

Low Night Temperature and Blast Disease Development on Rice

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ABSTRACT

Rice cultivars normally resistant to blast differentially succumb to the disease when exposed to varying periods of low night temperature (15 C) treatment before inoculation. The observations explain variation in

pathogenic race identification and variation in the performance of resistant cultivars in different regions.

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Blast of rice, caused by *Pyricularia oryzae* Cav., is one of the most important diseases of the crop, and occurs in almost all rice-growing regions of the world. Suryanarayanan (7) reported that rice seedlings grown at a low night temperature of 15-20 C showed profoundly increased infection and disease development compared with plants grown at night temperatures of 26 C or higher. The effect of low night temperature has been explained as the result of accelerated nitrogen metabolism of the host (6).

The inherent genetic variability of *P. oryzae* is well established (1, 5, 9). This paper reports on the effect of different periods of low night temperature on the susceptibility of differential host cultivars.

MATERIALS AND METHODS.—The isolate of *P. oryzae* employed in the pathogenicity trials came from a single spore from blast-infected rice leaves from Madras, India. It was virulent on the susceptible CO 13 *indica* variety and on certain species of Gramineae in repeated trials.

Five rice cultivars belonging to the set of international differentials were used for pathogenicity trials together with Lacrosse and CO 13, included for comparison.

Seedlings were raised in 8-cm plastic pots (8/pot) of Jiffy Mix (Products of America, Chicago, Ill.) in two growth chambers (Environmental Growth Chambers, Inc., Chagrin Falls, Ohio) with a 12-hr dark period and a 12-hr light period (22,500 lux at soil level). They were watered daily with tap water and fed weekly with 50 ml 0.5% Hyponex (Hydroponic Chemical Co., Copley, Ohio) per pot. Day temperature throughout was maintained at 30 C. In one chamber, the night temperature was 15 C, whereas in the adjacent chamber it remained at 30 C. The seedlings were raised for 14 days at 30 C, and then a series of treatments was begun by transferring batches of plants between chambers. There were three basic treatments, namely 12, 8, or 4 hr in the dark at 15 C with the balance of the dark period at 30 C, and these were carried out for 12, 8, and 4 days before inoculation. The treatments thus formed a graded series of exposures to 15 C in darkness, ranging from a total of 16 hr (4 days at 4 hr) to a

total of 144 hr (12 days at 12 hr). Seedlings were also grown at 30 C day and night as a control.

At the end of the low night-temperature treatments, on the 26th day, two sets of the seven cultivars (12-15 tillers each) for each treatment were inoculated at the leaf sheaths by a syringe inoculation technique (3) using a suspension of 2×10^4 conidia/ml. The plants were incubated in a moist chamber (98% relative humidity) at 25-27 C for 24 hr, then transferred to the chamber maintained at 30 C. The disease symptoms appeared on the emerging leaves above and below the needle punctures. On the 6th day after inoculation, disease ratings were made on the basis of the lesion development as proposed by the American and Japanese Cooperative Study (8).

RESULTS AND DISCUSSION.—The results are shown in Table 1. Three cultivars, Kanto 51, Lacrosse, and Caloro, were resistant in all treatments. The remaining cultivars, although resistant when kept at 30 C (except for the incubation period), varied in their resistance depending on the low night temperature to which they had been exposed. The cultivar CO 13 was susceptible in all treatments, whereas NP-125 became susceptible after all treatments except 16 hr (4 days at 4 hr); and Usen, after all except 16 and 32 hr. Lesion size and development on these cultivars did not vary significantly among the effective treatments. In contrast to these, Raminad Str. 3 showed a susceptible reaction only after 144 hr (12 days at 12 hr) of low night-temperature treatment. Although Kanto 51 and Lacrosse were resistant in all treatments, the reaction type varied in response to treatment. Figure 1 shows the reactions of representative leaves of all seven cultivars maintained at 30 C (0 hr at 15 C) and of leaves which received 144-hr (12 days at 12 hr at 15 C) treatment. The '—' or symptomless reaction on Kanto 51 and Lacrosse from the 0-hr treatment was accompanied by leaf lesions that were the result of needle injury at inoculation. The R lesions following the 144-hr treatment were quite pronounced on these two cultivars. The results show that low night-temperature

