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Bacterial Diseases of Rice: An Overview

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Bacterial diseases of rice are a major bottleneck towards a sustainable productivity. They are of paramount global importance, especially in the Asian countries. Extensive work has been done on the management of these diseases especially bacterial leaf blight which includes breeding for the tolerant varieties and chemical treatment. Several resistant genes have been isolated for the use in breeding of the future.

Key words: Rice, Bacterial Diseases, Resistance, Management.

Rice (*Oryza sativa* L) is an important cereal crop grown in different countries of the world. In India, it occupied 44.62m ha with a total production of 89.3MT, during 2009-10 (Anon, 2010). Out of 70 diseases known to attack the crop, 11 are of bacterial origin. These diseases are grouped into seedling, foliar, leaf sheath and grain and culm and root disease (Table 1).

The most important of these bacterial diseases, such as bacterial blight (BB) and bacterial leaf streak (BLS) can devastate a crop when environmental and cultural conditions favor disease development (Table 2).

Seedling Diseases

Seedling blight

Seedling blight is a relatively new disease that occurs in Japan and is associated with the production of seedling in nursery boxes. Seedlings for transplanting are raised in nursery boxes under conditions of high temperature and humidity that are conducive to infection.

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Symptoms

Early symptoms are characterized by a basal chlorosis and withering of the second or third leaves. Infected seedlings later become reddish brown and desiccated but do not exhibit a soft rot. With severe infection, root growth is retarded and seedlings easily lodge.

Causal organism

The disease is caused by *Pseudomonas plantarii*. The bacterium is a Gram-negative, non-spore forming, non-encapsulated rod, 0.7-1.0 x 1.4-1.9/ µm, with one to three polar flagella (Azegami *et al*. 1987a). *P. plantarii* produces a compound called tropolone, which is responsible for the retardation of root growth and leaf chlorosis of infected rice seedlings (Azegami *et al*,1987b).

Management

The application of iron compounds suppresses seedling blight, because the production of tropolone is inhibited in the presence of iron. A thionine producing gene from oat, coding for antimicrobial activity, when transferred to rice by biotechnological intervention, conferred resistance against the pathogen (Takayoshi *et al*,2002).

Bacterial Brown Stripe

Bacterial Brown Stripe also in known as

bacterial stripe, occurs in upland and wetland nurseries as well as in nursery boxes. Although it is widely distributed in the rice growing countries (Shakya *et al*, 1985), it does not cause much damage to production.

Symptoms

The symptoms of the disease in the seedling stage were divided into four types, viz, inhibition of germination, brown stripes occurring interveinally or along the midrib or leaf margins, curving of a sheath and abnormal elongation of mesocotyl. After the infected seedlings were transplanted to paddy fields, symptoms were masked. Natural occurrence of the disease was not seen after the tillering stage, except for the case where rice plants were submerged in the water by flood (Kadota and Ohuchi, 1983).

Causal organism

Bacterial brown stripe is caused by both *Pseudomonas avenae* and *P. syringae* pv. *panici*. *P. avenae* is a Gram-negative, non spore-forming, non-encapsulated rod. 0.92-2.4x0.5-0.7 um, with one or two flagella. *P. syrinage* pv. *panici*, is also a Gram-negative, spore forming, non-encapsulated rod (Shakya *et al*.1985).

Disease cycle and Epidemology

P.avenae is seedborne, and *P. syrinage* pv. *panici* is likely seed borne. Natural infection of *Panicum miliaceum*, *Hordeum vulgare* and *Setaria italica* by *P. syringae* pv. *panici* has been reported.

Management

Dry heat treatment at 65^{O}C for 6 days can eliminate the pathogen from seeds (Zeigler and Alvarez,1988). In nursery boxes, spraying of Kasugamycin can control the pathogen.

