A review of soybean rust from a South African perspective

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This review article describes the nature of the soybean rust pathogen, its interaction with the soybean host and documents some of the history of soybean rust in South Africa. Soybean rust has affected soybean cropping in parts of South Africa since 2001. The disease causes leaf lesions, which may progress to premature defoliation and ultimately result in grain yield loss in susceptible soybean genotypes. Chemical control measures have been successfully employed to limit commercial yield losses in South Africa; however, controlling the effects of this disease through host-resistance or tolerance mechanisms remains a long-term goal.

Key words: *Phakopsora pachyrhizi*, *Glycine max*, chemical control, tolerance

Introduction

Soybean rust, caused by the fungus *Phakopsora pachyrhizi* Sydow, was reported on soybeans (*Glycine max* L. Merr) in the Vryheid district of South Africa in February 2001,[1] and later identified in several other parts of KwaZulu-Natal (KZN) and Eastern Highveld production regions. Epidemics of soybean rust have since occurred in these areas every season to date (2008) and chemical control has become a standard commercial practice in the affected growing regions. Shortly after rust was identified in neighbouring Zimbabwe in 1998, a soybean rust workshop[2] was convened in Potchefstroom, South Africa, and a soybean rust task team was established to familiarize local researchers with the disease and develop a pre-emptive national soybean rust strategy. Through visits to Zimbabwe in the three-year period between the first outbreak in Zimbabwe and the first reported outbreak in South Africa, many local researchers gained valuable experience in identifying the disease and managing the epidemics.[3] Consequently, commercial losses in the first two seasons were far less than they could have been, as chemicals and protocols used in Zimbabwe were adopted until local research could support the soybean cropping industry.

The pathogen

There are approximately 80 species of *Phakopsora* known worldwide,[4] six of which occur on legumes. Soybean rust is caused by two species, *P. pachyrhizi* and, less commonly, *P. meibomiae* (Arthur) Arthur. The latter species (*P. meibomiae*), commonly known as the cause of Latin American rust or Legume rust, is found in the western hemisphere and is not known to cause severe yield losses.[5] The nomenclature history of these two species of rust is complex and their correct assignment in early reports, especially from Africa, remains uncertain.[4] The subject of this review is restricted exclusively to *P. pachyrhizi,* the cause of the disease known commonly as Asian soybean rust, or simply soybean rust hereafter.

Global distribution

Before 1992, soybean rust was known to cause significant losses in Asia and Australasia, inclusive of the following countries: Australia, India, Indonesia, Japan, Korea, Peoples Republic of China, Philippines, Taiwan, Thailand, Vietnam.[6] Not much was documented about the distribution of soybean rust in Africa before 1996 (given the problems with nomenclature); however, the following sequence of first reports[7] were confirmed: Uganda, Kenya and Rwanda, 1996; Zimbabwe and Zambia, 1998; Nigeria, 1999; Mozambique, 2000; South Africa, 2001. During 2001 *P. pachyrhizi* was detected in Paraguay[8] and this was followed shortly by confirmation of its presence in Argentina in 2002[9] and Brazil and Bolivia in 2003.[10] Uruguay, also a significant soybean producing country, recorded soybean rust for the first time in 2004.[11] Soybean rust was detected in Hawaii in 1994[12] which stimulated the convening of a workshop to discuss the potential threat that this held for the soybean crop in the U.S.A. As correctly predicted by the delegates of this workshop,[13] soybean rust had the potential to threaten crops on mainland U.S.A. In 2004, nine years later, Schneider *et al.*[14] confirmed the presence of soybean rust in the U.S.A. From detection in Louisiana in 2004, it spread to nine states by 2005, and was detected in 15 states in 2006.[15]

Alternative hosts

The soybean rust pathogen is known to naturally infect 95 species from 42 genera of legumes, inclusive of important weed species like Kudzu vine (*Pueraria lobata*) and major crop species such as common bean (*Phaseolus vulgaris*).[5] Such a broad host range is unusual amongst rust pathogens[5] which normally have a narrow host range. The significance of the numerous alternative host possibilities for the soybean rust pathogen is that these may serve as an inoculum reservoir or a 'green bridge' from one soybean planting season to the next.

