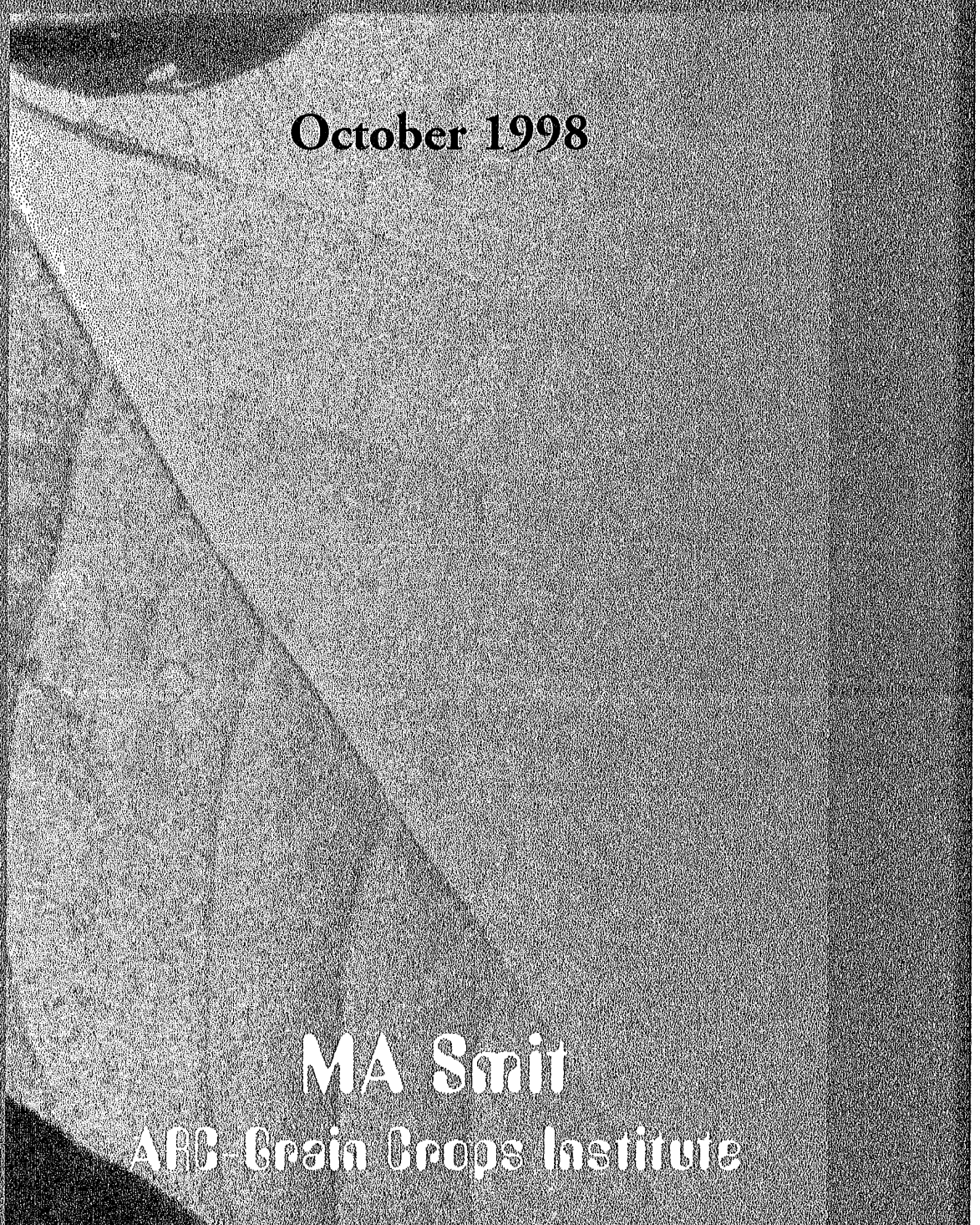
Proceedings of Workshop on Soybean Rust (*Phakopsora pachyrhizi*)



October 1998

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Sponsored by Protein Research Trust and Agricultural Research Council

PROCEEDINGS OF WORKSHOP ON SOYBEAN RUST (*Phakopsora pachyrhizi*)

ARC-Grain Crops Institute, Potchefstroom

Sponsored by Protein Research Trust and Agricultural Research Council

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PREFACE

Soybean rust is a devastating disease common to the SE Asia. It has also been reported in India and Australia. Two years ago scientists attending the All Africa Crop Conference in Pretoria reported that a outbreak of soybean rust in Uganda. Last year scientists reported the occurance of this disease in Zimbabwe.

There is no known resistance or tolerance for rust amongst RSA soybean varieties, and no chemicals are registered for protecting RSA commercial crops against the disease. Should the disease move South and infect local crops, research and industry will not be in a position to assist the National Oilseeds Producers Organisation to minimize the damage. The ARC therefore thought it wise to stage a rust workshop, invite a world authority on soybean rust to the workshop and to bring all interested parties and local expertise together in order to work out a strategy for South Africa.

Michiel Smit
Programme manager, ARC Soybean research

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1. INTRODUCTION: PLANT PATHOLOGY RESEARCH IN SOUTH AFRICA, PAST, PRESENT AND FUTURE

Dr K Pakendorf, Deputy Director, ARC- Grain Crops Institute

INTRODUCTION

The South Africa agricultural scientist has a proud record of successful basic and applied research achievements. This is best illustrated by the fact that no agricultural disaster in this country could be linked to a major pest or disease. This was achieved by the dedicated efforts of scientists of many scientific persuasions and the close integration of disciplines including crop management practices, crop rotations, genetic improvement through classical plant breeding as well as the discriminate use of organic chemicals. South Africa has been singularly fortunate in that international chemical companies have for many years been major role players in ensuring effective and optimal crop production in South Africa. This has all lead to the happy situation that this country has as yet never faced a famine, something which bedevils all the countries to the North of South Africa.

HISTORICAL

Plant breeding

Historically, the use of synthetic chemicals for pest control began in the 1940's and due to the effectivity of these chemicals, they were overzealously utilised in combatting major pests and diseases. Their use in modern agriculture has contributed significantly to yield increases in the 1940's to 1960's, culminating in the green revolution in India, Pakistan and Mexico. Clearly, the role of plant breeding in achieving this significant milestone must not be overlooked. The discriminate use of genetic resistance in all fields of agriculture has probably contributed more toward pest and disease control than any other scientific endeavour to date. Genetic control of plant diseases has stimulated much controversy, but also success, especially regarding the debate of single major genes versus multiple minor gene resistance. The use of single genes has had a vast impact in disease control in agricultural crops such as wheat, maize and groundnuts. However, it can be criticised since it also gave rise to new races of pathogens through the process of mutations. In this sense, it can probably be described as a boom or bust approach. In the sixties and seventies, controversy relating to multiple, or horizontal genetic resistance dominated scientific activity in especially wheat breeding. In this regard, JE van der Plank of South Africa,

played a leading role in defining and elaborating on the principles of this form of genetic resistance in combatting plant diseases in especially potatoes and wheat.

