CURRENT STATUS OF SOYBEAN RUST
(PHAKOPSORA PACHYRHIZI) - A REVIEW
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ABSTRACT
Soybean rust, incited by Phakopsora pachyrhizi, is the most serious soybean disease of economic importance in the Orient. It is known to occur in Asia, and in many countries of Europe, Africa, Australia and America. However, the distribution of rust in North America and Europe is restricted to small areas. Yield losses ranging from 10 to 80% are reported from different parts of the world. Phakopsora meloboeae also causes soybean rust in south of North America, Caribbean area and in south America. Epidemiological studies have been made on uredospore production, germination and infection process. Very meagre information is available on the occurrence and germination of telial stage and their role in life cycle of rust and in the epidemiology of the disease is still unknown. P. pachyrhizi has an extremely wide host range. Though, many fungicides have been reported effective against the rust but triazole derivatives themselves or in combination with other fungicides have proved their worth at large. A large number of soybean varieties and accessions have been screened and resistant sources have been identified across the various countries. Of late, DNA marker in combination with PCR have enabled marker-assisted selections to become a future practical method. In the present paper, the available information on distribution, magnitude, epidemiology and management of soybean rust is reviewed.

Soybean is an ancient crop with varied food and industrial uses. It is a promising source of vegetable oil, nutraceuticals and protein. Soybean cultivation is increasing consistently in the World. In the quest of higher and stable yield as well as quality produce the crop has received attention of a large number of researchers from diverse disciplines. Among the various biotic and abiotic factors leading to low productivity in soybean, diseases are the major factors. Almost all parts of a soybean plant are attacked by the diseases and more than 100 pathogens have so far been recorded on this crop. Out of them, only 35 pathogens are known to cause economic damage (Sinclair, 1983). Rust has been one of the major diseases taking a heavy toll. It is known to occur in Asia, and in many countries of Europe, Africa, Australia and America. Of late, rust has become a serious and economically important disease of soybean world wide causing 20-100% yield losses in India. The disease was earlier considered as of minor importance and was known to occur in low hills of UP, West Bengal and North East regions. After 1993 the rust scenario took a serious turn and every year started appearing in newer areas. Now rust is known to occur in almost all the states of NE region, Madhya Pradesh, Maharashtra, Karnataka, Andhra Pradesh. Tamil Nadu, Kerala, Rajasthan and Himachal Pradesh and posing a serious threat to soybean cultivation. To overcome the threat of rust and to minimize the accruing yield losses it becomes imperative to know more about the disease and its eco-friendly integrated management. In the present review an attempt has been made to compile and synthesize the important information on geographical distribution, yield losses, causal organism, epidemiology, host range, disease management, rust resistance gene and biological control and to reveal the gaps in knowledge on various aspects of this serious malady of soybean.

Geographical distribution: Rust disease was first recorded in Japan in the year 1902 by Nakanishiki who identified the fungus
as *Uredo sojae* (Cit. Yang, 1977). Later, Hennings (1903) confirmed the fungus as *Uredo sojae* on leaves of wild grown soybean (*Glycine soja*) collected from Tosa Province of Japan by Yoshinaga. However, the rust fungus in the western hemisphere on *Vigna* was reported in 1891, which was referred as *Uromyces vignae* (cited by Sinclair and Backman, 1989).

Report of soybean rust from India came as early as in 1906 from Poona on a host, which was wrongly identified as *Glycine hispida* (Sydow et al., 1906). Later, Butler (1953) clarified that the host was actually a species of *Mucuna* and therefore, the report of rust on *Glycine* sp. was incorrect. Sathe (1972) has re-examined the above specimen and concluded that the rust pathogen on *Mucuna* sp. was actually *Uromyces mucunae* and *U. sojae*. Thus, the first report of the occurrence of soybean rust in India was from Madras by Ramakrishnan (1951) who named it as *Uromyces sojae*.

