Genomic Approaches for Resistance Against Fungal Diseases in Soybean

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Abstract

Soybean (*Glycine max* (L.) Merr.), an essential leguminous crop, is plagued by several fungal diseases, which is a major worry for soybean farmers worldwide. Significant progress has been made in recent decades in the identification of pathogen-caused diseases, the sources of resistance, and the determination of genomic loci granting resistance to various diseases on linkage maps of soybean. To maintain the sustainability and expansion of soybean production globally, the application of genomics to disease-resistant soybean cultivars is a common goal. Marker-assisted selection and genomic selection have been shown to be effective methods for quickly integrating vertical resistance or horizontal resistance into improved soybean varieties. Vertical resistance is defined as R genes and major effect QTLs, whereas horizontal resistance is a combination of major and minor effect genes or QTLs. In this chapter, we have focused on some important fungal

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diseases of soybean, and genomic approaches like breeding, identification of QTLs, transcriptomics for differentially expressed genes (DEGs), metabolomics, and proteomics that confer resistance to fungal diseases in all major soybean production regions of the world are provided. We also emphasized the use of modern genomic tools by providing a thorough summary of significant resistance genes and QTLs for soybean improvement. The condensed genetic knowledge also illuminates the future directions for translational genomics research and expedited soybean breeding. The primary goals of soybean crop improvement are centred on the discovery of sources of resistance to various biotic as well as abiotic stresses and the use of these sources for additional hybridization and transgenic processes to generate new cultivars for stress management.

Keywords

Soybean · Fungi · Resistance · QTLs · Genomics

13.1 Introduction

Soybean (*Glycine max*) is an important legume crop recognized for its high seed protein and oil content (Chander et al. 2019). Having diverse climate adaptability and high protein content, it is cultivated in most part of the globe. A variety of food products and industrial food items are made from soybeans; in addition, it is also utilized as animal feed (Ratnaparkhe et al. 2022). In India, soya bean (*Glycine max* (L) Merrill) has been the most cultivated oil seed crop in terms of both production and area since 2005 (Gawai and Mangnalikar 2018). Soybean seeds are high in protein, oil, vitamins, and minerals, and they are an excellent source of vegetable oil and nutritious plant protein (Patil et al. 2018). Soybean accounted for 42% of total oil seed production in India and 25% of edible oil production. In India, soya bean is primarily produced in Madhya Pradesh, Maharashtra, Rajasthan, Karnataka, Telangana, Chhattisgarh, Nagaland, and Gujarat during a kharif season crop (Gawai and Mangnalikar 2018). According to Tripathi et al. (2022), about ten of the total number of fungal pathogens are consistently present in different regions of the world. Six of which are harmful particularly in India, namely, *Sclerotium rolfsii*, *Macrophomina phaseolina*, *Colletotrichum truncatum*, *Phakopsora pachyrhizi*, *Cercospora sojina*, and *Cercospora kikuchii*. Most of the diseased plants are treated with various chemicals to protect the crops and left their residual effects to the environment. It is better to find some resistant genotypes rather than using hazardous chemicals. Although identification of disease-resistant cultivar is difficult task, the modern molecular breeding tools could increase the efficiency to develop disease-resistant cultivars by transferring resistant gene to the genotype of our interest, developing mapping population, identification genomic regions/QTLs, etc. The resistance nature in soybean was found to be monogenic or polygenic (Tripathi

et al. 2022). The present study offers a glimpse into the genomic strategies used to identify the genes/markers linked to the targeted genes in soybeans that are resistant to fungal diseases.

13.2 Soybean Rust (SBR), Its Causal Organism, Important Symptoms, and Economic Loss

In the southern hemisphere, primarily in Asia (Taiwan, Thailand, Japan, and India), Africa, and South America, a potentially fatal foliar disease caused by two meticulously associated obligate fungal species, *Phakopsora pachyrhizi* Sydows and *P. meibomiae* (Arthur), is posing a serious threat to soybean cultivation (Langenbach et al. 2016). The specific ability of *P. pachyrhizi* to infect a wide variety of crop species, a total of 95 plants from 42 genera of the family *Papillionaceae*, presents significant management issues for soybean rust disease (Bromfield 1984). This disease has varying impact on soybean output as it may cause up to 80% yield loss in the zones favourable for growth and proliferation of the causative organism (Hartman et al. 2005).

13.3 *Rhizoctonia* Root Rot, Its Causal Organism, Important Symptoms, and Economic Loss

Rhizoctonia root rot is a soil-borne fungal disease caused by *Rhizoctonia solani* Küuhn. It causes up to 60–70% yield losses in India, 30–60% yield losses in Brazil, and 30–45% yield losses in the USA (Ciampi et al. 2008).

13.4 Brown Stem Rot (BSR), Its Causal Organism, Important Symptoms, and Economic Loss

The soil-borne fungus *Cadophora gregata* is the primary cause of BSR, a serious disease of soybeans (Harrington and McNew 2003). The fungus prevents water and nutrients from moving through the stem of soybean plants, which is essential for their normal growth and development. The majority of BSR illness cases are only detectable after complete pod formation (McCabe et al. 2018). Nutrient deficiency is the most common diagnosis for this illness. Recently, McCabe and Graham (2020) presented a diagnostic strategy based on genes and their network for quick and precise identification to combat misidentification of BSR. The management of this condition may benefit from this strategy. BSR has been cited as the cause of a 38% yield reduction in soybean harvests (McCabe et al. 2018).

13.5 Powdery Mildew (PMD), Its Causal Organism, Important Symptoms, and Economic Loss

The fungus *Microsphaera diffusa* (Paxton and Rogers (1974) causes powdery mildew. The main signs of this illness are infected soybean leaves that have a white, powdery coating. The rate of photosynthesis is decreased by more than 50% as a result of this coating (Dunleavy 1978). In addition, approximately 35% yield reduction occurs along with deteriorated soybean seed quality (Phillips 1984). The powdery patches are first visible on the leaves, but after a few days, they quickly cover the entire leaf and defoliate (Silva 2004).

