Analysis and Quantification of Soybean Rust Epidemics from Seventy-Three Sequential Planting Experiments

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ABSTRACT

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Epidemics of soybean rust (caused by *Phakopsora pachyrhizi*) were studied in Taiwan by sequentially planting soybean cultivars G 8587 and TK 5 at weekly intervals in 1980 and 1981. For most planting dates, lesions were first observed at soybean growth stage V2 or V3 (unrolled leaf occurred at second or third nodes on main stem), indicating the presence of inoculum near the time of planting. The development of disease was affected by the growth of soybean, of which the growth periods varied from 83 days to 207 days for different planting dates. On the autumn-seeded crop, disease reached the maximum level 40–60 days after planting. On the summer-seeded crop, disease onset was 20–40 days later than that of spring- or autumn-seeded crop, and disease reached the maximum level 80–100 days after planting. Physiological days were used as a measure of biological time for both the soybean plant and the rust pathogen. Total leaf area per plant was closely described using plant physiological day as an independent variable with R^2 greater than 0.88 (n = 215–235). A disease model using physiological day of plant and physiological day of pathogen as independent variables was used to calculate the disease development with data pooled from all the plantings, and R^2 = 0.69 (n = 696) for G 8587 and 0.75 (n = 589) for TK 5. Defoliation was significantly explained with regression on disease severity (R^2 = 0.625–0.809, $P < 0.0001$); however, error increased when disease severity was low. Estimation of yield loss using area under disease progress curve (AUDPC) as an independent variable resulted in a greater R^2-value than using area under defoliation curve for each cultivar. The results were significantly improved when AUDPC adjusted for planting date effect was used as an independent variable for the regression.

Soybean rust (caused by *Phakopsora pachyrhizi* Sydow) results in significant yield losses in eastern hemisphere countries (4,22,26). The importance of the disease has increased due to the increased soybean production and expansion of the crop to new regions (4,26).

Soybean rust is one of the major diseases of soybean in Taiwan, where soybean is grown throughout the year and rust occurs in all seasons (4). Of the three recognized cropping seasons (spring-, summer-, and autumn-seeded) the disease causes the most damage in spring- and autumn-seeded crops (4). Commonly, the disease causes severe premature defoliation which decreases yield (4). Casey (6) concluded from a 2-yr field study that extended free moisture and moderate temperature (18–26 C) are necessary for the disease development, whereas extreme temperatures (greater than 30 C) retard the development of the disease. Effect of plant growth on soybean rust development has been reported by several researchers (7,23,29). Tschanz (29) suggested that the physiological age of the soybean plant may play a significant role in affecting soybean rust development. This has been a difficult phenomenon to quantify, and the cause-effect relationship is not clear.

The effect of plant growth on disease development has been considered a challenging problem in plant disease epidemiology (24). Different approaches to demonstrate the interaction of pathogen and plant growth have been tried (12,17,18,24,28). Since plant parasites are inherently dependent on their host for existence, Lalancette and Hickey (18) suggested that plant growth itself can be used as a measure of biological time for modeling disease development. A relative biological lifetime of soybean was defined by Tschanz et al (30) to aid in examining the effect of plant growth stage on soybean rust development.

Soybean rust has been considered a potential exotic threat to soybean production of the United States (4), and several studies have been conducted to assess the potential impact of the disease on the soybean production of North America (3,16). However, these assessments were basically qualitative because quantitative information of disease epidemics was not available. Studies on population dynamics of soybean rust should be continued to provide quantitative information between disease occurrence and environmental factors. Such information will help us assess the potential epidemics of the disease in the United States (26). The objective of this study was to analyze and quantify the soybean rust in relation to the development of soybean plant and weather variables as part of a project to assess the potential impact of soybean rust to soybean production in the United States.

MATERIALS AND METHODS

Planting, inoculation, and weather record. Experiments were conducted in 1980 and 1981 at the Asian Vegetable Research and Development Center (AVRDC), Taiwan. Soybean cultivars TK 5 and G 8587, which have determinate and indeterminate growth type, respectively, were planted. G 8587 is more photoperiod sensitive and matures later (length depends on the planting time) than TK 5. In order to create different environmental and plant physiological windows, a sequential planting experiment was done. Each planting time was considered as a treatment. In 1980, planting began on 7 January (day 7) and continued through 9 December (day 343) at weekly intervals with a total of 46 planting times for each cultivar. In the 1981 experiment, planting started on 21 December 1980 (indicated as day -10) and continued through 21 September 1981 (day 264), for a total of 27 planting dates for each cultivar.