The name *Phakopsora sojae* was first given by Sawada as early as in 1933, which was subsequently renamed as *P. pachyrhizi* by Hiratsuka (1935, 1936). The first authentic report of rust in India was available from Pantnagar in 1970 (Sarkhoy et al., 1972). Later on rust was seen in low hills of UP and Kalyani in Bengal (Singh and Thapliyal, 1977). Till 1974 the rust remained restricted in and around Pantnagar and subsequently disappeared from India. However, in 1980, after a lapse of almost 5 years the rust resurged in high altitude areas of Meghalaya and later from Assam plains in North east regions of India (Maiti et al., 1981; Sharma, 1990). Since then rust is occurring in almost all Northeastern hills region in epiphytotic form. Till 1993, the rust remained confined to this region and afterwards it spread to other soybean production areas. Till date rust has been observed in the districts of Betul, Chhindwada, Seoni, Hoshangabad, Narsingpur, Jabalpur, Indore, Bhopal, Sehore, Dewas, Ujjain, Dhar, Vidisha etc. of Madhya Pradesh, Sangli, Kolhapur, Satara, Beed, Latur and Nagpur in Maharashtra, Dharwad, Belgaum and Bijapur in Karnataka, Jhalawar and Kota in Rajasthan and also in certain pockets in Andhra Pradesh, Tamil Nadu, Kerala and Himachal Pradesh.

Its occurrence is more widespread in Asia, which incidentally is the probable centre of origin of its primary host, the soybean. Yet, the information so far available from the continent is far from adequate (Bronfield, 1977). Sydow and Sydow (1914) in Formosa (Taiwan); Baker (1914) and Reinking (1918, 1919 a, b) from Philippines; Reinking (1919 c); Tu (1932) from Southern China; Hiratsuka (1935 a) from Korea; Reichert (1939) from Palestine (Israel) and Ito (1941) and Park (1941) from Sri Lanka. The incidence of soybean rust was observed in Nepal in 1984 (Joshi, 1985). Tai (1947) described the morphology of uredospores and teliospores of *P. pachyrhizi* collected in western provinces of Yunnan and Huanan of China. In the neighbouring Thailand and Cambodia, however, the disease could be spotted more than a decade later (Lützenberger et al., 1962; Puckdeedindan, 1966; Sangawongse, 1973). Ovchinnikova (1970) detected the incidence of soybean rust in Soviet Far East. The disease has been widely reported from Japan. Hiratsuka and Yoshinaga (1935) and Hiratsuka and Sato (1953) have given the distribution of soybean rust in Tosa, Iyo, Chikugo provinces, Mt. Fuji area and Ryukyu Islands.

Outside Asia the disease has been recorded in countries of Europe, Africa, Australia and America, but many reports need further confirmation. It occurrence on soybean has also been reported from Tanzania and Zambia (Sinclair and Shurtleff, 1975), and from Zimbabwe in February 1998 (Levy, 2004).
*Phakopsora pachyrhizi* has been spotted on cowpea leaves too from other African countries like Ghana, Sierra Leone, Sudan, Tanzania and Uganda (Bromfield, 1977). On the African continent, the distribution of soybean rust was not well known before 1996, but since then it was found in Uganda, Kenya, and Rwanda, in Zambia and Zimbabwe during 1998, Nigeria in 1999, Mozambique in 2000, and South Africa in 2001 (Levy, 2003). Soybean rust, caused by *P. pachyrhizi* Syd., was first reported in South Africa in February, 2001 and its molecular identification confirmed that the South Africa race of soybean rust is *P. pachyrhizi*, and not the less virulent new world species, *P. meibomiae* (Pretorius et al., 2001).

In Australia *P. pachyrhizi* was first recorded on soybean in 1934 (Simmonds, 1966) but it assumed serious proportions only after 1971 (Keogh, 1974). In Lockyer valley in the South Queensland the disease occurred at high intensity (Kochman, 1977). However, in Americas, the first authentic record of *P. pachyrhizi* on soybean came from Puerto Rico in 1976 and later from Colombia (Sinclair and Backman, 1989). In Paraguay soybean rust was first detected in commercial field in March 2001, in the district of Pirapa, State of Itapua (Morel et al., 2004). Argentina confirmed its occurrence in early 2002 (Rossi, 2003 and Ivancovich, 2004). The pathogen was found later on in most of the soybean growing areas of Brazil in 2003 and appeared late in the season in Bolivia (Yorinori et al., 2003; Navarro et al., 2004). The only report of soybean rust rust from North America was from Georgia (USA) (Sinclair, 1977), which is yet to be confirmed. The only report of its occurrence in Europe is by De Guerpel (1942).

