

Title: COMPENDIUM OF SMALL MILLETS DISEASES

Editors:

Dr. A. NAGARAJA,
Pathologist & TPL,
Project Coordination Cell,
AICSMIP, UAS,
GKVK, Bangalore – 560 065

Dr. J. KUMAR
Director Research,
University of Horticulture and Forestry,
Ranichauri, Uttarakhand -249 199

Dr. K.T. KRISHNE GOWDA
Project Coordinator,
All India Coordinated Small Millets Improvement Project
ICAR, UAS, GKVK Campus, Bangalore –560 065

Published by:

Dr. K.T. KRISHNE GOWDA
Project Coordinator (Small Millets), P.C. Cell,
All India Coordinated Small Millets Improvement Project
ICAR, GKVK Campus, Bangalore –560 065, India

Year of Publication: 2007

Number of Copies: 200

Printed by: Raghu Print System
Alumni Association Building
UAS, Hebbal, Bangalore-24

COMPENDIUM OF SMALL MILLETS DISEASES

AUTHORS

A. NAGARAJA
J. KUMAR
A. K. JAIN
Y. NARASIMHUDU
T. RAGUCHANDER
BIJENDRA KUMAR
B. HANUMANTHE GOWDA



Project Coordinating Unit
All India Coordinated Small Millets Improvement Project
UAS, GKVK Campus, Bangalore -560 065, India

2007

CONTENTS

#	DESCRIPTION	PAGE
1.	Introduction	
2.	Diseases of Finger millet	
3.	Diseases of Kodo millet	
4.	Diseases of Foxtail millet	
5.	Diseases of Little millet	
6.	Diseases of Barnyard millet	
7.	Diseases of Proso millet	
8.	Pathometry	
9.	Selected References	
10.	Title index	

FOREWORD

Small millets as a group include several grain crops viz., finger millet (ragi), kodo millet (kodo), foxtail millet (kangni), barnyard millet (sawan), proso millet (cheema) and little millet (kutki) that are popular as 'nutri-cereals' owing to their high calcium, iron, fibre and other quality aspects. With a long history of cultivation of more than 5000 years, these crops, grown in many states of the country, are quite important in areas of their production as dry land crops, as well as for tribal and hill agriculture, providing staple food for the people of the region. These crops, though, occupy relatively a lower position among food crops; they are important as food security crops at the regional as well as farm level. They further contribute to the widening of the food basket, which at present is narrow because of heavy dependence on a fewer food crops. The resilience exhibited by these crops is helpful in their adjustments to different kinds of ecological niches

Small millets are hardy crops and show quick rejuvenating capacity to various biotic and abiotic stresses. However, some diseases such as blast and smut take heavy toll of the crops each season in many parts of the country. A few other diseases like green ear and viruses in ragi were a potential threat to the cultivation of the crop. Though not economical, ediphenphos and kitazin were recommended for blast control. Realizing the importance of the need for varieties with disease endurance, resistance breeding was given priority and thus superior types like VL 149, GPU 45, GPU 28 and GPU 48 have been released for commercial cultivation. Further, over the years, some of the minor diseases have also become important especially when conditions turn favourable and cause losses to various degrees. Proper understanding of all such diseases affecting small millet cultivation in different parts of the country is thus necessary before crop protection strategies are evolved to sustain the production. Recently, bio agents like *P. fluorescens* and *T. viridae* have been found effective in disease management. Used for seed treatment and / or for spraying are highly ecofriendly and are useful in green agriculture. In this context, preparation of the **Compendium of Small Millets Diseases** is very timely and I would like to compliment the efforts made by the authors in its preparation.

I am sure, the compendium would prove useful to all those engaged in the small millet research, extension and production in the country.

S. N. Shukla
Assistant Director General (FFC)

Place: New Delhi
Date: March, 2007

PREFACE

Agro ecology and health are very important global issues. A exacting agro climatic zone produces a particular type of food that has a matching ecological cycle. Cultivation of traditional crops like small millets (viz., finger millet, kodo millet, foxtail millet, barnyard millet, proso millet and little millet), considered to be poor man's food, have strong bearing with ecology, economy and sociology of a region. These crops though occupy lower position in food crops but are important ecological food security crops for different agro climatic level. These coarser grains are also called nutri-cereals, because of higher nutritive values. They are one of the cheapest sources of dietary energy, in the form of proteins and carbohydrates. Millets give more calories to the body in comparison to the wheat and rice and hence are also called the 'energy food'.

The resilience exhibited by these crops is helpful in adjusting themselves to different kinds of ecological niches. Thus, it is important to enhance production and productivity of these crops to ensure food and nutritional security to the people living in harsh and difficult terrains. Utilization of these crops is mainly as food for human consumption. The straw is often a precious fodder for bovines. The grain is consumed in the traditional way and almost the entire produce is utilized at the farm/ village level. Being eco-friendly crops, they are suitable for fragile and vulnerable ecosystems.

Small millets although known to successfully cope up with losses due to biotic and abiotic causes, under vulnerable conditions some disease take a heavy toll of the crops in various ecological zones. For example, blast takes a heavy toll of the finger millets, wherever grown. The disease problems of millets including finger millet were first reviewed by the well-known pathologist T. S. Ramakrishnan (1963) in the monograph 'Diseases of millets' and subsequently by B. S. Pall and co-authors (1980) in their book 'Diseases of lesser millets'. Recently, T. B. Anil Kumar and co-authors (2003) in their publication have also addressed the 'Diseases of finger millet' but no such compilation has been made on diseases of other minor millets. During the last few years, considerable information has been generated on disease problems of small millets and their management. Therefore, an attempt has been made to compile the available information with pictorial depictions so as to help small millets researchers, extension specialists in the proper diagnosis of the diseases. Further, a separate chapter has been included on pathometry that includes the appropriate scale to be adopted and rating of the germplasm and test entries against each important disease.

It is hoped that this compilation would help scientists, teachers and students involved in small millet research to get ready and detailed account of small millet diseases.

Though utmost care has been taken to make the text error free, however some omissions might have been there. It is expected that all the shortfalls, errors etc., would be brought to the notice of the authors for further improvement.

AUTHORS

ACKNOWLEDGEMENT

This compendium has been possible due to the contributions of all the earlier pathologists, who worked on small millet crops and made recommendations. We wish to place on record our gratitude to former pathologists Dr. T. B. Anil Kumar from the Project Coordinating Unit, UAS, GKVK, Bangalore and Dr. N. D. Sharma former Professor and Head, Department of Plant Pathology, JNKVV, Jabalpur, Madhya Pradesh.

We acknowledge with sincere thanks and gratitude to Dr. S. N. Shukla, Asst. Director General (FFC), ICAR, Krishi Bhavan, New Delhi for his encouragement and also for writing the foreword.

We are indebted to the Indian Council of Agriculture Research (ICAR), Krishi Bhavan, New Delhi for providing necessary financial support for the preparation of this compendium through the Project Coordinator, AICSMIP, UAS, GKVK, Bangalore.

We wish to express our deep sense of gratitude and sincere thanks to Dr K.T. Krishne Gowda, Project Coordinator, All India Coordinated Small Millets Improvement Project (AICSMIP) for his keen interest and effective interventions in inspiring us to write this compendium and for the timely publication.

A. Nagaraja,
J. Kumar,
A. K. Jain,
Y. Narasimhudu,
T. Raghuchander
Bijendra Kumar &
B. Hanumanthe gowda.

INTRODUCTION

Small millets, referred to as 'Nutri-cereals', comprise of finger millet, little millet, foxtail millet, kodo millet, barnyard millet and proso millet, and are an important group of dry land field crops. They belong to the family Poaceae (Graminae) and are mostly cultivated as rain fed crops on marginal soils under poor to neglected management practices. Besides being high quality fodder crops, owing to their very high nutritive value, they have become popular in human diet. In India, finger millet occupies 16.42 lakh ha with an annual production of 19.35 lakh tonnes whereas other minor millets (excluding finger millet) occupy 10.70 lakh ha with around 5.29 lakh tonnes production.

All these small millet crops are known to be less prone to diseases though a variety of fungal, bacterial, viral and nematode problems have been known and cited in the literature. Many of these diseases do not warrant chemical control measures and these crops besides being eco-friendly are ideally suited for 'green agriculture'. However, some of the diseases, like blast of finger millet and brown spot on majority of these crops, cause widespread losses each season. Other location specific diseases, such as green ear of foxtail millet and smuts at times cause considerable yield losses. Over the years, considerable information has been generated on various aspects of diseases affecting small millets in different parts of the country. It is thus necessary that the available information on small millet diseases is put together so as to become a ready reference to all those involved in small millet research.

The present compilation is a comprehensive account of all relevant information on small millet diseases. Some of the major diseases occurring on these crops are covered under the subtitles historical account, losses, causal agent, symptoms, epidemiological factors and latest management practices. Other diseases that are so far minor and have not been properly worked out have also been listed. Explicit symptoms of most of the diseases have been depicted to help in the field identification of the diseases. A separate chapter on pathometry has been included to help researchers working on these crops in the proper evaluation of the test entries for disease resistance. At the end, a comprehensive bibliography has been added to provide reference to detailed information available in the literature.

DISEASES OF FINGER MILLET

Finger millet [*Eleusine coracana* (L) Gaertn] commonly known as *ragi*, *mandua*, *nagli*, *kapai* and *madua* is widely cultivated extending from Tamil Nadu in the South to Uttarakhand in the North; Gujarat in the West to Orissa in the East. Its production is extended to Northeastern regions including that of Sikkim. Among the 23-24 million ha of millets area, ragi occupies about 2.7 million ha with an annual production of 2.6 million tons and productivity of around 1400 kg/ha. Karnataka state has the highest area of about 1 million ha (60% of the total), followed by Maharashtra, Orissa, Tamil Nadu, Andhra Pradesh and Uttarakhand each with 10-12% of the share. The crop is grown in different seasons in different parts of the country, mostly as a rain fed crop. The crop does not suffer much from diseases however; blast is a major production constraint at times causing heavy yield losses.

BLAST

Historical account: The disease was reported for the first time in India, from Tanjore delta of Tamil Nadu by McRae (1920). It is not only widely distributed in almost all the finger millet growing regions of the world, but also is most destructive. In India, the disease is prevalent wherever finger millet is grown and is one of the major diseases causing recurring yield losses in all the states.

Losses: In India, the disease appears every year causing an average annual yield loss of around 28 per cent. Sundaram *et al.*, (1972) in a review on the status of diseases of finger millet, considered blast as the 'number one' loss causing disease in Andhra Pradesh, Haryana, Madhya Pradesh, Maharashtra and erstwhile Mysore state.

Symptoms: The disease occurs at all the stages more so as leaf, neck and finger blast. In the nursery or in direct sown crop, the death of the germ lings is common, especially if infected seeds are used for sowing. When the young seedlings are infected, they give a burnt appearance due to severe leaf blight and death that result in to burnt patches.

Leaf blast- The disease appears on leaf lamina with typical spindle shaped spots (Figure). Under congenial conditions, such spots enlarge, coalesce and leaf blades, especially from the tip to base, give a blasted appearance. Severe leaf blast could warrant fungicidal application if the temperature and humidity are favourable. Well-developed lesions may measure 0.5 cm x 2 cm.

Neck blast- The pathogen also attacks the culms, especially at nodal region resulting in blackening of that area. The most damaging stage of the disease, however, is when the pathogen attacks at the neck region. Two to four inches of the peduncle almost immediately below the ear turns brown and later black due to fungal infection (Figure). An olive grey growth of the fungus may appear in this area. Infection at seed setting stage may result into sterility while delayed

infection may produce underdeveloped seeds. The ears hang down from the stalk at the point of infection and sometimes may break away.

Finger blast- The infection on fingers usually begins from the apical portions and runs towards the base (Figure). The extent of damage depends on the stage of infection and the weather conditions. At times, the entire length of the ear is affected. The pathogen also attacks the seeds resulting in shrivelled and blackened seeds, even on otherwise apparently healthy ears.

Pathogen: *Pyricularia grisea* (Cke.) Sacc. (Per. Stage: *Magnaporthe grisea*)

Hypha is hyaline and septate, but as the fungus grows older, the hypha becomes brown. The size of the hyphal cell ranges from 1.5–6.0 μ in length. Under high humidity, large number of conidiophores and conidia are produced giving a dirty brown colour to the lesion. Generally, the growth of the pathogen is relatively more on the upper surface, which makes the spot darker. The conidiophores may emerge either through stomatal opening or directly from the epidermal layer. Conidiophores are simple, septate, basal portion being relatively darker. Conidia produced acrogenously, one after another, are hyaline and obpyriform in shape. Conidia are three celled, the middle cell being wider and darker, and measure 19-31 μ x 10-15 μ . Generally, the end cells germinate giving out germ tubes. Formation of terminal or intercalary chlamyospores is common. They are globose, thick-walled, olive brown measuring 4-10 μ in diameter. The pathogen grows luxuriantly on oatmeal, potato dextrose, bean meal and ragi meal agar. It produces abundant dark coloured chlamyospores in culture. Under laboratory conditions, the pathogen produces fertile perithecia.

Epidemiology: The initial inoculum of the fungus generally comes from infected seeds. Intensity of the disease depends on the weather conditions. A temperature of 25-30⁰C, humidity of 90 per cent and above, cloudy days with intermittent rainfall, are favourable for the development and rapid spread of disease. If there are, continuous rains at the time of heading heavy losses to the crop occur.

Control: Two sprays of Edifenphos or Kitazin (0.1%) with first at the time of ear emergence and the second 10 days later or an initial spray of 0.05% Carbendazim followed by a spray of Mancozeb (0.2%) 10 days later are effective for the control of blast.

Seed treatment with Tricyclazole (8 g/kg seed) followed by sprays of two plant extracts of notchi and Prosopio are effective.

Two sprays of Saaf (0.2%) with first spray at 50 per cent flowering followed by the second after 10 days were found effective in reducing the blast and increasing the grain yield.