Integrated pest management

Lead by entomologists, researchers began to recognise the problems associated with dependence on the extensive use of insecticides in the fifties. They developed concepts leading to what is now commonly referred to as integrated pest management (IPM). The central principle of IPM is the economic threshold concept, which holds that the mere presence of a pest population does not necessarily indicate an economically damaging situation, where benefits will exceed the cost of control. In principle IPM is an ecologically based pest control strategy that relies on natural mortality factors, such as natural enemies, weather and crop management. It seeks control tactics that disrupt these factors as little as possible. Traditionally the term IPM has been linked to entomology, but is currently also used in the fields of plant pathology and weed science. The common thread of all IPM programmes is the concept of an economic threshold below which pest populations or damage is tolerated. In terms of plant pathology, IPM programmes still face major challenges, especially with regard to the control of soil-borne diseases.

Alternate mechanisms

The use of natural control mechanisms, such as naturally occurring antagonists, phytoalexins and cross resistance mechanisms, have and still are important possible control measures which have stimulated much research and discussion, but have as yet not proven to be of major importance in commercial crop production systems. With the vocal lobbying of the disciples of the green environment and the pundits of alternative agricultural production systems, this form of disease control has received increased emphasis and attention in the past decade. In spite of being vocally derided by agricultural scientists in particular, the environmentalists have at least achieved a rethink on the excessive use of chemicals in combatting pests and diseases.

Molecular biology

The emergence of biotechnology and genetic engineering has offered plant scientist novel tools in manipulating and controlling plant diseases. Unfortunately, the purists who advocate the use of biotechnology as a revolutionary and long-term strategy to control plant diseases fail to realise that even alien genes, inserted into certain host plants, still act according to classical Mendelian laws of inheritance. The basis of molecular biology is founded on the principle that in all living organisms, genetic information is carried in the now well known DNA double helix, and that this genetic information can be manipulated at will by utilising prescribed laboratory procedures. It

must be clearly emphasised that biotechnology is merely a tool in achieving a desired state, and that it is not a desired state in itself.

Molecular biology and molecular genetics have now become a major focus of interest in many fields of agricultural endeavour. The question might now be raised as to whether molecular biology has become a new bandwagon onto which everybody wants to climb in order to be fashionable? There is a definite danger that the emphasis is shifting to fundamental research, away from applied or mission oriented production research. Currently, it is in vogue to emphasise molecular techniques as being synonymous with good or sophisticated research, while looking down upon applied plant pathological endeavours. This debate is not new, but has existed in plant pathology for many years. Horsfall (1959) felt that the science of our discipline was underdeveloped, and Stevens (1961) thought that the art of plant pathology was thriving, but that the science was essentially non-existent. In this total controversy, it might be apt to remark that plant pathology is a derivative subject, bordering on crop fields (e.g. agronomy, horticulture, forestry) on the one hand, and source fields (e.g. botany, soils, plant physiology, biochemistry, genetics, mathematics) on the other; it must be firmly rooted in the source field but must solve complex problems in agriculture. Too frequently biologists have come to equate fundamental with difficult and applied with easy research, although actually the reverse is often true. The question of successful plant pathology programme can be defined in the words of SD Garrett, the eminent soil microbiologist/pathologist, who cogently commented "the best monument to a plant pathologist is a forgotten plant disease".

The above brief resumé of scientific activities with regard to plant disease control in South Africa has been achieved through the active cooperation of various disciplines and institutional organisations. Here mention must be made of academic institutions who have succeeded in training highly skilled scientists, agricultural companies such as chemical companies, seed companies, agricultural cooperatives, the previous Department of Agriculture and currently the Agricultural Research Council. In order to achieve this success, the mediation of extension officers of these various institutions have been extremely important in order to provide the South African farmer with the correct technology in order to produce cost effectively.

2. EPIDEMIOLOGY AND CONTROL STRATEGIES FOR SOYBEAN RUST

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SOYBEAN RUST

I. INTRODUCTION

Soybean, *Glycine max* (L.) Merrill, is an important food, feed and industrial crop in Asia. The total soybean area in Asia was about 13 million ha in 1991/92 and it increased to 15 million ha in 1996/97. It represents almost one fourth of the total world area. The total soybean production in Asia was 15.6 million t in 1991/92 and 20.5 million t in 1996/97. It is equal to 14.5% and 15.5% of the world production in1991/92 and 1996/97, respectively.

The average yield of soybean in Asia is 1.35 t^{-ha} compared to the world average of 2.10 t^{-ha}.

Soybean is used in many different ways as a food. Soybean milk, many varieties of tofu (been curd), tempeh, natto, beansprouts, green vegetable soybean, protein film, soy sauce, soybean oil, nutri-nuggets, soynuts are a few of the popular food items commonly used in many Asian countries.

Many biotic and abiotic factors are responsible for the low yield of soybean in Asia. Among the biotic factors insect pests and diseases are the major cause for yield reduction. Soybean rust, caused by *Phakopsora pachyrhizi* Sydow is one of the major diseases. The yield loss due to soybean rust disease can vary from 10% to almost 100% depending on the severity of the disease and the growth stage of the crop at which the infection occurs. In a few countries like Australia, and India, soybean rust infection occurred in epidemic proportions but became almost insignificant disease after a couple of years. In recent years, soybean rust has become a serious disease in India and Africa. In a simulated yield loss model study in the USA, Kuchler, et al. (1984) have shown that in the event of severe soybean rust attack the estimated loss due to the disease will be US$7.2 billion! Assuming a conservative yield loss of 11% due to soybean rust, the loss in terms of US$ will be 2.23 billion.

II. DISTRIBUTION

Soybean rust was first reported in Japan in 1902 (Kitani and Inoue, 1960). Four years later in

1906, it was reported in India. In 1914, Papua New Guinea, Philippines and Taiwan recorded the pathogen. The first report of soybean rust in Australia was in 1934 from Queensland (Bromfield, 1984). The disease was observed and reported subsequently in many other Asian countries (Table 1).

Table 1: Geographical and seasonal distribution of soybean rust caused by *P. pachyrhizi* in the Eastern Hemisphere.

Country	Epiphytotic year	Months
Australia	1934, 1971	April – May
Cambodia	1962	-[1]
China	1940 or earlier	August
Taiwan, China	1914	Mar. – May; Sept. – Jan.
Ethiopia	(1976)	-
India	1906; 1970	April – May; Aug. – Sept.
Indonesia	1960	April – May; Oct. – Dec.
Japan	1902; 1948	Aug. – Oct.
Korea	1935 or earlier	July – Sept.
Okinawa	1948	Mar. – May
Papua New Guinea	1914; 1958	-
Philippines	1914; 1966	Aug.; Dec. – Jan.
Sri Lanka	1951	-
Thailand	1966	Mar. – Apr.; Sept. – Oct.
USSR	1957 or earlier	-
Vietnam	1948 or earlier	Aug. – Sept.

[1]- month not recorded.

As early as 1913, soybean rust was first collected and reported in Puerto Rico by Stevens (Arthur, 1915). The pathogen has been reported to cause disease in many other countries in the tropics and subtropics of the Americas (table 2), but not as seriously as in Asia.

Table 2: Reported occurrence of *P. pachyrhizi* in Western Hemisphere.

