

**REVIEW PAPER**

**BAKANAE OF RICE -AN EMERGING DISEASE IN ASIA**

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**ABSTRACT**

Bakanae or foot rot disease caused by *Fusarium fujikuroi* Nirenberg is an important emerging disease of rice across the world. It is responsible for high yield losses ranging from 3.0-95.4% and its incidence varies with regions and cultivars grown. It is one of the emerging problems in rice, particularly in basmati/scented rice in India during recent years and becoming more serious threat to sustainable rice production in other parts of the rice growing world. Currently, seed treatment with fungicides is the most important disease management strategy used worldwide after the use of resistant varieties. Various aspects of the disease covering history, significance, pathogen survival, variability to genomics and management issues that will be useful for researchers and other stakeholders to bring fruitful research on yield loss assessment, epidemiology, host-pathogen interaction, racial profiling, decision support system and integrated sustainable disease management practices which are still lacking about the disease have been discussed in greater detail.

**Keywords:** Bakanae, *Fusarium fujikuroi*, Significance, Variability, Genomics, Resistance, Management.

**INTRODUCTION**

Rice is a major food commodity after wheat and maize throughout the world. Globally, it is cultivated on 166 mha of land with an annual production of about 745.17 mt of paddy and average productivity of 4.48 t/ha (FAO, 2014). It is estimated that 880 mt of rough rice need to be produced by 2025 with an increment of 70% to satisfy the burgeoning population requirement (Lampe, 1995). In India, the total area under rice cultivation during the year, 2013 was 42.41 mha with annual production of 104.40 mt of paddy and average productivity of 3.59 t/ha (www.iasri.res.in). It has been estimated that by the year 2021, India needs to produce 113.3 mt rice to meet the increasing food demand of the country (Kumar, 2009). Higher rice yield can only be obtained through improved cultivars and integrated crop and pest management technologies (FAO, 1995; Kumar, 2013; Prajapati *et al.*, 2013). Major constraints for the realization of higher yields of rice crop are its susceptibility to insect-pests, diseases and abiotic stresses. However, the diseases caused by fungi, bacteria, viruses and nematodes are serious threats to sustain higher production and yield stability in India. Now-a-days, the scenario of rice diseases is changing due to extensive use of fertilizers, irrigations, pesticides and high yielding cultivars. The minor diseases like, false smut, bakanae, sheath rot and grain discoloration which were of less significance earlier, are now emerging as serious threat to rice cultivation. Bakanae disease caused by *Fusarium fujikuroi* Nirenberg [*F. moniliforme*

(Sheld.), telomorph: *Gibberella fujikuroi* Sawada, Wollenweber] emerged as a significantly important disease in recent times in Asia and other rice growing countries of the world (Singh and Sunder, 2012). The incidence of bakanae disease has increased steadily, particularly on aromatic rice cultivars in all rice growing states of India which causes severe qualitative and quantitative yield losses to rice crop (Bashyal *et al.*, 2014; Gupta *et al.*, 2014). The research on bakanae disease and its sustainable management are the need of the hour and must be given top priority for disease free quality seed production, realizing higher yield potential of aromatic rice and to get edge in rice trade at international market before it is too late. Hence, the current research review is an attempt to provide the information on various aspects related to bakanae disease of rice to help researchers and other stakeholders to bring long-term coherence for this emerging disease.

**History:** Bakanae disease was first identified during 1828 in Japan. Shotaro Hori (1898) first time demonstrated the fungus *Fusarium heterosporium* Nees induced the bakanae symptom in rice plants. It was later put in the genus *Gibberella* under the name *G. fujikuroi* (Sawada) (Ito and Kimura, 1931). In 1917, Sawada indicated that the elongation of rice seedlings might be due to some stimuli derived from bakanae fungus hyphae. Subsequently, Kurosawa (1926) demonstrated the hypertrophic and elongation effect from culture filtrate of dried rice seedlings, rice plants and other sub-tropical weeds which was also called as "bakanae effect" of

fungus and owing to this, disease is called as bakanae disease. Kurosawa also concluded that bakanae fungus secretes a chemical which suppress the chlorophyll formation, root growth and encourage stem elongation. Seto (1928) named the disease 'Bakanae Byo' which was caused by a *Fusarium* sp. isolated from infected tissues of rice plants and the culture filtrate of the causal fungus able to produce the bakanae symptom, as mentioned by Kurosawa. There has been disagreement among plant pathologists for the nomenclature of bakanae fungus. In 1930s, the imperfect stage of the fungus was described by H.W. Wollenweber (1935) as *Fusarium moniliforme* (Sheldon) and the perfect stage *Gibberella fujikuroi* (Sawada) Wr. The terms 'Fujikuroi' and 'Saw' in *G. fujikuroi* (Sawada) Wr. were derived from the name of two distinguished Japanese plant pathologists, Yosaburo Fujikuro and Kenkichi Sawada (Watanabe and Umehara, 1997).

#### Geographical distribution and economic significance:

The bakanae fungus *F. fujikuroi* has become widely distributed in tropical as well as temperate environment and occurring throughout rice growing regions of the world (Fig.1). It is one of first disease of rice described scientifically responsible for yield losses ranging from 3.0-95.4% varied with regions and cultivars grown (Pavgi and Singh, 1964; Kanjanasoon, 1965; Ou, 1987; Hajra *et al.*, 1994; Singh and Sunder, 2012). The disease has been reported from the South East Asia, Africa, America and European countries (Desjardins *et al.*, 2000). It is economically important in the Asian rice growing areas owing to the significantly large amount of yield losses estimated at approximately 20% in epidemic areas (Cumagun *et al.*, 2011). It is also a serious problem in countries like, Japan, Taiwan, Thailand, Turkey, California and Philippines (Nelson *et al.*, 1993; Webster