Seed treatment with *Trichoderma harzianum* and one spray of *Pseudomonas fluorescens* @ 0.3% at the time of flowering followed by second spray 10 days later can control all three blasts.

Use of blast resistant varieties, like GPU 28, GPU 26 and VL 149 coupled with Carbendazim seed treatment @ 2g/kg increases yield.

BROWN SPOT (SEEDLING BLIGHT or LEAF BLIGHT)

Historical account: Seedling blight or leaf blight of finger millet is next only to blast in terms of severity and distribution. The disease was first noticed by Butler (1918) to cause foot rot, seedling blight or leaf blight of ragi in different parts of India.

Symptoms: Infected seeds when used for sowing may not germinate at all due to pre-emergence rot of the seeds. In case such seeds germinate, post emergence rot is common. Where seeds or seedlings do not die or when healthy seedlings are subject to attack by the inoculum from outside, the characteristic symptom on the leaf lamina is the appearances of brown to dark brown spots (Figure). The pathogen infects leaves more readily from the upper surface or between the leaf and leaf-sheath. Infection takes place through the stomata, the epidermal cells or more frequently through certain epidermal outgrowths.

The spots are generally oval in shape and measure 8-10 mm in length and 1 to 1.5 mm in breadth. Often these spots coalesce giving the appearance of blighting of leaf, especially towards tip, that are ultimately killed prematurely. The fungus affects leaf sheath and culm especially at nodal joints. The area at the junction of leaf sheath and leaf blade is usually affected resulting in dark brown discolouration. Foot rot is a common symptom in a severely infected plant under favourable weather conditions.

Symptoms are also seen on leaf sheath (Figure), especially in older plants, wherein the woolly growth of the fungus can be seen in the centre of the lesion, especially under high humid conditions. Heavy infection results in premature death of the leaf. When the infection occurs on neck and fingers, often under high humid conditions, neck may break and hang on to the plant. Dark tan lesions are seen initially, which may extend up and down. The pathogen attacks ear head, fingers as well as grains. Affected grains may not develop fully and shrivel. The disease in such situation results in heavy losses in yield.

Pathogen: *Drechslera nodulosum* Berk and Curt. [Perfect Stage *Cochliobolous nodulosus* (Berk and Curt) Subram. and Jain]

The conidiophores are erect and at times curved, unbranched, produced in plenty in the older portion of the spot and rarely seen in the expanding area. Conidiophores generally emerge through stomata but it is not uncommon to see them emerging directly from epidermal cells. These may measure anywhere between 80-250 μ in length and 5-7 μ in width with many septa. Conidia are sub cylindrical or obclavate, straight or curved 3-10 septate and measure 40-114 μ X 11-21 μ . They are borne at the tip of the conidiophore either singly or one after another. As many as 11 conidia may be formed on one conidiophore. They are thick walled and light russet – green in colour.

The pathogen produces sexual fruiting bodies in culture, which possess long cylindrical beaks, black in colour and spherical in shape. These ascocarps (perithecia) measure 276-414 μ in diameter. The asci are short and straight with 1 to 8 ascospores with rounded apex. They measure 120-193 μ x 14-17 μ .

Epidemiology: The pathogen survives in unsterilized soil for over 18 months and the spores on grains are reported to be viable for a year. The optimum temperature for infection is 30- 32⁰C though the disease can occur between 10 to 37⁰C. High humidity and intermittent rains during the period of emergence of ear and before grain formation causes heavy ear infection and reduction in yield.

Control: The damage by the pathogen to the seedlings especially in nursery under warm humid conditions or to the developing seeds in the ear is quite considerable. Need based spraying of Mancozeb 0.2% control the disease.

CERCOSPORA LEAF SPOT

Historical account: Cercospora leaf spot of finger millet is one of the important foliar diseases restricted to Himalayan region in India and Nepal. Munjal *et al.*, (1961) studied the specimen collected during October 1959 by J.N. Kapoor from Kathgodam, Nainital and gave the descriptions and nomenclature of the fungus.

Losses: The disease if occurs immediately after heading, can reduce the yield up to 40 per cent and 1000 seed weight by 21 per cent (Pradhanang, 1994). However, there will be no loss in yield when the disease incidence is around 25 per cent (Pradhanang and Abington, 1993).

Symptoms: The disease occurs most severe in the month of June in the early sown crop. Infection generally starts from the older leaves and spreads to the young leaves. Thus, severity of disease decreases from older to younger leaves. Initial symptoms appear as reddish brown specks with yellow halo and are easily confused with those of Helminthosporiose or blast (Figure). Later, several such specks coalesce to form large lesions. In some cases the lesions enlarge, to assume eye shaped spots measuring 15 x 3mm and look like those of blast. Such leaves give burnt appearance. At the time of crop maturity, severely infected leaves turn completely necrotic, shrivel and dry. At this stage, the plants look completely blighted. Such symptoms can also be seen on the stem, leaf sheath and fingers. Dark brown scattered spots appear on the stem. Later, these spots coalesce to form a necrotic lesion on the stem or fingers.

Pathogen: *Cercospora eleusinis*

Conidiophores olivaceous brown, tip dilutely coloured, straight to curved, geniculate, septation at long intervals, not branched, spore scar prominent; measuring 4-5 X 27-300 μ . Conidia acicular, hyaline to sub hyaline, indistinctly multiseptate, straight to curved, base truncate, tip sub acute and measure 3-4 X 50-260 μ .

Epidemiology: The disease is generally restricted to cooler regions. In Nepal the disease is restricted to midhills where mean daily temperature does not exceed 20⁰C and the rainfall is generally high.

Control: Late sowing in the month of July decreases the disease incidence and severity. Fields should be cleaned of the infected crop refuge. Spraying the crop with Carbendazim @ 0.05% reduces infection.

DOWNY MILDEW

Historical account: This disease was reported for the first time in India by Venkatarayan (1946) on ragi from the erstwhile Mysore state. A detailed picture of symptomatology was given by Thirumalachar and Narasimhan (1949) who identified the pathogen as *Sclerospora macrospora*.

Crop loss: But for its sporadic occurrence, the disease could be destructive leading to total crop failure owing to malformation of the affected ears.

Symptoms: The white cottony growth, characteristic of many downy mildews, is generally not seen in the downy mildew of finger millet. As a result, the asexual phase quite often goes unnoticed.

Downy mildew affected plants are generally stunted with shortened internodes and profuse tillering. The leaves are crowded giving a bushy appearance to infected plants. The seedlings infected early in the season may be killed. Often, pale yellow translucent spots are seen on leaves of affected plants. The most striking feature of the disease however, is partial or complete proliferation of the spike let into leafy structures that often result into a brush like structure (Figure).

Pathogen: *Sclerophthora macrospora* (Sacc.) Thirum., Shaw and Naras.
(Syn. *Sclerospora macrospora* Sacc.)

Mycelium of the fungus is coenocytic and hyaline. Sporangiohores akin to vegetative hyphae, sympodially and successively branched, and haploid in nature. Sporangia are lemon shaped, measure 60-100 x 43-64 μ and are borne singly at the apices of sporangiophore. They germinate indirectly by releasing 24-48 zoospores, which are unequally biflagellate. Oospores are spherical 35-70 μ in diameter, with granular content. The oospore germination is indirect by the formation of a big lemon shaped sporangium, which liberates 24-48 zoospores.

Epidemiology: A temperature of 25-30⁰C is favourable for disease development. During night, when the temperature is around 22-25⁰C, many sporangia are produced which release zoospores. The pathogen is internally seed borne.

Control: Since systemic infection occurs at seedling stage as in case of many other downy mildews, seed treatment with chemicals like Apron 35 SD @ 2.5- 3.0 g/kg, would control systemic infection. Sprays may not be economical.

FOOT – ROT

Historical account: Coleman (1920) was the first in India to record the occurrence of *Sclerotium rolfsii* from the then princely state of Mysore. Subsequently it was reported from the former Madras Presidency, Coimbatore and Orissa.

Crop loss: Up to 50 per cent loss was recorded at Rampur, Nepal (Batsa and Tamang, 1983).

Symptoms: The disease appears randomly in the field. The infection occurs around the collar region, the infected area being restricted to two to three inches above ground level. Normally, at a stage when plants are flowering or setting seeds, the plants are attacked due to the debility of the stem as the movement of photosynthetic material is towards sink.

The basal portion of affected plant immediately above the ground initially appears water soaked due to infection by the pathogen. Later on it turns brown and subsequently dark brown with a concomitant shrinking of the stem in the affected region (Figure). Profuse white cottony mycelial growth occurs in this area (Figure). Soon small roundish white velvety grain like structures starts appearing in the fungal matrix. They grow, become mustard seed like, turn brown and these are the sclerotial bodies. Meanwhile the leaves lose their lustre, droop and dry. Ultimately, the plant dries up prematurely.

Aetiology: *Sclerotium rolfsii* (Sacc.) Curzi. (Per. Stage *Pellicularia rolfsii*)

The pathogen has a very wide host range and thus is present in almost all soils. At the end of the crop season, enormous sclerotial bodies are produced from the growth that had occurred on the host plant. The sclerotia find their way to soil, more through rainwater from field to field. Generally, *S. rolfsii* is a weak or an opportunist pathogen, capable of attacking the host plant when it is debilitated.

Epidemiology: Sandy loam soils favour disease incidence and the pathogen survives better at low soil moisture levels. The disease incidence is more during warm and dry months.

Control: The ideal way of managing disease could be through resistant hosts. Chemical control may not be economical as the pathogen is soil inhabitant. However, Vitavax may be effective in controlling foot rot of finger millet.

SMUT

Historical account: Kulkarni (1922) recorded this disease from Malkapur in 1918 in the then princely state of Kolhapur, described the pathogen and identified it as *Ustilago eleusine*. Later, this disease was noticed in the districts of Surat, Nasik and Ratnagiri of the then Bombay Presidency and subsequently from Mysore and Madhya Pradesh states.

Losses: Although no estimate on yield loss is available, there can be heavy loss if more number of grains are affected, besides blackening of the total produce.

Symptoms: The smut makes its appearance, generally a few days after flowering. The smutted grains can be seen scattered at random in the ear. The severity shall normally be less than or around one per cent of the grains. The affected ovaries are transformed into greenish gall like bodies, which are several times bigger in size than the normal healthy grains (Figure). In the initial stages, greenish swollen grains 2-3 mm in diameter that project beyond the glumes are evident. With the progress of the disease, the infected grains become swollen and reach a diameter of 16 mm. The greenish outer tunica of the sorus gradually

turns pinkish green and finally to dirty black on drying. Some times the affected grains are single or may be grouped into patches of varying size and are frequently confined to one side or towards the base or apex of the head and show signs of rupturing at several places.

Pathogen: *Melanopsichium eleusinis* (Kulk.) Mundk. and Thirum.
(Syn, *Ustilago eleusine* Kulk.)

The spores are globose or subglobose, auburn coloured and measure 7-11 μ with a mean of 9.5 μ . The epispore is rough and minutely pitted (Mundkur, 1939). They germinate by forming a septate promycelium producing both lateral and terminal sporidia. Promycelium first protrudes out as a small papilla, which gradually elongate into a stout germ-tube. Optimum temperature for spore germination is 25⁰c.

Control: The disease is more in late sown summer crop. Sowing in January avoids the disease.

DAMPING OFF

Historical account: The disease was reported from Annamalai in Tamil Nadu, India (Raghunathan, 1968). The disease appears in ill drained nursery/fields, especially during rainy months.

Symptoms: The symptoms of the disease are characterized by yellowish brown discolouration of the hypocotyl region at the ground level. Later on, such discolouration spreads to stems as well as roots and finally the seedlings collapse. Such seedlings when gently pulled get broken at the collar region.

The pathogen: *Pythium aphanidermatum*

The mycelium is hyaline, highly granular, coenocytic, profusely branched and measure 1.1 to 8.6 μ in width. The oospores are spherical, smooth and thin walled, measuring 14.1 to 19.2 μ in diameter. The walls of the oospore are 1.5 to 3 μ in thickness. Sporangia are terminal or intercalary, simple or digitately branched, loculate structures varying in shape and size.

Control: In case of transplanted ragi, seedlings in nursery can be protected by growing on raised beds with proper drainage. Practicing light irrigation or watering and drenching soil with suitable fungicides such as Copper oxy chloride, Captain, Hiram or Metalaxyl compounds prove effective, but expensive in disease management.

BANDED BLIGHT

Historical account: Lulu Das and Girija (1989) for the first time reported sheath blight of ragi from Vellayani in Kerala, India, where it occurred in a severe form. Subsequently, the disease was observed in a severe form in 1993 in the experimental plots of Birsa Agricultural University, Ranchi, Bihar.

Symptoms: The disease is characterized by oval to irregular light grey to dark brown lesions on the lower leaf sheath. The central portions of the lesions

subsequently turn white to straw with narrow, reddish, brown border. Such spots at later stages are distributed irregularly on leaf lamina. A temperature of around 28-30°C and a relative humidity of 70 per cent or above favours rapid disease development where these lesions enlarge rapidly and coalesce to cover large portions of the sheath and leaf lamina. At this stage, the disease symptom is characterized by a series of copper or brown colour bands across the leaves giving a very characteristic banded appearance. The mycelial growth along with white to brown sclerotia can be observed on and around the lesions. Later on, the leaves dry up and plants appear blighted.

On peduncles, fingers and glumes irregular to oval, dark brown to purplish brown necrotic lesions are formed. Early infection on peduncle or near finger base is somewhat similar to neck rot resulting in poor grain filling. If the sheath is infected before peduncle emergence, then the fingers are disorganized and reduced in size. Infected glumes produce smaller and shrivelled grains. Thus, the symptoms produced on every part of the plant give a characteristic banded appearance, due to which the disease has been named as banded blight (Dubey, 1995).

Pathogen: *Rhizoctonia solani* Kuhn. [Basidial stage: *Thanatephorus cucumeris* (Fr.) Donk.]