13.6 *Fusarium* Wilt (FW), Its Causal Organism, Important Symptoms, and Economic Loss (FW, Also Known as Sudden Death Syndrome, SDS)

For the first time, wilted soybean plants were diagnosed in May 2014 in commercial fields at Osijek (Slavonia County) and are caused by the fungal pathogen *Fusarium oxysporum* Schlecht. emend. Snyder and Hansen (Duvnjak et al. 2016). The symptoms of wilting in soybean plants were interveinal chlorosis of leaves, mortality of shoots, and external and internal browning at the base of stems but no symptoms in roots. Due to SDS, yield reductions of up to 5–15% have been seen in the USA (Luo et al. 2001). Due to the disease's frequently environment-sensitive, unpredictable, and irregular disease appearance as well as its time-consuming and expensive treatment, sudden death syndrome resistance is difficult to control in the field (Gibson et al. 1994). Resistance to SDS is partial, and partial disease resistance has advantages over total resistance in terms of consistency and yield compatibility (Yuan et al. 2002).

13.7 Downy Mildew, Its Causal Organism, Important Symptoms, and Economic Loss

Soybean downy mildew (SDM) is one of the major fungal diseases caused by *Peronospora manshurica* (Dong et al. 2018). The onset of symptoms is greatly influenced by the environment and is favoured by high humidity and temperatures of 20–22 °C (Phillips 1999). According to Taguchi-Shiobara et al. (2019), 33 different downy mildew races have been identified so far in the USA. In epidemic years, the average yield loss ranged from 6 to 15% (Dong et al. 2018).

13.8 Anthracnose, Its Causal Organism, Important Symptoms, and Economic Loss

The common soybean disease anthracnose is brought on by the fungus *Colletotrichum truncatum* (Schw.) Andrus & W.D. Moore (Sinclair and Backman 1989). The anthracnose disease caused a yield loss of 16–25% in India (Boufleur et al. 2021). Although several other species are also recognized as anthracnose causal agents, *C. truncatum* has been thought to be the primary cause of the anthracnose disease in soybeans.

13.9 Soybean White Mould (SWM), Its Causal Organism, Important Symptoms, and Economic Loss

One of the most devastating fungal diseases is soybean white mould (SWM) caused by the fungus *Sclerotinia sclerotiorum* (Lib) de Barry which can be found in southern Canada and the Upper Midwest of the USA (Kandel et al. 2018). According to Koenning and Wrather (2010), SWM causes significant yield losses and ranked fourth from the top 28 soybean producing US states. *Sclerotinia sclerotiorum* overwinters in resting structures known as sclerotia in the soil and debris (Yang et al. 1998). However, ascospores that initially touch down on fragile plant parts, such flower petals, are what caused infections of soybean in field situations. They become colonized by ascospores, which subsequently move downhill to infect and girdle the main stem, causing the plant to eventually perish. In addition, necrotic leaves, bleached lesions on stems and pods, white fluffy mycelial growth, and the appearance of black sclerotia on the leaves, stems, and pods are the common symptoms of infected plants (Chen and Wang 2005).

13.10 *Phomopsis* Seed Decay, Its Causal Organism, Important Symptoms, and Economic Loss

Phomopsis seed decay (PSD) of soybean is the primary cause of poor seed quality and causes a significant yield loss in most soybean-growing countries (Sinclair 1993). PSD is more likely to occur in environments that are hot and humid, and it typically gets worse when early maturing cultivars are planted early in the season. Significant symptoms include shrivelled, elongated, or cracked look and a chalky texture, but seed infection is typically asymptomatic.

13.11 *Cercospora* Leaf Blight (CLB)/Purple Seed Stain, Its Causal Organism, Important Symptoms, and Economic Loss

Cercospora leaf blight (CLB)/purple seed stain is a foliar fungal disease of soybean caused by *Cercospora kikuchii* (Albu et al. 2016). Reddish patches on leaves are one of the symptoms. Additionally, these hues intensify and cause soybeans to flower too

early. *Cercospora kikuchii* also reduces the marketability, processing potentials, germination, and vigour of seed (Kashiwa and Suzuki 2021). In soybeans, this fungus is the source of causing both *Cercospora* leaf blight (CLB) and/or purple seed stain (PSS) disease. In contrast to CLB, which affects leaves and petioles, PSS affects seed pods and seeds. A distinctive abrasion with a dark purple colour is one of these signs. The pathogen's synthesis of cercosporin led to the development of this lesion (Callahan et al. 1999). Because it degrades the quality of the seed, purple seed stain is a major barrier to its profitable marketability (Li et al. 2019). Various study groups in India have observed yield loss due to purple seed discolouration at different percentages, including 15–30% (Gupta et al. 1999) and 36–80% (Gupta et al. 2014). It is a disease that Americans find undesirable due to economic yield losses (Doupnik 1993).

13.12 Charcoal Rot, Its Causal Organism, Important Symptoms, and Economic Loss

Charcoal rot is caused by soil-borne fungus *Macrophomina phaseolina* and also causes significant yield reduction in soybean (Tripathi et al. 2022). This disease was first time reported in 1949 in the USA, and it was assumed that the presence of two toxins, *phaseolina* and *botryodiplodin*, are responsible for the infection caused by *M. phaseolina* in crops (Ramezani et al. 2007). *M. phaseolina* can infect the vascular system by growing and multiplying under favourable environmental circumstances in plants. It obstructs the movement of water and nutrients toward the leaves in the second step, which results in disease symptoms and further premature leaf death (Gupta and Chauhan 2005). Microsclerotia return to the soil after the crop is harvested and remain there for at least 2 years (Reis et al. 2014). Soybean crops have only exhibited little resistance to *M. phaseolina* (Pawlowski et al. 2015). Due to polygenic inheritance, it is challenging to breed soybean cultivars resistant to charcoal rot (Coser et al. 2017).

13.13 *Phytophthora* Rot and Stem Rot, Its Causal Organism, Important Symptoms, and Economic Loss

Phytophthora sojae is a soil-borne pathogen that causes *Phytophthora* root and stem rot diseases. The soybean crop is affected throughout the years by this disease. It is more devastating in flooded areas (Bernard et al. 1957). *Phytophthora root rot* often results in a yield loss of 35–40%, but under extreme circumstances, it can even result in a loss of 100% of the crop. The most effective strategy for controlling this disease is the creation of resistant cultivars.

In disease management strategies, it is better to find resistance genes or screening resistant cultivars rather than going for chemical application. Advances in plant breeding techniques, application of molecular markers, identification, and expression analysis of target genes linked to disease resistance have opened multiple ways

for the modification of the targeted genomic regions of desired genotypes or cultivars (Fig. 13.1). Here we have attempted to explain integrated genomics for several fungal disease management and identification of some resistant lines/genotypes cultivars (Tables 13.1 and 13.2).