TABLE 1. Blast development on rice cultivars after varying periods of low night temperature (15 C)

Rice cultivar	Reaction types ^a									
	Treatment periods ^b									
	Control	12 days			8 days			4 days		
	Constant 30 C	12	8	4 hr	12	8	4 hr	12	8	4 hr
Raminad Str. 3	R	S	R	R	R	R	R	R	R	R
NP-125	R	S	S	S	S	S	S	S	S	R
Usen	R	S	S	S	S	S	S	S	R	R
Kanto 51	—	R	R	—	R	—	—	R	—	—
Lacrosse	—	R	R	—	R	—	—	R	—	—
Caloro	R	R	R	R	R	R	R	R	R	R
CO 13	R	S	S	S	S	S	S	S	S	S

^a R = Resistant. S = Susceptible. — = No symptom.

^b Treatments began after 14 days at 30 C.

treatment leads to a partial breakdown of resistance.

Plants that received 144 hr of low night temperature were 10-15% shorter than those grown at a constant 30 C. Intermediate treatments had effects between these extremes. Whether low night temperatures altered the physiological age of the host tissue or affected host metabolism in some other way was not studied. We also do not know whether the effect of low temperature is independent of the dark period. We suspect that low day temperatures may

have similar effects. Ohata et al. (4) found that rice plants exposed to 19 C day and night for 6-12 days before inoculation were more resistant than control plants kept at 26 C. However, by 6-18 days after treatment, the situation was reversed and the cold-treated plants were more susceptible because by that time their rate of growth was greater than that of the control plants. Such changes in growth rate could also have occurred in our material at the time of inoculation, and may have contributed to variation in