At the research farm of AVRDC, a field was divided into plots of 3 × 5 m with 2-m corn strips planted between plots. Treatments were randomly assigned to the plots. Five raised beds of 1 × 3 m were made in each plot. Soybean was then planted in two rows per bed at a density of 400,000 plants/ha. Overhead irrigation was applied weekly. Soybean rust was allowed to develop naturally since the urediniospores are present in southern Taiwan all year (29).

A weather station was established in the field for recording daily maximum and minimum temperature and average temperature at 2-hr intervals using a thermometer.

Seasonal development. For each planting date, disease assessment began as soon as rust was first observed. Each plot was divided into four equal sections. Disease severity, which was defined as percentage of diseased leaf area, and percentage of defoliation were rated for each section. The plot mean was calculated from the data of four sections. Rust was assessed at weekly intervals for at least 8 wk until there was no further disease increase. Times of assessment varied with planting date.

To examine disease progress in the spring, summer, and fall cropping seasons, disease severities from plants seeded at day 40, 161, and 252 were plotted against days after planting. To examine the seasonal variation of disease development, the apparent infection rate (r) (32) was calculated for each planting date. The r-values were then plotted against the date of planting. The relationship between disease severity and defoliation was determined by regression using disease severity as an independent variable and defoliation as a dependent variable for each cultivar × year combination.

Pustule development. From December 1979 to March 1981, the effect of plant growth on pustules per plant was studied at AVRDC by sequentially planting G 8587 and TK 5 a total of 13 times at approximately 30-day intervals. At each planting, soybean was sown in plots of 6 × 7 m with a density of 400,000 plants/ha. Plots were separated by 2-m corn strips. When 50% of the plants reached every soybean growth stage from V1 to R8 (8), five plants per plot were randomly selected. The number of pustules per plant was counted, and total leaf area of each plant was measured with a Li-Cor area meter (Model LI-6000, Li-Cor Inc, Lincoln, NE). The relationship between leaf area per plant and accumulated physiological days (see below) was analyzed with regression analysis.

Quantification of disease development. Disease development was related to physiological days of pathogen and crop. The physiological day (D_i) can be linearly related to temperature as (10,35):

$$D_i = \begin{cases} 0, & \text{if } T_i < T_{min} \\ (T_i - T_{min})/(T_{opt} - T_{min}), & \text{if } T_{min} \le T_i < T_{opt} \\ (T_{max} - T_i)/(T_{max} - T_{opt}), & \text{if } T_{max} \ge T_i > T_{opt} \\ 0, & \text{if } T_i > T_{max} \end{cases} \quad (1)$$

in which the T_i is average daily temperature; T_{max}, T_{opt}, and T_{min} are maximum, optimum, and minimum temperature for the growth of an organism (pathogen or host), respectively; $D_i = 1$ if $T_i = T_{opt}$. In our study, the three values were obtained from literatures as $T_{max} = 45$, $T_{opt} = 30$, and $T_{min} = 7$ for soybean (35) and $T_{opt} = 22$, $T_{max} = 30$, and $T_{min} = 4$ for *P. pachyrhizi* (6,19,20). The cumulative physiological day (X) from initial day (t_0) to tth day after planting is written as:

$$X = \sum_{i=t_0}^{t} D_i \quad (2)$$

For the rust pathogen, 17 days after planting was arbitrarily taken

TABLE 1. Duration of crop growth, maximum leaf area per plant, maximum rust pustules per plant, and growth stage (GS) at which disease was first detected on soybean planted from 4 December 1979 to 12 March 1981 in Taiwan

Cultivar	Planting date[a]	Days of growth	Maximum leaf area/plant (cm^2)	First growth stage with lesion	Maximum lesions/plant
G 8587	−28	121	285	V[b]3	571
	−1	121	800	V2	13,835
	32	110	1,230	V3	20,595
	60	95	1,175	V3	37,382
	100	207	2,540	V2	44,789
	122	174	3,790	V2	51,047
	151	147	3,385	V3	58,401
	183	115	3,445	V3	66,207
	214	96	1,950	V4	40,309
	246	83	1,345	V3	24,822
	275	88	1,010	V3	27,992
	304	95	815	V2	12,856
	336	124	985	R5[c]	597
TK 5	−28	116	755	V2	4,778
	−1	107	700	V2	9,673
	32	95	1,080	V2	20,342
	60	84	1,075	V3	69,291
	100	93	1,315	V2	47,695
	122	104	1,510	V2	20,587
	151	101	1,930	V4	68,182
	183	83	2,340	V4	74,663
	214	76	1,125	V3	37,468
	246	76	1,310	V3	34,473
	275	77	1,365	V3	41,469
	304	88	725	V2	27,218
	336	122	1,215	R3[d]	1,548