Epidemiology of soybean rust

The presence of a susceptible host, viable pathogen spores and suitable environmental conditions are requisites for the development of a soybean rust epidemic. The optimum temperature for urediniospore germination ranges between 12 and 27°C, depending on the source of the research.[16–18] Urediniospore germination is greater in darkness, with light either inhibiting or delaying germination.[18] A further requirement for urediniospore germination is a period of leaf wetness. This period is considered to be about 6 h when this occurs within the optimal temperature range.[19] The optimum temperature for uredinia formation is reported by Kochman[20] to be 17°C (night) or 27°C (day). Uredinia form on the leaves nine days post infection (DPI) under these conditions, with the urediniospores maturing two to three days later.[21]

Symptoms of soybean rust

First symptoms of soybean rust could be described as small water soaked lesions which develop into grey, tan to dark brown, or reddish brown lesions (uredinia) particularly on the abaxial leaf surface.[22] The colour of the lesion is dependent on lesion age and interaction with the host genotype.[6] Red-brown (RB) lesions with little sporulation indicates a semi-compatible reaction, whereas tan lesions with much sporulation (Fig. 1) indicates a fully compatible reaction. During the early stages of development, before sporulation, soybean rust may be confused

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Fig. 1. Tan sporulation of soybean rust on the lower leaf surface of a susceptible soybean genotype.

with bacterial pustule disease [*Xanthomonas campestris* pv *glycines* (Nakano) Dye].[22] Soybean rust symptoms generally occur first on the leaves at the base of the plant and progress up the canopy as the disease severity increases. Increased lesion density leads to leaf yellowing and ultimately premature leaf senescence, resulting in yield losses primarily through reduced grain size.[23]

Effect of soybean rust on yield

There is a dearth of published information on the effects of soybean rust on soybean yields in South Africa. Researchers that have published data relating to the effects of soybean rust on yield have recorded considerable variability over seasons and genotypes.[24,25] McLaren[25] evaluated all the commercial soybean genotypes over two seasons and concluded that there was no tolerance of economic value amongst them. He also observed that the yield loss sustained in shorter maturity genotypes was lower than the longer maturity genotypes. This observation confirmed the earlier work of Caldwell and McLaren[24] who had come to a similar conclusion but had conducted their research on only one genotype per maturity class, leaving some doubt as to whether the effect was genotype specific or maturity-group related.

Initial indications from the research of Caldwell and McLaren[24] showed that planting date did influence the yield loss, but their two seasons' data were not sufficient to substantiate a trend. Soybean rust symptoms were more severe in the 0.45 m than in 0.90 m row spacing, and this was attributed to poorer fungicide penetration into the canopy.[24] McLaren[25] found that disease severity, as measured by the area under the disease progress curve (AUDPC), was poorly correlated to yield loss percentage. Mean yield loss for 2003/04 season was 31.1% or 1.68 t ha^{-1} and in 2004/05 season it was a devastating 60.9% or 3.4 t ha^{-1}. Genotype ranking for yield loss percentage between the two seasons was substantially different, highlighting the considerable variability of soybean rust epidemics over seasons and the difficulty in selecting for improved genotypic response.

Distribution and spread of soybean rust in South Africa

There has not yet been a formal attempt to survey the distribution of soybean rust in South Africa; however, the reports of positive identification of soybean rust sent in by members of the soybean rust task team have been collated for the period 2001–2008 (Table 1). The reports increased in frequency over the years surveyed, likely as a result of more scientists becoming involved in reporting rather than an increase in disease incidence. The distribution of locations with one or more soybean rust reports have been plotted on a rainfall map of South Africa (Fig. 2). The area with the highest incidence of soybean rust reports coincides with the high rainfall region east of the Drakensberg mountain range. Del Ponte *et al.*[26] showed that cumulative rainfall in the period after initial rust detection was positively correlated to disease severity, which probably accounts for the similarity in the rainfall and soybean rust distribution patterns. During the 2006 season, reports of soybean rust were obtained atypically far west of the normal distribution, but mostly too late in the season (Table 1) to have a significant impact on yield.

The collated reports are probably not ideally suited to making judgements on the progression of the disease, because the date of the report is not always a good indication of the start of the epidemic. However, in seasons that had sufficient reports to substantiate a trend (2006–2008), first reports for the season generally started in the east and progressed westward. While this may indicate a closer proximity to the inoculum source in the east of the production region, weather conditions favouring infection and development of symptoms may simply occur earlier in the season in the east compared to the west.

