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Disease Dispersal Gradients of Rice Blast from a Point Source

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點接種源으로부터 벼 稻熱病 擴散의 傾斜

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ABSTRACT

Rates of lesion development over time and disease gradients over distance for blast disease on the two rice varieties, Brazos and M-201 were significantly affected by two different cultural conditions, upland and flooded conditions. Flooding rice field plots lowered the rates of lesion increase and flattened the disease gradients for both varieties. Despite absence of statistically significant differences in the rate of lesion increase between four sampled distances from infection focus, rate of lesion development tended to be slightly greater as distance from the infection focus increases. Rate of lesion increase was greater with more susceptible variety M-201 than with Brazos. Disease gradient was steeper for M-201 than for Brazos. As blast disease progressed, disease gradients became flattened regardless of variety due to the infections originated from secondary foci. Between two empirical disease gradient models examined, Kiyosawa & Shiyomi model was fitted better over Gregory model. Rates of blast isopath movement under upland conditions were calculated as approximately 0.2m/day and 0.4 m/day for Brazos and M-201, respectively. The results in this study suggest that differences in varietal resistance to blast could be detected by measuring disease gradient as efficiently as by measuring infection rate.

Key words: rice, disease gradient, epidemiology, *Pyricularia oryzae*, isopath.

要 約

벼 品種 Brazos와 M-201의 稻熱病에 對한 單位時間당 感染速度와 點接種源으로부터의 單位 거리당 擴撒傾斜는 밭논과 물논의 두 栽培條件에 따라 크게 달랐다. 물논栽培는 稻熱病의 感染速度를 늦추고 擴散傾斜를 緩慢하게 하였다. 點接種源으로부터 거리별로 4地點에서 測定된 稻熱病의 感染速度는 거리에 따른 統計的인 有意差는 없었지만 點接種源으로부터의 거리가 멀어짐에 따라 빨라지는 傾向이었다. 品種別 感染速度는 Brazos보다 더 罹病性인 M-201 品種에서 높았고 擴散傾斜도 M-201 品種에서 가파른 傾向이었다. 그러나 稻熱病이 進展함에 따라 生成된 二次傳染源 때문에 稻熱病 擴散傾斜는 두 品種에서 모두 緩慢해졌다. 調査된 擴散傾斜의 두 經驗的 모델 中에서 Kiyosawa와 Shiyomi 모델이 Gregory 모델에 比하여 統計的 適合性이 높았다. 밭상태에서 單位時間當 稻熱病 isopaths 移動거리는 Brazos와 M-201 品種에서 각각 0.2 m/日와 0.4 m/日로 測定하였다. 以上의 結果, 稻熱病에 對한 品種抵抗性의 差異는 感染速度 뿐만 아니라 擴散傾斜의 測定에 의하여 效果的으로 感知될 수 있다고 생각된다.

INTRODUCTION

Two main approaches in analysis of plant disease epidemics are to characterize disease progress in time and disease spread in space (2, 3, 11). In this type of analysis, simple mathematical equations are often used as models to describe the relationships between disease spread and epidemiological time (11) or distance (2,4). The equations are frequently transformed to simple linear regression equations to obtain infection rates in unit time or disease gradients in unit distance. Either infection rates or disease gradients have been used as parameters to evaluate cultivar resistance to certain diseases in fields (1, 9, 10). Among few researchers who examined the relationships between infection rates and disease gradients in an epidemic, MacKenzie (10) and Berger and Luke (1) reported that disease gradients did not differ between varieties that had different infection rates. In addition, Berger and Luke (1) calculated the rate of isopath movement and used it as a parameter of spatial spread to rank variety resistance.

The disease gradients of rice blast have been studied little so far. Therefore, the purposes of this study were to characterize the dispersal gradients of blast disease under two different commercial cultivation conditions and to examine the relationships between infection rates and gradients of the disease. Some other epidemiological studies relating to this research have been published (5, 6, 7).

MATERIALS AND METHODS

Two rice varieties; M-201, susceptible to blast, and Brazos, partially resistant to blast, were drill-planted with drills spaced 18cm apart. Planting rate was 112kg/ha and fertilizer was applied at planting at the rate of 672 kg/ha of 20-10-10 (N-P_2O_5-K_2O). A split-plot design was employed with two irrigation practices, flooded or unflooded simulating commercial paddy or upland conditions, assigned to main plots and the two varieties assigned to subplots. Subplots each consisting of 29-row drill strips 4.6m long were separated by a 0.9m alley. Main plots were isolated from each other by 1.5m wide earthen levees and 6.1m wide ditches so that each main plot could be irrigated independently. In flooded plots, the water was maintained at 5-15cm in depth. The upland (unflooded) plots were flushed when necessary for plant growth. Water treatments started from 33 days after planting and were replicated six times.

