Plant Protection Bulletin 34 : 109~124, 1992

Epidemiology of Soybean Rust and Breeding for Host Resistance

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(Accepted for publication : March 9, 1992)

ABSTRACT

Wang, T. C., and Hartman G. L. 1992. Epidemiology of soybean rust and breeding for host resistance. Plant Prot. Bull. 34: 109-124

Soybean rust, caused by *Phakopsora pachyrhizi*, is a major disease limiting soybean production primarily in the tropics and subtropics of Asia. Research at the Asian Vegetable Research and Development Center (AVRDC) has focused on monitoring disease development; evaluating yield losses; obtaining basic information on the biology of the fungus; and on finding sources of resistance and developing these sources into breeding lines. Rust was monitored on one moderately resistant and three susceptible lines at five locations in Taiwan during three separate seasons. Apparent infection rates were similar within lines over locations and seasons. Several experiments showed that soybean maturation was significantly positively correlated to the rate of rust development whereas effects due to the enviroment and the host genotype were not as highly correlated. To compare soybean lines, methods were developed to compensate for differences in host maturities. The best method used the relative soybean life time (RLT) as a time element from 0 to 100. The time between planting and maturity was converted to a percentage of the soybean life cycle completed. Factors related to pathogenic diversity of the fungus, and the effect of environmental parameters were studied. Nine races were identified from forty-two isolates using a differential set consisting of 11 lines. The predominant races were complex with multiple virulence factors for compatibility on the differentials. Studies on leaf wetness and temperature indicated that the optimum temperature for urediospore germination was 15~ 25℃ ; the minimal dew period for infection was 6 hours at 20~ 25℃ and 8-10 hours at 15~ 17.5℃ ; and a mean night temperature below 15℃ greatly reduced lesion numbers or completely prevented lesion development. Field studies showed that precipitation was a critical factor in the development of epidemics. It was used to predict rust severity, and was more important than frequency and intensity of the infection period which consisted of leaf wetness, temperature, and their interaction. Difficulties associated with identifying and quantifying rate-reducing resistance and the ineffectiveness of race-specific resistance have brought about techniques to develop higher soybean yields with tolerance to rust. Also techniques were developed to better quantify and understand the components involved in partial resistance. In other studies, new sources of resistance were identified in accessions of the wild perennial *Glycine* species.

(Key words: *Phakopsora pachyrhizi*, infection rates, perennial *Glycine* spp., yield losses, races)

INTRODUCTION

Soybean rust, caused by *Phakopsora pachyrhizi* Sydow, is one of the major diseases limiting soybean yield in the tropics and subtropics of Asia. *P. pachyrhizi* occurs in both the Eastern and Western Hemispheres including Africa. The disease causes significant economic losses in Australia, India, Indonesia, Japan, Korea, Peoples Republic of China, Philippines, Taiwan, Thailand, and Vietnam (Fig. 1) (1,3,20). Yield losses ranging from 10 to 80% have been reported (8). Rust may be regarded as a threat to most soybean producing

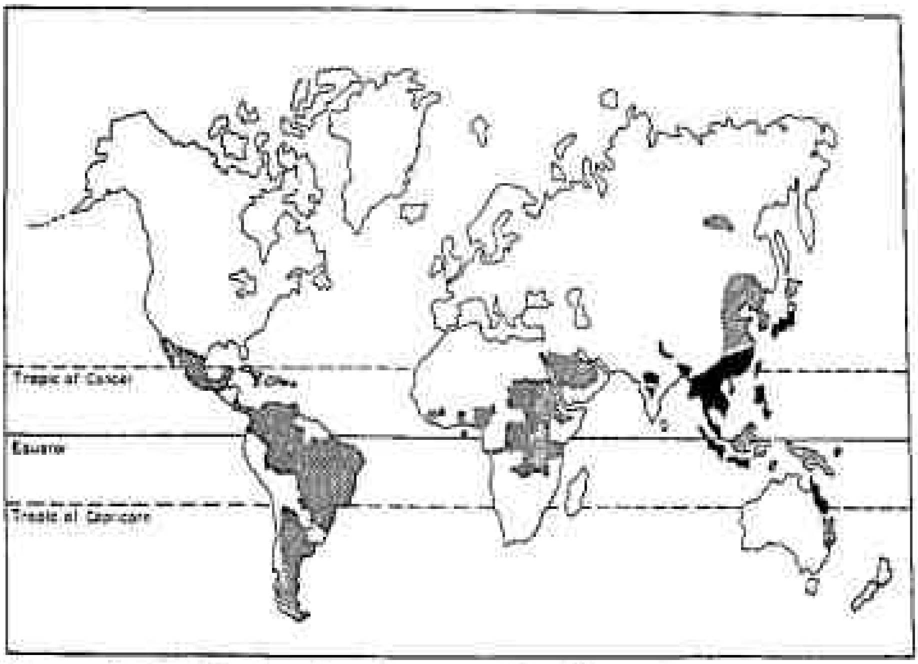

Fig. 1. Geographic distribution of soybean rust (shaded areas) with darker shaded areas representing countries where soybean rust has been reported to cause considerable yield reduction (20).

regions of the world, although isolates from Asia were reported to be more virulent than those from other locations(5).

In Taiwan, rust frequently causes severe losses to soybeans not protected by fungicides. In general, severe rust has been observed in Chiayi, Hualien, Kaohsiung, Pingtung, Tainan, and Yunlin counties. Yield losses as high as 50% have been reported (25). Heavily infected plants have fewer pods and lighter seed(9, 26). Marketable yields are even less because of poor seed quality (10). In Hualien county, rust is a limiting factor to soybean production in the spring season.