13.14 Integrated Genomic Approaches for Developing Resistance Against Fungal Disease in Soybean

13.14.1 Screening and Identification of Soybean Genotype/Germplasm Resistant to Fungal Disease

Screening of different genetic materials like pre-breeding lines, germplasms, accessions, etc. has tremendous importance in search of sources for disease resistance in both fields as well as in laboratory condition.

There have been numerous research organization attempts to screen soybean germplasm for the presence of fungal disease resistance in soybean. Recently, Nataraj et al. (2020) evaluated 225 soybean genotypes and identified five genotypes as highly resistant, and they are EC 538828, EC 34372, EC 457254, AKSS 67, and Karune. In addition to this, the genetics of anthracnose resistance in three F2 populations descended from the resistant parents EC 34372 × JS 95-60, EC 457254 × JS 95-60, and AKSS 67 JS 95-60 which showed that the resistance in all three resistant parents was controlled by two key genes interacting in a complimentary manner. Similar study by Sajeesh et al. (2014) identified DSb 12 as an anthracnose-resistant genotype.

13.14.2 Identification of QTL(S)/Genomic Loci Conferring Resistance to Fungal Disease in Soybean

Biparental mapping populations are made up of a group of individuals resulting from inter- or intraspecific crossing between two parents. Such recombinant lines are mostly used to provide pre-breeding sources for use in crop improvement, and they constitute a potent technique for analysing the genetic underpinnings of complex traits in crops (Tripodi 2021). Recently, Chanchu et al. (2022) reported a single QTL, *qSBR18.1*, for SBR resistance by evaluating a recombinant inbred line (RIL) population comprising of 108 lines developed from a cross between a susceptible cultivar Sukhothai 2 (SKT2) and CM5.

For BSR, the BSR resistance genes in soybean have been mapped by a number of researchers using marker-assisted breeding. The *Rbs3* gene was initially mapped by Lewers et al. (1999) using 320 recombinant inbred lines (RIL) developed from a cross between BSR 101 and PI 437.654. The same study was also verified by Klos et al. (2000) using SSR markers. Later study, SSR markers were used by Bachman et al. (2001) to map the *Rbs1* and *Rbs2* genes on chromosome 16 in soybean. In addition, Perez et al. (2010) have identified some novel sources of BSR resistance. In

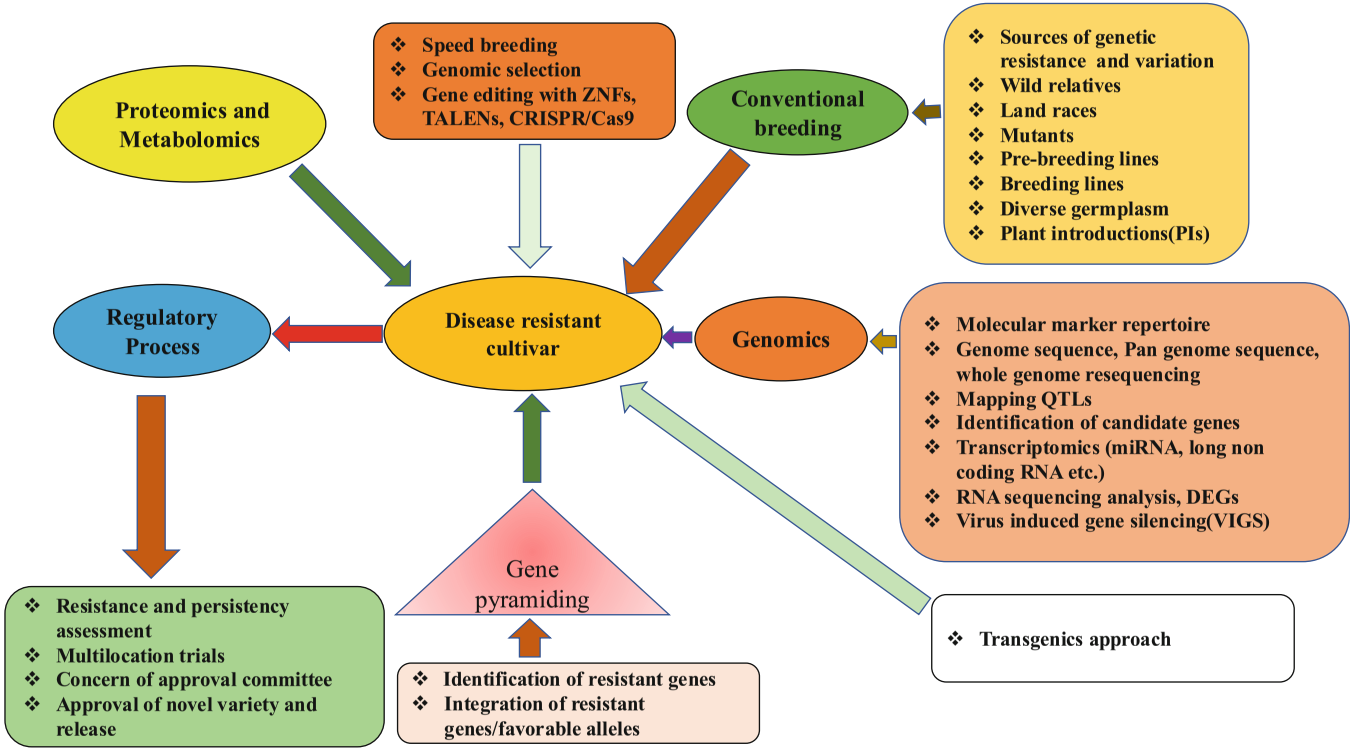

Fig. 13.1 Different genomic approaches for disease improvement in soybean

Table 13.1 List of identified genomic regions/quantitative trait loci (QTLs) conferring resistance to various fungal diseases in soybean

