MARDI Res. Bull. 3, 2: (19 – 24)

THE SURVIVAL, SPREAD AND HOST RANGE OF *RHIZOCTONIA SOLANI*, THE CAUSAL ORGANISM OF SHEATH BLIGHT DISEASE OF RICE*

K.M. CHIN** and W.C. LIM***

Accepted for publication on 29 April, 1975

RINGKASAN

Pokok-pokok tumpangan bagi *Rhizoctonia solani*, kebolehan hidup dan rebakannya di antara musim ke musim padi telah diselideki dalam beberapa percubaan. Beberapa corak tanaman untuk mengawal penyakit ini disyorkan.

INTRODUCTION

Sheath blight disease of rice was first reported in Malaysia as a minor disease by THOMSON in 1936. Recent observations however suggest that the disease is severe in the same locations every season. Studies were therefore conducted to determine the methods of survival and spread of the pathogen between crops with the view of developing cultural techniques for controlling the disease.

METHODS AND MATERIALS

(I) Survival in the soil

Cultures of the fungus on 2 cm. straw segments were obtained as described by CHIN (1973), and sclerotia were produced on potato dextrose agar plates. These were then buried one centimetre deep in padi soil contained in pots. An equal number of pots were either maintained in a moist condition or allowed to dry.

During intervals of 2, 4, 8, 16, 32 and 64 weeks, twenty pieces each of infected straw and sclerotia were recovered and rinsed in padi field water. They were tested for infectivity by inoculating onto six week-old padi plants of variety RD 3. The number of infected plants were determined ten days after inoculation.

(II) Host range

Seventeen weed species commonly found in rice fields around Bumbong Lima, North Malaysia, were collected and transplanted into pots. The speices were identified with the aid of "Malayan Wild Flower Parts I & II" (HENDERSON, 1954 and 1959) and "A Handbook on Padi-field Weeds" (SAMY *et al.*, 1968). Four crop species which are grown as rotational crops on rice fields were also used as test plants.

Five plants per pot of each species were inoculated with straw inoculum. The same number were used as controls.

---

*Part of a M.Sc. Thesis submitted to the Universiti Sains Malaysia by the Senior author.

**Pathologist, Rice Research Unit, MARDI.

***Lecturer, School of Biological Sciences, Universiti Sains Malaysia Penang, Malaysia.

All the plants were incubated inside polythene chambers to maintain a high humidity for five days. A species was considered susceptible if inoculated plants developed lesions which were absent on the control plants.

(III) Spread and infectivity of the pathogen

Sclerotia and infected straw segments prepared as described above were used throughout the experiments.

(a) Sclerotial germination and mycelial growth on soil and water surfaces

Sclerotia and infected straw segments were floated on water from the rice field, distilled water or placed on moist rice field soil contained in petri dishes. Observations of mycelial growth were made.

The experiment was repeated with the addition of healthy leaf sheath segments (from two month-old RD 3 var. rice plants) either on the soil or water surface.

(b) Mycelial growth through soil

(i) Sclerotia and infested straw were buried one centimetre deep in fresh rice field soil in petri dishes. Healthy leaf sheath segments were placed on the soil surface directly above the buried inoculum.

Observations were made daily for the appearance of mycelia above the soil surface or the infection of the sheath segments.

(ii) The experiment was repeated as in b(i) but with the lids off for 4 days after which fissures appeared on the soil surface due to drying. Healthy leaf segments were placed on the soil surface. The soil was moistened again and the petri dish lids replaced. Observations were continued for another four days.

(c) Sclerotial germination after burial in the soil

Sclerotia were buried in soil contained in petri dishes without lids. After ten days the soil was moistened and the sclerotia were recovered and placed on rice field water or distilled water. Observations on germination and mycelial growth were made.

(d) Infectivity of buried inoculum

Infested straw and sclerotia were placed in the soil both in contact with and near to roots of two-month old RD 3 var. rice plants in pots. Observations were made daily for the appearance of symptoms on the lower sheaths of the plants.

RESULTS

(i) Survival in the soil

The results of the experiment are presented in *Table 1.* The sclerotia were found to be infective for thirty two weeks under both wet and dry conditions. However from the sixteenth week onwards sclerotia buried in dry soil were less infective than those in wet soil.

