

Asian Soybean Rust Resistance: An Overview

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Abstract

Asian soybean rust caused by *Phakopsora pachyrhizi*, occurs in all soybean production regions of the world. Rust is the most destructive foliar disease of soybean and can cause yield losses of over 80%. To date, six race-specific resistance genes have been identified in plant introductions. However, races of *P. pachyrhizi* able to overcome the resistance conferred by these genes have evolved. Due to the limited availability of resistant varieties, fungicide application is the only management tool available for farmers, which significantly raises the production cost and the risk of environmental and human contamination. Thus, the transfer of resistance genes through classical breeding followed by marker-assisted selection allows the development of resistant varieties and their use as an efficient and cost-effective method for controlling soybean rust. The objective of this review is to provide a broad overview of the Asian soybean rust resistance, and a useful tool to guide future researches as well.

Keywords: Asian soybean rust; *Phakopsora pachyrhizi*; Soybean; *Glycine max*; Genetic resistance; *Rpp* gene.

Introduction

Soybean is one of the most important economic crops as a source of protein and oil. Biotic stresses such as pathogens, insects and weeds can cause negative impacts on its production. Among the diseases, Asian soybean rust caused by *Phakopsora pachyrhizi* (Sydow & Sydow) is the most destructive, and over 80% losses are common when environmental conditions are conducive to disease development (Figure 1). The infected plants undergo defoliation and early maturation in relation to non-infected plants, which causes reduction in weight and quality of the grains. Due to the limited availability of resistant varieties, the fungicide application is the only management tool available for farmers, which significantly raises the production cost and the risk of environmental and human contamination (Figure 2). In addition, some pathogen populations have shown increased tolerance to certain fungicides [1]. Thus, the search for resistant varieties is a critical strategy for economically and environmentally sustainable control.

Asian soybean rust is present in the most countries growing soybeans. The first report of rust epidemic was around 1914 in Southeast Asian countries. However, until the middle of last century the rust fungus reports occur only in East Asia and Australia. In 1970 it was reported in India [2], in 1976 in Puerto Rico [3] and in 1994 in Hawaii [4]. Probably the introduction of soybean rust in Africa in 1975 took place from urediniospores transported by air currents from west of India [5]. In 2001, the fungus was first detected in Brazil and Paraguay [6-9] and in 2003 in Argentina [7-10]. In the continental US, the pathogen was first reported in 2004 [11], and the rust epidemics have been limited to the southern states, with reduced movement toward producing regions of the North [12]. There are no reports of negative economic effects due to soybean rust in the United States, although there are up to 33% yield losses documented in experiments with no fungicide application [13]. In Argentina, soybean rust epidemics occur every year, usually late in the growing season and significant losses are limited to a few north locations of the country [10].

The main difference in the effect of the rust between Argentina, Brazil and the US is due to the unique environmental characteristics of Brazil that increase the rust risk. The environmental condition in Brazil is much more conducive to rust development than that in the

US and Argentina. Uredinia survive only in areas of southern US and northern Argentina due to low temperatures during the winter at high latitudes [14]. The high rainfall regime in the summer in Brazil increases substantially the risk of losses caused by Asian soybean rust when compared with Argentina and the US. In Brazil, the main factors for rust epidemics are a great soybean field extension and the continued monoculture, which favors urediniospores production and dissemination, and accelerates race shifts. The favorable climate, fungicide ineffectiveness, high plant density, sowing period from September to January, pathogen survival in volunteer plants of soybean and secondary hosts, are additional factors that decrease efficiency of control measures of the disease [15].

