**REVIEW ARTICLE**



# **Present status of bakanae of rice caused by** *Fusarium fujikuroi* **Nirenberg**

**Ram Singh1 · Pankaj Kumar2 · G. S. Laha3**

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#### **Abstract**

Bakanae disease of rice caused by *Fusarium fujikuroi* Nirenberg (Syn: *F. moniliforme* (Sheld.) [Teleomorph: *Gibberella fujikuroi* Wineland] is known to occur in almost all the rice growing countries. The disease causes substantial yield losses. In India, it is a major production constraint in north western parts especially in Basmati rice growing belt. The yield loss due to this disease has been reported to range from 3% to almost complete loss depending on extent of infection, variety and weather conditions. This review summarizes the available information on various aspects of the disease viz., history and geographical distribution, symptoms, economic importance pathogen biology, disease epidemiology and diferent management practices along with future lines of work.

**Keywords** Foot rot · Bakanae · Rice (*Oryza sativa* L.) · *Fusarium fujikjroi*

## **History and geographical distribution**

The disease is said to have been known in Japan since 1828. It has also been called as Fusarium blight, elongation disease, Fusariosis, white stalk in China, palay lalake (man rice) in British Guiana, foot rot in the Philippines, otoke nae (male seedling) in Japan, bakanae in USA, Africa and Australia, foot rot and bakanae in French Equitorial, Africa and Ceylon and foolish plant or foot rot in India (Singh and Sunder [2012](#page-9-0)).

The disease has been reported from almost all the rice growing regions of Asia, Africa, north America, south America and central American countries including countries in the carribean region (Anonymous [2018;](#page-7-0) Gangopadhyay [1983;](#page-7-1) Ou [1985;](#page-9-1) Khokhar [1990](#page-8-0); Singh and Sunder [2012](#page-9-0)). The disease has also been reported from most of the European countries though rice is not cultivated in a large area in Europe. However, the disease is widespread in countries like

 $\boxtimes$  Pankaj Kumar bodlapankajbodla@gmail.com

- <sup>1</sup> CCSHAU Rice Research Station, Kaul 136 021, India
- <sup>2</sup> Department of Plant Pathology, CCSHAU, Hisar 125 004, India
- <sup>3</sup> Department of Plant Pathology, ICAR-IIRR, Hyderabad 500 030, India

India, Bangladesh, China, Nepal and Laos in Asia, Guyana and Suriname in South America and in Australia (Anonymous [2018\)](#page-7-0).

Though the disease has been detected from most of the rice growing states of India, bakanae was considered as a minor disease in India till 1990. However, during last nearly three decades, it has emerged as one of the most serious diseases of rice in Punjab and Haryana, especially on basmati rice varieties. There are many reports of its occurrence in moderate to severe form in diferent districts of Haryana and Punjab during 2004–2012, mainly on basmati rice varieties (Laha et al. [2016](#page-8-1)).

## **Symptoms**

*Fusarium fujikuroi* causes diferent types of symptoms starting from pre-emergence seedling death to grain infection at maturity. Bakanae means bad or naughty seedling referring to abnormal elongation or foolish seedling. The disease occurs both in the nursery bed and transplanted crop and also in ratoon crop. The infected seedlings are thinner and elongated than the normal seedlings and have chlorotic stems and bear yellowish leaves. These infected seedlings may die eventually early in the season. However, some plants surviving up to maturity bear only sterile panicles. The bakanae infected feld can be easily identifed from a distance as it remains uneven throughout the crop season. White or pink mycelial growth and adventitious roots are also observed on the lower internodes of the infected plants.

Brown discolouration of tissues is also observed at lower nodes of diseased plants facilitating an easy separation of the root system at collar region resulting in foot rot symptoms (Ou [1985](#page-9-1); Singh and Sunder [2012\)](#page-9-0). In the end of season, small scattered pycnidia can also be seen in lower sheaths which become blue to black colour. Perithecia are also produced on diseased plants under favourable conditions (Sun and Snyder [1978](#page-9-2)). The various types of symptoms (such as elongated seedlings, stunted seedlings etc.) produced by the pathogen are determined by the production of relative amount of growth hormones (gibberellic acid and fusaric acid) by the pathogen (Webster and Gunnell [1992](#page-9-3); Singh and Sunder [2012](#page-9-0)).

#### **Economic importance**

Disease has been reported to 3% to all most complete loss in grain yield of rice in diferent countries depending on disease incidence, time of infection and cultivars (Singh and Sunder [2012\)](#page-9-0). The disease is more devastating on specialty basmati rice which earns substantial foreign exchange (Bashyal et al. [2014](#page-7-2)). It is known to reduce grain yield by 20–50% in Japan, 70% to almost complete loss in Australia, 3.7–14.7% in Thialand, about 25% in Bangladesh, 5–23% in Spain, 3.0–95.4% in India, 40% in Nepal, and 6.7–58.0% in Pakistan (Yasin et al. [2003;](#page-10-0) Singh and Sunder [2012](#page-9-0); Gupta et al. [2015\)](#page-7-3). In addition to direct yield loss, the disease signifcantly deteriorates the quality of rice (Bashyal and Aggarwal [2013](#page-7-4)).