TABLE 1. *INFECTIVITY (%) OF SCLEROTIA AND INFESTED STRAW SEGMENTS RECOVERED AFTER BURIAL FOR DIFFERENT PERIODS OF TIME

Time (weeks buried)	Sclerotia		Infected Straw	
	Wet Soil	Dry Soil	Wet Soil	Dry Soil
0	85	95	90	95
2	65	85	40	70
4	80	50	30	90
8	55	55	0	65
16	55	10	0	15
32	40	10	0	10
64	0	0	0	0

*Infectivity = % of plants inoculated which develop disease symptoms

Straw inoculum was infective for thirty-two weeks under dry conditions. When buried in wet soil however, infectivity was lost after the fourth week.

(ii) Host range

A summary of the results are presented in *Table 2.* The various species have been grouped under their respective families. Information on the habitat and abundance of the plants is also tabulated.

Large, irregular, greenish grey lesions (similar to that of sheath blight of rice) appeared on most of the species inoculated. In general, on grasses e.g. *Echinochloa colona,* the symptoms were closest in appearance to that on rice and appeared on both sheaths and leaf blades. On 'fleshy' plants like *Limnocharis flava* and *Jussiaea repens,* water soaked lesions rapidly became necrotic resulting in rotting of leaves, leaf stalks or stems. The sedges (eg. *Eleocharis variegata*) were less susceptible to infection and only small lesions developed.

Abundant sclerotia were sometimes produced on dead tissue e.g. on leaves of *Limnocharis flava* which had been killed by the pathogen.

(iii) Spread and infectivity of the pathogen

(a) Sclerotial germination and mycelial growth on soil and water surfaces

Sclerotia and infested straw produced mycelia on both rice field water and distilled water. These mycelia were able to float and spread on the water surface. On contact with healthy leaf sheath segments the hyphae produced typical blight lesions.

Mycelia were also readily produced on the soil surface by both sclerotia and infested straw.. Disease lesions developed on leaf sheath segments placed on the soil surface close to the inocula.

TABLE 2. HOST RANGE OF *RHIZOCTONIA SOLANI* THE CAUSAL ORGANISM OF SHEATH BLIGHT OF RICE

Family	Botanical Name	*Habitat	**Occurrence	***Susceptibility
Cyperaceae	*Eleocharis variegata*	P	2	+
	Cyperus haspan	B & P	1	+
	Fimbrystylis miliacea	B & P	1	+
	Scirpus grossus	B & P	2	+
Gramineae	*Sacciolepsis myosuroides*	B	1	+
	Paspalum conjugatum	B	1	+
	Paspalum scrobiculatum	B & P	1	+
	Echinochloa colona	B & P	2	+
	Digitaria marginata	B	1	+
	Eragrostis uniloides	B	2	+
	Axonopus compressus	B	2	+
	Zea mays	Off-season crop		+
	Sorghum vulgare	Off-season crop		+
	Saccharum officinarum	Off-season crop		+
Alismataceae	*Limnocharis flava*	P	2	+
	Sagittaria guyanensis	P	2	–
Pontederiaceae	*Monochoria vaginalis*	P	2	+
	Eichornia crassipes	P & C	1	+
Araceae	*Pistia stratiotes*	P	2	+
Onagraceae	*Jussiaea repens*	P	2	+
Solanaceae	*Capsicum annuum*	Off-season crop		+

*P : Padi field B : Batas (Earth bunds around padi fields) C : Irrigation canal
**1 : Occassional 2 : Common
***+ : Susceptible – : Not susceptible

(b) Mycelial growth through soil

The fungus was not able to grow through wet rice field soil. Buried sclerotia and infested straw failed to produce mycelia and infect healthy sheath segments placed on the soil surface.

Although the buried sclerotia and infested straw were not able to produce aerial mycelia through wet soil, on drying, the soil surface developed fissures through which the fungal mycelia were able to grow. Healthy sheath segments which were then placed on the soil surface developed lesions after three days.

(c) Sclerotial germination after burial in the soil

Sclerotia which had been buried for ten days germinated readily when recovered and placed on distilled water or rice field water.

(d) Infectivity of buried inocula

No lesions appeared on aerial parts of mature RD 3 var. rice plants when inoculum was buried in contact with and near to their roots.

DISCUSSIONS

Generally in West Malaysia, the interval between harvesting of one rice crop and transplanting of the next is not more than two months in double* cropping areas. In single-cropping areas, the land may be fallow or planted with other crops for six to eight months each year. Since the fungal sclerotia were found to be infective up to thirty two weeks in the soil, the disease may be carried from one crop to the next by buried sclerotia, particularly in double cropping areas. In single cropping areas, the inoculum potential due to buried sclerotia however may be reduced by up to 72% during the fallow season. This is consistent with field observations that the disease is more severe in double cropping areas than in single cropping areas.