Dubey (1995) observed the production of perfect state in nature on diseased plants as dirty white growth of hymeneal layer during September – October, when high humidity (above 80%) and moderate temperature (26 ± 2°C) prevail.

SHEATH BLIGHT

Historical account: The first recorded occurrence of a pathogenic mushroom on finger millet causing sheath blight of ragi was reported from Coimbatore, Tamil Nadu, India in 1974 (Parambaramani *et al.*, 1975).

Symptoms: The first symptom of the disease is the appearance of characteristic circular to elliptic, necrotic patches on the sheaths about 5 to 15 cm above the ground level. As the disease progresses, the sheaths are stuck or bound together, with the mycelium of the fungus to the stem eventually leading to wilting and death of the plants. The discoloured or dried up outer leaves could easily distinguish the diseased plants in the field. On lower sheaths of the dead plants, small sporophores of the mushroom are noticed.

Pathogen: *Marasmius candidus* Bolt.

The mushroom is white, delicate, leathery and dry, does not easily decay, but shrivel up in dry weather and revive in wet weather or when placed in water.

Control: No information is available.

LEAF SPOT

History: Shaw (1921) recorded the occurrence of *Curvularia lunata* (*Acrothecium lunatum*) on many small millets including finger millet from Pusa. Later it has been reported from Madras, Kerala and Assam.

Symptoms: The fungus produces small spots on finger millet, *Setaria italica* and *Panicum frumentaceum*.

C.O. *Curvularia lunata* (Walker) Boedijin (Syn. *Acrothecium lunatum* Walker)

The conidia are curved, knee-shaped, 2-4 septate with large middle curved cell. It measures $18.22\mu \times 5.7\mu$ and grows on a large variety of media. In non-synthetic media, greater development of aerial hyphae takes place, but the spores are usually smaller, whereas in synthetic media, lesser development of aerial hyphae takes place, but the spores are bigger.

RUST

History: Occurrence of a rust on finger millet was reported only recently in 1976 from Meerut in Uttar Pradesh (Dubish and Singh, 1976). There was no further report of this disease from any part of the world until 1996 when it was reported from Bangalore, Karnataka (Channamma *et al.*, 1996).

Symptoms: The symptoms appear as small, brown, broken pustules linearly arranged on the upper surface of the top leaves. The Urediniospores are pedicellate, globoid or broadly ellipsoid with thick, smooth walls and cinnamon brown in colour. The spores measure $24\mu \times 26.25\mu$, with 3-4 germ pores approximately equatorial.

C.O. *Uromyces eragrostidis* Tracy. Other details of the disease have not been thoroughly worked out.

BACTERIAL DISEASES

Historical: The first known occurrence of bacterial disease in finger millet is probably the one that was reported by Mehta and Chakravarty (1937) from Uttar Pradesh. However, they did not identify the pathogen. Rangaswami *et al* (1961) reported a bacterial disease from Tamil Nadu, which they identified as *Xanthomonas eleusinae*. Later on Patel and Thirumalachar (1965) reported yet another species of *Xanthomonas* from Gujarat. Billimoria and Hegde (1971) reported a species of *Pseudomonas* from Karnataka.

1. Leaf Spot: A leaf spot was noticed during the rainy season of 1960 in Chidambaram taluk, South Arcot district of Tamil Nadu, which was found to be caused by a bacterium. Rangaswami *et al* (1961) who studied the disease and the causal agent reported it to be due to hitherto unknown species of *Xanthomonas*, which they named as *X. eleusinae*.

Symptoms: Linear spots are seen on both upper and lower surfaces of the leaf blade and spread along the veins. The spots measure 2 to 4 mm long, but often

extend up to one inch or more. In the beginning, spots are light yellowish brown, but soon become dark brown. In advanced stage, the leaf splits along the streak, giving a shredded appearance. All the leaves, including the tender shoots, in a plant are affected. The bacterium, mainly affects the leaves, but at times characteristic streaks may be found on the peduncle of the ear head. These streaks are narrow, 5 to 10 mm in length and appear sub-cuticular.

Pathogen: *Xanthomonas eleusinae* Rangaswami, Prasad, Eswaran

The bacterium is a short rod, $1.8 - 2.7 \times 0.8 - 1.0\mu$ with a single polar flagellum, aerobic, gram negative, non-capsulated, non-spore forming and non-acid fast. It forms dull yellow slimy and shiny colonies on nutrient agar and growth in nutrient broth is turbid with pellicle formation. Gelatine is rapidly liquefied but starch is not utilized. Litmus milk turns neutral and is coagulated. Nitrate is reduced, H_2S produced but ammonia and indole not produced. It gives positive lipolytic activity and negative MR and VP tests. It utilizes lactose as a carbon source, with acid production and little or no gas formation.

2. Bacterial Blight: Desai *et al* (1965) insisting that the bacterial pathogen reported by Rangaswami *et al* (1961) on ragi was not a *Xanthomonas* species, reported a bacterial blight disease on ragi that was widely prevalent in Gujarat.

Symptoms: The plants are susceptible to infection at all stages of growth. If infection takes place during early stages of growth, the plants become yellow and show premature wilting. Infection first appears as water soaked, translucent, linear, pale yellow to dark greenish-brown streaks, 5 to 10 mm long, and running parallel to the midrib of the lamina. The hyaline streak later develops into a broad yellowish lesion measuring 3 to 4 cm and turns brown. When the infection is heavy, particularly in the early stages, the entire leaf turns brown and withers away.

Pathogen: *Xanthomonas coracanae* Desai, Thirumalachar and Patel.

Bacterium appear as short rods with rounded ends, usually single, occasionally in pair, measuring $1.1-1.8 \times 0.5 - 0.7\mu$, motile by a polar flagellum, gram negative, encapsulated, no endospore and non-acid fast. Colonies on potato dextrose agar plates are circular with entire margin, smooth, pulvinate, butyrous and glistening yellow. Growth on nutrient agar and potato dextrose agar slants is moderate to abundant, filiform, convex, glistening, smooth opaque, butyrous and lemon yellow; medium unchanged.

3. Leaf Stripe: Billimoria and Hegde (1971) observed a disease on ragi during 1969-70, which was found to be due to bacterium. The disease appeared in serious proportion in and around Bangalore district, Karnataka, India.

Symptoms: The common symptom of the disease is brown coloration of the leaf sheath especially from base upwards. The affected portion of the lamina invariably involves the midrib and appears straw coloured. This symptom spreads to about three fourths the lamina and then abruptly stops or in some cases reaches the leaf tip. Occasionally the strips of infected areas are seen to proceed along the margin of the lamina, leaving the central portion, including the

mid rib healthy. The bacteria are readily detected in the phloem vessels. Infected plants can be recognized from distance by characteristic drooping of the leaves.

Infected culms show a light brown discolouration along one side. In some cases, this discoloration begins from the base, but in most instances, it begins two to three inches above the base and extends to the leaf sheath proper. There is, however, no apparent reduction in girth or turgidity of the affected culms as compared to the healthy ones. Plants less than a month old are usually free from the disease. The bacterium is systemic and soil borne.

Causal organism: *Pseudomonas eleusineae*

The bacterium is a short rod, found singly or in pairs, measuring 0.83 – 2 μ by 0.31 – 0.42 μ , capsulated with one or two monotrichous flagella. Gram negative, non-spore forming and non-acid fast. It strongly hydrolyses starch, produces acid but no gas from glucose, turns plain milk alkaline and shows mild lipolytic activity. The bacterium does not liquefy gelatine reduces nitrate, V.P. and Methyl red test positive. Does not produce indole and does not hydrolyse casein.

VIRUS DISEASES

Two calamities of viral disease problem occurred on finger millet in Karnataka, first in 1940's and then in 1960's causing near total loss of the crop yield. In 1982, Nagaraju and co-workers reported yet another but very distinct virus that is known to be pathogenic to finger millet. Thus, three viruses viz., Sugarcane mosaic, Maize streak and Mottle streak have been known to occur on ragi in India.

1. Ragi Mottle Streak

During mid 1960's there was a severe disease problem in epidemic proportions all through Southern Karnataka. Govindu *et al.*, (1966) studied this disease and thought it was due to combined effect of a Virus and Helminthosporium Sp. Later Mariappan *et al.*, (1973) reported a ragi streak disease from Tamil Nadu, which was also transmissible by *Sogatella* sp and they regarded this virus to be a strain of the virus reported from Karnataka.

Losses: Maramorosch *et al.* (1977) observed 50-100 per cent losses in certain areas due to mottle streak.

Symptoms: The infected plants exhibit regular dark-green areas all along the leaf veins when the plants are 4-6 weeks old. Other symptoms on leaf include chlorosis and streaking. In some cases occasional yellowing to almost albino symptoms are also observed. However, in the lower leaves, the symptoms are of mottle type in the form of white specks and the affected plants are generally stunted bearing small ears.

Pathogen: Ragi mottle streak virus

Short rod-like, bacilliform particles seen in the perinuclear position in cells from all parts examined, including the epidermis mesophyll, and the conducting element. The particles were very abundant and therefore easily detected. The particles measure 80 nm in cross section and 285 nm lengthwise. The particles were enveloped, bacilliform and spiked corresponding to the morphology of rhabdoviruses.

The virus is transmitted by two species of jassids viz., *Cicadulina bipunctella* and *C. china*, *C. bipunctella* was able to transmit up to 82 per cent. The minimum acquisition-feeding period is 48 h and the minimum inoculation-feeding period is 24 h. The virus can persist in the insect for 8 days. Only a section of the population of the vector *C. bipunctella* transmitted the virus in high percentage and the virus is carried in the leafhopper in a persistent manner

Epidemiology: The adverse climatic conditions of high maximum – minimum temperatures, relatively low rainfall and less average relative humidity in all the affected districts from August to November during 1945 and 1965 were thought to have led to enormous increase in vector population.

2. Ragi Severe Mosaic

Historical: The ragi crop in Southern Karnataka and the border districts of Andhra Pradesh was affected by a severe mosaic in *kharif* 1966. In certain pockets like Hiriyur Taluk in Chitradurga district and Devanahally taluk of Bangalore district the disease was so severe that the farmers abandoned their crop as heavily diseased plants failed to set seed (Joshi *et al.*, 1966). The epidemic that occurred in 1960's was mainly due to continuous cropping of ragi, coupled with abnormal weather factors, which had favourable impact on vector population (Keshavamurthy and Yaraguntaiah, 1977).

Symptoms: The virus induces mosaic symptoms, which are more clear and pronounced on young leaves. Infected plants remain stunted and the ears of severely affected plants malformed. Such plants produce few seeds of smaller size, which reduces the yield considerably. In addition, the affected plants appear pale yellow due to severe chlorosis and in severe cases become brownish-white. Thus, the entire field appears yellow and can be readily distinguished from non-infected stands from a distance. Stunted plants do not recover, develop roots at nodes, generally do not produce ears and if produced remain mostly sterile.

Subbayya and Raychaudhuri (1970) observed severe mottling, chlorotic streak, general chlorosis and yellowing of leaves. They also observed profuse lateral shoots and aerial adventitious roots, stunting of plants, fewer flower and poor seed formation.

Pathogen: *Sugarcane mosaic virus*

The virus has a thermal inactivation point of 50-55⁰C, dilution end point between 1:500 to 1:750 and longevity *in vitro* of 10-12 h at room temperature (30-36⁰C), 30 h at 24-26⁰C and 5 days at 7-10⁰C. It withstands continuous freezing for 8 days and desiccation for 34 h in infected leaves. Particles were flexuous rods with an average length of 667 ± 8m μ and an approximate diameter of 12-14

mp. The virus, thus, was identified as a strain of sugarcane mosaic virus (Subbayya and Raychaudhuri, 1970).

The virus is neither seed-borne nor soil-borne. Paul Khurana *et al.*, (1973) studied the virus-vector relationship employing *Longiunguis sacchari* as vector. The optimum acquisition-feeding period was found to be 5 minutes and optimum transmission feeding period was found to be one hour.

The aphid acquires the virus in one minute. Pre-acquisition fasting increases the efficiency of the vector and the maximum transmission is obtained within 1^{1/2} to 2 h fasting. Even a single aphid transmits the virus and the optimum is 10 aphids per plant. Post-acquisition fasting decreases the efficiency of the vector and the virus is found to be non-persistent in *R. maidis* since it was retained only for one hour after acquisition. The incubation period of the virus is found to be influenced by temperature but not by the age of the host. Ragi plants of all ages are susceptible but the severity of infection decreases significantly with increase in the age of the host. (Subbayya and Raychaudhuri, 1970).

3. Ragi Streak

Historical: During the year 1974-75, a virus disease producing streaking and yellowing of leaves and stunting of ragi plants in the fields around Bangalore was observed and the virus was found not to be transmitted either through mechanical sap inoculation or aphid species tested (Anon, 1975). Ragi plants affected with such symptoms were noticed in a wider geographical area viz., in the districts of Chitradurga, Mandya, Bangalore, Tumkur and Hassan in the subsequent surveys during 1977-78 and 1978 –79. The incidence ranged from 5 to 45 per cent. Subsequent studies revealed this virus to be different from all those reported earlier on finger millet and was found to be a strain of maize streak virus,

Losses: The loss in grain yield depends on the age at which the virus infects the crop. Similarly, the virus infection results in a drastic reduction in 1000-grain weight depending upon the stage of plant infection. The loss in 1000-grain weight was 84, 63, 27 and 24 per cent when the infection occurred at 30, 40, 50 and 60 days old seedlings. However, there was no significant change in number of tillers except where infection occurred on 10 days old seedlings when there was a significant increase in seedling number (Nagaraju, *et al.*, 1982).

Symptoms: Symptoms appear on unfolding young leaves as pale specks or stripes of different size. The specks coalesce involving larger areas resulting in chlorotic bands running almost the entire length of the leaf parallel to the midrib. These bands are occasionally interrupted by dark green areas. The new emerging leaves of both the main shoot and the tillers show number of well defined chlorotic streaks having almost uniform width running parallel to the midrib throughout the length of the leaf lamina.