Rust fungus *P. pachyrhizi* occurs in most of the soybean growing areas of the globe except North America. Early reports showed that soybean rust isolate from the Western Hemisphere were different than Eastern Hemisphere. Based on the morphological characteristics the rust fungus in the Eastern Hemisphere was established as *P. pachyrhizi* and *P. meibomiae* in Western Hemisphere (Hartman et al., 2004).

The first molecular differentiation of these species was reported in 2002. However, the *P. pachyrhizi* was identified as more aggressive species in new geographical location beyond Asia and *P. meibomiae* as less virulent species in Western Hemisphere (Hartman et al., 2004). Rust caused by *P. pachyrhizi* is one of the major diseases of soybean in many Asian countries (Hartman et al., 2004). It is also severe in Brazil (Yorinori et al., 2003).

**Yield losses:** Rust in these years is recognized as the most destructive disease of soybean in both Eastern and Western Hemispheres (Sinclair, 1977). In the Eastern Hemisphere, it is a major constraint in soybean cultivation, affecting the crop both in the tropics and sub-tropics (Yang, 1977). Though reports of losses on account of rust are ranging from 10 to 90%, under congenial conditions it can cause substantial loss in yield (Gupta et al., 1999; Gupta, 2004). Yield losses reported in different countries have been given in the Table 1.

**Causal organism:** The nomenclature of soybean rust fungus has remained a subject of taxonomic controversy for almost three decades. The causal fungus was first reported as *U. vignae* in 1891 by Bresadola in the Western Hemisphere (cited by Sinclair and Backman, 1989) and as *U. sojae* by Hennings (1903) in the Eastern Hemisphere, obviously because of the close resemblance of its uredospore with those of *Uromyces*. In 1906, Sydow et al. recorded the occurrence of telial stage. Later, Sydow and Sydow (1914) recorded the pathogen on *Pachyrhizus erosus* (L.) Urban and named it as *P. pachyrhizi*. However, the fungus continued to be referred by different names even after. Arthur (1917)
transferred *U. vignae* of *Bresadola* to *P. vignae*. Sawada (1928) described soybean rust pathogen as *P. sojae*. The controversy seems to have ended after Hiratsuka (1932) adopted the name *P. pachyrhizi* for soybean rust fungus with *P. vignae* and *P. sojae* as synonyms.

The confusion relating to nomenclature could also be seen in Indian reports. The first report of soybean rust came from Poona in 1906, which was subsequently found incorrect due to wrong identification of the host as *Glycine hispida* (Sydow et al., 1906). Reports of Butler (1953) and Sathe (1972) later clarified that the reported fungus was actually *Uromyces mukuense* and the host was *Mucuna sp*. The next report on soybean rust in India was by Ramakrishnan (1951) who referred the fungus as *U. sojae* although the names *P. pachyrhizi* and *P. sojae* for soybean rust were already adopted by Hiratsuka (1932) and Sawada (1933) respectively.

Another species, *Phakopsora meibomiae* was found to be the causal organism of soybean rust in south of North America, Caribbean area and South America down to Argentina. Similarly, the causal agent of rust in Africa has not been described taxonomically. *P. pachyrhizi* is more aggressive than *P. meibomiae*.

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**Table 1. Yield loss due to rust in different parts of world**

