Int. J. Tropical Plant Diseases 9 (1991): 35-52
Today & Tomorrow's Printers & Publishers, New Delhi-5; Printed in India

PEARL MILLET RUST - PRESENT STATUS AND FUTURE RESEARCH NEEDS

S.D. Singh and S.B. King
Cereals Program
International Crops Research Institute for the Semi-Arid Tropics (ICRISAT)
Patancheru, Andhra Pradesh 502324, India

ABSTRACT

The present status of rust *(Puccinia species)*, which is an important disease of pearl millet *(Pennisetum glaucum)*, is reviewed. The disease is reported from many pearl-millet growing countries, but information is lacking from many others. There is confusion regarding the identity and distribution of the *Puccinia* species and subspecies that cause this disease. All the spore stages of this polymorphic rust are known. Annual recurrence is through aeciospores produced on eggplant *(Solanum melongena)* and/or through urediniospores.

Many sources of resistance, including some that are controlled by single dominant genes are available, but limited emphasis has been placed on breeding rust-resistant cultivars. Chemical control of the disease is possible, but does not seem practical. Biological and cultural control methods appear to have little current application. Several areas for future research are suggested.

INTRODUCTION

Pearl millet, *Pennisetum glaucum* (R.) Br., is an important cereal of the semi-arid tropics, grown for food and feed on about 26m ha in the world. Rust is one of the important diseases of pearl millet. A number of species of *Puccinia and* varieties of *Puccinia substriata* have been reported as rust pathogens on pearl millet in different parts of the world. In India, rust occurs in all areas where pearl millet is cultivated. The disease has usually been considered a minor problem because of its late appearance, generally after the grain-filling stage, causing little or no loss in grain yield. However, it can occur as early as the seedling stage and in such cases substantial reduction in grain yield and fodder yield and quality can occur (Rama-

Submitted as Journal Article No. 1018 of the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Andhra Pradesh 502324, India.

krishnan, 1963; Monson et al., 1986; Singh, S.D., unpublished). The disease has become a serious problem in recent years in India (Muthusamy and Raghupathy, 1980). The reasons for this include factors like large scale cultivation of high-yielding but rust-susceptible cultivars, large scale seed production during the summer season, and overlapping cropping, particularly in Tamil Nadu and Gujarat, India. The objectives of this article are to review published work, identify research deficiencies, and propose research needs to generate information for the control of this disease, should it become a limiting factor to pearl millet production in future.

GEOGRAPHIC DISTRIBUTION

Rust is known to occur on pearl millet in many countries of Asia (India, Srilanka and Pakistan) and Africa (Chad, Congo, Ethiopia, Ghana, Guinea, Cote d'Ivoire, Kenya, Malawi, Mozambique, Nigeria, Senegal, Sierra Leone, South Africa, Sudan, Tanzania, Uganda, Zambia and Zimbabwe) (Commonwealth Mycological Institute, 1967); and Niger (Jouan and Delassus, 1971). It has also been reported in Georgia, USA (Luttrell, 1954; Wells *et al.*, 1973b).

ECONOMIC IMPORTANCE

There is not much information on yield losses caused by the rust. Sokhi *et al.* (1978) appear to be the first to report that a fungicide, Dithane M-45, when applied to prevent rust development, increased yield components (including number of tillers bearing panicles) over the untreated check. Singh and Sokhi (1983) reported that rust reduced the average number of panicles per plant, grain yield per plant, and 1000 grain weight in both slow-rusting and fast-rusting cultivars. However, the reductions were comparatively more in fast-rusting cultivars than in slow-rusting ones. In Gujarat state (India), rust appears to be a major yield reducer in the post-rainy season crop grown for seed production (H.R. Dave, personal communication). In USA, severe impairment in fodder quality and yield has been reported due to rust (Wells et al., 1973a, Monson et al., 1986). Rust infection appears to affect fodder quality by affecting the mineral content of infected leaves. Sivaprakasam *et al.* (1976) reported that rust infection increased the nitrogen and calcium content of pearl millet leaves, but decreased phosphorus and magnesium content, whereas the potassium content remained unaffected. In general substantial reduction in yield can occur if the disease attacks at the seedling stage (Rachie and Majumdar, 1980).

THE PATHOGEN

i. Taxonomy

ported by Zimmerman in 1904 on pearl millet (*Pennisetum typhoides* Staph and Hubb.) at Amani, East Africa. Barclay (1891) used the name *P. penniseti* Barclay, which, according to Butler and Bisby (1931), was without formal description and which he probably confused with *P. purpurea* Cooke on sorghum. The same rust was observed from several parts of India by Butler (1918) on pearl millet. Ramachar and Cummins (1965) considered *P. substriata* Ell. & Barth. var. *indica* var. nov., whose aecial stage occurs on *Solanum melongena* L. (Ramakrishnan and Soumini, 1948), to be the common rust on Pennisetum in India. They ascribed rust occurring in Africa is *P. substriate* Ell. & Barth var. *penicillarie* (Speg.) Stat. nov. but commonly referred to as *P. penniseti* Zimm. They further reported that var. *indica* differs from var. *penicillariae* in having small and very tardily dehiscent telia and narrower and usually paler teliospores. All but one Indian specimen that they had examined were similar; they had no specimens from elsewhere. They considered *P. substriata* to be a collective species on the order if *P. recondita* Rob. ex Desm. and *P. graminis* Pers. They recognized ffive morphological varieties of *P. substriata* whose aecial stages probably all occur on *Solanum* spp.