and Gunnell, 1992; Cumagun *et al.*, 2011). Ito and Kimura (1931) reported up to 20 – 50% losses in Japan and Kanjanasoon (1965) found 3.7-14.7% loss in Thailand. The disease was observed for the first time during 2006 from Macedonia by Karov *et al.* (2009). In California, bakanae disease was first time observed during the year 1999 and by 2001; it spread rapidly in the majority of the rice production areas along with the threat of significant yield loss (Anderson and Webster, 2005). In Pakistan, the bakanae disease was first time reported by Khokhar (1990) and now it has become a major disease since last decade causing 10-50% yield losses (Bhalli *et al.*, 2001; Khokhar and Jaffrey, 2002; Ghazanfar *et al.*, 2013). In Bangladesh up to 25% yield loss has been reported in susceptible cultivars (Hossain *et al.*, 2011). In Nepal, local and improved varieties of rice have been found susceptible to the bakanae disease along with 40% yield reduction (Desjardins *et al.*, 2000). Surek (1997) observed 10-15% incidence of bakanae on various cultivars of rice in Turkey. In India, the prevalence and incidence of bakanae disease has been reported particularly on basmati rice cultivars (Bashyal *et al.*, 2014; Gupta *et al.*, 2014). The yield losses ranging from 15-25% have been reported from Uttar Pradesh, Assam, Andhra Pradesh, Tamilnadu, Haryana and Punjab states of India. (Pavgi and Singh, 1964; Rathaiiah *et al.*, 1991; Pannu *et al.*, 2012; Sunder *et al.*, 2014). Apart from yield losses studies, the bakanae pathogen was also found to be associated in highest percentage (1-24%) in seeds of different basmati rice cultivars (Butt *et al.*, 2011; Bashyal and Aggarwal, 2013) and showed the profound effect of the disease on seed quality. The observations and surveys from 2008-2014 in Northern states of India also showed the incidence of bakanae disease ranged from 1.2-40%, especially in basmati rice cultivars (Gupta *et al.*, 2014).



Fig. 1. Geographic distribution of bakanae disease

**Symptomatology:** Bakanae disease of rice is also known as “foot rot” in India, “white stalk” in China, “man rice” in the Philippines and “bakanae-byo or elongation disease” in Japan. Bakanae is a Japanese word which means bad or naughty seedling referring to the abnormal elongation, “thin noodle seedling”, “foolish seedling” or “stupid rice crop” (Sun and Snyder, 1981). The disease is caused by one or more *Fusarium* species and complex of disease symptoms including seedling blight, root rot, crown rot, stunting, and the most classical symptoms of etiolation, hypertrophy effect or excessive elongation of infected plants, foot rot, seedlings rot, grain sterility, grain discoloration with ultimate effect on yield and seed quality have been recorded from different regions of the world (Sun and Snyder, 1981; Ou, 1985; Webster and Gunnell, 1992; Desjardins *et al.*, 2000). Sasaki (1973) reported lesion formation on rice leaves, however, Sun (1975) considered that no vegetative parts above ground are sites for the infection. Sasaki (1976) also reported the presence of elongation symptoms on ratoon rice plants in Japan. The bakanae infected seedlings appeared as taller with chlorotic stems and leaves which become yellowish-

green to pale in colour in later stages. Diseased plants showed yellowish green elevated flag leaves with more horizontal orientation. The disease can build up in a crop very fast and a field with bakanae will remain uneven throughout the growing season. Therefore, it can be observed in nursery beds as well as in field from a distance (Fig. 2A & B). It attacks basically on all parts of the plant *viz.*, roots, crowns, stems, leaf sheaths and panicles throughout the season. Infected plants may die during crop growth period, however, some plants survive up to maturity and they have few tall, thin and lanky tillers with white empty chaffy panicles. Generally, panicles topple down due to their weight and plants die prior to their maturity (Webster and Gunnell, 1992). Pink sporodochia of the pathogen could be observed at the junction of the palea and lemma of the damaged grains. The development of adventitious roots from lower nodes of stem has been reported from India and the disease was described as foot rot (Thomas, 1931). Whitish-pinkish fungal growth also has been noticed from lower portion of the infected plants which contaminated the seed during harvesting (Fig. 2C) (Surek and Gumustekin, 1994).



### Host range of the pathogen

**Primary hosts:** Primary hosts of bakanae pathogen have been reported as rice, maize, barley, sorghum, sugarcane, wheat, pine, rye and asparagus from Asia, Africa, South East Asia and United States (Hsieh *et al.*, 1977; Kuhlman, 1982; Puhalla and Spieth, 1985; Wulff *et al.*, 2010; Petrovic *et al.*, 2013).

**Alternate hosts:** Alternate hosts *viz.*, tomato, cowpea, banana, subabool, proso millet, early water grass and barnyard grass have been found susceptible to bakanae disease and may also serve as reservoir of inoculum in the field (Anderson and Webster, 2005; Carter *et al.*, 2008).

**Isolation and pathogenicity:** Isolations of the pathogen was made on common medium (Potato Dextrose Agar), selective medium (Peptone Pentachloronitrobenzene Agar) and natural medium (Spezieller Nährstoffarmer Agar, Carnation Leaf Agar) for fungal culture (Zainudin

*et al.*, 2008a; Dal Pra *et al.*, 2010). Normally, the fungal cultures were incubated at  $25 \pm 1$  °C for 8 days, while, Burgess *et al.* (1994) also obtained good sporulation and growth of cultures after incubation in a room lighted with near-ultraviolet wave lengths and fluctuating temperatures regime *i.e.*, 25 °C during day and 22 °C in night under 12 hour photoperiod. In addition, the soil dilution method has also been used to isolate pathogen inoculum from soil around root zone of infected plants (Sharma and Singh, 1997; Saremi and Farrokhi, 2004; Saremi *et al.*, 2008). A perusal reveals that bakanae disease of rice is predominantly caused by *F. fujikuroi* (Nirenberg, 1976) being more virulent than others. However, other *Fusarium* spp. in pathogenicity tests also shown to induce bakanae symptom on rice seedlings and plants (Zainudin *et al.*, 2008a; Dal Pra *et al.*, 2010; Wulff *et al.*, 2010). Seed inoculation was found to be the most suitable method for assessing bakanae disease development compared to soil inoculation method

(Agarwal *et al.*, 1989; Zainudin *et al.*, 2008a; Karov *et al.*, 2009; Amatulli *et al.*, 2010; Quazi *et al.*, 2013).