<table>
<thead>
<tr>
<th>Country</th>
<th>Yield loss (%)</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Brazil</td>
<td>Up to 70</td>
<td>Silva et al. (2004)</td>
</tr>
<tr>
<td>India</td>
<td>10-90</td>
<td>Hartman et al. (2004); Gupta et al. (1999); Gupta (2004)</td>
</tr>
<tr>
<td>Japan</td>
<td>15-40 Up to 40</td>
<td>Bromfield (1984); Hartman et al. (2004)</td>
</tr>
<tr>
<td>Paraguay</td>
<td>&gt; 60</td>
<td>Morel et al. (2004)</td>
</tr>
<tr>
<td>South Africa</td>
<td>&gt; 60</td>
<td>Caldwell and McLaren (2004)</td>
</tr>
<tr>
<td>South China</td>
<td>10-50</td>
<td>Hartman et al. (2004)</td>
</tr>
<tr>
<td>Taiwan</td>
<td>20-30</td>
<td>Liu (1966)</td>
</tr>
<tr>
<td></td>
<td>70-80</td>
<td>Hsu and Wu (1968)</td>
</tr>
<tr>
<td></td>
<td>18-68</td>
<td>AVRDC (1976)</td>
</tr>
<tr>
<td></td>
<td>20-63</td>
<td>Yeh et al. (1981); Yeh (1983)</td>
</tr>
<tr>
<td></td>
<td>48-90</td>
<td>Tschanz and Tsai (1982)</td>
</tr>
<tr>
<td></td>
<td>20-80</td>
<td>Bromfield (1984)</td>
</tr>
<tr>
<td>Thailand</td>
<td>Upto 80</td>
<td>Poopongkul (2004)</td>
</tr>
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**Epidemiology:** Most of the earlier work on epidemiology and disease cycle of *P. pachyrhizi* was conducted in the orient. The temperature in the range of 15-25°C in the presence of free moisture on the leaf surface is essential for the germination of uredospore. The maximum infection was found at 20-25°C with 10-12 hours dew period (Kitani and Inoue, 1960; Hsu and Wu, 1968; Marchetti et al., 1975). Kochman (1979) also studied the effect of temperature on disease development and survival of the uredospores. He found that the maximum disease development occurred under a temperature regime of 17-27°C. Regarding survival of the uredospores, he concluded that the germination was significantly reduced when uredospores were exposed to temperature of 28.5-42.5°C for eight hours. Earlier, Ilag (1977) studied the effect of temperature and light periods on germination of uredospores and their survival. He found normal germination between 10-20°C, however, the germ tube remained shorter between 5-15°C and attained normal length between 15-30°C. The germ tube attained the maximum length under 15 hours. Darkness and nine hours light at 20°C. He also found that uredospores stored under different condition lost their germinability after 2-8 days. Kumar and Verma (1985) reported that...
uredospores stored in polythene bags at 18-25°C gradually lost their germinability in 15 days. However, when the spores were suspended in 1000-ppm sucrose solution, significant increase in germination percentage was observed. Melching et al. (1979) also compared the four rust isolates and found that all the four cultures produced lesions in seven days and secondary uredospores in nine days. However, the Indian culture produced more lesions per unit leaf area. The mean lesion areas on upper and lower leaf surfaces were similar in Indian, Taiwanese and Indonesian cultures but were smaller in the Australian culture. The number of uredia per lesion was also less in the Australian culture. However, the uredospores of all the cultures were similar in length and width as well as in germination potential. The virulence and aggressiveness of the four rust isolates on soybean were also compared by Bromfield et al. (1980).

Sharma (1990) studied the effect of host-age on the incidence and severity of rust and indicated that generally the disease appeared in the first half of September, irrespective of the host-age however, its appearance was delayed on August sown crop, which indicated that climatic factors like lesser rainfall might also determine its time of appearance. However, by early October, the disease attained higher severity on both in May and June sown crop.

The histology of the susceptible-pathogen relationship between the soybean plant and the rust fungus has been studied (Bonde et al., 1976; McLean, 1979 and McLean and Byth, 1981). Bonde et al. (1976) found that the uredospores started germinating within two hours after inoculation in a dew chamber at 20°C. Appressoria developed within two hours and the earliest penetration was observed in seven hours. The penetration occurred directly through the cuticle with the help of a trans-epidermal vesicle, which subsequently emerged from the invaded cell and formed intercellular mycelium in the mesophyll. Mclean and Byth (1981) observed that the number of uredospores germinated on leaf surface, number of appressoria formed and the number of host cells penetrated differed among susceptible and resistant soybean cultivars. Bonde and Brown (1980) examined isolate from Australia, India, Philippines, Taiwan and Puerto Rico on cv. Wayne. They observed that the isolates were indistinguishable in their pre- and post- penetration colonization phases and morphology of uredinia. The only difference could be observed in the germpore appearance, which may be due to thinner germpore plugs. Further, the isozymes of P. pachyrhizi isolates from Eastern and Western Hemisphere were compared (Bonde et al., 1988). Morphological difference between their anamorphic and teleomorphic stages of rust fungus was based on layering of telia and wall thickness of teleospores, which were studied by Ono et al. (1992). However, the molecular differentiation of the P. pachyrhizi and P. meibomiae was reported for the first time by Frederick et al. (2002), which was based on four and two sets of polymerase chain reaction respectively.