Sources of resistance have been reported and to date six dominant genes have been identified: *Rpp1* [16], *Rpp2* [17], *Rpp3* [18], *Rpp4* [19], *Rpp5* [20] and *Rpp6* [21]. These genes are not effective against all populations of *P. pachyrhizi* [22]. The transfer of resistance genes through classical breeding or through marker-assisted selection allow to develop resistant varieties and their use as an efficient and cost-effective method for soybean rust control. Therefore, studies on the inheritance of resistance in soybean to *P. pachyrhizi* are very important for varieties development. Many breeding programs have used some identified resistance genes with success, although the diversity of rust isolates present in the nature is a continuous challenge. The objective of this review is to provide a broad overview of the soybean rust resistance, and a useful tool to guide future researches as well.

The Host

Glycine max (L.) Merr, originally from northeastern China, has

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Figure 1: Soybean crop during the pod fill stage severely infected by *Phakopsora pachyrhizi* (A), and the same crop field a week later, presenting intense defoliation (B). Location of pictures: Campo Verde city, Mato Grosso state, Brazil, February-2010 (W55°16'30"/S15°35'6").



Figure 2: Aspect of soybean canopy with (A) and without (B) fungicide application to control *Phakopsora pachyrhizi*. It is noticeable the high amount of leaves above ground where the fungicide was not applied. Location of pictures: Campo Verde city, Mato Grosso state, Brazil, February-2010 (W55°16'30"/S15°35'6").

emerged as a domesticated specie by the eleventh century BC. Soybean belongs to the *Magnoliophyta* Division, *Magnoliopsida* Class, *Fabales* Order, *Fabaceae* Family, *Faboideae* Subfamily, *Glycine* genus [23]. *Glycine max* is a domesticated specie over the 18 known species of the genus *Glycine*, and its diploid number of chromosomes is forty. Soybean is cultivated in temperate and subtropical regions as oil and protein source.

Soybean is a legume species that has a great adaptability to different latitudes, climate and soil conditions. Their grains are important source of protein and vegetable oil for human and animal diet. Soybeans contain about 20% oil and 40% protein in the form of essential amino acids. The oil is used mainly for human consumption and as an industrial raw material, and the beans after oil extraction are used for animal feed [24].

In Brazil, the introduction of soybeans in a commercial scale occurred in the 1930's, using American varieties originally adapted to cultivation in the southern US. However, the large increase in area and production of soybeans took place in the 80's, when the success in breeding allowed the expansion of culture to the Cerrado areas [25]. From an area equal to 1.29 million hectares and a production of 2.2 million tons in 1980, the region began to grow to 5.08 million hectares, with a production of 10.3 million tons in 1989. Currently soybean is one of the most important crops produced in Brazil and accounts for about 46% of the grain produced in the country. In the 2014-2015 season, the Brazilian soybean production reached 96 million tons, an increase of

11.5% over the previous season [26]. Exports by the production chain of the oilseed reached nearly US \$31 billion and represented 31% of total exports from Brazil agribusiness [27]. Worldwide soy is cultivated in about 118 million hectares, with production estimated at 315 million tons [28]. Brazil is the second largest producer and accounts for about 30% of world production of oilseeds. The US is the first producer, with an output of 108 million tons in an area of 33.6 million hectares, representing 34% of total world production.

The Pathogen

The fungus that causes Asian soybean rust belongs to the *Fungi* Kingdom, *Basidiomycetes* Class, *Uredinales* Order, *Phakopsoraceae* Family, *Phakopsora pachyrhizi* specie. To date, the described fungus stages were uredinial, teleomorphic and basidial. The aecial stages have not been reported yet [29]. Like all fungi of the group, *P. pachyrhizi* is a biotrophic pathogen that requires a living host to grow and survive.

Naturally, *P. pachyrhizi* infects 31 species in 17 genera of legumes [30]. The main hosts are *Glycine max*, *G. soja*, *Pachyrhizus erosus*, *Pueraria lobata* and *Vigna unguiculata*. According to Yeh et al. [31] and Bromfield [2] the fungus could attack up to 87 and 95 hosts, respectively.