**Pathogen identification:** Bakanae disease of rice was first described in 1828 in Japan (Ito and Kimura 1931), however, it is not cleared till date which *Fusarium* species is associated with multiple symptoms of the disease. These *Fusarium* species could differ with geographic as well as on climatic variations. The causal organism was first time identified by Hori (1808) as *F. heterosporium* Nees. The sexual stage of the fungus was described as *Lisea fujikuroi* by Sawada (1917) which was amended in 1931 to *Gibberella fujikuroi* by Ito and Kimura, who also identified the asexual stage as *F. moniliforme* (Sun and Snyder, 1981). Initially, the pathogen responsible for the bakanae disease of rice was identified as *F. moniliforme* Sheldon (Snyder and Hansen 1945; Booth 1971; Nirenberg, 1976, Ou, 1987), later it was re-identified as *F. fujikuroi* Nirenberg (Nirenberg, 1976), the anamorph of *G. fujikuroi* Sawada. Some early authorities considered *G. fujikuroi* to be a single species (Snyder and Hansen, 1945) however, this taxon comprise a number of distinct species, now collectively termed the *G. fujikuroi* species complex (GFSC). Lately, Hsieh *et al.* (1977); Kuhlman (1982) and Leslie *et al.* (1996) have used formation of the sexual stage to distinguish mating population, or biological species within this group. Secondary metabolites, particularly mycotoxins and mating populations (MPs) are important characters for identification of *Fusarium* to species and sub-species levels (Leslie and Summerell, 2006; Zainudin *et al.*, 2008b). Studies on chemotaxonomic criteria indicated that each *Fusarium* species has a specific profile of secondary metabolite. Thus, physiological studies, especially by using chemotaxonomic criteria may serve as supplements to morphological characteristics in delimitation of *Fusarium* species (Nelson *et al.*, 1993). Normally, different species of *Fusarium* produced different profiles of secondary metabolites viz., *F. verticillioides* produce fumonisin B1 (FB1) and little or no moniliformin (MON), whereas *F. fujikuroi* produce gibberellic acid (GA<sub>3</sub>), FB1 and MON (Marasas *et al.*, 1986; Zainudin *et al.*, 2008b). The information on secondary metabolites is useful for assessing the risk of mycotoxin contamination in rice as well as for assisting in the identification of closely related species such as *F. fujikuroi* and *F. proliferatum*. *F. fujikuroi* regarded as the most virulent species causing bakanae disease, produced excessive gibberellin hormones which is responsible for internode elongation of bakanae infected plants (Zainudin *et al.*, 2008b). However, gibberellin was low or absent in *F. proliferatum* (Desjardins *et al.*, 2000). The increase in plant height might be due to the ability of the inoculated species either genetically or under mutation to produce gibberellin. Proctor *et al.* (2010) reported five strains of *F. proliferatum* that could produce gibberellins and

assumed that the strains might be a hybrid (mutant) between *F. fujikuroi* and *F. proliferatum*, and the gene for gibberellic acid biosynthesis might be transferred to the hybrid strain. This assumption is further supported by Cumagun *et al.* (2011) who suggested that mixed reproduction and gene flow might be responsible for formation of new genotype. The other explanation for bakanae disease symptoms and excessive increase in plant height might be due to production of metabolites other than gibberellins. Desjardins *et al.* (2000) reported the bakanae disease due to *F. proliferatum*, which induce elongation and production of gibberellins in infected plants. Phylogenetically, *F. proliferatum* and *F. fujikuroi* are closely related species which were commonly distinguished through mating types, chemotaxonomic criteria and molecular markers (Leslie and Summerell, 2006).

Morphologically *F. fujikuroi* forms white mycelia that become grey violet or magenta with age. Sporodochia normally are absent but, if present they are pale orange. When sporodochia are present, they contain macroconidia. Many isolates formed very few or no sporodochia or lose this ability following repeated subcloturing. Macroconidia are relatively slender with tapered apical and poorly developed basal cell with 3 to 5 septa. Microconidia are oval or club shaped with flattened base with 0 to 1 septa. False heads and chains of short to medium length are produced from polyphialides, which may proliferate and often form monophialides, whereas, chlamydospores are absent (Leslie and Summerell, 2006).

Mating populations (MPs) are also used to solve these taxonomic difficulties and to distinguish the species. Mating pattern within each of these MPs are heterothallic and governed by two alleles at a single mating-type locus. Members of the same MPs are sexually fertile with one another but not with members of different MPs. *F. fujikuroi* is an important pathogen with wide variability in pathogenicity, wide host range, and in many *Fusarium* spp., various formae specialis (f. sp.) and physiological races (Zemankova and Lebeda, 2001). Sun and Snyder (1981) produced perithecia by crossing strains of *F. fujikuroi* in the laboratory. Subsequently four reproductively isolated groups of *G. fujikuroi* designated as A, B, C, and D. The mating group C was genetically interfertile strains from rice (Hsieh *et al.*, 1977; Kuhlman, 1982). Mating population C (MP-C) (anamorph, *Fusarium fujikuroi*) was first identified among rice strains from Taiwan (Hsieh *et al.*, 1977). It has been also found responsible for bakanae disease in Italy (Amatulli *et al.*, 2010; Dal Pra *et al.*, 2010). Mating population A (MP-A) (anamorph, *F. verticillioides*, Synonym: *F. moniliforme*) and mating population D (MP-D) (anamorph, *Fusarium proliferatum*) have been isolated from rice from Asia, and MP-D has been isolated from rice from Africa, Australia, and the United States

