Citation: Thakur, R.P. 1990. Basic research on management of pearl millet diseases. Pages 343-358 in Basic Research for Crop Disease Management (Vidhyasekaran, P. ed.). Daya Publishing House, New Delhi 110 006, India. (CP 442)

CHAPTER 28

BASIC RESEARCH ON MANAGEMENT OF PEARL MILLET DISEASES

R.P. THAKUR

International Crops Research Institute for the Semi-Arid Tropics, (ICRISAT)
P.O. Patancheru, A.P.-502 324, India

Introduction

The philosophy and approach of plant disease management have been greatly influenced by the evolution of an Integrated Pest Management concept, developed originally as an integrated control approach for insects(57). This concept has developed into a holistic, multidisciplinary management system that integrates control methods, on the basis of ecological and economic principles, for pests of all classes that coexist in an agroecosystem(13). Disease management is an integral part of the crop management system and it implies that diseases are inherent components of the agroecosystem that must be dealt with on a continuous knowledgeable basis. Management is based on the principle of maintaining the damage or crop loss below an economic injury level. Plant disease epidemics result from the conjunction of host, pathogen, weather and time(73) and variations in any of these factors greatly influence the course of an epidemic.

Pearl millet (*Pennisetum glaucum* (L.) R. Br.), an important cereal crop of the semi-arid tropical parts of India, is grown annually on about 11 million ha, mainly in the states of Andhra Pradesh, Gujarat, Haryana, Karnataka, Madhya Pradesh, Maharashtra, Rajasthan, Tamil Nadu, and Uttar Pradesh. Pearl millet suffers from a number of fungal, bacterial, and viral diseases, witchweed (*Striga asiatica*), and nematodes(31). Diseases that are considered economically important, in order of their loss causing potential, are downy mildew (*Sclerospora graminicola* Sacc. Schroet.) ergot (*Claviceps fusiformis* Loveless) smut (*Tolyposporium penicillariae* Bref.), and rust (*Puccinia penniseti* Zimm.). Although these diseases have been known in India for a long time, they reached economic damage level only in the late 1960s and the early 1970s when F_1 hybrids occupied a relatively large area in the farmers' fields (67). The higher susceptibility of hybrids is attributed to their greater genetic uniformity than the open-pollinated varieties(39).

In order to develop a practical disease management program knowledge on the biology of the pathogen, epidemiology of the disease, and genetics and mechanism of disease resistance are essential. In this paper an attempt is made to review and

analyse the information available on each of the four diseases indicated above, and discuss the priority areas needing research to achieve integrated disease management in pearl millet.

Downy Mildew

Biology

Reproduction: Reproduction in S. *graminicola* is both sexual and asexual. Oospores, the sexual spores, are produced in the host tissue from a combined infection by two sexually compatible mating type zoospores(24). Oospores are thick-walled, spherical, light to dark brown, long-lived, resting spores which enable the pathogen to survive long, hot-dry, crop-free periods in the field. Although germination of oospores *in vitro* has been difficult to obtain, some reports(25, 62) claim that oospores germinate by the germ tube, germ sporangia, and by sporangiophores, by the extrusion of small, multinucleate bodies and by the formation of vesicles.

The process of asexual reproduction is dependent upon the supply of photosynthate in the infected host organ, temperature and relative humidity (RH). Sporangia, the asexual spores, are produced on the tips of sporangiophores emerging from the abaxial surface of the infected leaves. They germinate directly by release of motile zoospores through a pore produced in operculum in the apical region of sporangium. Zoospores, once liberated, normally swim in a thin film of water before undergoing encystment and retraction of the flagella. Each sporangium produces 3-8 zoospores(63) and they germinate by producing a germ tube and appressorium while still in water(38). Zoospores are fragile and ephemeral and they seldom survive at temperatures above 32°C and below 14°C(63).

Cytology: Cytological studies on *S. graminicola* have been reported by Shetty(43). Nuclei from the hyphae located in the leaf tissue migrate into the knob-like structures of sporangiophores that emerge from stomata. Subsequently, the nuclei from sporangiophores migrate into the sporangia as soon as these are formed. Normally 3-5 nuclei enter each sporangium, but occasionally as many as 13 nuclei have also been observed. All the nuclei are functional and a zoospores is formed around each of them. Liberation of zoospores from the sporangium is completed within 5-10 min and within 30-45 min a germ tube is produced from a zoospore and the nucleus migrates into the tube. When appressoria develop, nuclei occupy the apical region of the germ tube.

Shetty and Ahmed(44) reported differences in the nuclear contents of two pathogenic races of *S. graminicola*.. The pathogenic race specific to NHB 3 had 2-5 nuclei per sporangium while the other race specific to 'Kalukombu' had 3-13 nuclei, 6 being the most common.

Maintenance of culture/inoculum. S. graminicola is an obligate biotroph. Attempts to grow the fungus in axenic culture using tissue culture techniques have met with limited success(9, 38, 64). Callus culture obtained from the downy mildew infected stem-tip and panicle of pearl millet, developed mycelia after 20-25 days. After profuse mycelial growth sporangia were produced and subsequently oogonial and antheredial

structures developed. The fungus culture can be maintained on callus by repeated subculturing for up to 5 years without losing its virulence(43).

Transmission of downy mildew from infected to healthy calli occurred through contact within 3 days and from infected callus to healthy seedling within 1 week. Young seedlings of a susceptible pearl millet line were successfully infected by placing them on a heavily sporulating leaf for 4-5 h (27). The seedlings were transferred to the Murashige and Skoog medium supplemented with nutrients and incubated at 20 $\pm$ 1 C with a 12 h photoperiod. Callus initiation occurred in 3 days and mycelial growth of *S. graminicola* in 6 days. This technique can be used for screening pearl millet genotypes for downy mildew resistance, and from resistant calli plantlets can be regenerated to produce resistant plants(43). However, the effectiveness of such resistance under field conditions is yet to be demonstrated.

