a1)	Greenhouse test/isolate *ZM01-1*	F2:3 (100)	70%	PI 561356	Kim et al. (2012)
	Rpp6	Satt324	33.25 cM (5,927,346 a2)	Growth chamber/isolates *MS06-1, LA04-1*	F2:3 (104)	R gene	PI 567102B	Li et al. (2012)
		GSM0374 and GSM0427	(5,998,461 to 6,160,481 a1)	Greenhouse test/isolate *GA12*	F5:6 (184)	R gene	PI 567068A	King et al. (2016)
	–	BARC-016867–02,359 and BARC-048761–10,703	(51,814,496–52,157,617a2)	Field inoculation (Ha Noi, Vietnam)	F6:7 (250)	12.5%	DT2000 (PI 635999)	Vuong et al. (2016)
		Satt288_BARC-024489–04,936	(51,127,425–55,000,817a2	Field inoculation (Ha Noi, Vietnam)	F6:7 (250)	9.6%	DT2000' (PI 635999)	Vuong et al. (2016)

Table 12 (continued)

MLG (Chr.)	Locus/ allele name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
MLG L (Chr. 19)	*Rpp7*	W82 x PI between the markers GSM0546 and GSM0463;	(39,462,291 to 39,616,643 a1)	*AU79-1 (Australia), CO04-2 (Armenia, Columbia), GA12-1 (Georgia, USA), HW98-1 (Hawai), IN73-1 (India), LA04-1(Lousiana, USA), TW72-1 (Taiwan), VT05-1 (Vietnam), ZM01-1 (Zimbabwe)*	F2:3 (90/ 100)	R gene	PI 605823	Childs et al. (2018b)
		5601 T x PI between markers GSM0461 and GSM0468	(39,462,291 to 39,616,643 a1)	*AU79-1 (Australia), CO04-2 (Armenia, Columbia), GA12-1 (Georgia, USA), HW98-1 (Hawai), IN73-1 (India), LA04-1(Lousiana, USA), TW72-1 (Taiwan), VT05-1 (Vietnam), ZM01-1 (Zimbabwe)*	F4:5 (114)	R gene	PI 605823	Childs et al. (2018b)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[b]Phenotypic variations explained by the molecular markers

and wet weather conditions (Hoffman et al. 1998; Kurle et al. 2001; Peltier et al. 2012; Sun et al. 2020). For example, in 2004 and 2009, Sclerotinia stem rot caused yield losses of 1.63 and 1.61 million Mt, respectively, in the USA alone (Peltier et al. 2012). More recently, over 1.08 million Mt of production losses were recorded in 2014 in the North Central USA and Ontario, Canada (Allen et al. 2017). The disease steadily ranked among the top 10 most destructive diseases associated with yield losses in the northern USA and Ontario, Canada (Allen et al. 2017).

Horizontal resistance is the only type of soybean resistance identified for Sclerotinia stem rot. The first report for horizontal resistance identified three minor QTLs (explaining 6.5–9.6% of phenotypic variations) on linkage groups M, K, and C2 using a bi-parental population of 152 F3 derived RILs (Kim and Diers 2000). More recently, the assembly of the soybean reference genome and advancements in GWA have enabled more accurate dissection of genomic regions associated with resistance to Sclerotinia stem rot (Schmutz et al. 2010). For example, Bastien et al. (2014) identified four significant markers for resistance, which were located at chromosomes 1, 15, 19, and 20, explaining 6.3–14.5% of phenotypic variations. The locus on Chr. 15 (renamed *qRss15-01* in this review) was further validated in an F4:5 RIL population where significantly shorter lesions were observed for 24 resistant genotypes. In another GWA study, a major locus was identified and validated on Chr. 13 (*Qswm13-1*, and renamed *qRss13-01* in this review), which explained 23.33% of phenotypic variations (Zhao et al. 2015). From 2014 to 2021, a total of nine GWA studies have been published (Bastien et al. 2014; Iquira et al. 2015; Zhao et al. 2015; Wei et al. 2017; Wen et al. 2018; Boudhrioua et al. 2020; Sun et al. 2020; Jing et al. 2021; Zou et al. 2021). Combining the studies of QTL mapping and GWA, 14 loci have been validated from at least two mapping studies (Table 11). The 14 loci were distributed at 11 chromosomes (1, 4, 6, 8, 9, 10, 12, 13, 15, 17, and 19) and contributed as high as 32% of the phenotypic variations. These validated loci may be of high priority for soybean breeders to use for improving partial resistance to Sclerotinia stem rot. In addition to the validated QTLs, more than 200 QTLs have also been identified and may be validated in the future (Supplementary Table 3),

Soybean rust

Asian soybean rust (ASR) caused by *Phakopsora pachyrhizi* (Sydow. & Sydow.) is one of the most destructive diseases in soybean. When environmental conditions are conducive for disease development, ASR spreads fast, causing severe crop damage, leading to significant seed

quality reduction and yield losses of as much as 80% (Yorinori et al. 2005). Losses vary upon weather conditions, genotype, and the maturity stage at the time of infection (Wang and Hartman 1992) and are mainly attributed to premature leaf fall, reduced green leaf area in the canopy, reduced dry matter accumulation and reduced harvest index (Kumudini et al. 2008). Soybean rust can also be caused by *P. meibomiae*, which resembles *P. pachyrhizi* in both symptoms and spore appearance. Yet the rust caused by *P. meibomiae* occurs mainly in South and Central America and causes little damage on soybean. This review will be focused on ASR.

ASR is primarily diagnosed with a magnifying glass or microscope, but the polymerase chain reaction (PCR) reaction is also useful when sporulating pustules are not visible (Frederick et al. 2002). The key feature of ASR is the appearance of uredinia and urediniospores. Therefore, it is recommended that infected leaf samples be incubated in a humid chamber and left overnight to enhance rust development and sporulation for accurate diagnosis.

Many management strategies have been proposed to control ASR, including cultural practices, nutrition management, biological and fungicide applications, and host genetic resistance (Tadesse 2019). The application of fungicides is the preferred management tool used by farmers in regions where ASR is prevalent, but it increases production costs and environmental footprint. Since host plant resistance appears as an affordable method for managing ASR, considerable efforts have been directed toward screening soybean germplasm for resistance to *P. pachyrhizi* and the development of resistant cultivars.

Resistance to ASR

Screening for reaction to ASR can be carried out in the field, in locations where the presence of inoculum and environmental conditions are appropriate for disease development, or in the greenhouse with controlled inoculations and incubation at high relative humidity (Childs et al. 2018a). In the latter case, it is necessary to collect and maintain the *P. pachyrhizi* isolates to be used in the inoculations. Spores can be stored in sub-zero freezers, but, as an obligate parasite, inoculum must be produced on living soybean seedlings.

Resistance to ASR in soybean plants is evaluated based on the presence or absence of lesions, color of the lesions, number of uredinia per lesion, and level of sporulation (Bromfield 1984). More recent studies have evaluated resistance using quantitative traits (Bonde et al. 2006; Walker et al. 2011; 2014). During a compatible interaction in a susceptible soybean plant, abundant sporulation and tan lesions occur, whereas in incompatible interactions (resistance), lesions are reddish-brown (RB) with less sporulation. Immune reactions (IM) have also been observed without visible lesions (Bromfield, 1984).

However, it has been pointed out that the number of uredinia per lesion and the level of sporulation are not necessarily correlated with the color of the lesion (Yamanaka et al. 2015a). Yamanaka et al. (2010) analyzed five traits including lesion color, the number of uredinia per lesion, frequency of lesions that had uredinia, frequency of open uredinia, and level of sporulation, and observed high correlations between all the traits except the color of the lesion. In this sense, Yamanaka et al. (2016) selected the number of uredinia per lesion, the frequency of lesions that had uredinia, and the level of sporulation to assess the degree of resistance.

Resistance or susceptibility studies focus on understanding the defensive response. To date, eight major resistance genes (*Rpp1-7*, *Rpp1-b*) have been mapped (Table 12) (Childs et al. 2018b; Hossain 2019). But these *Rpp* gene-mediated resistances against ASR have been overcome in nature several times. For example, the soybean resistance provided by *Rpp1* and *Rpp3* was defeated by the *P. pachyrhizi* MT isolate only two years after ASR was first detected in Brazil (Pierozzi et al. 2008).

The improvement effort to know the physical location of the *Rpp* genes (resistance to *P. pachyrhizi*) is a great challenge today. However, despite the publication of the soybean genome (Schmutz et al. 2010), no *Rpp* gene has yet been cloned. For this reason, other authors have tried to identify the candidate genes linked to the *Rpp3* gene through a massive transcriptomic approach, using NILs populations. These genes are mostly related to phenylpropanoid branch isoflavonoid pathway-specific phytoalexin, glyceollin biosynthesis (Hossain 2019).

The presence of multiple virulence genes in the pathogen population and the lack of multiple resistance genes in the host give the soybean rust pathogen a competitive advantage. Therefore, the deployment of specific single genes for resistance is unlikely to be a successful strategy (Jarvie 2009).

Although varieties with pathotype-specific resistance genes were released, the stability of this resistance is uncertain since the large number of races of this fungus already described demonstrates the great variability of the pathogen. Understanding the molecular mechanisms involved in defense responses is of primary importance to plan strategies to control stress and, consequently, to increase the adaptation of plants to limiting conditions. Molecular markers have been considered tools for a large number of applications ranging from the location of a gene to the improvement of plant varieties through MAS. Also, the analysis of the soybean genome has generated a large amount of information and several databases with

molecular markers are being generated that could be used for genetic improvement (Vuong et al. 2016; Tadesse 2019).