Data collection points in each subplot were marked with garden stakes in transects oriented in eight compass directions (N, S, E, W, NW, SW, NE, SE). The points were 0.61, 1.22, 1.83 and 2.44m from the plot center. Therefore each plot had a total of 32 data collection points.

To initiate epidemics, a plant inoculated with the isolate 74L2 of race IH-1 of *Pyricularia oryzae* having four susceptible type lesions was introduced 8 days after the beginning of water treatment into the center of each subplot as a point source of inoculum. Number of susceptible type lesions developed on the plants at the data collection points was counted at 3-4 day intervals.

Rates of lesion increase in each subplot were calculated from a linear regression of ln X with time in days, where X is number of susceptible type lesions.

Disease gradients in each subplot were obtained using two empirical models:

$$Y = a X^b \text{ (reference 2)}$$

$$Y = c \exp(-dX) \text{ (reference 8)}$$

where Y = number of susceptible type lesions; X= distance from the infection source; a,b,c and d are constants. Both equations were transformed to as follows:

$$\ln(Y) = \ln(a) + b \ln(X)$$

$$\ln(Y) = \ln(c) - d X$$

Above equations were used to obtain disease gradients by regressing transformed number of lesions ($\ln(Y)$) against transformed distance ($\ln(X)$) or untransformed distance (X) from the infection source. Coefficients of determination (R^2) of the two linear regression models were used to compare the model fitness.

RESULTS

Lesion development at sampled distance. Rates of lesion increase were higher in upland plots than in flooded plots on both varieties (Fig. 1). Lesion development was particularly retarded under flooded conditions with Brazos. Blast lesions were developed more rapidly on M-201 than Brazos under flooded conditions but were developed at similar rate on both varieties under upland condi-

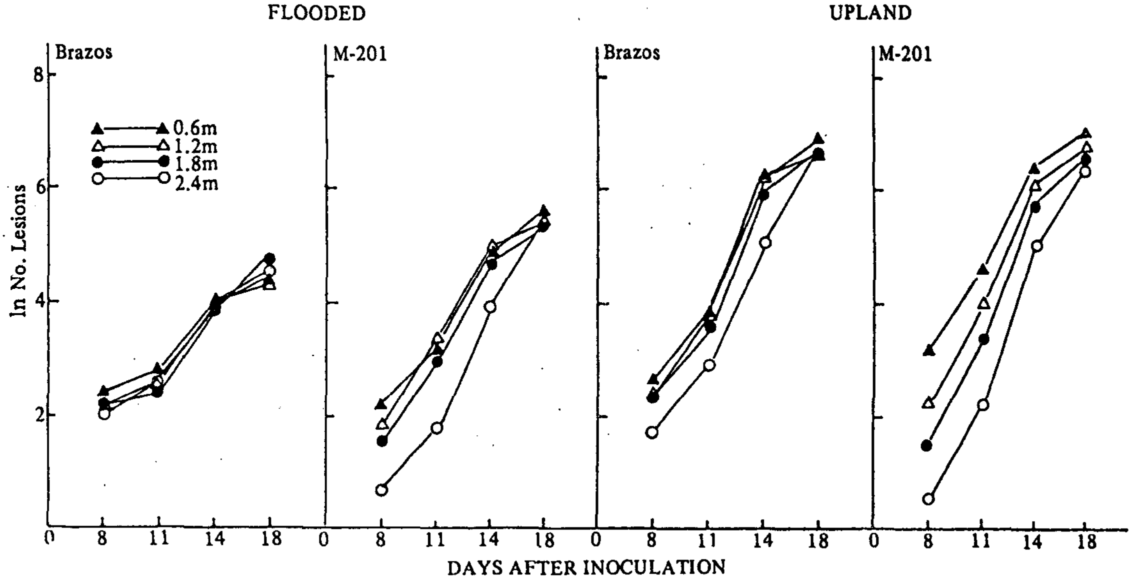

Fig. 1. Lesion increase of *Pyricularia oryzae* on the varieties, Brazos and M-201 grown under flooded and upland conditions at four sampled distances; 0.6, 1.2, 1.8 and 2.4m from infection focus during 18-day period following inoculation. Simple linear regression coefficients of each line under the same variety and the same cultural condition were not significantly different at P=.95 level.