### **Pathogen and its variability**

The organism causing bakanae was originally identifed as *Fusarium heterosporum* Nees and its ascigerous stage was identifed as *Lisea fujikuroi* by Sawada. Ito and Kimura ([1931](#page-8-2)) transferred *L. fujikuroi* to the genus *Gibberella* as *Gibberella fujikuroi* (Sawada) Ito with *Fusarium moniliforme* Sheldon as its anamorph. Later, *F. moniliforme* was re-identifed as *Fusarium fujikuroi* Nirenberg (Nirenberg [1976](#page-8-3)). Initially, *G. fujikuroi* was considered to be a single species; however, phylogenetic studies revealed that the taxon is composed of a several species known as *Gibberella fujikuroi* complex (Kim et al. [2012\)](#page-8-4). This species complex is consisted of at least 11 biological species of *Fusarium* from section *Liseola* (Nirenberg and O'Donnell [1998;](#page-9-4) Leslie and Summerell [2006;](#page-8-5) Kovacevic et al. [2013](#page-8-6)). At least three members of *G. fujikuroi* complex viz., *Fusarium fujikuroi* Nirenberg, *F. proliferatum* (Mats.) Nirenberg and *F. verticillioides* (Sacc.). Nirenberg have been reported to be associated with bakanae symptoms in rice (Jeon et al. [2013](#page-8-7)). However, there role in causing bakanae disease is not fully understood (Wulff et al. [2010](#page-9-5)). We have restricted our discussion to *F. fujikuroi* as it has been found to be the most dominant and virulent species causing bakanae disease of rice.

## **Morphological, physiological and pathogenic variability**

*Fusarium moniliforme* strains have been reported to exhibit signifcant variation with respect to production of gibberellins, fusaric acid, pectic enzymes and pathogenicity (Candau et al. [1991;](#page-7-5) Li et al. [1993](#page-8-8); Ou [1985;](#page-9-1) Sidhu [1983;](#page-9-6) Sunder and Satyavir [1998](#page-9-7); Thakur [1974](#page-9-8); Wada et al. [1990](#page-9-9)). In Japan, 56 isolates of *F. moniliforme* collected from diferent hosts were characterized with respect to mating, production of microconidia, chlamydospores, gibberellins and fusaric acid (Singh and Sunder [2012\)](#page-9-0). Similarly, Thakur ([1974](#page-9-8)) classifed 48 isolates Indian isolates of *F. moniliforme* into four groups on the basis of their variable growth rate. Subsequently, Sunder and Satyavir ([1998](#page-9-7)) categorized 28 fungal isolates into fve groups based on their virulence and  $GA<sub>3</sub>$  production while, Lone et al. ([2016\)](#page-8-9) characterized 20 isolates of the pathogen and categorized then into six pathogenic groups based on their reaction pattern on a set of diferential rice lines.

In Japan, Wada et al. [\(1990](#page-9-9)) reported that the isolates sensitive to ergestrol biosynthesis inhibitors (pefurazoate) and resistant to benomyl did not produce gibberellins but all thoese isolates produced fusaric acid. The less sensitive isolates to trifumizole have been reported to be less pathogenic due to production PF smaller amount of gibberellin like substances (Hamamura et al. [1989](#page-7-6)). In China, Pan et al. [\(1997\)](#page-9-10) also reported reduced mycelial growth, sporulation and pathogenicity in carbendazim tolerant isolates.

A wide variation among 66 isolates of *G. fujikuroi* on seedling elongation was reported by Nishikado and Matsumoto ([1993](#page-9-11)). Some of the isolates caused dwarfing of rice seedlings, while the others caused elongation or had no efect on seedlings height. Sharma and Bagga ([2007](#page-9-12)) have also observed variation in symptoms production by four isolates of the pathogen. Puyam et al. ([2017\)](#page-9-13) observed that the genetic variability index of 38 isolates of *F. moniliforme*, collected from diferent Basmati cultivars in Punjab, ranged from 25 to 77% and the isolates were divided into two major clusters. Sunder and Satyavir ([1998](#page-9-7)) and Ma et al. ([2008](#page-8-10)) reported a positive correlation between  $GA_3$  production and bakanae while Thakur ([1974\)](#page-9-8) observed a similar relationship between pectic enzymes and foot rot development.