Epidemiology

Pathogen factors: Both seed-borne and soil-borne inocula are known to be a source of primary infection. Surface contamination of seed with oospores lying in glumes and pericarp is reported to be a major source of seed inoculum(75). The internal seed transmission of the disease has been a subject of controversy for a long time. In one study(45) mycelium present in the embryo caused seedling infection, and a linear relationship was observed between the number of infected embryos and infected seedlings, while in another(76) a successful transmission of the disease was not obtained under controlled conditions from the seeds suspected to be infected with *S. graminicola*. The subject has recently been reviewed(75). Nevertheless, the evidence in favour of internal seed transmission of the disease warrants the exercise of caution in the movement of pearl millet seed from the crops infected with downy mildew. A procedure has been laid out(75) to eliminate the possible chances of seed transmission of the disease. The procedure includes: harvest physiologically mature seed from downy mildew free plants, thoroughly sun-dry to 10% moisture level, remove all glumes, husks, and debris, surface sterilize seeds in 0.1% $HgCl_2$ for 10 min followed by several washes in sterile distilled water, redry the seed, and treat it with metalaxyl at 2 g a. i. kg^{-1} seed.

Failure to germinate oospores *in vitro* has been the major limitation in the quantitative estimation of the infective potential of oosporic inoculum in soil, although oosporic inoculum is used to infest soil in field and pot experiments, and in fact the "sick-plot" technique of screening for downy mildew resistance is based entirely on oosporic inoculum in the soil. Subramanya *et al.*(60) reported 31% seedling infection with oospores alone and 68% with both oospores and sporangia. The role of oospores in the secondary spread of the disease, however, is likely very limited.

Sporangia produced on the infected plants play a major role in the secondary spread of the disease(55, 60), although this remained doubtful for a long time(37). Under severe disease pressure the infected seedlings die within 15-20 days and such seedlings may not contribute to oosporic inoculum in the soil. The infected plants that survive produce sporangia and later oospores in the necrotic tissues. As the plant grows the infected leaves senesce and fall to the ground adding numerous oospores

through leaf debris in the soil. Air-borne oospores are also deposited between the glumes of the seed and are carried on the seed as external contaminant. The asexual phase of the pathogen is very efficient; the sporangial cycle repeats every 24 h under favourable conditions, the number of sporangia produced per unit infected leaf surface is large, and spore dispersal and dissemination is rapid.

Environment: Environmental factors, particularly temperature and RH, influence sporulation to a great extent. Sporulation occurs between 14 and 30 C and the maximum is at 23 C and 100% RH(37). Light is also known to influence sporulation, and a minimum of 2 h exposure to light of infected leaf prior to incubation at 24 C and 100% RH is essential for sporulation and with increasing light duration sporangial production per unit lesion areas increases(43). Production 35000 sporangia cm^{-3} of lesion and 11 crops of sporangia were reported (38). Sporangia are forcibly discharged from sporangiophores up to 2.5 m (23) and sporangial liberation occurs continuously at 24 C and 100% RH in darkness. The viability of air-borne sporangia is influenced by prevailing temperature, RH, and wind speed before they are deposited on a suitable substrate. About 50% sporangia remain viable for at least 1 h at 98% RH, 22 C, and 50 m min^{-1} wind speed (59). Singh and Williams (55) record[illegible] dispersal of sporangia up to 340 m during the rainy season but the disease spread occurred only up to 80 m from the inoculum source during the dry season, while Mayee and Sirasker(22) recorded spread of the disease up to 2 km from the source of inoculum. Sporangia that fall to the ground, under favourable conditions, can liberate zoospores in wet soil which can infect plants through roots and root hairs. Sporangia deposited on the soil have been reported to remain infective upto 5 h (36), and these can be source of secondary inoculum as well.

Infection Process: Infection of the pearl millet seedlings by zoospores occurs through roots, root hairs, coleoptiles, and the young meristematic tissues enclosed in the leaf whorl(61). Germinating zoospores develope an appressorium and a tube-like infection peg that develop into primary vesicles which later develop as intercellular hyphae (43).

Pathogenic Variability: Intervarietal differences in susceptibility to *S. graminicola* in pearl millet was for the first time reported by Bhat (8). A pearl millet hybrid, NHB 3, was reported resistant at Mysore but susceptible at other locations in India. Similar observations were made in West Africa (15) and in India again on some other pearl millet lines (44). Results of several years of multilocational testing of pearl millet lines in the International Pearl Millet Downy Mildew Nursery in several countries in W[illegible] Africa and India clearly indicate the differential susceptibility of a set of pearl millet lines (54). Studies at the University of Reading, UK, provide strong evidences of the existence of different pathotypes of *S. graminicola* in India and West African countries (4-6,54). *S. graminicola* isolates from West African countries were generally more aggressive than those from India, and among the West African isolates, those from Nigeria were the most aggressive. An isolate from Zambia resembled Indian isolates in virulence, but differed in its reaction on a hybrid, BJ 104, in which it produced normal susceptible reaction as opposed to stunt reaction produced by all the other isolates from India and West Africa. In India, NHB 3, which is known for its high

sceptibility has shown resistant reaction at one location, Durgapura, Rajasthan, ice 1981(54). This shift in the disease reaction of NHB 3 from susceptibility to sistance over a period of few years, when NHB 3 was out of cultivation, can be ributed to a change in the virulence of the pathogen population in the absence of HB 3 at that location. Differential reactions of MBH 110 and 852 A/B to *S. minicola* isolates from ICRISAT Center, Mysore, and Aurangabad provide further idence of the existence of different pathotypes (S.B. King, ICRISAT, personal mmunication).

sistance

The mode of inheritance of resistance to downy mildew is not very clear. In me studies resistance was reported to be governed by single- or double-dominant nes(2, 14, 53) while in other cases by polygenes with additive and nonadditive ects(7, 46, 51). These differences among genotypes for resistance to downy mildew uld be either due to true genetic differences or due in part to other factors, such as use of heterozygous host genotypes, variable pathogen populations, variable ulum concentrations, different inoculation methods, and differences in weather ditions(54). To obtain meaningful results these variabilities have to be reduced d experiments need to be conducted in controlled environments.

It is well known that pearl millet plants are usually more susceptible to *S. minicola* at the seedling stage than at the later stages of growth, indicating a kind 'adult plant resistance' phenomenon. It means that if plants are protected for about weeks at the seedling stage, downy mildew incidence can very well be reduced. Seed atment with a systemic fungicide, metalaxyl, to control downy wildew works on the ne principle of seedling protection. In certain cases, however, metalaxyl has been nd ineffective and this is probably because of inactivation of the active ingredients exposure to high soil temperature, drought stress, or the evolution of a metalaxyl-sistant *S. graminicola* population.