Soybean rust has been a serious disease in Southeast Asia for many years and before 1980 there were relatively few reports on its epidemiology. Recognizing the paucity of information about this disease in the Eastern Hemisphere, and the potential danger to soybean production in the United States, the United States Department of Agriculture (USDA) Plant Disease Research Laboratory and the Asian Vegetable Research and Development Center (AVRDC), started a cooperative project in 1978 to study the epidemiology of soybean rust. In this report we highlight some of the data collected during this cooperative period, and use other published reports and data primarily collected at AVRDC to discuss the epidemiology of soybean rust and the utilization of host tolerance/resistance.

EPIDEMIOLOGY

Apparent infection rates. A series of field experiments were conducted to determine disease progress under different environmental conditions (16) Seeds of three susceptible lines, Taita Kaohsiung No. 5 (TK#5), Tainung No 4 (TN#4) and Shih-shih (G-38); and one moderately resistant line, PI 230971 (G8587), were planted at AVRDC, Hualien District Agricultural Improvement Station (DAIS) near Hualien, Kaosiung DAIS in Pingtung, Taitung DAIS near Taitung, and Taiwan Agricultural Research Institute (TARI). The sites were selected because they were located in soybean production areas or at experiment stations where soybeans were grown. A modified Horsfall-Barratt rating system (10) was used to assess disease severity. The apparent infection rates (r) were calculated according to van der Plank's formula(26) : $r=[1/t_2-t_1][\log_e(x_2/(1-x_2))-\log_e(x_1/(1-x_1))]$ where x = disease proportion from 0 to 1 at time t. The apparent infection rates were calculated when disease severity was between 10~90%.

The apparent infection rates ranged from 0.1114 to 0.2197 and were not significantly different for lines within and over locations. Although the apparent infection rates were similar, the onset of disease varied because of different planting dates. All lines at Hualien DAIS had 10% rust severity approximately 25 days earlier than at other locations. Rust severity on susceptible lines at other locations, reached 10% at 67~73 days after planting while the moderately resistant line had 10 % severity at around 92 days after planting. Disease progress curves generated from data collected at AVRDC showed that rust development on line G8587 was delayed compared to the other lines. Because of the delay in rust development, line G8587 was initially considered moderately resistant, and it appeared that this line may have gene (s) for race specific resistance that delayed the onset of the epidemic. However, since the apparent infection rates were similar to susceptible lines, it was evident that the delay in the epidemic was related to its longer maturity over the susceptible lines. With this information studies were conducted to determine how host maturity influences the rate of rust development.

Effects of soybean growth on rust development. Field observations at AVRDC demonstrated that physiological development of the soybean plant plays an important role in soybean rust development (7). Lines that are very susceptible and mature later than other susceptible lines appear to have less rust at the same number of days after planting. Rust severity on late-maturing susceptible lines will be equal to the rust severity on earlier-maturing susceptible lines, but at unequal days after planting.

To determine the effect of maturity on soybean rust development, lines G38 and G8587 were grown in the field under an average day length of 11.2 hours (10.7~11.9 hours) and under a 14-hour day length by extending the natural day length with tungsten lamps (18). The days to full maturity (growth stage R8) were significantly affected by day length (Table 1). The different day lengths caused greater differences in the number of days in the reproductive growth stages than in the vegetative growth stages. Under longer day lengths, the growth stages from R1 to R8 developed later for both lines.

The rate of rust development was delayed under 14-hour day lengths on both lines (Fig. 2). This delay was related to the delay in the physiological development of the plant. There was a highly significant ($p < 0.01$) correlation between the rate of rust development to the number of days after planting and to the relative soybean life time (RLT) (Table 2). RLT was calculated by: (days after planting/day to full maturity)×100. When RLT was used as a regression variable, rates of rust development (regression coefficient) on the two soybean lines did not significantly differ regardless of day length whereas rates did significantly differ when days after planting was used as a regression variable.

Host maturity was delayed by using different day lengths which also delayed the onset of rust and reduced the rate of rust development. From field observations,

Table 1. Number of days to first flower (growth stage R1) and to full maturity (growth stage R8) of two soybean lines under two day lengths.

Line	Day length (hours)[1]	Days to R1	Days to R8
G 8587	11.2	28 d[2]	99 b
	14.0	34 a	120 a
G 38(Shih-shih)	11.2	29 c	83 d
	14.0	32 b	95 c

1. Day length in the field ranged from 10.7 to 11.9 hours (average 11.2) and was extended to 14 hours by tungsten lamps.
2. Values followed by different letters differ at the 5% level according to Duncan's multiple range test.

