Rhizoctonia Solani: The Cause of Patchy Stunting of Wheat in Tanzania

[1]Kuwite*, C. A., and L. Piening[2],

[1]Selian Agricultural Research Institute, P.O Box 6024, Arusha, Tanzania.
[2]5016-58st. Lacombe, Alberta, T4L 1K7 Canada.

Abstract

Patchy Stunting of Wheat (PSW) has occurred in wheat at the Hanang Wheat Complex (HWC) in Northern Tanzania since early in the 1970's. In studies conducted with the objective of identifying the cause of PAW, nine random plant and soil samples were taken from three sites in each of the seven farms comprising the HWC. Sclerotia-like structures (nodules) which mostly occurred at the root tips of infected plants were counted on five plants of each sample. There were significantly more nodules on the roots of plants collected from the centre of PSW patches than from the periphery or from the adjacent healthy-looking plants in the 1994 test ($r=0.279$, $P=0.02$) and in the 1995 test ($r=0.41$, $P=0.01$). The nodules invariably yielded ***Rhizoctonia solani*** *on potato dextroses agar. Wheat seedlings of the cultivator Viri developed typical symptoms of PSW when seeds were planted in pots of soil infested with nodules from PSW infected roots or with R. solani from culture media.* ***Rhizoctonia solani*** *always resulted from the roots of diseased plants. The Isolation of* ***R. solani*** *from PSW infected plants and subsequent pathogenic positive tests with this pathogen suggest that this fungus is the causal agent of PSW in Tanzania.*

Keywords: Rhozoctonia solani, Sclerotic, Nodules, Patchy stunted wheat

Introduction

Patchy stunting (PSW) on wheat (*Triticum aestivum* L.) has been observed on wheat and barley at the farms of the Hanang Wheat Complex (HWC) of Northern Tanzania since the early 1970's (Tinline 1977 pers comm., Kuwite *et al.*, 1995). Over 30,000 ha of wheat is grown annually of which 5 - 10% is affected by PSW. Diseased wheat was observed on all soil types at the HWC namely Verticals, Mollisols, Inceptisols and Alfisols.

Symptoms of PSW first appear on wheat in the field 4 - 5 weeks after sowing. Diseased plants occur in irregularly shaped patches of one to 10 or more metres in diameter. Infected plants are stunted and yellow and as the disease progresses, the yellow leaves turn brown. Where moisture is sufficient, the stunted plants remain alive, with the youngest leaves remaining green. Infected plants produce no tillers and ears and generally die if moisture is insufficient. The patches of stunted wheat tend to occur at the same location each year; being slightly larger in drier years and smaller in wet years. Roots of infected plants are brown. Small soil-covered Sclerotia-like bodies (nodules) are frequently observed at the root tips of infected plants. Previous research has shown that nutrient deficiencies or nutrient imbalances in the soil, poor soil structure, nematodes, termites, soil-borne aphids and soil toxins were not associated with PSW (Tinline, 1977; Tinline, 1982 pers comm.). Since 1987, studies into the causes of PSW have focused mainly on mycological agents. *Rhizoctonia solani* is nearly always isolated from infected roots and from the nodules. Other fungi such a *Bipolaris Sorokiniana, Fusarium oxysporum, Periconia spp. and Fusarium spp* have been isolated less consistently than *R. solani* from plants showing symptoms of PSW.

*Corresponding author

The objective of this study was to identify the etiological agent of PSW by isolating and growing the causal agent on culture media and to transmit the symptoms of the disease by inoculating healthy seedlings with Sclerotia from PSW infected roots and the mycelium of the isolated causal agent from culture media.

Materials and Methods

Sampling and isolation

Randomly collected plants from PSW affected areas were obtained in 1994 and 1995 by loosening the soil with a fork in order to collect plants with intact roots and adhering nodules. Five plants were collected from each of the following areas: the centre and the periphery of an affected (patchy) area and from healthy wheat growing in an adjacent area about 1 - 2m away from the periphery of the patchy area. Three randomly selected PSW affected areas were sampled from each of the seven farms at the HWS. Nodules were counted on wheat roots taken from the HWC farms 4 - 6 weeks after planting. Comparison between the number of nodules per plant from the centre, the periphery and the adjacent healthy - appearing area were done using Person correlation test. The plants were washed, dried and stored in paper bags before examination for a duration varying between one month and two years.

Nodule counts

Samples for nodule counts were taken from the farms 4 - 6 weeks after planting. Five plants which were collected from each of the centre and the periphery of the PSW areas and from adjacent healthy appearing plants were washed in running tap water. Nodules were counted on the roots of each of the five plants collected from three sites within the affected sites on each of the seven farms. Correlation between the number of nodules per plant at the centre, the periphery and the adjacent healthy - appearing area were done using Person correlation test. The study was conduced in 1994 and repeated in 1995.