Recently a new type of resistance called "recovery resistance" has been ntified(18). This resistance is expressed by producing symptom-free leaves on the in shoot which show downy mildew infection on primary leaves, or producing nptom-free secondary shoots from the plants showing downy mildew on the main ot. This phenomenon is in contrast to the generally believed concept for systemic ection in downy mildew. This recovery resistance has been shown to be heritable offers a kind of immunity in the plants. This kind of resistance seems very useful, more studies are needed to understand the mechanism and durability of this sistance before it could be utilized a resistance breeding program.

got

logy

production: Both sexual and asexual reproductions occur in *C. fusiformis*. cospores, the sexual spores, are produced from germinating sclerotia that are ned in the infected florets of pearl millet. Sclerotia are brown to dark brown or k and of variable shapes and sizes. Considerable morphological variations were

reported among *C. fusiformis* isolates collected from different parts of India(12). Host genotypes and environmental factors, particularly temperature, humidity, and frequency of rain showers seem to influence sclerotial morphology. Germination of sclerotia has been erratic, and germination in field and laboratory has been reported with varying degrees of success(21, 30, 69). On germination, sclerotia produce stipes and globular capitula on their tips. Numerous pyriform perithecia are embedded in the peripheral somatic tissue of the capitulum. Asci are interspersed with paraphyses in perithecia. Asci are long, hyaline, and operculate with a narrow base. Each ascus contains eight ascospores which are long, hyaline, nonseptate, and thin-walled.

Conidia produced in the honeydew from infected florets are the asexual spores of the fungus. *C. fusiformis* produces generally two types of conidia, primary or macro-and secondary or microconidia(32, 47), but in some cases production of tertiary conidia has also been reported(29, 47). Macroconidia are fusiform and hyaline, whereas microconidia are globose and hyaline. Macroconidia from fresh honeydew germinate readily within 16 h at 25 C(12, 69), and are usually more infective than microconidia. Both macro and microconidia germinate by producing one to several germ tubes that bear macro or microconidia at the tips.

Cytology: Cytological information on *C. fusiformis* is very limited. Prakash *et al*(29) reported that ascospores are uninucleate and that, on germination, the nucleus from the ascospore migrates into the primary conidium and from there into secondary conidia without undergoing any division. In some ascospores the nucleus divided into two giving rise to two primary conidia. Their inoculation studies indicated that only the last-formed conidia were infective irrespective of their origin from ascospores or conidia.

Maintenance of culture/inoculum: *C. fusiformis* is a biotrophic pathogen. The fungus can easily be cultured on artificial media. The most commonly used medium is Kirchoff's (sucrose 100 g, potassium dihydrogen phosphate 1 g, asparagine 1 g, magnesium sulphate 0.25 g, and 1 L distilled water). Maximum growth and sporulation on Kirchoff's medium occurred at pH 6.5-7.5 at 25 C, and sporulation was inhibited at pH 9(42). Asparagine and magnesium sulphate were found to be the best sources of nitrogen and sulphur, respectively, but sulphates of barium, bismuth, sodium, and copper were toxic.

Growth of *C. fusiformis* on Kirchoff's medium is usually very slow; mycelial growth occurs within 7 days and sporulation within 2 weeks at 25 C. This medium is suitable for maintaining the culture of the fungus but not useful for increasing inoculum. Luxuriant growth and sporulation of *C. fusiformis* was reported(20) on modified calcium nitrate medium (calcium nitrate 2 g, dihydrogen potassium phosphate 2.5 g, magnesium sulphate 1.25 g, maltose 2%, and peptone 1.2 g in 1 L distilled water). Honeydew droplets appeared on the mycelial growth 25 days after inoculation on the medium at 26 $\pm$ 2 C.

Epidemiology

Pathogen Factor: Sclerotia left over in the field at harvest or mixed with seed serve as primary inoculum for the next crop. These sclerotia germinate, following rain showers

irrigation to produce ascospores that become air-borne and infect pearl millet icles at flowering. In addition to ascospores, conidia contained in the cavities and the surface of sclerotia can survive from one crop season to the next and cause ction(65). Honeydew appears within 1 week after inoculation and conidia, both ro and micro, contained in the honeydew are the major source of spread of the ase in a season(47). Conidia are disseminated by splashing rains, wind, physical tact with healthy panicles and, probably, by a number of insects. The role of cts in disease transmission has been reported under controlled conditions(41, 74), ough it is difficult to assume that insect transmission of ergot can occur in nature. cts feed on honeydew and mostly visit panicles that are at anthesis to collect en grains, and they probably never visit panicles having fresh stigmas. It is refore highly unlikely that disease transmission can occur through conidia carried insect bodies.

Several collateral hosts are reported for *C. fusiformis*(32). Recently *Panicum idotale*(66) and *Cenchrus ciliaris*(52) have been reported from Hisar, Haryana and rgapura, Rajasthan, respectively. These grasses grow the year round and bear eydew and sclerotia, and thus serve as a source of primary inoculum for the pearl let crop.

ironment: Weather conditions at the flowering stage of the crop are important in ot epidemiology. High RH (70-100%), an overcast sky with reduced sunshine rs, frequent drizzling rains, and cooler nights (18-20°C) are conducive for ergot elopment(3, 16, 35, 48). Chahal and Dhindsa(11) reported that uniform ribution of rains during the period of flowering favors ergot development. More cise information on macro- and microclimatic conditions are needed to better lerstand the role of weather factors in ergot epidemiology.

ction Process: Infection in pearl millet florets takes place through fresh mas(28, 68) and, once stigmas wither either due to pollination or ageing, infection revented(68). Withering of stigmas occurs because of development of localized striction in style either due to pollination or ageing(78). Conidia germinate on the ts of stigma protruding from the floret. Subsequently penetration and passage of hae down the styloidia follow the path normally taken by the pollen grain(77). onization of the ovary by the fungus proceeds predominantly through the xial wall towards the vascular tract supplying the ovary. Hyphae remain rcellular throughout invasion of the stigma and colonization of the ovary. eydew exudation from the florets marks establishment of the sphacelium 4-5 days r inoculation.

hogenic Variability: Variation in aggressiveness was reported(12) among eight ates of *C. fusiformis* collected from the same cultivar grown in the same season n eight different locations in India. There is, however, no definitive evidence to gest the existence of distinct pathotypes. Pathogenic variability in *C. fusiformis* is bably difficult to demostrate because resistance is largely based on the pollination-tection mechanism, and not on the differential physiologic inhibition of the hogen at the cellular level.