[a] 1 January = 1.
[b] V = Node on the main stem at which an unrolled leaf occurred.
[c] R5 = Plants began to seed.
[d] R3 = Plants began to pod.

as the initial day of infection because natural inoculum caused infection as soon as the first leaf appeared.

Disease severity at time t (Y_t) can be defined as:

$$Y_t = f(X_1, X_2) \quad (3)$$

in which X_1 and X_2 are the cumulative physiological days for soybean plant and the rust pathogen, respectively; $f(X_1,X_2)$ consisted of a linear combination of X_1, X_1^2, X_2, X_2^2, X_2^3, and X_1X_2. STEPWISE procedure with STEPWISE option of SAS statistical package (24) was employed to select the best linear combination of the equations based on significance of estimated parameters. Regression results were examined using residual plots, coefficients of determination (R^2), and F-values.

Determination of yield loss. A check plot was established for each planting date to estimate the effect of disease on soybean yield. The plots were sprayed with mancozeb 3 (Dithane M-45, Rohm and Haas Company, Philadelphia, PA) at weekly intervals to maturity. At maturity, soybeans were harvested from sprayed and unsprayed plots and threshed with a thresher. Seeds were air-dried and then weighed. Moisture was measured and plot yield was adjusted to 13% moisture. The plants of G 8587 from the first 30 planting dates were not harvested in 1980.

Regression analysis was used to examine the effect of disease on yield. Because planting date may affect the length of the plant growth period, the attainable yield, and the length of the epidemic period, planting date was adjusted so that the disease effect on soybean yield from different planting times could be compared. Relative yield loss and relative area under the disease progress curve (RAUDPC) (9,36) were used to make this adjustment to develop yield loss equations. The relative yield loss (L) was calculated as (healthy yield–diseased yield)/healthy yield. Healthy yield corresponded to the sprayed plot yield. The RAUDPC was calculated as:

$$\text{RAUDPC} = [\sum_{i=1}^{n} (Y_i + Y_{i-1})(t_i - t_{i-1})/2]/[(t_n - t_0)100] \quad (4)$$

in which n is times of disease assessment, t_i is days after planting for ith disease assessment, and Y_i is disease severity. We assume that $Y_0 = 0$ and $t_0 = 17$. For defoliation, area under the defoliation curve (AUDC) and relative AUDC (RAUDC) were calculated using defoliation ratings as the variables (Y_i) in equation 4. The regression model for yield loss was:

$$L = B_0 + B_1 Z \quad (5)$$

in which Z represents area under the disease progress curve (AUDPC), RAUDPC, AUDC, or RAUDC, and B_0 and B_1 are regression coefficients. All computations were done with SAS statistical package (25).