Strategies for ASR resistance

The introgression of vertical resistance through classical breeding followed by MAS allows the development of resistant varieties and their use as an efficient and cost-effective method to control soybean rust (Tadesse 2019). An example to highlight is the pyramiding of several *Rpps* genes in a single line. Yamanaka et al. (2015a, b) managed to develop highly resistant experimental lines with stacks of three genes: *Rpp2* + *Rpp3* + *Rpp4* and *Rpp2* + *Rpp4* + *Rpp5*.

Pathotype-specific resistance genes and molecular markers are known to facilitate selections. However, the resistance provided by major genes tend to be broken rapidly; thus, research should be focused on the role of quantitative minor genes (QTLs) which are more likely to provide durable resistance to this highly variable pathogen.

To date, only one attempt to enhance resistance ASR based on transgenic technology has been recorded (Soto et al. 2020). In this study, constitutive expression of the *NmDef02* gene from *Nicotiana magalosiphon* demonstrated significantly increased resistance in soybean against *Phakopsora pachyrhizi* in field experiments.

The most recent and novel attempt to control this disease is the treatment of liquid suspension of cellulose nanofibers (CNF) to plants before inoculation with the pathogen. The authors suggest that this application changes the hydrophobicity of the leaf surface, suppressing *P. pachyrhizi* CHSs (chitin synthases) expression related to chitin formation, which are associated with reduced formation of pre-infection structures (Saito et al. 2021).

Frogeye leaf spot, Cercospora leaf blight and purple seed stain

There are three soybean diseases caused by *Cercospora* spp.: frogeye leaf spot (FLS), Cercospora leaf blight (CLB), and purple seed stain (PSS). FLS, caused by *C. sojina* Hara, is an important foliar disease in soybean in the USA, Brazil, and China (Laviolette et al. 1970; Bernaux 1979; Dashiell and Akem 1991; Akem and Dashiell 1994; Ma 1994; Mian et al. 1998). Symptoms start on leaves as small, light brown circular spots which develop into a darkish brown to reddish margin (Dashiell 1991). In addition to foliar symptoms, *C. sojina* can cause lesions on pods and infect soybean seeds. FLS is favored by warm temperatures and frequent rainfalls (Phillips 1999) and remains active throughout the growing season (Laviolette et al. 1970; Kim et al. 2013), which make FLS a major disease in the southern USA as well as in some regions of the Midwestern USA (Yang et al. 2001; Mengistu et al. 2002; Mian et al. 2008). Yield losses can range from 10 to 60% mainly due to the reduction in photosynthesis and leaf area by necrotic lesions and/or premature defoliation (Laviolette et al. 1970; Bernaux 1979; Dashiell and Akem 1991; Akem and Dashiell 1994; Ma 1994; Mian et al. 1998). Screening methods for FLS include field evaluations with natural inoculum or with inoculations, and greenhouse inoculations of seedlings (Mian et al. 2008; Mengistu et al. 2012). Mian et al. (2008) proposed a set of 12 differential cultivars to determine races of *C. sojina*. With these differentials, they described 11 races from a collection of 93 *C. sojina* isolates collected in the USA. Three resistance genes (*Rcs*, Resistant to *C. sojina*) have been identified including *Rcs1*, *Rcs2,* and *Rcs3* (Table 13) (Athow and Probst, 1952; Athow et al. 1962; Phillips and Boerma 1982). *Rcs3* appears to confer resistance to all known races of *C. sojina* in the USA. *Rcs3* was further fine mapped on Chr. 16 (MLG J) (Mian et al. 1999; Missaoui et al. 2007a, b). In recent years, *Rcs*(PI 594,891) and *Rcs*(PI 594,774) were fine mapped and approved by the Soybean Genetic Committee as QTL that confers resistance to FLS (Hoskin 2011; Pham et al. 2015); In addition, two major QTLs were mapped on chromosomes 6 and 8, respectively, conferring resistance to *C. sojina* race 2 (ATCC 44,531) (Sharma and Lightfoot 2014); *Rcs15-02* was mapped on Chr. 6 (MLG C2); the ss715594329—ss715594474 interval was mapped on chromosome 6 (MLG C2) (Smith 2021); the ss715610717—ss715610843 interval was mapped on chromosome 11 (MLG B1)(Smith 2021); the ss715614578—ss715615158 interval was mapped on chromosome 13 (MLG F) (McAllister et al. 2021); and *Rcs15-01* was mapped on Chr. 19 (MLG L) (Lee 2021).

CLB and PSS are two closely related diseases caused by the same or similar pathogens. The causal agent of both CLB and PSS was identified as *Cercospora kikuchii* (Matsumoto & Tomoyasu) M. W. Gardner (Matsumoto and Tomoyasu 1925; Walters 1980); however, recent studies have found *C. flagellaris* and *C. sigsbeckiae* were the primary species associated with both diseases in the southern USA. CLB begins as a purpling of the upper leaves starting during seed development. This purpling can cover the entire leaf surface. Symptoms can advance to blighting where the entire leaf becomes chlorotic and necrotic with the leaflets falling off leaving the petioles attached. The pathogen produces a toxin, 'cercosporin', whose production requires light exposure. As a result, CLB symptoms begin at the upper end top of the plant and progress to the lower leaves. In severe cases, the whole plant may be defoliated. Yield losses for PSS have been estimated at 0.12–0.28 million Mt (Allen et al. 2017) whereas CLB

Table 13 Soybean loci conferring resistance to frogeye leaf spot (caused by *Cercospora sojina*) and Cercospora leaf blight/purple seed stain

Disease name	Causal agent	MLG (Chr.)	Locus name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
Frogeye leaf spot	*Cercospora sojina*	–	*Rcs1*	–	–	race *1*	F2	R gene	Lincoln	Athow and Probst (1952), Pham et al. (2015)
		–	*Rcs2*	–	–	race *2*	–	R gene	Kent	Athow et al. (1962), Pham et al. (2015)
		–	–	–	–	Greenhouse test	F2	R gene	Ransom, Lee, and Stonewall	Pace et al (1993)
		MLG A1 (Chr. 5)	–	Satt276	5,158,623	Greenhouse test/race *2*	F5:14 (94)	13%	Forrest	Sharma and Lightfoot (2014)
		MLG C2 (Chr. 6)	–	ss715594329–ss715594474	39,188,086—43,688,393	–	–	–	–	Smith (2021)
			-	Satt319—Satt079	38,049,354—44,503,658	Greenhouse test/race *2*	F5:14 (94)	52%	Essex	Sharma and Lightfoot (2014)
			Rcs15-02	–	–	–	–	–	–	Smith (2021)
		MLG M (Chr. 7)	–	Satt323	10,465,123	Greenhouse test/race *2*	F5:14 (94)	4%	Essex	Sharma and Lightfoot (2014)
			–	B35H07	–	Greenhouse test/race *2*	F5:14 (94)	5%	Essex	Sharma and Lightfoot (2014)
		MLG A2 (Chr. 8)	–	Satt589	5,182,879	Greenhouse test/race *2*	F5:14 (94)	11%	Forrest	Sharma and Lightfoot (2014)
			–	Satt632—A2D8	8,223,512	Greenhouse test/race *2*	F5:14 (94)	15%	Essex	Sharma and Lightfoot (2014)
		MLG K (Chr. 9)	–	Satt555	8,020,345	Greenhouse test/race *2*	F5:14 (94)	10%	Forrest	Sharma and Lightfoot (2014)
			–	Sat_116	–	Greenhouse test/race *2*	F5:14 (94)	11%	Forrest	Sharma and Lightfoot (2014)
		MLG O (Chr. 10)	–	Satt259	–	Greenhouse test/race *2*	F5:14 (94)	4%	Essex	Sharma and Lightfoot (2014)
		MLG B1 (Chr. 11)	–	ss715610717—ss715610843	4,338,907—5,248,257	–	–	–	–	Smith (2021)
			–	Satt444	29,759,281	Greenhouse test/race *2*	F5:14 (94)	6–12%	Forrest	Sharma and Lightfoot (2014)
		MLG H	–	Satt293	36,036,485	Greenhouse test/race *2*	F5:14 (94)	6%	Essex	Sharma and Lightfoot (2014)

Table 13 (continued)

Disease name	Causal agent	MLG (Chr.)	Locus name	Tightly linked/ flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
		(Chr. 12)								
		MLG F (Chr. 13)	*Rcs(PI 594,774)*	Satt663–Satt114	25,936,631–28,912,864	Greenhouse test	F2:3 (195)	R gene	PI 594774	Hoskins (2011)
			–	ss715614578–ss715615158	28,207,736–31,449,060	–	–	–	–	Smith (2021)
			Rcs(PI 594,891)	Satt114–Sct_033	28,912,864	Greenhouse test	F2:3 (110)	R gene	PI 594891	Hoskins (2011)
			–	CFR2	–	Greenhouse test/race *2*	F5:14 (94)	9%	Forrest	Sharma and Lightfoot (2014)
		MLG J (Chr. 16)	–	Satt249	1,149,373	Greenhouse test/race *2*	F5:14 (94)	8%	Essex	Sharma and Lightfoot (2014)
			Rcs3	Satt244—Satt547; AZ573TA150 and AZ573CA393	33,818,897–34,035,180	Greenhouse test/all known races	F2:3 (123)	R gene	Davis	Phillips and Boerma (1982), Boerma and Phillips, (1983), Mian et al. (1999), Missaoui et al. (2007a, b), Pham et al. (2015)
		MLG G (Chr. 18)	–	CGG-SCAR	–	Greenhouse test/race *2*	F5:14 (94)	6%	Forrest	Sharma and Lightfoot (2014)
		MLG L (Chr. 19)	–	Satt446	1,678,377	Greenhouse test/race *2*	F5:14 (94)	4–5%	Forrest	Sharma and Lightfoot (2014)
			Rcs15-01	–	–	–	–	–	–	Lee (2021)
		MLG I (Chr. 20)	–	Satt440	46,787,225	Greenhouse test/race *2*	F5:14 (94)	15%	Essex	Sharma and Lightfoot (2014)
Cercospora leaf blight/ Purple seed stain	*Cercospora kikuchii*	MLG G (Chr. 18)	*Rpss1*	Sat_308 and Satt594	6.6 and 11.6 cM (11,426,775–22,375,695)	Field test	F2 (148)	R gene	PI 80837	Jackson et al. (2008)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax2.0*

[b]Phenotypic variations explained by the molecular markers

causes an estimated yield loss of 23% in the USA (Wrather et al. 1997). On seed, infection causes a purpling of the seed coat. Seed infection is usually not associated with yield loss but can reduce seed germination and may lead to infected seedlings. Although both CLB and PSS are favored by high moisture and warm temperatures during early pod development (Jones 1968; Schuh 1990), the occurrence of these diseases appears to be independent of each other (Orth and Schuh, 1994; Walters 1985).