Fungal diseases	Causal organism	Material used	Linkage groups/ chromosome number	Tightly linked markers/ flanking markers	Molecular marker used	Position of QTLs on chromosome (cM)/bp	Locus/ QTLs name	Isolates used	% Phenotypic variation	References
Rust	*Phakopsora pachyrhizi*	F2:3 (173)	Chr.03	Sat_275 and Sat_280	SSR	40.81–43.45 cM	*Rpp5*	*BRSMS Bacuri*		Garcia et al. (2008)
		F2 (86)	Chr. 06	Sat_263 and Sat_238	SSR	118.67–117.45 cM	*Rpp3*	*Japanese T1–2*	70%	Hossain et al. (2015)
		F6 (240)	Chr. 08	Satt409 and Satt429	SSR	145.57–162.02 cM	*QTL Asian soybean rust 2–1*	Local isolate (Georgia, USA)	10%	Harris et al. (2015)
		F6:7 (250)	Chr. 06	Sat_312 and BARC-203517–05,442	SSR	27,940,542–36,131,665 bp	Rpp3	Field tests (FL, US)	8.40%	Vuong et al. (2015)
		F2 (106)	Chr. 18	Sat_064 and AF162283	SSR	108.69–87.94 cM		*Isolate E1-4–12*	65–67%	Yamanaka et al. (2016)
		F5:6 (184)	Chr. 18	GSM0374 and GSM0427	SSR	5,998,461–6,160,481 bp		*Isolate GA12*		King et al. (2016)
		F2:3 (90/100)	Chr. 19	GSM0546 and GSM0463	SSR	39,462,291–39,616,643 bp	Rpp7	*AU79-1 (Australia), CO04-2 (Armenia, Columbia), GA12-1 (Georgia, USA),*		Childs et al. (2018)
		RIL(108)	Chr. 18(G)	T001855631m—sc21_3420	SSR	123 cM	qSBR18.1	–	37.55	Chanchu et al. (2022)
Rhizoctonia root rot	*Rhizoctonia solani*	PI 442031	A2	Satt177	SSR	–	–	–	–	Ishiwata and Furuya (2020)
			C2 M	Satt281	SSR	–	–	–	–	Ishiwata and Furuya (2020)
			M	Satt245	SSR	–	–	–	–	Ishiwata and Furuya (2020)
Brown stem rot	*Cadophora gregata*	F2:3 (73)	Chr. 16	Satt215	SSR	28,944,536–28,944,665 bp	Rbs1	Green house assay	28%	Bachman et al. (2001)

(continued)

Table 13.1 (continued)

Fungal diseases	Causal organism	Material used	Linkage groups/ chromosome number	Tightly linked markers/ flanking markers	Molecular marker used	Position of QTLs on chromosome (cM)/bp	Locus/ QTLs name	Isolates used	% Phenotypic variation	References
		F2:3 (73)	Chr. 16	Satt431	SSR	36,221,174–36,221,397 bp		Green house assay	74%	Bachman et al. (2001)
		F2:3 (77)	Chr. 16	Satt244	SSR	33,818,897–33,819,094 bp	Rbs2	Green house assay	67%	Bachman et al. (2001)
		F2:3 (77)	Chr. 16	Satt431	SSR	36,221,174–36,221,397 bp		Green house assay	46%	Bachman et al. (2001)
		F6:7 (320)	Chr. 16	K375	SSR	67.3–69.3 cM bp	Rbs3	Green house assay	62%	Lewers et al. (1999)
		F6:7 (320)	Chr. 16	B122	SSR	53.8–55.8 cM bp		Green house assay	45%	Lewers et al. (1999)
Powdery mildew	*Microsphaera diffusa*	BRS135 (cultivar)	Chr. 16	GMES6959 Satt_393	SSR	4.6 cM–7.1 cM	*PMD*	–	–	Gordon et al. (2007)
		PI 567301B	Chr. 16	BARCSOYSSR_16_1291	SSR	3.3 cM	*PMD*	–	–	Jun et al. (2012)
		PI 243540	Chr. 16	Sat_224 BARC-021875-04228	SSR	1.3 cM–9.6 cM	*Rmd*	–	–	Kang and Mian (2010)
		ZH24		Gm16_428	SSR	–	*ASR*	–	–	Zhou et al. (2022)
Fusarium wilt/SDS	*Fusarium oxysporum / fusarium virguliforme*	F6:13 (50)	Chr. 2	ss107920774–ss107912689	SSR	30.0–36.0 cM	qRfv02-01	*Isolate Mont1*	5.20%	Abdel Majid et al. (2012)
		Advanced breeding lines (300)	Chr. 2	ss244884978	SSR	49,773,810 bp	qRfv02-01	*Field test (MI, US)*	6.40%	Wen et al. (2014)
		F7 derived RIL (200)	Chr. 2	BARC-041581–08,046—BARC-046084–10,230	SSR	93.341–02.59 cM	qRfv02-01	*Isolates Clinton 1B, Scott F2II 1a and Scott B2*	8.40%	Swaminathan et al. (2016)
		F5:11 (100)	Chr. 3	OF041600	SSR	–	qRfv03-01	*Field test (IL, US)*	10%	Chang et al. (1996), Chang et al. (2018)

		F6:13 (50)	Chr. 3	ss107912585–ss107920575	SSR	38.3–42.6 cM	qRfv03-02	*Isolate Mont1*	9.90%	Chang et al. (2018)
		F4 derived (129)	Chr. 4	ss245526764–ss245561373	SSR	48.56–83.86 cM	qRfv04-01	Field test (MI, US)	3.7–5.3%	Tan et al. (2018)
		F4 derived (153)	Chr. 4	ss245560843–ss245567348	SSR	47.29 Mb–48.08 Mb	qRfv04-01	Field test (MI, US)	6.36%	Tan et al. (2019)
Downy mildew	*Peronospora manshurica*	F6 and F7 (112)	Chr. 2	WGSP02_0160–WGSP02_0170	SSR	50 Mb	QRpm2-1	Field test (Japan)	8–10%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (155), F5 and F6 (190), F6 and F7 (112)	Chr. 3	WGSP03_0040–WGSP03_0070	SSR	–	QRpm3-1	Field test (Japan)	18–72%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (155)	Chr. 4	WGSP04_0120–WGSP04_0140	SSR	–	QRpm4-1	Field test (Japan)	4%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (155)	Chr. 6	WGSP06_0200–WGSP06_0210	SSR	–	QRpm6-1	Field test (Japan)	8%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (189), F9 and F10 (231), (F5 and F6 (190)	Chr. 7	WGSP07_0060–WGSP07_0070	SSR	5 Mb	QRpm7-1	Field test (Japan)	6–91%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (155)	Chr. 8	WGSP08_0110–WGSP08_0130	SSR	20 Mb	QRpm8-1	Field test (Japan)	13–24%	Taguchi-Shiobara et al. (2019)
		F9 and F10(231)	Chr. 11	WGSP11_0100–WGSP11_0120	SSR		QRpm11-1	Field test (Japan)	4%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (112)	Chr. 12	WGSP12_0120–WGSP12_0130	SSR	35 Mb	QRpm12-1	Field test (Japan)	6–8%	Taguchi-Shiobara et al. (2019)

