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Invention mechanisms and management source of false smut of rice caused by *Ustilaginoidea virens*- A Review

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Abstract

False smut of rice caused by *Ustilaginoidea virens* (Cooke), perfect sexual stage *Villosiclava virens*. Rice False Smut (RFS) early known as a minor disease but this time it is very severe in the majority of rice-growing areas around the world, as epidemics form. The disease incidence ranged from 5- 85% and major grain yield loss of 0.2% to 49% respectively in terms reduction in quality as well as quantity threatens food safety due to its production of mycotoxin. After flowering, its symptoms are appeared when the fungus transforms into globose structures or yellowed carbonaceous masses of individual grains unfit for consumption. The infection cycle and invasion mechanism, including the primary and secondary source of RFS infection involved different factors affecting for invention mechanisms of disease occurrence, including the infection time and pathway. Due to knowledge of epidemiology and invention mechanisms, resistance source, disease aggravates, proper management strategies needed to be framed to control the false smut disease. Improvement in completing the management source of false smut needs more emphasis. This review summarizes the present status of RFS, biochemical mechanism invention, resistant source of cultivars, and management of false smut in rice.

Keywords: Rice, false smut, *U. virens*, invention mechanism, management source.

Introduction

Rice is the most staple food commodity among the global population. It is mother source of energy and it covers more than 9 % of earth's arable land. It share 21 % of global per capita energy and 15 % of global per capita protein (FAO, 2016) ^[40]. *Ustilaginoidea virens* is Cooke, (1878) ^[33] in Tirunelveli district of Tamil Nadu the first time reported the causal agent of false smut of rice. Tanaka *et al.*, (2008) ^[105] suggested the *Villosiclava virens* as teleomorph of *U. virens*. Its outbreak might be possibly due to high input cultivation, increased use of hybrid varieties, and climate change (Lu *et al.*, 2009) ^[77]. There are different viewpoints on the source of infection in the early, but a more consistent view was that the overwintering sclerotia and chlamydospore are the main original resources of infection. There are many factors affect the occurrence and prevalence, for example, the climate conditions, temperature, humidity, hours of illumination, especially the "key growth stage of rice" (KGSR, i.e., the late booting stage and begin heading to flowering period are the susceptible period of rice to RFS) encounter the local climatic conditions which suitable for the occurrence of RFS. The disease hampers quality and quantity of rice grains as well as affects the germination percentage of the infected seedlings (Sanghera *et al.*, 2002) ^[97]. Pathogen on sick grains produces antimetabolic cyclic peptides, ustiloxin from its chlamydospores, poisonous to both humans and animals (Nakamura *et al.*, 1994; Koiso *et al.*, 1994) ^[81, 60]. Disease incidence ranged from 5- 85% and yield loss of 0.2% to 49% has been reported depending upon the rice cultivars and intensity of disease (Dodan and Singh 1996; Ladhakshmi *et al.*, 2012; Kumari and Kumar, 2015) ^[38, 65, 63]. Rice cultivars demonstration significant difference in quantitative resistance still no variety is yet to have complete resistance to *U. virens* (Biswas, 2001; Li *et al.*, 2008; Huang *et al.*, 2015) ^[18, 71, 54]. False smut can be effectively managed through cultural, biological, and chemical control. Various workers (Pannu *et al.*, 2010; Mohiddin *et al.*, 2012) ^[87, 80] have reported the efficacy of several fungicides for the management of false smut.

Symptoms

The usually disease occurred sporadically in the field and in an individual ear only a few grains are initially infected while severe infection many smut balls aggregate together (Singh, 1990) ^[102]. If infection occurs before fertilization, most of the glumes remain sterile without any visible sign of infection. Initially typical whitish creamy color smut balls occur infection occurs after fertilization then convert in large, velvety, green smut balls (Pseudomorph) develop (Kulkarni and Moniz, 1975) ^[61]. Initially the smut balls are small and remain confined between the glumes. They gradually increase and become 0.8 cm to 1 cm or more in diameter enclosing the floral parts. Young smut spores' balls are flattened horse-shaped, smooth, whitish creamy, light yellow, and covered by a membrane. Later, change the membrane bursts colour and the colour in orange, yellowish-green, olive green, and finally the greenish-black. The false smut balls are covered are three outer layers, each at different stages of development of smut balls. The innermost covered is yellowish with radiating mycelial and spores in the process of formation. The next layer is orange colored consisting of mycelial and spores. The outermost layer is green and consists of matures spores together with remaining fragments of mycelial (Ou, 1985) ^[83]. The pathogens cause sterility of the spikelets neighboring the smut balls (Hashioka, 1971) ^[50] reduced the grain weight and germination of affected grains. The sclerotial production in smut balls has been observed by severe; workers (Hashioka *et al.*, 1951; Ikegami, 1963; Sharmaa and Joshi, 1975; Singh and Dube, 1976; Rathaiah and Bhattachariya, 1993; Ladhakshmi *et al* 2012) ^[49, 57, 98, 101, 95, 654].