There is no literature on how the soybean rust pathogen survives from one season to the next in South Africa; however, Caldwell and McLaren[24] established that it required a live host and did not survive on soybean stubble. Since most of the soybean production regions receive significant frosts in winter, the pathogen is presumed to overwinter in frost-free areas within the country. Soybean rust epidemics in the KZN midlands normally originate from a few clearly distinguishable foci within a field, which would infer that initial infections have been started by a low concentration of windborne urediniospores. Infections that have resulted from urediniospores generated from within these foci, are a lot more uniform, clearly a function of inoculum concentration around these foci.

Pivonia and Yang[27] used a mathematical model to predict the likelihood of year-round survival of *P. pachyrhizi* across the world based only on historical temperature and moisture data. Host availability and presence of an inoculum source were not considered. They found that conditions for the survival of *P. pachyrhizi* were very favourable all along the east and southern coasts of South Africa. Since this area does not coincide with the soybean production area, it is likely then that the soybean rust pathogen survives the winter in this area on the many possible alternative hosts. Pretorius *et al.*[28] established that Kudzu vine (*Pueraria lobata*) was one of the alternate hosts of *P. pachyrhizi* that provided a green bridge in South Africa for the survival of the pathogen through winter in the frost-free areas. It is speculated

Table 1. A compilation of soybean rust reports made to the soybean rust task team.

Date	Location	Reporter
8 Feb 2001	Vryheid	H. Oellerman
6 Mar 2001	Howick	K. Horne
9 Mar 2001	Ahrens	F.J. Kloppers
14 Mar 2001	Greytown	J.A. Jarvie
– Mar 2001	Amersfoot	Unconfirmed
– Mar 2001	Ermelo	Unconfirmed
– Mar 2001	Piet Retief	Unconfirmed
4 Feb 2002	Cedara	E.D. Du Preez
15 Feb 2002	Amsterdam	J.L. Purchase
15 Feb 2002	Greytown	J.A. Jarvie
8 Jan 2003	Cedara	E.D. Du Preez
8 Jan 2003	Karkloof	E.D. Du Preez
14 Feb 2003	Greytown	J.A. Jarvie
26 Jan 2004	Cedara	E.D. Du Preez
26 Jan 2004	Karkloof	E.D. Du Preez
16 Feb 2004	Greytown	F.J. Kloppers
8 Apr 2004	Ermelo	P. Kruger
3 Jan 2005	Cedara	E.D. Du Preez
11 Jan 2005	Karkloof	E.D. Du Preez
3 Feb 2005	Winterton	E.D. Du Preez
3 Feb 2005	Weenen	E.D. Du Preez
24 Feb 2005	Greytown	J.A. Jarvie
9 Mar 2005	Winterton	N. Hackland
1 Feb 2006	Karkloof	S. Tweer
1 Feb 2006	Cedara	S. Tweer
2 Feb 2006	Greytown	E.D. Du Preez
3 Feb 2006	Sudwala Cave	Z.A. Pretorius
9 Feb 2006	Piet Retief	M. Craven
16 Feb 2006	Vryheid	M. Craven
24 Feb 2006	Morgenzon	M. Craven
27 Feb 2006	Amersfoot	W. van Wyk
3 Mar 2006	Winterton	M. Craven
14 Mar 2006	Normandien	M. Craven
14 Mar 2006	Kinross	M. Craven
15 Mar 2006	Kroonstad	F.J. Kloppers
30 Mar 2006	Kestell	M. Craven
3 Apr 2006	Potchefstroom	M. Craven
24 Apr 2006	Bothaville	F.J. Kloppers
25 May 2006	Letsitele	J.A. Jarvie
25 Jan 2007	Cedara	A. Liebenberg
26 Jan 2007	Piet Retief	M. Craven
31 Jan 2007	Greytown	F.J. Kloppers
1 Feb 2007	Vryheid	M. Craven
1 Feb 2007	Morgenzon	M. Craven
12 Feb 2007	Merrivale	N.C. van Rij
22 Feb 2007	Normandien	M. Craven
1 Mar 2007	Bergville	E.D. Du Preez
8 Mar 2007	Besters	E.D. Du Preez
25 Jan 2008	Cedara	N.C. van Rij
28 Jan 2008	Greytown	S. Tweer
7 Feb 2008	Vryheid	M. Craven
7 Feb 2008	Baynesfield	P.M. Caldwell
5 Mar 2008	Normandien	M. Craven
26 Mar 2008	Seven Oaks	J.A. Jarvie
7 Apr 2008	Winterton	J.A. Jarvie
7 Apr 2008	Groblersdal	J.A. Jarvie