(continued)

Table 13.1 (continued)

Fungal diseases	Causal organism	Material used	Linkage groups/ chromosome number	Tightly linked markers/ flanking markers	Molecular marker used	Position of QTLs on chromosome (cM)/bp	Locus/ QTLs name	Isolates used	% Phenotypic variation	References
		F5 and F6 (189)	Chr. 13	WGSP13_0080–WGSP13_0120	SSR	–	QRpm13-1	Field test (Japan)	3%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (189)	Chr. 14	WGSP14_0050–WGSP14_0060	SSR	–	QRpm14-1	Field test (Japan)	4%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (190)	Chr. 15	WGSP15_0130–WGSP15_0140	SSR	–	QRpm15-1	Field test (Japan)	3%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (190)	Chr. 16	WGSP16_0090–WGSP16_0100	SSR	–	QRpm16-1	Field test (Japan)	3%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (189)	Chr. 18	WGSP18_0150–WGSP18_0160	SSR	50–60 Mb	QRpm18-1	Field test (Japan)	11–16%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (155)	Chr. 19	WGSP19_0150–WGSP19_0170	SSR	–	QRpm19-1	Field test (Japan)	7%	Taguchi-Shiobara et al. (2019)
		F5 and F6 (189)	Chr. 20	WGSP20_0100–WGSP20_0130	SSR	–	Rpm20-1	Field test (Japan)	4%	Taguchi-Shiobara et al. (2019)
		F6 and F7 (112)	Chr. 20	WGSP20_0090–WGSP20_0100	SSR	–	Rpm20-2	Field test (Japan)	5%	Taguchi-Shiobara et al. (2019)
Cercospora leaf blight/ purple seed stain	*Cercospora kikuchii*	F2 (148)	Chr. 18	Sat_308 and Satt594	SSR	6.6 cM and 11.6 cM	Rpss1	Field test	–	Jackson et al. (2008)
		PI 80837	Chr. 18	Satt115 Satt340	SSR	44.1 cM–46.8 cM	*Rpss*1	–	–	Albu et al. (2016)
Charcoal rot	*Macrophomina phaseolina*	Maturity group I-V (130)	Chr. 2	Satt644	SSR	38,221,027 bp	–	Isolate S8	12%	Ghorbanipour et al. (2019)
		USDA PI lines (459)	Chr. 4	ss715588228	SSR	4,307,731 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)

		F2:3 (140)	Chr. 5	–	SSR	25,338,390 bp	–	Isolate Conway	–	da Silva et al. 2019
		USDA PI lines (459)	Chr. 6	ss715593307	SSR	14,918,492 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)
		F2:3 (140)	Chr. 8	–	SSR	7,511,708 bp	–	Isolate Conway	–	da Silva et al. 2019
		USDA PI lines (459)	Chr. 9	ss715604575	SSR	45,369,206 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)
		Maturity group I-V (130)	Chr. 11	Satt359	SSR	32,411,307 bp	–	Isolate S8	–	Ghorbanipour et al. (2019)
		USDA PI lines (459)	Chr. 12	ss715613120	SSR	492,020 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)
		USDA PI lines (459)	Chr. 14	ss715618004	SSR	219,725 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)
		F2:3 (140)	Chr. 15	Gm15_01842053 and Gm15_03051337	SSR	1,842,060 bp	–	Isolate Conway	29.40%	da Silva et al. 2019
		F2:3 (140)	Chr. 16	Gm16_28961127 and Gm16_30493887	SSR	29,328,591–30,862,012 bp	–	Isolate Conway	25.40%	da Silva et al. (2019)
		USDA PI lines (459)	Chr. 18	ss715631726	SSR	51,751,797 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)
		Maturity group I-V (130)	Chr. 19	Sat_124	SSR	50,728,020 bp	–	Isolate S8	11%	Ghorbanipour et al. (2019)
		USDA PI lines (459)	Chr. 20	ss715638424	SSR	43,471,723 bp	–	Isolate from Iowa soybean field	–	Coser et al. (2017)

(continued)

Table 13.1 (continued)

Fungal diseases	Causal organism	Material used	Linkage groups/ chromosome number	Tightly linked markers/ flanking markers	Molecular marker used	Position of QTLs on chromosome (cM)/bp	Locus/ QTLs name	Isolates used	% Phenotypic variation	References
Phytopthora rot		L88-8470	Chr. 3	Satt159	SSR	1.2 cM	*Rps*1a	–	–	Gordon et al. (2007)
		L76-1988	Chr. 16	Sat_393	SSR	0.5 cM	*Rps*2	–	–	Lewers et al. (1999)
		L83-570	Chr. 13	Sat_317	SSR	0.1 cM	*Rps*3	–	–	Bernard et al. (1957)
		L852352	Chr. 16	Sat_004	SSR	4.3 cM	*Rps*4	–	–	Klos et al. (2000)
		L85-3059	Chr. 16				*Rps*5	–	–	Bachman et al. (2001)
		L89-1581	Chr. 16	Sct-187 Sat_372	SSR	0.3 cM–0.4 cM	*Rps*6	–	–	Mueller et al. (1978)
		L93-3258	Chr. 3	Satt152	SSR	6.7 cM	*Rps*7	–	–	Rincker et al. (2016)
		PI 399073	Chr. 13	Satt154	SSR	4.0 cM	*Rps*8	–	–	Paxton and Rogers (1974)

SSR simple sequence repeat, *cM* centi Morgan *bp* base pair

Table 13.2 List of some identified different fungal disease-resistant genotypes/lines/cultivars of soybean developed through various genomic approaches

