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BLAST DISEASE OF RICE: A COMPREHENSIVE REVIEW

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Rice (*Oryza sativa*) is a staple food for over half of the world's population, making it a vital crop for global food security. However, rice cultivation faces numerous challenges, with blast disease, caused by the fungal pathogen *Magnaporthe oryzae*, being one of the most formidable. Blast disease, caused by the fungus *Magnaporthe oryzae*, remains a significant threat to global rice production and food security. This paper provides a comprehensive overview of blast disease in rice, including its biology, epidemiology, host-pathogen interactions, impact on rice production, and management strategies. Additionally, recent developments in research and sustainable approaches to mitigate the disease's impact are discussed.

Keywords : Rice, Blast, Magnaporthe oryzae.

Introduction

Rice blast caused by fungus Magnaporthe oryzae, is generally considered the most important disease of rice worldwide because of its extensive distribution and destructiveness under favourable conditions (Luo et al., 1998; Netam et al., 2011 Wilson and Talbot, 1994). The disease is generally considered the most important worldwide disease in all the rice growing regions of the world and has been reported in more than 85 countries (Rao, 1994). Blast disease is also known rice rotten neck and rice seedling blight (Talbot, 2003; Zeigler, et al., 1994). Rice blast spores can infect plants at all growth stages, from seedlings to maturity. The blast disease affects almost all parts of the rice plant and occurs in different crop growth stages, starting from nursery to harvesting. The disease was first reported as "rice fever" in China by Soong Yingshin in 1637 (Wang, 2014), and later, it was reported from Japan by Imochi-byo during 1704. It was first recorded in 1913, and the first devastating epidemic was reported in 1919 in the Tanjore delta of erstwhile Madras state (Padmanabhan, 1965). Later, the disease has been reported to occur in different regions of India (Padmanabhan et al., 1970; Rathour et al., 2004). The

annual losses due to rice diseases are estimated to be 10-15% on an average basis worldwide Annegowda 2021. Blast can infect rice from the seedling stage through maturity and can cause complete loss of seedling in seedbed and epidemic in the field. Infection leads to lesions on most of the plant including leaves, leaf collar, stems, and nodes, internodal parts of culms, panicles and grain. Although P. oryzae infect all foliar tissues, infection of the panicle can lead to complete loss of grain. The disease may also called leaf blast, collar rot, node blast panicle blast or rotten neck blast depending on the portion of the rice plant infected (Zeigler et al., 1994; Thurston, 1998; Webster, 2000). Symptoms develop on all above ground plant parts. Lesions or spots are the most common symptom, which are usually 1-1.5 cm long and 0.3-0.5 cm wide (NSW, 2012).

Symptoms

The fungus may attack at all the level of crop development. Symptoms develop on all above ground plant parts on leaves, nodes, rachis, glumes (Castilla *et al.*, 2009; Manandhar 1996). Lesions or spots are the most common symptom. The characteristic, isolated bluish-green, necrotic lesions, with water soaked

appearance are formed on leaf blade. Lesions are usually 1-1.5 cm long and 0.3-0.5 cm wide.

Leaf blast

Leaf lesions start as small white, grey or bluetinged spots. Under moist conditions lesions enlarge quickly to either oval or diamond-shaped spots or to linear lesions with pointed ends, grey or white centres and narrow brown borders (Figure 1). The lesions or spots first appear on leaves as minute water soaked brown specks, then rapidly enlarge, become elliptical, elongated diamond shaped or eye shaped pointed at both ends with white or greyish centre, reddish brown margin, sometimes with a yellow halo (IRRI). Severe infections may lead to death of leaves and whole plants. Lesion reduce the net photosynthetic rate and impaired the transport of water or nutrients or both and consequently affect the leaf tissues situated near the lesions (TeBeest, 2007). Leaf blast infections provide inoculum for panicles to become infected.

Collar rot

If a rice blast lesion is located at the junction of the leaf blade and leaf sheath the entire leaf can be killed. The leaf collar lesion discolours to brown and the leaf blade dies (Figure 2). The infection starts from near the base of the flag leaf near the leaf sheath. At later stage, infection proceeds upward to the leaf that girdles the flag leaf which turned brown, dry and fall off.