that this frost-free area then provides the initial inoculum source each season for the inland areas that have summer conditions favourable for the development of soybean rust. The consistency with which the epidemics have occurred since 2001 (Table 1) would tend to support the postulation that the source of urediniospores is, at the very least, regional and that local epidemics are not reliant on major weather phenomena for the deposition of urediniospores from the tropics of Africa.

Chemical control

Emergency registration of a number of chemicals made it possible for farmers to control epidemics during the first two seasons that soybean rust affected production in South Africa.[29] Much debate in South African soybean workgroups revolved around the difference in rates used in Zimbabwe compared to the recommended chemical rates in South Africa. The fear existed that sub-optimal doses of chemical would promote the build up of pathogen resistance to the active ingredients that controlled soybean rust. With pathogen diversity and variability clearly demonstrated in host-pathogen relationships,[5] this was a valid concern. Du Preez and Caldwell[29] evaluated effective dosage rates, timing of application and frequency of applications. This research contributed towards a leaflet being published[30] that made recommendations to soybean producers regarding control of soybean rust and included the registered chemicals. Du Preez and Caldwell[29] established that effective chemical control varied in a time range from 10 d (triforine) to 19 d (flusilazole/carbendazim), which supported the generalization that spray intervals should be no longer than 21 d apart, and that between one and three sprays may be required. They also concluded that some chemicals (flusilazole/carbendazim) had limited curative action, whereas others (azoxystrobin) were only effective in preventative applications. This conclusion was very important to the national strategy used to control rust. If control was primarily preventative, then the timing of fungicide applications in the absence of symptoms would be crucial, a conclusion that was also reached by several other researchers.[31] A reliable indicator of first spray was required, since spraying too early would mean unnecessary additional sprays, and spraying at first symptom would result in yield losses. As part of the national strategy to control soybean rust in South Africa, a series of 10 soybean indicator plots were planted throughout the production region, using early planting dates and genotypes which represented the extremes of maturity range for the country. These plots were not sprayed with fungicide and were monitored on a weekly basis from January through to April[32] for the presence of rust, both *in situ* and via leaf samples in the laboratory. These plots were used as sentinel plots to give producers advance warning of the presence and severity of the disease in an area. Producers were notified of the first presence of soybean rust in their area via cell phone SMS or alerts on farm radio programmes.[32] The system of sentinel crops is currently also one of the methods being applied in the U.S.A.[33] for the advance warning of the presence of the disease. Systems that recommend spraying at predetermined soybean growth stages, for example at flower or at 60 days after planting (dap) as in Zimbabwe,[34] do not take into consideration that the timing and severity of epidemics may have considerable seasonal variation. This could result in unnecessary spraying in some seasons. Hartman,[15] however, reported that there were occasional yield benefits to spraying fungicides in the absence of rust which may make this system both cost effective and simple to apply.

In 2005, a report from Washington State University[35] claimed that Roundup herbicide (glyphosate) had been found to have fungicidal action on *P. pachyrhizi* under laboratory conditions. Owing to the popularity of Roundup Ready (RR) soybean genotypes in South Africa, Kloppers and Jarvie (unpubl. data) performed a pilot study with sequential sprays of Roundup on an experimental RR genotype to establish whether there was a need to pursue this avenue of research further. The preliminary results showed that pre-flower applications of Roundup had no effect on soybean rust severity, but post-flower applications visibly reduced the premature defoliation due to rust. Since Roundup, when used as a herbicide, is primarily applied to soybeans at a pre-flower stage, it was felt that these findings would have little practical applicability and this line of research was not pursued further. The results of this pilot study were later confirmed by independent research conducted in the U.S.A. by Jurick and co-workers.[36] In their study, control of soybean rust by applications of Roundup at the R2 and R4 stage significantly improved yield over the untreated control, but the yield benefit and control of the disease was inferior to that of conventional fungicide (azoxystrobin) applications.