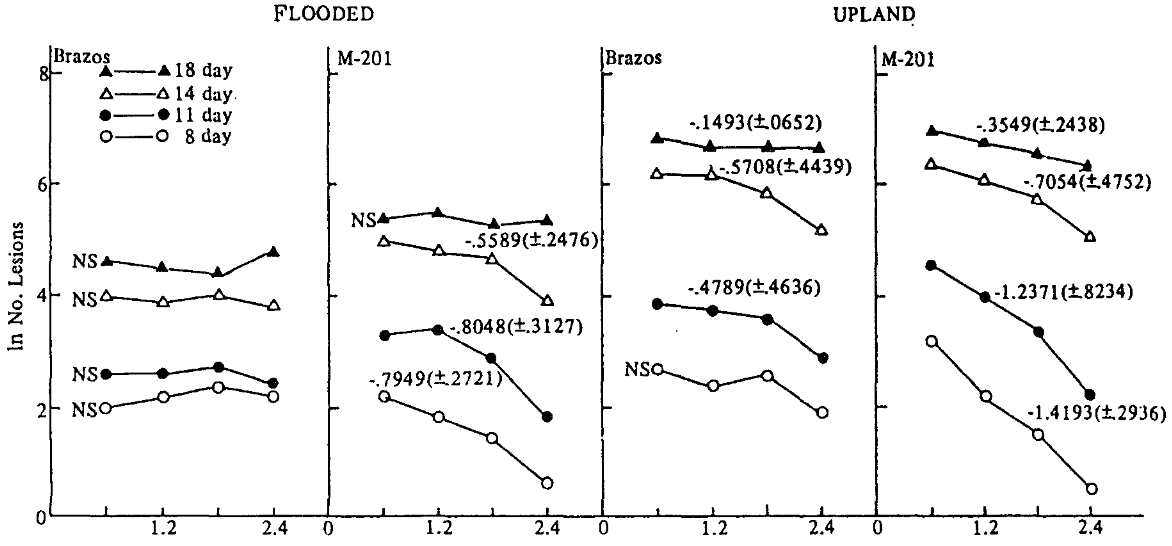

Fig. 2. Disease gradients for *Pyricularia oryzae* on the varieties Brazos and M-201 grown under flooded and upland conditions at 8, 11, 14 and 18 days after epidemic initiation. Each point is the average of 48 ratings (8 direction x 6 replications). Regression coefficients of linear model for each line were indicated with 95% confidence intervals. NS means no statistical significance of the linear model.

tions. Number of lesions developed on both varieties tended to decrease as distance from the infection focus increases, although magnitude of differences in lesion number between sampled distances differed with variety and treatment. This tendency was most obvious with M-201, particularly under upland conditions. Blast fungus developed lesions at about similar rate at all sampled distance from the initial focus in both varieties; i.e. the slopes of the lines at each distance did not differ significantly for each variety and treatment combination (Fig. 1).

Disease gradients. Among two empirical models used, Kiyosawa & Shiyomi model described better the relationships between lesion development and distance from the infection focus (Table 1), and therefore was used to obtain disease gradients (Fig. 2). Both models were not applicable for Brazos under flooded conditions, where disease gradients rarely formed (Table 1). Two models also failed to fit actual disease progress in unit distance for M-201 under flooded conditions examined 18 days after inoculation, and for Brazos under upland conditions 8 days after inoculation (Table 1). In other cases, both models were statistically significant for depicting the disease gradients. Model fitness was always greater with Kiyosawa & Shiyomi model having higher value of coefficients of determination. Under the same cultural conditions, both models fitted disease gradients better with more susceptible variety M-201. Disease gradients were also steeper with M-201 under the same cultural conditions (Fig. 2). As epidemic progressed, disease gradients became flattened for both varieties.

Rate of blast movement of isopath (annuli of same amount of disease from the infection foci) was calculated as a measure of blast spread over both distance and time. Under flooded conditions, blast fungus on Brazos developed the same number of lesions at 1.8m distance 8 days after inoculation as at 2.4m distance 11 days after inoculation. To obtain rate of isopath movement, the interlinear distance (2.4-1.8m = 0.6m) was divided by the number of days needed to reach the same severity (11-8day = 3 day). So rate of siopath movement of blast in this case was 0.2m/day. In the similar way, rate of blast isopath movement for M-201 was calculated as 0.4m/day under both upland and flooded conditions. Rate of isopath movement for Brazos under upland conditions was not obtained because no identical amount of disease reached at any sampled distance and at any date of examination.

DISCUSSION

Rate of lesion increase and disease gradients were affected significantly by variety and cultural conditions in the present study. Flooding increased the level of blast resistance of the varieties resulting

Table 1. Fitness of two empirical disease gradient models as indicated by coefficient of determination (R^2) of the models for rice blast disease on the varieties Brazos and M-201 grown under flooded and upland conditions

Days after epidemic initiation	Coefficient of determination (R^2)							
	Upland				Flooded			
	Model I[a]		Model II[b]		Model I		Model II	
	Brazos	M-201	Brazos	M-201	Brazos	M-201	Brazos	M-201
8	NS[c]	.9629	NS	.9956	NS	.8536	NS	.9984
11	.6728	.8494	.8328	.9558	NS	.5663	NS	.7513
14	.6415	.8466	.8141	.9546	NS	.6869	NS	.8365
18	.9163	.9692	.9640	.9980	NS	NS	NS	NS

[a] $\ln(Y) = \ln(a) + b\ln(X)$ (2), where Y=number of lesions; X=distance from the infection focus; a and b are constants.