The infected plants in the field produce comparatively more number of tillers and bear yellowish sickly ear heads, often bearing few shrivelled seeds. The plants infected very early in the crop growth stage die before they bloom.

Pathogen: *Eleusine* strain of maize streak virus

Cicadulina chinai is able to transmit the disease. The viruliferous leafhopper remains infective throughout its life period. The minimum inoculation-feeding period is 30 minutes. Single viruliferous leafhopper is able to transmit the virus.

NEMATODE PARASITES

Several nematodes viz., *Helicotylenchus*, *Trichodorus*, *Pratylenchus*, *Rotylenchulus*, *Heterodera*, *Criconemoides*, *Macroposthonia*, *Meloidogyne* have been reported to parasitize finger millet.

1. *Heterodera* spp.

During 1972, Occurrence of a cyst nematode was recorded at Hebbal, Bangalore, India by Setty (1975) for the first time on ragi. However, earlier, *Heterodera marioni* was reported on this crop (Ayyar 1933, 1934).

The main symptoms are the stunting of plants and yellowing of leaves in patches. The affected plants show unthrifty growth even under optimum conditions of moisture and nutrition, and can easily be pulled out. The cysts embedded or attached to the roots of the affected plants can be seen by naked eye.

C.O. *Heterodera gambiensis*

The cysts are lemon shaped, measure 800- 960 μ X 450 -600 μ while the second stage larvae are 400-540 μ long.

2. *Rotylenchulus reniformis* Linform and Oliveira

Chandrasekaran (1964) in his survey found finger millet to be susceptible to *R. reniformis*. Increased populations of this nematode had positive correlations with the reduction in height of plants, top weight, root weight and yield. Rajagopal (1965) found the association of high population of *R. reniformis* with stunted grassy patches of finger millet.

According to Krishnappa *et al.* (2002), 4.8 per cent of cropped area to ragi is affected by *Rotylenchulus reniformis* in Karnataka, and green manuring was highly effective in reducing nematode population.

In addition to *Heterodera* and *Rotylenchulus*, Narayana Swamy and Govindu (1966) reported the natural occurrence of *Helicotylenchus*, *Trichodorus* and *Pratylenchus* species from several places in Karnataka. Mohanty and Das (1976) studied the physiology of parasitism of *Criconemoides ornatus*, the ring nematode in ragi.

Control: The nematodes are best controlled by soil amendments like poultry manure or neemcake or by applying granular insecticides viz., Phorate 10G or Carbofuron 3G.

DISEASES OF KODO MILLET

Kodo millet (*Paspalum scrobiculatum* L.) is one of the small seeded grain cereal crop predominantly grown in India and West African countries by tribal and poor people in the lands of poor fertility status with no or low cash inputs. In India, the crop is grown over 0.7 million ha with a large area in the states of Madhya Pradesh, Chhattisgarh, Maharashtra, Uttar Pradesh, Gujarat and Tamil Nadu. Colloquially known as *kodo*, *varahu*, *haraka* and *arikal*, the crop has rich medicinal values and provides protein, fibre, carbohydrate and minerals required for the body growth. A number of diseases like head smut, ergot, rust, kodo poisoning etc. have been reported in kodo millet, which results in quantitative and qualitative loss.

HEAD SMUT

Historical account: Head smut of kodo millet was first reported from Queensland, Australia (McAlpine, 1910). Subsequently, the disease has also been reported in Eastern and Southern Asia. In India, the disease is reported from Chamta ghat and Monghyer, Bihar, Madhya Pradesh, Karnataka, Andhra Pradesh and Tamil Nadu. At present, the disease is endemic in all the kodo millet growing areas of the country; its occurrence and severity vary with the place, environment and host variety.

Crop loss: Butler (1918) recorded heavy losses in grain yield due to head smut in kodo millet. Viswanath (1992) reported 30 to 40 per cent loss in grain yield due to the disease. Jain and Yadava (1997) revealed that losses in yield increase linearly with increase in disease incidence and estimated a loss of 13.15 to 32.98 per cent at 13.95 to 40.15 per cent smut incidence in few varieties.

Symptoms: Characteristic symptoms of the disease appear as the crop approaches to flowering. Infected plants are stunted and almost all the panicles in the infected plants are converted into a long sorus ranging from 2.1 to 14.6 cm long and 0.1 to 0.6 cm broad (Figure). In early stage, the entire sorus remains surrounded by a creamy membrane. Some times, the sorus remains enclosed in the boot leaf and does not emerge fully. The sori destroy the whole inflorescence except the fibro-vascular bundles, the rachis of which persists in the form of a bundle of fibres. At maturity, the membrane of the sorus bursts and exposes the black mass of spores. Ahmed (1991) also observed necrotic streaks on the boot leaf covering infected panicles.

Causal organism: *Sorosporium paspali-thunbergii* (Henn.) Ito (Syn. *Sorosporium paspali* McAlp

Teliospores of the fungus are borne in loose spore ball like masses of 60 x 30 μ in diameter and disintegrate into individual spores on getting little pressure. Individual spores are sub-globose, angular to roughly pear shaped, dark to yellowish brown with thick smooth wall and measure 11-18 x 8-12 μ in diameter. Teliospores remain viable for seven months.

Disease cycle: Head smut of kodo millet is both seed and soil borne. For getting uniform infection, seedling inoculation with viable sporidial suspension resulted

into maximum disease. Mixing of seed with viable teliospores @ 2-3 g per kg seed before sowing also produces significantly higher smut incidence. Seedling infection takes place by penetration of germ tube through the cell wall. After entering in the seedling, the mycelium spreads inter and intra cellular in the host tissues and the fungus becomes systemic. It enters the meristematic tissues and finally infects the ear head. No sign of hypertrophy or hyperplasia of infected cells was observed. Hyphae are established in all the infected tissues except xylem vessels (Ahmed, 1991).

Control: As the disease is seed and soil borne, seed treatment with chemicals such as Carboxin, Carbendazim, Mancozeb, Chlorothalonil and Thiram were found effective in controlling the disease.

ERGOT OR SUGARY DISEASE

Historical account: Ergot infection in kodo millet was first recorded from Burma (Butler and Bisby, 1931). In India, the disease was first reported from Kodaikanal in South, Assam in the North and Gwalior in Madhya Pradesh (Ramakrishnan and Sundaram, 1950). Later on, the disease was also reported from Italy (Grasso, 1952). The disease is prevalent in the wild as well as cultivated forms of *Paspalum scrobiculatum*. The sphaelial stage is not harmful, but mature sclerotia cause paralysis and death in cattle, horses and sheep. The alkaloids present in the sclerotia have no therapeutic value.

Crop loss: The disease directly reduces the grain yield by replacing grain with sclerotia of the fungus and lowering quality of produce by contaminating grains with sclerotia, but no authentic report on crop loss is available.

Symptoms: The characteristic symptoms of the disease appear only after emergence of panicle. Few or all flowers in an inflorescence may be infected. The most obvious external sign of the disease is the exudation of a thin to viscous, sweet, sticky fluid from the infected flowers that gives the name sugary or honeydew disease and overflow over the lemma and palea. The droplets are soon hardened into reddish brown crusts. A plectenchymatous mass of fungal growth replaces the ovary. As the panicle mature, dark grey sclerotia or ergot replace the kernels and this is the most conspicuous symptom of the disease.

Causal organism: *Claviceps paspali* Stev. & Hall

A large number of conidia are present in the honeydew. These are formed at the tips of closely arranged conidiophores produced on the surface of the plectenchymatous mass. Conidia are hyaline, oblong, granular or gullulate measuring 15 x 5 μm (9 – 18 x 3 – 6 μm). Conidia germinate within three hours and secondary conidia are formed from the tips of the germ tubes, which are smaller measuring 8 x 4 μm (6 – 12 x 3 – 6 μm). The hard structures known as sclerotia project out between the palea and lemma are dark, oblong measuring 1.5 – 2.5 mm x 1 – 2 mm. Germination of the sclerotia from *Paspalum scrobiculatum* has not been observed, but the developmental stages of the strains on other species of *Paspalum* have been studied in detail. On germination, a number of pinhead like stromatoid structures bearing numerous

sunken perithecia in the terminal swollen portion are produced from each sclerotium. The asci are hyaline, cylindrical, clavate measuring 130 x 33 μm (90 – 180 x 2 – 5.4 μm). The ascospores are hyaline, slender and measure 85 - 145 x 0.5 – 1 μm (101 x 0.5 – 1 μm) are produced in each ascus. Air borne conidia or ascospores infect the spike lets at an early stage.

Control: Rouging of infected panicles and eradication of the collateral hosts can minimize the primary inoculum and no other control measure has been tried so far.

RUST

Historical account: The disease was first recorded on kodo millet in India from Himalayan hills at Kanaighat (Sylhet) and Kumaon hills. Afterward the disease was also recorded from Coimbatore in India and Ceylon.

Crop loss: No authentic report is available.

Symptoms: The erumpent, oval, brown uredia are formed on the upper surface of the leaf blade and on the leaf sheath. The brown coloured telia are formed on the under surface of the leaf blade and on the leaf sheath during December – February in Coimbatore condition. Telia remain covered by the epidermis for a long time. Under sheltered conditions, uredia are present throughout the year on the grass hosts and from where it can pass readily on the cultivated kodo millet.

Causal organism: *Puccinia substriata* Ellis & Barth. (Syn. *Uredo paspalis-scrobiculati* Sid.)

The uredospores develop in uredia and are sub-globose to elliptic, stalked, single celled, finely echinulated, light brown coloured, measure 31 x 25 μm (28 - 34 x 22 – 31 μm) with four equatorial germ pores. Paraphyses have been observed in the Uredia. Teliospores are two celled, oblong, rounded at both ends and with smooth uniform thick wall. Spores measure 40 x 22 μm (28 – 47 x 19 – 28 μm). The pedicels are short and coloured. Mesospores measuring 25–37 x 15 – 25 μm are common. Marginal groups of paraphyses are also present in the telia.

The uredospores germinate readily in water drops within three hours by producing one or more stout germ tubes. The incubation period ranges from 8 to 12 days. The rust readily infects the grass as well as cultivated hosts. The Teliospores have no resting period and germination take place in 48 hours.

Control: Eradication of the grass hosts is partly useful in control of the disease, but no work has been carried out so far in this disease on control aspect.

UDBATTA DISEASE

Historical account: in India, Butler and Bisby (1931) first reported the disease. Subsequently it was recorded from Koraput and Kalahandi in Orissa and Jabalpur, Madhya Pradesh during 1966 and 1974 respectively.

Crop loss: There is no authentic study regarding crop loss assessment.

Symptoms: The affected panicles are transformed into a compact agarbatti like shape, hence the name “Udbatta”.

Causal organism: Butler and Bisby (1931) recorded *Ephelis japonica* P. Henn. However, *Ephelis oryzae* Syd. (Teleomorph: *Balansia oryzae-sativae* Hoshioka) was also recorded on the same host by Mohanty and Mohanty (1957) from India.

Control: The information about the management of Udbatta disease in kodo millet is meagre. Pall and Nema (1976) screened fifty genotypes of kodo millet at Jabalpur (Madhya Pradesh) under natural conditions and recorded IPS 45, 196, 342, 381, 365, 368, 387, 140 and Niwas 1 as highly resistant.

Management practices which are helpful in minimising the disease viz., removal and burning of affected panicles, keeping bunds free from graminaceous weeds that serve as collateral hosts and pre-sowing seed treatment with carbendazim @ 4g kg⁻¹ seed may be followed.

KODO MILLET POISONING

Historical account: The grains produced in certain seasons cause giddiness, vomiting, unconsciousness, difficulty in swallowing and rarely death of humans and cattle after their consumption as food. Even cattle employed in threshing of such ears are known to exhibit uncoordinated movements. It is also known as Matona in North India and *Kiruku Varagu* in South India. Instances of stock poisoning have also been reported. This phenomenon is found only in ears produced during rainy weather or if premature grains are harvested.

Symptoms: The affected plant parts like leaf, stem, ears and grains show a whitish fungal growth during reproductive stage of the crop. Initially, small patches of fungus are seen, which grow very fast in humid environment or just after rains.

Causal organism: Kodo millet poisoning is supposed to be caused by a number of fungal species. Bhide and Aimen (1959) reported that glumes, lemma and palea contained poisonous alkaloids. *Phomopsis paspali* was isolated from kodo millet ear heads in the western ghats of Maharashtra (Pendse, 1974). Culture of the fungus contains two toxins named Paspalin P I and Paspalin P II (Pendse, 1974 and Deshmukh *et al.*, 1975). *Aspergillus flavus* (Lalitha Rao and Husain, 1985) and *A. tamari* (Lalitha Rao and Husain, 1985) were also found associated with kodo millet grains. Both the fungi produces cyclopiazonic acid (CPA) (Lalitha Rao and Husain, 1985). In addition to CPA, *A. flavus* also produces Aflatoxin B 1 and all these toxins are believed to be responsible for the kodo millet poisoning. However, systematic work on causal agents, metabolites produced and their reaction need attention.

Management: The fungus spreads rapidly in humid environment. As the early harvest remain in the field for drying, which receives rains at times, thus, harvested heaps should be protected from rains. Traditional practice of threshing by moistening the plants before threshing should be avoided and only dried harvest should be threshed. Unripe or premature grains should not be harvested.

Storage of premature crop harvest and its storage as cylindrical heap before threshing also make the grain / straw mouldy. The winnowing of infected grains also helps in reducing the spread of the disease. In case of kodo millet poisoning use of some antidotes like juice of banana stem, the astringent juice of the guava or the leaves of *Nyctanthus arbortristis*, 'tamarind' water or butter milk are in common practice locally.