Name of diseases	Genotypes/lines/cultivars/ resistant genes	Approaches	References
Soybean rust	SRE-Z-11A, SRE-Z-11B, SRE-Z-15A	Breeding	Langenbach et al. (2016)
	PI 441001	Breeding	Bromfield (1984)
	USP 97-08135	Breeding	Hartman et al. (2005)
	PI 416764, PI 462312, KS 1034	Breeding	McLean and Byth (1980)
	TGx 1993 4FN, TGx 1995 5FN, PI 594538A	Breeding	Cheng and Chan (1968)
	PI 594723, PI 594538A, PI 587880A, PI 230970, PI 459025A	Breeding	Hidayat and Somaatmadja (1977)
	PI 200492	Breeding	McLean and Byth (1980)
	PI 230970	Breeding	Cheng and Chan (1968)
	PI 462312	Breeding	Bromfield and Hartwig (1980)
	PI 459025B	Breeding	Hartwig (1986)
	PI 200456	Breeding	Wilcox et al. (1975)
	PI 567102B	Breeding	Li et al. (2012)
	PI 605823	Breeding	Alloatti et al. (2015)
	PI 594538A	Breeding	Calvo et al. (2008)
	70 differentially expressed proteins	Proteomics	Zhang et al. (2014)
Rhizoctonia root rot	AGS-129, G00056	Breeding	Kofsky et al. (2021)
	PI 442031	Breeding	Ishiwata and Furuya (2020)
Brown stem rot	PI 84946-2, PI 437833,PI 437970, L84-5873, and PI 86150	Breeding	Rincker et al. (2016)
Powdery mildew	BRS135 (cultivar)	Breeding	Gordon et al. (2007)
	PI 567301B	Breeding	Jun et al. (2012)
	PI 243540	Breeding	Kang and Mian (2010)
	ZH24	Breeding	Zhou et al. (2022)
	Djakl	Breeding	Dunn and Gaynor (2020)
Downy mildew	52 differentially expressed genes	Transcriptomics	Zhu et al. (2018)
Cercospora leaf blight/ purple seed stain	PI 417361, PI 504488, PI 88490, PI 346308, PI 416779, PI 417567, PI 381659, PI 417567, PI 407749	Breeding	Rahman et al. (2018)
	PI 80837	Breeding	Alloatti et al. (2015)

(continued)

Table 13.2 (continued)

Name of diseases	Genotypes/lines/cultivars/ resistant genes	Approaches	References
Phomopsis seed decay	PI 82,264		Walters and Caviness (1973)
	PI 181,550		Athow (1987)
	Delmar		Crittenden and Cole (1967)
	PI 200,501 and Arksoy		Ross (1986)
	PI 80,837, PI 417,479, and PI 360,841		Brown et al. (1987)
Anthracnose	EC 538828,EC 34372, EC 457254, AKSS 67, and Karune	Breeding	Nataraj et al. (2020)
	DSb 12	Breeding	Sajeesh et al. (2014)
Charcoal rot	JS 20-98, JS 20-34, MAUS 162	Breeding	Zhang et al. (2014)
	1219 DEGs	Transcriptomics	Deshmukh and Tiwari (2021)
Phytophthora rot	L88-8470	Breeding	Athow and Laviolette (1982)
	L76-1988	Breeding	Lewers et al. (1999)
	L83-570	Breeding	Bernard et al. (1957)
	L85-2352	Breeding	Klos et al. (2000)
	L85-3059	Breeding	Bachman et al. (2001)
	L89-1581	Breeding	Mueller et al. (1978)
	L93-3258	Breeding	Rincker et al. (2016)
	PI 399073	Breeding	Paxton and Rogers (1974)
	Zaoshu18	Breeding	Moellers et al. (2017)
	E00003	Breeding	Boudhrioua et al. (2020)
	46 differentially expressed proteins	Proteomics	Zhang et al. (2011)
	90 differentially accumulated metabolites	Metabolomics	Gordon et al. (2007)

further study, the genes conferring resistance to BSR were mapped on chromosome 16. These results led to the conclusion that soybean BSR resistance is caused by just one gene (McCabe and Graham 2020). Using mapping populations developed by crossing the resistant sources "Bell," PI 84946-2, PI 437833, PI 437970, L84-5873, and PI 86150 with either the susceptible cultivar Colfax or Century 84, three BSR resistant genes, Rbs1, Rbs2, and Rbs3, have been discovered and located on chromosome 16 (Rincker et al. 2016).

For powdery mildew (PMD), according to study, three alleles were present at the *Rmd* locus on the inheritance of host plant resistance to PMD, and they are *Rmd*, *Rmd-c*, and *rmd* (Lohnes and Bernard 1992). In the soybean cultivar PI 243540,

Kang and Mian (2010) found that a single dominant gene contributes to PMD resistance at all stages of soybean plant development. They discovered the gene *Rmd* PI 243540 from the cultivar PI 243540 to be situated between the SSR marker *Sat 224* and SNP marker *BARC-021875-04228* over the course of their investigation. The PMD resistance gene *Rmd* was linked to both markers at distances of 9.6 and 1.3 cM, respectively. The use of genetic markers for molecular characterization and diversity analysis among soybean genotypes for powdery mildew resistance has undergone a number of attempts. SSR analysis was utilized by DeMore et al. (2009) to find PMD resistance gene-linked markers in an F_2 population, derived from a cross between MGBR95-20937 IAC-Foscarin 31 and MGBR-46 EMBRAPA 48. In their investigation, two SSRs *Sat 366* and *Sat 393* were discovered and situated 9.41 cM and 12.45 cM away from PMD resistance genes, respectively. More recently, Zhou et al. (2022) examined adult plant resistance (APR) to PMD in soybean using recombinant inbred lines (RILs) populations created from crossing Zhonghuang 24 (ZH24) and Huaxia 3 (HX3). The outcomes showed that a single dominant locus controlled PMD resistance.

In case of FW, quantitative trait loci have significant role of controlling *Fusarium* wilt resistance. Studies reported four genes in a cluster with two duos in close proximity or two genes in a cluster with each gene exhibiting pleiotropy are responsible for triggering resistance to *Fusarium* wilt (Stephens et al. 1993). Another study under greenhouse conditions reported single dominant gene *Rfs1* may be responsible for controlling SDS resistance (Hnetkovsky et al. 1996). Similarly, a small number of significant QTLs govern some levels of resistance (Triwitayakorn et al. 2005). Consequently, a number of QTLs may also function as a qualitative locus (Anderson 2012). In same study, the candidate genes *QRfs1* and *QRfs2* are identified for two loci, and both offered resistance against root infection and leaf scorch, respectively (Anderson 2012). According to Fronza et al. (2004), QTLs on linkage group G conferred nine LGs (A2, C2, D2, F, G, I, J, L, and N) for resistance to root infection (*Rfs1).* According to Soybase (2010) report, more than 56 records of QTLs for *Fusarium* wilt in soybean have registered. Similarly, Fronza et al. (2004) reported multiple trait loci for resistance on chromosome number 18 (linkage group G), using four populations of almost isogenic lines and nine DNA markers. In linkage group G, it was hypothesized that three to four genes, namely, QRfs-, *QRfs1*-, *QRfs2*-, and *QRfs3*-rich islands, transfer resistance (Anderson et al. 2014). Similar study reported QTLs, namely, *BARC-Satt163*, *BARC-Satt080*, and *BARC-Satt307* for resistance that were identified on linkage groups G, N, and C2, respectively (Zou et al. 2005). Recombinant inbred lines with presentations that were environmentally stable and contained all three QTLs for resistance were considerably more resistant than other recombinant inbred lines. With a significant impact on the QTL *Rfs1*, the SSR marker *Satt183* has been found to provide resistance to SDS on molecular linkage group J (Sanitchon et al. 2004). The SSR marker *Satt183* has been found to provide SDS resistance (56% variance) on linkage group J. The SSR marker *Satt183* found to be most significant robust marker associated with QTL for *Rfs1* (Sanitchon et al. 2004).