Most of the reports of soybean rust have been based on uredial stage of Phakopsora pachyrhizi, which has so far been recorded on 87 hosts. The occurrence of telial stage has been reported only on six hosts (Sydow and Sydow, 1914; Keogh, 1974, 1976; Ormines, 1978), which may be due to the absence of telial formation in the tropics. Telia on soybean cv. TK-5 were observed in the field in Taiwan (Yeh et al., 1982), while Kitani and Inoue (1960) observed teliospores on soybean in field in Japan. Occurrence of telia under greenhouse condition was reported by Bromfield (1977) and Hsu and Wu (1968). Yeh et al. (1981 a, b, 1982), Yeh (1982) described a method of induced formation of
teliospores on soybean and on other hosts and studied the factors affecting their formation. They subjected the inoculated plant to 12 hours photoperiod, 60-100% RH and 15-24°C temperature. In field, teliospores were produced only when the average daily value of minimum and maximum temperature was lower than 20°C and 29°C, respectively. Later, Poolpol and Pupipat (1985) during their study on the morphology and development of *P. pachyrhizi* could induce teliospore formation on 11 more legumes inoculated and incubated in an incubator programmed on a 12 h photoperiod at 6000 lux at 20°C to 22°C day cycle and 17°C night cycle under 100% RH for 60 days. However, Dufresne et al. (1987) found production of telia after 21 and 30 days in a Taiwan isolate and after 34 and 35 days in a Puerto Rico isolate at 10°C and 15°C incubation temperature respectively. The incubation period for telial formation also varied but little with varying light intensities.

Reports of the telial stage have contributed to a better understanding of the taxonomy of *P. pachyrhizi* but their role in the life cycle of the rust fungus and the epidemiology of the disease is still unknown, though attempts to induce their germination have succeeded (Koch and Hope, 1987). They could create appropriate conditions under which the teliospore could germinate and form basidia and basidiospores in the laboratory. Although, the inductive treatments closely resembled the environmental conditions in the field, which may suggest that the teliospores might be giving rise to basidiospores in nature also. Yet, information on the infectivity of basidiospores and the host(s) they can infect are still lacking, without which the life cycle of *Phakopsora pachyrhizi* will continue to remain incompletely understood.

**Host range:** *Phakopsora pachyrhizi* has an extremely wide host range since it infects large number of dicotyledonous plants such as the common bean (*Phaseolus vulgaris*), wild soybean (*Glycine ussuriensis*), and yam bean (*Pachyrhizus erosus*), both in the field and in the laboratory (Yang, 1977). Keogh (1974) has made a rather thorough study of the host plants indigenous to Australia. A list of the hosts for *P. pachyrhizi* Syd. is compiled (Keogh, 1974; Kitani and Inoue, 1960; Lin, 1966). However, the nine isolates were separated into six pathogenic groups differing mainly in their reaction types with and without sporulation or no infection on *Vigna unguiculata*, *P. vulgaris*, and *Pachyrhizus erosus*. Burdon and Speer (1984) established a set of differential Glycine hosts for the identification of *P. pachyrhizi* races. Further, Burdon (1987) found nine races *P. pachyrhizi* in two natural populations of *G. canescens*.

Legume species have been identified as host of *P. pachyrhizi* in Thailand were *Canavaria gladiata*, *Cajanus sp.*, *Centrosema pubescens*, *Pachyrhizus erosus*, *Vigna mungo*, *Dolichos lablab*, *Phaseolus aureus*, *P. lathyloides*, *Pueraria thunbergiana*, *Pisum sativum*, *Vigna sinensis* (Poolpolgul and Surin, 1980).

**Disease management:** Bulk of information available on management of rust disease pertains to fungicidal control only. Information on other aspects like epidemiology, economic threshold, durable varietal resistance, and life cycle of the fungus are meagre, which do not permit development of an effective integrated pest management system for the disease. Most of the reports on fungicidal control have come from Far East countries where the disease has been a problem for many years (Sinclair and Dhingra, 1975 and Sinclair, 1977). Efficacy of lime-sulphur sprays in the control of rust was studied by Kitani et al. (1960 a, b) in Japan.