#### **Sensitivity to fungitoxicants**

Wide variations in the sensitivity of diferent strains *of G. fujikuroi* to diferent fungicides have been reported by several workers. Several strains of *G. fujikuroi* resistant to benzimidazoles (benomyl, carbendazim) and trifumizole have been detected by various workers from Japan (Hamamura et al. [1989](#page-7-6); Ishii and Takeda [1989](#page-8-11); Ogawa and Takeda [1990](#page-9-14); Omatsu et al. [1990\)](#page-9-15) and China (Hollomon et al. [1996\)](#page-8-12). Some of these strains could tolerate as high as 1000 ppm of the benzimidazole fungicides (Yasuda [1986\)](#page-10-1). A considerable amount of variation in sensitivity of the pathogen strains to pefurazoate (Wada et al. [1990](#page-9-9)), benomyl and hygromycin B (Yan et al. [1993\)](#page-9-16), carbendazim and prochloraz (Chen et al. [2007\)](#page-7-7), methoxy ethyl mercury chloride and carbendazim (Sunder et al. [1998](#page-9-17)) have been reported. Hamamura et al. [\(1989\)](#page-7-6) observed that the carbendazim sensitive isolates of the pathogen produced higher quantity of gibberellin like substances and caused high incidence of bakanae while less sensitive isolates produced smaller amount of gibberellin like substances resulting in reduced virulence though both groups produced similar amount of fusaric acid. Chen et al. ([2007](#page-7-7)) reported that resistance in *F. moniliforme* to carbendazim was stable after these strains were transferred 20 times on carbendazim-free potato sucrose agar. However, resistance to prochloraz decreased substantially after passing the strains 5–20 times on fungicide-free medium. Inoculation of plants with a mixture of conidia (1:1) from resistant and sensitive strains revealed that the carbendazim resistant strains had stronger competitive ability than the sensitive ones on rice plants while reverse was observed in prochloraz resistant strains. Iguchi and Takeuchi ([1988\)](#page-8-13) reported crossresistance in benomyl tolerant strains to thiophanate-methyl and thiabendazole.

#### **Heterothallism, genetics, sexuality and heterokaryosis**

The members of *G. fujikuroi* species complex are generally heterothallic in which sexual reproduction requires interaction between two mating types having diferent MAT allele, viz., MAT-1 and MAT-2 allele (Leslie and Summerell [2006](#page-8-5)). The heterothallic nature of the fungus was confrmed by Snyder and Sun ([1973\)](#page-9-18) in the USA and later, Chang and Sun ([1975\)](#page-7-8) in Taiwan succeeded in obtaining perithecia by mating two compatible ascospores under artifcial condition. Mating information among the isolates of *Fusarium* in section Leiseola provides important information on the genetic diversity and population genetics. *Gibberella fujikuroi* is a species complex comprising of at least 11 diferent mating populations (MPs) that are denoted by letters A through K. These are MP-A (*F. verticillioides;* Syn: *F. moniliforme*), MP-B (*F. sacchari*), MP-C (*F. fujikuroi*), MP-D (*F. proliferatum*), MP-E (*F. subglutinans*), MP-F (*F. thapsinum*), MP-G (*F. nygamai*), MP-H (*F. circinatum*), MP-I (*F. konzum*), MP-J (*F. gaditjirrii*) and MP-K (*F. xylarioides*) (Kovacevic et al. [2013](#page-8-6)). Some of these mating populations have been given *Gibberella* species names that parallel the names of their *Fusarium* anamorphs. MP-C (*F. fujikuroi*), frst identifed from Taiwan (Hsieh et al. [1977](#page-8-14)), has also been found to cause bakanae in Italy (Amatulli et al. [2010;](#page-7-9) Dal Pra et al. [2010\)](#page-9-19). The mating populations MP-A and MP-D have been found associated with bakanae disease of rice in Asia; MP-D in Africa, Australia and United states (Gupta et al. [2015](#page-7-3)) while, other mating populations viz., *F. moniliforme, F. proliferatum, F. verticillioides, F. sacchari* and *F. subglutinans* in Malaysia, Indonesia, Nepal, India, Pakistan, Bangladesh, Iran and Italy (Gupta et al. [2015\)](#page-7-3). Among various mating groups of *F. moniliforme* viz., A, B, C, D, E and F strains of group C are confned to rice (Puhalla and Spieth [1983\)](#page-9-20). In China, out of 37 *Fusarium* isolates of rice, 35 isolates belonged to A, D and F mating populations while two isolates could not be crossed with any of the non-mating populations. In pairing tests of 35 isolates for vegetative compatibility, 25 were heterokaryon and self-compatible and could be classifed into 20 vegetative compatibility groups (VCGs). The genetic diversity for the members belonging to D, E and F mating populations were 0.7, 0.8 and 1.0, respectively (Zhang et al. [1998\)](#page-10-2).