Sathe (1969) reported another species of rust, *Puccinia stenotaphri* Cummins, on pearl millet from Pune, India. The telia of this rust are foliicolous, blackish in color, depressed, spreading linearly, and evident on both surfaces of the leaves. *P. stenotaphri* differs from *P. substriata* var. *india* in having paraphysate uredinia and non-erumpent telia. Recently, *P. substriata* Ell. & Barth var. *decrospora* var. nov. has been reported on *Pennisetum americcnum* in Nigeria (Eboh, 1986). This taxon reportedly differs appreciably from all *P. substriata* varieties by its longer teliospores which have up to five cells. Clearly, there appears to be some variability and possibly some confusion about the taxonomy of the species of pearl millet rust.

ii Spore sacs and spore stages.

Butler (1918) suggested that pearl millet rust probably has dispensed with the aecial stage. However, Ramakrishnan and Soumini (1948) reported the alternate host as *Solanum melongena* (egg plant), and later *S. melongena* var. *insanum* Prain., *S. pubescence* Willd., *S. torvum* Sw. and *S. xanthocarpum* Schrad and Wendl., were also reported as alternate hosts (Ramakrishnan and Sundaram, 1956). *S. melongena* was reported as an alternate host of *P. substriata* var. *indica* in Georgia also (Wells, 1978), where it is believed to play an important role in the initiation of the disease each year. With the discovery of alternate hosts, this rust has all five spore stages.

a.1. Uredinia

Uredinia are formed subepidermally and they generally appear on the adaxial surface of the leaf, and sometimes on the stem. At maturity they rupture the epidermis resulting in the exposure of urediniospores. Urediniospores are borne on

hyaline pedicels which are up to 60 μm long (Ramakrishnan and Soumini, 1948). Pedicels have been reported to be shorter than urediniospores by Butler (1918), but to be longer by Ramakrishnan and Soumini (1948).

Paraphyses, mainly along the margin, have been seen in a number of sori. These are cylindrical or slightly inflated at the apex, flexuous, incurved, and stouter and longer than the pedicels. Sometimes they are faintly tinted brown. The paraphyses are not clearly visible in scrapings but can readily be recognised in sections; their number varies among sori (Ramakrishnan and Soumini, 1948). Total absence of paraphyses was reported by Butler (1918), and Ramachar and Cummins (1965) supported this view when they gave the name *P.substriata* var.*indica* as*P.substriata* is characterized as having no paraphyses.

a.2. Urediniospores

Urediniospores are generally oval, pyriform or elliptic with four equatorial germpores and measure 35 x 25 μm (25-42 x 21-30 μm). The spores have yellowish brown walls and sparse echinulations which are abundant near the apex (Ramakrishnan and Soumini, 1948).

a.3. Development

The urediniospore germtube penetrates pearl millet leaves through stomata and produces hyphal cells in the sub-stomatal cavity. These cells, which are generally cylindrical and regularly binucleate, form the uredinial initial. After repeated division, these cells produce compact tissue of binucleate cells and give rise to uredinial basal cells or primordia. The primordial cells elongate vertically producing the spore-initial, by the formation of two unequal binucleate cells through the process of conjugate division of the original nuclear pair. The upper cell becomes the urediniospore and lower cell the stalk-cell. This process is repeated several times, ultimately giving rise to full-fledged pedicellate urediniospores comprising a fully developed uredinium (Kulkarni, 1958).

b.1. Telia

The telia are amphigenous, blackish-brown, often in groups, and are generally on the abaxial leaf surface and also sometimes on stems. They are formed subepidermally and remain covered by the epidermis for a long time before bursting. In many cases, the sori are divided into two or more compartments separated by a wall of sterile tissues. These partitions are cellular and do not resemble paraphyses (Ramakrishnan and Soumini, 1948). A technique for the production and purification of telia has been described (Kapooria, 1970).

b.2. Teliospores

The teliospores are dark brown, generally 2-celled, cylindrical to club-shaped, broad in the upper portion, and tapering towards the base with the lower cell larger than the upper one. The apex is flattened, round or blunt, thickened up to 10 μm and often darker in color than the rest of the wall. The spores measure 49 x 21 μm (33-59 x 13-30 μm). The pedicel is colored, and generally short, 9 μm (4-19 μm) (Ramakrishnan and Soumini, 1948; Ramakrishnan, 1963). The presence of colorless pedicels was, however, reported by Butler (1918). Considerable variability in teliospores has been reported by Kapooria (1973a). He found that one of the pure line isolates produced 1- to 5-celled teliospores and 1- to 3-celled teliospores were produced by all the isolates he tested. The spores also varied in shape, size and thickness of wall.

b.3. Development

The telia develop either in old uredinia or independently as new structures from the binucleate basal cells through a process similar to that for uredinia. The binucleate basal cells undergo division and cut off vertical rows consisting of three cells which are regularly binucleate. The upper two cells form the 2-celled teliospores and the lower one develops into a stalk. The teliospores are binucleate in the initial stages; the conjugate nuclei, however, undergo a change and ultimately fuse in mature teliospores to form diploid nuclei (Kulkarni, 1958).

c.1. Spermogonia and spermatia

Spermogonia are genrally formed on the adaxial surface of leaf blades and sometimes on petioles of eggplant, the alternate host. They appear as minute, orange-yellow dots with glistening drops of nectar on the leaf surface. Spermogoinia are sub-epidermal, measure 100 x 140 μm and have a deep orange color. Paraphyses are present which may be straight and colored. Spermatia are oblong or elliptic, hyaline and measure 4-8 x 2-3 μm (Ramakrishnan and Soumini, 1948). They each contain a single haploid nucleus and are produced on simple, basal cells lining the inner wall (Kulkarni, 1958).

c.2. Development

The basidiospore germtube penetrates the eggplant leaf directly and gives rise to primary multicellular regularly uninucleate hyphae (gametophytic mycelium). The hyphae grow between the palisade cells of the host and force their way between the epidermal and palisade layers. These subepidermal hyphae branch rapidly and form a continuous mat of mycelium with a layer of polygonal cells at the top.