Location	Year reported
Brazil	1940 (Thurston, 1940)
Colombia	1913 (Kern et al. 1933)
Costa Rica	1976 (C.Y. Yang, AVRDC, Taiwan, personal communication)
Cuba	1926 (Seaver and Chardon, 1926)
Georgia (USA ?)	1922 (Sinclair and Backman, 1989)
Guatemala	1940 (Cummins, 1943)
Mexico	1917 (Sinclair and Backman, 1989)
Puerto Rico	1913 (Arthur, 1915)
St. Thomas	1926 (Seaver and Chardon, 1926)
Trinidad	1926 (Seaver and Chardon, 1926)
West Indies	1975 (Stevenson, 1975)

In Africa, *P. pachyrhizi* has been observed on "various legumes" other than soybean in Ghana, San Tome, Sierra Leone, Sudan, Tanzania, Uganda and Zaire. In Nigeria, *P. pachyrhizi* was collected from cowpea, however, adjacent soybeans were unaffected by the pathogen. Bromfield (1984) speculated that the *P. pachyrhizi* observed in Nigeria may represent a *forma specialis* different from that of Eastern Asia and from that in Americas.

III. SYMPTOMS

The symptom begins as a water soaked chlorotic polygonal lesion, which develops, into tan or reddish brown in color. The color of the lesion may become darker as the lesion ages. Individual lesions may coalesce to present an enlarged lesion. The leaves will turn abruptly yellow and abscission of the leaves occurs, especially in severe infection. Petioles and young stems may also get infected. Cotyledons and primary leaves may also show symptoms when inoculum and environment are favorable. The symptoms are abundant on older leaves especially in abaxial (lower) surface.

The lesions may contain one to several erumpent, globose, ostiolate uredia. The number of uredia per lesion increases with lesion age. The urediospores are exuded through the central pore in the uredium. The spores are in clumps.

Soybean rust symptoms appear similar to bacterial pustule. Through a hand lens or a microscope soybean rust will reveal the uredium and urediospore. However, in bacterial pustule, a fissure and necrotic tissue alone is visible.

In early morning, on a windless day, if a soybean rust infected leaf is gently tapped the spore dust will fall off. On the other hand, the bacterial pustule infected leaf will have only water soaked lesion.

Soybean genotype and pathogen race interaction results in either tan (TAN) lesion with profuse uredia or dark reddish brown (RB) lesion with zero, one or two uredia and extensive necrosis. Tan lesion is a compatible interaction, RB is semi-compatible and small, grayish flecks are incompatible interaction.

IV. PATHOGEN

The soybean rust disease is caused by the fungus, *Phakopsora pachyrhizi* Syd. & P. Syd belonging to the family Melampsoraceae, order Uredinales. The fungus was first described from the infected leaf of yam bean, *Pachyrhizus erosus* in 1913 in Taiwan (Kitani and Inoue, 1960). The fungus was observed in 1902 itself in Japan.

Uredosori are densely scattered, small, rounded and about 200 μ m diameter; paraphyses are long, and it has a circular pore. Urediniospores are globose to subglobose, ovate or ellipsoid, echinulate, light yellow-brownish, 20-28 x 17-23 μ m. Teleutosori scattered, aggregated, irregular, erumpent, small, blackish blood red; teleutospores 4-6 above each other, clavate, angulate.

The synonyms of *P. pachyrhizi* are:
Uredo vignae Bres
Uredo sojae Hennings
Uredo concors Arthur
Physopella concors Arthur
Physopella vignae Arthur
Phakopsora sojae Fujikuno
Phakopsora sojae Sawada
Uromyces sojae (Not Sydon) Miura
Uromyces sojae Sawada

Pycnia (spermogonia) and aecia are unknown for this fungus.

1. Uredinial stage:

Uredinia are "pimple like" on abaxial surface of soybean leaves. Urediniospores are erumpant through a pore at the top of the "pimple like" dome. Spores tend to clump together under calm field conditions.

Under optimum conditions the Urediniospores germinate to form a single germ tube. The subsequent appressorium formed at the end of the germ tube produces an hyphal tube, which penetrates directly through the cuticle into an epidermal cell. The penetrating hypha is termed transepidermal vesicle (Keogh, 1974). A second penetrating structure forms and penetrates through palisade tissue into spongy mesophyll of the leaf. Haustoria are formed within the palisade layer into the spongy mesophyll, vascular parenchyma and epidermis.

The sequence of events in the development of soybean rust from the time the urediniospore lands on the host is given in table 3.

Table 3: The sequence of events over time in the development of soybean rust.

Event	Time	sequence
a uredospore lands on soybean leaf surface over epidermal cell	0	hpi[1]
germ-tube development (5-400 um)	12	hpi
an appressorium-cone formed	16	hpi
penetration hyphae formed	16	hpi
first hyphal septum formed	18-20	hpi
primary hyphae produced	18-20	hpi
collapse of epidermal cell	24	hpi
haustorium formed (haustorium encasement)	24-48	hpi
branching into secondary hyphae	48-72	hpi

mycelia development inside spongy mesophyll and the intercellular space	3	DPI[2]
collapse of appressorium and penetration hyphae	4	DPI
necrotic lesions appear on leaf	6	DPI
runner hyphae passing through mesophylls	7	DPI
hyphae aggregate; uredial primordia formed consists of compact layer of 2-3 cell column	9	DPI
wall thickened (sporogenous tissue, pedicel and uredospore; Uredospores mature)	11-12	DPI

[1]hpi – hour post infection
[2]DPI – day post infection.

The comparative development of *P. pachyrhizi* in resistant and susceptible soybean has been studied in detail by Keogh (1978), McLean (1979, 1981) and Keogh, et al. (1980).

2. Telial stage:

Instead of uredinia and urediniospores, the fungus shifts to the production of telia and teliospores under specific conditions.

Telia are formed sub-epidermally in the midst of uredinia and they are crust-like. They contain two to five layers of teliospores. The telia may occur singly or in clusters. To begin with they are honey-brown in color and turn to brown and black with age. Teliospores are single celled and smooth walled.

V. CONDITIONS FOR SOYBEAN RUST DEVELOPMENT

The presence of a compatible host plant, a viable pathogen, its spore germination on the host plant, appressorium formation and penetration of the leaf tissue are essential for a successful

infection and disease development. The development of the host and the pathogen are influenced by environment. Therefore, in the management of soybean rust, as in any other diseases, a proper understanding of the triangular factors, namely host, pathogen and environment, and their inter relationship are essential.

1. Urediniospores:

Table 4: Reports on temperature requirement for Urediniospore germination.

Temperature °C			
Range tested	Optimum	No germination between	Reference
20	20	-	Bonde et al. 1976
24	24		McLean (1981), McLean and Byth (1981)
8-32	21-27	<8 and >32	Kitani and Inoue (1960)
9-26	21	4 and 32	Hsu and Wu (1968)
5-35	15-20	<5 and >33	Keogh (1974)
5.5-31 (light)	12-21	<9 and >28	Melching and Bromfield (1975)
5.5-31 (dark)	15-25	<10 and >28.5	Marchetti, et al. (1976)

From the above studies, it is obvious that the urediniospore germination is adversely affected when the temperature is below 10°C and above 28°C. The spores prefer darkness for germination. Light either inhibits or delays urediniospore germination.