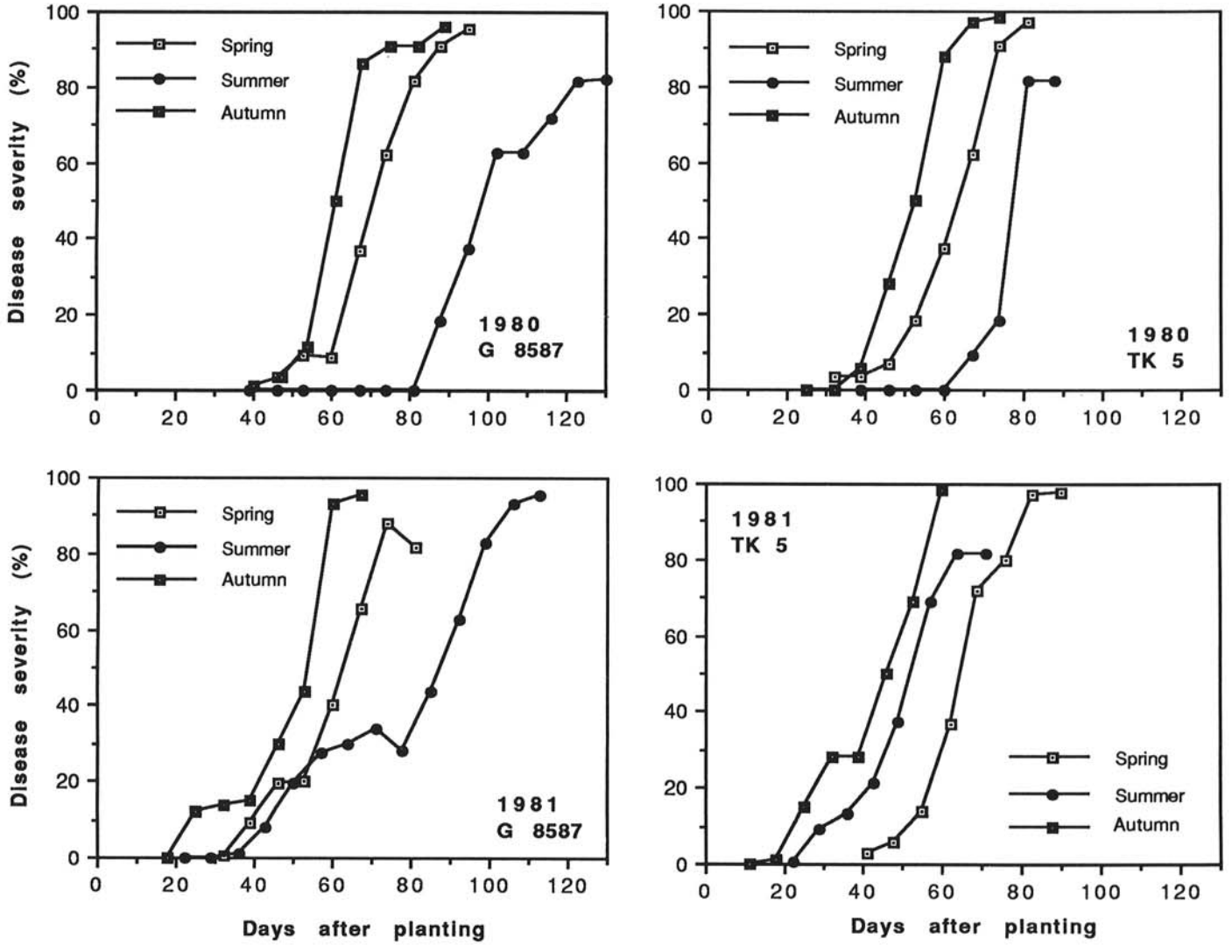

Fig. 1. Progress of soybean rust (caused by *Phakopsora pachyrhizi*) on two cultivars of soybean seeded at three cropping seasons: spring, summer, and autumn, during 1980 and 1981 in Taiwan.

RESULTS

Seasonal progress. The soybean growth period varied from 95 to 207 days for G 8587 and from 76 to 122 days for TK 5 (Table 1). The maximum leaf area per plant for different planting dates varied from 285 to 3790 cm^2 for G 8587 and from 700 to 2340 cm^2 for TK 5 (Table 1).

Rust pustules were first observed for most planting dates when an unrolled leaf occurred at the second or third nodes on the main stem (V2–V3) (Table 1). For the plantings during winter (day 336), disease was first observed on G 8587 when plants began seed (R5) and on TK 5 when plants began pod (R3). The maximum numbers of pustules per plant among different planting times varied from 571 to 66207 on G 8587 and 1548 to 74663 on TK 5 (Table 1).

Epidemics during the spring, summer, and autumn cropping seasons were different (Fig. 1). Disease onset was earliest on the autumn-seeded crop, and latest on the summer-seeded (Fig. 1). Disease in the autumn-seeded crop reached the greatest disease level within the shortest time. These results were similar for the 2 yr, except that 1981 spring-seeded TK 5 plants had a later disease onset time than summer-seeded plants.

The apparent infection rate fluctuated between values of 0.01–0.25/day for different planting times (Fig. 2). In 1980, high r values occurred on spring- and autumn-planted crops. On TK 5, r values were usually higher than those of G 8587 for the summer planting. In 1981, fluctuation patterns of the r values were similar between the two cultivars.

Quantifying rust epidemics with physiological days. Leaf area per plant was significantly correlated with physiological days of soybean (Fig. 3). In G 8587, the relationship was described with a third order polynomial equation with $R^2 = 0.922$ ($n = 215$). The maximum physiological day was 137, and leaf area started to decline at about 100 physiological days. TK 5 had a maximum physiological day equal to 71, and its leaf area growth was described with a simple linear regression with $r^2 = 0.885$ ($n = 235$) (Fig. 3).