The growing mycelium tends to converge from all sides towards a central point where the spermogonal initial develops. The mycelium now breaks up and forms pseudoparenchymatous tissues made up of polygonal cells and surrounds the entire host cavity, where ultimately the spermagonium is located. The epidermis covering the stat is cut off from its natural food supply and breaks giving rise to an spermogonium ostiole require 5-6 days to develop and as they attain maturity paraphyses appear as long, straight bristles at the mouth of the ostiole (Kulkarni, 1956). In spite of the simultaneous occurrence of the oozing of the spermogonial nectar and the presence of flexuous hyphae at the mouth of the spermatium, copulation between spermatia and flexuous hyphae has not been observed (Kulkarni, 1956).

d.1. Aecia and aeciospores

Aecia are usually formed on the abaxial surface of eggplant leaf blades and are arranged in irregular, concentric, expanding circles. Sometimes they are also formed on leaf petioles and rarely on the adaxial surface of leaf blades (Ramakrishnan and Soumini, 1948; Wangikar and Raut, 1972). Each aecium projects beyond the leaf surface by about 1 mm, has a salmon orange color, and is columnar. The peridium, consisting of hyaline, polygonal cells, is prominantly verrucose on the inner surface. Aeciospores are catinulate, globose to angular, yellowish orange, smooth, and measure about 21 x 18 μm (16-25 x 12-21 μm) (Ramakrishnan and Soumini, 1948).

d.2. Development

In the middle layers of infected eggplant leaf blades, wefts of hyphae appear. The initiation of the dicaryon occurs here by the process of fusion of adjacent pairs of hyphal cells. The resulting diploid hyphal cells together with a mass of pseudoparenchymatous cells form the aecial primordium which is made up of binucleate basal cells and pseudoparenchymatous cells. The basal cells produce chains of aeciospores, while the pseudoparenchymatous cells produce the peridium. After peridial formation, the basal cells divide into unequal cells, the larger forming aeciospores, and the smaller becomes intercalary cells. The repeated division of the basal cells and nuclei ultimately gives rise to chains of aeciospores interspersed with interclary cells (Kulkarni, 1958).

iii. Germination and viability of spores

a.1. Urediniospores

Uredinisopores germinate readily by producing one or more germtubes (Ramakrishnan and Soumini, 1948). Germtubes are stout, flexuous, sometimes branched and often enlarged at the tip. Kulkarni (1958) obtained urediniospore germination

in 3-4 h in hanging drops of sterile water. Germtubes originated through germpores and branching occurred with high humidity. Misra and Prasad (1971) reported observing up to four germtubes arising from a single urediniospore, although one germtube was most common.

Urediniospore germination occurred at temperatures ranging from 6°-30°C, being 100% at 20°C. No germination occurred at 35°C or at 5°C (Misra and Prasada, 1971). Uredinisopores germinated at pH 3-9, though optimum was pH 7 (Misra and Prasada, 1971). Dalela (1961) studied the effect of pH and found that 22.12% and 11.79% of the urediniospores did not germinate at pH 4.6 and 9.2 respectively. Both high and low pH appear to be inhibitory to urediniospore germination.

Viability of urediniospores over time was studied by several workers. Ramakrishnan and Soumini (1948) obtained from 90 to 100% germination soon after collection but spores kept on air-dried specimens at laboratory temperature (28-30°C) completely lost viability in 30 days. There was a quicker loss of viability when the specimens were stored at high humidity (75-100%) or very low humidity (below 5%). Basu-Chaudhury (1955) reported that germination was lost in just 12 h at 49-50°C and in 8 days. at 41-42°C. Misra and Prasada (1971) reported viability of urediniospores for 23 weeks at 5-8°C, while at 0° and 25°C viability decreased faster. Kulkarni (1958) reported that urediniospores remained viable under laboratory conditions (25-27°C) on dry leaves for 30 days followed by rapid deterioration in viability. It appears that urediniospores may best retain viability at relatively low temperature (5-10°C). Effects of sub-zero temperatures on urediniospore viability have not been reported.

a.2. Teliospores

Germination of freshly formed teliospores has been under dispute. Ramakrishnan and Soumini (1948) obtained only 5-10% germination of freshly formed teliospores after 72-100 h in drops of sugar solution. This was disputed in the same year by Prasada (1948) who obtained germination of teliospores without any rest period. A rest period of 3-5 months was found necessary for germination by Kulkarni (1958). However, Misra and Prasada (1972) obtained 100% germination of freshly formed teliospores at 20-22°C after 48 h of wetting and drying of infected leaves.

Misra and Prasada (1972) reported the formation of typical 4-celled basidia. However, Kulkarni (1953) reported the occurrence of only 2-celled basidia with quadrinucleate basidiospores. Basidia may develop from one or both cells. Sterigmata are formed from the upper end of each cell on which eliptical or round basidiospores are produced.

Kulkarni (1958) reported that teliospores in dry material can remain viable for 6-7 months at laboratory temperature (25-27°C), whereas Prasada (1948) reported teliospore viability for 6 months when kept dry at 5-8°C.

a.3. Aeciospores

Aeciospores germinated in 3-4 h in hanging drop cultures of sterile water. Germtubes were simple or branched, vacuolated in the lower portion, and densely granular at the tip. The aeciospores retain viability for 20-30 days in dry leaves stored at laboratory temperature 25-27°C) (Kulkarni, 1958).