Symptoms caused by soybean rust are different from those caused by other types of rusts. The uredinia are the fruiting bodies, which produce the urediniospores released through an ostiole. The symptoms of soybean rust are characterized by small brownish to dark brown lesions, with one or more uredinia mainly on the bottom side of the plant leaflets. The lesions tend to be angular with 2 to 5 mm in diameter. May also occur on petioles, pods and stems. The color of the lesions varies with age and with the interaction between the host genotype and the pathogen isolate. The new lesions are initially light brown, becoming darker with the age [32].

The urediniospores are the primary inoculum of soybean rust. These are asexual, small, lightweight spores, which are removed from uredinia when the infected leaf surface is dry. After removal, the air currents can transport the spores over long distances, which explain its spread from one field to another. In the presence of water and temperature between 21 and 25°C, the urediniospores deposited on the host leaf surface begins the germination process and infection [32]. The penetration of the parasite occurs directly through the cuticle and epidermal cell wall of the host. The direct penetration rather than through the stomata, is a characteristic that differentiates *P. pachyrhizi* from other rusts fungi. The colonization begins shortly after penetration, the primary branching hyphae gives rise to formation of a dense mycelium filling the intercellular spaces and inserting haustoria in the mesophyll and epidermal cells. The fungus reproduction begins approximately at eight days after infection, and its first evidence is the hyphae aggregation forming the uredinia primordium. Uredinia has light brown to red brown appearance. In about 3 to 4 days, it starts the production of urediniospores. According Alves et al. [33], the latency period length varies with the temperature, being shorter for temperatures around 23°C, in agreement with previous studies of Melching et al. [34].

Pathogen Variability

Regarding the variability of the pathogen, the asexual production of urediniospores suggests a low genetic diversity, with a small number of clonal lineages and recurrent events [35]. In general, data from different molecular markers shows that the genetic diversity in *P. pachyrhizi* is low between large geographic areas and high when considering the variation within local populations. The high capacity of dispersion over long distances enables virulent genotypes of *P. pachyrhizi* move

quickly between different populations [36]. In Nigeria, *P. pachyrhizi* has approximately 90% of their genetic diversity within the soybean fields, with little diversity distributed among the fields [35,37-39].

On the other hand, the presence of resistance genes in the host did not lead to reduction of frequency of virulence genes in pathogen populations. The probable explanation for this is that asexual way is not the unique reproduction mechanism of *P. pachyrhizi*. The anastomosis of the germ tube and hyphae, and the possibility of parasexual cycle [40,41], contribute to increase the genetic diversity of the pathogen. Thus, breeding for soybean rust resistance, should consider the likelihood of conversion of avirulent to virulent isolates. In addition, the local development of varieties containing only one resistance gene exposes it to these pathogen populations containing the full range of genetic variation present at regional level.

The size of the genome of *P. pachyrhizi* was estimated between 300 and 950 Mb depending on the analysis method used [42]. Igor Grigoriev provided similar information, suggesting a genome greater than 850 Mb [43]. However, due to ignorance about the degree of heterozygosity between or within the dikaryotic fungus genome, this size can be overrated. It is possible that the genome of *P. pachyrhizi* has sustained a high level of gene duplication during evolution with an important activity of transposable elements, which can explain the enormous size of the estimated genome for that species.

Resistance

The plants generally have two main defense mechanisms against pathogens, race-specific and race-nonspecific resistance. Race-specific resistance is controlled by single R genes and generally less durable. In contrast, race-nonspecific resistance is a polygenic trait and more durable [44].