Node Blast

Infected nodes appear black-brown and dry (Figure 3). An infection at the node often results in the stem breaking. node portion of the culms turn brown or black and the portion above the infected node may die and breakdown as the xylem and phloem vessel of plant blocks which affects the nutrient and water supply above the infected portion (www.ipm.ucanr. edu.). The fungus produces abundant spores on the lesions (Padmanabhan, 1974 and Manibhushanrao, 1994). (Ram *et al.*, 2007) reported that when the last node is attacked, it causes partial to complete sterility.

Neck rot

Neck rot may result in death of an entire panicle (Figure 4). Symptoms appear at the base of the panicle, starting at the node. The tissue turns brown and shrivels causing the stem to snap and lodge. The node immediately below the ear forms greyish brown lesions and can cause girdling. The node immediately below the ear is infected and become dark brown to black in colour, the symptom is called neck infection. The infected panicles often break and fall off, or the whole inflorescence may break off at the rotten neck. No grain is formed if infection of the neck occurs before milky stage whereas poor quality grains are formed if the infection occurs later (www. knowledgebank.irri. org). it is the most destructive phase of the disease and is found at the reproductive and ripening stage of the crop (Bonman *et al.*,1991).

Panicle blast

Panicles which do not break or fall off as a result of neck rot may turn white to grey. Partially infected panicles may show grey-brown lesions among the panicle branches and on the stems of florets. Florets which do not fill turn grey. In case of panicle infection gray brown lesions can be easily found on panicle branches, spikes and spikelet. Over time panicle branches breaks at the lesion presence spot.



Fig. 1 : Rice Blast TNAU agritech Porttal source



Fig. 2 : Nodal blast



Fig. 3 : Neck blast

The Pathogen

The blast disease of rice is caused by Pyriculria oryzae (Cavara) (synonym: Pyricularia grisea) (Cook) Sacc., anamorph of Magnaporthe grisea (Hebert) Barr. (Synonym: Magnaporthe oryzae) (Webster and Gunnell, 1992; Zhou et al., 2007). It is filamentous ascomycetes, when young possess hyaline mycelium. On maturity, the colour of mycelium changes to olive brown. The mycelium may be inter or intracellular within the host tissues. The fungus reproduce asexually as well as sexually. The asexual life cycle begins when the hyphae of the fungus produces fruiting structures and sporulates to give rise conidiophores and conidia. The conidiophores are given out through the stomata or through the epidermal cells, singly or in the clusters. The conidiophores are septate, with two to four septa, slender, rarely branched greyish in colour. Conidia are produced terminally. There are seven to nine conidia are produced on each conidiophore. Shape of the conidium is obpyriform or obclavate, hyaline, septae and with a small basal appendage. The conidium size ranges from 14 to 40 μ in length and 6 to 15 μ in width. The fungus grows hyphae inter or intracellular within the leaf and form lesions. Each cell of conidium is uninucleate and the nucleus contains two large and two small chromosomes (Chou and Li 1965). It is hyaline to pale olive in color and measures 14-40×6 -134m (mostly 19-23×7-94 cm) and has a basal hilum protruding outwards. Conidia are released from the conidiophores by dew or rain water and are dispersed by air currents. Most conidia travel only 1 to 2 m from their original source before falling on other species or other rice plants. When favorable temperature is obtained, these conidia's germinate within three to four hours by forming several germ tubes in the presence of free water. These germ tubes form appressoria, from which infection pegs emerge and penetrate the host cells. The process of germination and penetration can be completed within 7 to 8 hours and after about four days of conidium germination, lesions appear on the leaves of the plants and in 6 to 7 days a new crop of conidium is formed.