Based on natural field inoculum, Srisombun and Supapornhemin (1993) reported resistance to PSS in the soybean cultivar 'SJ2' and that this resistance may be due to a single dominant gene. Resistance to PSS was also reported in PI 80,837, PI 417,274, PI 417,460, and the cultivar 'Gnome' (Wilcox et al. 1975; Ploper et al. 1992). The resistance in PI 80,837 was attributed to a single gene on linkage group G, *Rpss1* (Jackson et al. 2006, 2008) (Table 13). Additional PIs were identified as resistant sources to both CLB and PSS (Alloatti et al. 2015) or only to PSS (Li et al. 2019). Several studies of population genetics have found differences in genetic structure among populations and pathogenicity of groups throughout the Americas (Almeida et al. 2005; Cai et al.2009; Lurá et al. 2011). It is unknown if the reactions of these soybean lines to CLB and PSS will remain consistent with the new species of *Cercospora* associated with these diseases.

Charcoal rot

The worldwide distributed charcoal rot disease of soybean is caused by *Macrophomina phaseolina* (Tassi) Goid (Smith and Wyllie 1999). *M. phaseolina* is a soilborne plant pathogen causing disease infection in more than 500 plant species (Su et al. 2001; Mengistu et al. 2007). Charcoal rot is one of the primary diseases of soybean in the USA and Canada (Bandara et al. 2020; Roth et al. 2020) resulting estimated yield losses between 0.73 and 2.0 million Mt from 2010 to 2014 (Allen et al. 2017). Disease severity is favored by the increase in soil and air temperature (28–35 °C) (Mengistu et al. 2014), and symptoms include stunted growth, leaf chlorosis, premature yellowing and early maturation, or incomplete pod filling (Gupta et al. 2012; Mengistu et al. 2016). Management strategies include crop rotation with non-host crops, such as cotton, wheat, and barley that can lower inoculum load in the soil, and avoidance of water stress especially during the reproductive stage of soybeans. (Almeida et al. 2003; García-Olivares et al. 2012; Vibha 2016). Biological control with Trichoderma isolates has been proposed by researchers as a possible alternative to control charcoal rot (Khalili et al. 2016; Orojnia et al. 2021). However, host plant resistance is the most viable method to control the disease (Mengistu et al. 2011; Coser et al. 2017). Little is known regarding the genetics and heritability of the pathogen and there is a lack of reliable and efficient screening method for this disease (Mengistu et al. 2008). Until 2018, no soybean genotype having a high level of resistance to *M. phaseolina* had been identified (Mengistu et al. 2018). Recently, a report by Nataraj et al. (2019), summarized eleven soybean genotypes identified as moderately resistant to charcoal rot along with pedigree information. Reznikov et al. (2019) found that cv. 'Munasqa RR' carried superior resistance to *M. phaseolina.* In addition, the University of Missouri-Fisher Delta Research Center has released varieties showing superior resistance to charcoal rot (Chen et al. 2020, 2021b). Based on field research studies conducted over the last several years, over 2,000 soybean genotypes have been screened for CR resistance, and of these genotypes, approximately 25 have been identified as having moderate resistance against charcoal rot (Mengistu et al. 2007, 2011, 2013). Recently, Mengistu et al. (2021) screened a set of 120 soybean accessions known to have resistance to one or more races of SCN. Twelve of these accessions have been identified to have moderate charcoal rot resistance combined with resistance to SCN. These accessions are archived and will be available through the Germplasm Resources Information Network (GRIN) system of the USDA. Even though moderately resistant cultivars have been identified, the lack of identifying a complete resistance has delayed the progress to better understanding the genetics of resistance. Most of those genotypes were screened using at least one of the six screening methods for the disease assessment including: colony-forming unit index (CFUI); root stem severity (RSS); percent height of stem discoloration (PHSD); foliar symptoms (FS); cut-stem inoculation method; and seed plate assay (SPA) (Mengistu et al. 2007; Twizeyimana et al. 2012; da Silva et al. 2019). Of all these methods, CFUI and RSS have been the stay methods for charcoal rot assessment currently used in the field.

Recently, QTL mapping and GWA studies were reported on multiple genomic regions harboring horizontal resistance to charcoal rot in soybean, which may be used to facilitate breeding and MAS against this pathogen (Table 14) (Coser et al. 2017; da Silva et al. 2019, 2020; Ghorbanipour et al. 2019). More efforts are needed to identify complete resistant sources and develop tightly linked molecular markers to facilitate breeding resistant varieties.

Table 14 Soybean loci conferring resistance to charcoal rot (caused by *Macrophomina phaseolina*)

MLG (Chr.)	Linked/flanking markers	Marker position/bp	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
MLG D1b (Chr. 2)	Sat_169	37,813,855 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	10% number of microsclerotia in stem, 13% amount of charcoal rot disease, 5% severity of charcoal rot disease	–	Ghorbanipour et al. (2019)
	Satt644	38,221,027 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	12% 100 grain weight	–	Ghorbanipour et al. (2019)
MLG C1 (Chr. 4)	ss715588228	4,307,731 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	Satt607	8,165,631 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	9% 100 grain weight	–	Ghorbanipour et al. (2019)
	Sat_404	13,613,713 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	8% pod weight	–	Ghorbanipour et al. (2019)
	Satt190	16,738,759 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	9% number of microsclerotia in stem, 7% amount of charcoal rot disease, 7% severity of charcoal rot disease	–	Ghorbanipour et al. (2019)
	Satt361	32,617,784 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	9% pod weight	–	Ghorbanipour et al. (2019)
	Sat_357	33,970,110 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	10% 100 grain weight	–	Ghorbanipour et al. (2019)
	Sat_416	40,624,709 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	11% number of microsclerotia in stem, 12% amount of charcoal rot disease	–	Ghorbanipour et al. (2019)
MLG A1 (Chr. 5)	–	25,338,390 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	–	PI 567562A	da Silva et al. (2020)
MLG C2 (Chr. 6)	ss715593307	14,918,492 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	Sat_238	43,990,660 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	8% pod weight, 13% grain weight, 8% grain yield	–	Ghorbanipour et al. (2019)
	Satt460	44,049,891 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	9% grain weight, 8% number of microsclerotia in stem, 10% severity of charcoal rot disease	–	Ghorbanipour et al. (2019)
	Satt079	44,503,658 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	11% grain weight, 8% 100 grain weight	–	Ghorbanipour et al. (2019)
	Sct_028	46,273,196 a1	Field inoculation/isolate S_8	Maturity group I-V (130)	6% grain weight	–	Ghorbanipour et al. (2019)
	Sat_252	48,211,009 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	11% pod weight, 12% 100 grain weight, 10% amount of charcoal rot disease	–	Ghorbanipour et al. (2019)

Table 14 (continued)

MLG (Chr.)	Linked/flanking markers	Marker position/bp	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
MLG A2 (Chr. 8)	–	7,511,708 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	–	PI 567562A	da Silva et al. (2020)
	ss715601990	42,490,418 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	ss715602087	43,618,993 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
MLG K (Chr. 9)	ss715604575	45,369,206 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
MLG B1 (Chr. 11)	Satt359	32,411,307 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	10% number of microsclerotia in stem, 9% amount of charcoal rot disease, 11% severity of charcoal rot disease	–	Ghorbanipour et al. (2019)
MLG H (Chr. 12)	ss715613120	492,020 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	ss715612760	37,527,844 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
MLG B2 (Chr. 14)	ss715618004	219,725 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	–	2,442,086 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	–	PI 567562A	da Silva et al. (2020)
MLG E (Chr. 15)	Gm15_01842053 and Gm15_03051337	1,842,060 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	29.4%	PI 567562A	da Silva et al. (2019)
MLG J (Chr. 16)	Gm16_28961127 and Gm16_30493887	29,328,591–30,862,012 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	25.4%	PI 567562A	da Silva et al. (2019)
	Gm16_35973543 and Gm16_37078478	36,476,386–37,570,986 a2	Cut-stem inoculation technique/isolate *Conway*	F2:3 (140)	8.84%	PI 567562A	da Silva et al. (2019)
MLG G (Chr. 18)	ss715631726	51,751,797 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	ss715631906	53,502,168 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
	ss715632099	54,829,750 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)

Table 14 (continued)