Types of Smut Ball

They are different types of smut balls, density, and distribution on infected panicles Ikegami (1961) ^[56] classified smut balls by colour as yellow, yellowish-green, greenish-yellow and green; the colour was essentially age-dependent, the aged the balls were, the darker was the colour. Some literature term yellow balls as orange or orange-yellow (Rani, 2014) ^[92] and green as greenish-black or black (Rani, 2016) ^[93]. Most literature presented dark balls (green or greenish-black) as the advance stage (by colour change over time) of light-colored (yellow or orange) balls (Rani, 2014) ^[93]. Ikegami (1961) ^[56] further observed that smut balls formed under conditions of 25 °C showed the highest percentage of yellow balls, whereas the high occurrence of greenish balls observed under temperatures lower than 25 °C. He found yellow balls consisted of three layers (thick yellowing brown in the outermost, light yellow in the middle and white in the center), whereas green ones had two layers (dark green in the outermost and yellow and white in the center). There were two pathways of colour formation in smut balls, according to Ikegami (1961) ^[56] first one is the appearance of the colour at the time just after the formation of the balls (inner alterations), and the another one is the colour changing after occurrence prompted by environment (light and temperature). Does not any information is available on whether the types of smut balls by origin are strain-related of the pathogen or not but some literature has mentioned the existence of 'white' smut balls (Honkura *et al.*, 1991) ^[52].

Inoculation Methods

According to Fujita *et al.* (1989) ^[43] inoculated a conidial suspension (4×10^6 Conidia/ml) of *U. virens* at the panicle emergence stage. It is incubate in a moist cabinet chamber initially at 15°C for 2 days and then at 26°C with 100 %

humidity for 5 days. Lu *et al.* (1996) ^[78]. Suggested that the panicle emergence stage is the sensitive infective stage and conidia are the main infective type. Ladhakshmi *et al.* (2012) ^[65] injected the conidial suspension of the pathogen during the panicle emergence stage of the rice variety TN1 and typical smut balls were produce on the panicles 15 days after inoculation. Tang *et al.* (2013) reported that after filtering the hyphae, conidia could be collected from the filtrate by centrifugation and re-suspending the conidia in the fresh Potato Sucrose medium to a density of $2-5 \times 10^5$ conidia/ml and used as the inoculum. The inoculated plants were placed in a plant growth chamber for 2 days at 20 °C with 85-95 percent relative humidity and then for 5 days at 25 °C with 3000 lux illuminances from white fluorescent tubes. Different developmental stages of rice plants were injected with 1 to 2 ml suspension of conidia and chlamydospores at a concentration of 10^6 /ml. Booting and panicle emergence stages were optimum infection stages (Anonymous, 2012) ^[6]. Conidial suspension (2×10^5 conidia/ml) was sprayed at panicle emergence stage in the evening hours. The rice panicles were sprayed with sterile distilled water served as control and RH maintain 95 % a week. The maximum percent of infection and smut balls per panicle observed in genotype PR-116 (26.56 % and 1.6) (Rani *et al.* 2016) ^[92]. Nevertheless, the conidia and suspension of hyphal fragments obtained by breaking the mycelia with a high-speed blender were also used as inoculum in some lab (Lu *et al.* 2009) ^[77]. Zhang *et al.* (2003) ^[126] injected the artificial culture of *U. virens in vitro* during panicle emergence stage of rice at nightfall.