Heterokaryosis (vegetative compatibility) has been reported to occur in rice isolates of *F. moniliforme* under laboratory and feld conditions (Singh and Sunder [2012\)](#page-9-0) and was studied by using *nit* mutants (Sidhu [1986](#page-9-21)) and artifcially induced auxotrophs (Puhalla and Spieth [1985](#page-9-22)). Puhalla and Spieth [\(1983](#page-9-20)) found that the vegetative compatibility was controlled by nuclear genes (*het* or *vic* genes) and was heterogenic and allelic in all the four mating groups. Group C strains had at least four *vic* loci (Puhalla and Spieth [1985](#page-9-22)). A total of nine genes were reported for *G. fujikuroi* mating group A. Puhalla and Spieth [\(1985\)](#page-9-22) identifed 12 VCGs among the isolates of group C in China and Taiwan while Sidhu [\(1986\)](#page-9-21) and Sunder and Satyavir ([1998\)](#page-9-7) categorized 38 and 28 isolates of *F. moniliforme* in 13 and 10 VCGs, respectively using *nit* mutants. Isolates from different or same VCGs varied considerably in virulence and  $GA<sub>3</sub>$  production. Sunder and Satyavir ([1998\)](#page-9-7) did not find any relationship of vegetative compatibility with virulence pattern and  $GA_3$  production. *Nit* mutants, lacking the enzyme nitrate reductase, were recovered on chlorate containing media and their frequency of appearance in culture was strain and environment dependent (Sidhu [1986;](#page-9-21) Klittich and Leslie [1988\)](#page-8-15). The sectoring frequency on chlorate medium varied between 0.83 and 10/colony in diferent isolates of *F. moniliforme* from rice (Sunder and Satyavir [1998](#page-9-7); Sidhu [1986;](#page-9-21) Klittich and Leslie [1988](#page-8-15)) per colony in isolates of mating group A. Klittich et al. [\(1988](#page-8-16)) indicated that sectoring is controlled by many genes. They also reported that *nit* mutations occurred in 7 loci namely, *nit* 1 (a structural gene for nitrate reductase), *nit* 3 (a regulatory gene specifc for the nitrate-reduction pathway) and *nit* 2, *nit* 4, *nit* 5, *nit* 6 and *nit* 7 commonly called *Nit* M (genes controlling the production of a molybdenum containing co-factor necessary for nitrate reductase activity). Klittich and Leslie [\(1988](#page-8-15)) identifed phenotypic classes (*nit* 1, *nit* 3 or Nit M) of *nit* mutants on media containing various nitrogen sources.

Hamamura et al. [\(1991](#page-7-10)) observed that trifumizole sensitive isolates representing the same geographical area generally formed a heterokaryon, whereas isolates from diferent areas were not complementary. Isolates less sensitive to trifumizole were frequently complementary regardless of geographical origin, but these were never complementary with sensitive isolates.

In Spain, Cerda et al. [\(1994](#page-7-11)) reviewed the research work done on development of genetic tools for studying *G. fujikuroi* and described the main features of the gibberellin biosynthesis pathway in *Gibberella*. Diferent workers reported the heterothallic nature of sexual reproduction of *G. fujikuroi* and identifed sex factors viz. hermaphrodite, female, male and neuter. Two alleles *A* and *a* controlled the compatibility reactions (Singh and Sunder [2012\)](#page-9-0). Min [\(1986\)](#page-8-17) confrmed the chromosome number  $(n=8)$  in *F. moniliforme*. Inheritance of mating type, colony colour, morphology and growth rate has been reported to be controlled by single nuclear gene (Sidhu [1983](#page-9-6)). The gene for colony colour and growth rate were linked whereas, other genes showed independent assortment. Chaisrisook and Leslie [\(1990\)](#page-7-12) stated that nuclear gene controlling perithecial pigmentation in *G. fujikuroi* was expressed maternally.

#### **Host range**

*Fusarium fujikuroi* has a wide host range. Beside rice, the pathogen is known to attack wheat (Randhawa et al. [1993](#page-9-23)), mango, pineapple, cotton and pine (Amoah et al. [1995](#page-7-13); Singh and Sunder [1997\)](#page-9-24), barley, *Panicum miliaceum* (Rangaswami [1979](#page-9-25)), sugarcane, maize, sorghum, fg, asparagus (Hsieh et al. [1977\)](#page-8-14). In Taiwan Sun ([1975\)](#page-9-26) reported that neither the pathogen strains from rice did infect crops like sugarcane, maize, sorghum and asparagus nor the isolates from these crops infect rice. However, Amoah et al. ([1995\)](#page-7-13) reported that maize and rice isolates were cross-pathogenic in Ghana. In China, *F. moniliforme* var. *zhejiangensis* has been found to infect the seedlings of wheat, barley, maize (Luo [1995;](#page-8-18) Wang et al. [1990\)](#page-9-27), sorghum, sudan grass and soybean (Luo [1995\)](#page-8-18), water melon and pea (Wang et al. [1990](#page-9-27)). In addition tomato, subabool (*Leucaena leucocephala*), banana, cowpea, barnyard grass and early water grass have also been reported to serve as alternate host of the pathogen (Anderson and Webster [2005](#page-7-14); Carter et al. [2008](#page-7-15)).