OTHER FUNGAL DISEASES

A large number of leaf spot diseases incited by graminicolous *Drechslera* Ito (with sympodial and indeterminate conidiophore) have been reported under the generic name *Helminthosporium* Link ex Fries (with determinate conidiophore and conidia are obclavate developing laterally often in verticils). Various species involved are given below with their present nomenclature status:

Drechslera state of *Trichometasphaeria holmii* Luttrell = *D. holmii* (Luttrell) Subram. & Jain (*Helminthosporium holmii*) causes leaf spots. *Drechslera* state of *Trichometasphaeria turcica* Luttrell (*Helminthosporium turcicum* Pass.) is the cause of leaf blight disease. *Drechslera victoriae* (Meehan & Murphy) Subram. & Jain causing leaf spot on kodo millet from Bagha, Champaran, Bihar, India. *Alternaria alternata* (Fr.) Keissler causing leaf blight. *Dimmersporium* (= *Asterina*) *erysiphoides* Ell. & Ev. causing leaf spot from Bassein, Maharashtra, India (Uppal, *et al*, 1935). *Macrophomina phaseolina* (Tassi) Goid reported to cause root rot. *Rhizoctonia solani* Kuhn, the incitant of sheath blight disease of rice also infects the *Paspalum scrobiculatum* and produces symptoms. *Uredo paspali-scrobiculati* Syd. Leaf rust from Kumaon, U.P., India. *Ustilago rabenhorstiana* Kuehn, infects inflorescence from Shilong, Meghalaya, India.

BACTERIAL DISEASES

1. Bacterial Leaf Streak

Historical account: In India, bacterial leaf streak of kodo millet was first reported from Jabalpur, Madhya Pradesh (Nema *et al*, 1978). So far, this was not noticed in other kodo millet growing areas of the country.

Crop loss: Quantitative yield losses have been reported.

Symptoms: The disease is characterized by pale yellow streaks measuring 0.5 to 1.0 mm that run parallel to the veins of leaf. Later, the streaks enlarge to 1.0 – 1.5 mm x 3 – 4 cm lesions, which ultimately turn brown. In severe infection, the entire leaf turns brown and withers away. The leaves may be shredded along the length. Streaks may also form on shoots and peduncle of panicles (Nema *et al*, 1978).

Causal organism: *Xanthomonas* species

Control: Varieties JK 41, JK 62, CO 2, CO 3, T 1 and IPS 14 have been reported to be resistant to bacterial leaf streak. Two foliar sprays of Streptomycin sulphate @ 300 ppm, first just after the appearance of the disease and

subsequently after an interval of 15 days has been reported to be effective against the disease along with 2.5 fold increase in yield (Pall, 1983).

2. Other bacterial diseases

Xanthomonas campestris (Pam.) Dowson f. *oryzae* Uyeda & Ishiyama has been reported as epiphytic on leaf of kodo millet from Cuttack and may cause leaf blight.

NEMATODE DISEASES

Meloidogyne incognita (Kofoid & White) Chitwood produces galls of different size in *Paspalum scrobiculatum* and has been reported from Aligarh (U.P.) as new host (Alam *et al*, 1973).

Paspalum scrobiculatum was reported a good host for *Tylenchorhynchus vulgaris* as a new host record.

PHANEROGAMIC PARTIAL ROOT PARASITE

Historical account: Witch weed (*Striga* spp.) has a very wide ecological range and have been found associated with the kodo millet plants. In India, it is reported from Andhra Pradesh, Karnataka and Madhya Pradesh. In a field survey programme, average incidence of *Striga* species varied from 1.6 to 2.0 per cent with 66.7 to 100.0 per cent frequency of incidence recorded in Rewa, Satna and Sidhi districts of Madhya Pradesh during the years 2001 and 2003.

Crop loss: Losses in grain yield due to infestation of *Striga* species depend primarily on the number of *Striga* plants attacking the crop and level of host resistance. Jain and Tripathi (2005) reported 42.4 to 65.8 per cent loss in grain yield per plant due to infestation of *Striga densiflora* in kodo millet.

Symptoms: The infestation of *Striga* species appears in the field after emergence of *Striga* plants from the soil. The under ground portion of *Striga* plant remain attached to the roots of host plant by haustoria, from which the parasite absorb water and nutrients. The attacked plants are stunted with poor aerial growth and bear lanky panicles. If the infestation occurs in early stage, the plants may dry up before the flowering (Yadava and Jain, 2006).

Causal organism: *Striga asiatica* (L) Kuntze and *S. densiflora* Benth. were found associated with the roots of kodo millet.

Control: Weeding or hand pulling of *Striga* plants before flowering is the cheapest and effective method for its eradication (Yadava and Jain, 2006). Improved kodo millet varieties viz. JK 41, GPUK 1, GPUK 3 and GPUK 5 were found least affected with *Striga* species (Reddy and Dastagiraiiah, 1987 and Jain and Tripathi, 2002). Application of nitrogenous fertilizers also reduces the infestation of *Striga* species (Pesch *et al*, 1983).

DISEASES OF FOXTAIL MILLET

Foxtail millet [*Setaria italica* (L.) Beauv.] also known as, German, Italian, Siberian millet is one of the oldest crops cultivated for hay, pasture and grains. It is called by different colloquial names as *kangni*, *navane*, *tenai*, *korra* and *rata*. At present, its cultivation is confined to semi arid regions in the states of Andhra Pradesh, Karnataka, Chattisgarh and concentrated pockets in Tamil Nadu. Although no major diseases, a few diseases like blast, rust, smut, brown spot, downy mildew and Udbatta have been reported on this crop

BLAST

Historical account: The disease was reported from Tamil Nadu in 1919, though Nishikado recorded this disease from Japan in 1917.

Crop loss: If the disease occurs in severe form, the grain loss could be around 30-40 per cent.

Symptoms: Plants up to 40 days old are highly susceptible to blast. On leaves symptoms develop from a small pin head water soaked yellowish dot that turns within 2 – 3 days to circular or oval shaped spot with greyish centre surrounded by dark brown margin (Figure). The spots measure 2-5 mm in diameter. The spots coalesce and make the leaves to dry up. When the node is affected, it turns black and breaks at the nodal junction. Lower leaves have to be examined for the symptoms of the disease. The disease starts from lower to upper leaves.

Causal organism: *Pyricularia setariae* Nisikado.

The conidiophores emerge through epidermal cells or stomata. Several conidia are formed one after another from each conidiophore. They are sub hyaline, 3 celled obpyriform and measure 19-30 μ . X 9-15 μ . Germ tubes are formed from the end cells on germination. Thick walled, brown, globose chlamydospores are developed at the tips of the germ tubes. The fungus grows well on agar media and host leaf extract. The fungus is seed borne and to some extent soil borne (Palaniswamy *et al.*, 1970), infecting finger millet, pearl millet and wheat also.

Epidemiological requirements: The factors that influence blast epidemics are, susceptible variety, availability of inoculum to initiate the disease, excessive application of nitrogen fertilizer, cloudy and drizzling weather or dew resulting in continuous leaf wetness for more than 10 h, night temperature between 15-24^oC and relative humidity above 90 percent.

Control: Growing blast tolerant varieties, avoiding excess nitrogen and destroying the weeds are best measures. When initial blast spots are seen immediate spraying with effective fungicides like Carbendazim 50 WP @ 1g /1l or Ediphenphos 50 EC @ 1ml /1l or combination product of Carbendazim + Mancozeb @ 1g/1l of water has to be resorted to intercept further development of

the disease. Top dressing of nitrogen has to be taken up after the fungicidal spray.

DOWNY MILDEW OR GREEN EAR

Distribution: Downy mildew or green ear of *Setaria* has been reported from Japan, China, Russia, Manchuria, the south-eastern countries of Europe, America, and India. In India, it is prevalent in Maharashtra, Tamil Nadu, Bihar, Karnataka, Andhra Pradesh and Kashmir.

Crop loss: It causes loss up to 50 per cent in certain years. Takasugi and Akaisahi (1933) reported that the losses go up to 20 per cent in Manchuria.

Symptoms: The primary infection starts with the seedling as systemic and secondary infection occurs on older plants, which are local. Primary infection causes chlorosis of the plant and the leaves turn whitish. The terminal spindle fails to unroll, becomes chlorotic and later turns brown and is shredded (Figure). Whitish bloom of sporangiophores and sporangia develop on the surface of the affected leaves under humid conditions (Figure). The affected plant rarely comes to flower. When the infection is mild, the plants may develop ear heads, but the floral parts are proliferated in to green leafy structures (Figure), hence the name "green ear". In a spike let all, the parts are converted in to green leafy structures of variable size. Some times only a portion of the ear head may be affected; the remaining may be normal and produce grains. Secondary infection causes chlorotic lesions on the younger leaves on which downy fungal growth may be seen under humid conditions.

Causal organism: *Sclerospora graminicola* (Sacc.) Schroet.

The fungus is an obligate parasite. Primary infection is mainly from soil-or seed borne oospores. The sporangia are produced on the host and spread by wind throughout the field causing secondary infection when favourable conditions prevail. The mycelium can be seen in the roots, stem, leaves and the modified ears. The hyphae are hyaline, non-septate and inter-cellular. Haustoria are sent in to the cells of the host tissues. Sporangiophores emerge through the stomata. Their size is governed by temperature and humidity. The sporangia are hyaline and thin walled, elliptical, with a distinct point at the free end. Oospores are produced in the proliferated structures and in the mesophyll tissue of the affected leaves. The oospores remain viable for 10 years.

Epidemiological requirements: The intensity of infection is influenced by the temperature and moisture content of the soil. The optimum soil temperature for infection is 20-21°C, the minimum temperature is 12-13°C and the maximum temperature is 30°C. Intensity of infection is also influenced by time of sowing. The intensity will be high in the early sown crop than late sown crop. High relative humidity and high soil moisture content favour severe disease incidence.

Control: Growing resistant varieties, collection and removal of infected plant debris re ideal. Seed treatment with Ridomil MZ 72WP @ 3 g/ Kg⁻¹ of seed helps in eliminating seed borne infection and protects the young seedlings from infection by the soil-borne inoculum.

RUST

Historical account: The disease was first recorded by Yoshino from Japan. It is very common on *Setaria* and has been observed in Europe, Asia and Africa. In India, it is prevalent in the states of Maharashtra, Uttar Pradesh, Madhya Pradesh, Tamil Nadu, Karnataka, Andhra Pradesh and Bihar. During certain years, it becomes epiphytotic and causes extensive reduction in grain yield. In 1944, it appeared in a very severe form in Andhra Pradesh and Karnataka states.

Crop loss: The disease affects the crop at all stages, however, the crop damage is more when infection occurs before flowering.

Symptoms: Numerous minute brown uredosori appear on both sides of the leaf (Figure). Rust pustules are oblong, brown, often formed in linear rows (Figure). They are also produced on the leaf sheaths, culms and stems. If the infection is severe premature drying of leaves and poor grain set are observed. If the infection commences before flowering the damage will be more.

Causal organism: *Uromyces setariae*

The fungus has both uredial and telial stages. Uredospores measure $26 \times 20 \mu$. ($20-34 \mu$. $\times 14-24 \mu$.) and germinate producing one or more germ tubes capable of infecting the host. The telia are formed on the leaf blade, leaf sheath and stem and are larger in size than uredia. They measure $22 \times 17 \mu$. ($17-26 \mu$. $\times 14-20 \mu$.) The Uredospores are pedicellate, oval or globose, echinulate, yellowish brown having 3-4 pores. The teliospores are single celled, pedicellate, oblong, globose or polygonal, yellowish brown with a smooth thick wall, which is much thicker at the apex than at the base. The uredospores infect the host and produce uredia in 7-10 days. The fungus can perpetuate in uredial stage on collateral hosts. The rust appears within 20-25 days of sowing and the intensity increases as the plants grow older. The telia appear at the time of maturity of the crop.

Epidemiological requirements: Low temperature and high relative humidity are favourable for severity of the disease. During December and January months, the intensity of the disease will be high, and cause extensive reduction in grain yield.

Control: Growing resistant varieties, removal of collateral hosts and sprays of Mancozeb @ 2.5 g/ l of water, immediately after observing the symptoms are some measures.

SMUT

Distribution: The disease is common in India, China, Europe and Manchuria. In India, it has been reported from Karnataka, Andhra Pradesh, Tamil Nadu and Maharashtra.

Crop loss: The disease causes heavy losses to the crop. In Romania, the damage caused by the disease is very high. In China, the loss caused by the

disease varies from 8-50 per cent of the grain yield and in Manchuria, the loss reported is up to 50 per cent. Sundararaman (1921) reported 75 per cent infection in grains of *Setaria*.

Symptoms: The fungus affects most of the grains in an ear, some times terminal portion of the spike may escape (Figure). The sori are seen in the flowers and basal parts of palea. The sori are pale greyish in colour and measure 2-4 mm in diameter. When the crop matures, the sori rupture and produce dark powdery mass of spores.

Causal organism: *Ustilago crameri* Korn.

The spores are dark brown and angular or round in shape and smooth walled measuring 7-10 μ . in diameter. The fungus is externally seed borne. Soil-borne infection has also been observed. The invading hyphae are systemic mainly concentrated towards the apical portions and at the time of flowering replace the ovaries producing septate hyphae, which are transformed in to chlamydospores.

Epidemiological requirements: Low temperature and high relative humidity are favourable for disease incidence in severe form.

Control: Growing resistant varieties, removal of infected ears and seed treatment with Carbendazim @ 2g/kg⁻¹ of seed should be done.

LEAF SPOT OR BLOTCH

Historical account: The disease was first recorded in Japan and Formosa in the year 1906. Later, the disease appeared in severe form in New Jersey (Haenseler, 1941).

Crop loss: During favourable weather, the disease causes considerable loss in grain yield.

Symptoms: Brown leaf spots appear, which enlarge, coalesce and cover the entire leaf blade. Finally, the leaves dry up. Lesions also appear as blotches.

