J Indian Bot Soc.67 (1988) 97-100

Cross Protection in Rice to Sheath Blight

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(Accepted June 1987)

Incompatible interaction (12 h) of rice leaves with rice-grain culture of the avirulent *Rhizoctonia solani* (R7) isolate from potato gave significant protection (90%) against differentially virulent *R. solani* isolates (R1 and R5). Three rice cultivars, namely TKM 9, Ponni and IR 50 acquired high degree of resistance to sheath blight in response to prior-inoculation with avirulent isolate.

Key Words - Avirulent Incompatible Protection Rice Virulent.

Sheath blight caused by *Rhizoctonia solani* is one of the major fungal diseases of rice. It has of late become a severe constraint to rice cultivation in India. The peculiar versatility of the pathogen tended to defeat attempts to breed for resistance and practically no resistant variety is available. The subterranean nature of *R. solani* rendered the chemical control ineffective. Since these classical approaches to disease control have not been feasible, use of biocontrol agents and induction of host resistance will be the viable alternatives to control rice sheath blight.

Ever since Chester's review in 1933, cross-protection has been demonstrated in several hosts. Of late, systemic immunization of plants to viral, bacterial and fungal pathogens (Kuc, 1982) has been reported.

Induction of resistance in rice to *Pyricularia oryzae* (Watanabe, 1951) and *Helminthosporium oryzae* (Trivedi & Sinha, 1976) has been known. Recently, acquisition of resistance in rice to *R. solani* in response to biotic (Kalaiselvi *et al.*, 1986) and abiotic stimuli (Waheeta *et al.*, 1987) has been reported. In this study the efficacy of an avirulent *R. solani* isolate to induce resistance in rice against differentially virulent isolates has been examined.

MATERIALS & METHODS

Rice cultivars TKM 9, Ponni and IR 50 (60 day-old), virulent *R. solani* isolates R1 and R5 (ATCC Nos. 48502 and 48505) respectively (Zuber & Manibhushanrao, 1982) and avirulent isolate R7 from potato (Waheeta *et al.*, 1987) were used. Sclerotia collected from 5-day-old PDA grown culture of R1/R5 were used for inoculation (compatible interaction). Rice seeds (cv. IR 20) were boiled in water (1:25, w/v) for 30 min, supplemented with dextrose (5%) and autoclaved. The sterilized prepration was inoculated with a 5 mm disc of 5-day-old PDA-grown culture of R7. The rice-grain culture (7 day-old) as well as the aqueous mycelial suspension of R7 prepared by macerating the mycelial mat obtained from liquid culture (potato dextrose broth) was used for inoculation.

Leaf segments (12-15 cm) were floated on 50 mL of kinetin solution (5 ppm) kept in 20 cm diameter Petri plates. The leaf bits were initially inoculated with either a drop of the suspension or single grain culture of R7 at two sites followed by the sclerotia of R1/R5 isolate at 12, 24 and 48 h after the primary inoculation at the site of R7 interaction or 2-3 cm away. Appropriate controls with three replicates were maintained. Inoculated rice leaf bits cleared in chloral hydrate were observed under a microscope. The number of lesions per leaf and the average lesion length in cm were recorded at 24 h interval starting from 48 h after challenge inoculation till 120 h. Disease severity index (DSI) was determined by multiplying the average lesion length in cm with the average number of lesions per leaf and the percentage protection obtained was calculated as follows: DC - DP/DC x 100, where DC and DP are the DSI on control and prior-inoculated respectively.

RESULTS & DISCUSSION

The isolate R7 from potato did not exhibit any direct antagonism to the virulent isolate in dual culture on PDA. On rice leaves (TKM 9, PONNI and IR 50) mycelium from R7 inoculum (rice-grain culture) grew as early as 12 h after inoculation, but did not spread further rapidly. Microscopic observation revealed that R7 neither produced infection cushions nor penetrated the host through stomata. However, small yellow spots (2-3 mm) which later turned brown developed at the site of inoculation.

Prior inoculation of excised rice leaves (TKM 9) with the rice grain culture of R7 allowed very little spread of the challenger (R1) leading to significant mitigation (90%) of sheath blight severity, while the mycelial suspension of R7 did not offer any such protection. Interestingly, even 12 h

Table 1. Efficacy of the Inducer Inoculum in Disease Suppression on TKM 9.