## **Disease cycle**

Bakanae is a monocyclic disease. The pathogen is primarily seed borne. Howevere, it can also survive in soil in infected crop residues as thick-walled hyphae or macro-conidia. The soil borne inoculum is of less signifcance as it is relatively short lived (Watanabe [1974;](#page-9-28) Singh and Sunder [2012](#page-9-0)). The fungus is both internally and externally seed-borne and the recovery of the fungus was found to be more in lemma followed by palea (Manandhar [1999;](#page-8-19) Kumar et al. [2015](#page-8-20)). The pathogen survived in endosperm and embryo to the extent of 15.00 and 6.25% (Kumar et al. [2015](#page-8-20)). In India and Pakistan, bakanae incidence and level of highest infestation of *F. moniliforme* in naturally infested samples ranged from 0.5 to 20% (Bashyal et al. [2015\)](#page-7-16) and 21.75% (Raza et al. [1993](#page-9-29)), respectively. Sowing of 100% infected seeds resulted in production of 30–68.4% diseased seedlings (Yu and Sun [1976](#page-10-3); Yasin et al. [2003](#page-10-0); Singh et al. [2018](#page-9-30)). Likewise, the disease spread from infected to healthy seedlings was observed to be 4.4, 5.3 and 6.9% when healthy and infected seedlings were transplanted alternately in a row, in alternate rows and simultaneously in a hill (Singh et al. [2018](#page-9-30)). In artifcially inoculated soil also, the disease development was observed to the extent of 8.5% (Singh et al. [2018](#page-9-30)).

Seed infection occurs through secondary air-borne conidia and ascospores which are discharged from diseased plants during heading to harvest stage. The fungus grows intercellular in stigma and anthers within 48 h which ultimately reaches and covers the ovary (Kagiwata [1963](#page-8-21); Yu and Sun [1976](#page-10-3)). Fungus remained viable in seeds and infected plants only for 4–10 months (Kanjanasoon [1965](#page-8-22)). On the other hand, the survival of pathogen in seed has been reported for 10–28 months (Dodan et al. [1994;](#page-7-17) Misra et al. [1989](#page-8-23), Sunder and Satyavir [1997\)](#page-9-31) and for 22–26 months at 10 °C and 40% relative humidity (Manandhar [1999\)](#page-8-19). Seed harvested during wet season had more *G. Fujikuroi* infected seed than those harvested during the dry season regardless of cultivar and number of inoculations with conidial suspension during anthesis (Manandhar [1999](#page-8-19)).

The fungus infects rice seedlings through roots and the base of the stem and becomes systemic multiplying within the infected tissues. The pathogen was found to be discontinuously distributed in infected seedlings (Nisikado and Kimura [1941](#page-9-32); Thomas [1931\)](#page-9-33) with a maximum recovery from the basal 3–4 inches of the stem (Singh and Sunder [2012](#page-9-0)).

Chan et al. [\(2004](#page-7-18)) observed that sprouting period was the most efective period for disease occurrence. They observed positive correlations between disease incidence and soaking temperature  $(r=0.8757)$  and disease incidence and sprouting temperature ( $r = 0.9570$ ) in a temperature range of 28–34 °C. The most important sprouting temperature was 34 °C and the most important inoculation time for soaking and sprouting period was 18 and 24 h, respectively. The correlation coefficient between disease incidence and soaking time in these two periods were 0.9620 and 0.8947, respectively. Bud phase was most suitable phase for inoculation of the pathogen.

Sunder and Satyavir ([1997](#page-9-31)) reported that the pathogen also survive in diseased debris for 10–28 months and served as a primary source of inoculum. Though soil inoculum is not very signifcant in areas where one rice crop is taken in year, the fungus can survive for a limited period in soil through conidia, ascospores, thick-walled hyphae (Sun [1975\)](#page-9-26) and sclerotia (Sharma and Singh [1978\)](#page-9-34). Yu and Sun ([1979\)](#page-10-4) reported the survival of *F. moniliforme* up to 280 days in dry soil [10% moisture holding capacity (MHC)], which decreased to 115 days at 45% MHC and only 45–70 days at 100% MHC. Mandal and Chaudhuri [1988](#page-8-24)) and Sunder and Satyavir [\(1997\)](#page-9-31) also recorded longer survival of the pathogen in comparatively dry soil (5–10% MHC). On the other hand, Kanjanasoon [\(1965\)](#page-8-22) observed that survival of pathogen in soil is limited. He reported that freshly inoculated soil resulted in 93% seedling infection while, the percentage of infection drastically reduced to 0.7% and 0.0% after 90 and 180 days of inoculation respectively.

### **Factors afecting disease development**

Intensity of the disease has been aggravated by changes in the climatic parameters. Bakanae incidence was more when rice nursery was raised during summer season and in dry seed beds as high temperature and relatively humidity favoured the disease development. More disease has been reported in upland nurseries than in fooded conditions (Anonymous [1975](#page-7-19); Hashioka [1971\)](#page-7-20). In Thialand and Ceylon, disease was found to be more severe in hot season and at low water levels. Mandal and Chaudhuri ([1988\)](#page-8-24) reported a reduction in pathogen population in soil due to application of higher dose of nitrogenous fertilizers. Sprouting period has been observed to be the most efective period for disease occurrence (Chan et al. [2004](#page-7-18)). The most important sprouting temperature was 34˚ C and the most important inoculation time for soaking and sprouting period was 18 and 24 h, respectively. Bud phase was most suitable phase for inoculation of the pathogen. The grain infection is known to increase at high temperature and poor insolation during fowering (Hino and Furata [1968;](#page-7-21) Takeuchi [1972](#page-9-35)).