HOST RANGE

Alternative or collateral hosts are important mainly as epidemiological bridges for the maintenance of urediniospore inoculum between crops of the main host, pearl millet. Several reports on the alternative hosts of pearl millet are available. These include reports of *P. penniseti* on *Pennisetum leonis, P. spicatum,* and hybrids of *P. typhoides* and *P. purpuream* (Ramakrishnan and Sundaram, 1956; Ramakrishnan, 1963), on *P. polystachyon* and *P. orientale* (Ramakrishnan and Sundaram, 1956; Dalela and Sinha, 1960), on *P. nigritarium* and *P. pycnostachyum,* (Kapooria, 1973b), and on *Panicum antidotale* and *Panicum maximum* (Sokhi, et al., 1984). More recently, *P. violaceum* was identified as an alternative host (Sokhi and Jhooty, Personal Communication). These hosts and the reported alternate hosts (*Solanum melongena* and other *Solanum* spp.) could be quite important epidemiologically posing a potential threat to pearl millet production.

CONTROL METHODS

i. Host-Plant Resistance

a.1. Screening procedures

Availability of field/laboratory screening techniques is a pre-requisite for the identification of resistance and its transfer into good agronomic backgrounds. Resistance sources identified so far have been based on natural field inoculations. At ICRISAT Center we compared several field inoculation procedures: (i) urediniospores supplied by earlier-planted infector rows, (ii) spraying urediniospores 25 and 40 days after planting, and (iii) spreading of uredinia-bearing leaves among test plants 25-30 days after planting. Among the methods, spraying of urediniospores twice, 25 and 35 days after planting, gave the best results. This, however, needs confirmation (S.D. Singh, unpublished).

The availability of urediniospores for use in screening is the key requirement. Uredeniospores can be made available by preservation or through rapid multiplication *in vivo.* Viability of urediniospores has been preserved for several months at ultra-low temperature of -75 to -86°C (H. D. Wells, Personal Communication). A detailed leaf culture technique has been developed (Sokhi, 1983). In this techni-

que, sporulating uredinia were maintained for 24 days on detached leaves that were maintained in water alone, or on a benzimidazole solution (40 ppm), or on a benzimidazole (40 ppm) and glucose (500 or 1000 ppm) solution. Fully developed uredinia were observed 10 days after inoculation and eight urediniospore crops were harvested at 3-day intervals thereafter. Top leaves of 40-day-old plants supported sporulation for 24 days whereas leaves from 32-, 25-, and 18-day-old plants sporulated for 15, 10, and 2 days, respectively. The study concluded that the flag leaf is most suitable for detached leaf culture.

a.2. Identification of resistance

Although rust was first reported on pearl millet in 1904, there has been little effort to breed for resistance. Suresh (1969) was the first to report high levels of rust resistance in open pollinated cultivars. Of the 34 cultivars tested, he reported PT826/4 and PT829/5 as resistant; PT829/3, PT833/2, PT833/4, PT835/6 and PT829/8 as tolerant; and others as susceptible. Ramakrishnan and Sundaram (1956) evaluated, during 1951-1954, many pearl millet cultivars and reported PT 81413 as highly resistant and PT 829 and MS 6897 as moderately resistant. *P. purpureum* was reported as immune. Their methods of inoculation and disease evaluation were not reported. All the screening was probably done under natural rust pressure. Recently, Govindrajan *et al.* (1984) reported an F_1 hybrid, IBH 1203 (PT 732 x PT 3832) as resistant to rust. The resistance in this hybrid came from the male parent.

A large number of germplasm accessions, particularly those originating from Nigeria, were evaluated and many resistant lines identified at ICRISAT Center (Singh and Williams, 1978). The stability of rust reactions of many accessions was evaluated with the help of cooperators in India and now many lines with a high degree of stable resistance to rust are available (Singh et al., 1983). Many germplasm accessions show combined resistance to downy mildew and rust (S.D. Singh, unpublished). There is also a report on the availability of slow rusting genotypes in pearl millet (Singh and Sokhi, 1983). This report needs confirmation.

Recently, ICML 11, a selection from 7042 (IP 2696), a land race from Chad, has been identified as rust resistant (Singh et al., 1987). Most of the rust-resistant material identified at ICRISAT Center have originated in W. Africa. A wild relative of pearl millet, *P. americanum* (L.) Leeke subsp. *monodii*, that showed immunity to rust, came from Senegal, W. Africa (Hanna *et al.*, 1982).

a.3. Utilization of resistance

canum (L.) Leeke. subsp. *monodii* to Tift $85D_2$ B_1, the maintainer line of Tift 85 D_2 A_1 (Hanna et al., 1987). Later, a rust-resistant hybrid, 'Tifleaf 2', was produced using this male-sterile line (Hanna et al., 1988). Efforts are underway to determine how resistance is controlled in other resistant genotypes of pearl millet and whether there are several single genes that can control rust. If major single gene resistance exists in other genotypes, and these genes differ from the one present in ICML 11, then an effort will be made to incorporate several dominant genes for resistance into a single genotype which might provide resistance to many populations of this rust and would possibly be long lasting. When two or more dominant genes for rust resistance are incorporated into a single pearl millet line, it would be desirable to determine a marker trait associated with each gene. The marker traits, if identified, could help ensure the transfer of the specific genes in the receiving plants.

a.4. Physiological specialization

Basu-Choudhury and Sinha (1955) appear to be the first to report on physiological specialization in *P. penniseti.* Their conclusions were based on the slight differences they observed in the infection reactions shown by 20 pearl millet varieties inoculated with rust samples collected from several parts of India. Their conclusions however, were not convincing, as all the varieties they used were susceptible. Better evidence of the occurrence of races was later provided by Ramakrishnan and Sundaram (1956). They found the presence of two types of uredinia: (i) uredinia isolated and scattered, and (ii) uredinia in groups on dark brown lesions. Inoculation with urediniospores from either type of uredinia consistently produced the same type of uredinia. All the cultivated varieties produce both types of uredinia. However, a *P. typhoides x P. purpureum* hybrid could be infected only with urediniospores from uredinia produced in groups on dark brown lesions. Kapooria (1978) also provided some evidence for the presence of physiological variability in this rust.