The Causal Agent of False Smut Rice

The Pathogen

The sound knowledge of the history, life cycle, infection mechanisms of pathogens and process for the management of rice false smut. Rice false smut disease caused by the fungal pathogen *Ustilaginoidea virens*, which produces both sexual ascospores and asexual chlamydospores in its life cycle (Wang *et al.*, 1992; Biswas, 2001) ^[110 and 118]. The anamorph form, *Ustilaginoidea virens* (Cooke) Takahashi, is widely accepted of false smut in rice and maize (Rush *et al.*, 2000; Abbas and Sciumbato, 2002) ^[96 and 1]. The teleomorph stage *Claviceps virens* Sakurai ex Nakata and *Claviceps Oryza-sativa* Hashioka because of the teleomorphic features of *U. virens* are similar to those of *Claviceps*. However, phylogenetic analyses using sequences of the large subunit of the ribosomal RNA gene suggested that members of *Ustilaginoideae* are distinct from the teleomorphic genera of *Clavicipitaceae* and should be recognized as a monophyletic group within Hypocreales (Bischoff *et al.*, 2004) ^[17]. Molecular phylogenetic studies have also revealed that *Ustilaginoidea* species formed a paraphyletic group, and not congeneric with *Claviceps* based on the ALDH1-1 gene, which encodes a member of the aldehyde dehydrogenase family (Tanaka and Tanaka, 2008) ^[108]. Based on morphological and biological characteristics of the teleomorph, Tanaka *et al.* (Tanaka *et al.*, 2008) ^[96] suggested *Villosiclava virens* as the new name for the teleomorph of *Ustilaginoidea virens* which is accepted and the name was used by recent reports (Ladhakshmi *et al.*, 2012; Fu *et al.*, 2012; Tang *et al.*, 2012; Ashizawa *et al.*, 2012) ^[65, 42, 103, 8]. According to scientific classification, the fungus is an ascomycete, not a basidiomycete ('true smut'), hence received its common name as 'false smut' and ascigerous stage of *U. setariae* and described the development of the fruiting bodies

on true sclerotia and the fructification of asci and ascospores but did not propose a new taxonomic name based on the teleomorph (Tanaka *et al.* 2008) [105].

Geographical Distribution

RFS disease occurs in almost everywhere of the world. Historically, it was considered a minor disease causing significant yield loss (Webster and Gunnell, 1992) [115]. Now a days its traditional rank has changed with extensive reports in almost all the rice-growing countries of the globe including Africa, China, Egypt and India. RFS is a developing potential disease in India, where increasing incidence being reported from fields since the early 2010s. In Indian and part of Bangladesh subcontinent, rice is grown year-round, broadly under three seasons: early monsoon ('Aus' rice), late monsoon ('Transplanted Aman or T. Aman' rice) and winter ('Boro' rice). RFSm is the disease of 'T. Aman' rice in Bangladesh (BRRI, 2013) [22] and parts of India (Shetty and Shetty, 1985) [99]. However, there are undocumented reports of its sporadic incidence in other two seasons also. Quantitative information on the seasonal incidence of the disease is unavailable.

Morphological Characters

Chlamydospore spore formed on spore balls are borne lately on minute sterigmata on radial hyphae and are spherical to elliptical, echinulate, olivaceous 4-6×3-5 µm with epispore 0.3 mm in thickness (Hashioka, 1971; Singh, 1990) [50 and 102], 3.5-5.5 µm (Sharma and Joshi 1975) [98], 4.20 to 6.54 µm (Baite *et al.*, 2014) [11]. The scanning electron microscopy revealed that the conidial wall was echinulate and ornamented with prominent spines. The spines were pointed at the top or irregularly curved and ranged from 359.9– 994.5 nm long. Characters are isolate based on colony of *U. virens* were grouped into three groups. Immature spores are smooth, hyaline, and prominent pointed warts characterize smaller but with maturity, the episode.

Primary Infection Source of RFS

Host pathogen interaction is the most important for the primary infection of pathogens. Rice false smut *U. virens* can survive in the form of mycelium, chlamydospore, sclerotium and RFS balls. The overwintering sclerotia germinated and produced ascospores, which caused rice false smut in the coming year (Liao, 1994) [73]. Inoculation of chlamydospore, ascospores and thin-wall conidia can successfully induce rice false smut disease (Chen *et al.*, 1995; Yao *et al.*, 2012) [29 and 121]. Zuo *et al.* (1996) [127] captured chlamydospores over the rice fields, suggesting that chlamydospores have a high ability of airflow dispersion and infection. Therefore, it is reduced that there is a pathogen source base or intermediate host suitable for the dormancy of chlamydospore outside rice fields. However, there is a different point of view on this point. Chen *et al.*, (1994) [30] believed that chlamydospores germinate in the soil and produced conidia, these conidia are spreading by source of wind and rain and caused the primary infection RFS.