During the early phases of infection, all pathogen classes deliver effector molecules into the plant cell to enhance microbial fitness. In response, plants have evolved a dynamic immune system to defend themselves against plant pathogens. The perception of pathogen associated molecular patterns (PAMPs) by plant extracellular pathogen recognition receptors (PRRs) is the first stage of plant basal defenses. The PAMPs are essential structures or components that are conserved throughout the whole classes of pathogens, including oligogalacturonides, ergosterol, bacterial flagelin, xylanases, cold-shock protein and lipopolysaccharides. Such response is referred to as PAMP-triggered immunity (PTI) and activates a myriad of process, including mitogen-activated protein kinase (MAPK) cascades, production of reactive oxygen species (ROS). According to the zig-zag model, proposed by Jones & Dangl [45], there are numerous PRRs in plants to recognize PAMPs and to initiate basal defense responses. However, some pathogens can secrete effectors to evade recognition by plant PRRs and to promote pathogen growth and virulence, called effector-triggered susceptibility (ETS). In response to ETS, host plants trigger R proteins to interact directly or indirectly with pathogen effectors or avirulence (Avr) proteins, and induce a stronger defense response, referred to as effector-triggered immunity (ETI) [44]. ETI triggers salicylic acid (SA) biosynthesis and signaling, leading to local and systemic acquired resistance (SAR) against biotrophic pathogens.

According to Van de Mortel [46], a biphasic gene response to *P. pachyrhizi* infection was seen in resistant and susceptible genotypes. The early transcriptional response observed in susceptible and resistant plants might represent a general response of soybean to the non-specific recognition of any pathogen, presumably by interaction with PAMPs. By contrast, the second likely response relates to R gene

detection of *P. pachyrhizi*, typically mediated by ETI pathway. Typically ETI culminates in hypersensitive cell death (HR) that retard pathogen growth, particularly in interactions involving haustorial parasites.

There are three types of soybean reaction to infection by *P. pachyrhizi*, which are associated with qualitative resistance genes (Figure 3). The first type is the immunity (IM) or complete resistance, without the presence of reproductive structures, such as uredinia or uredinospores. The second type is incomplete (or partial) resistance, which leads to the development of red brown (RB) lesions. According Parlevliet [47] and Ribeiro Do Vale [48], incomplete resistance allows some growth or reproduction of the pathogen in the host tissues. Finally, the tan colored lesions (TAN), indicative of susceptibility [49]. Either IM or RB reaction are initiated with the early perception of the pathogen avirulence proteins by plant R proteins, according the classical gene-for-gene resistance theory [50]. This incompatible interaction is followed by a localized programmed cell death, called hypersensitive response (HR), to limit the pathogen growth. On the other hand, TAN reaction means a compatible interaction, without the perception of the pathogen by the plant. Studies of host-pathogen interaction show that RB lesions tend to have longer latency period, smaller and fewer uredinia than the TAN lesions. Although RB type injury can also vary in color from light red to dark red, and sometimes have larger lesions than the TAN type. These observations suggest that the color of the lesions may not be a reliable indicator of susceptibility or resistance [2,51,52].

The wide variation in the type of reaction, color and intensity of sporulation observed in the field can be a difficulty factor in genotype characterization studies [53]. Although the type of lesions are widely used in detached leaves and seedlings inoculations studies, the appearance of rust lesions in adult plants is often different from that originally reported by Bromfield & Hartwig [17]. Besides the influence of the host genotype and levels of pathogen sporulation, the color of the lesions varies with their age, especially in the field where the infection events are continuous. In the work of Miles et al. [49], the IM and RB reactions were considered the unique forms of resistance expression. However, partial resistance also occurred in the interaction between *P. pachyrhizi* and soybeans, since differences were observed between the lines with TAN lesions. The variation in the number of uredinia is one of the parameters to be considered in the differentiation of genotypes with partial resistance and is inversely correlated with yield [2,51,54,55].