Disease severity was well predicted using physiological days of the plant and pathogen as independent variables (Table 2). Coefficients of determination (R^2) were 0.69 ($n = 696$) for G 8587 and 0.75 ($n = 589$) for TK 5. Terms in the regression equations were different between two cultivars. However, all models included the interaction between pathogen and plant physiological days (X_1X_2). Predicted values plotted against observed values showed that a great amount of unexplained variation was in the region of low or high disease severity.

Relationship between disease severity and defoliation. The results from both years showed a significant correlation ($R^2 = 0.625$–0.809) between disease severity and defoliation of soybean plants (Fig. 4). The regression coefficients (B_1) varied from 0.58 to 0.76, indicating that 100% severity caused less than 100% defoliation on average. A great amount of variation was found in the range of lower disease severity. As shown in Fig. 4, many plants with severity less than 25% had no defoliation, indicated by the data points at $Y = 0$. However, at severities less than 25%, defoliation was sometimes greater than 25%, particularly in 1980.

Yield loss. Defoliation and disease severity were significantly correlated with yield loss (Table 3). The regression using RAUDC as predictor provided a better fit than that using AUDC as predictor for both cultivars (Table 3). Values of R^2 obtained using RAUDPC as an independent variable were greater than those obtained using AUDC, RAUDC, and AUDPC as an independent variable (Table 3). Values of intercepts (B_0) for RAUDPC were

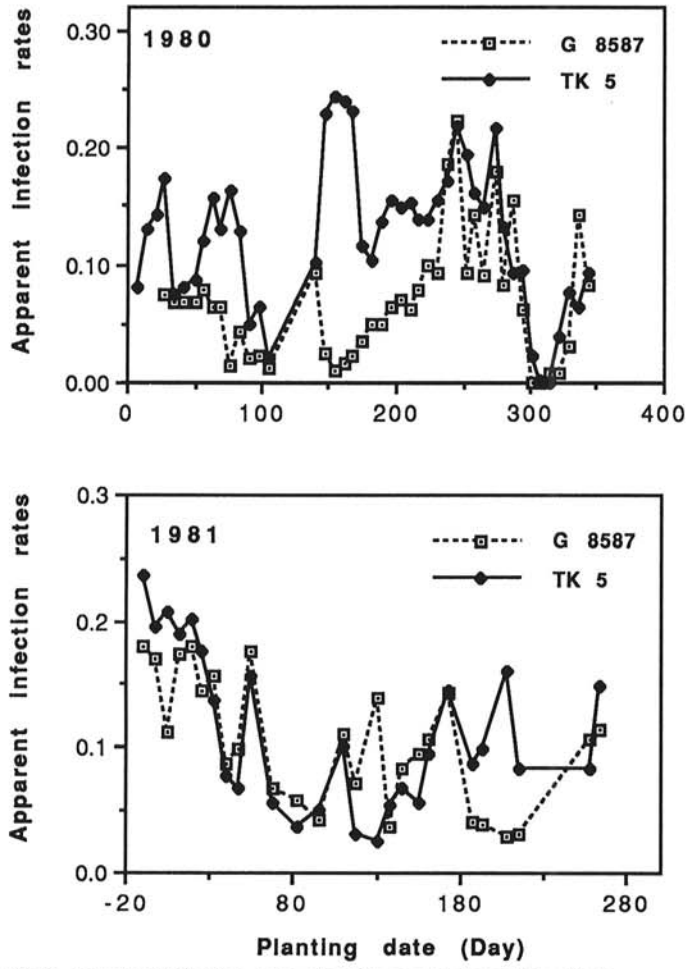

Fig. 2. Apparent infection rates of soybean rust (caused by *Phakopsora pachyrhizi*) on two soybean cultivars seeded at different times during 1980 and 1981 in Taiwan.