MLG (Chr.)	Linked/flanking markers	Marker position/bp	Testing methods/ Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
MLG L (Chr. 19)	Sat_124	50,728,020 a2	Field inoculation/isolate S_8	Maturity group I-V (130)	11% grain weight, 11% 100 grain weight, 11% grain yield	–	Ghorbanipour et al. (2019)
MLG I (Chr. 20)	ss715638424	43,471,723 a2	Field test and cut-stem inoculation technique/ isolate from Iowa soybean field	USDA PI lines (459)	–	–	Coser et al. (2017)
-	Satt512	–	Field inoculation/isolate S_8	Maturity group I-V (130)	8% pod weight, 10% 100 grain weight	–	Ghorbanipour et al. (2019)
	S63880-CB	–	Field inoculation/isolate S_8	Maturity group I-V (130)	11% grain weight, 7% grain yield	–	Ghorbanipour et al. (2019)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[b]Phenotypic variations explained by the molecular markers

Brown stem rot

Brown stem rot (BSR) is a devastating soybean disease caused by a soilborne fungus, *Phialophora gregata* (syn. *Cadophora gregata*), which was first discovered in central Illinois in 1944 (Allington and Chamberlain 1948; Harrington and McNew 2003). There are two different types of *P. gregata* pathogen identified (Type I and II): Type I causes pith browning and interveinal chlorosis and necrosis of leaves, but Type II only causes pith browning (Gray 1972; Harrington et al. 2003). The disease caused annual yield loss of 0.35 million Mt in the Northern USA (Allen et al. 2017; Klos et al. 2000), and yield reduction can reach as high as 38% (Bachman et al. 2001). The most effective strategy to control BSR is the introgression of resistance genes into soybean cultivars (Klos et al. 2000; McCabe and Graham 2020). From previous studies, three genes (*Rbs_1*, *Rbs_2*, and *Rbs_3*) for BSR resistance in soybean have been identified through allelism tests (Table 15) (Hanson et al. 1988; Willmot and Nickell 1989). Later, it was determined that all three genetic loci were in an overlapping region of Chr. 16 (28.9–36.2 Mb) (Lewers et al. 1999; Bachman et al. 2001). Recently, Rincker et al. (2016a) concluded that all three loci for BSR resistance were located in the same region, and that the resistance was conferred by a single gene based on their fine mapping (Rincker et al. 2016a) and GWA studies (Rincker et al. 2016b). To evaluate BSR resistance in soybean, Sebastian et al. (1983) established a greenhouse root-dip method, which has been modified and refined by further studies (Hanson et al. 1988; Willmot and Nickell 1989; Lewers et al. 1999; Bachman et al. 2001). Soybean PIs that have BSR resistance include PI 84,946–2, PI 86,150, PI 90,238, PI 95,769, PI 88,820, PI 424285A, PI 424,353, PI 424611A, PI 437,833, and PI 437,970 (Chamberlain and Bernard 1968; Tachibana and Card 1972; Hanson et al. 1988; Nelson et al. 1989; Wilmot and Nickell 1989).

Rhizoctonia damping-off and root rot

Rhizoctonia damping-off and root rot is an important disease in soybean and can cause pre- and postemergence damping-off, seed rot, root rot, hypocotyl lesions, and web blight (Dorrance et al. 2003; Rahman et al. 2020). The causal agent, *Rhizoctonia solani* Kuhn, is a soilborne necrotrophic complex species that can host corn, soybean, and other crops such as wheat and potato, suggesting that management of Rhizoctonia root rot by rotations between these crops may not be effective (Ajayi-Oyetunde and Bradley 2017, 2018). The isolates of *R. solani* can be classified into 14 anastomosis groups (AGs) and more

Table 15 Soybean loci conferring resistance to brown stem rot (caused by *Phialophora gregata*)

MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods	Population type (size)	PVE[b]	Donor source	References
MLG J	Rbs_1	Satt215	(28,944,536–28,944,665)	Greenhouse assay	F2:3 (73)	28%	L78-4094	Bachman et al. (2001)
(Chr. 16)		Satt431	(36,221,174–36,221,397)	Greenhouse assay	F2:3 (73)	74%	L78-4094	Bachman et al. (2001)
	Rbs_2	Satt244	(33,818,897–33,819,094)	Greenhouse assay	F2:3 (77)	67%	PI 437833	Bachman et al. (2001)
		Satt431	(36,221,174–36,221,397)	Greenhouse assay	F2:3 (77)	46%	PI 437833	Bachman et al. (2001)
	Rbs_3	K375	67.3–69.3 cM*	Greenhouse assay	F6:7 (320)	62%	PI 84946–2	Lewers et al. (1999)
		B122	53.8–55.8 cM*	Greenhouse assay	F6:7 (320)	45%	PI 84946–2	Lewers et al. (1999)

*GmComposite2003 genetic position (www.soybase.org)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax2.0*

[b]Phenotypic variations explained by the molecular markers

subgroups based on their genetic similarity. Different AGs may incite different symptoms of disease on soybean. For example, AG-2-2IIIB, AG-4 and AG-5 can cause seed rot, pre- and post-emergence damping-off, hypocotyl and root rot, and foliar blight on soybean, while AG-3, AG-7, and AG-11 cause very little damage (Ajayi-Oyetunde and Bradley 2018). The management of Rhizoctonia root rot may include clean seeds, tillage, fungicides, and deployment of resistant cultivars if possible. Unfortunately, currently there is no commercial resistant cultivars available to the market, and the genetic research against Rhizoctonia root rot is inadequate. Only three SSR markers, Satt281, Satt177, and Satt245 (Table 16) have been found associated with partial resistance to AG-4 isolate (Zhao et al. 2005), although more germplasm lines and soybean varieties have been identified as potential sources of resistance (Muyolo et al. 1993; Bradley et al. 2001; Sharma 2020).

Other fungal diseases

Taproot decline

Taproot decline is a disease caused by *Xylaria necrophora* sp. nov. (Garcia-Aroca et al. 2021), a recently identified pathogen that was overlooked since some of the symptoms were similar to other soybean root diseases including SDS and charcoal rot. This soilborne pathogen can affect seedlings; however, the symptoms in the field develop later in the season producing interveinal chlorosis followed by necrosis. It has been noted that *X. necrophora* will affect the root to the point that pulling plants from the ground causes the root system to break with black stroma visible on the root tissue (Allen et al. 2017). The disease is mostly managed with cultural practices, but cultivar trials are ongoing. The cv. 'Osage' (PI 648,270) has tolerance to this pathogen (Purvis 2019). Osage was developed in Arkansas and also has resistance to SDS, stem canker, and frogeye leaf spot (Chen et al. 2007).

Red leaf blotch

Red leaf blotch affects soybean plants in several Eastern, Central, and Southern African countries. The disease (also known as Pyrenochaeta leaf spot or blotch, and Dactuliophora leaf spot) can cause yield losses of up to 50% (Hartman et al. 1987, 2016). The causal agent is *Coniothyrium glycines* (R.B. Stewart) Verkley & Gruyter, a fungus previously named *Phoma glycinicola*, *Dactuliochaeta glycines*, *Dactuliophora glycines*, and *Pyrenochaeta glycines*. The disease affects foliage, petioles, pods, and stems, and may cause severe leaf blotching,

Table 16 soybean loci conferring resistance to Rhizoctonia damping off and root rot (caused by *Rhizoctonia solani*)

MLG (Chr.)	Locus name	Tightly linked / flanking markers	Marker position cM (bp)[a]	Testing methods / Resistance spectrum	Population type (size)	PVE[b]	Donor source	Reference
MLG C2 (Chr. 6)	-	Satt281	6,529,270	Greenhouse test / AG-4	F2(189), F4:5(23), F4:5(32)	11-39%	PI 442031	Zhao et al. (2005)
MLG M (Chr. 7)	-	Satt245	9,357,717	Greenhouse test / AG-4	F2(189), F4:5(23), F4:5(32)	6.8-14%	PI 442031	Zhao et al. (2005)
MLG A2 (Chr. 8)	-	Satt177	36.77cM *	Greenhouse test / AG-4	F2(189), F4:5(23), F4:5(32)	7-23%	PI 442031	Zhao et al. (2005)

[a] Marker position (bp) based on the Glycine max genome assembly version Gmax2.0

[b] Phenotypic variations explained by the molecular markers.

* GmComposite2003 genetic position (www.soybase.org)

defoliation, and premature senescence. Because of the potential negative consequences of this disease to US agriculture if introduced, *C. glycines* is listed as a select agent by the Federal Select Agent Program (Tooley 2017).

Since the 1980s, soybean germplasm has been evaluated under field conditions in African countries for reaction to red leaf blotch. Despite this extensive field testing, no sources of resistance have yet been identified among US soybean commercial cultivars, local lines, or exotic soybean lines. These evaluations were carried out in regions where red leaf blotch is endemic (Sinclair 1989). A field method to assess the infection of soybean by the pathogen was developed and used to evaluate cultivar reaction and efficacy of chemical control (Levy et al. 1990).

A seedling inoculation method has also been proposed which allows optimal infection in less space over a shorter period than field trials and without relying on the occurrence of natural inoculum and disease conducive environmental conditions. Soybean genotypes that represent nearly 90% of the genes present in US soybean were evaluated and found to be susceptible, which is consistent with previous field evaluations (Tooley 2017).

Studies are necessary to evaluate genetic variability within the pathogen population from different countries, and to assess potential interactions with soybean genotypes. With limited genomic information of the pathogens known, there are no molecular genotyping or detection methods available. Recently, the draft genome sequences of three *C. glycines* isolates were reported, enhancing the knowledge of this species (Blagden et al. 2019).