The incompatible interaction expressed as IM phenotype is mediated by *Rpp1* gene [56], while the resistance conferred by the other genes is characterized by the formation of RB lesions, and limited

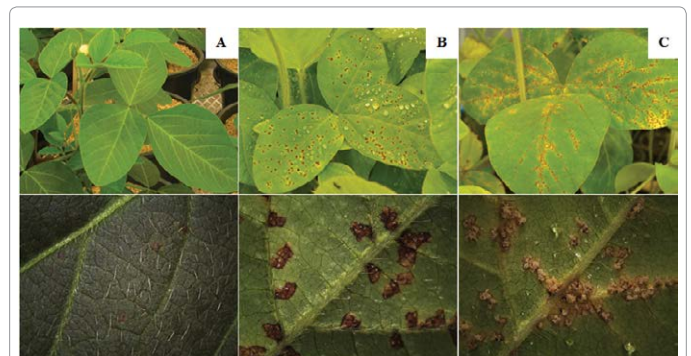


Figure 3: The three types of soybean responses to infection by *Phakopsora pachyrhizi*: Immunity or complete resistance (A), RB type lesions or incomplete resistance (B) and TAN type lesions indicative of susceptibility (C).

growth and sporulation [51]. Soybean varieties with partial resistance allow the development of a few lesions and limited sporulation during the growing season [55]. Race-nonspecific resistance has also been observed. It acts by reducing the amount and rate of rust development, even if the type of infection is similar to that produced in highly susceptible varieties [2]. This type of resistance can be effective against most of the pathogen population, being more useful than the race-specific resistance. The difficulties associated with race-specific and race-nonspecific resistance have led to the search for new types of resistance such as tolerance [57]. Tolerance is the relative ability of soybeans to produce under the stress caused by rust. This type has been used to minimize yield losses associated with soybean rust [58,59].

The first report of race-specific resistance in soybean to *P. pachyrhizi* occurred in the 70's [60]. In 1975, the US Department of Agriculture (USDA), in cooperation with the Asian Vegetable Research and Development Center (AVRDC), tested approximately 16,000 genotypes against a mixture of five isolates. They found 805 accessions with low severity or RB lesions, which were considered as a potential resistance sources. However, it was not possible to determine whether these resistant genotypes represented new sources of resistance genes or just alternative sources for the same four genes already known at that time. This is still an important question to be answered about the new resistance sources identified in recent studies. Studies conducted in Australia by McLean [61] and McLean & Byth [16] found that a single dominant gene controls resistance of PI 200492 to certain races of *P. pachyrhizi*. At the same time, Singh & Thapliyal [62] reported that PI 462312 also carries a single dominant gene.

The first report indicating the occurrence of variability in the pathogen population was in 1980. In tests carried out by Bromfield & Hartwig [17], the PI 200492 and PI 462312 showed susceptibility to an isolate from Taiwan and resistance to an isolate from India. On the other hand, PI 230970 and PI 230971 had just RB lesions when inoculated with isolates from Australia, India, Taiwan and Philippines. With the same set of varieties, Hartwig & Bromfield [18] indicated that *Rpp1*, *Rpp2* and *Rpp3* genes not had the same spectrum of reaction against different rust isolates. While *Rpp1* and *Rpp3* genes conferred resistance only to an isolate from India, the *Rpp2* gene conferred resistance to isolates from India and Taiwan. When combined, the *Rpp1* and *Rpp3* genes conferred intermediate reaction between immunity and incomplete resistance. With these results, they concluded that PI 200492, PI 230970 and PI 462312 carry different dominant resistance genes located on different locus. According to the authors', the isolates able to attack the PI 200492 have become prevalent in the fungal population in Taiwan. Since PI 230971 and PI 230971 were resistant to four isolates, these varieties become fundamentally importance for the breeding programs at that time. During this period, the Asian Vegetable Research and Development Center (AVRDC) recommended the varieties PI 200492, PI 462312, PI 230970, PI 230971, PI 293871A, PI 239871B, PI 459024, PI 459025, Tainung 4, Taita Kaohsiung No.5 and Wayne, as a set for rust isolates differentiation [63].

Between 2000 and 2010, with the incorporation of *Rpp1* and *Rpp2* genes in the Asian breeding programs, the resistance conferred by these genes was quickly defeated [64]. Similarly, the effectiveness of these genes was lost only two years after soybean rust had been reported in Brazil [7].