(Amoah *et al.*, 1995; Desjardins *et al.*, 1997). Group C is confined to the rice, while, the other groups have sugarcane, pine, rye, wheat, asparagus, corn or sorghum as their major hosts (Puhalla and Spieth, 1985). Kuhlman (1982) indentified a fourth mating population termed as D and described four varieties of *G. fujikuroi* viz., *moniliformis*, *subglutinans*, *fujikuroi* and *intermedia* basing on mating population, ascospores, perithecia size, phialide type and microconidial formations. Later, additional genetic studies have distinguished nine biological species or mating populations (designated by letter A to I) within the *G. fujikuroi* species complex (Leslie and Summerell 2006). *G. fujikuroi* species complex is generally designated as section *Liseola*, comprising of nine biological species designated as MP-A (*F. verticillioides*), 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*) and MP-I (*F. konzumi*) (Leslie and Summerell, 2006). *F. fujikuroi* causing bakanae disease of rice has been found frequently abundant and most virulent species in most of the studies all over the world (Zainudin *et al.*, 2008a & b; Amatulli *et al.*, 2010; Zainudin and Salleh, 2010; Nancy, 2002) while, other *Fusarium* spp. namely, *F. moniliforme*, *F. proliferatum*, *F. andiyazi*, *F. verticillioides*, *F. sacchari* and *F. subglutinans* also have been isolated and found to be associated with bakanae disease from various countries viz. Malaysia, Indonesia, Nepal, India, Pakistan, Bangladesh, Iran and Italy (Agarwal *et al.*, 1989; Khokhar, 1990; Desjardins *et al.*, 2000; Saremi and Farrokhi, 2004; Zainudin *et al.*, 2008a & b; Dal Pra *et al.*, 2010; Wulff *et al.*, 2010; Bashyal and Aggarwal, 2013; Quazi *et al.*, 2013).

**Survival, dispersal and disease cycle:** Survival of pathogen is the first event leading to pathogenesis. Bakanae is a monocyclic disease and the pathogen is seed and soil borne both, whereas, seedborne inoculum is more significant source as soilborne inoculum is reduced rapidly passage of time (Kanjansoon, 1965; Ou, 1985). The pathogen is dispersed predominantly with infected or infested seeds, although infected crop residues from the previous season also have been found to be capable of initiating the disease in the field (Watanabe, 1974). Infested seed is primary source of inoculum and act as main means of spreading the disease from field to field (Anderson and Webster, 2005). The pathogen survives under adverse conditions as spore on the coat of infected seed and as thick walled hyphae or macroconidia in infested crop residue in the soil (Watanabe, 1974; Ou, 1987; Saremi and Farrokhi, 2004). Studies on survival of *G. fujikuroi* in soil and residue under *in vitro* and *in vivo* conditions revealed that pathogen population in naturally infested soil declined rapidly with increased storage period. Kanjansoon (1965) in Thailand showed that

artificially inoculated soil caused 93% infection immediately after inoculation, however, after 90 days of the inoculation only 0.7% infection occurred and no infection resulted after 180 days of inoculation. This indicated that the fungus does not survive in soil for longer period. Sun (1975) reported that fungus survives in soil for about four months in the form of thick-walled hyphae or macroconidia. Kanjansoon (1965) also found that the fungus is viable in seeds and other parts of the diseased plants up to 4–10 months at room temperature and more than three years at 7 °C in cold storage. The fungus survived in infected stubbles kept in soil under natural environmental conditions up to four months (Biswas and Das, 2003; Pannu *et al.*, 2012). It confirmed that the pathogen survives long enough (ten months) to carry over between seasons, particularly in situations, where rice is grown for consecutive years in the same field. Bakanae incidence was found very low when clean seeds were planted in fields with a history of the disease (Karov *et al.*, 2009).

The conidia are disseminated by wind and water causing new infections in the rice field. The production of conidia on diseased or dead culms in the field coincides with the flowering and maturity of the crop (Seto, 1937). Therefore, seed infections occur through airborne ascospores during the flowering stage of the crop or conidia that contaminate the seed during harvesting. Ascospores and conidia adhering to the seed act as primary source of inoculum, germinate and infect seedlings through the roots and crown (Sun, 1975). The fungus becomes systemic within the plant but does not systemically infect the panicle. It can parasitize the plants without producing visible symptoms and can be isolated even from healthy looking seeds. Generally, the seed borne inoculum provides an initial site or focus for secondary infections. Under favorable conditions, infected plants have the capacity to produce numerous conidia that subsequently infect neighboring plants. Infection may also take place through spores and mycelium that are left in the water used for soaking seeds to stimulate germination before sowing (Karov *et al.*, 2009).

**Predisposition factors:** The effect of climate, especially rainfall and temperature, on the abundance of *Fusarium* species has been reported by various investigators (Burgess *et al.*, 1996; Saremi and Farrokhi, 2004). The optimum temperature for pathogen infection is 27-30°C and for disease development 35°C which is also highly favorable for seedling growth. Wind or water easily carries the conidia from one plant to another. Higher dose of nitrogenous fertilizers reduces the pathogen population in soil (Mandal and Chaudhuri, 1988). The disease incidences were greater in dry nurseries and summer crop than wet nurseries and spring crop as disease expression is favored by high temperatures and relatively humidity.

Transplanted rice plants display more symptoms than those grown from broadcast seeds (Saremi and Farrokhi, 2004).

### Secondary metabolites

**Gibberellic acid:** Gibberellic acid (GA<sub>3</sub>) is an important plant hormone produced by the bakanae fungus. Yabuta and Sumiki (1938) isolated a crystalline compound from culture filtrate of *G. fujikuroi* named as gibberellins. The GA<sub>3</sub> metabolic pathway genes were identified in fungi, bacteria and Arabidopsis, however, Sakamoto *et al.* (2004) reported in cereal plants. The genetic and biochemical background of GA biosynthesis by *F. fujikuroi* has been well characterized (Rojas *et al.*, 2001; Tudzynski *et al.*, 2003). GA biosynthetic genes are organized in a gene cluster (Linnemanstons *et al.*, 1999) which consist four cytochrome P450 monooxygenase genes (P450-1 to P450-4) and one GA4 desaturase gene (des) (Rojas *et al.*, 2001; Tudzynski *et al.*, 2003).

**Other secondary metabolites:** Secondary metabolites have been produced by different species of *Fusarium* or members of mating populations such as fumonisin B1 and B2 produced by *F. verticillioides* and *F. proliferatum* (Marasas *et al.*, 1986; Desjardins *et al.*, 2000), fusaric acid (Bacon and Hinton, 1996), beauvericin and fusaproliferin (Reynoso *et al.*, 2004), moniliformin (Leslie *et al.*, 1996; Desjardins *et al.*, 2000) and gibberellins (GAs). Gibberellin and fuminosin have been found most potent secondary metabolites for disease production and pathogenicity of *G. fujikuroi* species complex on rice which may depend on the balance of toxins and growth regulators (Amoah *et al.*, 1995; Zainudin, 2008b).