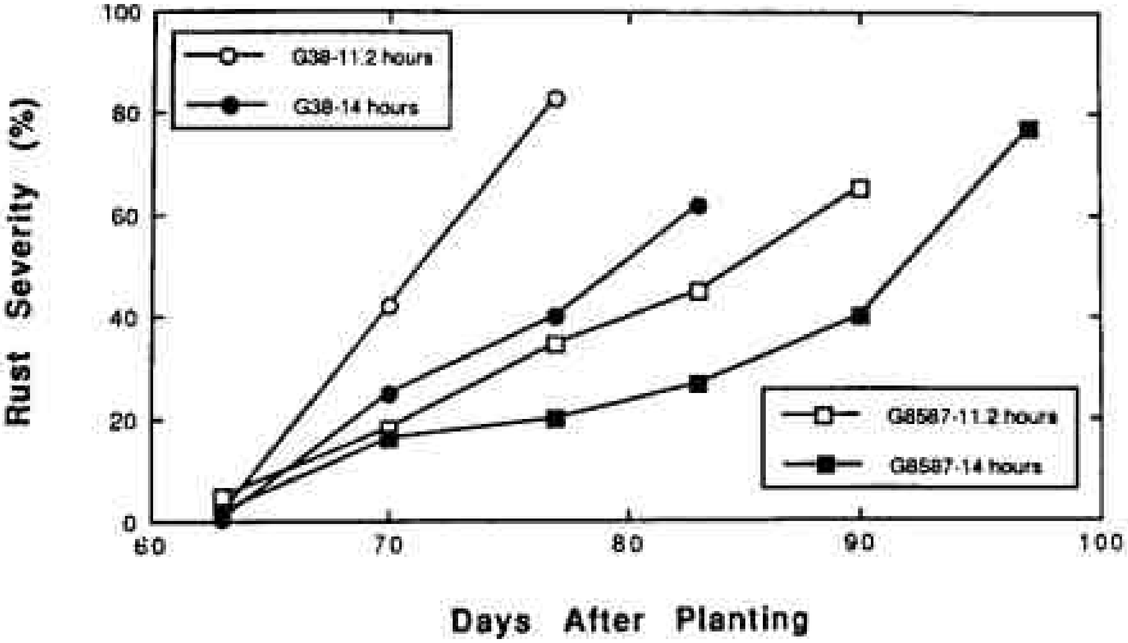

Fig. 2. Rust severity of two soybean lines grown under an average day length of 11.2 hours (natural photoperiod) and under 14 hours of day length.

Table 2. Regression and correlation coefficients of apparent infection rates on days after planting (DAP) and relative life time (RLT) for two soybean lines grown under two day lengths.

		Line			
		G 8587		G 38	
Regression variables[1]	Day length[2] (hours)	Apparent infection rates 3	Correlation coefficient	Apparent infection rates[4]	Correlation coefficient
DAP	11.2	0.3756 a	0.9800 **	0.2300 a	0.9203 **
	14	0.2815 b	0.9639 **	0.1803 b	0.9214 **
RLT	11.2	0.3116 b	0.9801 **	0.2379 a	0.9202 **
	14	0.2674 b	0.9638 **	0.2163 a	0.9289 **

1. Relative life time = (DAF/DFM) × 100; where DAP = Days after planting and DFM = Days to full Maturity.
2. Day length in the field ranged from 10.7 to 11.9 hours (average 11.2) and was extended to 14 hours by tungsten lamps.
3. Values followed by different letters differ at the 1% level according to Duncan's multiple range test.
4. Values followed by different letters differ at the 5% level according to Duncan's multiple range test.

we have noticed that early maturing lines have rust severity earlier with higher rates of increase than later maturing lines. This interaction between host development and rust increase occurs in all field experiments and confounds rust ratings between lines. To evaluate and compare rust development on soybeans without correcting for the differences in host maturity can led to erroneous conclusions about host resistance and environmental parameters related to disease development.

Serveral methods can be utilized to correct for differences in host maturity. By using maturation time of soybean or RLT as the time element, we have found that it helps to alleviate the problem associated with evaluating lines with different maturities. When rates of rust were com- pared using RLT, it partially or completely corrected the differences in rates of rust development in lines with different maturities(20).

Effects of temperature and dew period on rust development. Studies on how the environment influences rust development primarily have been based on greenhouse or growth chamber experiments. Uredospores of *P. pachyrhizi* were reported to germinate between 10 and 28.5 ℃ with an optimum range of 15～25℃(12). Minimum time of dew periods required for infection was 6 hours at 20～25℃ and became progressively longer at higher and lower temperatures. Twelve hours of dew was required at 10～14.4℃ and 26.6～27℃. Longer dew periods caused more disease.

To determine the relationship of rust development to environmental conditions in the field, lesion numbers were counted at various growth stages on a determinate cultivar, TK#5, and on an indeterminate line, PI 230971, from 4 December 1979 to 1 December 1980. Temperatures that were favorable for growth and development of soybean plants also in general favored an increase in rust. Greater leaf areas generally supported greater lesion numbers (Table 3). During the months with night temperatures above 27 ℃ lesion numbers tended to decrease.

The increase in lesion number was influenced by temperature; and was further complicated by precipitation, and by frequency and duration of dew. Mean temperatures during dew periods were recorded to determine the number of infection periods that occurred during each month from December 1979 through March 1981. Fewer infection periods and also decreased lesion numbers were associated with low temperatures at the beginning and the end of the year (Table 4). Low mean night temperatures of < 15℃ reduced lesion development and also adversely affected the growth and development of soybean plants.

It also has been shown that extended periods of leaf-surface wetness of approximately 10 hours per day and moderate temperatures (18～26℃) were necessary for severe epidemics(8). Temperatures of < 15℃ and/or > 30℃ along with dry conditions retarded the development of rust, and prolonged temperatures above 27 ℃ inhibited the fungus even when leaf surface wetness was adequate(8).