Much of the evidence supporting the concept of physiologic specialization in pearl millet rust pathogen is not very convincing, although it seems reasonable to expect that physiological races could exist in pearl millet rust due to its heteroecious nature (Kulkarni, 1956, 1958). If the conclusions of Dalela and Sinha (1962, 1964), that *P. penniseti* is homothallic and that hybridization does not occur are in fact valid, the potential for pathogenic variability in this pathogen may be greatly reduced. However, it is still advisable that researchers, extension workers, and producers be alert to the possibility of new races of the pathogen developing, should the time come when rust-resistant pearl millet cultivars are grown extensively by farmers. This is possible because other means of creating pathogenic variability in rust pathogens, have been reported (Christenson, 1961).

a.5. Inheritance of resistance

Resistance is dominant over susceptibility (Hanna et al., 1982, 1985; Andrews et al., 1985 and Sokhi et al., 1987). The resistance in these cases was demonstrated to be controlled by single dominant genes, except in one entry, 700481-23-2, in which complementry gene action was reported (Sokhi et al., 1987). It is not known whether these genes are the same or different. The gene identified by Andrews et al. (1985) was designated as Rppl.

ii. Chemical Control

Attempts have been made to control pearl millet rust by chemicals. Though a wide range of chemicals including antibiotics, have been tried, only one systemic fungicide namely Plantvax, has been tested. Both *in vivo* and *in vitro* tests have been used to test the effectiveness of chemicals.

In vitro tests: These tests have investigated largely the effect of chemicals on urediniospore germination. Kapooria (1972) evaluated 21 chemicals representing fungicides, antibiotics, plant growth regulators, and chelating agents. Dithane M-22, Ziram, O-phenanthroline, and Ceresan effectively reduced urediniospore germination at 10, 100, and 1000 ppm, whereas Dithane S31 was effective at 100 and 1000 ppm, and Cupramar and 1BA at 1000 ppm only. Bahadur et al. (1975) obtained complete inhibition of urediniospore germination in 100 ppm of 2-methyl benzoic acid anilide (75% WP), 2-iodine benzoic acid anilide (50% WP), and 2, 5-dimethyl Furan-3-carboxylic acid (50% WP). Parambaramani et al. (1971) reported that Plantvax and Vitavax in 1, 10, 100, and 1000 ppm, gave complete inhibition to urediniospore germination compared to 96% germination in the check.

In vivo test: Sinha and Dalela (1963) reported a decrease in rust intensity with spray application of 2, 4-D (10 ppm) to pearl millet plants at 3-4 h or 24 h after inoculation, and Sulfadiazine (100 ppm) and Streptomycin (1000 ppm) were effective when sprayed 3-4 h or 24 h after inoculation, or 24 h before inoculation. Soil application of these chemicals were ineffective, probably due to poor uptake.

Kapooria (1972) tested 21 chemicals by applying them 24 h before inoculation, at inoculation, and 24 h after inoculation on seedlings at the 3-4 leaf stage. Dithane M-22, Dithane S31, Cupramar, and Phenanthroline were most effective when applied 24 h before and at the time of incoulation. Post inoculation sprays were generally ineffective. Conversely, he reported that post inoculation spray with Ziram promoted disease incidence. Bahadur et al. (1975) reported that 2-methyl benzoic acid anilide (75% WP), 2-iodine benzoic acid anilide (50% WP), and 2,5-dimethyl furan-3-carboxylic acid (50% WP) at 100 ppm controlled rust to <5% levels when applied as pre-, post- or simultaneous with inoculation. Muthusamy and Raghupathy (1980) reported that spraying of copper oxychloride at 600g/acre, 30 and 55 days after

planting , spraying with bayleton 75g/acre, 30 days after planting, and pre-soaking of seeds in bayleton (2g/kg seed), were all effective in controlling ruse. Muthusamy and Raghupathy (1982) also obtained good rust control by spraying wettable sulphur at 800g/acre 30 and 55 days after planting.

To control plant pathogens, chemicals are commonly used because their application is generally easy and they are effective. However, use of chemicals to control rust on pearl millet is questionable because there may not be an economic return from the treatment, and application may not be possible considering the areas of the crop and people who grow it. Although research on the use of chemicals, especially systemic fungicides, to control rust of pearl millet could produce useful results for some situations, it seems doubtful that this area of research would result in recommendations that would be useful, given the present method and scale of pearl millet production.

iii. Cultural Control

Various cultural pratices are known to reduce the severity of diseases. Since pearl millet rust has so far been considered a minor disease, it received very little attention for its control by cultural methods. There are reports on the effect of fertilizers on rust incidence. Kandasamy et al. (1971) evaluated six rates of N_2; 0, 40, 80, 120, 160 and 200 kg/ha in soil with basal application of K_2O (murate of potash), 50 kg/ha and P_2O_5 (single super phosphate), 50 kg/ha, on six pearl millet cultivars, HB-1, HB-3, HB-4, J934-5, K-559 and X3. Rust severity was increased with the increase in N_2 levels from 0-40 kg/ha. Further increase in N_2 levels had no significant effect on rust severity and the cultivars responded alike. When we carefully examined the data of this experiment, we found that although there was a significant increase in rust severity with the increase in N_2 levels from 0. 40 kg/ha, the actual increase in rust was only 4%, from 40.8 to 44.8% severity. This difference is not sufficient to discontinue the use of N_2 in order to offset the disadvantageous effect of rust. We, therefore, fully agree with Sivaprakasam and Pillayarsamy (1975) who concluded that N_2 application is not practical to control rust in pearl millet.

Adjustment in planting date. which in effect, allows the susceptible stage of crop to escape the congenial environment for the development of the disease, has been reported to reduce some diseases including ergot and downy mildew of pearl millet (Nene and Singh, 1976).