**Variability:** A significant variability has been observed in symptom development and production of gibberellin and fusaric acid. Symptom development is greatly influenced by the amount of inoculum, different isolates of the pathogen, nutritional conditions and production of gibberellin and fusaric acid (Nyvall, 1999; Sharma and Bagga, 2007; Amtaulli *et al.*, 2010; Kaur *et al.*, 2014). Nisikado and Matsumoto (1933) observed significant differences in pathogenicity of 66 strains of *G. fujikuroi* isolated from rice on the basis of overgrowth of seedling. Low inoculum level results in etiolating symptoms due to pathogen's gibberellin production upon infection of the host. Strains that produce high level of gibberellin growth hormone makes rice seedlings and plants to grow tall, thin, yellow and die. However, high level of fusaric acid cause stunted and chlorotic seedlings followed by root and crown rots, which eventually die (Amoah *et al.*, 1995; Karov *et al.*, 2009). A positive correlation existed between gibberellin production in culture and bakanae disease (Sunder and Satyavir, 1998), and pectic enzymes production and foot rot development (Thakur, 1974). Ma *et al.* (2008) reported a significant correlation between

the length of seedling treated with gibberellic acid and bakanae symptoms. Bashyal and Aggarwal (2013) also reported variation in morphology and pathogenicity of the *F. fujikuroi* isolates collected from rice seeds. Matua *et al.* (1976) from Japan identified 56 variants of *F. moniliforme* isolated from different hosts based on the production of microconidia, macroconidia, chlamydospores, gibberellin and fusaric acid. In India, Thakur (1974) characterized 48 isolates of *F. moniliforme* in four groups based on variable growth rate. Sunder and Satyavir (1998b) divided 28 isolates into five groups with respect to virulence and gibberellic acid production. Kaur *et al.* (2014) revealed that all isolates of bakanae pathogen produced gibberellic acid, however, only 45% isolates produced fusaric acid. Fungicide-induced pathogen resistance against (benzimidazole) benomyl and carbendazim has been reported which may be due to their successive applications as a seed disinfectant (Ogawa, 1988). Wada *et al.* (1990) reported that ergosterol biosynthesis inhibitors (perfurazoate) sensitive and benomyl resistant isolates have not produced gibberellins, while, fusaric acid produced by all type of isolates. The isolates which are less sensitive to triflumizole duly produced smaller quantity of gibberellins and showed reduced pathogenicity (Hamamura *et al.*, 1989). Similarly, Pan *et al.* (1997) reported less mycelial growth, sporulation and low pathogenicity in carbendazim tolerant strain.

**Molecular diagnostics:** Identification of *Fusarium* species based on morphological characteristics alone remains incomplete and inconclusive (Leslie and Summerell, 2006). Therefore, there is need to integrate molecular characterization as additional criteria with morphological characteristics for *Fusarium* species categorization associated with bakanae disease. *F. fujikuroi*, belonging to *G. fujikuroi* species complex (GFSC) is the most abundant *Fusarium* species causing bakanae disease followed by *F. proliferatum*. Multiple alignment of translation elongation factor (TEF) gene sequences of different *Fusarium* spp., showed a deletion of six nucleotides in *F. fujikuroi* sequence and a two nucleotide polymorphism in the same region of *F. proliferatum* sequence. These elements of variability were used to develop a conventional and real-time PCR assay for diagnosis. The species specific primer pairs (Fuji1F/TEF1R and Proli1F/TEF1R) gave a product of 179 and 188 bp for *F. fujikuroi* and *F. proliferatum*, respectively. Primer specificity was confirmed by analyzing the DNA of the most representative species of the GFSC and strains of *Fusarium* spp. isolated from rice plants and seeds. These specific primers were successfully used to detect fungal presence directly from infected rice tissues or seeds and providing a rapid tool for the early detection of pathogen contamination (Amatulli *et al.*, 2010). A comparison at the DNA

sequence level provides accurate classification of fungal species and elucidates the evolutionary, ecological relationships among diverse species (Mule *et al.*, 2005). Quazi *et al.* (2013) identified *F. proliferatum* based on molecular identification using species specific primer pairs and confirmed with sequencing (Mule *et al.*, 2005; Leslie and Summerell, 2006). *Fusarium* spp. were commonly characterized at species level by translation elongation factor 1- (TEF) gene sequencing (Dal Pra, 2010; Wulff *et al.*, 2010; Petrovic, *et al.*, 2013), internally transcribed spacer regions in the ribosomal repeat region (ITS1 and ITS2) and  $\alpha$ -tubulin (tub2) (O'Donnell *et al.*, 1998; Bashyal and Aggarwal, 2013). Other molecular techniques, such as RAPD (Amoah *et al.*, 1995), AFLPs (Petrovic *et al.*, 2013) and CHEF gel karyotypes (Xu *et al.*, 1995) have also been used to differentiate the members of *G. fujikuroi* species complex. These studies revealed that *G. fujikuroi* complex has been delineated into three lineages, designated as the African, Asian, and American clades (O'Donnell *et al.*, 1998). All sequences does not work equally for all species with *TEF1* gene [primer ef1 (5'-ATGGGTAAGGA (A? G) GACAAGA C-3') and primer ef2 (5'-GGA (G? A) GTACCAGT (G? C) ATCATGTT-3')] being the most widely accepted across the genus, however the ITS regions do not work well within the *Liseola* section.