The initial infections occur on leaves usually around tillering and appear as diamond, football, or spindle shape lesion with pointed ends. Once it is established in the host plant the fungal hyphae sporulates and produce asexual spores (Kim, 1994). Sexual reproduction occurs when two strains of opposite mating types meet to form a fruiting structure known as perithecium in which ascospores is formed (Dean *et al.*, 2005). This fungus also produces chlamydospores in the culture medium which are smooth, thick walled and 5-124 in diameter. Sclerotiums are also formed by this fungus (Padmanabhan, 1965). This fungus produces a toxin, pyricularin, which is stimulating to plant growth in high dilutions, but is phytotoxic in high concentrations. The pathogen also produces other toxins like, apicolinic acid, pyriculol and tenuazonic acid. Umetsu et al., 1974 stated that, tenuazonic acid plays a role in the formation of necrotic spots on blast infected leaves Apart from this, this fungus secretes pectinolytic enzyme, which helps in dissolving the cell walls. This fungus is non-s (non-stromatic) and produces a dark colored spherical ascocarp with a long ascillicle whose ascill is lined up by the periphysis and the ascus apex has a dome-like refracting structure. The young ascocarp is vertical. Filled with pseudoparenchyma of swollen cells arranged in rows. The developing ascus has a slightly thickened, probably bitunicate wall, which is rapidly shed at an early stage. Ascospores are four-celled and often have pointed ends. The outer cells are lighter than the middle cells. So far, 32 functional strains of Pyricularia grisea have been identified in India, out of which strains IC3 and ID 1 are prevalent in most of the paddy growing states.

Disease cycle

The primary mode of infection of this disease is not known for sure, but it is likely that the fungus survives in the seeds and remains of diseased plants. The mycelium of the fungus remains alive in infected dry straw for one or two years, but when the straw is buried in the ground, it is easily destroyed by moisture and microbial action. Although, infected crop debris is a major source of primary inoculum, infested seeds are also considered the important source (Thurtson, 1995). At the time of collection of seeds, the fungus remains in the dormant stage and to some extent, this can be important in the growth of the fungus later in the season. Faivre-Rampant et al., 2013 stated that the infested seeds produce diseased seedlings which die and serve as primary inoculum. Conidia are produced and released by overwintering fungus during the period of high relative humidity (>90%). This disease is seed borne as well as air borne. Mycelium and conidia overwintered on straw and seeds as well as on the collateral hosts like Panicum repens, Digitaria marginata, Eleusine coracana, E. indica, Panicum sp., Setaria sp. etc. which may serves as primary inoculums spreading primary infections (Paudel et al., 2017). Secondary infection of the disease spread by wind borne conidia.

The age of the plant also affects the severity of the disease. The seedling stage and then the planting stage are most susceptible to the disease and as the plants mature, the effect of the disease reduces. Khan and Libby (1958) Reported that the optimum temperature

for lesion development was 27-29 °C and the minimum temperature was 14-15 °C. Saifulla *et al.* (2011) find out that Rice blast severity reduced gradually with increased in minimum temperature from 190 C to 260 C. Tebeest *et al.* (2007) reported that period of high moisture of 12 hours or more with temperature of 24 C was highly favorable for the development of the disease.

Use of excess nitrogen in plants also makes them susceptible to diseases. The more nitrogen accumulated on the leaves, the greater will be the effect of the disease, because due to less nitrogen, the absorption of silica reduces, due to which the plant is less affected by the disease. In plants whose supply of nitrogen is reduced, more absorption of silica has been observed, due to which the plant remains disease resistant (Kumagaya et al., 1957). Phosphorus and potash alone or mixed with nitrogen have no effect on the disease. The age of the plant also influences the intensity of infection. The fungus can infect the leaves of seedlings 25 days old but not of those three months old. Upper younger leaves of seedlings are found to be more susceptible than the middle or lower older leaves. With the increase in age the resistance to infection also increases till the ear emerges when the plants become susceptible once again to neck and nodal infections.

Integrated disease management

The most usual approaches for the management of rice blast disease are fertilizers and irrigations, plantation of resistant varieties, alteration of planting time and application of fungicides. To prevent this disease, the following measures should be taken, Seeds should be selected from disease free crops and as far as possible, certified seeds should be sown by purchasing them from a reliable source.