Effects of irrigation and precipitation on rust development. A multi-locational experiment was conducted to assess the effect of environment on soybean rust development in three seasons. Rust deve-

Table 3. Regression coefficients of leaf area (cm^2) and lesion number per plant over time for 13 planting dates of two soybean lines.[1]

Date planted	TK #5		G 8587	
	Leaf area (cm^2)	Lesion number per plant	Leaf area (cm^2)	Lesion number per plant
79-12-4	10.1	45.8	2.0	2.4
79-12-31	11.5	120.6	9.5	134.5
80-2-1	18.4	315.6	16.5	220.3
80-2-29	18.6	1514.5	21.1	602.7
80-4-9	26.8	928.0	17.9	302.9
80-5-1	25.5	268.6	29.8	380.2
80-5-30	36.2	1048.7	35.8	649.3
80-7-1	41.3	1758.3	49.3	984.7
80-8-1	28.4	704.5	39.2	844.3
80-9-2	30.0	851.5	32.6	641.7
80-10-1	30.1	878.4	24.9	603.6
80-10-30	13.3	626.5	15.2	262.8
80-12-1	16.0	58.5	10.8	-[2]

1. Numbers are averages of five plants per sample in four replications. Data were recorded from every planting date when each new growth stage developed.

2. No data recorded.

Table 4. Number of days with the minimun infection period for soybean rust development and average mean night temperature of 16 months.

Date (year-month)	Number of days with infection period[1]	Average mean night temperature (C)[2]
1979-12	14	14.8
1980-1	9	13.7
1980-2	2	14.5
1980-3	10	19.0
1980-4	15	20.7
1980-5	13	23.4
1980-6	5	24.6
1980-7	11	25.7
1980-8	20	25.1
1980-9	22	23.8
1980-10	28	22.0
1980-11	18	19.1
1980-12	8	13.9
1981-1	11	12.9
1981-2	12	15.5
1981-3	17	19.2

1. Based on mean night temperature and estimated minimum dew hour required for rust infection.

2. Average of bihourly data from 8:00 p.m. to 6:00 a.m.

lopment varied according to prevailing rainfall patterns. Rust developed most rapidly at sites where rainfall was more evenly distributed throughout the season and was slower in development at sites where rainfall occurred in uneven patterns[20]. In seasons with low rainfall, rust initiation was delayed, and the rate of development was reduced. Precipitation that occurred almost daily at one site promoted early and rapid rust development.

To determine the effect of precipitation and irrigation on rust development, field soybeans were either overhead or furrow irrigated, or were not irrigated in a two-season study. Rust severity increased more in fall 1980 than in fall 1979 (Fig. 3A). Within each year, plots that were overhead irrigated had more rust than plants in plots that were furrow or not irrigated. Precipitation was necessary for the early development of rust, even when soybeans were overhead irrigated (Fig. 3B). The type of irrigation used in 1979, a drier year compared to 1980, related to the onset of the epidemic and also to how rapidly it developed.

From this study, it was concluded

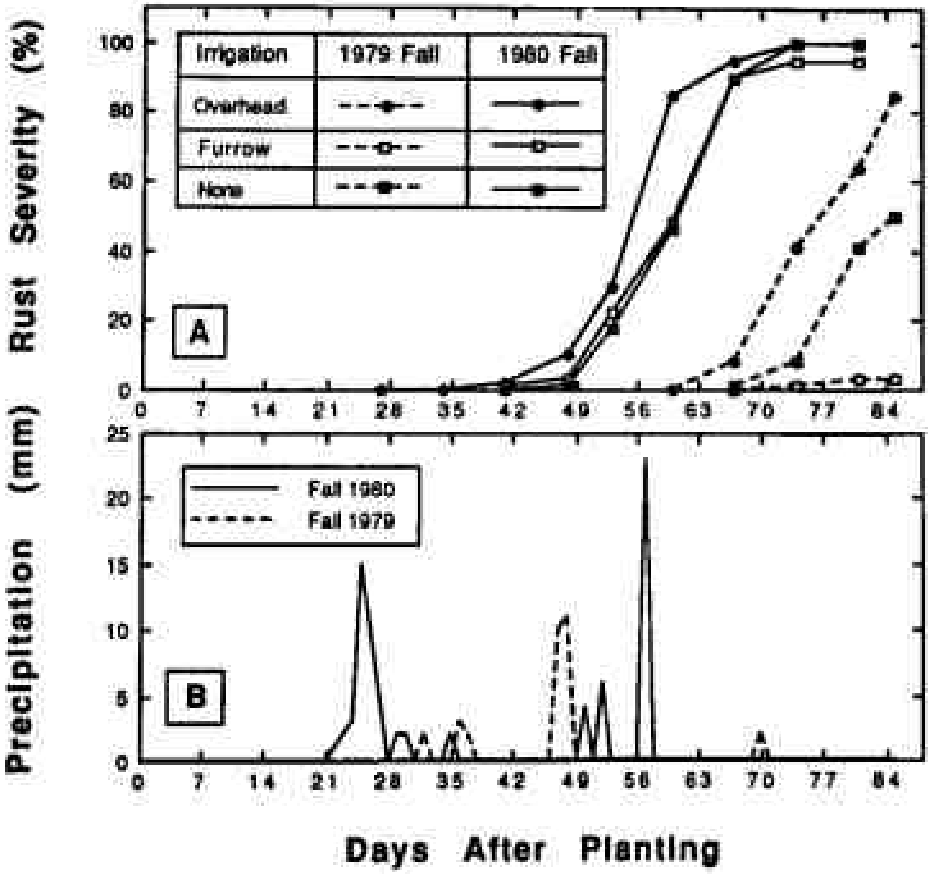

Fig. 3 Rust severity in plots that were either overhead or furrow irrigated or not irrigated (A) and corresponding precipitaion (B) in two seasons.

that precipitation was an important factor for the development of rust, and was more critical than the frequency and intensity of infection periods, which frequently occur during the normal soybean growing season although not always during the short period between rapid rust development and completion of the epidemic. Infection periods may be most important during the early stages of an epidemic or when precipitation is absent. The frequency and duration of infection periods probably intensifies the rust epidemic. Based on these results, a system for predicting rust epidemics and optimum scheduling for fungicide applications may be developed.