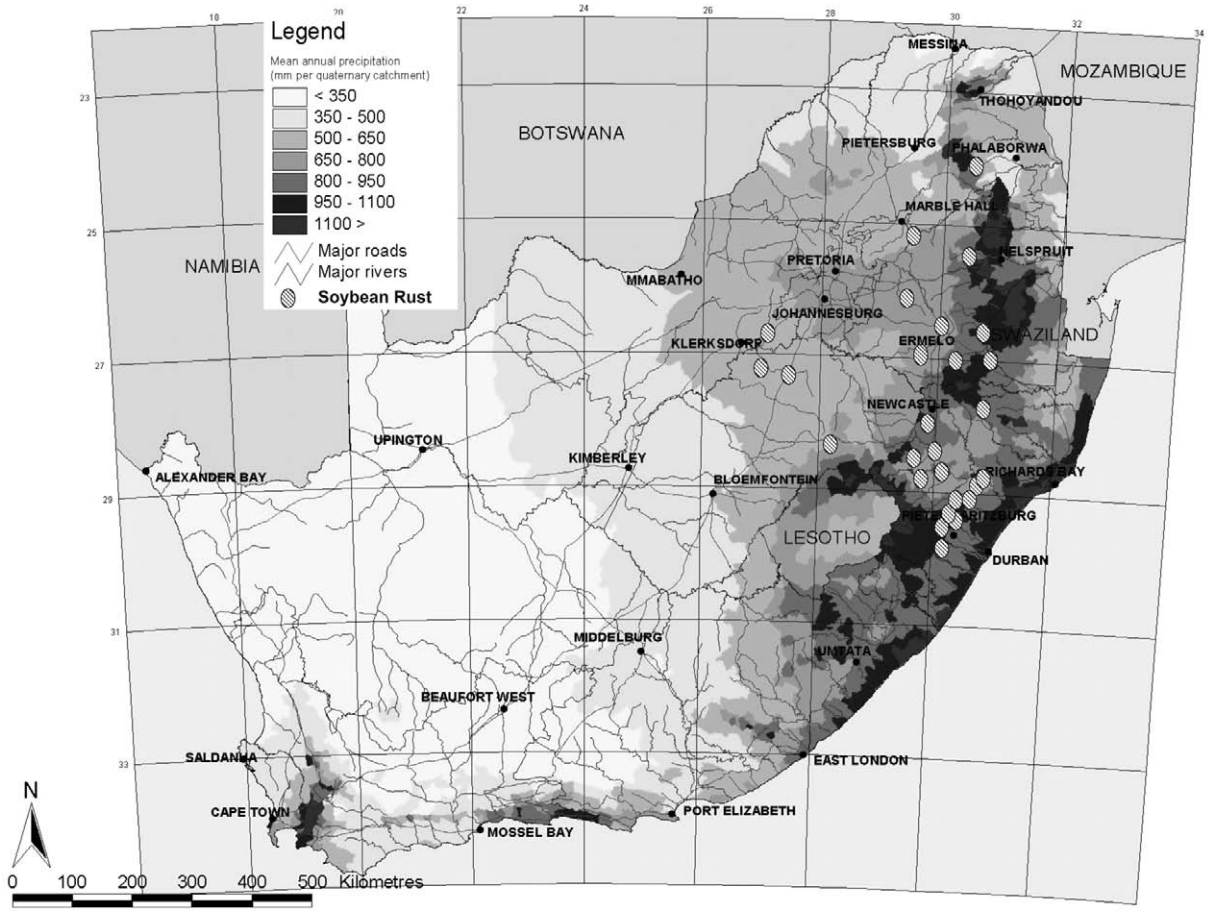

Fig. 2. Distribution of locations with one or more reports of soybean rust during the period 2001–2008, superimposed on the annual rainfall map of South Africa (Source: Surface Resources of South Africa, 1990).

