IV. BIOLOGY OF THE SOYBEAN RUST PATHOGENS

A. Taxonomy of the casual agents

Two species of the genus *Phakopsora* infect soybeans. *Phakopsora pachyrhizi* Sydow is the Australasian species (4-6) and *Phakopsora meibomiae* (Arthur) Arthur is the tropical Latin American or New World species (6) (Appendix I-C). There is a third rust fungus reported on soybeans from sub-Sahara Africa, but the correct name for this species is still uncertain. Details on the taxonomic differences between the two described species are presented (6) (Appendix I-C).

B. Alternative hosts

Phakopsora pachyrhizi has been reported to produce natural infections on 31 plant species in 17 genera of legumes, and 60 species of plants in 26 additional genera when inoculated (6-8) (Appendix I-C). There is a large number of synonyms reported on these numerous hosts. Similarly, *Phakopsora meiboniae* has as many synonyms and a wide host range, which includes 42 species in 19 genera (6) (Appendix I-C). Twenty-four species in 19 genera are common alternative hosts for both fungal species. Among these many hosts is the vining weed *Pueraria lobata* commonly known as kudzu vine, a common weed pest in the southeastern U.S.

C. Symptomology

The most common symptom is tan to dark brown or reddish brown lesions or spots with one to many raised round uredia with an apical opening (Appendices I-A & -C). The disease develops first with small, water-soaked lesions, which gradually increase in size, turning from gray to tan or brown (Appendix I-E). They assume a polygonal shape restricted by leaf veins and may eventually reach a size of 2 to 3 mm^2. Lesions can appear on petioles, pods, and stems, but are most abundant on leaves, particularly on the under surface. The number of uredinia per lesion increases as lesions age.

D. Mycological description (4-6)

1. Teleomorph: *Phakopsora meibomiae* (Arthur) Arthur: telia are irregularly 1- to 4 (-5)-spore layered. Teliospores have walls that are cinnamon to light chestnut-brown and 1 .5 to 2 µm thick but thickened apically to 6 µm in the outermost spores.

Anamorph: *Malupa vignae* (Bresadola) Ono, Buritica & Hennen, comb. nov.: uredinial sori minute, scattered or in groups on discolored lesions, subepidermal in origin; urediniospores obovoid to broadly ellipsoid, 16 to 31 x 12 to 24 µm, germ pores four to eight or 10 mostly scattered on an equitorial zone and the walls are minutely or densely echinulate, colorless to pale yellowish brown.

2. Teleomorph: *Phakopsora pachyrhizi* H. Sydow & Sydow: telia are crustose, 2- to 7-spore layered, chestnut-brown to chocolate-brown, and subepidermal.

Anamorph: *Malupa sojae* (P. Hennings) Ono, Buritica & Hennen, comb. nov.: uredinial sori minute, scattered or in groups on discolored lesions, subepidermal in origins; urediniospores are obovoid to broadly ellipsoid, 18 to 34 x 15 to 24 µm, germ pores on an equitorial zone or scattered and the walls are densely echinulate, colorless to pale yellowish brown.

V. GEOGRAPHICAL DISTRIBUTION OF THE PATHOGENS ON SOYBEANS

Soybean rust occurs in Africa, Australasia, the Caribbean, Hawaii, and Central and South America. *P. pachyrhizi* is present in Australasia where it predominates in Eastern Australia, Eastern Asia and the islands between those land masses including Japan, The Philippines, and Taiwan. The disease occurs in India and Nepal, although its frequency and distribution are not defined. *P. meibomiae* is found in the Caribbean, and South and Central America although there is little information on its frequency and distribution. There is also a soybean rust in Africa, which at this time is suspected to be *P. pachyrhizi* with unknown frequency and distribution. The species recently found in Hawaii is apparently *P. pachyrhizi*, although its definitive identification is under consideration.