Cultural management

Rice blast has become more difficult to control because of the pathogen's ability to survive and multiply in harsh environmental conditions and easily spread to new fields (Araujo, 2000). Destroying diseased crop debris reduce the over wintering inoculum in the field. Bastiaans, 1991 stated that rice blast fungus also survive in unfavorable condition so proper field sanitation by removal or burning of plant stubbles, long term crop rotation, summer ploughing and removal of collateral hosts is necessary. Application of nitrogen above the recommended rate and as a single application significantly increases disease incidence and disease severity. Average leaf blast incidences at the panicle primordia stage were 73% in high-N, 60% in normal-N and 43% in split-N treatments (Long et al., 2000). Therefore, it is

recommended to apply an optimum dose of nitrogen in split doses based on soil testing results. Use of healthy seeds reduces seed borne infection, Seeds should be selected from disease free crops and as far as possible, certified seeds should be sown by purchasing them from a reliable source. Flooding of the field to avoid water stress can reduce disease development. The collateral grass hosts present around the field should be detected and destroyed.

Nutrient Management

Managing blast disease in rice involves several strategies, including nutrient management. Providing the right balance of nutrients, especially nitrogen (N), phosphorus (P), and potassium (K), is essential. Excessive nitrogen can make plants more susceptible to blast, so it's crucial to avoid over-fertilization (Long *et al.*, 2000). Adequate levels of micronutrients like zinc, manganese, and copper can enhance plant immunity against blast. Foliar applications or soil amendments with micronutrient fertilizers can help maintain optimal levels. Silicon has been shown to enhance rice resistance to blast disease. Silicon fertilizers can be applied to the soil or foliage to improve plant defenses against blast (Ahn and Mukelar, 1986).

Resistant varieties

The best way to control disease is to grow disease resistant varieties (Leung et al., 2003). If disease resistant varieties are not available then sowing disease tolerant varieties will also be useful. Blast resistant varieties of rice; Such as Pusa-205, PusaNR-162, PusaNR-166 (IET 9206), PusaNR-381 (IET 9208), Abhay (95), IR 64, CR 1002, Co 4, TKM-1, Co-29, Co30, T-603, T-141, A-67, A-9 etc. and moderately resistant varieties like- SYRER. -2 (85), Ratnagiri-1 (85), Ratnagiri-2 (155), Pantsankar Paddy-1 etc. should only be grown. In the year 1989-90, improved varieties of blast disease resistant rice cultures were developed;Like- IET 8901, IET 10418, IET 9892, IET 8584, IET 9801, IET 9986, IET 9287, IET 9361, IET 9380 and IET 9941 etc. and in the year 1990-91, IET 11481, IET 11479, IET 11483, IET- 11490, IET-11517, IET-11449, IET-10420, IET-11471 etc. have been identified. In the year 1996-1997, the paddy variety Pant Sankar Dhan-1 and in the year 2001-2002, Pant Dhan 16, Yamini and PR 113 etc. were made moderately resistant and Vivek Dhan 82 and Harsan Sarai. Twelve elite germplasm viz; HPR-917, HPR933, HPR-977, HPR- 1001, HPR-1009, HPR1020, HPR-1062, HPR- 1064, HPR- 1153, HPR- 1155, HPR-1161 and HPR-1174 and six released varieties viz; Himalaya 741, Himalaya 799, Himalaya 2216, RP-2421, IR 64

and Palam Dhan 957 resistant against rice blast (Sharma, 2006).

About 100 quantitative blast Resistance (R) genes and more than 350 quantitative trait loci QTLs have been identified in rice and 19R genes have been successfully cloned and characterized (Ballini *et al.*, 2008; Sharma *et al.*, 2012). Recently, it has been found that balancing high disease resistance and yield can be done through epigenetic regulation of paired antagonistic Nucleotide-binding Leucine-Rich (NLR) receptors (Deng *et al.*, 2017).