Section IV Soybean resistance to bacterial diseases

Bacterial blight

Soybean bacterial blight caused by *Pseudomonas savastanoi* pv. *Glycinea* Coerper (formerly *Pseudomonas syringae* pv. *glycinea*) is a widespread soybean disease. Although bacterial blight is not a major suppressor of soybean yield in the USA (Williams and Nyvall 1980; Hwang and Lim 1992), the interaction between soybean and the pathogen was well known as a model system to study gene-for-gene host-parasite relationships (Huynh et al. 1989). Five resistance genes/alleles have been identified named *Rpg1-b*, *Rpg1-r*, *Rpg2*, *Rpg3*, and *Rpg4*, conferring resistance to the corresponding *Psg* avirulence factors AvrB, AvrRpm1, AvrA, AvrC, and AvrD, respectively (Staskawicz et al. 1987; Keen and Buzzell. 1991; Ashfield et al. 1998; Khan et al. 2011; Whitham et al. 2016). The *Rpg1-b* and *Rpg1-r* genes were located on MLG F (Chr. 13) (Ashfield et al. 1998) and have been cloned in

Table 17 Soybean genes/loci conferring resistance to bacteria diseases

Disease Name	Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	PVE[b]	Donor source	References
Bacterial blight	*Pseudomonas syringae* pv. *glycinea*	MLG F (Chr. 13)	*Rpg1-b* (RGA-84B)	Flanked by K644 and B212. RFLP markers R45, php2265. php2385 co-segregated with *Rpg1*	67.18–69.83 cM *	AvrB	R gene	Norchief, Harosoy, PI 132207, Merit, BSR 101, Williams82,	Mukherjee et al. (1966), Staskawicz et al. (1987), Ashfield et al. (1998), Ashfield et al. (2004)
			Rpg1-r (P21f22_29)	Flanked by K644 and B212. RFLP markers R45, php2265. php2385 co-segregated with *Rpg1*	67.18–69.83 cM *	AvrRpm1	R gene	Flambeau, PI 96983	Ashfield et al. (1998), Ashfield et al. (2014)
			Rpg2	Loosely linked with *Rpg1*	–	AvrA	R gene	Merit	Keen and Buzzell (1991), Whitham et al. (2016)
		–	*Rpg3*	Linked with *Rpg4*	–	AvrC	R gene	Merit, Flambeau	Keen and Buzzell (1991), Whitham et al. (2016)
		–	*Rpg4*	Linked with *Rpg3*	–	AvrD	R gene	Flambeau	Keen and Buzzell (1991), Khan et al. (2011), Whitham et al. (2016)
Bacterial pustule	*Xanthomonas axonopodis* pv. *glycines*	MLG D1a (Chr. 1)	–	ss715580342	(53,136,582 a2)	–	–	–	Chang et al. (2016)
		MLG K (Chr. 9)	–	Satt137	(5,753,983 a1)	–	5.5%	Keunolkong	Seo et al. (2009)
		MLG O (Chr. 10)	–	Sat_108	(48,199,089 a2)	–	Single recessive gene	PI 96188	Kim et al. (2011)
		MLG B1 (Chr. 11)	–	ss715609404	(26,963,752 a2)	–	–	–	Chang et al. (2016)
		MLG B2 (Chr. 14)	–	Satt556	(38,859,467 a2)	–	7.3%	Keunolkong	Seo et al. (2009)

Table 17 Soybean genes/loci conferring resistance to bacteria diseases

Disease Name	Causal agent	MLG (Chr.)	Locus name	Tightly linked/flanking markers	Marker position cM (bp)[a]	Testing methods/ Resistance spectrum	PVE[b]	Donor source	References
		MLG D2 (Chr. 17)	*rxp*	Satt014 and Satt372; Satt486; Rxp17-700; SNUSSR17_9 and SNUSNP17_12	(6,475,946—7,542,029 a2)	–	Single recessive gene	CNS (PI 548445), Young, Coker237	Feaster (1951), Hartwig and Lehman (1951), Bernard and Weiss (1973), Hwang and Kim (1987), Palmer et al. (1992), Narvel et al. (2001), Kim et al. (2004), Kim et al. (2010), Yang et al. (2011), Chang et al. (2016)
		–	–	Satt135 and Satt397	(6,156,526—11,724,482 a1)	–	20.9%	Keunolkong	Seo et al. (2009)
		MLG I (Chr. 20)	–	Satt496	(27,664,504 a2)	–	2.7%	Keunolkong	Seo et al. (2009)

*GmComposite2003 genetic position (www.soybase.org)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01* (a1), or *Gmax2.0* (a2), only starting position is shown for SSR markers

[b]Phenotypic variations explained by the molecular markers

2004 and 2014, respectively (Ashfield et al. 2004, 2014). *Rpg2* is loosely linked with *Rpg1*, and *Rpg3* is linked with *Rpg4* at 40.5 ± 3.2 recombination units (Table 17) (Keen and Buzzel. 1991).

Bacterial pustule

Soybean bacterial pustule is a common disease in regions with warm and wet conditions (Bernard and Weiss 1973; Kennedy and Tachibana 1973; Matsuo et al. 2017). The causal agent, *Xanthomonas axonopodis* pv. *glycines*, can cause small, pale green spots with elevated pustules in the center of lesions, which can grow into large necrotic lesions causing premature defoliation (Kennedy and Tachibana 1973; Narvel et al. 2001). The first identified resistance gene is *rxp* from cv. 'CNS' and was initially mapped between Satt014 and Satt372 on MLG D2 (Chr. 17) (Feaster 1951; Hartwig and Lehman 1951; Bernard and Weiss 1973; Hwang and Kim 1987; Palmer et al. 1992; Narvel et al. 2001). Further studies narrowed the *rxp* locus down to a 33 kb genomic region between markers SNUSSR17_9 and SNUSNP17_12, with two candidate genes identified (Kim et al. 2010). In addition, another single recessive resistance gene was identified from PI 96,188. The gene was located on MLG O (Chr. 10) and was closely linked with Sat_108 (Kim et al. 2011). QTLs have also been reported against bacterial pustule (Van et al. 2004; Seo et al. 2009; Chang et al. 2016). For example, Seo et al. (2009) reported four QTLs on chromosomes 9, 14, 17 and 20, explaining 2.7–20.9% of phenotypic variations (Table 17).

Section V Soybean resistance to virus diseases

Soybean mosaic virus

Soybean mosaic virus (SMV) is a major global viral pathogen in soybean that can compromise the soybean value chain by causing expressive yield losses of up to 90% in severe outbreaks (Ren et al. 1997a; Wang et al. 2001). SMV is widely distributed in soybean-growing countries including Brazil, Canada, China, Japan, Korea, and the USA (Cho and Goodman 1979; Li et al. 2010b, 2015b). In China, the occurrence of SMV is gradually increasing throughout the country and it currently represents the most prevalent disease in soybean with annual yield losses reaching over 50% (Zhang et al. 1980, 2015b). Typical SMV symptoms include reduced seedling viability and vigor, flower abortion, reduction of pod set, seed number, and seed size (Hill et al. 1987; Ren et al. 1997b; Gunduz

et al. 2004). The severity of the symptoms is dependent on the host genotype, virus strain, plant stage at infection, as well as environmental factors (Bos 1972).

SMV is classified into strains based on its virulence and observed symptoms and differs between countries. In the USA, SMV isolates are classified into seven strains (G1–G7), where G1 is the least virulent affecting only susceptible genotypes whereas G7 is the most virulent capable of infecting both resistant and susceptible soybean genotypes (Cho and Goodman 1979). In China, SMV is classified into 21 groups (SC1–SC21) according to geographical regions and individual genotypes responses (Moon et al. 2009; Li et al. 2010b). Genetic resistance is the most efficient strategy to control SMV (Gunduz et al. 2004). To date, four independent loci for SMV resistance, *Rsv1*, *Rsv3*, *Rsv4*, and *Rsv5* have been identified (Kiihl and Hartwig 1979; Buzzell and Tu 1984; Buss et al. 1997; Li et al. 2010c; Klepadlo et al. 2017) although most of the modern commercial cultivars are susceptible to SMV, particularly to more virulent strains (Table 18) (Zheng et al. 2005a, b; Shakiba et al. 2012a).

Rsv1 is the first SMV resistance locus identified and was mapped on Chr. 13 (MLG F). It represents the most common resistance locus in soybean germplasm (Kiihl and Hartwig 1979), conferring resistance to less virulent strains (G1-G3) and susceptibility to more virulent strains (G5-G7). A total of ten unique alleles have been identified including *Rsv1, Rsv1-t, Rsv1-y, Rsv1-m, Rsv1-k, Rsv1-r, Rsv1-s, Rsv1-n*, *Rsv1-h,* and *Rsv1-c* (Kiihl and Hartwig 1979; Roane et al. 1983; Chen et al. 1991, 2001, 2002; Shakiba et al. 2013). *Rsv3* was mapped on Chr. 14 (MLG B2) and confers resistance to more virulent strains (G5-G7) while susceptible to less virulent strains (G1–G4) (Tu and Buzzell 1987). The *Rsv3* locus contains at least six alleles identified in 'OX686', 'Harosoy', 'L29', PI 61,944, PI 61,947, and PI 399,091 (Buzzell and Tu 1989; Buss et al. 1999; Gunduz et al. 2001; Shakiba et al. 2012b; Cervantes-Martinez et al. 2015). *Rsv4* was mapped on Chr. 2 (MLG D1b) and confers complete resistance to all SVM strains (Buss et al. 1997; Ma et al. 2002; Gunduz et al. 2004). A total of four alleles have been identified from 'V94-5152', PI 88,788, and 'Beeson' (Buss et al. 1997; Ma et al. 2002; Gunduz et al. 2004; Shakiba et al. 2013). Since the reaction (hypersensitive reaction) observed in *Rsv1* and *Rsv3* is different from that in *Rsv4*, it is suggested that *Rsv4* has unique molecular defense mechanisms (Ma et al. 2002; Gunduz et al. 2004; Saghai Maroof et al. 2008). Recently, Klepadlo et al. (2017) suggested that *Rsv1-y* should be named as an independent locus *Rsv5* because of segregation in resistance to SMV in progenies derived from PI 96,983 (*Rsv1*) and 'York' (*Rsv1-y*).