The optimum temperature for Uredinia development and sporulation is 17 °C night and 27 °C day. The results of several studies showed that appressorium formation requires almost similar optimum temperature as the spore germination. In the presence of dew the appressorium formation is better but it is not a necessary condition.

Maximum number of lesions was observed following artificial inoculation on leaves kept at 20-25 °C with 10-12 hr of dew and at 15-17.5 °C with 16-18 hr dew. The minimum period

for infection was 6hr at 20-25 °C and 8-10 hr at 15-17.5. No lesions were observed at 27.5 °C.

Table 5: Temperature requirement for Uredinia development (UD) and sporulation (S).

Temperature °C (Duration)		RH	Time to UD and S	Reference
Day	Night			
25 (12)	20 (12)	-	9	Keogh, 1974
27 (13)	17 (11)	75-80%	9	Kochman, 1976
17 (13)	7 (11)	75-80%	14	Kochman, 1976
22 (13)	12 (11)	75-80%	11	Kochman, 1976
32 (13)	22 (11)	75-80%	11	Kochman, 1976

From the first infection, the uredinia formation continued for 56 days. The number of spores released from each lesion over a period of 39 days varied with isolates. Taiwanese and Indonesian isolates produced more than 6,000 spores while the Indian and Australian isolates produced 3,900 and 2,000 spores, respectively.

The number of spores produced over a 36-day period on cultivar, TK 5 was 12,646 per lesion while on the resistant cultivar, PI 230971 was 9,396 in Taiwan.

2. Teliospores:

Teliospores have been observed on several hosts in different locations (table 6).

Table 6: Hosts on which teliospores were observed.

Host	Location	Reference
G. max	Japan	Kitani and Inoue, 1960
G. max	Taiwan	Yeh (1981)
Pachyrhizi angulatus	Taiwan	Sydow and Sydow (1914)
Crotalaria linifolia	Australia	Keogh (1974)

Desmodium rhytidophyllum	Australia	Keogh (1974)
Canavalia villosa	Guatemala	Cummins (1943)
Meibomia supina	Puerto Rico	Arthur (1915)
Lab Lab pupureus	Puerto Rico	Vakili (1981)
Rhyncosia minima	Brazil	Bromfield (1984)
G. max	Brazil	Bromfield (1984)
G. wightii	Brazil	Bromfield (1984)
Phaseolus lunatus	Brazil	Bromfield (1984)
Phaseolus vulgaris	Brazil	Bromfield (1984)
Lab Lab purpureus	Brazil	Bromfield (1984)
G. max	Taiwan	Yeh et al. (1982)

Yeh et al. (1982) reported that for telia production in soybean, the mean temperature should not exceed 25 °C for at least 15 days. Similarly, in the containment house in the plant disease research laboratory in Beltsville, Maryland abundant telia occurred when the temperature was around 20 °C.

At night temperatures of 5, 10, 15 °C, the telia developed in 20, 14 and 10 days, respectively (Kitani and Inoue, 1960; Hsu and Wu, 1968). All the research results indicated that when inoculated plants are maintained at 10-15 °C telia production can be induced in situ or in detached leaves of soybean as well as in other legumes (Yeh, 1981; Yeh et al. 1981; Yeh et al. 1982).

No one has reported the germination of teliospores of *P. pachyrhizi*. Therefore, information on the sexual phases of this pathogen is lacking. It is unclear whether the pathogen is Autoecious or Dioecious.

From inoculum source, the mean rate of spread of soybean rust is about 1m per day (Kitani and Inouye, 1960). However, based on a detailed three seasons study from 1974 to 1977, the rate of spread of the disease varied from 0.03m to 0.45m/day (Casey, 1979).

Casey (1979) concluded that "Extended periods of leaf surface wetness of approximately 10 hrs per day and moderate temperatures (18-26 °C) were necessary for the development of a severe epidemic. Extreme temperatures (greater than 30 °C and/or less than 15 °C) and/or dry conditions retarded the development of the rust. Furthermore, prolonged temperatures above 27 °C appeared to inhibit the fungus even when leaf-surface wetness was theoretically adequate.

From initial inoculation, when conditions are favorable 50% defoliation can occur in about 45 days and in 55 days complete premature defoliation can be expected.

At AVRDC, the Feb/March and September planted crops experience heavy dew on the leaves every day for nearly 40 to 65 days during the growing period with moderate temperature which is excellent for soybean rust development. However, the July planted crop experiences heavy rain and very high temperature and therefore the disease development is retarded.

Research at AVRDC has shown that variation in the growth and maturation of soybean plants is directly related to variations in rust development and rate of rust development. Delay in host maturity delays rust development and reduces the rate of development.

VII. HOST RANGE

More than 95 species in 42 genera in the family Fabaceae have been reported as hosts of *P. pachyrhizi*. Although symptoms have been observed in many hosts, many species do not support sporulation. Such species, which support sporulation alone, are epidemiologically important in disease initiation and spread.

Some legume species may be listed as hosts in one location but in another location or in another season, it may not be listed as hosts due to pathogen differences or differences in host genotypes. Results of greenhouse and field studies may also differ.

Species other than *G. max* which support sporulation of *P. pachynhizi* have been reported by Bromfield (1984).

The species on which *P. pachyrhizi* sporulates and the species other than *G. max* reported to be hosts of *P. pachyrhizi* but for which documentation concerning their ability to sporulate is unclear or lacking is reproduced from Bromfield (1984) in tables 7 and 8.

Table 7: Species, other than *Glycine max*, on which *P. pachyrhizi* has been reported to sporulate.

Species	Remarks
Alysicarpus glumaceus	
Cajanus sp.	

Cajanus cajan (L.) Millsp.	Pigeon pea. Found throughout tropical and subtropical regions and warmer temperature regions from 30 N to 30 S.
Calopogonium mucunoides Desv.	Native to tropical America. Widely cultivated and often escaped in Old World tropics, So. America, Central America, and West Indies.
Canavalia gladiata (Jacq.) DC	Swordbean. Used as cover crop, forage, vegetable. Cultivated in Far East and India. Widespread in tropical Africa, America, Asia, and Australia.
Canavalia maritima	
Centrosema pubescens Benth.	
Crotalaria anagyroides HBK	
Crotalaria dissaromoensis	
Crotalaria linifolia	
Crotalaria pallida Ait.	Smooth crotalaria. Widely cultivated throughout tropics for pasture, forage, cover crop. In Americas from Mexico to Brazil, West Indies, Florida to Mississippi.
Crotalaria sp.	
Delonix regina (Boj.) Raf.	
Desmodium discolor (?)	
Desmodium rhytidophyllum	
Desmodium triflorum (L.) DC	Three-flower beggarweed
Desmodium varians	
Dolichos axillaris E. Mey	
Glycine canescens F.J. Herm	In Australia
Glycine clandestina	In Australia
Glycine falcata	In Australia
Glycine latrobeana (Meissn.) Benth.	In Australia
Glycine soja (syn. G. ussuriensis)	In China, USSR, Korea, Japan, Taiwan