A study was conducted to determine the effect of relative humidity on the development of rust. In a 2-year study, Muthusamy *et al.* (1981) concluded that pearl millet crop planted at fortnightly intervals beginning 22 March to 22 June developed only 0-7% rust intensity, while crop planted during other months of the year developed 15-74% rust. The differences in rust severity were attributed to the differences in relative humidity which was less during March to June com-

pared to the other months. This finding, though important, does not have much practical use since much of pearl millet is planted during July to January.

iv. Biological Control

Use of microorganisms naturally occurring on plant surfaces which may directly or indirectly affect spore germination and subsequent processes leading to disease development is being widely researched in several patho-systems. Though excellent success has been achieved in controlling certain insect pests by biological agents, its impact on disease control has been so far discouraging.

Rust fungi, in general, are infected with several fungi including *Darluca filum*, *Tuberculina vinosa* and *Verticillium lecanii* (Kranz, 1981). *D. filum* and *Tuberculina* species have been reported on *P. penniseti* from India (Sundaram, 1962). In an extensive study Kapooria and Sinha (1969) mixed urediniospores of *P. penniseti* with the conidia of *Fusarium, Helminthosporium, Penicillium, Chaetomium, Choanephora, Curvularia, Alternaria, Aspergillus, Rhizopus, Talaromyces* and *Trichoderma.* Urediniospore germination was reduced from 89% to 48%. Urediniospore germination was also reduced when mixed with pollen grains of pearl millet. Spores of *C. globosum, F. oxysporium, T. koningii, A. japonicus* and *A. niger* when mixed with the urediniospores of *P. penniseti* reduced the mean number of uredinial pustules per plant from 13.2% (in controls) to 1.6%, 4.4%, 4.7%, 5.8% and 7.0% respectively (Kapooria and Sinha, 1969).

Very little information available on this aspect clearly shows that biological control of rust is not an important research area. This appears to be correct keeping in view the economic importance of the disease and the status of the crop.

CONCLUSIONS

It is evident that certain aspects such as life cycle and cytology of the pathogen are well studied. However, research on several basic and applied aspects (epidemiology, taxonomy, screening technique, yield-reducing potential, chemical control, and host-plant-resistance, etc. is inadequate. This lack of research may be due to (i) pearl millet is not considered as important a food crop as are several other creals, (ii) it is usually grown by poor farmers who live at the subsistence level, (iii) rust has not been recognized as an important disease of this crop. We propose that the following areas should receive attention.

RESEARCH NEEDS

i. Assessment of losses

Precise experiments should be conducted to determine how much yield loss (grain, stover) the disease can cause, and under what conditions.

ii. Susceptibility to rust with plant age

Pearl millet is susceptible to rust infection at all stages of growth (Ramakrishnan, 1963; Singh, unpublished), though genotypic differences may exist. However, this fact has not yet received general acceptability. This in turn will help to remove the misconception about its yield-reducing potential.

iii. Retention of viability of urediniospores

Several reports on this aspect are available but these results require confirmation and precise methodology needs to be determined. A reliable method for retention of viability of urediniospores for at least 1 year would be especially useful for research involving artificial field and laboratory inoculation and screening.

iv. Infection process

In order to develop artificial screening procedures, and to study resistance mechanisms, information on pathogen germination, penetration, and establishment together with information on the optimum and essential environmental requirements (temperature, dew period, and humidity) are needed.

v. Screening procedure

All source of rust resistance so far reported have been identified under natural rust inoculation. As natural disease pressure is generally unpredictable of the time of appearance and severity, the data collected from such screenings are often suspect. Reliable laboratory and field-screeing techniques are required that provide adequate rust disease pressure at the correct stage of host susceptibility.

vi. Rating scale

Cobb's modified scale developed for the assessment of wheat rusts (Melchers and Parker, 1922) has been used. However, this scale may not be the most suitable for assessing rust severity on a crop such as pearl millet. Research attention should be given towards improving the scale for its application to pearl millet rust, by taking into consideration aspects such as size of pustules and spore production.

vii. Taxonomy

It is not yet precisely known whether the species and subspecies occurring in

India, Africa, and the USA are the same or different. The extent of variation in morphological and other criteria used for taxonomic determinations should be identified and their geographical distribution determined.

viii. Physiological specialization

Aspected of an obligate parasite, physiologic races in pearl millet rust are reported. Research should be conducted to further substantiate the existence of races and a set of differentials should be identified.

ix. Identification and utilization of resistance

Several lines with high degree of stable resistance have been identified. A continued search has to be made to identify new rust resistant sources, particularly those originating from diverse geographic regions. Rust-resistant sources identified in India, particularly at ICRISAT Center, are effective in India. Rust resistance has also been identified at Tifton, Georgia, USA, in a wild relative of pearl millet and this resistance has been transferred to pearl millet (Hanna et al., 1982). Unfortunately, it is not known for sure whether resistance identified in India is effective in USA and *vice versa,* or whether the resistant sources identified at the two places are effective in Africa. This obviously calls for an International Rust Resistance Testing Program involving India, USA and the African countries in order to identify resistances stable at all these places. Such testing could also throw some light on the qualitative variations in the pathogen populations. The identity and use of slow rusting needs exploration. It is important that once sources of stable resistance are identified, they are used in breeding programs.

x. Genetic studies

Studies conducted at Georgia (Hanna *et al.*, 1982, 1985) and at ICRISAT Center (Andrews *et al.*, 1985) show that pearl millet rust is controlled by single dominant genes. Whether these two genes are the same or different needs to be determined. It is likely that other genes for resistance can be found in accessions of germplasm.

ACKNOWLEDGEMENT

We thank Dr. K.D.M. Luther and Mr. S.S. Navi for their help in literature collection.

REFERENCES

Andrews, D.J., Rai, K.N. and Singh, S.D. 1985. A single dominant gene for rust resistance in pearl millet. *Crop. Sci. 25* : 565-566.