Causal organism: *Cochliobolus setariae* (Ito and Kuribayashi) Drechsler ex Dastur. [*Helminthosporium setariae* Sawada]

The conidiophores are simple, erect, cylindrical brown, slightly swollen at the base and geniculate at the apex. They measure 72-199 μ X 5-6 – 9 μ .. The conidia are acrogenous ellipsoid to obclavate fusoid, straight or slightly curved, pale to moderately dark brown and thin walled. The spores measure 39-120 μ . X10-18 μ . Four to ten septa are present and there is no constriction at the septum.

Epidemiological requirements: Cold weather is favourable for the incidence of disease in severe form.

UDBATTA DISEASE

The affected panicles are transformed into a compact agarbatti like shape, hence the name “Udbatta”.

Causal organism: *Ephelis oryzae* Syd. (Teleomorph: *Balansia oryzae-sativae* Hoshioka).

BACTERIAL DISEASES

1. Bacterial Blight or Bacterial spot: This disease has been reported from the USA. The disease is seen as small, greyish-green spots with brown margin or light to dark brown spots on the leaf blade and leaf sheath.

Causal organism: *Pseudomonas albopreceptans* Rosen

The bacterium is a rod measuring 0.6-1.8 μ , occur singly or in pairs, no spores are formed, capsulated, motile with one polar flagellum, Gram negative, aerobic, optimum temperature 30-35 $^{\circ}$ C with minimum and maximum at 0 and 40 $^{\circ}$ C respectively. Thermal death point 41-43 $^{\circ}$ C. Other details not available.

2. Bacterial brown stripe: The disease, which is prevalent in Japan and Formosa, is seen as long, narrow, dark brown streaks on the leaf blade. Centrally shoot is usually involved in a top rot accompanied by foetid smell.

Causal organism: *Pseudomonas setariae* (Okabe) Savul.
[Syn. *Bacterium setariae*]

The bacterium is rod shaped with rounded ends, occurs singly or in pairs or as short chains. Measures 1.8-4.4 μ x 0.4-0.8 μ , motile with one or two flagella, Gram negative, aerobic, optimum temperature 31-34 $^{\circ}$ C with minimum and maximum at 5 and 42 $^{\circ}$ C respectively. Thermal death point 55-56 $^{\circ}$ C.

DISEASES OF LITTLE MILLET

Little millet (*Panicum sumatrense* Roth ex Roemer and Schultes), locally known as *kutki*, *samai*, *samalu* and *same* is cultivated through out India in more than 0.5 million ha under extreme climatic conditions of tribal agriculture. Its cultivation is in the states of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, Jharkhand, Madhya Pradesh, Orisssa and Gujarat. The crop is hardy and can with stand both water logging and drought conditions. Grain smut, rust and Udbatta are important fungal diseases that cause considerable loss in some years. Few phytonematodes also infect the crop but bacterial and viral pathogens are not reported.

GRAIN SMUT

Historical account: The disease is quite prevalent in early maturing genotypes of little millet. In India, the disease was first reported by Sharma and Khare (1987) from Dindori district of Madhya Pradesh. Later on, the disease was also reported from other parts of the country in sporadic form.

Crop loss: Studies on yield losses is very limited. Sharma and Khare (1987) noticed up to 50 per cent plants/ grains affected by the pathogen. Jain *et al* (2006) reported 9.8 to 53.5 per cent reduction in grain yield per plant, 4.2 to 16.6 per cent in plant height and 6.4 to 38.9 per cent in panicle length.

Symptoms: Symptoms are visible at grain formation stage. The affected ovary is converted into smut sorus, but does not increase in size than the normal grain (Figure). The glumes are pushed apart by the transformed spore balls (sori). The sorus is covered by thin dull delicate membrane, which is easily pushed away leaving exposed sorus. The spores are easily blown away leaving nothing inside the glumes. Some of the late developing grains remain greenish and increase in size slightly over the normal grains. Such greenish healthy appearing grains release spores on pressing (Sharma and Khare, 1987).

Causal organism: *Macalpinomyces sharmae* K. Vanky (Syn. *Tolyposporium* sp.).

Ovaricolous sori of the fungus are inconspicuous. They are hidden by the floral envelopes, ovoid, 0.8-1 to 2 mm covered by a thick light to dark brown peridium of host and fungal origin, which ruptures irregularly to expose the blackish brown granular mass of spore balls intermixed with sterile cells. Spore balls are variable in shape and size, mostly irregular 25-70 x 30-100 µm, dark reddish brown, composed of many rather permanently agglutinated spores. Individual spores are subglobose, ovoid usually polyhedral irregular, 6.5–9 x 7–10.5 µm, yellowish brown, thin walled, finely and densely punctuate verruculose. Sterile cells are subglobose, ovoid to slightly irregular and larger than the spore (9–12 x 11-17 µm), smooth, single or in loose groups, sub hyaline to light yellowish brown. Spore on germination produces one or two septate basidia bearing lateral and terminal basidiospores. Basidiospores are ovoid to long, ellipsoidal 1.5-2.5 x 3-8 µm, hyaline and germinate like yeasts (Vanky, 1995).

Control: The disease can be controlled by adopting resistant cultivars, cultural practices and chemicals. Genotypes namely DPI 2394, PLM 202, OLM

203, DPI 2386 and CO 2 were found resistant to grain smut. Early sowing (1st fortnight of July) recorded the maximum smut incidence and susceptibility index. However, highest grain yield was also recorded in the earlier planting. Early maturing cultivars were found susceptible to grain smut as compared to late maturing. Seed treatment with Carboxin or Carbendazim @ 2 g per kg seed has been found to be economical and effective.

RUST

Distribution: The disease has been reported from India, Philippines and Ceylon (Cummins, 1971; Pall *et al*, 1980 and Haider, 1997).

Crop loss: Systematic work on crop loss has not been carried out.

Symptoms: Numerous, minute, narrow brown pustules arranged in linear rows appear on the upper surface of the leaves.

Causal organism: *Uromyces linearis* Berk. and Br.

The brown uredia develop on upper surface of the leaves. The uredospores are round or subglobose, 22-28 x 20-25 µm in size, brown, echinulate with four equatorial germ pores. The telia are black in colour. Teliospores are globose, smooth, thick walled and 20-32 x 18-25 µm in size with persistent long thick pedicels. The wall is thickened at the apex up to 11 µm. One germ pore is clear at the apex. Fresh uredospores germinate readily but not the Teliospores.

Control: No work has been carried out to control the rust disease in little millet.

UDBATTA

Distribution: In India, the disease was first reported from Bhubaneswar (Orissa) in 1965 and about 40-50 per cent, plants were found infected. Later, the disease was found prevalent to a greater intensity during 1966 and 1967.

Crop loss: No any report is available.

Symptoms: The diseased plants are conspicuous by their malformed inflorescence bearing greyish white fructifications of the fungus. In the infected panicles, the spike lets are found to be glued to one another and to the main rachis by the viscid spore masses, which harden into a crust. Black sclerotial masses are formed on mature panicles. The inflorescence of the healthy plant is a loose panicle measuring 30 to 40 cm long where as in the diseased plant, the spike lets become glued into a cylindrical structure and the length of the panicle gets reduced to 15-23 cm long.

Causal organism: *Ephelis oryzae* Syd. (Teleomorph : *Balansia oryzae-sativae* Hoshioka).

The mycelium of the fungus is septate, hyaline to dirty grey and later becomes dark coloured. The conidia of the fungus are needle shaped, hyaline, septate measuring 13-35 x 1-2 µm.

Control: No work has been carried out.

OTHER FUNGAL DISEASES

Drechslera state of *Cochliobolus nodulosus* Luttrell = *D. nodulosa* (Berk. & Curt.) Subram. & Jain (*Helminthosporium nodulosum* Berk & Curt. apud Sacc.) isolate of finger millet infects producing blight symptoms. *Drechslera* state of *Cochliobolus miyabeanus* (Ito & Kuribayashi) Drechsler ex Dastur (*Helminthosporium oryzae* Breda de Hann.) infects little millet. *Rhizoctonia solani* produces sheath blight symptoms.

Tilletia narasimhanii Thirum & Safee produces broadly, ellipsoid, 2-2.5 mm long and 1-1.5 mm wide galls replacing grain, but these smut galls are seen only when keen observations are made. This smut was first noticed in little millet variety Gariaband.

NEMATODE DISEASES

Little millet is reported to be a good host for *Meloidogyne incognita* and *Tylenchorhynchus vulgaris*.

Ragi cyst nematode, *Heterodera delvi* can infect little millet crop (Krishnaprasad *et al*, 1980).

Panicum miliare (*P. sumatrense*) is reported to be good host for spiral nematode, *Helicotylenchus abunaamai* (Padhi and Das, 1982).

DISEASES OF BARNYARD MILLET

Locally known as *modira*, *sawa*, *kudraivali* and *oodalu*, barnyard millet [*Echinochloa frumentacea* (Roxb.) Link] is grown for both grain and fodder purposes in India. It is quite popular in the hills of Uttarakhand as a component of tribal agriculture. It is also grown on a lesser scale in the states of Bihar, Tamil Nadu, Maharashtra and Madhya Pradesh. Of the different smuts affecting the crop, grain smut is important.

HEAD SMUT

Distribution: This disease has been reported from India and United States. In India, it has been recorded from Madhya Pradesh and Uttarakhand.

Symptoms: The infected inflorescence is deformed or destroyed. In addition, the smut also produces gall-like swellings on the stem, the nodes of young shoots and in the axils of the older leaves. Sometimes, twisted, deformed clusters of leafy shoots with aborted ears may develop (Figure). The gall-like swellings are covered by a hairy rough membrane of host tissue and may be up to 12 mm in diameter.

Pathogen: *Ustilago crusgalli* Tracy and Earle

Epidemiology: The disease occurs late in the season when the crop is about to mature. During this time, the temperature ranges between 20-25°C.

Control: Treat seeds before sowing with Carbendazim or Thiram @ 2g/kg seed. Rogue out infected plants from the field.

GRAIN SMUT

Distribution: The disease is prevalent in Nyasaland, Eastern Europe and many states of India such as Uttar Pradesh, Bihar and Maharashtra. The disease has been reported to cause heavy losses to crop in Madhya Pradesh. In nature, smut incidence has been found to range from 0-75 per cent on different species of barnyard.

Symptoms: The disease is ovaricolous but not all the grains in an ear may be affected. The affected ovaries enlarge two to three times of their normal size (Figure), and their surface becomes hairy.

Pathogen: *Ustilago panici-frumentacei* Bref.

Epidemiology: The disease occurs at the time of grain formation when the temperature ranges between 20 to 25°C.

Control: Pre sowing treatment of seeds with Carbendazim or Thiram @ 2g/kg seed is recommended.

LEAF SPOT OR BLIGHT

Historical account: Drechsler in the year 1923 first recorded the disease from the USA. The disease has been reported from Japan, United States and China.

Symptoms: The disease appears as isolated, dark brown, scattered and spindle shaped spots, measuring 0.5 to 3.0 mm X 0.25 to 1.5 mm, on flag leaves. Afterwards, several such spots coalesce and cover the entire leaf, which becomes grey and dried up. The spots are dark brown to grey in colour and are surrounded by yellow halo. Just after the appearance of the lesions, dark points are visible in the centre. Under humid conditions, fungal growth is visible on these spots. In severe form, the leaves show blighting. Similar spots can also be seen on the leaf sheath.

Causal organism: *Helminthosporium monoceros* Drchsler.
(Syn. *Helminthosporium crusgalli* Nisikado and Miyake)

Epidemiology: The disease is most common under humid conditions.

Control: Since the primary infection comes from seed borne inoculum, seed treatment with systemic fungicides before sowing helps in control. Spraying of copper fungicides helps in reducing the disease intensity.

LEAF BLAST

Symptoms: The symptoms appear on the young seedlings in the field. The spots are spindle to circular shaped and are of different sizes. Initially the spots have yellowish margin and greyish centre. Later, the centres become ash coloured. Under humid conditions, an olive-grey overgrowth of fungus develops at the centre of the spots. Conidiophores and conidia are present in the overgrowth. In the beginning, the lesions are isolated but afterwards they may soon coalesce.

Causal organism: *Magnaporthe grisea* (Anamorph: *Pyricularia grisea*)

Epidemiology: A temperature of 25-30⁰C, humidity of 90 per cent and above, cloudy days with intermittent rainfall, are favourable for the development and rapid spread of disease.

Control: Treat seeds with Thiram, Mancozeb or Carbendazim @ 2g/kg seed 24 hours before sowing. Sowing early in the season, replacing 25% N with either FYM or compost reduces the blast severity.

DISEASES OF PROSO MILLET

Panicum miliaceum L. is the true millet called by different names viz., French millet, Hog millet, Common millet and Proso millet else where and in India as *Baragu*, *Varagu*, *Panivaragu*, *Cheena* and *Cheen*. The crop is grown in sporadic patches from the Himalayas in the North to Tamil Nadu in the South. Major diseases do not damage the crop, but some fungal diseases like sheath rot, leaf spot, smut, rust and Udbatta; a bacterial stripe and a nematode are reported on this important dry land crop.

SHEATH ROT: Caused by *Rhizoctonia solani* Kuhn. was observed for the first time by Rajagopalan *et al.* (1992) at the College of agriculture, Vellayani, Trivandrum during Jun-Aug 1991.

Symptoms- The disease starts at the tillering stage of the crop and continues in the succeeding stages. First symptoms appear as greyish green lesions on the leaf sheath between soil level and leaf blade. The lesions are ellipsoid or ovoid, 2-3 cm long that become greyish white with brown margins (Figure). The disease spreads upwards causing blighting of leaf sheath and leaf blades.

HEAD SMUT: This disease is widely distributed in millet growing areas in Europe and Asia. It is known to be caused by *Sporisorium destruens* (Schlechtend) K. Vanky (Syn. *Sphacelotheca destruens*).

Symptoms- Smut sori first become evident as the panicles emerge. The entire inflorescence is modified into a sorus enclosed by a greyish-white false membrane. The membrane ruptures as the plants mature, exposing the dark-brown spore mass and the vascular tissues of the smutted panicle.