Glycine tabacina (Labill.) Benth.	In Australia, So.China, Oceania, Ryukyu Island, Taiwan
Glycine tomentella (syn. G. tomentosa)	In Australia, So. China, Papua New Guinea, Philippines, Taiwan
Hardenbergi violacea	
Kennedia coccinea	In Australia
Kennedia prostrata	In Australia
Kennedia rubicunda	In Australia
Lablab purpureus (L.) Sweet (Syn. Dolichos lablab)	Hyacinth bean. Grown in tropical subtropical areas for food, forage green manure. Cultivated throughout the warmer parts of the world. In U.S., cultivated as annual in gardens from which escapes and becomes naturalized.
Lespedeza bicolor	
Lespedeza juncea	
Lotus major	
Lupinus angustifolius L.	Blue lupine. Used for forage, silage, green manure and late winter grazing. Introduced and widely grown in Australia, So. Africa, No. Europe, S.E. Unites States.
Lupinus hirsutus L.	
Macroptilium atropurpureum (DC) Urb.	Siratro. Among the most important pasture legumes in Australia, Brazil, & Mexico. Grown naturally from So. Texas through Central America to Colombia, Argentina, and Peru at altitudes from sea level to 2,000 m.
Macroptilium bracteatum (syn. M. bracteatus)	
Macroptilium lathyroides (syn. Phaseolus lathyroides)	
Macrotyloma axillare (E. mey. Verde)	
Medicago arborea	

Melilotus officinalis Lam.	Yellow sweet clover. Cultivated in Europe and America for forage, hay, pasture, soil improvement. Native from Europe to central Asia and western China, south to No. Africa. Introduced in No. and So. Amercia.
Melilotus speciosus	
Mucuna cochinchinensis A. Cheval	
Neonotonia wightii (Arnott) Lackey (syn. Glycine wightii; G. javanica)	
Pachyrhizus erosus (L.) Urban (syn. P. angulatis, P. bulbosus)	Yam bean. Widely cultivated in Central America, China, India. Native to Mexico and south to Guatemala and Nicaragua. Introduced and widely cultivated in the tropics and subtropics.
Phaseolus coccineus L.	Scarlet runner bean. Perennial in tropics. Probably native to Mexico or Central America or both. Cultivated in many tropical areas and as an annual crop in many temperate zones, e.g., UK.
Phaseolus lunatus (syn. P. limensis)	Lima bean. Native to tropical America from Mexico south to Brazil. Introduced to Asia, Africa.
Phaseolus vulgaris L.	Common bean. Native to New World. Widely cultivated in temperate zone, subtropics, and tropics
Pisum sativum	
Psoralea tenax	
Pueraria lobata (Willd.) Ohwi (syn. P. thunbergiana)	Kudzu. Primarily grown for pasture, hay, silage. Native to northeastern Asia. Adapted in U.S. south Virginia and Kentucky west to Oklahoma and Texas.
Rhynchosia sp.	
Rhynchosia minima (L.) DC	
Sesbania sp.	

Sesbania exaltata (Raf.) Rydb.	Colorado River hemp. Native to coastal region of So. U.S., W. to Arizona and So. California and Lower California.
Sesbania vesicaria Elliott	
Teramnus uncinatus	
Trifolium incarnatum	Crimson clover. Introduced into U.S. from Italy in 1819. Widely grown in southern U.S. as an overwintering annual seeded in late summer. Provides winter and spring grazing in mild-winter areas.
Trigonella foenum-graecum L.	Fenugreek. Widely cultivated as a condiment crop. Native to Mediterranean region Near East. Cultivated in No. Africa and India Grow in fog belt of California.
Vicia dasycarpa Ten (syn. V. villosa Roth)	Woolypod vetch. A valuable winter vetch for the U.S. cotton belt.
Vigna mungo (syn. Phaseolus mungo)	Urd. Black gram. Introduced in So. U.S., West Indies, and other tropical areas but cultivated extensively only in India.
Vigna radiata (L.) Wilczek (syn. Phaseolus radiatus)	Mungbean. Long cultivated in India. Introduced early into So. China, Indochina, Java, more recently into East and Central Africa, West Indies, U.S.
Vigna unguiculata (L.) Walp. (syn. V. sinensis)	Cowpea. Long cultivated in Africa and Asia. Now widely cultivated throughout tropics and subtropics. Grown in southeastern U.S.

VII. PHYSIOLOGIC RACES

The first evidence of the presence of races in *P. pachyrhyizi* on soybean came from the work of McLean and Byth (1976). Race 1 was virulent on Wills and avirulent on PI 200492. Race 2 was virulent on both cultivars.

Using three genotypes with known dominant loci for resistance to soybean rust and four different rust isolates from different sources Bromfield et al. (1980) identified four different races of the pathogen (table 8).

Table 8: **Rust infection types induced by four races of *P. pachyrhizi* on three soybean differentials.**

	Infection type[a] on differential host			
Culture	PI 200492 (Gene Rpp1)	PI 230970 (Gene Rpp2)	PI 462312[b] (Gene Rpp3)	Race
Taiwan-72-1	TAN	RB	TAN	PDRL-1
India-73-1	0	RB	TAN	PDRL-2
PR-Comp	RB	RB	RB	PDRL-3
Taiwan-80-2	TAN	TAN	TAN	PDRL-4

[a]TAN = tan-colored lesion: 0.4 mm^2, usually with two to five uredinia per lesion.
RB = reddish-brown lesion: 0.4 mm^2, usually with zero to two uredinia per lesion.
0 = absence of macroscopically visible signs or symptoms.
Abaxial surface of leaves observed two weeks after inoculation.
[b]PI426312 = Ankur.

Again, 9 isolates were tested against 12 different host genotypes and their reactions are presented in table 9 (Bromfield, 1984).

Table 9: **Infection types induced on selected *G. max* accessions by selected Plant Disease Research Laboratory isolates of *P. pachyrhizi*.**

Accession	Isolate								
	Aust 72-1	Aust 79-1	India 73-1	Phil 77-1	Taiw 72-1	Taiw 80-1	Taiw 80-2	Brazil 80-1	PR-Comp
200490	0	...	0	TAN	TAN	TAN	...	...	...
200492 (Rpp1)[b]	0	0	0	TAN	TAN	TAN	TAN	RB	RB
230970 (Rpp2)[b]	RB	RB	RB	RB	RB	RB	TAN	RB	RB
230971	RB	...	RB	RB	RB	RB	...	...	...
462312 (Rpp3)[b]	RB	RB	RB	TAN	TAN	TAN	TAN	RB	RB

200452 (A)	...	...	RB	...	...	...	...	...	...
200466	...	...	RB	...	...	...	...	...	...
200488	...	...	RB	...	...	...	...	...	...
200525	...	...	RB	...	...	...	...	...	...
459024 (A)	...	...	RB	TAN	TAN	TAN	...	...	...
459025	...	...	RB	RB	RB	RB	RB	...	...
Wayne	TAN	TAN	TAN	TAN	TAN	TAN	TAN	RB	RB

[a]Infection types are defined in Table --.