Foliar diseases

Bacterial blight

History and Distribution

Bacterial blight is one of the most devastating diseases of rice worldwide and is found both in tropical and temperate regions. It was first recorded in Japan in 1884. In the 1960s, bacterial blight became prevalent in other rice-growing regions of Asia with the introduction of high yielding cultivars line TNI and IR8, which were susceptible to the disease. In addition to Asia, the disease occurs in Australia, Africa, Latin America, The Caribbean and the United States. Economically, it has had the greatest impact in Asia, where several epidemic have occurred in the past three decades and West Africa, particularly in Niger, where irrigated rice was extensively damaged in 1982 (Ou, 1985; Mew, 1989). Under favorable conditions susceptible varieties can undergo more than 70% crop loss (Mew *et al*,1993; Mew and Vera Cruz, 2001).

Economic Importance

The disease is responsible for a loss of 20-30% in different countries. In India, losses in yield varied from 6-60% in different states depending upon stage and severity of infection and type of cultivars (Ou 1985; Singh *et al*, 1977).The disease occurred in an epidemic form in the Palghat district in Kerala and resulted in huge mortality of the crop Reddy *et al*, (1979) found a linear relationship between disease severity and grain yield and developed a critical point model to predict crop losses associated with disease.

Symptoms

There are three main symptoms caused by bacterial blight- leaf blight, wilt or Kresek and yellow leaf or pale yellow.

Leaf blight, the most common syndrome, generally occurs from the maximum tillering stage onward. It begins as water-soaked stripes on the leaf blades. The stripes increase in length and width, become yellow and then white, and may coalesce to cover the entire leaf blade. Drops of bacterial exudates may be observed on young lesions. Older infected leaves may appear grayish from the growth of saprophytic fungi. Small, circular lesions with water-soaked margins may also form on the glumes with severe infections. Infected plants produce fewer and lighter grains and the grain is of poor quality.

The wilt syndrome, known as 'Kresek' is the most destructive manifestation of the disease found between the temperature 28^{0}C and 34^{0}C. It occurs in the tropics from the seedlings to the early tillering stage. Leaves of infected plants wilt and roll up, turning grayish green. The leaves then turn yellow to straw-colored and wither, and entire plant generally dies. Plants that do survive are stunted and yellowish. Total crop failure is not uncommon with Kresek.

In the tropics, yellow leaf or pale yellow syndrome is associated with bacterial blight. The youngest leaf of the plant becomes uniformly pale yellow or has a broad yellow stripe. With be yellow leaf, the bacteria are not present in the leaf itself

but can be found in the internodes and crowns of affected stems.

Causal organism

Bacterial blight is caused by *Xanthomonas oryzae* pv. *oryzae (Xoo)*. The bacterium is Gram-negative, non-spore forming rod. 0.55 x 3.5-2.17µm and mono-trichous flagellate. It is aerobic and grows best at a temperature and pH of 25-30^{0}C and 6.5-7.5 respectively. The isolate of bacterium varied in the rate of utilization of different carbon and nitrogen sources (Ou, 1985). Iron has been found to enhance the virulence of *X.oryzae* pv. *oryzae* (Ansari abd Sridhar, 2001).

Pathogenic Variation

Differences in virulence were observed among the isolates of *Xoo*. Mew (1987) reported a comprehensive list of pathotypes prevalent in Asia. Accordingly, there were seven pathotypes in Japan, six in Philippines, nine in Indonesia, two in Thailand, three in India, two in Korea and two in Nepal on a set of 10 differential cultivars (Kinmaze. Kogyku, Rantaj- emas. Was Aikoku 3, Java 14, IR8, IR 20, IR 1545-339-2-2, Cas 209 and DV 85).Reddy and Reddy (1992) collected 150 isolates from 25 locations in India and classified them into two pathotypes: pathotype I was avirulent on DV 85 but virulent on Cemposelak and Java 14 and pathotype II was virulent on DV 85 but avirulent on Cemposelak and Java 14. Pathotype I was further divided into sub-groups i.e. pathotype Ia and Ib, respectively based on the avirulence or virulence on another differential IR 20.They reported that pathotype Ia was prevalent in Punjab, Eastern Uttar Pradesh, and Maharashtra, to which IR 20 was resistance while pathotype 1B was prevalent in Andhra Pradesh, Bihar, Gujarat, Haryana, Kerala, Orissa, Tamil Nadu and Western U.P while pathotype II was limited to West Bengal. DNA fingerprinting of 67 isolates of *Xoo* collected during 1994 and 1995 from 18 locations in India belonged to a single lineage representing (Yoshitola *el al.* 1997). No single gene is effective against all the pathotypes in India.