VI. EPIDEMIOLOGY OF SOYBEAN RUST

Most of the known epidemiology of the two pathogens that cause soybean rust is based on research from *P. pachyrhizi*, the species that presumably predominates in Australasia. There is considerably less research on rust epidemics caused by *P. meibomiae* and proper documentation on yield losses is lacking. Under field conditions, *P. pachyrhizi* can infect soybean leaves early in the season. Precipitation, 6-hour dew periods or longer, and moderate temperatures enhance rust severity. After initial infection, the disease progresses slowly and may take several weeks for urediniospores to increase. Overhead irrigation increases rust severity as does precipitation which aids in its spread and provides leaf moisture for infection. Lower leaves often are heavily infected before the upper leaves become infected. The pathogen progresses rapidly from lower to upper leaves when plants begin to flower. At that time, lower leaves senesce earlier than lower leaves on noninfected plants, and heavily infected plants may mature up to 2 weeks earlier.

Disease progress curves (DPC) are used to monitor rust epidemics based on recording severity of rust on leaves over time. These curves can be used to obtain values for the area under the disease progress curves (AUDPC). The AUDPC and DPC are used to compare epidemics when studying infection rates, testing soybean lines for their reactions to rust, and testing fungicide efficacy, irrigation and other factors that influence rust development. The rate of rust development also is dependent on soybean maturation. Later maturing lines often have less rust than earlier maturing lines when evaluated at the same time, because of the differences in soybean maturation. A delay in plant maturation by increasing the photoperiod during the reproductive growth stages cause a simultaneous delay in the rate of rust development. Erroneous conclusions may occur when assessing rust severity if corrections are not made for differences in host maturity. To correct for differences in host maturity, relative life time as the time element may partially or completely delete the differences in maturation. The interrelationship of the host and the pathogen must be considered when evaluating disease progress curves for resistance, forecasting and modeling, and in fungicide control experiments. Along with assessments of leaf severity, factors like defoliation and/or percentage of green leaf area which accounts for defoliation, are useful in comparing treatments whether they are fungicide applications or tests of different cultivars and lines.

VII. EFFECT OF THE PATHOGENS ON SOYBEANS

Yield losses of up to 80% have been reported from experimental trials in many countries throughout Australasia. Heavily infected plants have fewer pods and lighter seeds. Marketable yields are even less because of poor seed quality. One unpublished report from central India indicated losses of up to 90% occurred in severely affected areas (M. S. Bhale, personal communication).

A number of reports have quantified disease parameters like leaf severity, defoliation, pustule counts, and area under curves to yield components. A critical point model, using leaf severity at flowering, was shown to be a good predictor of yield loss. Others have regressed disease parameters measured over time to yield and yield losses. This kind of information is useful for understanding the relationship of disease to yield and for predicting yield losses in soybean production areas that so far do not have the disease, such as the continental U.S.

VIII. POTENTIAL THREAT TO U.S. CONTINENTAL SOYBEANS

Although the pathogen has not been found in the continental U.S., its occurrence in Hawaii has renewed interest in the event that this disease reaches the mainland. It has been predicted that losses greater than 10% could occur in nearly all of the soybean growing areas, with greater losses in the Mississippi delta and southeastern coastal areas. Losses in the main soybean production area in the midwest would depend on several factors including its capacity to move during the season and the establishment of the fungus (its ability to survive from one growing season to the next). Since these factors are unknown and difficult to test, the best estimates of losses are speculative, but are informative for analyzing the potential threat of rust in the continental U.S. The best control now is to prevent the pathogen from entering into the continental U.S. If the pathogen should occur in the continental U.S., then fungicide control and eradication of the fungus should be top priority. In the event that the pathogen occurs from season to season, then the eradication of alternative hosts along with rapid development and deployment of host resistance and/or tolerance should be implemented.