[b]Gene conferring specific resistance.

The soybean germplasm (in addition to those listed above) which may be useful as differentials in *P. pachyrhizi* race "identification" research as mentioned by Bromfield (1984) are:

PI 181567	PI 227687
PI 200455	PI 229358
PI 200465	PI 240667 (HY 2217)
PI 200474	PI 285089
PI 200476	PI 341352
PI 200477	EC 11695 (UPSM-91)
PI 200487	EC 36956 (UPSL-85)
PI 219653	EC 50081 (UPSM-168)
PI 224268	Tainung 4
PI 224270	

VIII. DISEASE MANAGEMENT

1. Chemical means

Initial research to manage soybean rust focused on chemical means. Among the chemicals evaluated, dithane M-45 or mancozeb @2.0 kg/ha can be used as a prophylactic and once

every three-week spray appears to effectively keep the disease under control and reduces the yield loss. Triadimefon and Bayleton also known to give good control and can be applied less frequently than mancozeb.

2. Cultural practice:

By choosing specific planting dates or by using either early maturing cultivars or cultivars with a short pod-filling stage, soybean rust infection can be avoided or minimized. Control of weed hosts and selection of sites where soybean rust does not occur are other means by which one can minimize infection.

3. Host plant resistance/tolerance:

Some cultivars or breeding lines yield well inspite of severe rust infection. Such cultivars are known to be tolerant.

Specific or vertical resistance is governed by one or more alleles and is generally expressed as hyper sensitive response.

Horizontal or general resistance, assumed to be governed by several loci each having a small, additive effect, reduces the rate of development and amount of rust.

In 1960s, three accessions were identified as resistant to soybean rust. They are PI 200492, PI 200490 and PI 200451 (Cheng and Chan, 1965). Utilizing PI 200492, three improved cultivars with resistance to soybean rust were developed: Tainung No. 3, Tainung No. 4 and Kaohsiung No. 3 (Shanmugasundaram, 1976).

Singh and Thapliyal (1977) using three resistant parents, UPSL 18, UPSL 85, UPSS 3 provided genetic data for a dominant allele for resistance.

McLean (1981), McLean and Byth (1981) also reported that resistance to soybean rust in PI 200492, Tainung No. 3 and Tainung No. 4 using Australian isolate Q-1, was dominant to susceptibility. The research results of McLean and Byth suggested that Tainung No. 4 has probably two different resistant alleles. PI 200492 and Tainung No. 3 have the same resistant allele. Similarly, PI 200492 and Tainung No. 4 have one common resistant allele. McLean and Byth (1981) assigned the designation Rpp1 for the dominant allele conditioning rust resistance in PI 200492.

From the screening of germplasm at AVRDC in cooperation with Plant Disease Research Laboratory at Beltsville, Maryland and Dr. Hartwig, PI 230970 and PI 230971 were

identified to have semi compatible reaction (RB type).

Hartwig and Bromfield (1983) reported the relationship between the alleles for specific resistance in PI 200492, PI 230970, and PI 462312 (Ankur) using rust isolates India 73-1 and Taiwan 72-1. The infection types produced are shown in table 10.

Table 10: Infection types observed for three resistant genotypes with rust isolates.

Acc. No.	Infection type	
	India 73-1	Taiwan 72-1
PI 200492	O[a]	TAN
PI 239070	RB	RB
PI 462312	RB	TAN

[a]Immune or nearly immune.

Based on their genetic study, Hartwig and Bromfield (1983) assigned allelic designation for each genotype as follows:

PI 200492	Rpp1	Rpp1	rpp2	rpp2	rpp3	rpp3
PI 230970	rpp1	rpp1	Rpp2	Rpp2	rpp3	rpp3
PI 462312	rpp1	rpp1	rpp2	rpp2	Rpp3	Rpp3

In 1986, Hartwig reported a fourth major allele in PI 459025. PI 459025 was found to be resistant to the soybean rust isolate Taiwan 80-2, Taiwan 72-1 and India 73-1. The genotype designation of PI 459025 was rpp1 rpp1 rpp2 rpp2 rpp3 rpp3 Rpp4 Rpp4.

In scarlet runner bean (*Phaseolus coccineus*) reciprocal cross differences in resistance reaction suggested Vakili (1977, 1979) to postulate that maternal plasmagenes influence on soybean rust reaction. It is unclear whether such response is present in *G. max*.

Historically, at least in the rust disease utilization of single gene resistance have given rise to cultivars whose resistance is short-lived. Loss of resistance is due to the occurrence of new race or races and change in the virulence of the existing race. The route to a reliable management of the disease is not via single gene resistance.

Variability for soybean rust reaction exists in wild annual and perennial *Glycine* species.

A number of accessions in *G. soja*, *G. canescens*, *G. clandetina*, *G. tabacina*, and *G. tomentella* have been observed (Burdon, et al. 1981a). How useful the resistance sources from wild species are questionable.

Breeding for durable soybean rust resistance combined with other desirable agronomic traits including yield is the major goal of soybean breeders. In the early years soybean breeders in Taiwan, Australia, India and many SE Asian countries developed cultivars which have specific resistance (table 11).

Table 11: Cultivars developed using specific rust resistant alleles.

Cultivar name	Country	Resistant source
Tainung No. 3	Taiwan (1968)	PI 200492
Tainung No. 4	Taiwan (1971)	PI 200492
Kaohsiung No. 3	Taiwan (1971)	PI 200492
Ankur	India	PI 462312
Dowling	U.S.A. (1978)	PI 200492

Recognizing the instability of specific resistance the plant pathologists and plant breeders were looking for alternate approaches. Rate reducing or slow rusting was explored. But it is very difficult to measure in a segregating population.

Therefore, tolerance was the major approach used by most people. The mechanism involved in tolerance probably includes a combination of specific resistance to one or more isolates, slow rusting and general resistance.

At AVRDC, the segregating population is subjected to severe soybean rust stress in the field by artificial inoculation. Single plants with good pod and seed development are selected inspite of soybean rust and advanced to next generation. The procedure is repeated in subsequent 'inbreeding' generations. Finally the stable pedigrees are evaluated with and without fungicide treatment. Those with high yield potential under no fungicide treatment are selected as tolerant selections.

A rust tolerance index (RTI) was found to be a better predictor of soybean rust tolerance and high yield potential.

$$RTI = (Yp \times Ys)/ (Y_p)^2$$

Where Yp is the yield of a given genotype in no rust field (fungicide protected);
Ys = yield of a given genotype in rusted field (no fungicide);
Y_p = mean yield in no rust field.

The rust intensity can be determined from the ratio between the mean yield in the rusted field and mean yield in no rust field

$$RI = 1 - (Ys/Y_p)$$

The higher the value of RTI for a genotype, the higher is the rust tolerance and yield potential. The RI is also included in the estimation of RTI.

Genotypes can be conveniently grouped into four groups based on their performance in rust and no rust environments; Group A. genotypes perform equally well in rust and no rust environment. Group B. genotypes, which do well only in no rust environment. Group C. genotypes yield relatively higher in rust environment and Group D. genotypes that perform poorly in both rust and no rust environment.