For DM, 31 quantitative trait loci (QTL) were identified using five populations of RILs, derived from ('Natto-shoryu' × 'Tachinagaha' (NT), 'Nattoshoryu' × 'Suzumaru' (NS), 'Satonohohoemi' × 'Fukuibuki' (SF), 'Kinusayaka' × 'COL/Akita/2009/TARC/1' (KC), and 'YR-82' × 'Harosoy' (YH) grown across location and years (Taguchi-Shiobara et al. 2019).

For SWM, the use of molecular markers in conjunction with field studies has opened up new possibilities because they are independent of environmental factors. Recently Kandel et al. (2018) reported ten significant QTLs by single marker analysis that could be used as source of resistance to develop SWM-resistant cultivars. Another study by Moellers et al. (2017) reported 58 SNP-based loci had main effects, and some others had epistatic effects that were related to SWM resistance.

For PSD, employing progenies derived from the cross between resistant cultivar 'Taekwangkong' and the susceptible cultivar 'SS2-2' yielded two QTLs for PSD resistance under greenhouse condition (Sun et al. 2013).

For purple seed stain disease (PSS), the only partially resistant sources for PSS that have been reported are PI 80, 837, and SJ2 (Roy and Abney 1976; Ploper et al. 1992). According to Jackson et al. (2006), a single dominant gene *Rpss1*, on linkage group G, was shown to be responsible for resistance to *C. kikuchii* in the cultivar PI80837. In this study, the potential resistant gene was located between the flanking markers *Sat 308* and *Satt594* away from resistant genomic loci of 6.6 cM and 11.6 cM, respectively, on linkage group G. The use of such molecular markers in PSS resistance study will aid the advantages in marker-assisted breeding and selection (Jackson et al. 2008). Similarly, two SSR molecular markers *Satt115* and *Satt340* that are associated with resistance of purple seed stain have been identified in an association mapping study by evaluating two population derived from the cross of PI 80,837 (resistant) with AP 350 and MO/PSD-0259 (Alloatti et al. 2015).

For charcoal rot, in a recent study, a total of 140 $F_{2:3}$ lines derived from the cross PI 567562A (resistant) PI 567437 (susceptible) were genotyped, and QTL mapping analysis revealed one QTL on chromosome 15 and two QTLs on chromosome 16 for resistance to *M. phaseolina* (da Silva et al. 2019) (Table 13.1).

For *Phytophthora* root and stem rot of soybean, the mapping of molecular markers conferring resistance to the disease on different linkage groups has advanced since the introduction of the soybean linkage map. Several studies reported different genes responsible for resistance against of *P. sojae*. The resistant genes *Rps1*, *Rps2*, *Rps3*, *Rps4*, *Rps5*, *Rps6*, *Rps7*, and *Rps8* have been mapped on linkage groups N, J, F, G, G, G, N, and F, respectively, from different studies (Cregan et al. 1999; Sugimoto et al. 2007; Bernard and Cremeens 1981; Demirbas et al. 2001; Buzzell and Anderson 1981; Sugimoto et al. 2011). In addition, one RFLP marker, *pT-5*, found to be associated with the *Rps5* gene (Athow and Laviolette 1982). However, in addition to these resistant genes, soybean also has several partial resistance-related genes (Akem 1996). Some more genomic loci/QTLs have been reported by several researchers and documented which are elaborated in Table 13.1.

13.15 Genome-Wide Association Studies (GWAS) for Identification of Potential Candidate Gene(s) Associated with Resistance to Fungal Disease

With the help of molecular markers, GWAS have been successfully used to understand the genetic architecture of panels of germplasm lines and to pinpoint regions of the soybean genome linked to various disease resistance and also useful for marker-assisted selection in breeding programme. In an association study, 256 germplasm accessions from various countries were examined with several years across the location for their responses to soybean rust (SBR) along with susceptible controls and plant introductions (PIs) with *Rpp* genes at known loci (Walker et al. 2022). According to GWAS analysis, 31,114 SNPs were found, and 8 significant SNPs in 8 genomic areas on 7 chromosomes were found. Eight genomic areas, including previously unreported parts of chromosomes 1, 4, 6, 9, 13, and 15, as well as the *Rpp3* and *Rpp6* locus, were found to be related with SBR resistance on 7 chromosomes (Walker et al. 2022). Linkage map analysis with SSR markers revealed significant marker association to rust resistance in the linkage group (LG) C2 in cultivar FT 2 (Cregan et al. 1999). Another study reported a resistance gene situated in between the flanking marker *Satt134* and *Satt460* on LG-C2 and has been mapped in the cultivar Hyuuga (Monteros et al. 2007). Similarly, *Rpp3* was also located at the same location as reported by Hyten et al. (2007). On LG-G, between flanking markers *Sct 187* and *Sat 064*, 1 cM interval has been identified as the location of the *Rpp1* locus (Hyten et al. 2007). The *Rpp4* locus was located on chromosome 18 in linkage group G by 1.9 cM distance (Silva et al. 2008) and 2.8 cM (Garcia et al. 2008) from SSR marker *Satt288*, respectively. Meyer et al. (2009) reported *Rpp4C4* (PI 459025B) was highly expressed in the resistant genotype, while the expression of the other intrusive genes was essentially undetectable. According to the results of reverse transcription polymerase chain reaction sequencing, *Rpp4C4* considered to be the single candidate gene for *Rpp4C4*-mediated rust resistance. Molecular marker was used to increase the resistance against SBR of Vietnamese elite soybean cultivar (Khanh et al. 2013). In the same study, the *Rpp5* gene of SBR resistance was successfully incorporated into a popular Vietnamese soybean variety HL203 by using molecular markers in a backcross breeding technique. The *Rpp5* locus was discovered and to be located in the N linkage group between the flanking markers *Sat 275* and *Sat 280*. Further, based on the molecular information, Maphosa et al. (2012) asserted that the three resistance genes *Rpp2*, *Rpp3*, and *Rpp4* were effectively pyramided in pairwise combinations in the soybean F_2 generation.