Biological control agents and biopesticides

Biological control agents and biopesticides offer sustainable promising alternatives for disease management of blast disease in rice cultivation. These eco-friendly approaches harness natural mechanisms to suppress pathogen populations and reduce disease severity without adverse effects on the environment or human health. In the context of blast disease, several biological control agents and biopesticides have been studied and implemented with varying degrees of success. Here, we explore some of the key examples and their potential for integrated pest management strategies: Trichoderma species are well-known biocontrol agents that colonize the rhizosphere and exert antagonistic effects against various plant pathogens, including M. oryzae (Bhusal et al., 2018). They produce cell wall-degrading enzymes and antifungal metabolites that inhibit pathogen growth and induce systemic resistance in plants. Application of Trichoderma-based biopesticides has shown promising results in reducing blast severity and improving yield in rice fields. Gliocladium virens (Soilgard-5g)and Trichoderma harzianum (Bioderma-5g) Trichoderma viride (Ecoderma-5g)as seed treatment per kg and foliar sprays per liter thrice at tillering, booting and panicle initiation stage most effective in reducing the disease incidence (Anonymous, 2000, Hossain and Kulkarni 2001; Sharma, 2006). Certain strains of Pseudomonas bacteria possess biocontrol activity against M. oryzae through the production of antimicrobial compounds and competition for nutrients and colonization sites on plant surfaces. Additionally, some Pseudomonas spp. can trigger induced systemic resistance in rice plants, enhancing their ability to withstand blast infection. Bioformulations containing Pseudomonas-based biopesticides have demonstrated efficacy in field trials for blast disease management. P.fluorescens (Bioshield-5ml), most effective in reducing the disease incidence (Anonymous, 2000, Bacillus species, such as Bacillus subtilis and Bacillus amyloliquefaciens, are renowned for their biocontrol potential against a wide range of plant pathogens. They

produce antimicrobial peptides, volatile organic compounds, and enzymes that inhibit fungal growth and promote plant growth and health. Biopesticides formulated with Bacillus-based products have been evaluated for blast disease suppression, showing promising results in reducing disease incidence and severity. Extracts from botanical sources and essential oils derived from plants possess antimicrobial properties and have been investigated as potential biopesticides for blast disease management. Compounds such as neem oil, garlic extract, and cinnamon oil have shown inhibitory effects against M. oryzae both in vitro and in field trials. biopesticides namely, Achook (5ml), Spictaf (4.5 ml), Neem-Azal (3 ml), Neem gold (10 ml) Nimbicidine (5ml), Wanis (5 ml) and tulsi leaf extract (10 ml) are effective in disease management of blast disease (Jiwan et al., 2019). Utilizing plant-derived biopesticides offers a sustainable approach to blast disease control while minimizing environmental impacts. Garlic extract at higher doses and neem extract at 4 ml/15 ml PDA medium inhibit the mycelial growth of Magnaporthe grisea (Khanzada et al., 2012).

Chemical management

Chemical management of blast disease in rice can be an effective tool for reducing yield losses and ensuring crop health. However, it should be used in conjunction with other control measures as part of an integrated pest management strategy to minimize environmental impact and promote long-term sustainability. Fungicides can be applied using different methods, including aerial spraying, groundbased spraying, and seed treatments. Aerial spraying is commonly used for large-scale rice cultivation, while ground-based spraying may be more suitable for smallholder farmers. Seed treatments with fungicides can also protect young seedlings from blast infection during germination and early growth stages. Seed treatment with carbendazim @ 2g/kg + spraying of tricyclazole @ 0.06% + spraying of plant extract of Ocimum sanctum @ 15%, 7 days of first spray + spraying of Pseudomonas fluorescens @ 0.4 g/l after 7 days of first spray (Varaprasada et al., 2018) Seed treatment with Tricyclazone at 1 g/kg of seed or Thiram or carbendazim at 2 g/kg of seed isalso effective in controlling blast disease. Fungicide used to control blast fungus are Prebandazole, Dithane M-45, Carbendazim, Tricyclazole, Isoprothiolane, Edifenphos, Iprobenphos, Blasticidin, and Kasugamycin. Continuous use of fungicides can lead to the development of resistant fungal strains, compromising their effectiveness over time. To mitigate fungicide resistance, farmers should adhere to

recommended application rates, avoid consecutive applications of the same fungicide class, and incorporate non-chemical control methods into their disease management practices. It is essential for farmers to follow label instructions and adhere to regulatory guidelines when using fungicides to manage blast disease. Proper storage, handling, and disposal of fungicides are critical to prevent environmental contamination and minimize risks to human health and safety. Integrated approaches that reduce reliance on chemical fungicides should be encouraged to promote sustainable rice production systems.