Resistance

Genetics of ergot resistance is relatively complex. Resistance has been reported to be recessive and polygenically controlled(71). More genetic studies are needed to clearly understand the inheritance pattern for properly utilizing the resistance.

Resistance to ergot in most ergot-resistant lines seems to operate through short protogyny, rapid self-anthesis, and stigmatic constriction either due to ageing or pollination(68,78). Susceptibility to ergot, on the other hand, is governed by longer protogyny (>48 h), and delayed pollination/fertilization. Higher susceptibility of F1 hybrids than open-pollinated varieties under natural conditions is attributed to longer protogyny and more synchronous tillering and flowering. In open-pollinated varieties because of asynchrony in tillering and flowering, pollination continues for a longer time that reduces ergot infection. This pollination/fertilization-mediated resistance phenomenon can be utilized to control ergot in pearl millet. Significant control of ergot in pearl millet hybrids obtained(72) by strategically providing pollen to the flowering hybrid crop by manipulating planting dates of the test hybrid and an ergot-resistant pollen donor line, or mixing the seeds of the two in proper proportion.

Smut

Biology

Reproduction: *T. Penicillariae* reproduces both sexually and asexually. It is assumed that sexual reproduction takes place through plasmogamy between any two sporidia of compatible mating types giving rise to dikaryotic mycelium as occurs with several other cereal smuts. The characteristic teliospores are produced from the dikaryotic mycelia in the host tissue. The individual teliospores are typically globose to subglobose, and yellowish brown. A large number of teliospores are held together in a sporeball. Sporeballs are of various shapes and sizes, and the number of teliospores per sporeball varies from 200 to 1400 (58). Individual teliospores germinate to produce a typical four celled promycelium on which basidiospores are borne either laterally or terminally.

Asexual reproduction is by budding of basidiospores or sporidia produced on the promycelium. Sporidia are hyaline, single-celled and spindle-shaped. Maximum germination (67%) of teliospores occurred at 30°C and minimum (16%) at 15°C(58). A significant positive correlation was found between teliospore germination and temperature up to 30°C.

Cytology: Nuclear activity in *T. Penicillariae* is typical of smut fungi. The cycle proceeds from the resting stage or diplophase (teliosore) to vegetative or haplophase (sporidia) to the parasitic dikaryophase (infective hyphae). In contrast to the other seed-borne smuts, dikaryophase in *T. Penicillariae* is much shorter, about 2 weeks. The diplophase or resting stage in the form of teliospores is much longer, about 8-9 months, and the vegetative phase for about 2-3 days on the host surface (R.P. Thakur, unpublished).

Maintenance of Culture/Inoculum: *T. Penicillariae* is a biotroph and has successfully been grown on such simple media as potato extract, potato agar, carrot extract, or

rrot agar with and without sugar.(58) Growth on potato or carrot agar is ostly sporidial, and maximum growth is obtained at 30-35°C within 5 days ter inoculation on the medium. Growth is very slow below 20 and above 40°C. owever, Pathak and Shekhawat(26) found maximum growth on Brown's medium at °C at pH 7.5. Among the carbon sources, inulin supported maximum growth llowed by dextrose.

pidemiology

athogen factor: Teliospores in the soil left over from the previously infected panicles id teliospores adhering to the seed surface are the main source of primary inoculum. nder favourable conditions of soil moisture, teliospores germinate to produce illions of sporidia which become air-borne and infect the flowering panicles of pearl illet. The incubation period is usually about 2 weeks and latency about 3 weeks. mut sori, initially appears shiny on infected panicles; they later turn brown to dark rown at maturity, and rupture to release masses of teliosporeballs. These teliospores re deposited on the ground or carried by the wind to the neighbouring fields and, nder favourable conditions, can cause infection. Secondary spread of the disease hin a crop is minimal because of a prolonged latent period, by which time lowering is almost complete. Significant reduction in smut infection due to rapid ollination has been demonstrated(70). It seems that under natural conditions smut nfection is greatly reduced due to the availability of enough pollen in the field when he weather is dry at flowering time; but, when flowering coincides with wet weather, nther dehiscence is adversely affected and pollen grains are washed, and sporidia ecome abundant in the atmosphere. This results in reduced competition between ollen grains and sporidia and thereby severe smut infection occurs (R.P. Thakur, npublished).

Environment: Weather factors, particularly temperature and RH, are most important n smut epidemiology. A temperature range of 20-35°C and >80% RH at the time of lowering are congenial for smut development. A longer duration of warm weather >30°C for more than 12 h a day is more favourable than shorter periods. Based on he natural occurrence of smut it is now very clear that smut requires warmer weather han ergot (R.P. Thakur, unpublished). However, more precise information on weather factors influencing smut development are needed.

Infection Process: Bhatt(10) studied the infection process of *T. Penicillariae* in detail. he hyphae penetrate the flower through emerging stigma and reach the upper wall of the ovary, traversing the whole length of the style without lateral spread. The mycelium is binucleate, inter and intracellular, exhibiting slight branching with two to four-lobed haustoria. The hypha advances downward through the ovary wall and finally invades the ovule. Before the whole tissue is involved, the walls of the hyphae begin to gelatinize to form the sporeballs.

Pathogenic Variability: As a rule, smuts are highly variable and *T. penicillariae* should be no exception. However, there is no definite evidence so far on the existence of different pathotypes in this pathogen.

Resistance

Resistance to smut in pearl millet is reported to be governed by either single or double genes(79). Studies conducted at ICRISAT Center indicated that inheritance of smut resistance was simple with a dominant effect(18).

Mechanism of resistance to smut is not known except that pollination-mediated resistance, as in ergot, also operates for smut in pearl millet(70). More research is needed to understand the histopathological and biochemical aspects of the resistance mechanism.

Rust

Biology

Reproduction: *P. penniseti* is a macrocyclic, heteroecious rust. Uredia and telia are formed on pearl millet, and pycnia and aecia on the alternate host, *Solanum melongena*. Since pycnial and aecial stages are not directly related to pearl millet, the discussion will mainly be confined to the uredial and the telial stages.