Inoculum	Disease severity index (DSI) h after R7 inoculation 12	24	48
Control (R1)	7.35	4.55	4.14
Mycelial suspension (R7) + R1	7.53	7.51	7.76
* Grain culture (R7) + R1	0.34 (95)	0.19 (96)	0.113(97)
+Grain Culture (R7) + R1	2.5 (66)	--	--

* : R1 same site

+ : R1 different site

Values in parenthesis show percent protection

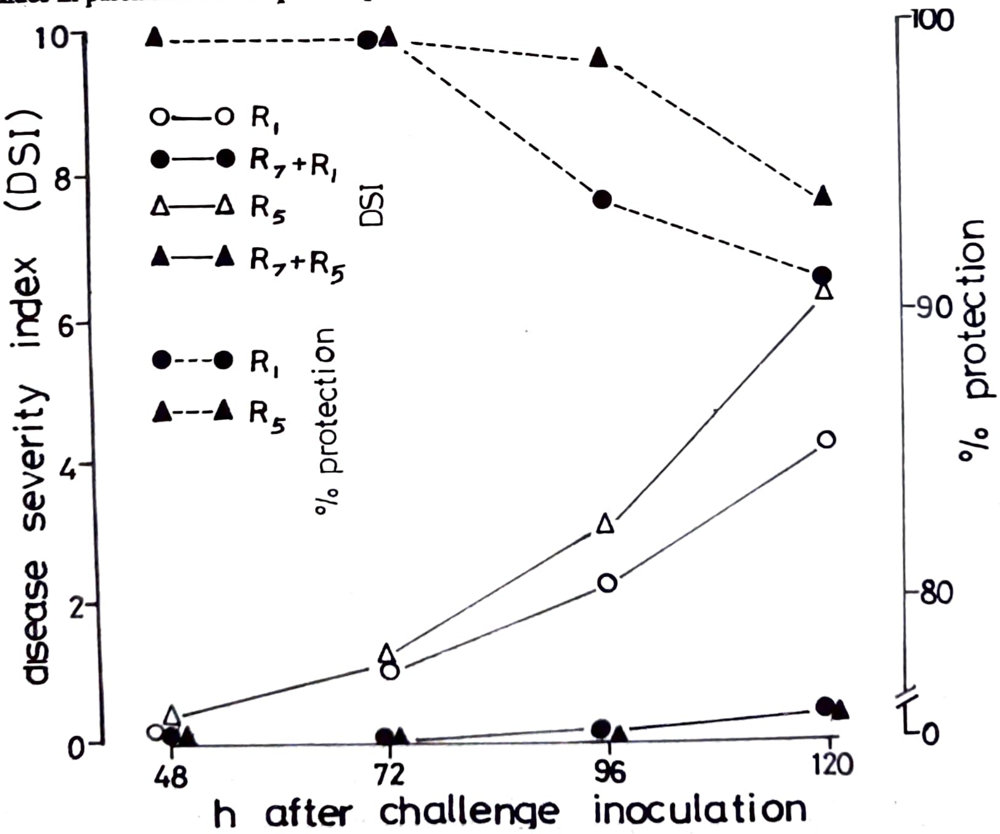

Fig.1. Efficacy of R7 against differentially virulent *R. solani* isolates

Table 2 Varietal Response to Incompatible Interaction

Variety	Treatment	Disease severity index (DSI) Interval (h) after challenge inoculation							
		48		72		96		120	
TKM 9	R5	3.6		6.5		8.9		9.5	
	R 7 + R 5	0	(100)	0	(100)	0.05	(99)	0.2	(98)
Ponni	R5	1.84		5.54		8.15		10.9	
	R 7 + R 5	0.07	(96)	0.3	(95)	0.97	(88)	1.33	(88)
IR 50	R5	0.87		2.81		6.87		8.44	
	R 7 + R 5	0	(100)	0.06	(98)	0.21	(97)	0.43	(95)