HOST RESISTANCE

Identifying resistance in soybeans to rust has been a major objective at AVRDC as over 9, 000 soybean accessions have been screened for resistance. Both rate-reducing and specific resistance occur in the soybean germplasm. In addition, nearly 300 accessions of wild perennial *Glycine* species have been evaluated for resistance(28).

Specific rust resistance. Three independent dominant genes for resistance have been identified in PI 200492, PI 230970, PI 462312 (Ankur)(2,11,13). The gene designation assigned to each of the specific resistant genes are: Rpp$_1$ (PI 200692), Rpp$_2$ (PI 230970), and Rpp$_3$ (PI 462312)(11,13). Three other soybean lines, Tainung#4, PI 459024 and PI 459025, and one *G. soja* line (PI 339871) are reported to have additional specific genes for resistance(4,15).

Our research indicates that PI 339871, PI 459024, PI 459025, and TK#5, Tainung #4 may also have single dominant genes for resistance(23). It was reported that in PI 339871 more than one gene may be present and may differ from that of PI 230970(23).

Physiological races of *Phakopsora pachyrhizi*. Three infection types have been described ; (1)TAN-tan lesions about 0.4 mm^2 with 2~4 uredia per lesion ; (2) RB - reddish brown lesions about 0.4 mm^2 with 0~2 uredia per lesion and; (3) 0 - absence of macroscopically visible signs or symptoms(9). Based on these infection types, races of the pathogen have been described(5,27).

Eleven lines, Ankur, PI 200492, PI 230971, PI 239871A, PI 239871B, PI 459024, PI 459025, TK#5, TN#4, and Wayne, that have been confirmed or suspected of having race-specific genes for rust resistance were used as a differential set to determine the physiological races of forty-two purified isolates. Based upon the infection type, these isolates were classified into nine races. Most isolates caused TAN-type lesions on at least seven of the lines. The data suggests that the predominant rust races were complex, and these races possess multiple virulence factors for compatibility on most of the lines.

At least one rust race from Taiwan has been reported to have three virulence genes(14). Other isolates from Taiwan were reported to cause rust with all known or suspected sources of specific resistance including PI 200492, PI 230970, PI 339871, PI 459025, and PI 462312(23). The majority of these rust isolates had multiple virulence genes.

The occurrence of multiple virulence genes in *P. pachyrhizi* is unusual because there are no soybean lines known to possess more than one specific resistance gene(23). The presence of multiple virulence genes in the pathogen population and the absence of multiple specific resistance genes in the host provides a competitive advantage when there is no selective advantage for excess virulence genes. Therefore, gene deployment techniques like gene rotation and pyramiding of specific resistance genes will most likely be ineffective in controlling soybean rust since the pathogen retains unnecessary virulence genes at a high frequency(23).

Rate-reducing resistance. Rate-reducing resistance has been effectively utilized to control some diseases including powdery mildew of wheat and late blight of potato. The advantage of rate-reducing resistance is that it is effective against all races of a pathogen. This is an important consideration with soybean rust since the rust pathogen apparently has already evolved into complex races with multiple virulence genes. The presence of rate-reducing resistance to rust in soybeans has been implicated(17) and verified(18), but has not been effectively utilized in soybean breeding programs.

A major impediment in developing lines with rate-reducing resistance has been the complications of evaluating lines from segregating populations or from accessions that have different maturities. Besides the physiological differences, there are differences in environmental conditions as plants mature under different time periods. An evaluation method which partially corrects for differences in host maturities uses the relative soybean life time (RLT) regressed on the logit transformation of rust severity as previously described. The rate of rust development is used to determine the level of rate-reducing resistance by comparing the slopes of the regression lines.

Seventeen advanced breeding lines which had previously been selected for being less susceptible to rust were tested for rate-reducing resistance(19). Apparent infection rates in one season had greater ranges between the lines (0.2075～0.3483) when the epidemic was delayed compared to the ranges in two other seasons when the epidemic was early (0.0998～0.1390 and 0.1080～0.1632). However, later developing epidemics may be more difficult to evaluate because of the reduced time frame of the epidemic. An interaction occurred between the rate of rust development and rust severity at RLT = 70%. This was primarily caused by lines TK#5, KS 535, G 5524 and SRE-Z-15C in the spring season; and AGS 62, 78-084 and SRE-Z-15C in the fall season which had differences in the rate of rust development to severity ratings compared to other lines (Table 5). Therefore, the rate of rust development alone does not always correlate well with the level of resistance. Lines SRE-Z-11A, SRE-Z-11B, and SRE-Z-15A had consistantly lower rates of rust development and lower severity ratings. These lines represent the best available levels of rate-reducing resistance and have been used as parents to improve resistance levels of soybeans.

Tolerance to soybean rust. The difficulties associated with identifying and quantifying rate-reducing resistance and the ineffectiveness of race-specific resistance necessitates evaluating soybeans for tolerance to rust. Tolerance can be defined as the relative yielding ability of soybeans under stress from rust.