However, some literature and cited gave information about the sclerotia were not found in some areas of some provinces in China, but the rice was infected by *U. virens* every year, so it was questionable that sclerotium was the major primary infection source (Wang, 1992; Chen *et al.*, 1995) [110 and 29]. On the other hand, Liu *et al.* (2009b) [75] demonstrated that the primary infection source of RFS mainly was the seeds with a pathogen, followed by the overwintering pathogen in soil. Most scholars abroad were consistent with the views that the

pathogens overwintering in the form of sclerotium and chlamydospore were the major primary infection sources, and the chlamydospores played a decisive role in the secondary infection (Ikegami, 1963; Ou, 1985) [57 and 82].

Secondary Source of Infection

The main key to secondary infection source of false smut is chlamydospores. The chlamydospores germinate in the soil and produced conidia, and the conidia caused secondary infection by spreading through wind and rain. Many studies have reported that there was a severe incidence of RFS in late-maturing varieties, and that the secondary infection may be the major factor (Chen *et al.*, 1994) [30]. A large number of chlamydospores could be captured over the rice field, and the number of spores was relatively increased with the arrival of the flowering period. The secondary infection source of RFS is mainly chlamydospores and conidia by air spread (Ou, 1985) [82].

Time and Pathway of Infection

A recent study has indicated that *U. virens* follow a specific route, with the hyphae colonizing the outer surface of the spikelets, and then entering the insides of speckles from the apex (Azhiwaza *et al.*, 2012) [8]. *U. virens* initially attached itself to the surface of the filamentous, and then formed several discrete structures, including mycelial and stroma infection hyphae. The infection periods and pathways of *U. virens* are not very clear yet. There are chief views of system infection in late booting to early heading stage infection (late-stage infection), or systemic infection, more advanced inoculation and detection technology have been employed recently, indicating that RFS infection is mainly primary infection in late booting stage and secondary infection in later stage i.e., after flowering of rice panicles.

Most researchers believed that the primary infection site was the floral organ of rice, and the infection period was between the middle and late booting stages to the early heading period (Xu *et al.*, 2001; Wang *et al.*, 2008; Chen *et al.*, 2013) [117, 111 and 27]. Liu *et al.* (2007) [74] believed that the main infection period of *U. virens* was between the big belly stage to begin heading period, but not the seed germination stages. Strong evidences support that the infection that occurred in the flowering stage was that artificial inoculation in the late booting stage could increase the diseased panicle rate substantially (Cai *et al.*, 2009;) [23]. At present, it was believed that 1-2 weeks before heading are the main invasion period of RFS (Li *et al.*, 1986; Wang, 1992; Guo *et al.*, 2000) [70, 110 and 47]. Du *et al.* (1990) [39] illustrated that seed treated with biocidal there was no effect of prevention and cure RFS, and inoculation with *U. virens* after germination of rice seeds also did not incur the disease, proving that *U. virens* is not systemic infection. It was found that the hyphae of *U. virens* could invade the spikelet apices, via a small gap between the lemma and palea.

Infection sites: Examination of serial semi-thin and ultrathin sections of infected spikelets showed that the primary infection sites of *U. virens* was upper parts of the three stamen filaments located between the ovary and the lodicules. In the booting stage, *U. virens* specifically infects the stamen filaments of rice, and thus it grows and develops into chlamydospores and finally formed smut ball. *U. virens* could not infect the ovary and anther, however, the secondary hyphae can occasionally infect the stigma and outer cells of lodicule (Dai *et al.*, 2005) [34]. Dodan *et al.*, (1996) [38] and Madhare *et al.*, (2008) [79] found that the conidiophore of

fungus *U. virens* could infect the ovary and single spikelet and then transformation into chlamydospore and false smut ball. A recent study has indicated that the *U. virens* follows a specific route, with the hyphae colonizing the outer surface of the spikelets, and then entering the inside of spikelets from the apex (Ashizawa *et al.*, 2012) [8]. Consistent with a previous report (Tang *et al.*, 2012) [106], *U. virens* initially attaches itself to the surface of the filamentous, and then formed several discrete structure, including mycelial stroma and infection hyphae.