**Genome sequencing:** *F. fujikuroi* is a biologically and phylogenetically distinct species in *G. fujikuroi* species complex (O'Donnell *et al.*, 1998). Jeong *et al.* (2013) presented the genome sequence of *F. fujikuroi* (strain B14) isolated from rice in South Korea. Genome sequencing of *F. fujikuroi* B14 was carried out using an Illumina HiSeq 2000-based whole-genome shotgun strategy. A total of 35,306,706 paired-end reads of ~3.57 Gb (101 nucleotide [nt] cycle, 486-bp average paired distance) were preprocessed and *de novo* assembled using CLC Genomics workbench 5.5. Initially, the assembly was 43,794,120 bp in length with 338 scaffolds (N50 678,621 bp, 48.3% GC, 1,079 contigs). After automatic gap closing using gap filler version 1.9 (www.baseclear.com) with the same reads, the final assembly consisted of 455 contigs in 333 scaffolds with a length of 43,810,516 bp exclusive of N's in remaining gaps. After masking repetitive sequences using a search against Repbase (www.girinst.org/repbase), 14,017 protein-coding genes were predicted using Augustus 2.5.5 (www.augustus.gobics.de) with *F. graminearum* parameters. Based on BLASTP, search against the UniRef90 database, significant matches (E value 10<sup>-5</sup>) were identified for 13,734 gene, while, 9,143 hits were derived from *F. oxysporum*. They also identified 576 tRNA genes using tRNA scan-SE (Lowe and Eddy, 1997). For comparative genomic analysis, the preprocessed Illumina reads were mapped to

chromosomal reference sequences for three known *Fusarium* species (www.broadinstitute.org/annotation/genome/fusarium\_group). *F. verticillioides* 7600 was most similar to B14 in terms of reference coverage (83%; 66.3% of reads were mapped). The percent coverage values of *F. oxysporum* 4287 and *F. graminearum* PH-1 were 57% and 29%, respectively. BLASTP analysis showed that 46.2% and 42.1% of the B14 genes matched those of *F. oxysporum* (total, 17,701 genes) and *F. verticillioides* (14,188 genes), respectively. Thus, *F. fujikuroi* B14 genome will contribute to a greater understanding of the biology and evolution of the *G. fujikuroi* species complex. The sequence determined in this study was deposited in the gene bank database with nucleotide sequence accession number ANFV00000000.

**Quarantine regulations:** Quarantine is a reasonable path of action to prevent inadvertent introductions of dangerous plant pathogens or to attempt eradication of new incursions. In California, the bakanae disease was first noticed during the year, 1999 due to strict quarantine regulations (Anderson and Webster, 2006). The Arkansas state plant board has declared the bakanae fungus a menace to the rice crop and its industry. The movement of *G. fujikuroi* infected articles from infested areas into Arkansas has been prohibited. A certificate issued by Arkansas state plant board indicating that a regulated article is not contaminated with *G. fujikuroi* strain, or has been treated in strict manner to eliminate the causal organism and such articles may be moved to any destination. Any rice seeds or plant parts that are to be used in a recognized research projects conducted by a state or federal program under the supervision of trained professional staff with proper safety programmes to prevent the accidental release or spread of the disease. As a condition of issuance of certificates the limited permits for the movement of regulated articles stipulating that any violation of these rules may be subjected to civil penalties under the authority of the Arkansas plant act of 1917 (www.rense.com). The Mississippi department of agriculture and commerce has also placed *G. fujikuroi* (bakanae strains) under quarantine regulations in the year, 2009.

### Disease Management

**Host Resistance:** Several screening methods have been tested and verified till date against bakanae disease of rice in different countries. Saremi and Farrokhi (2004) tested different cultivars of rice in Iran under naturally infested soil conditions having 1575 colony forming propagules (cfu g<sup>-1</sup>) in one gram soil to determine the relative resistance or sensitivity of cultivars in the fields. Aktas and Tunali (1986) and Quazi *et al.* (2013) used spore suspension for seeds and soil inoculation in pots before sowing for screening resistant germplasm. Haque

*et al.* (1979) inoculated sprouted paddy grain with spore suspension for 7-10 days in test tube resulted in higher bakanae infection as compare to field sowing. *In vitro* seedling screening method was also developed for selecting resistant rice germplasm against bakanae disease (Lee *et al.*, 2011). Zheng *et al.* (1993) screened rice germplasm for resistance through bud soaking in pathogen inoculum. Hossain *et al.* (2013) developed inoculum free varietal screening method through dipping of dry seeds in gibberellic acid to screen out susceptible rice varieties against the bakanae disease. Kim *et al.* (2014) developed an inoculation method for microconidia of *F. fujikuroi* using tissue embedding cassette and seedling tray for large scale screening of rice germplasm. Fiyaz *et al.* (2014) also standardized the high precision rapid methodology to screen the large number of rice germplasm against the bakanae disease. Seeds inoculation with spore suspension ( $1.0 \times 10^6$  spore/ml) and grown in protrays produced more distinct symptoms of the disease as compare to seedling inoculation method within 15 days after sowing.

**Quantitative trait loci (QTL) mapping and varietal resistance:** Rice germplasm carrying dwarf and semi dwarf gene *d29*, *sd6* or *sdq(t)* showed resistance to bakanae disease which can be utilized in rice breeding programme, however, rice germplasm carrying dwarf gene such as *sd1* and *d1* found susceptible to the disease (Ma *et al.*, 2008). The reaction of some genotype varied with crop growth stage. Lu (1994) reported seedling resistance in rice genotype Longjiao 86074-6 and moderately susceptible at adult stage. The genotype Qingxi 96 was moderately resistant at seedling stage and resistant at adult stage, however, Zupe 7, Dongrong 84-21, G-6, Sui 89-17 genotypes were found moderately resistant at both the stages. Khan *et al.* (1999) revealed that the resistance in rice against bakanae disease is monogenic, being recessive in cv. IR 6 and dominant in cv. KS 282. Yang *et al.* (2006) used japonica/indica double haploid population, derived from Chunjiang 06 and TN1 to analyze QTLs for resistance against bakanae disease by artificial inoculation at the budding stage. Two quantitative trait loci (QTLs) viz., *qB1* and *qB10* were identified on chromosome 1 and 10, respectively, which showed additive effect.