Barclay, A. 1891. Additional Uredineae from neighbourhood of Simla. *J. Asiat. Soc. Bengal. 60* (Part 4): 1-22

Bahadur, P., Singh, Seodhan, Sinha, V.C., Goel, L.B. and Sharma, S.K. 1975. Note on the laboratory evaluation of some systemic fungicides against cereal rusts. *Indian J. Agric. Sci. 45* : 274-275.

Basu-Chaudhury, K.C. 1955. Effect of temperature on the survival of the rust caused by *P. penniseti* Zimm. *Agra Univ. J. Res. Sci. 4* : 31-33.

Basu-Chaudhury, K.C. and Sinha, S. 1955. Physiologic specialization in *Puccinia penniseti* Zimm. on *Pennisetum typhoides* Stapf. & Hubb. *Agra Univ. J. Res. Sci. 4* : 575-578.

Butler, E.J. 1918. Fungi and Disease in Plants. Thacker Spink & Co., Calcutta, pp. 547.

Butler, E.J. and Bisby, G.R. 1931. Fungi of India. *Sci. Monogr. Coun. Agr. Res. India 1* : pp.237.

Christensen, J.J. 1961. Somatic variations in rust fungi. *Recent Adv. Bot. 5* : 571-574.

Commonwealth Mycological Institute. 1967. Distribution Maps for Plant Diseases No. 225, (Ed. 2, 1967), n.p., 1948-53.

Dalela, G. G. and Sinha S, 1960. Grasses as collaberal hosts I. Susceplibility of certain grasses to *Puccinia* pernninsiti Zimm. Proc. 47th Ind. Sc. Cong.: Part III

Dalela, G.G. 1961. Influence of pH on germination of uredospores and accidiospores of *Puccinia penniseti* Zimm. *Sci. and Cult. 27* : 196.

Dalela, G.G. and Sinha, S. 1962. Homothallism in *Puccinia penniseti* Zimm., a long cyclic rust and the function of pycnia. *Indian Phytopath. 15* : 156-161.

Dalela, G.G. and Sinha, S. 1964. Experiments on physiologic specialization in *Puccinia penniseti* Zimm. *Indian Phytopath. 17*(1) : 63-65.

Eboh, Don. O. 1986. A new variety of *Puccinia substriata* from Nigeria. *Trans. Br. mycol. Soc. 87* : 476-478.

Govindarajan, K., Nagarajan, C., Raveendran, T.S., Prasad, M.M. and Shunmugum, N. 1984. A new source of resistance to pearl millet rust disease. *Madras Agric. J. 71*(3) : 210.

Hanna, W.W., Wells, H.D. and Burton, G.W. 1982. Transfer of disease immunity from a wild subspecies to pearl millet. Agron. Abst., 1982, pp. 69.

Hanna, W.W., Wells, H.D. and Burton, G.W. 1985. Dominant gene for rust resistance in pearl millet. *J. Hered. 76* : 134.

Hanna, W.W., Wells, H.D. and Burton, G.W. 1987. Registration of pearl millet inbred parental lines, Tift 85 D2A1 and Tift 85 D2B1. *Corp. Sci. 27* : 1324-1325.

Hanna, W.W., Wells, H.D., Burton, G.W., Hill, G.M. and Monson, W. G. 1988. Registration of "Tifleaf 2" pearl millet. *Crop. Sci. 28* : 1023.

Jouan, B. and Delassus, M. 1971. Principales malades des mils et sorghos observees au Niger. *Agron. Trop. 26*(8) : 830-859.

Kandasamy, D., Sivaprakasam, K. and Krishnamurthy, C.S. 1971. An observation on the influence of different levels of nitrogen on the incidence of rust disease of pearl millet (*Pennisetum typhoides* Stapf. & Hubb.). *Plant and Soil.34* : 757-760.

Kapooria, R.G. and Sinha, S. 1969. Phylloplane mycoflora of pearl millet and its influence on the development of *Puccinia penniseti. Trans. Br. mycol. Soc. 53* : 153-155.

Kapooria, R.G. 1970. On the purification technique and production of telia in certain isolates of *Puccinia penniseti. Plant Dis. Reptr. 54* : 646-647.

Kapooria, R.G. 1972. On the chemical control of *Puccinia penniseti. Ghana J. Sci. 12* : 67-68.

Kapooria, R.G. 1973a. Morphological variability in teliospores of *Puccinia penniseti . J. Gen. Microbiol. 77* : 443-446.

Kapooria, R.G. 1973b. Studies on the host spectrum of *Puccinia penniseti. Ghana J. Sci. 13* : 153-154.

Kapooria, R.G. 1978. Studies on physiological variability in *Puccinia penniseti. Indian Phytopath. 31* : 372-374.

Kranz, J. 1981. Hyperparasitism of biotrophic fungi. *In* Microbial Ecology of the Phylloplane (J.P. Blakeman, Ed.). Academic Press, London. pp. 327-352.

Kulkarni, U.K. 1953. On the production of 2-celled basidia in *Puccinia penniseti* Zimm. *Sci. and Cult. 19* : 39-40.

Kulkarni, U.K. 1956. Initiation of dicaryon in *Puccinia penniseti* Zimm. *Trans. Br. mycol. Soc. 39* : 48-50.

Kulkarni, U.K. 1958. Studies on the development and cytology of *Puccinia penniseti* Zimm. *Trans. Br. mycol. Soc. 41* : 65-67.

Luttrell, E.S. 1954. Diseases of pearl millet in Georgia. *Plant Dis. Reptr. 38* : 507-514.

Melchers, L.E. and Parker, J.H. 1922. Rust resistance in winter wheat varieties. *U.S. Dept. Agril. Bull. 1046* : pp. 32.

Misra, A. and Prasada, R. 1971. Studies on uredospore germination and incubation period of bajra rust caused by *Puccinia penniseti. Indian J. Mycol. and Pl. Pathol. 1* : 103-107.