- : No Visible Symptoms Values in parenthesis indicate the percent protection

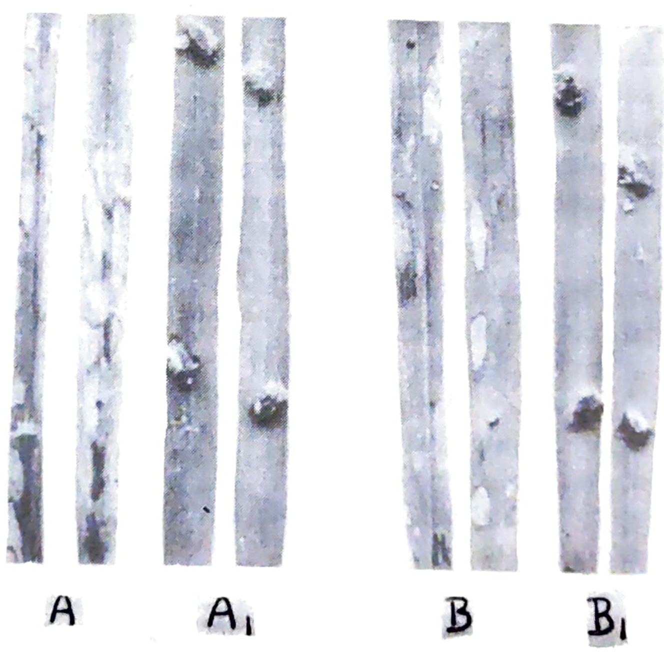

Fig. 2. Cross Protection in Rice Leaves Against Sheath Blight

A : Ponni leaves inoculated with R5 (Control)

A_1 : Ponni leaves prior interacted with R7 and inoculated with R5

B : IR 50 leaves inoculated with R5 (Control)

B_1 : IR 50 leaves prior interacted with R5 and inoculated with R5 prior inoculation with R7 effectively protected the rice tissue against R1 invasion (Table 1), and hence the same combination was followed in further experiments. Resistance induced in rice was basically localized as the degreee of protection decreased when R1 was inoculated a few cm away from the site of R7 inoculation (Table 1).

In addition to the less virulent isolate R1, the efficacy of R7 to induce resistance against a highly virulent isolate R5 was tested. Incompatible interaction conditioned the host tissue (TKM 9) effectively providing about 90% protection against both the isolates despite their differential virulence (Fig. 1).

Besides TKM 9, Ponni and IR 50 were also checked for their response to incompatible interaction (R7). Interestingly, the susceptible cultivars displayed high degree of acquired resistance to *R. solani* (R5) resulting in marked reduction of sheath blight severity (Table 2 and Plate 1).

By using mycelial discs of avirulent isolate R7, Waheeta *et al* (1987) demonstrated induced resistance in rice leaves against *R. solani* which offered about 80% protection. This study shows the efficacy of rice grain culture of R7 to give around 90% protection against two virulent isolates as well as in three different varieties, even with 12 h of prior interaction with the inducer. Quantity of the inducer inoculum and its ability to spread and interact with the host tissue much earlier appear to be responsible for the induction of resistance when grain culture was used. Presumably, the mycelial suspension lacked these abilities.

The mechanism(s) of induced resistance, namely, direct antagonism (Yarwood, 1956), physical obstruction (Kochman & Brown, 1975), formation of physical/chemical barriers and anatomical changes (Davis, 1966; Ouchi, 1983) varies with the host-pathogen-inducer combination. Further, biotic immunization of plants has recently been explained as a two step-process where the host is merely sensitized and the latent resistance mechanism is rapidly expressed in the face of pathogen attack.

Acquisition of resistance in rice to *H. oryzae* was attributed to the fungitoxicity generated in the host by the preinoculant (Trivedi & Sinha, 1976). Cross-protection in *Panicum repens* to *P. oryzae* has been explained in terms of competition between the two isolates for infection sites and interaction between the host and the avirulent isolate (Hilda & Suryanarayanan, 1976). However, in the present system, the inducer neither produced infection cushions nor penetrated the host through stomata. The sclerotia of the virulent isolate germinated and the mycelium spread rapidly forming typical infection cushions even during 24 h on the control leaves but not on the leaves previously inoculated with the avirulent R7 isolate. Influence of host exudates on *R. solani* growth and infection cushion formation is already known (Dodman, 1970). In the absence of direct antagonism and physical obstruction, nutrient competition and/or induced resistance appears to be a plausible mechanism as in the case of *Aqrostis palustris* - *R. solani* system (Burpee & Goulty, 1984).

Incompatible interaction of rice leaves with an *R. solani* isolate from groundnut as well as with *R. bataticola* also reduced the sheath blight severity considerably (unpublished). The underlying mechanism(s) and the feasibility of inducing systemic resistance in rice with either of these fungi or their products are being investigated.

Acknowledgement We thank Prof. A. Mahadevan, Director, C.A.S. in Botany, University of Madras, for facilities.

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