Mechanism of Infection

The route of *U. virens* penetrates rice panicles has long been a question of debate. A recent study utilized a transgenic starin expressing green fluorescent protein gene (GFP) (Ashiwaza *et al.*, 2012) [8] to observe the *U. virens*, and found that there were attachment and infection of the early reproduction stage of rice (Zhou *et al.*, 2003, 2006; Wang *et al.*, 2005a) [126, 125 and 112]. The GFP-labelled conidia of *U. virens* were injected into rice sheaths had invade spikelets through the apices, via the small gap between lemma and plelia had already reached all floral organs 144 hpi (Ashiwaza *et al.*, 2012) [8]. According to (Hu *et al.*, 2014) the primary site of *U. virens* colonization at the base of the filaments with the inner spikelets becoming infected by hyphae at 24 hpi after that hyphae reached its highest level at 168 hpi, before rice heading stage, as the infection extended upward from basal filamentous to the anther apex, and then enclosed all the floral organs to a velvety smut ball.

Predisposing Factors

It was demonstrated with many years' observation in the fields, the RFS incidence of the same rice variety in different years are quite different. If the resistant varieties of rice encounter rainy days during the booting period and begin heading stage, then the incidence of RFS will be intensified; on the contrary, if the susceptible varieties encounter dryness and high-temperature weather during these stages, then the incidence of RFS will decrease or even no be infected (Wang *et al.*, 2004) [109].

The epidemiological factor that rice plants encounter in susceptible periods or KGSR is one of the key factors determining the degree of RFS incidence. If KGSR encounter more rainy days, abundant rainfall, a short sunshine duration, the relative humidity (RH) was high (above 85%), the temperature was suitable (22-28 °C), and a small temperature difference between day and night, then the degree of RFS incidence was severe. If these factors were contrary, then the degree of RFS incidence was low (Yashoda *et al.*, 2000; Yang, 2007; Fei *et al.*, 2010) [122, 120, 41]. Pan *et al.*, (1997b) [84] found that the severity of RFS was closely related to the local accumulated sunshine hours and total rainfall in KGSR. If the number of sunshine hours was reduced and the rainfall was increased, then the morbidity of RFS was aggravated. The rain days and rainfall during the KGSR were positively correlated with the infected panicle rate, the correlation coefficient was $r = 0.8342^*$ and $r = 0.8826^*$, and the related equation was $Y = -6.7985 + 6.0538x$ and $Y = -2.6963 + 0.3652x$, respectively. RFS is negatively correlated with the daily mean temperature but positively correlated with humidity at the beginning heading stage. When the daily mean temperature was 23-24 °C and RH 82%-87% during the beginning heading period are conducive to the occurrence of RFS. Yashoda *et al.*, (2000) [122] that Low maximum temperature (< 31°C), low rainfall (< 5mm), high minimum

temperature (19 °C), was found to be favourable for disease development also supported it. (Jie *et al.*, (2014) [58] noticed that the effect of rice growth stage temperature, relative humidity and wetness duration on infection of rice false smut found that late panicle development stages had the highest percentage (90.00 %) of diseased panicle and highest level of disease (92.90 %) was obtained at 25 °C and 95 % RH with 120 hrs of wetness duration. But, Singh *et al.*, (1987) found that Incidence is favored by relatively low (20 °C) temperature and high relative humidity (>90%) coupled with well-distributed moderate rainfall during flowering, also by late sowing and high soil fertility. Reports on the effect of rainfall were conflicting; high disease intensity had been attributed to rainfall at heading (Cartwright *et al.*, 2002), but the opposite (low rainfall favoring the disease) had been reported (Dodan and Singh, 1996) [38].

Bhargava *et al.*, (2017) [16] reported that the disease incidence and disease severity index of false smut occurred at the temperature (23-32 °C), relative humidity (66-90 %), rainfall (5-8 mm) and sunshine (4.81-6.20 hrs). Chaudhari *et al.*, (2019) recorded that the data of meteorological during the subsequent two-year in the progress of the false smut due to closely similar weather relation. In 2018, the disease was initiated on the 37th Meteorological standard week (MSW). More dominant favourable weather conditions viz., maximum temperature (30.8, 32.7, 33.8 and 36.9 °C), average temperature (26.3, 27.3, 27.7 and 29.8 °C) and bright sunshine hours (6.8, 6.8, 8.1 and 8.6 hrs). After 40th MSW up to harvest, the disease incidence was continuously increased and recorded up to 6.68 percent disease incidence.