Rajagopalan and Bhuvaneswari [\(1964](#page-9-36)) and Kanjanasoon [\(1965](#page-8-22)) found more infection by sowing un-germinated seeds in infested soil in comparison to germinated seeds. The disease incidence has been observed to be more in basmati rice cultivars particularly when the nursery is uprooted under upland/vattar condition (Singh et al. [2018](#page-9-30)).

#### **Disease management**

#### **Host Resistance**

Host plant resistance is the most efective and practical approach for disease management. Several rice genotypes have been found resistant to bakanae disease by various researchers under artifcial inoculation conditions, which can be utilized as donors for incorporating bakanae resistance in rice cultivars (Table [1\)](#page-4-0).

There is a very limited knowledge on mapped loci governing resistance to bakanae disease. Yang et al. [\(2006](#page-10-5)) identifed two QTLs viz. QB 1 and QB 10 derived from the chinese japonica cultivar Chunjiang 06 on chromosome 1 and 10 while Naeem et al. ([2016\)](#page-8-25) identifed qBK1 and qB2 on chromosome 1 and 2 governing resistance to bakanae. Hur et al. [\(2015](#page-8-26)) identifed a major QTL (qBK1) using NILs derived from a cross between rice varieties Shingwang

<span id="page-4-0"></span>**Table 1** Rice genotypes showing resistance to foot rot and bakanae

Country	Resistance sources	References
India	CN 1722-4, Haryana Basmati 1, Haryana Mahak, HKR 08-425, HKR 12-406, KMR 1-41, MAUB 2014-1, MAUB 2014-2, NDR 6222, NDR 6345, NP 973-2, Pusa Basmati 1460 (Improved Pusa Basmati 1), Pusa 1557-06-28-188-1-17, RDN 02-01-8-18-11-9, RSK 1045, Shaan, SJR 70-3-2, SJR 129, Super Basmati	Kumar et al. $(2016a)$
	Co 18, Co 22, Adt 8, PTB 7, GEB 24, GSL-5, GSL-9, GSL-12, GSL-36, GSL-44, GSL-60, GSL-66, GSL- 67, GSL-68, IR 20, IR 26, IR 32, IR 38, IR 44, IR 45, Punjab Mehak, Pusa Basmati No.1	Kumar et al. $(2014)$
	HKR 96-561, HKR 96-565, HKR 07-40, HKR 07-53, HKR 08-13, HKR 08-21, HKR 08-22, MAUB 2009-1, Sunder et al. (2014) PAU 3456-46-6-1-1, PNR 600, RPDN 01-2-10-9	
	Athad, Appunu, BPT 5204, C 101A51, Chandana, Himju, IR 58025B, Panchami, PAU 201, Pusa 1342, Peeli Fiyaz et al. (2014) Badam, Suphala, Varun Dhan	
	GSL-5, GSL-9, GSL-12, GSL-36, GSL-44, GSL-60, GSL-66, GSL-67, GSL-68	Ahangar et al. $(2012)$
	Pakistan IR 6, KS 133	Ghazanfar et al. (2013)

and Ilpum on long arm of chromosome 1. Subsequently, Fiyaz et al. ([2016](#page-7-25)) detected two novel QTLs viz. qBK1.2 and qBk1.3 on short arm of chromosome 1. The major efect QTL designated as qBK1.2 having 55 annotated genes was mapped on 0.26 Mb region between RM5336 and RM10153. In addition, two novel genomic regions qBk1\_628091 and qBK4\_317509550 conferring bakanae resistance have also been identifed on chromosome 1 and 4 (Volante et al. [2017](#page-9-38)).

Basmati/scented rice varieties have been found more susceptible to bakanae than the non-scented rice cultivars (Singh et al. [2018](#page-9-30)). Coarse varieties were more resistant to bakanae disease than the fne ones (Ghazanfar et al. [2013](#page-7-24)). Rice genotypes carrying dwarf and semi-dwarf genes *d 29, Sd 6* or *Sdq (t)* exhibiting resistance to bakanae disease can be used in developing disease resistant rice cultivars (Ma et al. [2008](#page-8-10)).

The disease reaction of some genotypes are known to vary with crop stage. In China, Lu [\(1994\)](#page-8-29) observed that rice genotype Longjiao 86074-6 exhibited resistance at seedling stage and moderate susceptibility at the adult stage. Likewise, genotype Qingxi 96 showed moderately resistant reaction at the seedling stage but resistant at adult stage. However, the rice genotypes namely, Zupei 7, Dongrong 84-21, G-6 and Sui 89-17 were moderately resistant both at seedling and adult stages. Khan et al. [\(1999\)](#page-8-30) observed that resistance in rice genotype IR 6 was monogenic and recessive while dominant in KS 282.