The uredia are formed subepidermally and at maturity they rupture the epidermis which forms flecks around the uredosori. The uredospores are borne on hyaline pedicells interspersed with paraphyses mainly along the margin of the sori. The uredospores are oval, pyriform, or elliptical with four equatorial germ pores. The spores are yellowish brown with sparse echinulations.

The telia are amphigenous, often in groups and black. These are formed subepidermally, and unlike uredosori, teliosori remain covered for a longer time without bursting. Teliospores are dark brown, bicelled, cylindrical to club-shaped with a lower cell larger than the upper one. The apex is flattened, round or blunt.

Cytology: Kulkarni(19) made extensive cytological studies on *P. penniseti*. The uredospore germ tube penetrates the stomata and produces hyphal cells in the substomatal cavities. After repeated cycles of cell division, compact tissue of binucleate cells is formed which gives rise to uredial 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 cells become the uredospores and the lower stalk-cells. The process is repeated several times; the nuclear pairs are reorganized ultimately giving rise to pedicillate uredospores comprising the fully developed uredium.

The telia develop either in the old uredia or independently as a new structure from the binucleate basal cells. The binucleate basal cells undergo division and cut off vertical rows consisting of three rows that are regularly binucleate. The upper cells form the bicelled teliospores and the lower ones develop into stalks.

Maintenance of culture/inoculum: Like many other heteroecious rusts, *P. penniseti* has not been cultured on artificial media. Sokhi *et al.*(56) reported a detached leaf technique to maintain the culture of this fungus. On the detached leaves of pearl millet, infected with *P. penniseti*, supported on water alone, and a solution containing

benzimidazole (40 ppm) and glucose (0.05%), infective uredospores were maintained for 20 and 40 days, respectively, at 20°C under 78 ft candle light. Fully developed uredosori were observed on the 10th day after inoculation and 8 uredospore crops were harvested at 3-day intervals. Top leaves of 40-day-old plants sporulated for 24 days; the younger plants had a shorter period of sporulation. The flag leaf was found most suitable for the detached leaf culture.

Like in other cereal rusts uredospores in glass vials can generally be stored in liquid nitrogen for several years. Uredospores have been stored at -70°C for about 8 months without losing viability (C.S. Kousik, ICRISAT, personal communication).

Epidemiology

Pathogen factor: Uredospores from the main host or collateral hosts, and aeciospores from the alternate host serve as the primary inoculum source. Once pearl millet plants are infected, uredial production is fast, and the cycle is repeated every 7-8 days under favourable weather conditions. Rust usually appears on the lower leaves and its severity increases with increasing plant age, with maximum severity occurring towards the post-flowering stage of the crop. However, seedling infections in severe form have also been reported(56). Sharma and Pathak(40) reported post-inoculation exposure of plants to high humidity at 22°C under continuous light essential for obtaining maximum infection by *P. penniseti*. In nature, rust appearance is usually correlated with cool night and dry periods.

Uredospores germinate in 3-4 h in hanging drops of sterilized distilled water. Germination occurs at 15-30°C, and the maximum being at 20°C and no germination was observed below 5 and above 35°C. Uredospore germination was 100% soon after collection, but air-dried spores stored at 28-30°C lost viability in 30 days(33). When stored at 5-8°C, uredospores were viable for 30 days in dried leaves at 25-27°C.

In addition to *S. melongena,* the alternate host important for completion of the life cycle of the pathogen, several collateral hosts are reported which are important in the epidemiology of the disease. Several *Pennisetum* spp. including *P. leonis. P. polystacyion*, *P. oriantale*, *P. nigritarum* and *P. violaceum*, grasses commonly found around pearl millet fields are known to harbour the pathogen.

Pathogenic Variation: As applies with any other heteroecious rust, the existence of pathogenic races in this pathogen cannot be ruled out. However, very little information is available on this aspect. The presence of two types of uredia, isolated and scattered, and in groups, on dark brown lesions on naturally infected leaves of pearl millet are known (34). These two types of uredia were consistent for several generations. All the cultivars of pearl millet showed both types or uredial lesions, except the F_1 hybrid between *P. typhoides* x *P. purpureum* which showed only a grouped-type lesion indicating the existence of two distinct pathotypes. A rust-resistant line of pearl millet, ICML 11, with a single dominant gene identified at ICRISAT Centre, was found susceptible at Tifton, Georgia (H.D. Wells, personal communication), indicating that the ICRISAT and the Tifton isolates were different pathotypes. More research efforts are needed to determine the existence of pathotypes or races in this important pathogen.

Resistance

Inheritance of resistance to rust was reported to be monogenic dominant to the pathogen in India (1). Another dominant gene for resistance was identified in a wild relative of pearl millet, *P. americanum* subspecies *monodii* (Maire) Bruken from Senegal, at Tifton, Georgia, USA(17). This wild species is known to possess several useful attributes including resistance to downy mildew, smut and some leaf spot diseases(17).

A slow-rusting type of resistance to *P. penniseti* in pearl millet(50), was found to be controlled by several components, such as longer incubation and latent periods, smaller uredosori, and less uredospore production than those in the fast-rusting lines(49). This kind of resistance is usually stable and governed by polygenes. More studies are needed to clearly understand the mechanism of slow-rusting in this system to properly utilize it in the resistance breeding program.

Concluding Remarks

Considerable progress has been made on basic research on major pearl millet diseases in India during the past 10 years, but still there are many gaps in our knowledge of understanding the host-pathogen-environment-time interactions. Some of the important research areas that need attention are: 1. The role of *S. graminicola* oospores in downy mildew epidemiology; 2. Characterization of environments favourable for disease epidemics; 3. Pathogenic variation as influenced by changes in host genotypes in different agroecosystems; 4. The genetics and mechanism of resistance in hosts and the genetics of virulence in the pathogen; 5. The biochemical basis of resistance; 6. Edaphic and weather factors, and crop agronomy in relation to disease development in a given agroecosystem; 7. Application of tissue culture and genetic engineering techniques to understand the genetics of host-pathogen interactions; and 8. Development of an integrated crop management system through interdisciplinary efforts to sustain and increase the productivity and production of pearl millet.

References

1. Andrews, D.J. Rai, K.N. and Singh, S.D. 1985. A single dominant gene for rust resistance in pearl millet. Crop Science 25:565-66.

2. Appadurai, R., Parambaramani, C. and Natarajan, U.S. 1975. Note on the ineritance of susceptibility of pearl millet downy mildew. Indian J. agric. Sci. 45:179-80.