Since the disease is externally seed borne, only fungicidal seed treatment is effective (Kovacs *et al.*, 1997). Sharma and Sugha (1991) found Carboxin and Binomial to give good control of smut reducing the incidence by 99% and increasing the yield by 136%.

GRAIN SMUT: This disease is also known as covered or kernel smut. Most of the grains are transformed into white greyish sacs (smut sori). The sori are slightly pointed to oval and filled with black powder (chlamydospores).

The disease is caused by *Sphacelotheca sorghi* (*Ustilago crameri*). The ustilaginomycete perpetuates through contaminated seed and can be managed by seed treatment, early collection and burning of diseased ears on appearance and crop rotation for 2-3 years.

LEAF SPOT: caused by *Bipolaris panici-miliacei* (Syn. *Helminthosporium panici-miliacei*) is a seed transmitted disease. Seed infection causes seed rotting, coleoptile spot and seedling blight (Lee-DuHyung, 1997). The conidiophores are dark olivaceous brown, simple, cylindrical, geniculate and septate. The conidia are fusoid, dark olivaceous brown, tapering gradually toward the ends, straight to slightly curved, 3-3 distoseptate, and 29.4-155.4x10-26 µm in size with dark hilum included within the contour of the basal cell.

Growing resistant varieties such as RAUM-7 is recommended .

RUST: The disease is caused by *Uromyces linearis* Berk. and Br. Numerous minute brown uredosori appear on both sides of the leaf. Rust pustules are oblong, brown often formed in linear rows. They may also be produced on the leaf sheaths, culms and stems. In case of severe infection, premature drying of leaves and poor grain set is observed. If the infection commences before flowering the damage will be more.

UDBATTA: The affected panicles are transformed into a compact agarbatti like shape, hence the name "Udbatta". The disease is caused by *Ephelis oryzae* Syd. (Teleomorph: *Balansia oryzae-sativae* Hoshioka). Since the crop is of low returns and grown mostly on marginal poor fertility soils, no efforts are made on the disease management.

BACTERIAL STRIPE: The disease was observed for the first time at the South Dakota Agricultural Experimentation Station, Brookings in August 1917 by A.G. Johnson.

Narrow, brown, water soaked streaks extend from the blades of the leaves down to the sheaths and also on culms, where many streaks coalesce. The tissue turns brown and translucent. Abundant exudates is evident in the form of thin, white scales along the streaks. Similar lesions occur on the peduncles and pedicels of the panicle. The severity of the infection may not kill the plants, but individual leaves are partly or entirely browned. In some instances the entire top of the plant may be killed, tissue becomes soft and brown, especially where partly enclosed and protected by lower leaves and sheaths. In such case, new shoots or tillers may come out at the base.

Pseudomonas avenae (*Bacterium panic*) the causal bacterium is a short rod with rounded ends, arranged singly or in pairs, occasionally in chains. The bacterium is gram negative, measures 3.1 to 1.5 μ X 0.3 to 0.45 μ , produces no spores and has a polar flagellum. It is seed transmitted.

NEMATODE: Gokte *et al*, (1992) at NBPGR, New Delhi observed the seeds of proso millet to be infected with nematodes. The nematodes were identified as *Aphelenchoides besseyi* and up to 16 nematodes per seed were found localized beneath the glumes in anhydrobiotic state. Further, Gokte and Mathur (1993) suggested eradication by pre soaking of seeds in 1% H₂O₂ for 3h followed by hot water treatment at 48°C for 15 min. Without pre-soaking complete kill was also achieved by exposing to 50°C for 15 min.

PATHOMETRY / DISEASE QUANTIFICATION

Pathometry is the branch of plant pathology that deals with quantification or measuring plant diseases. Recording the disease a few times during the crop growth period is a bare necessity to plot its progress or evaluate varieties for their resistance. Disease can be measured by three ways:

Disease incidence- the number or proportion of plant units diseased (the number or proportion of plants, leaves, stems that show disease)

$$\text{Per cent Disease Incidence (PDI)} = \frac{\text{Number of infected plants}}{\text{Total number of units assessed}} \times 100$$

Disease severity- is the proportion of area or amount of plant tissue diseased

$$\text{Disease severity} = \frac{\text{Area of plant tissue affected}}{\text{Total area}} \times 100$$

Yield loss - is the proportion of the yield that the grower will not be able to harvest because the disease destroyed it directly or prevented the plants from producing it.

$$\text{Yield loss} = \text{Yield in disease free plot} - \text{Yield from disease affected plot}$$

Disease incidence is easy to assess as the number of infected unit plants out of the total in a unit area is counted. In case of severity, the assessment depends on the perception of symptoms by human eye. This perception depends on the capability to distinguish and read infection from the uninfected tissue. In addition, based on previous experience the material perceived is rated in a way that is easily communicable. Such rating should be consistent, in which case they can be numerically analysed and interpreted (Nagarajan, 1983).

In a few cases such as systemic viruses, smuts, Udbatta, green ear and neck blast, disease incidence has a direct relationship to the severity of the disease and yield loss. In many of the other diseases such as leaf blast, leaf spots, leaf blights and rusts in which plants are counted as diseased whether they are exhibiting a single lesion or hundreds of lesions, disease incidence may have little relationship to the severity of the disease or to yield loss. Although severity and yield loss, rather than disease incidence, are of greater importance to the grower, but for evaluating germplasm and varietal resistance incidence is considered important for superiority of performance and durability.

Scales adopted for evaluating/screening varieties and germplasm lines as applicable to different diseases are given below.

Striga: The per cent incidence is recorded by counting total number of plants infected with *Striga* species and working out the percentage

$$\text{PDI} = \frac{\text{Number of infected plants}}{\text{Total number of plants}} \times 100$$

Blast : For assessing leaf blast incidence, following scale is adopted:

Grade	Percentage leaf area affected
0	No symptoms on the leaves
1	Small brown specks of pinhead size to slightly elongate, necrotic grey spots with a brown margin, less than 1% leaf area affected.
2	A typical blast lesion elliptical, 5-10 mm long, 1-5% of leaf area affected
3	A typical blast lesion elliptical, 1-2 cm long, 5-25% of leaf area affected
4	25-50% of leaf area affected
5	More than 50% of leaf area affected with coalescence of the lesions

Neck blast and finger blast are recorded at dough stage of the crop. Neck blast is recorded as the percentage of ears showing infection on the peduncle and finger blast as the percentage of fingers affected is recorded.

$$\text{Percent neck blast} = \frac{\text{Number of ears showing infection on peduncle/neck}}{\text{Total number of ears in a unit area}} \times 100$$

$$\text{Percent finger blast} = \frac{\text{Number of infected fingers per unit area}}{\text{Number of fingers in five plants} \times \text{Total number of ears}} \times 100$$

The resistance or otherwise of the germplasm or the test entries to blast can be assessed using the following rating-

Sl. No.	Percentage Disease Incidence (PDI)	Reaction	Grade
1	0	Immune	I/HR
2	0.1-2.00	Highly resistant	HR
3	2.01-10.00	Resistant	R
4	10.01-25.00	Moderately resistant/susceptible	MR/MS
5	25.01-50.00	Susceptible	S
6	>50.00	Highly susceptible	HS

Following scale is used for screening the varieties/genetic material against **Brown Spot**

Grade	Percentage leaf area affected
0	No infection
1	Pinhead spots less than 1% leaf area affected
2	Pinhead spots 5-10% leaf area affected
3	Typical brown spots with grey centre 5-25% leaf area affected
4	25-50% leaf area affected
5	Large brown spots with grey centre more than 50% leaf area affected

For **Rusts** the following scale is adopted for visual rating

Grade	Percentage leaf area affected
0	No infection
1	Pustules covering 1% leaf area
2	Pustules covering 1-5% leaf area
3	Pustules covering 5-25% leaf area
4	Pustules covering 25-50% leaf area
5	Pustules covering above 50% leaf area

Per cent **Head smut**, **green ear** and **Udbatta** incidence is recorded by counting healthy and smutted plants in a unit area at maturity. Per cent, **Grain smut** incidence can be calculated by counting the number of infected plants among the total plant population and then computing the percentage

$$\text{PDI} = \frac{\text{Number of smut / green ear / head smut affected ears}}{\text{Total number of ears}} \times 100$$

To estimate the **grain smut** severity 1-6 rating scale recommended for small millets is used.

Grade	Percentage of smutted grains in the ear
1	Up to 1
2	1.1 to 5
3	5.1 to 10
4	10.1 to 25
5	25.1 to 50
6	> 50

The germplasm or the entries can be assessed for their resistance or susceptibility to head smut using the following scale-

Head smut incidence (%)	Reaction
0	Highly resistant (HR)
Up to 1	Resistant (R)
1.1 to 5	Moderately resistant (MR)
5.1 to 10	Moderately susceptible (MS)
10.1 to 20	Susceptible (S)
> 20	Highly susceptible (HS)

Assessment scale used for rating the severity of **ergot** infection in pearl millet may be adopted for rating the severity of ergot in kodo millet.

Grade	Description
1	No symptoms
2	< 5 per cent of ergot grains (grains became sclerotic)
3	6 – 10 per cent of ergot grains
4	11 – 20 per cent of ergot grains
5	> 20 per cent of ergot grains

SELECTED REFERENCES

- Ahmed, N.N. 1991. Biology of smut fungi of the minor millets *Paspalum scrobiculatum* L. and *Echinochloa frumentacea* (Robx.) Link in Karnataka. M.Sc.(Agri.) Thesis, University of Agricultural Sciences, Bangalore, India. pp 1-91
- Alam, M.M., Khan, M.A. and Saxena, S.K. 1973. Some new host records of root knot nematode, *Meloidogyne incognita* (Kofoid & White, 1919) Chitwood, 1949. *Indian J. Nematol.* **3**:159-160.
- Anonymous 1975. *Annual report of the virologist, AICRP small millets 1974-75*, UAS, Bangalore.
- Ayyar, P.N.K., 1933, Some experiments on the root gall nematode *Heterodera marioni* in South India. *Madras agric. J.*, **21**:97-107.
- Ayyar, P.N.K., 1934, Further experiments on the root gall nematode, *Heterodera marioni* in South India. *Indian J. agric. Sci.*, **3**:1064-1071.
- Batsa, B.K. and Tamang, D.B., 1983, Preliminary report on the study of millet diseases in Nepal. In : *Maize and finger millet*. 10th Summer Workshop 23-28 Jan, 1983, Rampur, Chitwan, Mysore
- Bhide, N.V. and Aimen, R.R. 1959. Pharmacology of a tranquilizing principle in *Paspalum scrobiculatum* grains. *Nature.* **153**:1735-1736.
- Billimoria, K.N. and Hegde, R.K., 1971, A new bacterial disease of ragi, *Eleusine coracana* (Linn) Gaertn in Mysore state. *Curr. Sci.* **40**:611-612.
- Butler, E. J. and Bisby, G. R. 1931. Fungi of India. *Sci. Monogra.*, XVIII, Calcutta.
- Butler, E.J., 1918, Fungi and Diseases in Plants, Thacker Spinck and Co. Calcutta pp 547.
- Chandrasekaran, N.M., 1964. *Studies on the reniform nematode Rotylenchulus reniformis with special reference to its pathogenicity on castor (Ricinus communis) and ragi (Eleusine coracana)*. M.Sc. (Agri.) thesis, Univ. Of Madras.
- Channamma, K.A.L., Viswanath, S. and Mantur, S.G., 1996, New record of *Uromyces* Sp on ragi from Karnataka. *Curr. Res.*, **25**:97.
- Coleman, L.C., 1920, The cultivation of ragi in Mysore. *Bull. Dep. Agric. Mysore.* Gen. Ser. 11.
- Cummins, G. B. 1971. *The rust fungi of Cereals, Grasses and Bamboos*. Springer Verlag, Berlin.
- Desai, S.G., Thirumalachar, M.J. and Patel, M.K., 1965, Bacterial blight disease of *Eleusine coracana* Gaertn. *Indian Phytopath.*, **28**:384-386.
- Deshmukh, P.G., Kanitkar, U.K. and Pendse, G.S. 1975. A new fungal isolate from *Paspalum scrobiculatum* Linn. with new biologically active metabolites. *Acta Microbiologica Academiae Scientiarum Hungaricae.* **12(3)**:253-242.
- Dubey, S.C., 1995, Banded blight of finger millet caused by *Thanatephorus cucumeris*. *Indian J. Mycol. Pl. Pathol.*, **25**:315-16.
- Dublish, P.K. and Singh, P.N., 1976, Phytopathogenic fungi of Meerut; Some new records for India. *Curr. Sci.*, **45**:168.
- Gokte, N. and Mathur, V.K. 1993. Treatment schedule for denematization of seeds of *Setaria italica* and *Panicum miliaceum* infected with *Aphelenchoides besseyi*. *Nematologica.* **39(2)**:274-276.