For *Rhizoctonia* root rot, the development of resistant genotype was aided by marker-assisted selection in combination with phenotypic selection in later generations. According to an association study, the identified SSR markers, *Satt177* on linkage group A2, *Satt281* on linkage group C2, and *Satt245* on linkage group M, found to be associated with the resistance to *Rhizoctonia* root rot (Tomar et al. 2011). Utilizing these three SSR markers for further screening revealed the allelic variation for resistance (Sserunkuma 2016). In this study, five alleles were

amplified by each of the three markers. These markers amplified uncommon alleles and were found to be highly polymorphic.

For FW, an association mapping strategy by using 282 soybean lines along with 1536 SNP markers was used by Bao et al. (2015) to locate the loci that differ in SDS resistance and were employed, and two new loci were identified on chromosomes 3 and 18. The findings of these studies have accelerated the value of association mapping in locating significant loci in soybean.

For SWM, genome-wide association study revealed a novel QTL on chromosome 1 which is associated with SWM resistance (Boudhrioua et al. 2020).

For charcoal rot, a set of 459 different plant introductions from the USDA soybean germplasm core collection were screened in the field and greenhouse, and GWAS revealed some putative candidate genes led to new source of resistance (Coser et al. 2017). Similarly, in an association mapping study using 130 different soybean varieties and lines, *Sat_252*, *Satt359*, *Satt190*, *Sat_169*, *Sat_416*, and *Sat460* markers were identified that are associated with the charcoal rot disease (Ghorbanipour et al. 2019).

13.15.1 Virus-Induced Gene Silencing

The molecular identification of resistance in plants uses a virus-induced gene silencing approach and can be used as an alternative transgenic approach for disease resistance. Using this method, Meyer et al. (2009) discovered the *P. pachyrhizi*-resistant accession PI459025B in soybean. Additionally, Pedley et al. (2018) employed this method to characterize *Rpp1* in a recent study. According to this study, *Rpp1* was situated on chromosome 18 between the flanking markers *Sct 187* and *Sat 064*. According to results, the *Rpp1* gene was found to be distinct among other *Rpp* genes as it provides an immune response to isolates of avirulent *P. pachyrhizi* and is known to produce ULP1-NBSLRR protein which is essential for the immunological response.

13.15.2 Gene Pyramiding

There are reports on the use of gene pyramiding in soybean to create resistance to soybean rust. Combining *Rpp2*, *Rpp4*, and *Rpp5* in one soybean genotype demonstrated greater resistance to SBR (Lemos et al. 2011). Similarly, *Rpp2*, *Rpp3*, and *Rpp4* were combined with cumulative resistance using the gene pyramiding strategy (Pedley et al. 2018). The gene pyramiding strategy to promote disease resistance in the soybean crop is clearly reflected in these results (Chander et al. 2019). Recently, it has been discovered that using marker-assisted selection in conjunction with line breeding can help create soybean cultivars that have ASR resistance genes. It contributed to the introduction of two new soybean varieties in Paraguay, namely, JFNC 1 and JFNC 2. Three all-stage resistance (ASR) genes, *Rpp2*, *Rpp4*, and *Rpp5*, were present in both cultivars (Kato et al. 2022).

13.15.3 Transcriptomics

RNA-seq analysis identified 52 differentially expressed genes (DEGs) DEGs, demonstrating soybean downy mildew (SDM) defence-responsive genes (Dong et al. 2018). These discoveries have opened the door for additional functional evaluation of potential candidate genes, which can then be exploited to create superior soybean cultivars with improved SDM resistance.

The differential expression of WRKY transcription factors (TFs) in SDM-high resistant (HR) and SDM-high susceptible (HS) genotypes was examined in order to provide new insights regarding the defence mechanism of soybean response to Pm infection. In addition, a total of 16 WRKY TFs were discovered to be specific in response to fungal inoculation, and 22 WRKY TFs were shown to be differentially expressed in HR and HS genotypes. The yeast one-hybrid (Y1H) experiment was used to test the capacity of the GmWRKY31 to bind the cis-acting W-box element in the promoter region of the GmSAGT1 gene, whose higher transcriptional expression was associated with increased SDM resistance (Dong et al. 2018).

13.16 Conclusion and Future Perspectives

Throughout this content, it has been discussed how fungal diseases affect soybean production globally and how much yield is lost as a result. Diseases have been consistently documented to cause significant yearly output losses in the millions of dollars in the literature for decades (Savary et al. 2019; Bandara et al. 2020). The most efficient and long-lasting method for managing disease in soybeans worldwide is genetic resistance, which serves as a crucial tenet supporting the global soybean value chain and food security. Since the discovery and use of molecular markers are intimately related to resistance genes, public and private soybean breeding programmes have consistently introduced vertical resistance through MAS. Despite the fact that our evaluation identified hundreds of key genomic areas that confer resistance to numerous fungal diseases, there are still other aspects of genetic resistance that need to be clarified and actively researched.

The development of high-density molecular markers based on next-generation sequencing (NGS) was made possible by advances in genomics. These markers quickly advanced and were affordable for use in both public and private breeding programmes (Song et al. 2013, 2020). The soybean genome has many novel regions that are significantly associated with resistance to various pathogens, according to genome-wide studies. Traits that were previously thought to be qualitative in nature have somewhat changed into quantitative traits, with major and minor alleles having small effects contributing to the observed phenotypes.

However, a successful genetic transformation mechanism is necessary for the generation of CRISPR/Cas9 transformants, though. Unfortunately, soybeans are a difficult commodity for plant transformation technology, and the majority of GE research are still in the early stages of development. Although a few studies have successfully demonstrated the introduction of ribonucleoprotein complex (Cas12a-

RNP) in soybean protoplast (Kim et al. 2017), significant efforts may be required to incorporate these tools into soybean.

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