Isolation of causal agent

Immediately prior to isolating the causal agent, roots and nodules were washed in running tap water for 5 minutes and rinsed five times in sterile water. Root segments and nodules were dipped momentarily in 80% ethanol followed by a dip in sodium hypochlorite (JIK Bleach, Reckitt Household Products) diluted 1:2 (v:v) in water. Roots dipped for five minutes and nodules for one minute. This was followed by five rinses in sterile distilled water after which the roots and nodules were plated on Potato Dextrose agar (PDA) in 9 cm diameter Petri dishes. The PDA was prepared from the broth of 200 g of freshly pealed, sliced, boiled potatoes, 20 g dextroses, 18 g BDH agar in one litre distilled water. Alternatively, the commercial PDA (Difco, Detroit) was used as prescribed. All cultures were incubated at room temperature in the dark for three days before being examined for fungus growth with a light or a dissecting microscope. Sections of hyphae that grew out from the roots and nodules were subcultured onto fresh plates of PDA to serve as cultures for pathological tests.

Infection studies

Three isolates of *R. solani* were used to inoculate wheat (*Triticum aestivum L.*) cultivator Viri in the screenhouse. The seeds were dipped momentarily in 80% ethanol and then placed in 30% sodium hypochlorite solution for one minute. Seeds were then rinsed twice in sterile distilled water. Inoculum was prepared from seven day old cultures of *R. solani* on PDA which was macerated in water in a Warring Blender for one minute at low speed and then for one minute at high speed. Segments of hyphae per ml of solution were counted in a haemocytometer and adjusted to 2 - 3 x 10^6 segments per ml of water. Seeds were dipped in the mycelia solution for 30 minutes prior to planting in noninfested soil in 15 cm diameter pots. The soil was watered to field capacity. The soil and wheat seeds were planted into the soil. Watering was as described above. Control seeds were not inoculated and were planted into noninfested soil and watered as described above. Records of plant growth were made eight weeks after planting.

Random samples of soil were collected to a depth of 15cm from PSW locations at the HWC and placed in 15 cm diameter pots in the screenhouse in order to determine the presence of the pathogen causing PSW. Non-infested Millisol soil was collected from hill tops in Northern Tanzania, about 300 km from where PSW occurs. The non-infested soil was also placed in 15cm diameter pots. Seeds of wheat cultivar *Viri* were planted in the pots. The experiment was conducted in a completely randomised design. After emergence, the plants were thinned to eight plants per pot. Data recorded included root length, shoot height and shoot weight in two months after planting. The study was conducted in 1994 and repeated in 1995.

Results

Rhizoctonia solani was consistently isolated from infected roots and nodules of plants from the PSW patches from all the seven farms of the HWC. Likewise, it was also isolated from nodules that had been stored at room temperature in Petri dishes for two years.

The nodules consisted of compact masses of mycelia. Tinline (1977 pers comm.) also isolated *Rhizoctonia spp.* from wheat plants at the HWC but did not consider it as the cause of PSW. Kuwite and others (1995) also isolated *Rhizoctonia spp.* from wheat from the HWC farms but the frequency of isolation was low when compared to the frequency in the present studies. The high frequency of isolation in these studies was thought to be due to the change in sampling methods (Piening 1992 pers comm.). The previous methods involved pulling plants from the soil without loosening or with minimal loosening of the soil with a fork and as a result, most of the diseased roots and nodules remained in the soil. Loosening the soil resulted in the collection of more intact roots with nodules which were not detected in earlier work.

The three isolates of *R. solani* collected for this study were pathogenic to the wheat cultivar *Viri* growing in pots. *Rhizoctonia solani* was also isolated from lesioned roots of plants growing from inoculated seeds and soil. The symptoms of wheat inoculated with *R. solani* or wheat growing in infested PSW soil in the screen house were similar to those of wheat growing in areas of PSW in the field and that *R. solani* was consistently isolated from root lesions and nodules suggest that *R. solani* is the causal age of PSW.

There were significantly more nodules from the roots of plants collected from the centre of PSW patches than from the periphery of from the adjacent healthy-looking plants in the 1994 test ($r=0.279$, $P=0.02$) and in the 1995 test ($r=0.41$, $P=0.01$) (Table 1). Shoot length and shoot weight were significantly reduced in PSW soil than compared to those in non-PSW soil (Table 2).

Discussion

Isolation of *R. solani* from the nodules of PSW infected plants and subsequent pathogenic positive tests with this pathogen suggest that this fungus is the causal agent of PSW in Tanzania. There were significant reduction in shoot height and weight of plants growing in PSW soil when compared to those growing in non-infested soil.