The pre-disposing effects of host nutrition on rust intensity at 3 levels of NPK fertilizers was studied by Sharma (1990),
Sharma and Verma (1995) and Sharma et al. (1996). The lowest rust intensity was recorded at 0:60:20, 0:30:0 and 40:30:40 NPK doses, while the highest intensity of the disease was observed at 60:0:20 followed by 60:0:40. Hung and Liu (1961) and Wang (1961) found best control of the rust by 5 weekly sprays of Dithane M-45 or Dithane Z-78 + wettable sulphur 0-318 B or Bordeaux mixture in Taiwan. Jan and Wu (1971) studied the effect of different fungicidal chemicals on the germination of uredospores in vitro as well as in vivo. Sangawangse (1973) found that Plantvax and Plantvax+Benlate reduced the defoliation significantly, but without any increase in the yield. Thapliyal and Singh (1974) on the other hand, reported best control of rust associated with yield increase due to Dithane M-45 and Dithane Z-78 followed by benomyl and plantvax. Quebral (1977) tested the efficacy of five fungicides in Philippines and found the Dithane M-45 was the most effective in controlling the disease as well as increasing the yield during both wet and dry seasons. Maiti et al. (1982) found saprol and delan more effective than Dithane M-45 and RH 124. They found that delan and saprol gave almost three-fold increase in grain yield. Later, Maiti et al. (1983) reported saprol as the most effective fungicide both in respect to disease control as well as yield increase. Junqueira et al. (1984) have studied the efficacy of some protective and curative fungicides for control of rust. They found that triforine and triadimefon had better curative effects than benomyl, while the latter gave better protection. Among the non-systemic fungicides, maneb gave the best control. Efficacy of nine fungicides and two modes of treatments were studied by Sharma (1990) and Sharma et al. (1998) to control rust. They observed the seed treatment followed by two sprays of Tilt or seed treatment followed by three sprays of Dithane M-45 were the most effective against rust.

Ruengwiset and Poonpolgul (1999) observed that the rust infection declined significantly after the 2nd spray (first at 35 and 2nd at 42 days after planting) of chlorothalonil (35 g/20 lit water) followed by mancozeb 80% WP (30 g/20 lit water) as compared to control. Severity of the disease significantly decreased by hexaconazole (40cc/20 lit water) at the 2nd spray followed by mancozeb as compared to control. However, the last spray (63 DAP) of hexaconazole trend to decrease and still showed significant difference (Suraponchai and Poonpolgul, 2002). Poonpolgul (2004) recommended that in Thailand the disease can be controlled by the three application of mancozeb (80% WP) at 7 day interval when the first symptom was found on the unifoliate leaves at flowering stage or when there were continuous shower for 3 days or when there were dew on leaf surface at least 3-4 hours in the cool morning. Out of eleven fungicide, flusilazole / carbenilazim had less disease than azoxystrobin in South Africa (Preez and Caldwell, 2004). The mixture of triazole with strobilurin showed the best control of soybean rust (Quezzoni et al., 2004), while Jain et al. (2004) reported the new fungicide Opera, a combination of strobilurin and triazole – Pyraclostrobin + Epoxiconazole, 133 + 50 g/l respectively gave better results in controlling soybean rust in South Brazil. However, the fungicides tetraconazole and tebuconazole were similarly efficient in reducing rust; severity and soybean productivity loss in Brazil (Blum et al., 2004; Ito et al., 2004).

Some isolated attempts have been made to control the disease by adjusting the sowing time for avoidance of the rust. Desborough (1984) found that rust infection occurred late in two seasons but reduced the yield only in one season and that too in the new variety, Fitzroy. Ribeiro Do-vale et al. (1985 a) observed that the crop sown in October remained free from rust up to the 88
stage of growth and they concluded that the long cycle cultivars exhibited higher disease severity.

Rust resistance gene: Specific resistance to P. pachyrhizi has been attributed to four single dominant genes (Rpp1, Rpp2, Rpp3, and Rpp4). Hartwig (1995) summarized that the Rpp1 is having immune reaction when inoculated with a few isolate, including India 73-1. Inoculation of most rust isolates on Rpp1 or the other genes produces a resistant red-brown (RB) lesion with no or sparsely sporulating uredinia.