Previous studies indicated that yields obtained when lines were grown under

Table 5. Apparent infection rates of rust and rust severity of 20 soybean lines in fall and spring, 1982, AVRDC.

Spring 1982			Fall 1982		
Line	Apparent infection rates[1]	Rust severity (%)[2]	Line	Apparent infection rates	Rust severity (%)
SRE-Z-15A	0.0998	22.8	SRE-Z-11B (AGS 182)	0.1080	42.2
SRE-Z-11B (AGS 182)	0.1014	18.6	78-084	0.1182	54.9
Shih-shih	0.1069	23.6	SRE-Z-11A (AGS 181)	0.1198	39.8
SRE-Z-11A (AGS 181)	0.1083	24.9	SRE-Z-15A	0.1233	43.8
TK #5	0.1113	40.3	SRE-Z-15C	0.1243	51.1
SRE-A-16	0.1126	20.4	TN #15	0.1261	45.5
KS-535	0.1154	32.5	SRE-A-16	0.1281	52.2
Hua-78-28	0.1163	28.3	Shih-shih	0.1317	50.3
AGS 62	0.1210	16.2	TK #5	0.1449	66.8
AGS 129	0.1214	26.9	KS-8	0.1449	71.0
TN #15	0.1245	26.7	AGS-66	0.1480	47.1
KS-741	0.1250	35.9	KS-535	0.1481	60.8
78-067	0.1252	32.1	Hua-78-28	0.1497	59.0
SRE-Z-15C	0.1267	18.5	KS-741	0.1504	56.2
KS-8	0.1276	30.6	TN #4	0.1509	56.7
G 5524	0.1293	16.6	AGS 62	0.1552	40.8
78-084	0.1299	26.4	78-067	0.1560	69.4
AGS-66	0.1311	21.7	SRE-Z-13 (AGS 183)	0.1586	64.9
TN #4	0.1358	30.6	G 5524	0.1629	49.3
SRE-Z-13 (AGS 183)	0.1390	27.2	AGS 129	0.1632	48.3
LSD (.05)[3]	0.0016			0.0013	

1. Based on relative soybean life time (RLT), calculated using van der Plank's formula(30).
2. Based on RLT = 70%.
3. Least significant difference.

fungicide-protection were not necessarily related to the yields obtained without fungicides. It was also observed that in the same environment, percentage yield loss based on yields from fungicide and non-fungicide-protected plots varied with lines and was not necessarily related to susceptibility (28). Tolerance to rust and significant variation in levels of tolerance within the soybean germplasm has been reported (20). Using some of the advanced lines, yields without fungicide protection ranged from 0.18 to 1.33 tons/ha with yield losses of 48 to 91% in spring season (Table 6). In the fall season, yields of the same lines without fungicide protection ranged from 0.22 to 0.93 tons/ha with yield losses of 58 to 90% (Table 6). The yield increase

Table 6. Yield of selected soybean lines in plots under fungicide and nonfungicide-protected conditions, and the associated yield loss in spring and fall of 1982 at AVRDC.

	Spring 1982			Fall 1982		
	Yield (t/ha)			Yield (t/ha)		
Line	Fungicide-protected	Nonfungicide-protected	Yield loss(%)	Fungicide-protected	Nonfungicide-protected	Yield loss(%)
SRE-Z-11A[1]	2.34	1.21	48.0	2.33	0.93	57.7
SRE-Z-11B[1]	2.58	1.33	48.1	2.11	0.84	60.2
78-067[2]	2.17	0.20	90.8	2.42	0.28	88.5
TK #5[2]	2.12	0.18	91.3	2.27	0.22	90.2
Shih-shih[3]	2.70	1.09	59.4	2.47	0.59	76.1
AGS 129[3]	3.29	0.72	78.2	3.74	0.55	79.6
Mean[4]	2.73	0.80	70.0	2.42	0.61	74.3

1. Lowest yield loss highest yield with rust.
2. Highest yield loss lowest yield with rust.
3. Check cultivars.
4. Total of 16 lines.

of the most tolerant line over the best check line was 59% in the fall season and 22% in the spring season. In this study, no correlation occurred in yields of plants in the fungicide-protected and yields of plants in the nonfungicide-protected plots. There was a significant interaction of yields between lines and fungicide treatment indicating that selection for high yields under fungicide-protection does not guarantee high yields under nonfungicide-protection. Selection for high yields under rust stress should not prevent later identification of high yielding lines under low levels of rust.

The variation in tolerance to rust can be exploited more readily than the variation that occurs in rate-reducing resistance. The measurement of yield may be more easily and acurrately assessed than disease severity in which assessments for rate-reducing resistance needs to be done weekly with pustule counts. A breeding scheme to incorporate tolerance to rust may involve early generation advance based on agronomic selections, mid-generation advance based on rust tolerance selection, and late generation advance based on rate-reducing resistance, tolerance, and agronomic selections (22). Early generation advance can be accomplished by either a bulk or single-seed descent method depending on the desired population size and diversity. Plants in the F4-F5 generations would be selected based on their level of tolerance and their seed bulked within families. Line selection would occur in the F6 generation and would be based on desired levels of tolerance and resistance. The F7 generation would be used to determine the homozygosity of the lines and their levels of rate-reducing resistance.