[b] $\ln(Y) = \ln(c) - dX$ (8), where Y and X are the same as noted in foot note a; c and d are constants.

[c] The model is not statistically significant by F-test.

lowered slopes of regression lines of lesion increase. Varietal differences in resistance to blast under both cultural conditions were also disclosed by the slope differences in lesion development. Differences in lesion development between the two varieties at various sampled distance appeared to be greater as resistance level of rice variety decreases. This was shown by wide intervals between regression slopes. Similar slopes of lesion increase between sampled distances observed in the same variety and the same cultural conditions indicate that disease progress can be effectively monitored regardless of distance from infection foci. Despite no significant differences in slopes of lesion increase between sampled distances, rate of lesion increase tended to be slightly greater as distance from infection foci increases, and thus initial severity of blast becomes less. This tendency was also reported in other research (9).

Disease gradients were steeper with more susceptible variety M-201. Steeper gradients were also observed under upland conditions, where blast was more severe. The observation was more evident where disease gradients did not form for more resistant variety Brazos under more resistant cultural conditions, flooded fields. Steeper disease gradients on susceptible variety relative to resistant variety have not been reported. Since this observation was based on only two varieties and two cultural conditions, it remains to be verified with more number of varieties having various levels of resistance. Reasons for absence of disease gradients observed on Brazos under flooded conditions are not clear. However, there was no evidence that conidia of *P. oryzae* dispersed differently from infection foci under flooded conditions compared to upland conditions, since disease gradients were obtained with the other variety M-201 under flooded conditions. Observed differences in disease gradients between upland and flooded conditions might come from the process after conidia of *P. oryzae* were deposited at infection courts on leaf surface. Rate of successful penetration by conidia deposited at infection courts under flooded conditions might not be consistent between the four sampled distances due to the changes associated with flooding. Disease gradients of *P. oryzae* under flooded conditions might be more apt to be influenced by some other factors rather than distance from infection foci. Evidence for this explanation could be found with increased error variance in disease gradients under flooded conditions compared to upland conditions.

Flattened disease gradients over time observed in this study were mainly due to the infections originated from secondary foci as epidemic progressed. This was also commonly observed in other researches (1, 9).

The results in this study suggest that difference in varietal resistance to blast could be detected by measuring either infection rate or disease gradient. Infection rates have been used as a common criterion for screening varietal resistance (11). Disease gradients have not been an efficient criterion to detect differences in resistance level between varieties on stem rust in wheat (10) and crown rust in oat (1) because of their insensitivity. With rice blast the present study suggests that higher infection rate goes together with steeper disease gradient that could be used as one criterion for comparing varietal resistance.

REFERENCES

1. BERGER, R. D. & LUKE, H. H. (1979). Spatial and temperal spread of oat crown rust. *Phytopathology* 69:1199-1201.
2. GREGORY, P. H. (1968). Interpreting plant disease dispersal gradients. *Ann. Rev. Phytopathol.* 6:189-212.
3. JEGER, M. J. (1983). Analysing epidemics in time and space. *Plant Pathology* 32:5-11.
4. KABLE, P. F., FRIED, P. M. & MACKENZIE, D. R. (1980). The spread of a powdery mildew of peach. *Phytopathology* 70:601-604.
5. KIM, CHOONG-HOE, RUSH, M. C. & MACKENZIE, D. R. (1985). Effect of water management on the epidemic development of rice

blast. *Phytopathology* 75:501.

6. KIM, CHOONG-HOE, RUSH, M. C. & MACKENZIE, D. R. (1986). Flood-mediated resistance to the rice blast disease. *Proc. Inter. Confer. Wetland Utilization for Rice Production in Subsaharan Africa.* at IITA, Ibadan, Nigeria, 4-8 Nov. 1985. p 151-160.
7. KIM, CHOONG-HOE, RUSH, M. C., MACKENZIE, D. R. & MARCHETTI, M. A. (1987). Effect of water-management on rice blast epidemiology and flooding dependent resistance. *Phytopathology* 77: (in press).
8. KIYOSAWA, S. & SHIYOMI, M. (1972). A theoretical evaluation of the effect of mixing resistant variety with susceptible variety for controlling plant disease. *Ann. Phytopathol. Soc. Japan* 38:41-51.
9. LIM, S. M. (1978). Disease severity gradient of soybean downy mildew from a small focus of infection. *Phytopathology* 68:1774-1778.
10. MACKENZIE, D. R. (1976). Application of two epidemiological models for the identification of slow stem rusting in wheat. *Phytopathology* 66:55-59.
11. VANDERPLANK, J. E. (1963). *Plant Disease: Epidemics and Control.* Academic Press, New York, 349 pp.