3. Arya, H.C. and Kumar, A. 1982. Ergot epidemic of pearl millet in Rajasthan. pp 439-51 *in* Recent Advances in the Biology of Microorganisms. (Bilgrami, K.S., Vyas, K.M. Singh, B. and Singh, M.P., eds.) Vol. 2. Dehra Dun, Uttar Pradesh, India: Bishen Singh Mahendra Pal Singh.

4. Ball, S.L. 1983. Pathogenic variability of downy mildew (*Sclerospore graminicola*) on pearl millet. 1. Host cultivar reactions to infection by different pathogen isolates. Ann. appl. Biol. 102:257-64.

5. Ball, S.L. and Pike, D.J. 1983. Pathogenic variability of downy mildew (*Sclerospora graminicola*) on pearl millet. II. Statistical techniques for analysis of data. Ann. appl. Biol. 102: 265-73.

6. Ball, S.L. and Pike, D.J. 1984. Intercontinental variation of *Sclerospora graminicola*. Ann. appl. Biol. 104:41-51.

7. Basavaraj, R., Safeeulla, K.M. and Murty, B.R. 1980. The role of gene effects and heterosis for resistance to downy mildew in pearl millet. Indian J. Genet. Pl. Br. 40: 537-48.

8. Bhat, S.S. 1973. Investigations on the biology and control of *Sclerospora graminicola* on bajra. Ph. D. thesis, University of Mysore, Mysore, Karnataka, India 165 p.

9. Bhat, S.S., Safeeulla, K.M. and Shaw, C.G. 1980. Growth of *Sclerospora graminicola* in host tissue culture. Trans. Br. mycol. Soc. 75:303-09.

10. Bhatt, R.S. 1946. Studies in Ustilaginales. I. The mode of infection of the bajra plant (*Pennisetum typhoides* Stapf.) by the smut *Tolyposporium penicillariae*. J. Indian Bot. Soc. 25:163-86.

11. Chahal, S.S. and Dhindsa, H.S. 1985. Relationship between ergot severity and rainfall distribution during flowering in pearl millet. Indian Phytopath. 38:329-31.

12. Chahal, S.S., Rao V.P. and Thakur, R.P. 1985. Variation in morphology and pathogenicity in *Claviceps fusiformis*, the causal agent of pearl millet ergot. Trans. Br. mycol. Soc. 84: 325-32.

13. Glass, E.H. 1975. "Integrated Pest Management: Rationale, Potential, Needs and Implementation", Spec. Publ. 75-2. Entomol. Soc. Am.

14. Gill, K.S., Phul, P.S. Chahal, S.S and Singh, N.B. 1978. Inheritance of resistance to downy mildew disease in pearl millet. Cereal Res. Comm. 6:71-74.

15. Girard, J.C. 1975. Downy mildew of pearl millet in Senegal, pp 59-73 *in* Proceedings of Consultants' group Meetings on Downy mildew and Ergot of Pearl Millet, 1-3 Oct 1975, ICRISAT, Hyderabad, India. Patancheru, A.P. 502 324, India: International Crops Research Institute for the Semi-Arid Tropics.

16. Gupta, G.K., Rao. G.V.S., and Sexena, M.B.L. 1983. Relationship between meteorological factors and the occurrence of ergot disease (*Claviceps microcephala*) of pearl millet. Tropical pest Management 29:321-24.

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

18. ICRISAT (International Crops Research Institute for the Semi-Arid Tropics). 1987. Annual Report 1986. Pearl Millet. pp 68-126. Patancheru, A.P. 502 324, India: ICRISAT.

19. Kulkarni, U.K. 1958. Studies on the development and cytology of *Puccinia penniseti* Zimm. Trans. Br. mycol. Soc. 41:65-73.

20. Kumar, A. and Arya, H.C. 1978. *In vitro* production of honeydew in *Claviceps fusiformis*. Curr. Sci. 47:781-82.

21. Loveless, A.R. 1967. *Claviceps fusiformis* sp. nov. the causal agent of an agalactia of sows. Tans Br. mycol. Soc. 50:15-18.

22. Mayee, C.D. and Siraskar, R.D. 1980. Epidemiological studies on downy mildew of pearl millet. Indian Phytopath. 33-149. (Abstr).

23. Melhus, I.E., Van Haltern, P. and Bliss, D.E. (1927). A study of the downy mildew *Sclerospora graminicola* (Sacc.) Schroet. Phytopathology. 17:57.

24. Michelmore, R.W., Pawar, M.N. and Williams, R.J. 1982. Heterothallism in *Sclerospora graminicola*. Phytopathology 72:1368-72.

25. Nene, Y.L. and Singh, S.D. 1976. Downy mildew and ergot of pearl millet. PANS 22: 366-85.

26. Pathak, V.N. and Shekhawat, B.S. 1980. Physiological studies on *Tolyposporium penicillariae*. Indian Phytopath 33:208-11.

27. Prabhu, M.S.C. 1985. Studies on *in vitro* cultures of downy mildew pathogens of bajra and sorghum. Ph.D. thesis, University of Mysore, Mysore, Karnataka, India.

28. Prakash, H.S., Shetty, H.S. and Safeeulla, K.M. 1980. Histology of carpel infection by *Claviceps fusiformis* in pearl millet. Proceedings of Indian Nat. Acad. Sci. B. 46: 708-12.

29. Prakash, H. S., Shetty, H.S. and Safeeulla, K.M. 1983. Germination and nuclear behavior of sexual and asexual propagules of *Claviceps fusiformis*. Trans. Br. mycol. Soc. 81:65-69.

30. Prakash, H.S., Shetty, H.S., Subramanyam, S., and Safeeulla, K.M. 1981. Standardization of sclerotial germination technique and infectivity of ascospores of *Claviceps fusiformis* Iov. Indian J. agric. Sci. 51:900-04.

31. Rachie, K.O. and Majmudar, J.V. 1980. Pearl Millet. University Park, Pennsylvania, USA, Pennsylvania State Univ. Press. 307 p.

32. Ramakrishnaan, T.S. 1971. Diseases of Millets. New Delhi, India: Indian Council of Agricultural Research. 152 p.

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

34. Ramakrishnan, T.S. and Sundaram, N.V. 1956. Further studies on *Puccinia penniseti* Zimm. Proc. Indian Acad. Sci. B 43:190-96.