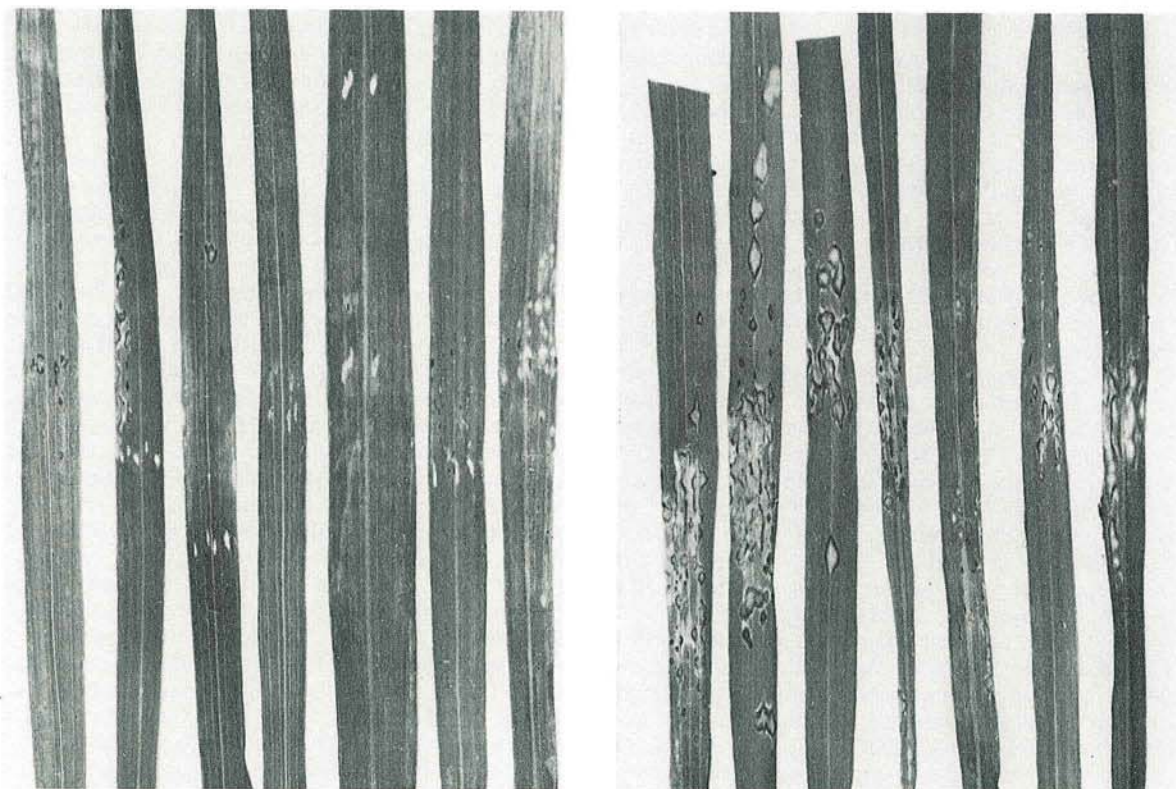


Fig. 1. Reactions of leaves of seven rice cultivars maintained at 30 C (0 hr at 15 C) prior to inoculation with *Pyricularia oryzae* (left) and of leaves which received 144 hr (12 days at 12 hr) of low night temperature, 15 C (right). Rice cultivars, left to right: Raminad Str. 3, NP-125, Usen, Kanto 51, Lacrosse, Caloro, and CO 13.

blast development such as that observed on Raminad Str. 3.

Evidently, preinoculation conditions affect the reactions of some of the standard differential cultivars and will therefore, unless controlled, introduce errors in pathogenic race identification. Since low night temperatures of 15-16 C are not uncommon in hilly areas in TamilNadu, India, during the rice growing seasons (2), our results also explain why severe infection sometimes occurs on cultivars which are satisfactorily resistant on the plains.

LITERATURE CITED

1. GIATGONG, P., & R. A. FREDERIKSEN. 1969. Pathogenic variability and cytology of monoconidial subcultures of *Piricularia oryzae*. *Phytopathology* 59:1152-1157.
2. GOVINDASWAMY, C. V. 1964. Temperature and blast disease incidence. *Madras Agr. J.* 51:255-257.
3. MANIBHUSHANRAO, K. 1971. Studies on the cultures of *Piricularia oryzae* Cav. I. On their morphology and pathogenicity on rice and certain spp. of Gramineae. *II Riso* 20:283-297.
4. OHATA, KAN-ICHI, K. GOTO, & T. KOZAKA. 1966. Effect of low air temperature on the susceptibility of rice plants to blast disease, with special reference to some chemical components in the plants. (In Japanese, English Summary) *Nat. Inst. Agr. Sci., Bull.* 20:1-65.
5. OU, S. H., & M. R. AYAD. 1968. Pathogenic races of *Piricularia oryzae* originating from single lesions and monoconidial cultures. *Phytopathology* 58:179-182.
6. SADASIVAN, T. S., S. SURYANARAYANAN, & L. RAMAKRISHNAN. 1965. Influence of temperature on rice blast disease, p. 163-171. *In* The rice blast disease. Johns Hopkins Press, Baltimore.
7. SURYANARAYANAN, S. 1958. Role of nitrogen in host susceptibility to *Piricularia oryzae*. *Curr. Sci.* 27:447-448.
8. UNITED STATES AND JAPAN COOPERATIVE STUDY. 1967. An international set of rice varieties for differentiating races of *Piricularia oryzae*. *Phytopathology* 57:297-301.
9. YAMASAKI, Y., & H. NIIZEKI. 1965. Studies on variation of rice blast fungus *Piricularia oryzae* Cav. I. Karyological and genetical studies on variation [In Japanese, English Summary]. *Nat. Inst. Agr. Sci. Bull. D.* 13:231-274.