Disease Cycle and Epidemology

X.oryzae pv. *oryzae* survives primarily in/on infected seeds, stubbles, straw, ratoons, self-sown plants and rhizosphere of winter crops and perennial wild plants, especially *Leersia oryzoides*, *Zizania latifolia*, *Leptochola chinensis*, *L. panacea*. and *Cyperus rotundus* and wild *Oryza* species *O.rufipogon* and *O. australiensis* (Devadath, 1982; Singh *et al*, 1980; Sundar and Dodan, 1989; Thrimurty and Devadath, 1981).

The bacterium invades through wounds caused by root development or any other injuries occurred during handling, insect attack or through natural openings like hydathodes and stomata on leaves and becomes systemic in the xylem of rice plant (Devadath and Rao, 1975; Nada *et al*, 1981). Infection is favored by a temperature of 25-30^{0}C, high humidity, shading, heavy dose of nitrogenous fertilizers, rain, flooding and severe winds. The bacterium can be disseminated by irrigation water, by splashing or windblown rain, by plant to plant contact, by trimming tools used in transplanting, and by handling during transplanting (Devadath, 1982).

Management

Management of bacterial blight, caused by *Xanthomonas oryzae* pv. *oryzae* comprises of i) use of host resistance ii) modification in cultural practices iii) biological control iv) use of natural products or botanicals extracts and v) use of conventional and non-conventional chemicals.

Host resistance

Genetic resistance is the most effective, practical and economic method of disease management. Considerable emphasis is being given on identification and incorporation of bacterial blight resistant gene donors in commercial cultivars using conventional breeding methods and molecular approaches.

Inoculation methods and rating scale

Development of an efficient inoculation method is pre-requisite for a successful resistance breeding programme. Various inoculation methods viz. needle pricking, modification of needle prickling, immersion/dipping, clipping, applying and spraying methods have been developed by various workers for evaluation of resistance (Ou,1985). The resistant genotypes are identified based on their reaction following 0-9 scale (Anonymous,1996).Young bacterial culture (1-2d old) at a concentration of 10^8 cells/ml has been preferred for inoculation to ensure infection (Ou,1985).

Varietal resistance

Testing and breeding of rice cultivars for resistance began long back in Japan. Most of the workers have evaluated rice genotypes for

resistance to bacterial blight using local bacterial isolate(s) with unknown virulence. Use of resistant varieties has given commendable control of the disease (Savary *et al* 2000), but durable resistance being eluding, breeding for resistant variety remains a continuous challenge (Bonman *et al*,1992).

However, several workers have identified bacterial blight donors against known virulence prevalent in the region. Currently though rich and diverse sources of resistance along with rapid and reliable screening techniques are available, a satisfactory management of BB has not been achieved. Ajaya and PR-112 have reasonable but not desirable level of resistance. 51-33-2.RP 2151-33-21-22, Ruchi, IR 54, RP 2151-40-1-11 (Aggarwal *et al.*,1997) and12 lines IRBB1, IRBB2, IRBB3, IRBB4, IRBB7, IRBB8, IRBB10,IRBB11, IRBB13, IRBB14, IRBB21 and IR24 have been recently identified by BANITO,2012 as resistant aginst 13 X. oryzae pv. oryzae strains from Togo.

A varied response of rice plants to *X. oryzae* pv *oryzae* at different growth stages has been reported. Resistance to the kresek phase was reported in many cultivars (Chand, 1986), Kaku and Kimura (1978) observed that TKM 6, Sigadis etc had seedling resistance while Kogyoku etc. had adult plant resistance (Mew, 1997). Horino (1981) demonstrated that IR 28 and Tetep had high level of resistance at both seedling ad adult plant stage to bacterial group 1. In general, the genes that confer resistance at seedling stage (*Xa4a, Xa5, Xa10*) remain effective in adult stage also. However, genes for adult plant resistance such as *Xa3, Xa4b, Xa6, Xa8, Xa9* and *Xa2I* do not necessarily provide resistance at the seedling stage indicating that the two types of resistance are distinctly distinguishable as demonstrated from the work of Khoshkdaman (2014) **.**

Apart from above Biochemical resistance due to high phenolic contents in Basmati-385 and Basmati-2000 was also suggeted by Khan (2014).