35. Ramaswamy, C. 1968. Meteorological factors associated with the ergot epidemic of bajra (*Pennisetum*) in India during the *Kharif* season 1967-a preliminary study. Curr. Sci. 37:331-35.

36. Ramesh, C.R. 1981. Studies on host and parasitic systems of the green ear disease of bajra. Ph.D. thesis, Univ. of Mysore, Mysore, Karnataka, India.

37. Safeeulla, K.M. 1976. Downy mildew of pearl millet. pp 85-95 *in* Proceedings of the Consultants' Group Meetings of Downy mildew and Ergot of Pearl Millet, 1-3 Oct 1975, ICRISAT Center, India. Patancheru A.P. 502 324., India: International Crops Research Institute for the Semi-Arid Tropics.

38. Safeeulla, K.M. 1976. Biology and control of the downy mildews of pearl millet, sorghum and finger millet. Mysore, Karnataka, India: University of Mysore, Downy Mildew Research Laboratory, p. 304.

39. Safeeulla, K.M. 1977. Genetic vulnerability: the basis of recent epidemics in India. Annals of the New York Academy of Sciences 287: 72-85.

40. Sharma, K.B. and Pathak, V.N. 1987. Studies on pearl millet rust. p 313 *in* Proceedings on the International Pearl Millet Workshop, 7-11 April, 1986. ICRISAT Center India, Patancheru, A.P. 502 324, India: ICRISAT

41. Sharma, Y.P., Singh, R.S. and Tripathi, R.K. 1983. Role of insects in secondary spread of the ergot disease of pearl millet (*Pennisetum americanum*). Indian Phytopath. 36: 131-33.

42. Sharma, R.K. Verma, O.P. and Pathak, V.N. 1984. Some physical and nutritional factors for the growth and sporulation of *Claviceps fusiformis* Lov. Cryptogamic Mycol. 5: 269-75.

43. Shetty, H.S. 1987. Biology and epidemiology of downy mildew of pearl millet. pp 147-60 *in*

Proceedings of the International Pearl Millet Workshop, 7-11 April, 1986, ICRISAT Center, India. Patancheru A.P. 502 324, India: ICRISAT.

44. Shetty, H.S. and Ahmed, R. 1981. Physiological specialization in *Sclerospora graminicola* attacking pearl millet. Indian Phytopath. 34:307-09.

45. Shetty, H.S., Mathur, S.B. and Neergaard, P. 1980. *Sclerospora graminicola* in pearl millet seeds and its transmission. Trans. Br. mycol. Soc. 74: 127-34.

46. Shinde, R.B., Patil, F.B. and Sangave, R.A. 1984. Resistance to downy mildew in pearl millet. J. Maharashtra Agr. Univ. 9: 337-38.

47. Siddiqui, M.R. and Khan, I.D. 1973. Renaming *Claviceps microcephala*, ergot on *Pennisetum typhoides* in India as *Claviceps fusiformis*. Trans. Mycol. Soc. Japan 14: 195-98.

48. Siddiqui, M.R. and Khan, I.D. 1973. Dynamics of inoculum and environment in relation to ergot incidence of *Pennisetum typhoides* (Burm) Stapf and Hubbard. Trans. Mycol. Soc. Japan 14:280-88.

49. Singh, B.B. 1981. Studies on rust of pearl millet caused by *Puccinia penniseti* Zimm. with special reference to disease resistance. M.Sc. Thesis. Punjab Agri. University, Ludhiana. 58 p.

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

51. Singh, F., Singh, R.K., Singh, R.M. and Singh, R.B. 1978. Genetic analysis of downy mildew (*Sclerospora graminicola*) resistance in pearl millet (*Pennisetum typhoides* (Brum.) S.& H.) Zeitschrift fuer Pflanzenzuechtung 81:54-57.

52. Singh, G., Vyas, K.L. and Bhatt, B.N. 1983. Occurrence of pearl millet ergot on *Cenchrus ciliaris* pers. in Rajasthan, Indian J. agric. Sci. 53: 481-83.

53. Singh, S.D. 1974. Studies on the downy mildew disease (*Sclerospora graminicola* (Sacc.) Schroet.) of bajra (*Pennisetum typhoides* (Brum.F.) Stapf and C.E. Hubb.). Ph. D. thesis, Indian Agricultural Research Institute, New Delhi, India. 126 p.

54. Singh, S.D., Ball, S. and Thakur, D.P. 1987. Problems and strategies in the control of downy mildew. pp 161-72 *in* Proceedings of the International Pearl Millet Workshop, 7-11 April. 1986, ICRISAT Center, India. Patancheru, A.P. 502 324, India, ICRISAT.

55. Singh, S.D. and Williams, R.J, 1980. The role of sporangia in the epidemiology of pearl millet downy mildew. Phytopathology 70:1187-90.

56. Sokhi, S.S., Rewal, H.S., Jhooty, J.S. and Brains, S.S. 1978. Investigation on rust of pearl millet. Indian Phytopath. 31:134 (Abstr.).

57. Stern, V.M., Smith, R.F. Van den Bosch, R. and Hayen, K.S. 1959. The integrated control concept. Hilgardia 29:81-101.

58. Subba Rao, K.V. and Thakur, R.P. 1983. *Tolyposporium penicillariae*, the causal agent of pearl millet smut. Trans. Br. mycol. Soc. 81:597-603.

59. Subramanya, S. and Safeeulla, K.M. 1981. Aerosol viability of sporangia of *Sclerospora graminicola*. Abstracts of the third International Symposium on Plant Pathology, 14-18 Dec, 1981, New Delhi. Indian Phytopathological Society, New Delhi, India: IARI.

60. Subramanya, S., Safeeulla, K.M. and Shetty, H.S. 1982. Importance of sporangia in the epidemiology of downy mildew of pearl millet. Proceedings of Indian Nat. Sci. Acad. 48: 824-33.

61. Subramanya, S., Shetty, H.S. and Safeeulla, K.M. 1983. Pearl millet downy mildew: biology of systemic infection by zoospores Proceedings of the Indian Nat. Sci. Acad. 49: 377-86.

62. Sundaram, N.V. and Gurha, S.N. 1977. Note on the rapid method of germination of oospores of *Sclerospora graminicola* (Sacc.) Schroet., the causal agent of pearl millet downy mildew. Indian J. agric., Sci. 47: 165-69.