Nutritional Management

Different fertilizer, dosage of application and application time of fertilizer significantly affect the occurrence and severity of RFS. The incidence of RFS was higher if more nitrogen fertilizer was applied and the application time was late (Pan *et al.*, 1993) [86]. Increasing the amount of nitrogen fertilizer significantly increased the rate of infected rice plants.

Fertilization habit of partial nitrogen, excess dosage of fertilizer and late fertilization will reduce the rice resistance ability to RFS (Zhao, 2006) [125]. The applied total quantity of nitrogen (X1), amount of panicle fertilizer (X2) and application time of panicle fertilizer (X3) strongly affected the incidence of RFS (Y), the regression equation was $Y = -93.053 + 3.393X1 + 9.265X2 + 3.711X3$, and the correlation coefficient $R = 0.8922^{**}$. Among all of these factors, the direct effect of the application amount of ammonia fertilizer on the RFS incidence was the highest, $P 0.1 = 0.393$ (Pan *et al.*, 1997a) [85]. If 600 kg/hectare of urea was used as panicle fertilizer, then the infected panicle rate was 17.5%, and increased by 34.6 % and 48.9 % compared with that of the 300 kg/hectare and no application of the panicle fertilizer (Chen *et al.*, 2009) [31]. When 165.0, 225.0, 232.5, 240.0 and 300.0 kg of pure N was used as the topdressing fertilizer (urea) per hectare at heading period, the infected rice panicle rates of RFS were 1.33 %, 1.97 %, 2.13 %, 2.33 % and 3.13 % respectively. Reasonable amounts ratio of nitrogen, phosphorus and potassium fertilizer were beneficial to increasing rice yield and reducing the occurrence and harm degree of RFS (Wang *et al.*, 2010; Hong *et al.*, 2013; Qing *et al.*, 2014) [113, 51 and 90].

Management Source of RFS Resistance Source

RFS resistance sources in India is very negligible due to its minor importance as a disease infecting rice while its cause severe effect on the world as an epidemic. The previous work on rice false smut resistance screening and molecular mechanism of false smut resistance is not sufficient (Zhang *et al.*, 2014) ^[123]. Resistance genes to rice false smut is less reported despite considerable efforts on screening of on-field resistance and quantitative trait loci (QTL) analysis (Guo *et al.*, 2012; Huang *et al.*, 2015) ^[48 and 54]. Various attempt has been made by researches to screen rice cultivars resistance to false smut. Singh and Singh (2005) ^[103] screened 98 genotypes in which 27 rice genotypes found resistant. Ashrafuzzaman (1974) ^[9] evaluate 91 false smut resistant lines and 4 were susceptible out of these 42 had been found resistant to false smut. Singh and Dube (1978) ^[101] from India observed more yield loss (~44%) in hybrid varieties compared to inbred varieties (~17 %). Barnwal and Singh (2011) ^[14] also from India found similar results, more yield loss in hybrid 'HKR-126' than inbred 'PA 6444'. Baruah *et al.* (1992) ^[15] was also reported that hybrid varieties were more susceptible to rice false smut (compared to inbred varieties). Ansari *et al.* (1988) ^[7] screened 22 cultivars grown under natural infection by RFSm in the Andaman Islands of India, the most susceptible ones were 'DR447-20' (49 % yield loss), while the most resistant ones were 'CR155-5029216' (0.04 % yield loss), 'CN758-1-1-1' (0.1 % yield loss). Xia *et al.*, (1989) ^[116] was also found that the extended angle of glume to be wider in hybrid rice and also the flowering phase to be longer in hybrid rice which resulted in more infection. He reported more incidence of false smut in hybrid rice lines. Rice cultivars, Taraori Basmati and CR-333-6-1. The crop planted on 10th June had the least incidence of false smut. False smut was maximum in CR-33-6-1 with 36.9 percent incidence and 0.6 percent smutted balls (Dodan and Singh, 1995) ^[36]. Rao (1955) ^[94] reported that the least percentage of smutted balls was recorded in case BS-158 (2.7) and the late duration cultivars were more severe. Ahonsi *et al.* (2000) ^[3] found that all the tested varieties reacted differently to false smut incidence. ITA 316 and Ex-China showed some resistance and IRAT 170 was completely free from infection. Lu *et al.* (2008) ^[76] reported that the number of smut balls on panicles or degree of blanking (chaffiness) is related to the level of resistance in the rice cultivars this was also confirmed by (Cartwright *et al.* 2003) ^[25]. Ladhalakshmi *et al.* (2012) ^[65] reported that 18 resistance of blast- resistance varieties/lines were mostly resistant against false smut. Panwar *et al.* (2012) ^[88] noted that early maturing rice varieties and hybrids (90-115 days) were found to low infected with the disease as compared to late maturing varieties (115-130 days) and disease severity ranged from 27.3-42.74 % whereas, it was ranged from 85.35-94.71 in rice hybrids. Based on a false smut score, four rice genotypes were screened by Mohiddin *et al.* (2012) ^[88], HRI 119 being the most resistant genotype with low disease incidence. Screening of 186 rice hybrids to false smut resistance were done by Yan *et al.* (2014) ^[119] identified few hybrids with low disease incidence. Li *et al.*, 2008 ^[71] mapped two major genes controlling rice false smut with polygene mixed model and the heritability of two major genes. Using near isogenic introgression line for disease resistance two QTLs were mapped (Xu *et al.*, 2002) ^[119]. Li *et al.* (2014) ^[63] detected eight QTLs (qFsr1, qFsr2, qFsr4, qFsr8, qFsr10a, qFsr10b, qFsr11 and qFsr12) on chromosomes 1, 2, 4, 8, 10, 11 and 12 controlling false smut resistance. Stable inheritance of disease-resistant loci on chromosome 10 and 11 over the years have been studied by