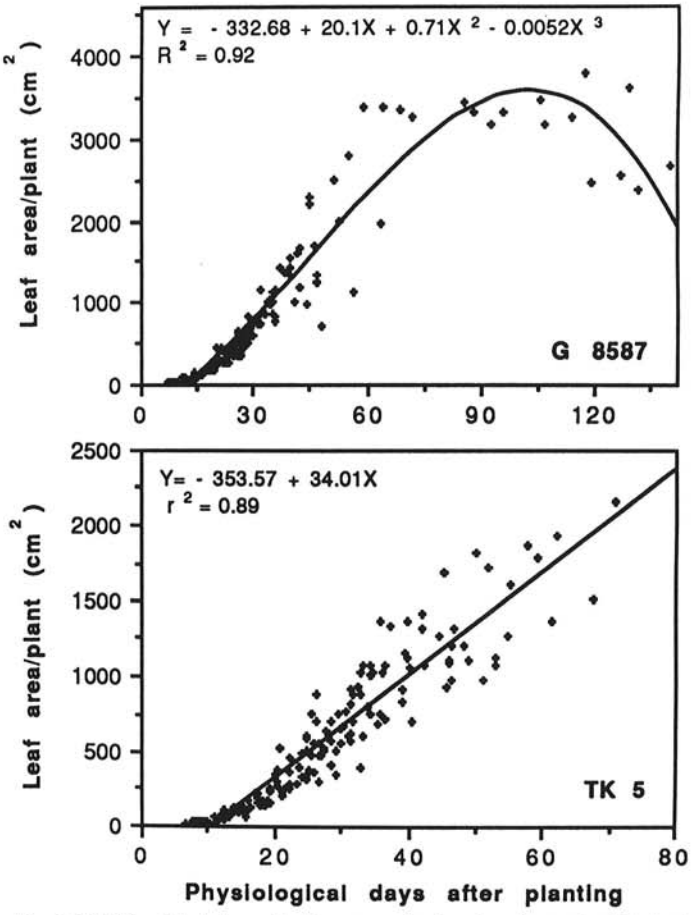

Fig. 3. Relationship between leaf area per plant and soybean physiological day for the data collected from 13 planting times from December 1979 to March 1981 in cultivars G 8587 and TK 5 in Taiwan.

smaller than those obtained by using the other three variables. In general, variables based on disease severity had greater coefficients of determination with yield loss than variables based on defoliation. The slopes and the intercepts for the model of RAUDPC were not significantly different between the two cultivars.

DISCUSSION

By sequentially planting two soybean cultivars different in growth throughout two years, we obtained a series of seasonal windows which were different from each other in weather and plant conditions. Based on the data, dynamic development of soybean rust in relation to plant growth and environment effects was outlined and quantified. The difference among these seasonal windows in disease development may be similar to the difference among different geographic regions. Application of disease model developed from these windows may enable us to estimate the epidemic potential of the pathogen at given regions of the United States using plant and pathogen physiological days as input variables.

The planting time experiment in the present study showed that the maximum disease level was attained closer to the time of planting in the spring and autumn crop than in the summer-seeded crop (Fig. 1). This difference may result from responses of the plant and pathogen to the summer environment. It has been reported (29,30) that in the summer the photoperiod sensitivity of soybean resulted in a longer growth period, which may have delayed disease development. The rust pathogen has also been reported to be sensitive to the change of photoperiod (14).

TABLE 2. Results of stepwise regression between soybean rust severity and the linear combination of physiological days of soybean plants (X_1) and pathogen (X_2) for data collected from 73 sequential planting experiments during 1980 and 1981 in Taiwan

		Partial regression coefficients							
Cultivar	df[a]	B_0	X_1	X_1^2	X_2	X_2^2	X_2^3	X_1X_2	R^{2b}
G 8587	690	−21.88	1.866	−0.034	−1.830	...[d]	−0.00050	0.077	0.69
		(2.97)[c]	(0.120)	(0.004)	(0.320)	...	(0.00008)	(0.004)	
TK 5	583	−16.47	1.272	−0.047	...	−0.093	0.00014	0.166	0.75
		(5.43)	(0.275)	(0.003)	...	(0.012)	(0.00005)	(0.007)	

[a] df is degrees of freedom for error.
[b] R^2 are coefficients of determination.
[c] Standard error of estimated parameter.
[d] Variable was not selected.