Misra, A. and Prasada, R. 1972. Mode of germination of teliospores of *Puccinia penniseti. Indian J. Mycol. and Plant Pathol. 2* : 174.

Monson, W.G., Hanna, W.W. and Gainers, T.P. 1986. Effect of rust on yield and quality of pearl millet forage. *Crop. Sci. 26* : 637-639.

Muthusamy, S. and Raghupathy, N. 1980. Efficacy of fungicides in control of rust disease of pearl millet. *Madras Agric. J. 67* : 838-840.

Muthusamy, S., Padmanaban, D., Nagarajan, R. and Raghupathy, N. 1981. Incidence of pearl millet rust in relation to sowing time and relative humidity. *Madras Agric. J. 68* : 527-529.

Muthusamy, S. and Raghupathy, N. 1982. Control of pearl millet rust with fungicides. *Pesticides 16* : 35-36.

Nene, Y.L. and Singh, S.D. 1976. Downy mildew and ergot of pearl millet. *PANS. 22* : 366-385.

Parambaramani, C., Vidhyasekaran, P. and Kandaswamy, T.K. 1971. Preliminary studies on the use of new oxathiin fungicides for the control of some rusts. *Madras Agric. J. 58*(8) : 705-706.

Prasada, R. 1948. Studies on the formation and germination teliospores of rusts. 1. *Indian Phytopath 1* : 119-126.

Rachie, K.O. and Majumdar, J.V. 1980. Pearl millet. Pennsylvania State University Press, University Park and London. pp. 307.

Ramachar, P. and Cummins, G.B. 1965. The species of *Puccinia* on the Paniceae. *Mycopath. et., Mycol. Appl. 25* : 7-60.

Ramakrishnan, T.S. 1963. Diseases of Millets, Indian Council of Agricultural Research, New Delhi, pp. 156.

Ramakrishnan, T.S. and Soumini, C.K. 1948. Studies on cereal rusts. 1. *Puccinia penniseti* Zimm. and its alternate host. *Indian Phytopath 1* : 97-103.

Ramakrishnan, T.S. and Sundaram, N.V. 1956. Further studies on *Puccinia penniseti* Zimm. *Proc. Indian Acad. Sci. Sect. 3*(B) : 190-196.

Sathe, A.V. 1969. A new rust record on *Pennisetum typhoides. Mycologia 61* : 198-200.

Singh, B.B. and Sokhi, S.S. 1983. Effect of rust of millet on yield components. *Indian Phytopath. 36* : 89-91.

Singh, S.D. and Williams, R.J. 1978. Review of pearl millet rust resistance screening. Mimeo, ICRISAT, India, pp. 23.

Singh, S.D. Reddy, P.M. and King, S.B. 1983. Report of the International Pearl Millet Rust Nursery (IPMRN). Progress Report : MP 106, ICRISAT, pp. 14.

Singh, S.D., Andrews, D.J. and Rai, K.N. 1987. Registration of ICML 11 rust resistant pearl millet germplasm. *Crop. Sci. 27* : 367-368.

Sinha, S. and Delela, G.G. 1963. Effect of certain chemicals on the rust development on bajra *(Pennisetum typhoides).* Symposium on: role of therapeutic treatment for controlling tolerant diseases. *Indian Phytopath. 16*(1) : 104-111.

Sivaprakasam, K. and Pillayarasamy, K. 1975. Effect of Nitrogen on the incidence of rust disease of pearl millet caused by *Puccinia penniseti* Zimm. *Madras Agric. J. 62*(4) : 221-223.

Sivaprakasam, K., Pillayarsamy, K., Jagdesan, N. and Kannapiran, C. 1976. Effect of rust infection on the mineral content of pearl millet leaves. *Madras Agric. J. 63* : 134-135.

Sokhi, S.S., Rewal, H.S., Jhooty, J.S. and Bains, S.S. 1978. Investigations on rust of pearl millet. *Indian Phytopath. 31* : 124.

Sokhi, S.S. 1983. Detached leaf culture of rusts of pearl millet and groundnut. *Indian Phytopath. 36* : 100-102.

Sokhi, S.S., Singh, S.J. and Singh, B.B. 1984. Urediae of the Punjab State. *Indian J. mycol. Plant Pathol. 14* : 160-162.

Sokhi, S.S., Bariana, H.S. and Pannu, P.P.S. 1987. A note on genetics of resistance in pearl millet against rust. *Indian Phytopath. 40* : 423-424.

Sundaram, N.V. 1962. Studies on parasites of rusts. *Indian J. Agric. Sci. 32* : 266-271.

Suresh, S. 1969. A preliminary study on the reactions of pearl millet *Pennisetum typhoides* (S and H) inbreds and open pollinated lines to "green ear" and "rust" diseases. *Madras Agric. J. 56* : 88-90.

Wangikar, P.D. and Raut, J.G. 1972. Notes on some plant diseases round about Akala. *The PVK Res. J. 1* : 139-140.

Wells, H.D., Burton, G.W. and Hanna, W.W. 1973a. *Puccinia substriata* var. *indica* a new rust with devastating potential on pearl millet in the Southeast. *Phytopathology 63* : 449.

Wells, H.D., Burton, G.W. and Hennen, J.F. 1973b. *Puccinia substriata* var. *indica* on pearl millet in the Southeast. *Plant Dis. Reptr. 57*(3) : 262.

Wells, H.D. 1978. Eggplant may provide primary inoculum for rust of pearl millet caused by *Puccinia substrata* var. *indica. Plant Dis. Reptr. 62* : 469-470.

Zimmerman, A. 1904. Unterschungen uber tropische pflanzenkrankheiten. *Ber. Land-uForstww in Deut. Ostafrika. 2* : 36.