Infested straw was found to be infective for 4 weeks in wet, and 32 weeks in dry soil. This suggests that the disease may also be carried over by infested straw or stubble, particularly in double-cropping areas. Infected plant remains should therefore be burnt after harvest to reduce the carry over of the disease to the next crop. Alternatively, these remains should be ploughed in and the soil kept wet in order to reduce the survival of the pathogen.

Since a large number of susceptible plants were found among common weeds of the rice field, it is also likely that these alternative hosts are responsible in part for the carry-over of the disease. *Limnocharis flava* and *Echinochloa colona* both of which are very common weeds in local padi fields, were found to be very susceptible. Lesions formed on the former also produced numerous sclerotia. This is significant as it increases the primary inoculum potential at the beginning of the season. Similarly, KOZAKA (1965) suggested that weeds may contribute to primary infection by the production of sclerotia.

The results of the experiment on alternative hosts also show that plants belonging to other families besides the Gramineae and Cyperaceae may be attacked by the fungus. This is in contrast with results obtained by KOHLI (1965). Natural infection of these weeds in the field has also been observed. Control programmes for the disease should thus include eradication of weeds. In particular fields which are being left fallow for long periods of time in order to reduce the inoculum potential should not be allowed to be overgrown with weeds.

The present study also shows that *Sorghum vulgare, Saccharum officinarum, Capsicum annuum* and *Zea mays* are susceptible to the disease. It is suggested that these crops should not be grown either as rotational or replacement crops in areas with a history of the disease.

Laboratory observations on the spread of the fungus showed that it was able to produce mycelia and grow on water or moist soil surfaces, but indicated that it was unable to grow through soil. Similar observations were made using potted plants. Inoculum buried in the soil

*Irrigated areas in which two crops of rice are grown a year

failed to infect leaf-sheaths placed on top of the soil. This is in agreement with findings by other workers (RYKER & GOOCH, 1938). However when the soil surface developed fissures due to drying, the fungus was able to grow up between these cracks. Since fissures of the soil surface are common in areas where irrigation facilities are inadequate it is possible that primary infection may not only be due to sclerotia being uncovered by cultivations (KOZAKA, 1965), but also to mycelial growth from buried inocula through these fissures.

Deep ploughing to bury fungal sclerotia and infected plant material followed by continuous flooding to prevent cracking of the soil surface are therefore recommended in the control of the disease.

ACKNOWLEDGEMENTS

The authors are grateful to Dr. W.P. Ting and B.H. Chew for useful discussions. They are also indebted to Cik H.A. Voon for technical assistance.

SUMMARY

The host range of *Rhizoctonia solani* and its ability to survive and spread between crops of rice were investigated in a series of experiments. Several cultural methods for the control of the disease are suggested.

REFERENCES

CHIN, K.M. (1973). Studies on the sheath blight disease of rice in West Malaysia. M.Sc. Thesis, Universiti Sains Malaysia, Penang, Malaysia. 67 pp.

HENDERSON, M.R. (1954). Malayan Wild Flowers (Monocotyledons) Mal. Nature Soc. Kuala Lumpur (1954). 357 pp.

HENDERSON, M.R. (1959). Malayan Wild Flowers (Dicotyledons) Mal. Nature Soc. Kuala Lumpur (1959). 478 pp.

KOHLI, C.K. (1965). Pathogenicity and host range studies on the paddy sheath blight pathogen *(Rhizoctonia solani* Kuhn.) J. Res. (Punjab. Agric. Univ.) *3* : 848–54

KOZAKA, T. (1965). Ecology of *Pellicularia* sheath – blight of Rice Plant and its Chemical Control. Ann. phytopath. Soc. Japan *31* : 179–85.

RYKER, T.C. & GOOCH, F.S. (1937). *Rhizoctonia* sheath spot of rice. Phytopathology *28* : 223–46.

SAMY, J., WONG, A. & JAAFAR, M. BIN. (1968). A handbook on padi field weeds. Rice Research Centre, Dept. Agric. Malaysia. 41 pp.

THOMSON, R.A. (1936). Annual Report, Division of Mycology. Tech. Rep. Dept. Agric. SS. & F.M.S. (1936). 55 pp.