In addition to *Rsv1*, *Rsv3*, *Rsv4*, and *Rsv5*, several other genes named *Rsc5* (Karthikeyan et al. 2017), *Rsc7* (Yan et al. 2015), and *Rsc8* (Zhao et al. 2016) have been mapped on Chr. 2 (MLG D1b), and *Rsc3* (Yang et al. 2013), *Rsc14Q* (Ma et al. 2011) and *Rsc15ZH* (Li et al. 2020) on Chr. 13 (LG F) for resistance to Chinese SMV strains. Due to differences in SMV strain classification systems between USA and China, likely *Rsc3*, *Rsc14Q* and *Rsc15ZH* share the same locus of *Rsv1* whereas *Rsc5, Rsc7, Rsc8* share the same locus as *Rsv4* (Table 18). Although rare, the combination of the four resistance loci is naturally available in soybean genotypes and can be achieved through gene pyramiding. Combining multiple resistance genes may provide more effective and durable resistance and minimize the occurrence of resistance-breaking emerging populations.

Alfalfa mosaic virus

Alfalfa mosaic virus (AMV) is a member of the genus *Alfamovirus* in the family Bromoviridae. It has a worldwide distribution and infects more than 600 species in 22 dicotyledonous families, including agriculturally valuable crops such as alfalfa, tomato, lettuce, potato, soybean, and common bean. AMV is transmitted by more than 15 species of aphids, including the soybean aphid [*Aphis glycines* Matsumura (Hemiptera: Aphididae)], in a nonpersistent manner. It is also transmitted by mechanical inoculation and in some species, such as alfalfa and in reduced values in soybean, through the seed (Truol et al. 1985; Clark and Perry 2002; Hartman et al. 2016). Seed transmissibility was proven to be virus strain and host genotype-dependent in soybean (He et al. 2010).

AMV is known as a very complex virus which has four bacilliform particles, elongated with rounded ends. The particles are 18 nm in diameter and 30, 34, 43, and 56 nm in length. The viral genome consists of three single strands of RNA (2.0, 2.6, and 3.6 kb in length) and a fourth subgenomic RNA, known as RNA 4 encoding the coat protein (Hartman et al. 2016; Loesch-Fries 2021).

Symptoms caused by AMV in soybean range from mosaic to mottle patterns of contrasting mixes of bright yellow and dark. It is often referred to as a calico or flashy mosaic. Leaf malformation, stunting, reduced pod set, and seed coat mottling have also been mentioned. Depending upon soybean genotype, environmental conditions and strain of the virus involved, symptoms can either persist or disappear in the new tissues of infected plants (Mueller et al. 2007; Hartman et al. 2016).

Synergism between AMV and SMV has been reported. AMV symptoms are more severe and persist throughout the season in plants infected by both viruses. The observation that co-infection of AMV and SMV results in disease synergism suggests enhancement of potential that AMV

Table 18 Soybean loci conferring resistance to soybean mosaic virus (SMV)

MLG (Chr.)	Locus/allele name	Tightly linked/flanking markers	Marker position (bp)[a]	Testing methods/Resistance spectrum	PVE[b]	Population Type (size)	Donor source	References
MLG D1b (Chr. 2)	*Rsv4*	–	–	Greenhouse Screening SMV Strains G1–G7	–	F2:3 (117)	PI 88788	Gunduz et al. (2004)
		Barc-011147–00,855	8,380,603	Greenhouse Screening SMV Strains G1–G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		Barc-025955–05,182	9,689,348	Greenhouse Screening SMV Strains G1–G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		AW307114-indel	12,585,482	Greenhouse Screening SMV Strains G1–G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		Satt558	10,619,724	Greenhouse Screening SMV Strains G1–G7	–	F2 (255)	LR2	Hayes et al. (2000)
		Satt542	13,316,465	Greenhouse Screening SMV Strains G1–G7	–	F2 (255)	LR2	Hayes et al. (2000)
		BARCSOYSSR_02_0610	11,964,524	Greenhouse Screening SMV Strain SC8	–	F7:11 (184)	Kefeng No. 1	Wang et al. (2011)
		BARCSOYSSR_02_0616	12,070,465	Greenhouse Screening SMV Strain SC8	–	F7:11 (184)	Kefeng No. 1	Wang et al. (2011)
		Sat_254	11,168,955	Greenhouse Screening SMV Strain G7	–	F2 (561)	PI 486355	Hwang et al. (2006)
		ss244712184	11,613,852	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		ss244712591	11,685,678	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		ss244712651	11,693,196	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		ss244712652	11,693,604	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		ss244712653	11,693,900	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		ss244712671	11,697,977	Greenhouse Screening SMV Strains G1 and G7	–	F2 (766)	V94-5152	Klepadlo et al. (2017)
		R4at3	11,964,498	Greenhouse Screening SMV Strains G1 and G7	–	BC3F2 (309)	V94-5152	Ilut et al. (2016)
		Rat2	12,044,285	Greenhouse Screening SMV Strains G1 and G7	–	BC3F2 (309)	V94-5152	Ilut et al. (2016)
		Sms1	12,156,384	Greenhouse Screening SMV Strains G1 and G7	–	BC3F2 (309)	V94-5152	Ilut et al. (2016)
		Sm0	12,276,844	Greenhouse Screening SMV Strains G1 and G7	–	BC3F2 (309)	V94-5152	Ilut et al. (2016)
		BARC-021,625-04,157	12,623,066	Greenhouse Screening SMV Strain SC7	5.0%	Soybean Accessions (191) F7:16 (184)	Kefeng No. 1	Yan et al. (2015)

Table 18 (continued)

MLG (Chr.)	Locus/allele name	Tightly linked/flanking markers	Marker position (bp)[a]	Testing methods/Resistance spectrum	PVE[b]	Population Type (size)	Donor source	References
	Rsv4-b	–	–	Greenhouse Screening SMV Strains G7	–	F2 (616) F2:3 (289)	Beeson	Shakiba et al. (2013)
	Rsv4-v	Satt634 Satt296	11,441,849 12,975,935	Greenhouse Screening SMV Strains G7	–	F2:3 (403)	PI 438307	Klepadlo et al. (2016)
	Rsc5	Bin 352 Bin 353	11,300,000 11,800,000	Greenhouse Screening SMV Strain SC5	–	F7 (427)	Kefeng No. 1	Karthikeyan et al. (2017)
	Rsc7	Satt266 Satt634	14,288,241 11,778,505	Greenhouse Screening SMV Strain SC7	4.1–10.6%	Soybean Accessions (191) F7:16 (184)	Kefeng No. 1	Yan et al. (2015)
	Rsc8	ZL-42 ZL-52	12,060,386 12,091,080	Greenhouse Screening SMV Strain SC8	–	F2 (2122)	Kefeng No. 1	Lin et al. (2016), Zhao et al. (2016)
	qSC3/7-D1b	ss715580960–ss715581063	10,935,557–12,334,435	Greenhouse Screening SMV strains SC3 and SC7	54.2%	RIL (279)	Qihuang30	Chu et al. (2021a)
	qSC7-D1b	ss715581063–ss715581097	12,334,435–12,506,411	Greenhouse Screening SMV strain SC7	27.0%	RIL (279)	Qihuang30	Chu et al. (2021a)
	–	ss715583175	45,170,092	Greenhouse Screening SMV strain SC3	10.6%	Cultivars (302) and landraces (77)	–	Chu et al. (2021b)
MLG B1 (Chr. 11)	–	ss715608741	10,304,178	Greenhouse Screening SMV strain SC3	7.0%	Cultivars (302) and landraces (77)	–	Chu et al. (2021b)
MLG F (Chr. 13)	*Rsv1*	–	–	Greenhouse Screening SMV Strain SVM-1	–	F2 (1739)	PI 96983	Kiihl and Hartwig (1979)
		SoyHSP176	29,041,694	Greenhouse Screening SMV Strain G1	–	F2 (107)	PI 96983	Yu et al. (1996)
		3gG2-snp1	29,877,164	Greenhouse Screening SMV Strain G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		3gG2-snp2	30,402,642	Greenhouse Screening SMV Strain G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		N11PF-snp2	31,012,220	Greenhouse Screening SMV Strain G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		Barc-015435–01,966	32,607,605	Greenhouse Screening SMV Strain G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		BARCSOYSSR_13_1128	30,119,784	Greenhouse Screening SMV Strains SC3, SC6, SC7, SC17	–	F2 (783)	PI 96983	Yang et al. (2013)
		BARCSOYSSR_13_1136	30,464,888	Greenhouse Screening SMV Strains SC3, SC6, SC7, SC17	–	F2 (783)	PI 96983	Yang et al. (2013)
		BARCSOYSSR_13_1140	30,501,849	Greenhouse Screening SMV Strains SC3, SC6, SC7, SC17	–	F2 (783)	PI 96983	Yang et al. (2013)
		BARCSOYSSR_13_1155	30,880,128	Greenhouse Screening SMV Strains SC3, SC6, SC7, SC17	–	F2 (783)	PI 96983	Yang et al. (2013)