According to studies from Miles et al. [49], the rust severity was not related to the type of lesions. Among the varieties with RB lesions, PI 561356 and PI 594538A showed lower levels of severity than PI 230970, PI 423972 and PI 459025B, who were the varieties with higher rates of

severity. On the other hand, among the varieties with TAN lesions and low levels of severity, PI 548463, PI 548484 and PI 549017 produced similar numbers and sizes of uredinia as from the susceptible variety Williams, indicating no relationship between the severity of the disease and the reproduction of the fungus.

Walker et al. [53], studying the field reaction of reported resistance genes in the southern United States, noted that *Rpp1* gene, present in PI 200492, and *Rpp6* gene, in PI 567102B, still conferred high levels of resistance. The varieties carrying *Rpp2*, *Rpp3*, *Rpp4* or *Rpp5* showed incomplete resistance and moderate levels of rust development when compared to susceptible controls. Although *Rpp2*, *Rpp3* and *Rpp4* genes have conditioned lower levels of resistance, this moderate resistance was stable at the different sites. Regarding the resistant varieties reported in the literature, but whose genes have not been mapped yet, the resistance in PI 567104B was similar of PI 567102B (*Rpp6*). Coincidentally both these varieties came from the same research center in Indonesia, yet the relationship between them is unknown. PI 416826A, PI 417125, PI 567024, PI 567025A, PI 567034 and PI 605823 also showed good resistance to most field isolates of *P. pachyrhizi*. The authors reported no immunity among the varieties tested and a low frequency of sporulating uredinia, even in highly resistant accessions, such as PI 200492 and PI 567102B. This indicates that in some regions, the fungal population is heterogeneous and contains one or more isolates able to overcome the resistance conferred by *Rpp1* and *Rpp6* genes. They also observed that moderate resistance conditioned by certain genes make it difficult to distinguish between race-specific and race-nonspecific resistance. An example is the case of PI 506764 (Hyyuga), carrying resistance genes *Rpp3* and *Rpp5* [65], but whose classification field was the same of PI 200492 (*Rpp1*) and PI 462312 [*Rpp3*].

Recently, Chen et al. [66] identified a new resistance allele on chromosome 18, where several alleles of *Rpp1* family have been identified. According to the authors, the identified locus differs from those previously identified in PI 200492 [67], PI 587886 and PI 587880A [68], PI 561356 [69] and PI 594538A [70]. According to Ray et al. [68], the resistance observed in PI 587880A and PI 587886 is controlled by an *Rpp1* allele with incomplete dominance. The presence of RB lesions in heterozygous individuals (*Rpp1/rpp1*), and IM or TAN lesions in homozygous (*Rpp1/Rpp1* or *rpp1/rpp1*) individuals are indicative of incomplete resistance conferred by *Rpp1*. Furthermore, the presence of three classes of lesions (IM, RB and TAN) in mapping populations is also an indicative of incomplete dominance. In this sense, Smith et al. [71] also conclude that the gene action of an *Rpp1* allele mapped in PI 587905 can be dominant or incompletely dominant, depending on the age or development stage of plants. So far, resistance alleles to soybean rust were identified and mapped in different loci of chromosomes 3, 6, 16 and 18 (Table 1).

Resistant varieties are useful tools to reduce the economic losses caused by Asian soybean rust. However, varieties with a single resistance gene tend to be readily broken [2,64,72]. The effectiveness of the race-specific resistance is often of short duration, especially in biotrophic pathogens like *P. pachyrhizi*, with high virulence variability. Varieties with two pyramided resistance genes tend to express greater durability of resistance than varieties with a single gene [73,74]. Lemos et al. [75] and Yamanaka et al. [76] reported that a variety with three resistance genes (*Rpp2*, *Rpp4* and *Rpp5*) showed significantly higher resistance than its ancestor, showing that pyramiding strategy is effective to increase the rust resistance.