Mechanism of resistance

Kiryu and Mazuta (1995) found that cultivars with few, short, narrow and erect leaves have low infection than those having luxuriant growth and spreading leaves. Cultivars with hairy leaves showed maximum disease while the disease was very low in cultivars with glabrous leaves due to retention of more inoculums by the hairy cultivars (Premlatha Dath *et al*, 1977). Resistant varieties posses lower stomatal index than susceptible ones (Shukla and Gangopadhyay, 1981), A negative correlation has been observed between disease development and frequency of distribution of silicate cells in coastal region, (Kaul and Sharma. 1987).Resistant cultivars have a higher ratio of reducing sugars to total nitrogen, higher contents of polyphenols, lower contents of some free amino acids and production of non-specific phytoalexins. (Mahto *et al*, 1987, Ou, 1985). In Tetep, the bacterial seeds become irregular in shape and were immobilized by fibrillar material induced from cell wall of host. The development of such fibrillar material has been reported to be a defense mechanism associated with compatible reaction (Horino, 1981).

Genetics of resistance

Considerable information is available on genetics of resistance. It is suggested that only the existence of horizontal resistance along with the vertical component could help varieties with enhanced and sustainable resistance to BB. Pyramiding of genes and use of molecular markers in the screening of germplasm have been advocated for accurate and speedy assessment of germplasm to be used in resistance breeding. Resistance to BB is considered to be due to or a combination of two or more genes that are often described as dominant, recessive, inhibitory, complementary or polygenic. About 23 genes (*Xal-Xa23* are responsible for BB resistance. Gene combination *Xa4 + xa5*, *xa5 + xa2l* and *xa4 + xa5+ xa2l* conferred broad spectrum of resistance of all the isolates tested, supporting the strategy of pyramiding appropriate resistant genes. A marker assisted breeding strategy was employed for enhancing the resistance of two elite cultivars (Swarna and IR 64) to BB by pyramiding two / three specific resistance genes (xa5, xa13 and Xa21) through backcrossing. The pyramided lines manifested a wider spectrum and higher level of resistance than lines with only a single gene. To speed up the gene pyramiding process and to facilitate future marker aided selection, PCR markers were developed for the two recessive genes xa5 and xa13, and these were used to survey a range of rice germplasm. Many resistant (R) genes have been identified for resistance to *Xoo* in rice. *Xa4* which confers resistance to IR20 and other IR

varieties, developed at IRRI, was originally derived from an Indian cultivar TKM6. *Xa2l* is a resistance gene which confers broad spectrum resistance to all known races of *Xoo* in India and six Philippines races, was initially identified in *Oryza longistaminata* at Central Rice Research Institute, Cuttack, India (Sridhar *et al*, 1997; 2001, Huang *et al*, 1997; Khush *et al*, 1990; Ikeda *et al*, 1990; Zhang *et al*, 1998). Recent development reflect that *Xa3/ Xa26* confers resistance against the bacteria at both seedling and adult stage (Gao *et al*, 2010). An elite restorer line, R8012 including four target genes (*Pi25+Xa21+xa13+xa5*) was acquired by Xiao-deng, et al 2012.,in their same study they derived hybrid Zhong 9A/R8012 from the selected line showed stronger resistance to blast and BB, and higher grain yield than the commercial checks. In the line of strategies to develop resistant cultivars full-diallel mating design, by Habarrurema et al 2012 produced rice genotypes, as NERICA14, NERICA10 and NERICA4 having desirable GCA (Griffing's combining ability analysis) estimates, and were found to be, the best general combiners. Crosses CO39 x NERICA10 and NERICA14 xIRAT104 having favourable SCA (specific combining ability) values were found to be promising in developing the BLB resistant progenies.