The 3-D plots for two yield trials with and without rust is shown in Fig. 1 and 2. The genotypes in four groups are shown under the figures. The soybean rust intensity was slightly different in the two trials.

Trials conducted in different seasons show the instability of some of the genotypes in their rust tolerance and yield potential. Therefore, it is important to evaluate in different seasons and possibly in different locations before arriving at major conclusions.

IX. RESOURCES FOR AFRICA

Based on historical information on soybean rust, Bromfield (1984) speculated that soybean rust in Africa might be a different *forma specialis* since it was observed on hosts other than soybean. Since 1984 sporadic reports indicated possible occurrence of soybean rust in Africa. Now it appears it is true.

Known sources of resistance/tolerance to soybean rust are available which include PI 200492, Tainung No. 4, TK #5, PI 462312 (Ankur), PI 230970, PI 459028 (A), PI 459025, PI 200466, PI 200488, PI 339871, PI 200525, PI 200490, PI 230971, Shih Shih, PI 239871B, Wayne, PI 239871A, GC 00138-29, GC 6002-8-7-7-18, GC 84040-7-1, GC 86018-427-3, and SRE-C-56A.

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An extensive bibliography on soybean rust published by AVRDC is available. Rate reducing resistance was observed in SRE-Z-11A, SRE-Z-11B and SRE-Z-15A.

Knowledge of the pathogen, interaction of the pathogen and environment and interactions of the pathogen with hosts, both cultivated and wild, and the three way interactions are fragmentary and limited. There is lot of opportunity to conduct useful and meaningful research to better understand and enrich the knowledge base.

AVRDC will continue to serve as a resource base. However, research on soybean rust has been largely scaled down at AVRDC for want of sustained resources.

X. SUMMARY

Soybean rust in epidemic form at present is limited to the tropics and subtropics. Strains of the pathogen from the Orient are consistently more virulent and aggressive than those from the Americas and Africa that have been studied. The environmental factors required for favorable development of soybean rust, moderate temperatures and moist conditions, are present largely in the tropics and subtropics. Soybean rust is not seed borne. Autoecious or heteroecious nature of the pathogen is unknown. Presence of large number of wild and weed hosts appear to serve as the primary inoculum source. The monsoon wind current and the weather patterns play a vital role in carrying the inoculum although no experimental evidence is available.

Even if the soybean rust is introduced into the temperate countries, due to assumed lack of overwintering mechanism it may not get established. For example, there is lack of severe rust in Hokkaido compared to Kyushu in Japan. Uredinial stage cannot overwinter. Telial stage probably can. But its germination and reinfection has not been recorded.

A few races of the pathogen have been identified. Race specific resistant alleles have also been identified in the host. However, such resistance is short lived due to the presence of multiple virulence in the pathogen.

With the available knowledge on the management of soybean rust, it may be possible to manage the disease with minimum yield loss. Anticipating the disease prophylactic measures such as planting rust tolerant cultivars, advance application of suitable chemicals in a judicious manner are required. Resistant and tolerant sources of germplasm are available. Utilizing them it should be possible to develop rust tolerant cultivars within a reasonable period.

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3. REPORT ON SOYBEAN RUST IN ZIMBABWE

Rex Tattersfield, Head of Research, Seed Coop, Harare & Clive Levy, Plant Pathologist, CFU Commodities Technical Team, Harare

1. The disease arrived in Zimbabwe in late January 1998 but was not initially identified, being confused with bacterial pustule disease.

2. By the end of the season, probably 65% of the total hectarage was infected of which 30% was serious and yields were considerably reduced. The worst hit areas were Enterprise, Lions Den, Chinhoyi North and parts of the Mazoe Valley, especially near Glendale.

3. The occurrence of rust coincided with a very wet January with a lot of moist tropical air moving in from the north. It probably came from Uganda and neighbouring states where it is known to occur.

4. Identification was a problem initially, a description has been circulated to growers as follows:

   a) Initially rust appears on the lower leaves as a yellow discoloration (almost mosaic- like) on the upper surface. It rapidly moves upwards through the canopy.

   b) Numerous, small grey to russet coloured rufts of the fungus (at first appearance they resemble spider mites) are sen on the lower surface of affected leaves. These may been seen on both yellowed and green leaves.

   c) In more advanced stages of infection, the lower and middle canopy leaves become distinctly yellow and the plant look "diseased". On the under side of these, brown scarring is seen both as pin-points and more extensive lesions.

   c) In hot dry weather, a light brown cloud of spores (like dust) may be seen above the canopy when the wind blows or the canopy disturbed. Tip-burning and sun scorch of green leaves may be seen. Leaf drop will be premature and rapid.

5. The effect of the diseased on yield varied with severity, the results of some fungicide trials indicate how severe the disease can be.

Table 1: Yield response from controlling soyabean rust with effective fungicides at Rattray Arnold Research Station in conjunction with Agricura (Pty) Ltd.

	Trial 1	Trial 2	Trial 3
Planting Date	08.12.97	30.12.97	10.01.98
Cultivar	Roan	Roan	Soprano
Spraying dates in 1998	6/2 20/2	5/3 25/3	5/3 25/3 15/4
Yield with fungicide kg/ha	3099	1930	1553
Yield no fungicide kg/ha	1792	530	174
Yield increase from fungicide %	73	264	793
Yield decrease from disease %	42	73	89

Disease invaded all of the plants at the end of January. The relative yield reduction increased with delay in planting date showing that the earlier in the plant's life infection occurs the greater the yield reduction. In the worst case with the January 10th planting, infection started to appear pre flowering, in that case there was almost no yield without the use of a fungicide.

6. Control measures

a) **Cultivar resistance**

No cultivars nor breeding lines in the Seed Co programme showed any useful level of resistance or tolerance. A few lines appeared to rust slower than the rest but their yield were still considerably depressed by the disease. Seed Co will embark on breeding programme and has acquired lines reputed to have resistance from DeKalb, U.S.A., U.S.D.A, U.S.A., AVRDC, Taiwan and Indonesia. However, this material mist all be screened for reaction to the rust biotype in Zimbabwe and a breeding programme set up to develop resistant cultivars. One other possibility is to use the Pgip gene and certain other genes which can confer general resistance to fungi. An update on breeding procedures will be obtained by Jacob Tichagwa when he visits Thailand and Taiwan (AVRDC) in early November, and discussions will beheld in

Zimbabwe and South Africa in October with Dr Sundar Shanmugasundaram.

Any breeding procedure will take a number of years, probably 7-109, before resistant cultivars can be developed, so far some time to come control will have to be through the use of chemicals.

b) **Fungicides**

When the rust outbreak was confirmed, the Chemical Registration Authority was immediately notified of the magnitude of the epidemic, and told that COPA had circularised all its members to spray one of four chemicals to save their crops (this is not the normal procedure, but time was of the essence if the Authority did not agree, it had at least been informed). The Authority was also told to expect applications for emergency (temporary) registration from the chemical companies.