The main factors that determine which strategies and methods should be employed in rust resistance breeding programs include

VARIETY	GENE	CHROMOSOME (LINKAGE GROUP)	MOLECULAR MARKER	REFERENCE
PI 200492	<i>Rpp1</i>	18 (G)	Satt191 - Sat064	McLean & Byth, 1980; Hyten et al., 2007.
PI587866	<i>Rpp1</i>	18 (G)	Satt191 - Sat064	Ray et al., 2009.
PI 587880A	<i>Rpp1</i>	18 (G)	Satt191 - Sat372	Ray et al., 2009.
PI 561356	<i>Rpp1</i>	18 (G)	SSSR50 - SSSR1859	Kim et al., 2012.
PI 587905	<i>Rpp1</i>	18 (G)	Sat064 - SSR66/Satt191 - Sat_372	Hossain et al., 2014; Smith et al., 2015.
PI 594760B	<i>Rpp1</i>	18 (G)	Sat117 - Sct187	Garcia et al., 2011.
PI 594767A	<i>Rpp1</i>	18 (G)	Sat064 - Satt191	Hossain et al., 2014.
PI 594538A	<i>Rpp1-b</i>	18 (G)	Sat064 - Sat372	Chakraborty et al., 2009.
PI 230970	<i>Rpp2</i>	16 (J)	Satt255 - Satt620	Hartwig & Bromfield, 1980; Silva et al., 2008.
PI 224270	<i>rpp2</i>	16 (J)	Satt215 - Sat361	Garcia et al., 2008.
PI 462312	<i>Rpp3</i>	6 (C2)	Satt460 - Sat263	Hartwig & Bromfield, 1983; Hyten et al., 2009.
PI 416764	<i>Rpp3</i>	6 (C2)	Satt263 - Satt307	Hossain et al., 2014.
PI 567099	<i>rpp3</i>	6 (C2)	Satt460 - Staga001	Ray et al., 2011.
PI 506764	<i>Rpp3/Rpp5</i>	6/3 (C2/N)	Satt460 - Sat263/Satt275 - Sat280	Monteros et al., 2007; Kendrick et al., 2011.
PI 200487	<i>Rpp3/Rpp5</i>	6/3 (C2/N)	Satt460 - Sat263/Satt275 - Sat280	Garcia et al., 2008; Kendrick et al., 2011.
PI 471904	<i>Rpp3/Rpp5</i>	6/3 (C2/N)	Satt460 - Sat263/Satt275 - Sat280	Kendrick et al., 2011.
PI 459025	<i>Rpp4</i>	18 (G)	Satt288 - AF162283	Hartwig, 1986; Silva et al., 2008.
PI 459025B	<i>Rpp4</i>	18 (G)	Satt288 - AF162283	Silva et al., 2008.
PI 200456	<i>rpp5</i>	3 (N)	Satt275 - Sat280	Garcia et al., 2008.
PI 200526	<i>Rpp5</i>	3 (N)	Satt275 - Sat280	Garcia et al., 2008.
PI 567102B	<i>Rpp6</i>	18 (G)	Satt324 - Satt394	Li et al., 2012.

Table 1: Soybean varieties used as a source of resistance to *Phakopsora pachyrhizi* and the molecular information of the mapped genes in soybean genome.

the genetic distance between the varieties to be improved and the resistance donor, the screening methods, the genetics of resistance and the number of characteristics to be improved [77]. The soybean breeding for rust resistance has focused on qualitative genes, which have stability limitations in the control of diseases. Furthermore, the presence of multiple virulence genes in the pathogen population, and the lack of multiple resistance genes in the host gives a competitive advantage to rust, and makes the race-specific resistance less effective and short as compared to race-nonspecific resistance [78]. Ribeiro et al. [79] suggest that breeding for race-nonspecific resistance is more effective to achieve durable resistance. Currently, with the launch of new resistant varieties, it is expected the appearance of new rust races. Therefore, it is crucial to make continued efforts on identifying newer genes for resistance, developing and refining molecular markers for use in breeding programs for Asian soybean resistance.

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