Conclusion

Blast disease poses a significant threat to rice production worldwide, requiring integrated and sustainable management approaches for effective control. Continued research efforts aimed at understanding the molecular basis of host-pathogen interactions and the development of novel control strategies are essential for mitigating the impact of blast disease on global food security.

References

- Agriculture: Rice Pest Management Guidelines/ UC statement IPM Programe (UC IPM), www.ipm.ucanr.edu.
- Ahn, S.W. and Mukelar, A. (1986). Rice blast management under upland conditions. Progress in Upland Rice Research. Manila: *International Rice Research Institute*, 363-374.
- Anonymous (2000). Department of agriculture cooperation and farmers welfare Link http://pib.nic.in/newsite/Print Release.a spx? relid=186796
- Araujo, L.G.D., Prabhu, A.S., Freire, A.D.B. (2000). Development of blast resistant somaclones of the upland rice cultivar Araguaia. *Pesqui. Agropecu. Bras*, 35, 357– 367.
- Ballini, E., Morel, J.B., Droc, G., Price, A., Courtois, B., Notteghem, J.L. and Tharreau, D. (2008). A genome-wide meta-analysis of rice blast resistance genes and quantitative trait loci provides new insights into partial and complete resistance. *Mol. Plant-Microbe Interact.*, 21: 859-868.
- Bastiaans, L. (1991). Ratio between virtual and visual lesion size as a measure to describe reduction in leaf photosynthesis of rice due to leaf blast. *Phytopathol.* 81(6), 611-615.
- Bhusal, N.R., Acharya, B., Devkota, A.R., Shrestha, J. (2018). Field evaluation of Trichoderma viride for the management of rice leaf blast disease in Pyuthan district, Nepal. J Inst Agri An Sci., 35(1): 259-266.
- Blast (Node and Neck) IRRI Rice Knowledge Bank. www. knowledgebank.irri.org
- Bonman, J.M., Estrada, B.A., Kim, C.K., Ra, D.S. and Lee, E.J. (1991). Assessment of blast disease and yield loss in susceptible and partially resistant rice cultivars in two irrigated low land environments. Pl Dis 75:462-66