Based on this selection procedure,

fourteen SRE-lines (SRE=soybean rust epidemiology) have been selected and screened in the advanced rust tolerance trials. Some of these and other lines were recently evaluated in the field. The SRE-lines which were developed at AVRDC for their rust tolerance had the lowest levels of infected leaf area and were considered partially resistant based on pustule counts per leaf node (Table 7). Yield losses in these lines ranged from 29 to 85%. The SRE-lines also had less yield loss than the other high yielding lines (Table 8). Similar trends occurred in these lines for 100-seed weight losses. These SRE- lines represent the best available lines with tolerance to rust, and also are partially resistant based on pustule counts compared to susceptible checks.

CONCLUSION

The epidemiology of soybean rust has been a major focus of AVRDC research for nearly 20 years. There have been a number of important discoveries related to the interaction of soybeans, *P. pachyrhizi*, and the environment. However, there are still some questions to answer in regards to the origin of the initial inoculum that starts the epidemic; the races that predominate and their number of virulence factors; and the utilization of environmental parameters to forecast disease outbreaks.

Table 7. Leaf area infected, defoliation, and total number of pustules per plant, per leaf, and at node 7 on 12 soybean lines inoculated with *Phakopsora pachyrhizi*.[1]

Line	Leaf area infected (%)	Defoliation (%)	Total number of pustules	Pustule per leaf	Number of pustules at node 7
AGS 129	45	28	1776	41	104
AGS 181	76	41	3849	130	87
AGS 302	43	20	2209	66	53
GC 81118-8-4	54	29	2541	61	80
GC 82345-20-2	66	34	5934	168	176
GC 82349-6-1	53	41	2108	49	150
KS 8	64	36	2715	76	107
SRE B-15A	44	29	2272	70	83
SRE C-56A	25	24	803	23	25
SRE C-56E	31	24	709	19	29
SRE D-14C	35	16	2159	58	17
SRE D-14D	34	16	2100	54	51
Average	47	28	2431	68	78
LSD (P < 0.05)[2]	6	13	973	28	44

1. Based on growth stage R5-R6.

2. Least significant differences.

Table 8. Yield and 100 seed weight in fungicide-protected plots and in rust-inoculated plots, and their losses on 12 soybean lines inoculated with *Phakopsora pachyrhizi*.

Line	Yield (kg/ha)			100 seed weight (g)		
	Fungicide-protected	Rust-protected	Loss (%)	Fungicide-protected	Rust-protected	Loss (%)
AGS 129	2800	837	70	16.1	7.5	53
AGS 181	2279	766	66	17.1	10.0	42
AGS 302	2400	1050	57	21.2	12.5	41
GC 81118-8-4	2816	471	83	17.3	6.2	64
GC 82345-20-2	2864	726	75	19.5	7.8	59
GC 82349-6-1	3440	837	76	22.5	14.3	36
KS 8	3498	528	85	29.8	11.1	63
SRE B-15A	2386	1076	54	17.4	10.4	40
SRE C-56A	2567	1818	29	25.5	18.0	29
SRE C-56E	2625	1804	31	20.7	13.6	34
SRE D-14C	2804	1514	46	23.5	16.6	29
SRE D-14D	2605	1502	41	25.0	16.4	34
FLSD ($P \leq 0.05$)[1]			9			8
FLSD ($P \leq 0.05$)[2]	214			2.0		
FLSD ($P \leq 0.05$)[3]	263			2.3		

1. Differences between main plot means.
2. Differences between subplots within the same main plot.
3. Differences between subplots with different main plots.

To improve soybean lines that have resistance or tolerance to rust, techniques are needed to improve and allow for easy use of identifying and quantifying partial resistance. In addition, the utilization of sources of resistance from wild perennial *Glycine* species is no longer remote as recent advances in crossing *G. max* to other *Glycine* species will make the transfer of useful traits from the wild relatives to commercial soybeans more feasible [34]. In the future, we would expect this route to be most successful in utilizing host resistance to control soybean rust.

ACKNOWLEDGEMENTS

We would like to acknowledge Dr. A. T. Tschanz who has been instrumental in organizing many of the experiments on rust epidemiology.

LITERATURE CITED

1. Bromfield, K. R., and Yang, C. Y. 1976. Soybean rust: summary of available knowledge. p. 161-164. in: Expanding the Use of Soybeans, R. M. Goodman, ed., INTSOY Series No. 10, Univ. of Illinois, Urbana-Champaign, Illinois.