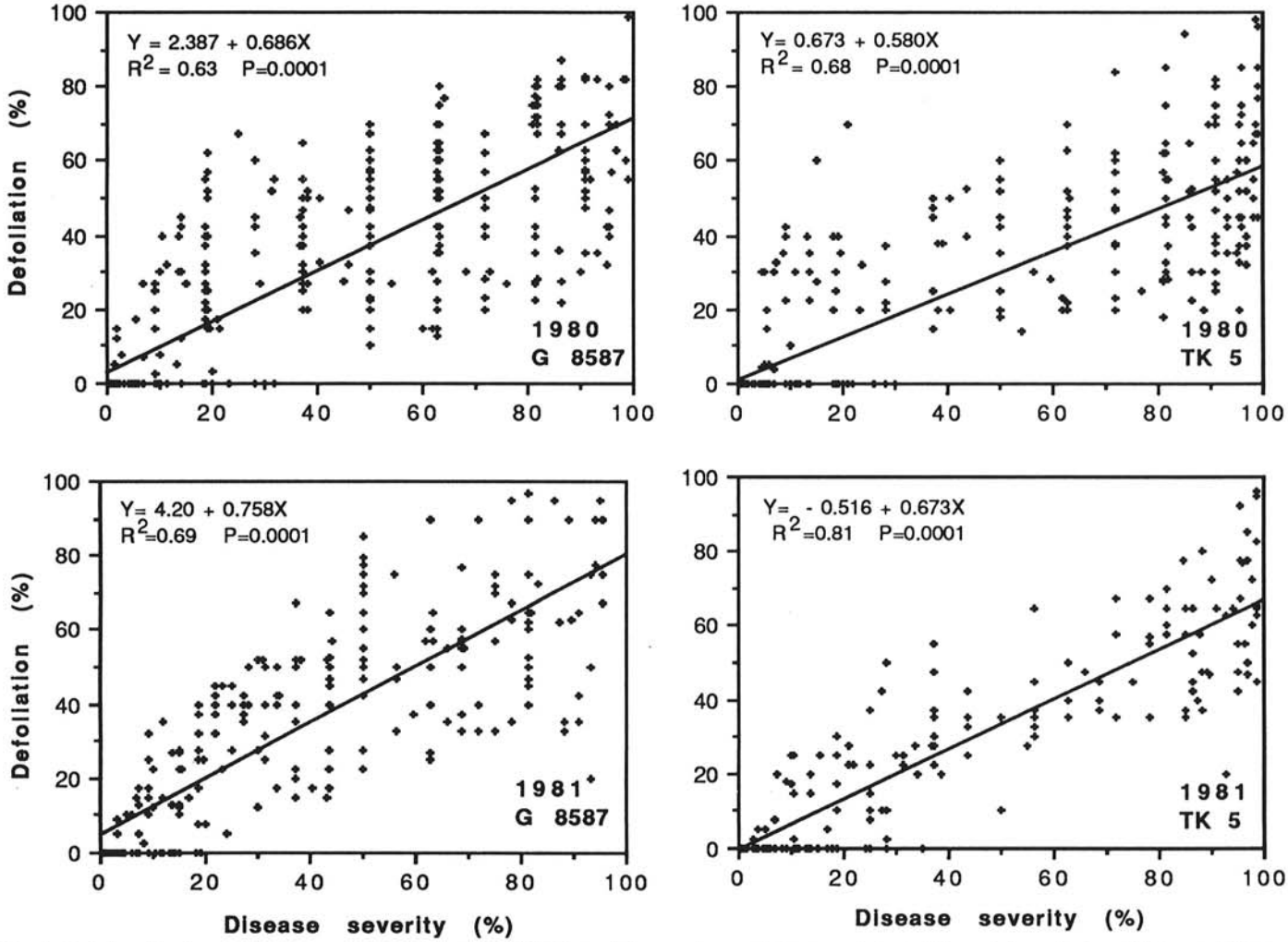

Fig. 4. Relationship between soybean rust severity and defoliation of two soybean cultivars seeded at different times during 1980 and 1981 in Taiwan.

High temperatures in the summer may have retarded disease development (6,15). Marchetti et al (19) reported that no infection occurred under greenhouse conditions when temperatures were over 27.5 C. Casey reported from field data in Australia that temperatures greater than 27 C inhibited the soybean rust fungus and that temperatures greater than 30 C retarded disease development (6). In southern Taiwan, average temperatures during summer are often above 28 C.

It is generally considered that leaf abscission is correlated with the number of pustules per leaflets (4). Results of our study (Fig. 4) showed a significant regression between disease severity and defoliation. However, despite the correlation between defoliation and disease severity, defoliation may not be as useful as disease severity to predict yield loss caused by soybean rust (Table 3). Defoliation may be greatly influenced by environment, even with constant disease severity. Vakili (31) observed that in the warm and rainy season in Puerto Rico, leaves with 5–6 pustules per leaflet were readily defoliated after slightly shaking the plant. However, the leaves were not easily dislodged in the cool and dry seasons, although there were up to 15 pustules per leaflet.