A number of these companies hastily designed and conducted fungicide screening trials at the end of the 1997-98 season. These were rather uncoordinated and varied in quality (the data was confidentially presented to COPA). They did indicate, however, that certain chemicals, especially the triazole group, were very effective in the control of soyabean rust. As a result of these trials six chemicals have been granted emergency (temporary) registration in the coming season:

CHEMICALS FOR THE CONTROL OF SOYABEAN RUST

TRADE NAME	CHEMICAL NAME	RATE/ha	COST/ha
Alto® 100SL	cyproconazole	300 ml	approx. $750.00
Folicur® 250EC	tebuconazole	1 000 ml	$902.40
Funginex®	triforine	1 500 ml	$877.50
Punch® Xtra	flusilazole/carbendazim	350-500 ml	$284.03-$405.75
Score® 250EC	difenoconazole	300-450 ml	$404.10-$606.15
Shavit®	triadimenol	500 ml	$390.00

Two to three sprayings of any of these chemicals will be necessary to control the disease.

Note: The cost stated is that of the chemical only per spraying (as at 31.08.98) - it does not include application. Prices are likely to increase with the currency devaluation.

(Alto®, Folicur®, Funginex®, Punch® Xtra, Score® and Shavit® are all registered trade names)

Having had discussions with the chemical companies, COPA has drawn up certain guidelines based on the following premises:

1. The pathogen will infect soyabeans much early than last season, and over a wider area. Infection will be governed by the prevailing environment.

2. Farmers generally will not be able to recognise early symptoms, and therefore, scouting will not be possible.

3. Chemical control is the only means to counter yield loss.

A) Where the risk of rust is high (last season's experience and ideal environmental conditions), but where good scouting is not possible, the farmer should apply an initial preventative spray 4-5 weeks after planting, and thereafter, probably two further sprays at 21-day intervals.

B) Where the risk of rust is low and/or where farmers can effectively scout for the early symptoms, they must react to a disease outbreak and apply the first fungicide treatment immediately, and thereafter, further sprays at 21-day intervals as necessary.

In order to give farmers early warning of rust in their areas, and to educate them on how to identify the disease, a series of rust "traps" (approx 0.25 ha plots) will be planted throughout the main growing area in late October to early November (3-4 weeks early than normal). These will be intensively scouted, and as soon as rust appears, warnings will be sent out and also provide the venue for discussion groups.

There will be a large research programme on fungicides and their use conducted by COPA, Seed Co and the chemical companies in the coming season.

4. QUESTIONS AND ANSWERS

Chaired by Dr K Pakendorf, ARC-Grain Crops Institute

1. **Rex Tattersfield: How do you select for tolerance in a breeding program, especially in early generations?**
Answer dr Sundar: Soybean rust affects the pod development and bean filling. A well adapted and high yielding cultivar is crossed with an accession with tolerance. From the F2-generation the population is subjected to rust stress without applying fungicide. Single plant selection is based on pod and seed development.Ill-filled and shrivelled podded plants are discarded. The selection intensity is about 5 to 10%. It is repeated in the subsequent generations until F5 or F6. Thereafter the inbred pedigrees are evaluated with and without the fungicide and tolerant selections are identified.

2. **Anthony Jarvie: The RSA has a typical variation in climate across seasons. How do we select for resistance in such conditions?**
Answer: You are then forced to do selections in different locations and hope for good rusting conditions.

3. **Julian Ward: How do you artificially inoculate soybean rust?**
Answer: A soybean rust nursary is maintained throughout the year by means of sequential planting of a susceptable cultivar. Early morning fresh spores are harvested by blending infected leaves in water. The desired number of spores per ml is determined and used for inoculation. A sticker is used to prevent run off. Infection is promoted by misting or other means of increasing the humidity around the inoculated plants.

4. **Rex Tattersfield: In Zimbabwe the fields are damp and wet in mid season and this seems to encourage rust outbreak if there is a inoculum source.**
Responce: Yes, those are ideal conditions and the inoculum was probably not present before!

5. **Klaus Pakendorf: The pathogen does not seem that aggressive if only 4 races have been reported?**
Answer Sundar: Bromfield has identified only 4 races. It is likely that there are many more races waiting to be identified. It is also unclear whether new races arise from bisexual or other means. Unlike wheat rust, there are very few researchers

working on soybean rust. Comment from Clive Levy: There is no selection preasure in soybeans for more races.

6. **Gerhard Scholtemeijer: Soybean rust does not seem to be a problem in any of the major soybean producing countries. Do we know if any of the US varieties are resistant to soybean rust?**
Answer Sundar: *We at AVRDC have screaned almost all the USA soybean varieties in batches since 1974. Wayne was the only variety with some level of resistance. Dowling was developed at AVRDC, Stoneville, MS and Texas A&M from accesion PI 200452 (resistant). Since then several leading USA varieties have been crossed with resistant and tolerant accesions and the progeny populations are ready for selection in the event of a rust epidemic.*

7. **Rex Tattersfield: It seems to me that the climate in Brazil should be ideal for development of rust?**
Answer Sundar: *The pathogen has been reported in Brazil but the particular race is not very aggressive. Bazilian temperatures also do not seem to be conducive for epidemic development of the disease.*

8. **Gerhard Scholtemeijer: will Zimbabwe screen their germplasm for resistance?**
Answer Rex Tattersfield: *We are busy collecting resistant sources and host differentials. We will think carefully and plan accordingly.*

9. **Munro Griesel: Does South Africa have climatic conditions conducive for rust outbreak?**
Answer Michiel Smit: *The high rainfall and high humidity conditions in the Berville to Newcastle area have the highest probability to develop the disease.*

10. **Schalk van Wyk: Could there be an interaction between rust and the frogeye fungus?**
Rex Tattersfield: *Frogeye susceptible varieties were also thumped by rust. It would anyway not be possible to manage the interaction should there be any.*

11. **Faan Botes: How does the pathogen survive?**
Answer Sundar: *The pathogen can only survive on a live host and will die out in temporate areas where frost occurs.*

12. **Tokkie Liebenberg: Will the symptoms for soybean rust be recognizable on other hosts?**
Answer Sundar: *Yes, the tan symptom will be easily recognizable.*

5. STRATEGY PLANNING

Chairman dr K Pakendorf, Deputy Director, ARC Grain Crops Institute

1. Schalk van Wyk recommends that South Africa not take the initiative in identifying the races involved and in screening germplasm but that the RSA rather support the Zimbabwe initiative.

2. Chris Loubser requests that a phase 1 ARC soybean cultivar trial be send to Harare for rust evaluation.

3. Clive Levy recommends that a rust trap be planted in the Northern Province to act as a early warning system.

4. Dr Sundar suggests that a session on soybean rust be scheduled at the WSRC in order for scientists from RSA and Zimbabwe to interact with those who have experience in dealing with the disease.

5. Schalk van Wyk and Michiel Smit are requested to compile a press release advising soybean producers on the outbreak of rust in Zimbabwe and to be on the lookout this coming cropping season.

6. Michiel Smit recommends that the ARC include rust resistance as an objective in their soybean breeding programme and maintain a progency population for use should there be an outbreak of rust in South Africa.

7. Rex Tattersfield emphasizes the need for regional interaction. Clive Levy suggests that the RSA budget for intraregional travel. It is recommended that RSA scientists visit Zimbabwe during February to experience the disease first hand.

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