IX. MANAGEMENT OF SOYBEAN RUST

A. Resistant and/or tolerant cultivars

Four dominant, independently inherited genes for resistance to *P. pachyrhizi-Rpp1, Rpp2, Rpp3, Rpp4*-have been identified in PI 200692, PI 230970, PI 462312 (Ankur), and PI 459025, respectively. These lines, as well as seven others, are suspected of containing genes for resistance. PI 239871A and PI 239871B (G. *soja),* PI 230971 and PI 459024, and the cultivars Taita Kaohsiung-5, Tainung-4, and Wayne have been used as differentials to identify nine races at the Asian Vegetable Research and Development Center, in Taiwan. The predominant race was compatible with three or more of the differentials, indicating that some races already possess multiple virulence factors to known and suspected genes for resistance (Appendix I-A & -B).

Genes for resistance also occur among the wild *Glycine* spp. from Australia. Efforts are b

being made to transfer this resistance to G . *max* (Appendix I-D & -E).

Rate-reducing resistance has been demonstrated; however, it is difficult to evaluate because the rate of rust development is dependent on soybean development and maturity. Currently, evaluation for this type of resistance is time consuming; this limitation prevents the use of this method for screening large populations (Appendix I-B).

R.L. Bernard, Department of Crop Sciences, UIUC, has released three lines, L85-2378 *(Rpp1)*, L86-1752 *(Rpp2 Rps1-k)*, and L87-0482 *(Rpp4)* for evaluating for resistance to the rust pathogen, *P. pachyrhizi* (Appendix Il-A). E. E. Hartwig, USDA, ARS, Stoneville, MS, has released D86-8286 for further evaluation and a second line which has PI 459025 as a source of resistance (Appendix Il-B). From the breeding program at Chiang Mai, Thailand, three tolerant cultivars have been identified: 8517-3-4, 8523-11-2, and 8520-102-7-1 (Appendix II-C).

L. 0. Vodkin, Department of Crop Sciences, UIUC, has isolated a RAPD marker located near the *Rpp1* gene. It is anticipated if the RAPD marker is linked to a resistant gene, it could be converted to a restriction fragment length marker that would provide a means for screening for resistant gene (Appendix II-D).

B. Chemical control

Information on well-planned programs for the chemical control of soybean rust is not available. As an example, the economic threshold of soybean rust is not known. The occurrence and severity of soybean rust varies among regions, from season to season, and even within regions in the same season. The epidemiology of soybean rust is not understood and precise forecasting of soybean rust epidemics is not possible with the present knowledge. It would be ideal to have this information in order to develop an effective, cost-saving fungicide spray program for the control of soybean rust that would use the minimum amounts of pesticides and labor.

All of the work on the use of fungicides for the control of soybean rust has been done earlier in the Eastern Hemisphere where the disease has been a problem for many years. A number of systemic and nonsystemic fungicides used as sprays or seed treatments, alone or in combination, have been tested and the data published. A summary of the compounds most widely used and the sources of the data are presented (Table 1).

The fungicides or combinations used as sprays that have offered the most promise are listed (Table 1). Two reports stated that benomyl was effective while another report stated that it was not. The rates used were often not stated. Dithane M-45 at 2.2 kg of 80% w/w per 200 to 400 1/hectare for spray application and 2.2 kg of 80% w/w per 50 l/hectare for aerial application is suggested for control of soybean rust in Australia (J. K. Kochman, Department of Primary Industries, Queensland, Australia, personal communication).

In general, for effective fungicide control of soybean rust, with any materials so far tested, at least three to five or more fungicide applications are required on a weekly or 10-day basis. Thus, spraying with fungicides for soybean rust is time-consuming and expensive. Some of the yield loss data from the endemic areas of soybean rust show that in certain areas the use of fungicides might be warranted where losses of 80% reductions have been recorded. However, when losses are 10 to 15%, it may be difficult to justify the cost of fungicides. More work must be done on timing, rates, number of applications, plant age and other factors affecting the use of fungicides for control of this disease.

C. Seed treatment

Since *P. pachyrhizi* is not seedborne in soy-beans, this suggests that other means are involved in the long-range dissemination of the pathogen. In addition, the urediniospores are short-lived away from host tissue. Thus, seed treatment per se cannot be considered as an effective means for direct control of this fungus on soybean seeds.