Modeling yield loss of soybean appears to be more difficult than for certain other crops because of the ability of soybean to regenerate tissue after being defoliated. The critical point model, using rust severity at plant flowering as a predictor, was reported to fit the yield loss data well (33). Because data in this experiment were collected from sequential planting times that did not have a uniform environmental background as in most yield loss experiments, critical point and multiple point models may not be adequate in our case. The AUDPC did not fit the data well (Table 3) because value of AUDPC varied with the period of plant growth or planting time. Therefore, the use of RAUDPC as a disease variable to adjust the planting time effect may be reasonable. Yang and Zeng (36) reported that in stripe rust of wheat, RAUDPC produced a very good fit to the yield loss data from different years and locations. Subba Rao et al (27) developed a leaf rust yield loss model on wheat using a modified RAUDPC as a disease variable. They suggested that this method gave a better fit and biologically meaningful parameters. However, the RAUDPC only adjusts the growth period effect. Like AUDPC, it cannot distinguish disease severity early and late in the season (5). Application of Hills et al's weighted AUDPC (11) or RAUDPC calculated from the key growth stages may provide better estimation if data of plant growth stage are available.

The physiological day or "heat unit approach" has been in use for over two centuries and is widely adopted in biology because of its value in satisfying practical needs, rather than for its accuracy (34). Numerous experiments both in agronomy (1,2,34,35) and plant pathology (10) have successfully used physiological age or degree days as the simplest time scale to measure development of crops and pathogens. In our study, the physiological days of the plant and the pathogen provide the inherently integrated information on the crop, pathogen, environment, and time. The 73 sequential plantings provided a broad diversity of environmental windows. With such variation, our model still explained as much as 75% variation in disease development. However, like other works which study population dynamics by collecting data on a population over time, our analysis also faces a dilemma in that the disease ratings as well as the leaf areas collected within one planting date were not independent from each other. Such dependence does not allow us to test our prediction model with current statistical techniques.

Variation that the model (equation 3) failed to explain may be from the random error as well as from effects that were not accounted for. Our model did not consider variation in plant resistance during the growing season. It has been reported that plant and leaf age affects susceptibility of plants to *P. pachyrhizi* (13,21). The simplicity of our model also does not include photoperiod sensitivity and moisture. Improvements are expected from the inclusion of these variables. It is possible that the model can also be improved by calculating physiological day with temperature at hourly intervals rather than daily intervals. Different minimum, maximum, and optimum temperatures could be used to calculate the soybean physiological day for different cultivars because each cultivar may react to the environment differently. Finally, the relationship between plant physiological day and pathogen physiological day in equation 3 was obtained empirically. Improvements may be possible if the above effects are accounted for in an analytical rather than an empirical method.

TABLE 3. Regression of relative soybean yield loss caused by *Phakopsora pachyrhizi* to area under defoliation curve (AUDC), area under disease progress curve (AUDPC), relative AUDC (RAUDC), and relative AUDPC (RAUDPC) for data collected from sequential planting experiments during 1980 and 1981 in Taiwan

Cultivar	Disease variable	df[a]	Regression coefficients[b] $B_0 + S(B_0)$	$B_1 + S(B_1)$	R^2[c]	$P > F$[d]
G 8587	AUDC	41	53.94 ± 6.13	0.0072 ± 0.003	0.11	0.0300
	RAUDC	41	43.34 ± 6.34	85.44 ± 21.54	0.28	0.0005
	AUDPC	41	34.00 ± 7.99	0.0154 ± 0.004	0.31	0.0001
	RAUDPC	41	22.29 ± 6.20	131.29 ± 17.51	0.58	0.0001
TK 5	AUDC	71	71.24 ± 7.23	−0.0037 ± 0.006	0.01	0.5546
	RAUDC	71	49.98 ± 6.38	73.31 ± 24.76	0.12	0.0040
	AUDPC	71	41.16 ± 7.05	0.0142 ± 0.004	0.19	0.0002
	RAUDPC	71	25.74 ± 5.84	110.89 ± 14.65	0.46	0.0001

[a] df is degree of freedom for error.
[b] B_0 and B_1 are the intercept and slope, respectively. $S(B_0)$ and $S(B_1)$ are standard errors for B_0 and B_1.
[c] R^2 are coefficients of determination of the regression.
[d] $P > F$ is probability of F-value.

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