- Gokte, N., Mathur, V.K, Rajam and Arjun Lal. 1992. *Panicum miliaceum*- A new host record for *Aphelenchoides besseyi*. *Indian Phytopathol.* **45**:
- Govindu, H. C., Shivanandappa, N. and Renfro, B.L., 1966. Observations on diseases of *Eleusine coracana* with reference to *Helminthosporium* disease. *Abstr. Internat. Symp. Pl. Path.* New Delhi. pp 48-49.
- Grasso, V. 1952. Le Claviceps delle Graminacee Italiane. *Ann. Sper. Agr. (N.S.)* pp 143.
- Haenseler, C.H., 1941. Helminthosporium leaf spot on millet in New Jersey. *Plant disease reptr.*, **25**:486
- Haider, Z.A. 1997. Little millet in India Agriculture: Progress and Perspectives. In Nat. semi. on *Small Millets : Current trends and Future Priorities as food, Feed and in Processing for Value Addition*, TNAU, Coimbatore. April 23-24, 1997.pp
- Jain, A. K. and Tripathi, S. K. 2002. Pathogenic variability in the isolates of *Sorosporium paspali-thunbergii* causing head smut of kodo millet. In *Advances in Fungal diversity and host pathogen interactions*. (Eds.) B. F. Rodrigues, H. N. Gour, D. J. Bhatt and N. Kamat. Dept. of Botany, Goa University, Goa. pp 85-89.
- Jain, A. K. and Yadava, H. S. 1997. Recent approaches in disease management of Small Millets. Proc. Nat. Semi. on *Small Millets – Current Research Trends and Future Priorities as Food, Feed and in processing for Value Addition*, held at TNAU, Coimbatore (T.N.) from 23-24 April, 1997. pp 31-33.
- Jain, A.K., Tripathi, S. K. and Singh, R. P. 2006. *Macalpinomyces sharmae* – A new threat for the cultivation of little millet in Madhya Pradesh. Proc. Nat. Symp. on *Emerging Plant Diseases, their diagnosis and management*, N.B.U., Siliguri (W.B.), Jan. 31 to Feb. 2, 2006. pp 31-32.
- Joshi, L.M., Raychaudhuri, S.P., Batra, S.K., Renfro, B.L. and Ghosh, A., 1966. Preliminary investigations on a serious disease of *Eleusine coracana* in the states of Mysore and Andhra Pradesh. *Indian Phytopath.* **19**:324-325.
- Keshavamurthy, K.V. and Yaraguntaiah, R.C. 1969. Further studies on the transmission of the virus component of the ragi disease complex in *Mysore*. *Mysore J. Agric. Sci.*, **3**:480.
- Kovacs, J. Koppnyi, M, Nemeth, N and Petroczi, I 1997. Occurrence and prevention of head smut caused by *Sporisorium destruens* (Schlechtend) K. Vanky in millet. *Noventyermes.* **46**(4):373-382.
- Krishnaprasad, K. S., Krishnappa, K., Setty, K.G.H., Reddy, B.M.R. and Reddy, H.R. 1980. Susceptibility of some cereals to ragi cyst nematode, *Heterodera delvi*. *Curr. Res.* **9**(6):114-115.
- Krishnappa, K., Ravichandra, N.G. and Reddy, B.M.R., 2002, Role of green manuring in the management of nematodes. In : *Potentials of green manuring in Rain fed Agriculture* (Eds.) Shankar, M.A., Manjunath, A., Gajanana, G.N., Mariraju, H. and Lingappa, B.S., pp 45-55.
- Kulkarni, G.S. 1922. The smut of nachani or ragi (*Eleusine coracana*). *Ann. Appl. Biol.* , **9**:184-6
- Lee-DuHyung. 1997. Morphological characters and seed transmission of *Bipolaris panici-miliacei* causing leaf spot of common millet. *Korean J. Pl. Pathol.* **13**(1):18-21
- Lulu Das and Girija, V.K., 1989, Sheath blight of ragi, *Curr. Sci.*, **58**: 681-682.

- Mariappan, V., Natarajan, C. and Kandaswamy, T.K., 1973. Ragi streak disease in Tamil Nadu. *Madras Agric. J.*, **60**:451-53.
- Maramorosch, K., Govindu, H.C. and Kondo, F., 1977. Rhabdo virus particles associated with a mosaic disease of naturally infected *Eleusine coracana* (finger millet) in Karnataka state (Mysore) South India. *Plant Dis. Repr.*, **61**:1029-31.
- Mc Rae, W., 1920, Detailed administration report of the government mycologist for the year 1919-20.
- McAlpine, D. 1910. *The smuts of Australia*, Melbourne. Government Printer. pp 285
- Mehta, P.R. and Chakravarty, S.C., 1937, A new disease of *Eleusine coracana*. *Indian J. agric. Sci.*, **7**:793-6.
- Mishra, A. P. and Mishra, B. 1969. *Helminthosporium holmii* on graminaceous hosts. *Indian Phytopath.* **22**:412-414.
- Mohanty, K.C. and Das, S.N., 1976, Free amino acids in the roots of finger millet plants infected with ring nematodes. *Indian Phytopath.*, **29**:434-436,
- Mohanty, U. N. and Mohanty, N. N. 1957. *Ephelis* on some new hosts. *Sci. & Cult.* **22**:465-468.
- Mundkur, B. B. 1939. A contribution towards knowledge of Indian ustilaginales. *Trans. Brit. Mycol. Soc.* **23**:105.
- Munjal, R.L., Lall, G. and Chona, B.L., 1961, Some *Cercospora* species from India-VI. *Indian Phytopath.*, **14**:179-190.
- Nagarajan, S. 1983. *Plant Disease Epidemiology*, Oxf. & IBH, New Delhi. Pp 267.
- Nagaraju, Viswanath, S., Reddy, H.R. and Lucy Channamma, K.A. 1982. Ragi streak, a leaf hopper transmitted virus disease in Karnataka. *Mysore J. Agric. Sci.*, **16**:301-305
- Nema, A. G., Kulkarni, S.N. and Pall, B.S. 1978. Bacterial leaf streak of kodo (*Paspalum scrobiculatum* L.). *Sci. & Cult.* **45**(9):365-366.
- Padhi, N.N. and Das, S.N. 1982. Host range of the spiral nematode, *Helicotylenchus abunaamai*. *Indian J. Nematol.* **12**(1):53-59.
- Palaniswamy, A., Govindaswamy and Vidyasekaran, P., 1970. Studies on blast disease of tenai. *Madras Agric. J.*, **11**: 649-693
- Pall, B. S. and Nema, A. G. 1976. Screening of kodo (*Paspalum scrobiculatum* L.) varieties against Udbatta disease. *JNKVV Res. J.* **10**(2):194.
- Pall, B. S., Jain, A.C. and Singh, S. P. 1980. Diseases of lesser millets. JNKVV, Jabalpur. pp 53-54.
- Pall, B.S. 1979. Quantitative losses due to bacterial leaf streak of kodo (*Paspalum scrobiculatum* L.). *Food farming & Agriculture.* **11**(1): 1
- Pall, B.S. 1983. Bacterial streak of kodo millet: Varietal resistance and chemical control. *MILWAI Newsl.* **2**:12.
- Pall, B.S. 1984. Quantitative loss due to bacterial leaf streak in kodo millet. *MILWAI Newsl.* **3**:15-16.
- Parambaramani, C., Subramanian, C.L. and Kandaswamy, T.K., 1975, Sheath blight and wilt or ragi (*Eleusine coracana* Gaertn) caused by *Marasmius candidus* Bolt. *Curr. Sci.*, **44**: 358.
- Patel, M.K. and Thirumalachar, M.J., 1965, Notes on some *Xanthomonas* species described from South India. *Curr. Sci.*, **34**: 436-37.
- Paul Khurana, S.M., Raychaudhuri, S. P. and Sundaram, N.V., 1973. Further studies on ragi mosaic in Delhi. *Indian Phytopath.*, **26**:554-559.

- Pendse, G. S. 1974. A note of new chemical compound isolated from a fungus hitherto unknown. *Experiential*. **30**:107-108.
- Pesch, C., Pieterse, A.H. and Stoop, W.A. 1983. Inhibition of germination in *Striga* by means of urea. Proceedings of the 2nd International Workshop of *Striga*, ICRISAT. pp 37-38
- Pradhanang, P.M. 1994, Quantification of the relationship between cercospora leaf spot disease (*Cercospora eleusine*) and yield loss of finger millet. Tech. Paper- Lumle Agricultural Centre No. 94/3, 12 pp 7 ref.
- Pradhanang, P.M. and Abington, J.B., 1993, Cercospora leaf spot disease (*Cercospora eleusine*) of finger millet in Nepal. In : *Advances in Small Millets* (Ed.) Riley, K.W., Gupta, S.C., Seetharam, A., Mushonga, J.N., Oxford IBH Pub. Co., New Delhi.
- Raghunathan, V., 1968, Damping-off of Green gram, Cauliflower, Daincha, Ragi and Cluster beans. *Indian phytopath.*, **21**:456-57.
- Rajagopal, B.K., 1965, *Further studies on the damage caused by the Reniform nematode Rotylenchulus reniformis Linford and Oliveira 1940 to ragi and castor*. M.Sc. (Agri.) thesis, Univ. of Madras, pp 62.
- Rajagopalan, B., B. Balakrishnan and Lu Lu Das. 1992. Sheath rot of *Panicum miliaceum* L. *Indian Phytopath.* **45**:279
- Ramakrishnan, T.S. and Sundaram, N.V. 1950. Ergot on two grasses from South India. *Sci. & Cult.* **16**:214.
- Rangaswami, G., Prasad, N.N. and Eswaran, K. S. S. 1961. Two new bacterial diseases of sorghum. *Andhra Agric. J.* **8**(6):269-272.
- Setty, K.G.H., 1975, Studies on ragi cyst nematode *Heterodera* Sp. Report of UNDP/ICAR Research Project, University of Agril. Sciences, Hebbal, Bangalore.
- Sharma, N. D. and Khare, M.N. 1987. Two new smut diseases in little millet (*Panicum sumatrense*) from India. *Acta Botanica Indica*.**15**:143-144.
- Sharma, P.N. and Sugha, S.K. 1991. Effectiveness of seed dressing fungicides for control of smut (*Sphacelotheca destruens*) of Proso millet (*Panicum miliaceum*). *Indian J. Agric. Sci.* **61**(9):692-693.
- Shaw, F.J.F., 1921, Report of the Imperial Mycologist Scient. Reports *Agric. Res. Inst. Pusa*, 1920-21 pp 34-40.
- Subbaya, J and Raychaudhuri, S.P., 1970. A note on a mosaic disease of ragi (*Eleusine coracana*) in Mysore, India. *Indian Phytopath.*, **23**:144-148.
- Sundaram, N.V., Palmer, C.T., Nagarajan, K. and Presscot, J.M., 1972, Disease survey of sorghum and millet in India. *Pl. dis. Repr.*, **56**:740-43.
- Sundaraman, S., 1921. *Ustilago crameri* Koern on *Setaria italica*. *Beauv Bull Agric Res Inst. Pusa* 97 pp 11.
- Takasugi and Akaisahi, Y., 1933. Studies on the downy mildew of Italian millet in Manchuria. *Res. Bull s Manchuria rly.W.* **11**:1-20.
- Thirumalachar, M.J. and Narasimhan, M.J., 1949, Downy mildew of *Eleusine coracana* and *Iscilema laxum* in Mysore. *Indian Phytopath.*, **2**:46-51.
- Uppal, B.N., Patel, M.K. and Kamat, M.N. 1935. The Fungi of Bombay. VIII. Private Publication. pp 1-56.
- Vanky, K. 1995. Taxonomical studies on Ustilaginales XII. *Mycotaxon*. LIV:215-238.
- Venkataraman, S.V., 1946, Diseases of ragi (*Eleusine coracana*). *Mysore Agri. J.*, **24**:50-57.

- Viswanath, S. 1992. Management of biotic factors (diseases). In 6th Annual Small Millets Workshop at B.A.U., Ranchi- Kanke (Bihar) from 30th April to 2nd May, 1992.
- Yadava, H.S. and Jain, A. K. 2006. *Recent advances in kodo millet*. ICAR, New Delhi pp 1-84.
- Yaraguntaiah, R.C. and Keshavamurthy, K.V., 1969. Transmission of the virus component of the ragi (*Eleusine coracana*) disease- complex in Mysore, India. *Plant Dis. Repr.*, **53**:361-363.

TITLE INDEX

- Finger millet** 1,
Diseases 2
Blast 2,
Pyricularia grisea
Brown spot 3
Drechslera nodulosum
Cercospora 5
Cercospora eleusinis
Downy mildew 5
Sclerophthora macrospora
Foot rot
Sclerotium rolfsii
Smut
Melanopsichium eleusinis
Banded blight
Rhizoctonia solani
Sheath blight
Marsmius candidus
Leaf spot
Curvularia lunata
Rust
Uromyces eragrostidis
Bacterial
Leaf spot
Xanthomonas
Pseudomonas
Blight
X. coracanae
Leaf stripe
P. eleusineae
Virus
Ragi mottle streak
Ragi severe mosaic
Ragi streak
Nematodes
Heterodera
Rotylenchulus reniformis
- Kodo millet**
Diseases
Head smut
Sorosporium paspali-thunbergii
Ergot/Sugary
Claviceps paspali
Rust
Puccinia substriata
Udbatta
Ephelis oryzae
Poisoning

Bacterial
 Leaf streak
 Xanthomonas
 Leaf blight
 X.c.oryzae
Nematodes
 Meloidogyne incognita
 Tylenchorhynchus vulgaris
Root parasite
 Striga
 S. asiatica
 S. densiflora

Foxtail millet

Diseases
 Blast
 Pyricularia setariae
 Downy mildew/ Green ear
 Sclerospora graminicola
 Rust
 Uromyces setariae
 Smut
 Ustilago crameri
 Leaf spot/ Blotch
 Helminthosporium setariae
 Udbatta
 Ephelis oryzae
 E. japonica
 Bacterial
 Blight/ Spot
 Pseudomonas albopreceptans
 Brown stripe
 Pseudomonas setariae

Barnyard millet

Diseases
 Head smut
 Ustilago crusgalli
 Grain smut
 Ustilago panici-frumentacei
 Leaf spot/ Blight
 Helminthosporium crusgalli
 Leaf blast
 Magnaporthe grisea

Little millet

Diseases
 Grain smut
 Macalpinomyces sharmae
 Rust
 Uromyces linearis
 Udbatta
 Ephelis oryzae

Nematode

Meloidogyne incognita

Heterodera delvi

Helicotylenchus abunaamai

Proso millet

Diseases

Sheath rot

Rhizoctonia solani

Head smut

Sporisorium destruens

Grain smut

Sphacelotheca sorghi

Leaf spot

Bipolaris panici-miliacei

Rust

Uromyces linearis

Udbatta

Ephelis oryzae

Bacterial stripe

Pseudomonas avenae

Nematode

Aphelenchoides besseyi