Modifications in cultural practices

Cultural practices like proper levelling of field, good drainage and flow irrigation, ploughing down of stubble and straw following harvest and removing of alternate hosts have been observed to minimize the incidence of bacterial blight. Growing of nursery on raised seed beds are advocated to prevent exposure of nursery to bacterial inoculum. Avoidance of excessive application of nitrogenous fertilizers particularly in organic form at tillering stage helped in minimizing bacterial blight incidence. A significant relationship of potassium with bacterial blight incidence has been demonstrated. Deficiency of phosphate and potassium, and excess of silicate have been reported to increase the disease (Reddy and Srivastava, 1975; Ou, 1985). Soil application of potash at 50kg/ha in two splits at 40 and 50 DAS effectively restricted the spread of BB and increased grain yield (Marimuthu, 1995).

Biological control

Erwinia herbicola and native strains of *Pseudomonas fluorescens* (biotype III) from roots of rice, pearl millet and citrus proved inhibitory to BB pathogen and reduced the disease development substantially (Anuratha and Gnanamanickam, 1987; Gnanamanickam *et al*,1999; Sivamani *et al*, 1987). Seed and seedling bacterization with plant growth promoting rhizobacteria, *Azosprillum brasilense* and *Bacillus poymyxa* individually and in mixture proved effective in reducing BB severity (Islam and Bora, 1998). Higher population of *Azotobacter* (1:1 ratio to pathogen) and two strains of N fixing bacteria, *Enterobacter cloacae* MR12 and *Alcaligenes paradoxus* R4 suppressed bacterial blight to a considerable extent (Yang *et al*, 1999). A novel strain of *Lysobacter antibioticus* (strain 13-1) was found to suppress the disease upto 69.7% in green house and 73.5%, 78.3%, and 59.1% respectively in field conditions (Ji *et al*, 2008).

The phylloplane microorganisms namely *Erwinia herbicola*, *Bacillus subtilis*, *Aspergillus* spp, *Sreptomyces* sp, *Micrpcoccus* sp etc suppressed bacterial growth and reduced BB incidence (Saikia and Chowdhury, 1993). *Bacillus subtilis*, *Pseudomonas fluorescens* and *Trichoderma harzianum* were found antagonistic to the pathogen and reduce the disease intensity significantly when used as spray, as combination of sprays with seed treatment, and pre soaking nursery treatment (Manmeet and Thind, 2002). In field evaluation, only *P.fluorescens* and *T. harzianum* reduced the disease significantly. Durgapal (1987) found that mixing avirulent cultures with virulent isolate of *Xoo* inhibited the growth of the virulent isolate and protected the plant against infection.

Use of natural products / botanical extracts

Very little information is available on evaluation of evaluation of plant extracts and other natural products against *Xoo* under *in vitro* and field conditions. Madhiazhagan *et al* (2002) have been reported that leaf extract of *Adhatoda vasica* was most effective in reducing BB incidence followed by *Curcuma longa*, *Allium cepa*, *Prosophis juliflora* and *Azadirachta indica*. Some neem products viz, nemadol, extracts of Neemcake and neem kernel have been found effective in reducing BB incidence. Of seven botanicals formulations namely, achook, neemgold, neemzal, tricure (neembased), ovis(*Lantana camara*), wanis

(*Cymbopogon sp*) and spictaf reduced bacterial blight severity by 20% (Eswamurthy *et al*, 1993, Singh and Sunder, 2001). Extracts of aak (*Calotropis procera*), bathua (*Chenopodium sp*.), santhi (*Trianthema monogyma*), onion (*Allium cepa*), sunflower (*Helianthus annus*), jamun (*Syzygium cumini*), rhizome of Ginger (*Zingiber officinale*) and processed tea (*Camellia sp.*) were found most effective against BB during *Kharif* 1998-2001 (Sunder *et al* 2001).

In field experiments, foliar sprays of fresh cow dung (50 kg / ha), hing (oleoresin of asa foetida, 0.2 g/l and cow dung extract significantly reduced BB incidence (Mary *et al*,2001; Das *et al,* 1998; Marimuthu, 1995). Prophylatic spray is reported to have a better control of the disease as compared to curative spray (Nair et al 2001). For unequivocal conclusion regarding mode of action of these natural products extensive field trials under high disease pressure need to be conducted.