#### **Agronomic practices**

Use of clean non-infected seed, hot water treatment, proper selection of geographical area, time and method of nursery sowing and transplanting, removal and destruction of infected plants from the feld and balanced fertilization help in reducing the incidence of bakanae disease by minimizing the inoculum and seed-borne infection of the pathogen (Gupta et al. [2015](#page-7-3)). Hot water treatment has been found efective in reducing the seed infection and bakanae incidence (Miyasaka et al. [2000;](#page-8-31) Yamashita et al. [2000](#page-9-39)). Bagga et al. [\(2007\)](#page-7-26) recorded minimum disease incidence in July and planted crop owing to the prevalence of lower temperature during infection. Higher levels of nitrogen and potassium and soil amendment with neem cake and press mud have also been found to suppress the bakanae incidence (Kumar et al. [2016a](#page-8-27)). Seedlings transplanted from nursery box had more bakanae compared to transplanting of rice seedlings from protected nursery (Sung and Yang [1985](#page-9-40)). Minimizing the root injury by uprooting paddy nursery in standing water has been found helpful in preventing the potential entry of bakanae pathogen and ultimately, in reducing the disease incidence. The disease incidence was signifcantly low when carbendazim treated and untreated seeds were sown under dry condition compared to conventional method of sowing sprouted seeds in puddled beds (Sunder et al. [2014\)](#page-9-37).

### **Bio‑control and botanical extracts**

The work on biological control of bakanae has been reviewd by Kumar et al. [\(2014](#page-8-28)) and Gupta et al. [\(2015](#page-7-3)). Several fungal and bacterial antagonists and botanical extracts have been reported to suppress the mycelial growth, conidial production & germination of *F. moniliforme* along with a significant reduction in disease incidence (Table [2](#page-5-0)).

Bhramaramba and Nagamani ([2013](#page-7-27)) and Sharma et al. ([2014](#page-9-41)) reported the antagonistic activity of *Trichoderma*

<span id="page-5-0"></span>**Table 2** Bio-control agents and botanical extracts found efective against *F. moniliforme*

Bio-control agents/botanicals extracts	Remarks
A. Bio-control agents	
<i>Pseudomonas fluorescens</i> isolates PF-9, PF-13 and <i>Bacillus thuring-</i> <i>iensis</i> isolate B-44	The antagonists produced lytic enzymes [Chitinase and $\beta$ -1,3-glucanase $\{endo-1, 3(4) - \beta-glucanase\}\$ , siderophores, salicylic acid and hydro- gen cyanide and suppressed fungal growth & bakanae incidence
Trichoderma asperellumSKT-1	-do-
<i>Talaromyces sp. KNB 422</i>	-do-
Bacillus subtilis and B. megaterium	$-do-$
Bacillus subtilis, Trichoderma harzianum and T. virens	Most effective in reducing bakanae. However, these were inferior to thiophanate methyl thiram 80 WP at 2 g/l
<b>B.</b> Botanicals extracts	
Extract of Azadirachta indica, Decalepis hamiltonii, Lawsonia iner- mis, Andrographis paniculata, Eucalyptus citriodora	Inhibited conidial production, germination and mycelial growth of $F$ . Moniliforme
Oil of Hedychium spicatum and Acorus calamus	Completely inhibited the growth at $1.0 \times 10^3$ ml/l and $0.5 \times 10^3$ ml/l, respectively and exhibited both fungistatic and fungicidal properties depending upon the concentration
Aerated vermicompost tea	Highly effective in controlling the disease in field trials

species to control bakanae disease of rice. Motomura et al. [\(1997\)](#page-8-32) observed that seed treatment with metabolites produced by five soil-borne bacteria efficiently inhibited bakanae development and were better than benomyl, trifumizole, pefurazoate and prochloraz. Dehkaei et al. ([2004\)](#page-7-28) observed that seed treatments with antagonists, such as *Bacillus subtilis, Trichoderma harzianum* and *T. virens*, prior to inoculation was better than their application after seed infection. Dry seed treatment of *T. viride* at 5 g/kg seed before sowing provided about 60% disease control (Kumar et al. [2016b](#page-8-33)). Lu et al.  $(1998)$  $(1998)$  $(1998)$  reported better efficacy of antagonists applied as seed treatment than spray application. They also observed improved efficacy of bio-control agents when used in combination. Treatment with FYM 10 t/ha+*Trichoderma*+*Pseudomonas* signifcantly lowered the disease incidence under felds conditions (Wyawahare et al. [2012\)](#page-9-42). Seed treatment with antagonistic yeasts and thermotherapy has given promising results (Matic et al. [2014\)](#page-8-35). Seed treatment and soil incorporation of *Pseudomonas aureofaciens* reduced bakanae incidence by 71.7 to 96.3% (Rosales and Mew [1997\)](#page-9-43). An efective disease control has also been observed by using the bacterial strains B-916 and P-91 in China (Chen et al. [1998](#page-7-29)). Kazempour et al. ([2007](#page-8-36)) reported that molecular analysis using PCR based RAPD method is useful to diferentiate strains of bio-control agents efective against *F. moniforme* at the intraspecifc level.