Li *et al.* (2011) ^[68]. Using a large set of random introgression lines (ILs) and field-based phenotyping, rice QRLs providing resistance to *U. virens* were identified. Four QRLs (qFSR-6-7, qFSR-10-5, qFSR10-2 and qFSR-11-2) have been identified with relatively larger and consistent effects across the two testing sites. Li *et al.* (2011) ^[68] identified QTL conferring resistance to false smut in IR28 from the chromosome 11 during preliminary mapping of 157 recombinant inbred lines. Channamallikarjuna *et al.* (2010) ^[26] suggested that the broad spectrum and durable disease resistance for sheath blight resistance might be associated with chitinase gene cluster. Rani *et al.* (2016) ^[92] evaluated 31 germplasm line for two year against *U. virens*. In first year, 8 inbred line were found to be completely free from disease. In second year, five germplasm are completely free from disease. Kumar *et al.* (2017) ^[62] evaluated 21 rice genotypes for resistance to false smut and found that four rice genotypes resistance i.e., Swarna Shreya, IR96321-1447-521-B-2-1-2, IR96321-1447-651-B-1-1-2, and IR 83294- 66-2-2-3-2 were immune or highly resistant against false smut.

Cultural Management

Initially transplanted rice had higher disease incidence as compared to late planting (Chhottaray. 1991; Dodan and Ram Singh., 1995) ^[32 and 36]. Conservation tillage, continuous rice cropping and moderate nitrogen fertility rates reduced false smut disease in susceptible cultivars (Brooks *et al.*, 2009) ^[20]. To escape severe damage, sowing date and heading period could be planned in such a way that flowering should not coincide with rainy period. The use of sclerotic free seeds for sowing and cleaning of bunds may help the farmers to reduce the initial occurrence of the disease. In respect of cultivation practices, furrow irrigated rice cultivation systems recorded less disease severity compared to flooded fields. The mechanism behind is the reduction on the survival period of chlamydospores in soil and occurrence of physiological changes in the host plant in response to shifting of rice cultivation from anaerobic to aerobic growing conditions (Brooks *et al.*, 2010) ^[21].

Fungicidal Management

In recent years, the disease has become a more severe threat to our rice cultivation due to the intervention of false smut epidemics. In order to minimize direct economic loss to farmers, suitable management practices have to be made to manage the disease. Yield loss of 44 % in Ratana and 17 % in IR8 was reported by Agarwal and Verma (1978) ^[2]. The control of rice false smut has mostly relied on fungicides. Various fungicides such as Wenqunig (a suspension of *Bacillus subtilis* in a solution of Validamycin), Copper Oxychloride, Cuproxat, Simeconazole, Tebuconazole, Copper Hydroxide, Difenoconazole and Hexaconazole have been reported for the control over 70 % of rice false smut disease (Ahonsi and Adeoti, 2003; Gao *et al.*, 2010; Liang *et al.*, 2014; Tripathi *et al.*, 2014. ^[3, 45, 72 and 107]. Bagga and Kaur (2006) ^[10] evaluated and reported fuji one 40 EC (0.1, 0.2 and 0.3 %) and Bavistin 50 WP (0.1 %) showed that reduced the infection of incidence when applied at the booting. Treatment with Blitox 50 WP (0.3 %) and tilt 25 EC (0.1 %) were most effective. Tasuda *et al.* (2006) ^[108] studied the application of fungicide simeconazole when applied 3 weeks before rice heading which was found to be more effective against rice false smut disease. Fungicides Triüoxystrobin 25 % + Tebuconazole 50 % and Propiconazole 25 EC were evaluated by *in vitro* and *in vivo* condition which showed 100 %