Table 18 (continued)

MLG (Chr.)	Locus/ allele name	Tightly linked/flanking markers	Marker position (bp)[a]	Testing methods/ Resistance spectrum	PVE[b]	Population Type (size)	Donor source	References
		Satt510	31,802,676	Greenhouse Screening SMV Strain G1	–	F2 (1056)	PI 96983	Gore et al. (2002)
	Rsv1-h	–	–	Greenhouse Screening SMV Strains G1, G5, G6, G7, G7A	–	F2 (794)	Suweon 97	Chen et al. (2002)
	Rsv1-r	–	–	Greenhouse Screening SMV Strains G1-G7	–	F2:3 (1041)	Raiden	Chen et al. (2001)
	Rsv1-k	–	–	Greenhouse Screening SMV Strain G1	–	F2:3 (1133)	Kwanggyo	Chen et al. (1991)
	Rsv1-t	–	–	Greenhouse Screening SMV Strain G1	–	F2:3 (1133)	Ogden	Chen et al. (1991)
	Rsv1-m	–	–	Greenhouse Screening SMV Strain G1	–	F2:3 (1133)	Marshall	Chen et al. (1991)
	Rsv1-n	–	–	Greenhouse Screening SMV Strains G1 and G6	–	F2:3 (239)	PI 507389	Ma et al. (2003)
	Rsv5	Satt114	28,912,864	Greenhouse Screening SMV Strain G1	–	F2:3 (3000)	York	Klepadlo et al. (2017)
	Rsc3	BARCSOYSSR_13_1128	28,919,973	Greenhouse Screening SMV Strain SC3	–	F2 (783)	PI 96983	Yang et al. (2013)
BARCSOYSSR_13_1136		29,264,742						
	Rsc14Q	Satt334	29,609,521	Greenhouse Screening SMV Strain SC14	–	F7 (231)	Qihuang No. 1	Bai et al. (2009), Ma et al. (2011)
		MY750	29,594,566					
	Rsc15ZH	–	27,801,314	Greenhouse Screening SMV Strain SC15	–	F8 (163)	Zhonghuang24	Li et al. (2020)
		–	27,864,011					
	qSMV13	ss715614844	29,741,893	Greenhouse Screening SMV strain SC3	8.1%	Cultivars (302) and landraces (77)	–	Chu et al. (2021b)
		ss715614844–ss715614864	29,741,893–29,839,120	Greenhouse Screening SMV strains SC3 and SC7	71.2–76.6%	F6:8 (193)	Kennong7	Chu et al. (2021b)
MLG B2 (Chr. 14)	–	ss715617664	13,092,389	Greenhouse Screening SMV strain SC3	19.0%	Cultivars (302) and landraces (77)	–	Chu et al. (2021b)
	Rsv3	–	–	–	–	–	OX 686	Buzzell and Tu (1989)
		Barc-012953–00,413	45,086,977	Greenhouse Screening SMV Strains G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		A519-snp2	46,937,343	Greenhouse Screening SMV Strains G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		A519-snp4	46,937,465	Greenhouse Screening SMV Strains G1-G7	–	Diverse Genotypes (47)	–	Shi et al. (2011)
		Satt063	45,993,857	Greenhouse Screening SMV Strains G5-G7	–	F2:3 (195)	L29, Tousan 140	Jeong et al. (2002)

Table 18 (continued)

MLG (Chr.)	Locus/allele name	Tightly linked/flanking markers	Marker position (bp)[a]	Testing methods/Resistance spectrum	PVE[b]	Population Type (size)	Donor source	References
		A519-f/r	46,937,953	Greenhouse Screening SMV Strains G5-G7	–	F2:3 (195)	L29, Tousan 140	Jeong et al. (2002)
		M3aSatt	47,090,758	Greenhouse Screening SMV Strains G5-G7	–	F2:3 (195)	L29, Tousan 140	Jeong et al. (2002)
		BARCSOYSSR_14_1413	46,944,330	Greenhouse Screening SMV Strains SC4	–	F2 (1047)	Dabaima	Wang et al. (2011)
		BARCSOYSSR_14_1416	47,007,588	Greenhouse Screening SMV Strains SC4	–	F2 (1047)	Dabaima	Wang et al. (2011)
		-	–	Greenhouse Screening SMV Strains G6 and G7	–	F2:3 (472)	Harosoy	Gunduz et al. (2001)
	Rsv3-n	Satt534	45,051,723	Greenhouse Screening SMV Strains G1 and G7	–	F2 (273) F2:3 (196)	PI 61944	Cervantes-Martinez et al. (2015)
	Rsv3-h	Satt063	45,993,857	Greenhouse Screening SMV Strains G7	–	F2 (616) F2:3 (289)	PI 61947	Shakiba et al. (2012a, b)
	Rsv3-c	Satt063	45,993,857	Greenhouse Screening SMV Strains G7	–	F2 (616) F2:3 (289)	PI 399091	Shakiba et al. (2012a, b)
MLG J (Chr. 16)	-	ss715625254	6,042,142	Greenhouse Screening SMV strain SC3	6.0%	Cultivars (302) and landraces (77)	–	Chu et al. (2021b)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax2.0*

[b]Phenotypic variations explained by the molecular markers

Table 19 Soybean loci conferring resistance to other soybean viruses

Causal agent	MLG (Chr.)	Locus name	Tightly linked markers	Marker position cM (bp)[a]	Testing methods/Resistance spectrum	Population type (size)	PVE[b]	Donor source	References
Alfalfa mosaic virus (AMV)	MLG J (Chr. 16)	*Rav1*	Sat_228	23,91 cM (3,049,971)	Greenhouse test/AMV-C field isolate, Wiconsin, USA (2001)	F4:7 (174)	79%	PI 153282	Kopisch-Obuch et al. (2008)
Soybean dwarf virus (SbDV)	MLG A1 (Chr.5)	*Rsdv1*	Sat_11 and Sct_13	–	Greenhouse and field tests	F6 (289)	79%	Wilis	Uchibori et al. (2009), Yamashita et al. (2013)
	MLG N (Chr.3)	*Raso1*	Gm03-11 and Gm03-12	(4,661,084–4,724,159)	*Raso1* confers resistance to foxglove aphid, but require additional genes for tolerance to SbDV	F2 (669, 576)	32% to aphid	Adams (PI 548502)	Ohnishi et al. (2012)

[a]Marker position (bp) based on the *Glycine max* genome assembly version *Gmax1.01*
[b]Phenotypic variations explained by the molecular markers

may become a serious viral disease of soybean (Malapi-Nelson et al. 2009).

Recommended management strategies include selection of resistant cultivars and the use of clean virus-free seed. Resistance to AMV in the Brazilian cultivars 'Pérola' and 'Planalto' and their common ancestor 'Hood' was reported to be controlled by a single dominant gene (Almeida et al. 1982). Two cultivars, 'Wuyuezha' and 'Baimaodou', were described as tolerant in China (Che et al. 2020). In the USA, resistance to AMV was found in PI 153,282. Genetic studies revealed the existence of one dominant gene, which was named *Rav1*, and DNA marker analysis allowed its location on a genetic map (Kopisch-Obuch et al. 2008) (Table 19).

Bean pod mottle virus

Bean pod mottle virus (BPMV), a member of genus *Comovirus* in the family *Comoviridae*, is a major viral pathogen of soybean first identified in Arkansas in 1951 (Walters 1958). The adult bean leaf beetle, *Cerotoma trifurcate* Forster (Coleoptera: Chrysomelidae), has been known as a main vector of BPMV, but it is also a destructive insect feeding on leaves, stems, and pods in soybean production regions in the USA (Pedigo and Zeiss 1996; Giesler et al. 2002). Plant responses to this pathogen can range from mild chlorotic mottling to severe mosaic on younger soybean leaves co-occurring with green stem symptoms (Giesler et al. 2002; Zheng et al. 2005b; Rodriguez and Thiessen 2020). BPMV can also cause plant stunting, leaf distortion, wilting, and reduced pods per plant and seed size and quality under severe infection (Myhre et al. 1973; Schwenk and Nickell 1980; Giesler et al. 2002). Soybean yield reductions resulting from BPMV infection have been reported as high as 52% (Hopkins and Mueller 1984; Gergerich 1999), and it can be maximized by the infection before V6 stage (Fehr et al. 1971) or the co-infections with soybean mosaic virus (Ross 1968; Rodriguez and Thiessen, 2020). Although Ross (1986) developed and released four BPMV-resistant soybean germplasm lines, these lines showed mild symptoms with systemic infections, and there is still no commercial soybean variety with BPMV resistance (Zheng et al. 2005b; Rodriguez and Thiessen 2020). Genetic loci for BPMV resistance have not been thoroughly investigated in soybean, but several studies have successfully engineered BPMV resistance in transgenic soybean plants by overexpressing ds-specific ribonuclease gene *PAC1* (RNase III family) from *Schizosaccharomyces pombe* and RNAi-based strategies (Reddy et al. 2001; Zhang et al. 2011; Yang et al. 2019). However, with previously identified 15 *G. soja* and 12 *G. tomentella* lines showing tolerance with

mild symptoms or no systematic infection to BPMV, soybean breeders may want to incorporate those useful genetic sources into *G. max* by interspecific crosses for further investigation in specific loci and molecular marker development in soybean (Zheng et al. 2005b). The virus infection assay for BPMV was well described by Zheng et al. (2005b) using four diverse isolates (*K-G7*, *K-Ha1*, *K-Ho1*, and *AR*) and enzyme-linked immunosorbent assay (ELISA).