Chemical control

Use of conventional chemicals

A large number of chemicals have shown inhibitory effects on the bacterium in the laboratory, but relatively few have been found effective in preventing or reducing disease incidence under field conditions. Bordeaux mixture and other copper compounds have been used in Japan since 1909. Mercury compounds alone and in mixture with copper compounds were also tried during 1950's. Since 1955, a number of antibiotics including streptomycin, streptocycline, penicillin, chloramphenicol etc have been reported to be effective against *Xoo* in laboratory (Ou, 1985), but very few proved effective in field (Wakimoto, 1962).

Several fungicides including thiram, captan, vitavax, difolatan, mercuric chloride, duter, kocide, MEMC, foltaf, fytolan, cumin, dithane Z-78 etc have been reported to inhibit the growth of bacterium *in vitro*. Among these, cumin, fytolan, mercuric chloride, duter, kocide, MEMC may reduce disease incidence in field (Balaraman and Rajagopalan, 1978).Copper hydroxide containing 35% metallic copper was reported to be very effective against BB (Bag *et al,* 2010).

Eradication of seed borne infection of *Xoo* has been reported by steeping the seeds in mixed solution of wettable ceresan (500-1000 ppm) and agrimycin 100(250ppm) or streptocyclin (27ppm) followed by hot water treatment at 52-55^{0}C for 20-30 min (Devadath, 1982). Agrimycin or plantomycin in combination with copper oxychloride has been reported to reduce the severity of bacterial blight (Mariappan *et al*, 1988).

Use of Non-Conventional Chemicals

Different kinds of non-conventional chemicals including synthetic organic bactericides and host defense activators have been developed and evaluated to manage the disease. Synthetic organic bactericides such as Sankel (Nickel dimethyl dithiocarbonate) etc are recommended for BB control (Mukherjee *et al*,1976). Submerged application of a systemic compound probenzole has been found effective against BB. The chemical induced resistance in rice plant through host mediation (Sekizawa and Mase, 1980).

Padmanabhan and Jain (1966) observed that chlorination of water through soil application of bleaching powder was economical and equally effective to five sprays of streptocycline and copper oxychloride in checking the disease spread. Subsequently, several researchers have advocated the use of stable bleaching powder to control the disease (Chand *et al*, 1979; Srinivasan *et al* ,1977). Preinoculation spray of plant growth regulators like 2, 4-D and NAA has been found effective in reducing BB severity. Nakashita (2003) reported that brasinolide, an important brassinolide induced resistance against BB in an appropriate manner.

As chemical control is not economically effective and cultural practices are not practiced by the farmers, bacterial blight remains a constraint on rice production and will affect the attainment of target of 103 mt by 2006-07 (Jayaraman and Verma, 2002).

An alternative to chemicals has been advocated in the form of an eco-friendly technology by Ahuja (1997) who reported that field trials in 30 areas in 1996 and 1997 have shown that herbal foliar sprays restricted the spread of disease and also increased the yield by 10-15q/ha.

Bacterial Leaf Streak

History and Distribution

It was first reported in Philippines in 1918 and was called the stripe disease. Fang *et al* (1957) reported this disease from China and gave it the current name bacterial leaf streak. The disease is widely distributed in tropical Asia and in West Africa in both lowland and upland rice growing areas. Besides Philippines and China the disease

has been observed in Thailand, Indonesia, Malaysia, India, Vietnam, Indonesia, Bangladesh and Cambodia. In India, it was first reported by Srivastava (1967). It is not either from Japan or from any other temperate countries.

Economic Importance

Estimate of yield loss from the disease range 1.5 to 17.1% depending on the cultivar and the climatic conditions (Opina and Exconde. 1971).

Symptoms

The disease can occur at any growth stage and initially appears as small as interveinal, water soaked streaks. The streaks are at first dark green and later become translucent. The streaks enlarge and coalesce and eventually become light brown. Numerous tiny yellow beads of bacterial excaudate are common the surface on lesions. Eventually, entire leaves turn brown and then grayish white and die. According to Shekhawat and Srivastava (1972) infection of florets and seeds results in brown or black discoloration and death of ovary, stamens and endosperm and browning of glumes. Seeds fail to mature but the rachis do not show any symptoms.