- Castilla, N., Savary, S., Veracruz, C.M. and Leung, H. (2009). Rice Blast: Rice Fact Sheets. International Rice Research Institute. pp. 1-3.
- Dean, R.A., Talbot, N.J., Ebbole, D.J., Farman, M.L., Mitchell, T.K., Orbach, M.J., Thon,M., Kulkarni, R., Xu, J.R., Pan, H., Read, N.D., Lee, Y.H., Carbone, I., Brown, D.Oh, Y.Y., Donofrio, N., Jeong, J.S., Soanes, D.M., Djonovic, S., Kolomiets, E., Renmeyer, C., Li, W., Harding, M., Kim, S., Lebrun, M.H., Bohnert, H., Coughlan,S., Butler, J., Calvo, S., Li-Jun, M., Nicol, R., Purcell, S., Nusbaum, C., Galagan, J.E. and Birren, B.W. (2005). The genome sequence of the rice blast fungus *Magnaporthe grisea*. *Nature*, 434: 980-986.
- Deepak Chikkaballi Annegowda, Mothukapalli Krishnareddy Prasannakumar, Hirehally Basavarajegowda Mahesh, Chethana Bangera Siddabasappa, Pramesh Devanna, Sahana Nagaraj Banakar, Haniyambadi Basavegowda Manojkumar and Siddegowda Rajendra Prasad 2021 Rice Blast Disease in India: Present Status and Future Challenges. Integrated Advances in Rice ResearchDOI: 10.5772/intechopen.98847
- Deng, Y., Zhai, K., Xie, Z., Yang, D. and Zhu, X. *et al.* (2017). Epigenetic regulation of antagonistic receptors confers rice blast resistance with yield balance. *Sci.*, 355, 962-965.
- IRRI Blast (leaf and collar) -IRRI Rice Knowledge Bank. www. knowledgebank.irri.org
- Jiwan Paudel, Saroj Belbase, Shrvan Kumar, Rivesh Bhushal, Ramu Yadav and Dipak Yadav (2019). Eco-friendly Management of Blast (*Magnaporthe oryzae*) of Rice. *Int. J. Curr. Microbiol. App. Sci.* 8(09), 2610- 2619.
- Khan, R.P. and Libby, J.L. (1958). The effect of environmental factors and plant age on the infection of rice by the blast fungus, *Pyricularia oryzae*. *Phytopathology*, 48, 25-30.
- Khanzada, M., Shah, G.S. (2012). In-vitro evaluation of fungicides, plant extracts and bio-control agents against rice blast pathogen Magnaporthe oryzae couch. *Pak J Bot.*, 44(5): 1775-1778.
- Kim, C.K. (1994). Blast management in high input, high yield potential, temperate rice ecosystems. In: Rice Blast Disease. Zeigler, R.S. and Leong, S.A. (eds.). CAB International, Wallingford, U.K.
- Kumagaya, S., Goto, Y., Hori, C., Matsuoka, M., Nakano, R. (1957). Annual change of silicate absorption and effect of calcium silicate in the rice plant. *Rept. Tokushima Agric. Exp. Stn.* 1957, 2, 13–14
- Leung, H., Zhu, Y., Revilla-Molina, I., Fan, J.X. and Chen, H. et al. (2003). Using genetic diversity to achieve sustainable rice disease management. *Plant Disease*, 87: 1156-1169
- Long, D.H., Lee, F.N. and TeBeest, D.O. (2000). Effect of nitrogen fertilization on disease progress of rice blast on susceptible and resistant cultivars. *Plant Dis.*, 84: 403-409.
- Long, D.H., Lee, F.N., TeBeest, D.O. (2000). Effect of nitrogen fertilization on disease progress of rice blast on susceptible and resistant cultivars. *Plant Dis.* 84(4), 403-409.
- Luo, Y, Teng, PS, Fabellar, N.G. and Tebeest, D.O. (1998). Risk analysis of yield losses caused by rice leaf blast associated with temperature changes above and below for five Asian countries. *Agriculture, Ecosystem and Environment*, 68:197-205.