Studies on varietal resistance screening revealed that basmati/scented germplasm and cultivars are more susceptible to bakanae disease as compared to non-scented rice cultivars (Sunder *et al.*, 1998; Bashyal *et al.*, 2012; Pannu *et al.*, 2012; Ghazanfar *et al.*, 2013; Gupta *et al.*, 2014). Currently, the disease has been found more severe on basmati rice cultivars viz., Pusa Basmati 1121, Pusa Basmati 1176 and Pusa Basmati 1509 in Northern part of India, however, other basmati rice varieties viz., Pusa Basmati 1401, Pusa 2511, CSR 30, Dehradun basmati and Pakistani basmati were also found to be

infected by the disease with 2.0-22.8% incidence (up to 40%) in India (Bashyal *et al.*, 2012; Gupta *et al.*, 2014). Sunder *et al.* (1998) screened 221 scented and non-scented rice genotypes through seedling root dip inoculation method. Amongst scented rice genotypes, only two genotypes namely, C 4-64 (green base) and Karjat x 13-21 were found resistant, however, in non-scented genotypes, four genotypes, viz., BR 1067-84-1-3-2-1, BR 1257-31-1-1, BR 4363-8-11-4-9 and IR 58109-109-1-1-3 were rated as highly resistant. Sunder *et al.* (2014) identified highly resistant genotypes namely, HKR 96-561, HKR 96-565, HKR 07-40, HKR 07-53, HKR 08-13, HKR 08-21, HKR 08-22, MAUB 2009-1, PAU 3456-46-6-1-1, PNR 600 and RDN 01-2-10-9 through seedling root dip inoculation method. Fiyaz *et al.* (2014) found genotypes namely, Athad apunnu, C101A51, Chandana, IR 58025B, Panchami, PAU 201, Pusa 1342, and Varun Dhan as highly resistant, whereas, BPT 5204, Himju, Peeli Badam and Suphala as resistant to bakanae disease. In Pakistan, Ghazanfar *et al.* (2009; 2013) screened various rice germplasm through seedling dip inoculation method revealed that IR-6 and KKS-133 as resistant with 18.80 and 19.82% plant infection, whereas, the varieties Bas-385 and Bas-Super with 61.99 and 61.04% plant infection exhibit susceptible reaction. In Iran, Saremi *et al.* (2008) and Saremi and Farrokhi (2004) resulted cultivar Binam as highly resistant and cultivars Kadous, Shafagh Fajr, Sahel and Shafagh were moderately resistant to the disease.

**Agronomical practices:** The effective means for the management of this disease is the use of clean non-infested seeds. Since the pathogen is mainly seed borne, therefore, clean seeds should be used to minimize the disease occurrence. Salt water can be used to separate light weight seeds from seed lots which may reduce seed borne inoculums (Coher and Lanoiselet, 2002). Destruction or burning of crop residues with known fields having infections of pathogen may provide some benefits by limiting the amount of inoculum that may carry over to the next crop (Gupta *et al.*, 2004). Proper selection of geographic area, use of organic matter, crop rotation, time and mode of planting, selection of resistant varieties, balanced fertilization and irrigation management help in minimizing the disease (Burgess *et al.*, 1993; 1996). Bagga *et al.* (2007) found minimum disease incidence in late planted rice crop by the end of July which may be due to low temperature during the infection period.

Nutrients may affect the survival of pathogens either through a direct toxic or stimulatory effect in the soil. Higher levels of nitrogen and potassium affect the survival and population of fungus. A combination of NPK, ZnSO<sub>4</sub> and FeSO<sub>4</sub> significantly reduced the survival of pathogen after eight months of incubation (Sunder and Satyavir, 1998a; Mandal and Chaudhuri, 1988). Panneerselvam and Saravanamuthu (1996)

reported the suppression of pathogen in soil amended with neem cake than in groundnut cake. Utmost care and handling should also be taken at the time of harvesting and threshing, proper drying and seed cleaning to prevent the spread of the disease (Saremi *et al.*, 2008). Avoiding the damage on root tissue or less root injury at the time of nursery uprooting/transplanting is helpful to preventing the potential entry of the pathogen which ultimately gives less incidence of bakanae disease in the field. This can be achieved by mechanical transplanting of rice that gives less transplanting shock, better tillering and uniform maturity of crop ([www.csisa.org/category/csisa-success-story/india](http://www.csisa.org/category/csisa-success-story/india)).

**Use of plant extracts:** Plant extracts are becoming increasingly popular for the management of plant diseases. Antifungal activity of different plant extracts has been reported earlier by several investigators against the number of plant pathogens and *F. moniliforme*. Yasin *et al.* (2003) found maximum inhibition of mycelial growth of *F. moniliforme* (60.65%) by the leaf extract of *Lawsonia inermis* followed by root extract of *Asparagus racemosus* (50.59%). The presence of antifungal principle lawsone (2-hydroxyl-1, 4, naphthoquinone) in the leaf extract of *L. inermis* has been identified (Tripathi *et al.*, 1978) which might have been responsible for fungal growth inhibition. Antifungal properties of leaf extract of *Andrographis paniculata* and *Lagerstroemia speciosa* and bark extract of *Eucalyptus citriodora* against bakanae disease has also been reported. *Lawsonia inermis* and *Asparagus racemosus* have been found an important plant species for their exploitation as potent natural fungitoxicants with broad spectrum activity for controlling bakanae disease of rice (Yasin *et al.*, 2003). Essential oils (EOs) from *Cymbopogon citratus*, *Ocimum gratissimum* and *Thymus vulgaris* were found effective against *F. moniliforme* in controlling the seed infection by 95-100%. The antifungal activity of EOs due to their contents of thymol, terpinene, p-cymene, carvacrol from *O. gratissimum*, linalool from *T. vulgaris* and citral from *C. citrates* has been reported (Nguefack *et al.*, 2007).

**Use of compost and vermicompost extracts:** Compost tea extracted from decomposed organic matter has been used from centuries for their beneficial effects on plant health (Brinton, 1995). Compost and vermicompost tea produced with active aeration typically dominated by bacteria which is a useful parameter in relation to plant disease suppression. Manandhar and Yami (2008) investigated aerated and non aerated compost and vermicompost tea for the suppression of bakanae disease of rice resulted maximum control of bakanae disease with aerated vermicompost tea followed by aerated compost tea (25.6 and 22.4% increase of healthy seedlings, respectively) and the least effect was obtained by non aerated vermicompost tea (13.6% increase of healthy seedlings).

## Seed treatments

**Hot water seed treatment:** Thermal treatment is best substitute for chemical pesticides in organic cultivation. It is effective against many fungal species, while chemical fungicides are more specific and target oriented in the control of one or few pathogens (Ora *et al.*, 2011). The cold-hot water treatment method has been used widely as seed disinfection for controlling seed borne diseases. However, the hot water treatment developed through omission of cold water treatment found equally effective for the control of bakanae disease (Jaehwan *et al.*, 2009). Miyasaka *et al.* (2000) and Yamashita *et al.* (2000) recommended the soaking of seeds into hot water at 60°C for 10 minutes before sowing for reducing the seed infections and bakanae incidence in nursery and fields.