Causal Organism

The disease is caused by *Xanthomonas oryzae* pv. *oryzicola*. The bacterium is a gram-negative, non-spore forming rod, 1.2x0.3-0.5 um with a single polar flagellum.

Disease cycle and Epidemology

According to Shekhawat and Srivastava (1972a) the pathogen can survive in infected seed from season to season to the next but not in the crop debris. The bacterium hibernates under the glumes in mature seed. First leaf carries the bacterium to the aerial parts from where secondary spread occurs, as in the bacterial blight, through wounds and stomata and multiplies in parenchymatous tissue. Bacterial exudate from lesions are disseminated primarily by splashing and wind blown rain and also by leaf contact and irrigation water.

All wild species of *Oryza* may be infected by *X.oryzae* pv. *oryzicola* and may serve as reservoirs of inoculum. Many of the fields in different states of India are also noticed to show streak symptoms earlier than the transplanted rice and may serve as the source of inoculum from season to season in both single and double cropped areas. The bacterium may also be able to survive in irrigation water.

Young rice leaves are more susceptible to the disease and become resistant with increasing age. High humidity for two to three consecutive days (RH 83-93%) or dew during morning hours is necessary for infections. If rains stop, spread of the disease is also retarded. Lesion enlargement is favored by moderate temperatures (26-30.5^{0}C) and retarded at lower temperature (below 22.4^{0}C) irrespective of relative humidity.

Management

According to Shekhawat and Srivastava (1971), since the disease is seed-borne, seed treatment i.e overnight soaking of seeds in 0.025% streptocycline solution and hot water treatment at 52^{0}C for 30 minutes are effective in eradicating seed infection. They have also reported that the sprays of Vitavax at 0.15-0.3% are effective in preventing infection and lesion development. Sankel, captan and fytolan were also effective to some extent. Banerjee *et al* (1984) recommended three sprays of 100ppm streptocycline or agrimycin 100 at intervals of 10 days starting from the earliest appearance of the disease. Ou *et al* (1970) screened 1118 varieties by critical inoculation and found that their reaction varied from resistant to very susceptible. None was immune, Only 140 varieties showed few and small lesions indicating resistant reaction. Most varieties were intermediate in reaction. In India IR-20, Krishna and Jagannath have shown good tolerance to the disease.

Halo blight

Halo blight, caused by *Pseudomonas syringae* pv. *oryzae*, was first reported in 1985 and is currently limited to Aomori Prefecture, Japan. The disease is characterized by circular, pale green to yellowish brown lesions, 2-10 um in diameter on leaf blades. The lesions are surrounded by a distinct halo and have a dark brown spot or stripe in the center. The lesions may coalesce to form large blotches. The disease has not caused serious damage to date (Kuwata, 1985).

Leaf sheath and grain rot

Sheath Brown Rot

Sheath brown rot has been reported in Asia, Latin America, South America, Central Africa and Madagascar. The disease has been reported to be widespread in irrigated rice between 1300-2000m elevations in Madagascar.

Symptoms

On seedlings, a systemic discoloration of the leaf sheath occurs, which may spread to the midrib or veins of the leaves. On the mature plants, symptoms typically occur on the flag leaf sheath from the booting to heading stage, become dry and the panicle withers. Glumes of panicles emerging from infected sheaths exhibit water soaked lesions that turn light brown. Grains of infected panicles are discolored, deformed or empty (Zeigler and Alvarez, 1987, 1990).

Causal Organism

The disease is caused by *Pseudomonas fuscovaginae*, which is a Gram negative, non-spore forming rod, 0.5-0.8x2,0 x 3.5um with one to four polar flagella (Tanii *et al* , 1976).

Disease cycle and Epidemology

P. fuscovaginae survives on rice seed at a low level and as an epiphyte on graminaceous weeds in rice growing areas. Disease development on mature plants is favored by cool day time temperatures (17-23^{0}C) that delay panicle emergence.

Management

Use of clean seed or seed treated with dry heat at 64^{0}C for 6 days is important in management of sheath brown rot. Antibiotics such as streptpmycin, alone or in combination with oxytetracycline also, can affectively manage sheath brown rot if applied at or a few days after panicle emergence.