inhibition to growth of fungal mycelium. Application of Prochloraz + Carbendazim followed by chlorothalonil were effective in controlling the false smut of rice (Mohiddin *et al.*, 2012)^[80]. Raji *et al.* (2016)^[91] reported that Propiconazole 25 EC (0.1 %) recorded lowest disease severity than other treatments and higher grain yields were obtained when sprayed at booting stage. Control of false smut was obtained by application of combination of Propiconazole + Difenconazole and *B. subtilis* with Validamycin. Spray of Propiconazole and Hexaconazole were effective in controlling the rice false smut (Barnwal *et al.*, 2010)^[14]. Application of EBI fungicides such as propiconazole have been reported to control rice false smut and sheath blight (Chen *et al.*, 2013)^[27]. Pramesh *et al.* (2017)^[89] also found that Azoxystrobin (18.2 %) + Difenconazole (11.4 %) SC was highly effective in the management of disease with least infected tillers and disease severity (3.43 %), (1.80) per cent, respectively. Bhargava *et al.*, (2017)^[16] It was also reported that in case of yield, Sabour ardhajal had minimum 31.42 q/ha whereas maximum yield was found in Sushak samrat (42.22 q/ha) and early sowing 15th June sown crop had maximum yield (41.20 q/ha).

Biological Control

Plant products such as leaf extracts and plant oils could also be used to control rice false smut. Raji *et al.* (2016)^[91] studied plant extracts under *in vitro* against rice false smut pathogen which was considerably inhibited by bulb extract of Garlic (*Allium sativum*), rhizome extract of Turmeric (*Curcuma longa*), leaf extracts of Lantana (*Lantana camara*) and Bael (*Aegle marmelos*), whereas essential oils of Lemon grass (*Cymbopogon flexuosus*) Cinnamon (*Cinnamomum zeylanicum*), and Palmarosa (*Cymbopogon martinii*) have completely inhibited the growth of *U. virens*. *Bacillus subtilis* (Liu *et al.*, 2007)^[74] was reported to be effectively against the disease. Kannahi *et al.* (2016)^[59] studied the antagonistic potential of 9 isolates of *Trichoderma viride*, *Trichoderma virens*, *Trichoderma harzianum* and *Trichoderma reesei* obtained from rice rhizosphere under *in vitro* condition and reported that all the isolates of *Trichoderma* have showed antagonistic activity against *U. virens* but among them isolate of *T. viride* showed maximum antagonistic potential. Andargie *et al.*, (2017)^[5] reported first time *Antennariella placitae* as a potential fungal endophyte effective in reducing the negative effects of rice false smut fungus (*Ustilagoideia virens*) both *in vitro* and *in vivo* condition.

Conclusion

Intensive cultivation of rice over depend on climatic condition, chemical fertilizers, changes in varietal profile and false smut disease of rice has emerged as one of the major threats to rice cultivation in World as well as India. The mapping of resistance genes and stable inheritance of resistance gene have been reported. Furthermore, molecular studies on isolation and characterization the QTLs responsible for resistance to false smut is needed. Development of resistant varieties through resistant QTLs and marker-assisted selection have to be considered for further research. Cultivation of resistant varieties is the best method to control disease. Another area of work that needs attention is the prediction or forecasting of the disease occurrence for better management of the disease. However, few varieties showing resistance against false smut have been reported till date, management through fungicides only has been found effective in managing false smut of rice at present and these varieties are

further used for the breeding purpose. Only prophylactic sprays of selected fungicides presently manage the disease. Among the fungicides tested, Triazole group of fungicides have been reported to most potential for the management of false smut. Other effective non- chemical means of control including the use of bio-control agents or plant products need to be explored.

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