**Chemical seed treatment:** Currently, the seed treatment with chemicals is the most common management practice for bakanae disease in India (Gupta *et al.*, 2014) and widely practiced in most of East Asia. The imperative control of bakanae disease has been obtained by soaking the seeds in 0.3% sodium hypochlorite solution for two hours then drained and soaked in fresh water reduces the inoculum load by 80 to 90% (Webster and Gunnell, 1992). Fungicidal seed treatment with benomyl, thiram, benomyl + thiram, carbendazim + thiram, carboxin + thiram, fludioxonil, mancozeb, iprodione + triticonazole, prochloraz, thiophanate - methyl and ipconazole at 1-2% of seed weight found effective in various countries viz., Japan, Taiwan, Korea, Turkey, Iran, Pakistan, Bangladesh, India, Nepal and Italy (Ou, 1987; Bagga and Sharma, 2006; Bagga *et al.*, 2007; Karov *et al.*, 2009; Ora *et al.*, 2011; Tateishi *et al.*, 1998; Ghazanfar *et al.*, 2009). Faired controls of bakanae pathogen achieved by immersion of diseased seeds in the suspension of benzimidazole fungicides for 10 minutes (Sasaki, 1987; Surek and Gumustekin, 1994). Triflumizole, propiconazole, ipconazole and prochloraz were found to be effective against strains that are resistant to benomyl and the combination of thiram and benomyl (Karov *et al.*, 2009; Tateishi *et al.*, 1998). Seed/seedling treatment followed by soil drenching of benzimidazole fungicide derosol (0.2%) was found highly effective in controlling the disease (Bhalli *et al.*, 2001). Nursery dip treatment with benlate found highly effective for the control of bakanae disease followed by topsin-M and carbendazim (Javed *et al.*, 1996). Bagga and Sharma (2006) found seedling treatment with carbendazim or benlate (0.1%) for 6 and 8 h, significantly reduced the disease incidence up to 92%. Propiconazole 25 EC at 0.05% was the most effective treatment in controlling foot-rot, but it showed signs of phytotoxicity along with reduction plant height and grain yield. Dip the seed in bavistin 50 WP (0.05%) plus streptocycline (0.01%) solution for 12 h and

smearing the seeds with talc formulation of *Trichoderma harzianum* (15 g/kg seed) immediately before nursery sowing and seedling root dip for 6 hrs in *T. harzianum* biopowder (15 g/liter water) was found most effective against bakanae disease (Pannu *et al.*, 2009). Xueming and Yinghua (1997) prepared a seed-immersing chemical from asomate (76-78 parts), triazoleone (19-21 parts) and thiazole (1.9-2.1 parts) for preventing bakanae disease. Hongfu and Zhiyuan (2007) invented a fungicide consists of aluminum sulphate as the active ingredient which controls the 85% bakanae disease (www.patent.ipexl.com/C2N/200510123045).

**Biological control:** Rosales and Mew (1997; 1998) from Philippines; Kazempour and Elahinia (2007) and Padasht *et al.* (2004) from Iran, Dhitikiattipong *et al.* (2011) from Thailand found fluorescent *Pseudomonas* effective under *in vitro*, greenhouse and in field conditions, indicated 0.30-6.8% disease incidence and up to 96.3% disease control. Seed soaking with selected strains of *Pseudomonas* and *Bacillus* protected the seedlings from *F. moniliforme* infections (Mew and Rosales, 1992; Rosales and Mew, 1997) owing to suppression of initial inoculums present on the seed. It might be due to detoxification of fusaric acid as shown by Toyoda *et al.* (1988) while, working on a mutant of *P. solanacearum* with a capacity to detoxify fusaric acid. It is possible that extracellular and heat-stable proteinases produced by rhizobacteria from the genus *Pseudomonas* might imparted in inactivation of polysaccharide hydrolases of pathogenic strains of *Fusarium* spp. (Pietr, 1990). Kazempour and Elahinia (2007) found *P. fluorescens* isolate F15 highly effective against *F. fujikuroi*. Studies on combined application of mixture of fungicide rovril TS with F15 antagonistic bacterial isolate for seed coating, soil drenching and seed coating + foliar spray showed significant decrease in disease incidence by 6.5, 6.75 and 5.5%, respectively. *Bacillus* spp. viz., *B. subtilis* and *B. megaterium* have also been found effective against bakanae disease (Luo *et al.*, 2005). The most common biocontrol agent *Trichoderma strictipilis*, *T. atroviride* and *T. neokoningii* reduces the vegetative growth of bakanae pathogen (Nagamani *et al.*, 2012; Bhramaramba and Nagamani, 2013). In India, *T. viride* and *P. fluorescens* alone and in combination was found effective for the management of bakanae disease of rice. Lower disease incidence was reported in fields treated with FYM 10 t/ha + *Trichoderma* + *Pseudomonas* (Wyawahare *et al.*, 2012). Therefore, biological control can be integrated with various other appropriate management strategies for sustainable management of bakanae disease of rice.

**Conclusion:** Bakanae caused by *Fusarium fujikuroi* (Nirenberg) is emerging as a serious disease of rice in India. High disease incidences (1.2-40%) result in greater yield losses (3.0-95.4%) across the rice growing countries

of the world. The pathogen primarily survives in seed but also known to survive in soil. Among various *Fusarium* species, *F. fujikuroi* has been found frequently abundant and most virulent species all over the world. Currently, seed treatment with fungicides is the most common management practice for bakanae disease control in India. Studies on varietal screening revealed that basmati and aromatic rice germplasm/cultivars are more susceptible to bakanae disease as compared to coarse grain non-scented cultivars. Some strains of *Trichoderma*, *Pseudomonas* and *Bacillus* have been found effective against the disease. A considerable amount of research work has been done on various aspects of bakanae disease in different countries of the world. However, further research on host-pathogen interaction, racial profiling, variability, QTL mapping, virulence pattern and biochemical and molecular aspects of pathogenesis are also essential and need to be prioritized. Integrated and sustainable disease management practices including disease resistance, potential antagonists and biodegradable chemical molecules with decision support system need proper attention to devise practical management of the disease.

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