Resistance

Screening for resistance

From the early 1960s through to the 1990s, much of the soybean rust research focused on resistance. Tschanz[37] reported that he and his co-workers at the AVRDC (Asian Vegetable Research and Development Centre) had, over the years, screened more than 9000 accessions for resistance to soybean rust. Hartwig[38] reported to have evaluated 1675 germplasm lines adapted to the southern U.S.A. for resistance to soybean rust in Taiwan. From this early screening work, it was clear that various levels of specific resistance, partial resistance and tolerance to soybean rust all occurred in soybean germplasm. One of the recent objectives of the USDA-ARS soybean rust research programme has been to evaluate the USDA germplasm collection for resistance. A set of 174 soybean genotypes, inclusive of the most important parental germplasm and the most promising sources of resistance, were screened against field populations of *P. pachyrhizi* in Brazil, China, Paraguay and Thailand.[39] South Africa also participated in this evaluation, where soybean rust symptoms on this set of germplasm were recorded in the 2002/03 and 2003/04 seasons at Greytown, KZN. No lines were found to be resistant at all locations. With the threat of soybean rust looming in the U.S.A. at that time, the search for resistance intensified further, eventually involving the screening of 16 595 accessions in the Fort Detrick containment facility.[15]

Under field conditions, early maturing soybean genotypes will have a higher disease rating earlier in the season than the equivalent later maturing genotype. The rate of rust development in these genotypes is also higher than that of later maturing genotypes, and if a correction for host maturity is not made, erroneous conclusions from field data will result.[40] To correct for maturity, relative life time (RLT) is calculated as the proportion of the life cycle completed relative to the complete life time (time from planting to harvest) of the genotype. Only rust severity ratings at comparable RLTs can be compared, which makes a single simple field severity rating meaningless unless all genotypes are of a similar maturity. McLaren[25] showed that disease severity, as measured by the area under the disease progress curve (AUDPC), was poorly correlated with yield loss. For this reason, disease severity ratings are seldom used as a measure of resistance.

Specific resistance in soybean

McLean and Byth[41] presented the first evidence of physiological races in *P. pachyrhizi* on soybean genotypes in Australia. Race 1 was virulent on Wills and avirulent on PI 200492. Race 2 was virulent on both varieties. Subsequent to this, considerable variation in isolate virulence (collected from the same field, as well as isolates from geographically-distant regions) has been shown to occur.[5] Three infection types have been described: the Tan lesion is a fully susceptible reaction; the resistant RB reaction is a red-brown lesion with no or few sporulating uredinia; and the absence of any macroscopic symptoms is immunity.[6] Eleven genotypes were used as a differential set to determine the physiological races of 42 purified *P. pachyrhizi* isolates by Wang and Hartman,[6] and based on the infection type they were able to identify nine races. The data suggested that the pathogen races studied were complex and that they possessed multiple virulence genes for compatibility on many of the differential cultivars. Bromfield[42] reported on a *P. pachyrhizi* race that had

three virulence genes, more than were necessary to overcome host resistance. More recent research[5] indicates that field pathogen populations are often mixtures of many races which may induce mixed infection types in the host. This is not uncommon in rust pathogens, as was shown to be the case with bean rust (*Uromyces appendiculatus*) where the more tropical locations (like South Africa) were found to induce greater race variability than more temperate climates.[43] It is not known how many races are commonly found in South African soybean fields, but since mixed infection types on the same plant have been observed, at least two races must be present. Variability in race virulence is also known to occur. In inoculation studies conducted under controlled conditions, researchers reported that recent isolates collected from southern Africa and South America were significantly more virulent than Asian isolates collected in the 1970s.[44] The most virulent isolate they reported was collected in Zimbabwe.

The specific resistance gene in PI 200492 was given the designation Rpp_1,[45] and since then three other independent dominant genes have been named: Rpp_2;[46] Rpp_3;[47] Rpp_4.[48] In Brazil, where the Rpp_1 and Rpp_3 genes are ineffective and Rpp_2 and Rpp_4 currently confer resistance, Neto[49] reported that many 'new' (unnamed) gene sources of resistance have been discovered. These were tested for allelism to Rpp_2 and Rpp_4, and of the 26 sources reported, 23 were found to be at different loci to Rpp_2 and Rpp_4. One of these sources of resistance was conditioned by a single recessive gene[49] from the variety Abura, and this has been incorporated in a variety (BR01-18437) destined for release in Brazil during 2008. Neto[49] also reported the preliminary findings that stacking Rpp_2 and Rpp_4 in a single genotype had no additive advantage in the expression of resistance.