- Manandhar, H.K. (1996). Rice blast disease: Seed transmission and induced resistance. Ph.D. thesis. The Royal Veterinary and Agricultural University.
- Manibhushan Rao, K. (1994). Rice Blast Disease. 1stEd., Daya Publishing House, Delhi. 179.
- Netam, R.S., Bahadur, A.N., Tiwari, U. and Tiwari, R.K.S. (2011). Efficacy of plant extracts for the control of (Pyricularia grisea) Blast of Rice under Field condition of Bastar, Chattisgarh. *Research Journal of Agricultural Science*, 2(2): 269-271.
- NSW (State of New South Wales through the Department of Trade and Investment, Regional Infrastructure and Services). 2012. Exotic Pest Alert: Rice blast. www.dpi.nsw.gov.au/biosecurity/plant.
- Padmanabhan, S.Y. (1965). Recent advances in the study of blast disease of rice. *Madras Agriculture Journal*, 564-583.
- Padmanabhan, S.Y. (1974). Fungal Diseases of Rice in India. 1st ed. Indian council of Agriculture Research, New Delhi. pp.15.
- Padmanabhan, S.Y., Chakravarti, N.K., Mathur, S.C., Veeraraghavan, J. (1970). Identification of pathogenic races of Pyricularia oryzae in India. *Phytopathology*. 60(11): 1574-1577.
- Paudel, M.N., Bhandari, D.R., Khanal, M.P. Joshi, B.K. Acharya, P. and Ghimire, K.H. (2017). CDD, ASON. Rice Science and Technology in Nepal (MN Paudel, DR Bhandari, MP Khanal, BK Joshi, P Acharya and KH Ghimire, (Eds). Crop Development Directorate (CDD), Hariharbhawan and Agronomy Society of Nepal (ASoN), Khumaltar. 2017
- Ram, T., Majumder, T.N.D., Mishra, B., Ansari, M.M and Padmavathi, G. (2007). Introgression of broad spectrum blast resistance genes into cultivated rice (*Oryza sativa* sp. indica) from wild rice *Oryza rufipogon*. *Current Science*. 92(2), 225-230.
- Rao, K.M. (1994). Rice Blast Disease. Daya Publishing House, Delhi, India.
- Rathour, R., Singh, B.M., Sharma, T.R., Chauhan, R.S. (2004). Population structure of Magnaporthe grisea from the north-western Himalayas and its implications for blast resistance breeding of rice. *Journal of Phytopathology*, 152(5): 304-312.
- Saifulla Khan, A.M., Khan, N.A. and Mohammad, Y. (2011). Effect of epidemiological factors on the incidence of paddy blast (*Pyricularia oryzae*) disease. *Pakistan Journal of Phytopathology*. 23(2),108-111.

- Sharma, T.R., Rai, A.K., Gupta, S.K., Vijayan, J., Devanna, B.N. and Ray, S. (2012). Rice blast management through host-plant resistance: Retrospect and prospects. *Agric. Res.*, 1, 37-52.
- Sharma, V. (2006). Integrated management of rice blast. Ph.D. thesis, Department of Plant Pathology, CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur (H.P.). pp. 1-117.
- Talbot, N.J. (2003). On the trail of a cereal killer: exploring the biology of Magnaporthe grisea. *Ann Rev Microbiol.*, 57(1): 177-202.
- Tebeest D.O., Guerber, C. and Ditmore, M. (2007). Rice blast. The Plant Health Instructor. doi: 10.1094/PHI-I-2007-0313-07.
- TeBeest DO, Guerber C, Ditmore M. Rice blast. The plant health instructor. 2007.
- Thurston, H.D. (1995). Tropical Plant Diseases. APS Press, Minnesota.
- Thurston, H.D. (1998). Tropical plant diseases second edition. APS press. American phytopathological society, 31–40.
- Umetsu, N., Kaji, J., Aoyama, K., Tamari, K. (1974). Toxins in blast-diseased rice plants. Agri Bio Chem., 38(10), 1867-1874.
- Wang, X., Lee, S., Wang, J., Ma, J., Bianco, T. and Jia, Y. (2014). Current advances on genetic resistance to rice blast disease. *Rice-Germplasm, Genetics and Improvement*, 23, 195-217.
- Webster, R.K. (2000). Rice Blast Disease Identification Guide. Department of plant pathology, University of California, Navis.
- Webster, R.K. and Gunnell, P.S. (1992). Rice blast. Pp. 14-17 In: Compendium of rice Diseases. Webster, R.K. and Gunnell, P.S. (eds.) Amercam phytopathological Society, St Paul, MN
- Wilson, R.A. and Talbot, N.J. (2009). Under pressure: Investigating the biology of plant infection by (*Magnaporthe oryzae*). Nat Rev Microbiol. 7(3), 185-195.
- Zeigler, R.S, Leong, S.A and Teng, P.S. 1994. Rice blast disease. CAB International Mycological Institute, Kew. UK
- Zeigler, R.S., Leong, S.A., Teng, P.S. (1994). editors. Rice blast disease. Int Rice Res Inst.
- Zhou, E., Jia, Y., Singh, P., Correll, J.C. and Lee, F.N. (2007). Instability of the Magnaporthe oryzae avirulence gene AVR-Pita alters virulence. Fungal Genetics and Biology.