Soybean vein necrotic virus

Soybean vein necrosis virus (SVNV) was first reported in Arkansas and Tennessee in 2008 (Tzanetakis et al. 2009) and is now found in 22 states in the USA as well as in Canada and Egypt (Zhou 2012; Ali and Abdalla 2013; Conner et al. 2013; Han et al. 2013; Jacobs et al. 2013; Smith et al. 2013; Kleczewski 2016; Abd El-Wahab and El-Shazly 2017; Escalante et al. 2018). It is now the most prevalent virus in North America (Zhou and Tzanetakis 2013). Symptoms caused by SVNV begin as clearing of the main leaflet veins that progressively become necrotic. When severe, these symptoms can expand to encompass the entire leaflet (Tzanetakis et al. 2009). Seeds of plants infected by SVNV can have lower oil and protein content (Groves et al. 2016; Anderson et al. 2017), with higher levels of linoleic acid and lower levels of oleic acid (Anderson et al. 2017). It is not known if the virus reduces overall yields. There is evidence of seed transmission (Groves et al. 2016), but SVNV is vectored primarily by thrips which transmit SVNV in a persistent and propagative manner (Zhou et al. 2013). The primary thrips vector is *Neohydatothrips variabilis*, but the thrips *Frankliniella tirtici* and *F. fusca* also transmit the virus at lower rates (Zhou et al. 2018).

Two studies have identified resistance related to SVNV. Zhou et al. (2020) compared the feeding preferences of *N. variabilis* on 11 soybean accessions and suggested breeders consider PI 547,422 as a source of resistance. In a more recent study, seven soybean genotypes were inoculated under controlled conditions using SVNV-infected thirps (*N. variabilis*), and their results suggested that the genotypes '51–23', '91–38', and 'SSR51-70' were resistant to SVNV and 51–23 was tolerant (some symptom development, but very low virus titer) (Zambrana-Echevarria 2021).

An alternative mechanism to control SVNV is blocking the vector-virus interaction via synthetic glycopeptides that compete with SVNV glycopeptides to reduce transmission of SVNV by *N. variabilis* (Zhou and Tzanetakis 2020). These peptides reduced the transmission of SVNV by at least 50% (Zhou and Tzanetakis 2020).

Soybean dwarf virus

Soybean dwarf virus (SbDV) was first noticed in Hokkaido in 1969 and remains a major soybean yield suppressor in northern Japan (Tamada et al. 1969; Harrison et al. 2005). The symptoms of SbDV include dwarfing (stunting), downward curling, rugosity, and interveinal yellowing of the leaves. *Rsdv1* is the only gene known to confer major resistance to SbDV (Uchibori et al. 2009; Yamashita et al. 2013). Another gene, *Raso1*, was found conferring resistance to foxglove aphid, a transmission vector of SbDV, but a further study indicated that *Raso1* needs at least one additional gene for resistance to SbDV (Table 19) (Ohnishi et al. 2012).

Conclusions and future perspectives

With the identification and implementation of molecular markers tightly linked with resistance genes, the introgression of vertical resistance through MAS became a practice routinely performed by public and private soybean breeding programs. Efforts to understand minor genes with small but accumulative effects for horizontal resistance will also be needed. What's more, to expand the sources of resistance and discover resistance genes and QTLs to ensure the sustainability of soybean production, continuous efforts are needed to screen diverse germplasm lines. For example, the USDA Germplasm Collection (GRIN) provides more than 20,000 soybeans accessions worldwide and more resistance sources can be expected to be identified. Germplasm lines and elite soybean cultivars with resistance to multiple diseases combined with high-yielding potential and desired agronomic traits are being developed. In addition, interaction among resistance loci, allelic and copy number variations, their interactions with environment, and impact on virulence of pathogens and disease development deserve close attention in future research.

Advances in genomics facilitated the introduction of next generation sequencing (NGS)-based high-density molecular markers which are quickly evolved and became available at an accessible cost for both public and private breeding programs (Song et al. 2013, 2020). Genome-wide studies revealed many novel regions of the soybean genome significantly associated with resistance to different pathogens, and traits that were often considered qualitative in nature evolved to some extent into quantitative traits with major and minor alleles with small effects contributing to the observed phenotypes. The rise of digitally smart-agriculture and the application of machine learning and artificial intelligence for characterizing the response of

breeding lines to specific diseases represented another breakthrough in breeding for genetic resistance. Disease assessment screening protocols often reported on categorical scales based on subjective ratings are gradually being replaced by precise quantitative metrics representing the observed phenotypes (Gazala et al. 2013; Khalili et al. 2020; Gui et al. 2021; Liu et al. 2021). In combination with advanced predictive analytics and mega environmental data, one can predict the response of soybean breeding lines to specific or multiple diseases in diverse environments, which can be a powerful tool to anticipate the deployment of resistant cultivars to potential disease outbreaks and extreme environmental conditions.

Throughout this review, the impact of pathogens in global soybean production and their respective yield losses have been discussed. Substantial yearly production losses in the order of billions of dollars due to diseases have been repeatedly reported in the literature for decades (Wrather et al. 1997; 2001; Allen et al. 2017; Savary et al. 2019; Bandara et al. 2020). Genetic resistance is the most effective and sustainable approach for the disease management in soybean globally, representing a critical pillar bolstering the global soybean value chain and food security. Although hundreds of significant genomic regions conferring resistance to multiple pathogens have been reported in this review, there are many components of genetic resistance still to be enlightened and continuously investigated. For instance, limited advancements have been achieved in understanding the pathogen infectious dynamics and underlying genetic regulations. The substantial shift and emergence of novel and/or resistance-breaking strains and emergence of pathogen races impose a threat to previously validated resistance genes. In addition, the pleiotropic effect of resistance genes and the interaction among those in terms of durable broad-based resistance levels, yield penalty, as well as environmental interactions are now becoming critically important due to the availability of big genomic data and emergence of advanced analytical algorithms (Patil et al. 2019).

Whole genome resequencing facilitated the characterization of diverse lines with superior haplotypes or alleles among unexplored germplasm which could be used to deploy durable resistance in plant breeding program. The future breeding era is likely to be genomics-assisted breeding (GAB) including marker-assisted recurrent selection (MARS), marker-assisted backcrossing (MABC), haplotype-based breeding, and genomic selection (GS) (Varshney et al. 2021). Trait-associated genes would be mapped with NGS-based trait mapping and system biology approach. Future genetic variations can be estimated by targeting induced local lesions in genomes (TILLING), Eco-TILLING populations, and multiparent advanced generation intercross (MAGIC) or can be created through genome/gene editing (GE). GE has been emerged at an unprecedented speed and probably become a primary technique for translating genomic information to improvement of the crop in the field. However, the success of the development of CRISPR/Cas9 transformants is subject to effective genetic transformation system. Unfortunately, soybean is a recalcitrant crop for plant transformation technology and most of the GE studies are in primary phase of development. Although a few studies have successfully show the introduction of Cas12a-RNP in soybean protoplast (Kim et al. 2017), enormous efforts may be needed to implement these tools into soybean.

All in all, the early establishment of the soybean research field, the vast availability of unexplored genetic diversity through soybean accessions, the breakthrough advancements in genomics and analytics, and the dynamism of the environment, pathogens, and host genetic background will significantly improve the efficiency and accuracy of global soybean breeding in the next decades, ensuring the sustainability and growth of soybean production worldwide.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00122-022-04101-3.

Acknowledgements We thank the funding support from United Soybean Board (Grant No. 2120-172-0153), National Sclerotinia Initiative, Michigan Soybean Promotion Committee, Missouri Soybean Merchandising Council, Arkansas Soybean Promotion Board, USDA National Institute of Food and Agriculture (Hatch project 1011788, Hatch project 1017862, NIFA grant No. MO-00063945 and Award No. 2019-67013-29370), AgBioResearch at Michigan State University (Project No. MICL02013), and University of Arkansas Division of Agriculture. We also acknowledge funding support from Ministerio de Ciencia, Tecnología e Innovación Productiva of Argentina (MinCyT), Plataforma BiotecSur-UE, Proyecto BiotecSojaSur I N° 127119; Proyecto Investigación Regional MERCOSUR BiotecSojaSur II-Convenio MinCyT Fundación InnovaT (2012)- Acta Comp. N° 52; PICTOs from Agencia Nacional de Investigación Científica y Tecnológica (ANPCyT) – Estación Experimental Agroindustrial Obispo Colombres (EEAOC), N° 2005-35332 and N° 2016-0098; and from the Grain Program of EEAOC, Projects GrF2, GrB2, and GrB4.

Author's Contribution HTN, DW, and PC conceived and supervised the review; FL is author team coordinator and led the sections of Phytophthora root and stem rot, Pythium damping-off and root rot, downy mildew, sudden death syndrome and Fusarium wilt and root rot, Sclerotinia stem rot, Rhizoctonia damping-off and root rot, Bacterial diseases, and soybean dwarf virus; SSC led the section of soybean cyst nematode; CCV led sections of root-knot nematodes and soybean mosaic virus; MPS led sections of stem canker and charcoal rot; AR led sections of Phytophthora root and stem rot, Pythium damping-off and root rot, and stem canker; DL led sections of reniform nematodes, lance nematodes, brown stem rot, and bean pod mottle virus; NL led sections of frogeye leaf spot and soybean mosaic virus; EMP led the section of soybean rust; all authors contributed to the writing and editing of the manuscript. All authors read and approved the final manuscript.

Funding The authors have not disclosed any funding.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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