The presence of multiple virulence genes in the pathogen population and the lack of multiple resistance genes in the host provides the soybean rust pathogen with a competitive advantage. The deployment of specific single genes for resistance is thus unlikely to be a successful strategy. As an example of gene failure, Hartman *et al*.[5] quoted the examples cited by Bromfield, where the Rpp_1, Rpp_2 and Rpp_3 lost their effectiveness in the field within 10 years of exposure. In Taiwan, Shanmugasudaram *et al*.[50] quoted examples of Tainung 3, Tainung 4 and Kaohsiung 3 (all cultivars containing Rpp_1) becoming susceptible within a few years of release. Genotypes PI 230970 and PI 230971 were identified as being resistant in Taiwan, and these were subsequently used as parents in crosses to generate a number of resistant lines (AGS 181, AGS 182, AGS 183, AGS 229, AGS 233, AGS 240, AGS 244, AGS 247). So too were the resistances of these lines short lived. Following that, new sources of resistance were identified in PI 459024, PI 459025 (Rpp_4) and PI 339871 (*G. soja*) but have all since been defeated.[5,50] In Brazil, Yorinori[10] had a similar experience with germplasm that had shown resistance in 2002 being susceptible in 2003.

The use of gene pyramiding and gene rotation is also unlikely to be a stable solution because the pathogen retains unnecessary virulence genes at a high frequency in its population.[51] In addition, resistance associated with the RB infection type is a semi-compatible host–pathogen reaction, which generally allows pathogen reproduction and has not been shown to significantly affect epidemic development.[51]

Partial resistance

Partial or rate-reducing resistance to soybean rust has been documented in soybean,[51] but it has not been widely employed because of complexities in assessment. Plants or genotypes maturing at different times cannot be compared to each other in the field because of the different environmental conditions that they are exposed to at similar growth stages. Physiological differences can be partially corrected for by regressing relative life time (RLT) on the log transformation of rust severity. The slopes of these graphs can be compared to identify the 'slow rusting' genotypes. Collecting the data required to generate these graphs is laborious and cannot be conducted on a large number of genotypes, limiting its practical application. Hartman *et al*.[5] suggested that measuring the latent period would help identify genotypes with a long latent period and hence a slower rate of rust development. The difficulties associated with identifying partial resistance and the ineffectiveness of specific resistance genes has led to the suggested use of tolerance as a breeding remedy for soybean rust.

Tolerance

Tolerance implies susceptibility, and can be defined as the relative ability of a genotype to yield under stress from rust.[6] Tolerance is a characteristic that can only be evaluated in the target environment while under rust stress, as it implies a measure of genotypic adaptation to that environment. Tolerance is of little value unless the genotype is high yielding in that environment and it maintains yield stability despite rust infections. Selecting for yield stability in the presence of rust is not an easy task[5] since over and above the normal genotype × environment interaction that breeders have to contend with for adaptation, seasonal variation in severity and timing of rust epidemics is superimposed. Whilst yield is normally the primary consideration, a consistent performance is also valuable to a producer, who may be willing to sacrifice some yield in order to achieve a stable yield over seasons.[52] Tolerance is traditionally assessed by comparing yields of paired plots of fungicide protected versus unprotected plots. The percentage yield loss between fungicide protected and unprotected plots is not necessarily correlated to rust susceptibility ratings or to rust development rates[5] and may be linked to other stress-tolerance mechanisms. Significant variation in tolerance levels exist in soybean, which could be exploited by breeders. From work conducted at the AVRDC in Taiwan, Hartman[40] demonstrated yield losses of 12 genotypes ranging between 29 to 85%. Based on reduced pustule numbers, the two genotypes that had the smallest yield losses (29% and 31%) could conceivably have had some form of partial resistance. This, when compared to a possible 85%, appears to be significant but in reality is still far too high for practical benefit on a commercial scale. In more recent research conducted in Brazil,[49] minor genes have contributed towards tolerance in the genotype EMGOPA 313, with yield losses in the order of magnitude where fungicide spraying would still be financially attractive.

Conclusion

High levels of tolerance or sustainable rust resistance in South African genotypes is not imminent, which means that for the foreseeable future control of soybean rust by a combination of chemical and cultural means will need to continue. An efficient warning system and effective fungicides have been instrumental in averting potentially large financial losses to producers. Whilst seasonal soybean rust epidemics will persist and control measures will continue to be required, the soybean rust crisis in South African soybean production is largely over as a result of the efforts of forward-thinking policy-makers and pro-active researchers.

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