PSW of wheat in Tanzania appears to be similar to crater disease in South Africa which is also caused by *R. solani* (Smith and Wehner 1986, Deacon and Scott 1985, Scott et al., 1979). However, whereas crater disease occurs only on very heavy black soils of the Springbok flats in South Africa (Deacon and Scott, 1985), PSW occurs on a variety of soil types including Vertisols, Mollisols, Inceptisols and Alfisols. The South African isolates form bead-like swellings composed of hyphae of *R. solani* (Deacon and Scott, 1985). The Tanzanian isolates also form similar structures hereby known as nodule-like structures. The significant correlation that was observed between the number of nodules observed at the centre and periphery of PSW spots and at near-by healthy-appearing plants agrees with Deacon and Scott's (1985) strong association between craters and beads formed by *R. solani*. Since compact mycelia of *R. solani* was observed in these nodule-like structures, most likely these are important survival structures for this pathogen.

Rhizoctonia solani also has been implicated in stunting diseases of cereals in other countries. In Australia, *R. solani* causes bare patch

Table 1: Number of nodules on roots from plants from the centre, periphery and from healthy plants adjacent to patches of stunted wheat in 1994 and 1995

Sample location	Number of samples[a]	Total number of Nodules[b]	Average number of Nodules
Centre	126	216	1.71
Periphery	126	114	0.90
Healthy looking	126	47	0.37

[a]Includes 63 samples collected in 1994 and 63 samples collected in 1995;
[b]Total number of nodules counted in all sites at five plants per site (126 sites in two years).

Table 2: Effect of soil (infested and non-infested) on root length and shoot weight in 1994 and 1995a

Soil type	Root length (cm)	Shhot length (cm)	Shoot weight (g)
PSW-affected	11.18	26.73[a]	0.92[a]
Non-PSW affected	11.76	0.77[b]	1.43[b]
Probability	ns	*	*
cv (%)	16.26	8.26	20.0

[a]From combined analysis of 1994 and 1995 data;
*Significant at P = 0.05.

of wheat (Neate, 1987) and in the U.S.A., it causes bare patches of small grains. In the USA, *R. solani* has also been reported (Weller et al., 1986) to have caused stunting of wheat in irregular patches and formed lesions that often girdled the roots of wheat plants. These lesions were found on primary and secondary roots, on the subcrown interposed and on basal leaf sheath (Weller et al., 1986). The root symptoms caused by the American isolates of *R. solani* are different from those observed in Tanzania. PSW affected roots in Tanzania show general browning without distinct lesions. In Australia *R. solani* form Sclerotia in the root zone but nodule-like structures were not reported by Neate (1987).

Conclusions

Our findings indicate that the patchy stunting of wheat caused by *R. solani* in Tanzania is similar to crater disease in South Africa and bare patch of wheat in USA and Australia in above ground symptoms. However, it is different from the USA and Australian isolates in root symptoms.

Acknowledgement

The authors wish to acknowledge the technical assistance of Bashir R. Makoko and Eugenia Kweka in all aspects of this study. We deeply thank R. A. Mckeague, formerly the team leader of the Tanzania-Canada wheat programme for inspiration. We thank Dr. F. C. Wehner of the University of Pretoria, and Mr. F. Ngulu of SARI for reviewing the manuscript. Funding for the project was provided by the Canadian Agency for International Development and the Government of Tanzania and is gratefully acknowledged.

References

Deacon J. W. and D. B. Scott. 1985. *Rhizoctonia solani* associated with crater disease (stunting) of wheat in South Africa. Trans. Br. Mycol. Soc. 85 (2): 319 - 327.

Kuwite C. A., Piening L., and L. Braun. 1995. Patchy Stunting of Wheat in the Hanang Wheat Complex, Tanzania. A mycological Aspect. pp 447 - 451, In: Tanner D.G.; Osman A., and Payne T. (ed) 1996. Proceedings of the ninth Regional Wheat Workshop for Eastern, Central and Southern Africa. Addis Ababa, Ethiopia. CIMMYT. 536 pp.

Neate S. M. 1987. Plant debris in soil as a source of Inoculum of *Rhizoctonia* in wheat. Trans. Br. Mycol. Soc. 88(2): 157 - 162.

Scott, D. B., Visser, C. P. N. and Erna, M.C. Rufenact. 1979. Crater disease of summer wheat in Africa dry lands. Plant Dis. reptr 63: 836 - 840.

Smith Elize M. and F. C. Wehner. 1986. Induction of crater disease of wheat in the field by artificial infestation of the soil with *Rhizoctonia solani*. Phytophylactica 18: 127 - 130.

Weller, D. M., Cook, R. J., Macnish, G. Bassett, E. N., Powelson, R. L., and R. R. Petersen. 1986. *Rhizoctonia* root rot of small grains favoured by reduced tillage in the pacific north west. Plant Disease 70: 70 - 73.