The inheritance of RB and TAN type reaction of four soybean crosses indicated inter-relationship between specific resistance genes in cultivars CM 60, PI 230970, PI 459025 and Wayne. Each cultivars carried a different single dominant gene for resistance and these genes were located at different loci. The genes for rust reaction were assigned to the four soybean parental types as followed: CM 60: Rpp1 Rpp1 rpp2 rpp2 rpp3 rpp3; PI 230970 and Wayne: rpp1 rpp1 Rpp2 Rpp2 rpp3 rpp3; PI 459025: rpp1 rpp1 rpp2 rpp2 Rpp3 Rpp3 (Poonpolgul, 1997).

A large number of soybean accessions have been screened in Taiwan, Thailand, Philippines, Indonesia and India (Tisselli et al., 1980). Cheng and Chan (1968) selected rust resistant soybean cultivar Tainung 3, which was found to perform better than the local checks. Sumarno and Sudjadi (1977) tested 50 soybean cultivars at 4 locations in Indonesia and found 8 varieties as moderately resistant. However, Lantican (1977) reported that the rust organism could continuously develop new strains leading to the breakdown of resistance rather quickly. It has suggest for greater collaborative effort to generate requisite information on: (i) prevalent strains of the rust organism in an area; (ii) identification of sources of genetic resistance against each strain and, (iii) a practical system for maintenance of each strain in pure culture. Lampang (1981) outlined the strategy for breeding soybean cultivars resistant to major diseases. McLean and Byth (1977) have also reported that the accessions PI 200492 and the cultivars derived from it (viz., Tainung 3, Tainung 4) gave immune reaction and accessions PI 224268 and PI 227687 also gave type I reaction while HY 2217 type R reaction both in field as well as glasshouse tests in Australia. Tschang et al. (1983) have indicated the possible existence of a rate reducing (RR) resistance, and they found variations in level of rate reducing resistance in 17 breeding lines, which were previously selected for their rust resistance. It appeared that quantitative characters control the RR resistance, since the variations were found to be continuous.
the investigation helped to identify several resistant and moderately resistant cultivars showing stability both over locations and years. Zambolin et al. (1983) and Riberio-Do-Vale et al. (1985 b) screened 34 Brazilian soybean cultivars. Tschang et al. (1985) evaluated five advanced cultivars previously selected for rust tolerance and yield potential at three different locations. Lack of durable resistance to rust has also led to mutation breeding.

As per current informations, the resistance against Phakopsora pachyrhizi is governed by four independent and dominant genes, which have been identified in PI 200492, PI 230970, PI 462312 (Ankur) and PI 459025 (Sinclair and Backman, 1989). Similarly, nine races have so far been identified at AVRDC, Taiwan. Genes for resistance have also been identified in some wild Glycine sp. from Australia.

In recent years, DNA markers in combination with PCR have enabled marker-assisted selection (MAS). RAPD marker located near the Rpp1 gene decides resistance to soybean rust. This RAPD marker would provide a means for screening for resistance gene at places where rust is not endemic (Vodkin, 1996).

Biological control: Biological controls offer great potential as an ideal component of IPM. There are many reports of using biological agents experimentally to control rust of soybean diseases. Some of the reported biological agents for soybean rust are Gliocladium roseum, Penicillium thomii and Trichotecium roseum (Kumar and Jha, 2002), Darluca filum, Tuberculina vinosa and Verticillium lecanii (Blakeman and Fokkema, 1982).

CONCLUSION

The literature indicated that two species of Phakopsora, P. pachyrhizi (an Australia and Asian species) and P. meibomiae (a new world species) are known to infect soybean in the world. The geographic range of P. meibomiae is restricted compared to that of P. pachyrhizi, which has been spreading to newer areas since last few years and now occurs in all major soybean growing areas except North America.

Rust of soybean caused by P. pachyrhizi, earlier known to be of minor importance became prominent after 1993 in India. As a very limited research was done on soybean rust, basic information on epidemiology and biology, resistant/tolerant varieties and effective and integrated management is lacking. So, concerted efforts are needed to find out the primary source of infection, race analysis, identification of resistant genes/genotypes against these races, inheritance studies and marker assisted selection. Various factors influencing the teleospore production, its germination and infection process should be worked out. Additional research in these areas will certainly uncover many more required information for the development of effective and sustainable integrated approach for the management of rust.

REFERENCES