2. Bromfield, K. R., and Hartwig, E. E. 1980. Resistance to soybean rust and mode of inheritance. Crop Sci. 20:254-255.
3. Bromfield, K. R. 1981. Differential reaction of some soybean accessions to *Phakopsora pachyhizi*. Soybean Rust Newsl. 4:2.
4. Bromfield, K. R., and Melching, J. S. 1982. Sources of specific resistance to soybean rust. (Abstr.) Phytopathology 72:706.
5. Bromfield, K. R., 1984. Soybean Rust. Monograph No. 11, The American Phytopathological Society, St. Paul, Minnesota, U.S.A.
6. Casey, P. S. 1981. The epidemiology of soybean rust - *Phakopsora pachyrhizi* Syd. Soybean Rust Newsl. 4:2.
7. Fehr, W. R., and Caviness, C. E. 1977. Stages of soybean development. Special Report 80. Iowa State Univ., Ames, Iowa p.1-12.
8. Hartman, G. L., Wang, T. C., and Hymowitz, T. 1992. Sources of resistance to soybean rust in perennial *Glycine* species. Plant Dis. 76:396-399.
9. Hartman, G. L., Wang, T. C., and Tschanz, A. T. 1991. Soybean rust development and the quantitative relationship between rust severity and soybean yield. Plant Dis. 75:596-600.
10. Horsfall, J. G., and Barratt, R. W. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
11. Hartwig, E. E., and Bromfield, K. R. 1983. Relationships among three genes conferring specific resistance to rust in soybeans. Crop Sci. 23:237-239.
12. Marchetti, M. A., Melching, J. S., and Bromfield, K. R. 1976. The effects of temperature and dew period on germination and infection by uredospores of *Phakopsora pachyrhizi*. Phytopathology 66:461-463.
13. McLean, R. J., and Byth, D. E. 1980. Inheritance of resistance to rust (*Phakopsora pachyrhizi*) in soybeans. Aust. J. Agric. Res. 31:951-956.
14. Singh, R. J., Kollipara, K. P., and Hymowitz, T. 1990. Backcross-derived progeny from soybean and *Glycine tomentella* Hayata intersubgenetic hybrids. Crop Sci. 30:871-874.
15. Sudjadi, M. 1980. Host range study for soybean rust in Indonesia. Soybean Rust Newsl. 3:32-33.
16. Tschanz, A. T., and Wang, T. C. 1980. Soybean rust development and apparent infection rates at five locations in Taiwan. Prot. Ecol. 2:247-250.
17. Tschanz, A. T., Wang, T. C., and Hu, L. F. 1980. Epidemic development of soybean rust and a partial characterization of resistance to soybean rust. Soybean Rust Newsl. 3:35-41.
18. Tschanz, A. T., and Tsai, B. Y. 1982. Effect of maturity on soybean rust development. Soybean Rust Newsl. 5:38-41.
19. Tschanz, A. T., Sheng, W. S., and Tsai, B. Y. 1982. Development of soybean rust resistance in advanced breeding lines. Soybean Rust Newsl. 5:34-37.
20. Tschanz, A. T. 1982. Soybean rust epidemiology (final report). AVRDC, Shanhua, Tainan, Taiwan. 157pp.
21. Tschanz, A. T., and Tsai, M. C. 1983. Evidence of tolerance to soybean rust in soybeans. Soybean Rust Newsl. 6:28-31.
22. Tschanz, A. T., and Shanmugasundaram, S. 1984. Soybean rust. in: World soybean research conference III, ed. R. Shibles, Boulder, Co, Westview Press. p. 562-567.

23. Tschanz, A. T., Wang, T. C., and Tsai, B. Y. 1986. Recent advances in soybean rust research at AVRDC. in : Soybeans in tropical and subtropical cropping system. ed. S. Shanmugasundaram and E. W. Sulzberger, AVRDC, Shanhua, Tainan, Taiwan. p237-245.
24. van der Plank, J. E. 1963. Plant Diseases: Epidemics and their control. Academic Press, New York, 349 pp.
25. Yang, C. Y. 1977. Past and present studies of soybeans rust incited by *Phakopsora pachyrhizi Syd. Bull. Inst. Trop. Agric. Kyushu Univ.* 2: 78-94.
26. Yang, X. B., Tschanz, A. T., Dowler, W. M., and Wang, T. C. 1991. Development of yield loss models in relationships of components of soybean infected with *Phakopsora pachyrhizi.* Phytopathology 81: 1420-1426.
27. Yeh, C. C. 1983. Physiological races of *Phakopsora pachyrhizi* in Taiwan. Jour. Agric. Res. China 32: 69-74.

摘　　要

王添成、Hartman, G. L. 1992. **大豆銹病流行病學及抗病育種**　植保會刊 34:109-124

由 *Phakopsora pachyrhizi* 所引起的大豆銹病是限制亞洲熱帶及亞熱帶地區大豆生產之主要病害。亞洲中心對大豆銹病之研究是集中於偵測病害之進展；評估產量損失；獲取病原菌之基本知識，找尋抗病材料進而將這些材料導入雜交育種之品系中。以一個中抗及三個感病品系，在臺灣五個地區之三個不同季節裡偵測銹病進展之結果，顯示這些品系之感染速率在不同季節及不同地區均相類似。幾個試驗顯示大豆的成熟度與銹病進展之速率顯著的成正相關，而環境及寄主基因型之影響則非呈高度相關。因此評估大豆品系之抗病性，亞蔬中心已研究出補救寄主成熟度差異之方法。最佳之方法就是採取大豆相對的生育期為一個時間要素，分為0～100。把播種到完全成熟之間的任何時間轉變成大豆生活史之百分比。關於病原菌病原性之差異，以及環境因子影響病害之進展亦曾加以研究。從所採集到42個菌株在11個鑑別品系上鑑定出9個生理小種。優勢的生理小種則呈多數毒性基因型之複合體。環境因子方面，最適合本病原菌夏孢子發芽之溫度為15～25℃。當溫度在20～25℃時，病原菌侵入感染所需之最短露期為6小時，但當溫度降為15～17.5℃時，則露期增長為 8～10小時。當夜間平均溫度低於15℃時，則大大地降低病斑數或完全阻止病斑之進展。雨量是大豆銹病流行進展之重要因子，因此，被應用於預測銹病之嚴重度，而且比葉面濕度及溫度或二者交互作用更為重要。由於發病速率減低之抗病性在鑑定與量度上之困難，以及生理小種專一性之抗病性的無效，導致發展對銹病具有耐性而且較高大豆產量之技術，並且也發展出更佳之量取及瞭解部份抗病成份之技術。另外，一些新的抗病來源已從多年生之野生大豆品種中鑑定出來，將作為抗病育種之材料。

（**關鍵字**：Phakopsora pachyrhizi，感染速率，多年生野生大豆，產量損失，生理小種）