Sheath rot

Sheath rot caused by *Pseudomonas syringae* pv *syringae* (syn *P. oryzicola*) is identical in symptomatology to sheath brown rot caused by *P.fuscovaginae*. It is the only reported sheath rot pathogen of rice in Chile and has been reported from Asia, Australia and Hungary (Zeigler and Alvarez, 1990).

Grain rot

Grain rot occurs in Japan, Korea and Taiwan. The disease is manifested as a grain rot of mature plants in the field and also as a seedling rot (Chien *et al*, 1983).

Symptoms

On seedlings, symptoms consist of a brown, water-soaked soft rot of leaf sheaths accompanied by wilting or soft rot of the leaves. On the panicle grains are shrunken and pale green, becoming dirty yellow to brown and dry. A brown margin between the infected and healthy parts of the grain is a diagnostic feature of the disease. A mild rot of the flag leaf sheath or the flag leaf sheath collar occurs (Zeigler and Alvarez, 1990).

Causal organism

Grain rot is caused by *Pseudomonas glumae*. The bacterium is a Gram negative rod 0.5-0.7x1.5-2.5 um with one to three polar flagella.

Disease Cycle and Epidemology

The bacterium is seed borne and invades the spaces between the cells in the outer epidermis and spongy parenchyma of the lemma. From seed it may grow epiphytically at a low level until panicle emergence. When its population increases rapidly on the grain. The disease is favored by high temperatures (28^{0}C) and high humidity.

Management

The pathogen may be eradicated from small seed samples with dry heat treatment of 65^{0}C for 6 days. Pre-treatment of rice seeds with a high concentration of (10^{10}cfu/ml) of the avirulent strain of *P.glumae* is the most effective method for reducing the incidence.

Bacterial Palea Browning

The disease occurs in Japan and affects grain quality. Disease incidence as high as 32% and reduction in 1000 seed weight by as much as 15% have been reported (Azegami *et al*. 1983).

Symptoms

Symptoms usually first appear at early. Initially, light brown, water soaked lesions occur on the lemma or palea. The lesions then turn dark brown, The discoloration occurs most frequently on the palea. Infected panicles have more immature grains and lighter grains at harvest, and infected grains become brown after milling.

Causal Organism

The bacterium responsible for the disease is *Erwnia herbicola*. It is Gram negative, and fermentative, with peritrichous flagella.

Disease cycle and epidemiology

High epiphytic population of *E.herbicola* are common on rice. The disease occurs when heading coincides with periods of rain and high temperature in the range of 30-35^{0}C. Disease incidence is increased in fields with high levels of nitrogen fertilization, especially at heating.

Management

No management for the disease is available.

Culm and root disease

Bacterial Foot Rot

Foot rot occurs in Japan, India, Bangladesh , Korea and the Philippines (Goto, 1979 a,b).

Symptoms

Leaf sheaths of infected plants typically exhibit a dark brown decay, and attached leaves turn yellow and wilt. Infection often begins in the ligules. The nodes, culms, and crown also become rotted, and infected tillers can easily be detached from the crown. Infected culms and internodes turn black. Roots attached to infected nodes decay and fall off. Bacterial ooze may be present inside the culms and infected plants have an unpleasant odor. In some cases, the young leaves of tillers that show no sheath browning may wilt as a result of systemic infection of the crown alone.

Causal Organism

The disease is caused by *Erwinia chrysanthemi*. The bacterium is a Gram-negative rod with four to six peritrichous flagella.

Disease and Epidemology

The bacterium has a wide host range. In Japan, Iris plants, which are common around rice fields, were shown to be susceptible to the bacterium and capable of serving as an inoculums source. The bacterium can also be isolated from the rhizosphere of healthy rice plants and thought to be disseminated primarily by the irrigation water. The bacterium enters rice plants through wounds in the root.

Management

No management for the disease is available.

CONCLUSION

Bacterial diseases of rice continue to intrigue the plant pathologists in its management as chemical control is really hard to devise. Specific bactericides are gradually coming to the fore but it has to be coupled with cultural, breeding and biotechnological techniques so as to usher in a polyphasic approach of the disease management.

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