Extracts of *Mimosopus elengi* and *Acacia nilotica* provided more than 85% growth inhibition of *F. moniliforme* (Mohana et al. [2011](#page-8-37)). Essential oils of *Cymbopogon citratus*, *Ocimum gratissimum* and *Thymus vulgaris* were found highly effective and curtailed the seed-borne infection of bakanae pathogen by 95–100% (Nguefack et al. [2007\)](#page-8-38). In Korea, extract of *Ginkgo biloba* outer seed coat has been reported to provide 80% reduction in bakanae incidence (Oh et al. [2016](#page-9-44)).

## **Chemical control**

Seed soaking in salt solution has been advocated to reduce the seed-borne inoculum by separating the light-weight, infected seeds from seed lots. Sunder et al. [\(1998](#page-9-17)) reported that soaking of seeds in MEMC  $(0.5 \text{ g/l})$ , MEMC 1 g/l) and propiconazole (2 ml/l) solution for 36, 24 and 24 h resulted in complete eradication of the pathogen from grains, respectively. Seed dressing or slurry treatment with benomyl, carbendazim, carbendazim + mancozeb, prochloraz, propiconazole, thiophanate-methyl, benomyl+thiram and thiophanate methyl+thiram, Shibaoke, Jinzhongling, kasugamycin, triforine, prochloraz, prochloraz+carbendazim, mancozeb+benomyl, carboxin+thiram, iprodione, iprodione+triticonazole, ipoconazole, fudioxonil, ferimzone, tebuconazole, trifoxystrobin+tebuconazole and kresoxim methyl have been found efective in reducing bakanae incidence and in enhancing grain yield of rice in diferent parts of the world. Seed treatment and soil drenching of benzimidazole fungicide Derosal and seed & seedling dip in Bavistin  $(0.2\%)$  proved highly effective in checking the bakanae disease. Soil drenching with carbendazim and difenoconazole have also been found effective in managing the disease. Seed treatment with Bavistin at  $0.05$  g + Streptocycline  $0.01$  g/l water for 12 h and with talc formulation of *T. harzianum* at 15 g/kg of seed and seedling root dip with T. *harzianum* at 15 g/l of water was most efective against bakanae disease (Singh and Sunder [2012;](#page-9-0) Kumar et al. [2014](#page-8-28), [2016b](#page-8-33); Gupta et al. [2015](#page-7-3); Hossain et al. [2015;](#page-8-39) Raghu et al. [2018\)](#page-9-45).

The efficacy of fungicides varied with seed treatment method and type of formulations. Wet seed treatment with organo-mercurial fungicides (Takeuchi [1972\)](#page-9-35) and EC formulation of trifumizole (Suzuki et al. [1994](#page-9-46)) provided better disease control than their dry treatment and WP formulation, respectively. This was due to presence of higher amount of fungicide on the seed and early permeation of EC treatments into seeds through the husk.

Seed dressing with Chitosan S-II, soaking of seeds in 500 ppm of pefurazoate and thermal (72° C for 5 min) and carbendazim treatment provided more than 90% disease control. Fungicides namely, trifumizole, propiconazole, prochloraz, NF 30, pefurazoate, captan+thiabendazole, trifumizole and phenyl pyrrole fungicide CGA 173506 have been reported to be highly effective for controlling benzimidazole (benomyl), thiophanate-methyl and thiobendazole tolerant strains of *F. moniliforme* which are frequently encountered in Japan (Kumar et al. [2014](#page-8-28)).

Seedling treatment with Bavistin or benomyl at 0.1% for 6 and 8 h has also been found very efective against bakanae (Bagga and Sharma [2006\)](#page-7-30). Propiconazole (Tilt 25 EC) at 0.05% was the most efective treatment in curtailing the disease. However, this fungicide showed phytotoxicity on rice. Foliar application of benzimidazoles (benomyl and carbendazim) at 0.1% signifcantly reduced the disease incidence, increased the grain yield and reduced the seed-borne infection by *F. moniliforme*. In addition, sand mix application of carbendazim in nursery bed at  $1 \text{ g/m}^2$  7 days before uprooting of seedlings and as seedling dip in 0.1% carbendazim solution for 3 h before transplanting has also been reported to impart efective disease control in transplanted crop (Kumar et al. [2014](#page-8-28); Sunder et al. [2014\)](#page-9-37).

#### **Conclusion**

Foot rot and bakanae of rice is widely distributed throughout the globe. Infected seedlings become lanky, pale yellow and taller than the healthy seedlings which ultimately die and result in substantial reduction in grain yield particularly in scented rice varieties. The pathogen is primarily seed-borne but also known to survive in soil. Various aspects of the disease have been studied well in diferent countries. However, further research is necessary on host–pathogen interaction, racial profling, development and commercialization of highly effective strains of bio-control agents, QTL mapping and virulence pattern, biochemical and molecular aspects of pathogenesis and disease resistance. Besides, characterization of environmental conditions for disease epiphytotics, formation and maturation of perithecia, effect of soil-applied pesticides, soil solarization and fooding on survival of the pathogen, role of internal seed-borne infection and soilborne inoculum in annual recurrence and epidemiology of the disease need further investigation. An integrated sustainable approach involving development of resistant varieties, use of efficient bio-control agents and effective bio-degradable fungicides along with selection of geographical area for seed production is required for better disease management.

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