63. Suryanarayana, D. 1965. Studies on the downy mildew disease of millet in India. Bulletin, Indian Phytopathological Society 3: 72-78.

64. Tiwari, M.M. and Arya, H.C. 1969. *Sclerospora graminicola* axenic culture. Science 163:291-93.

65. Thakur, D.P. 1983. Epidemiology and control of ergot disease of pearl millet. Seed Sci. Tech. 11:797-806.

66. Thakur, D.P. and Kanwar, Z.S. 1978. Ability of naturally incident *Claviceps microcephala* from *Panicum antidotale* to produce ergot symptoms in *Pennisetum typhoides*. Indian J. agric. Sci. 48:540-42.

67. Thakur, R.P. 1987. Diseases of pearl millet and their management. pp 147-58 *in* Plant Protection in Field Crops (M.V. Rao and S. Sithanandam eds.). Plant Protection Assoc. India. Rajendranagar, Hyderabad, India.

68. Thakur, R.P. and Williams, R.J. 1980. Pollination effects on pearl millet ergot. Phytopathology 70:80-84.

69. Thakur, R.P., Rao, V.P. and Williams R.J. 1984. The morphology and disease cycle of ergot, caused by *Claviceps fusiformis*, in pearl millet. Phytopathology 74: 201-05.

70. Thakur, R.P., Subba Rao, K.V., and Williams, R.J. 1983. Effects of pollination on smut development in pearl millet. Plant Pathol. 32: 141-44.

71. Thakur, R.P., Talukdar, B.S. and Rao. V.P. 1983. Genetics of ergot resistance in pearl millet. p 737 *in* Abstracts of contributed papers of the XV International Congress of Genetics, 12-21 Dec 1983. New Delhi, India. Pt 2. New Delhi, India: Oxford IBH Publishing Co.

72. Thakur, R.P., Williams, R.J. and Rao, V.P. 1983. Control of ergot in pearl millet through pollen management. Ann. appl. Biol. 103:31-36.

73. Van der Plank, J.E. 1975. "Principles of Plant Infection" Academic Press, New York.

74. Verma, O.P. and Pathak, V.N. 1984. Role of insects in secondary spread of pearl millet ergot. Phytophylactica 16:257-58.

75. Williams, R.J. 1984. Downy mildews of tropical cereals. Advances in Plant Pathology. 2:1-103.

76. Williams, R.J., Pawar, M.N. and Huibers-Govaert, I. 1980. Factors affecting staining of *Sclerospora graminicola* oospores with triphenyl tetrazolium chloride. Phytopathology 70:1092-96.

77. Willingale, J. and Mantle, P.G. 1987. Stigmatic constriction in pearl millet following infection by *Claviceps fusiformis*. Physiological and Molecular Plant Pathology 30:247-57.

78. Willingale, J., Mantle, P.G. and Thakur, R.P. 1986. Post pollination stigmatic constriction, the basis of ergot resistance in selected lines of pearl millet. Phytopathology 76: 536-39.

79. Yadav, R.P. 1974. Inheritance of smut resistance in pearl millet. Agra Univ. of Res. 23: 37-39.

Eradication of Agricultural Pests Diseases

Basic Research for Crop Disease Management

P. Vidhyasekaran

Physiology of disease resistance is the most fascinating field of plant pathology. Plants have their own built—in defence mechanisms against almost all microorganisms, but in a few cases the pathogen overcome the defence barrier with their offensive chemicals and cause diseases. It is a battle between microorganisms and plants, and sometimes the plants lose the battle. If the mystery of disease resistance is unravelled, scientists can engineer plants to win the battle even when the pathogens have an array of offensive chemicals. Several factors contribute to disease resistance, and by identifying the links between different mechanisms, we may be able to alter host physiology to ward off the pathogens by connecting or disconnecting some of the vital links. This book critically analyses the various physiological and biochemical processes involved in the defence mechanisms of various crops such as rice, wheat, maize, sorghum, finger millet, tomato, potato, beans, cucumber, chillies, cotton, sugarcane, tobacco, apple, orange, grapevine, mango and banana against fungal, bacterial and viral diseases. It reviews all of the published works critically and suggests future course of research.

Contents

Virus Disease Management

13. Virus Inhibitors from Plants and Crop Disease Management—H.N. Verma and V.K. Baranwal
14. Antiviral Principles for Virus Disease Management—P. Narayanasamy
15. Basic Research for the Management of Vectors of Plant Virus Diseases—S. Jayaraj
16. Basic Research on Behaviour of Insect Vectors for Developing Effective Virus Disease Management—M.S. Venugopal and K. Gunathilagaraj
17. Cross Protection in the Management of Virus Diseases—H.R. Reddy
18. Possibility of Controlling Virus Diseases by Plant Products—V. Mariappan

Bacterial Disease Management

19. Basic Research on Management of Bacterial Diseases of Plants—J.P. Verma

Epidemiology and Forecasting

20. Mathematical and Statistical Epidemiology—Mrinal K. Das Gupta and Sibaji Chakraborti
21. Basic Research on Plant Disease Forecasting—S.T. Tilak
22. Basic Research on Epidemiology of Rice Diseases—M.C. Nair and C. Gokulapalan
23. Basic Research on Epidemiology of Pearl Millet Diseases—V.N. Pathak and G.R. Kumhar

Resistance Breeding

24. Modern Approaches to Breeding for Disease Resistance—E.A. Siddiq

Biological Control

25. Mycorrhiza—A Tool for Biocontrol—Bushan L. Jalali

26. Soil Amendment for Crop Disease Management—K. Sivaprakasam

Management of Diseases of National Improvance

27. Basic Research on Downy Mildew of Cereals and Disease Management—H. Shekara Shetty
28. Basic Research on Management of Pearl Millet Diseases—R.P. Thakur
29. Basic Studies on Fungal Diseases of Rice for Developing Effective Management Practices—S. Gangopadhyay
30. Management of Sheath Rot of Rice—R. Ramabadran and R. Velazhahan
31. Basic Research on Rice Sheath Blight Disease-K. Manibhushan Rao, U.I. Baby and Y. Joe
32. Current Fungal Disease Problems of Important Vegetables in India—S.S. Sokhi, G.D. Munshi, R.K. Grewal, T.S. Thind and K.S. Sandhu
33. Management of Seed Borne Diseases-M.R. Siddiqui