



## **Blast Disease of Rice Caused by *Magnaporthe grisea* : A review.**

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### **Abstract**

*Rice is one of the major field crops raised in Assam. At present' rice occupies about 25.30 lakh hectares or 80% of the total cropped area of the state .It plays a significant role in the state economy. Rice is traditionally grown throughout the year. Assam occupies a special place in the rainfed rice production system in the eastern India by covering about 9% of the total rice area and contributes 8% to the food production. At the national level, the state contributes over 5% of rice area and 4% of rice production. Though rice is subjected to many fungal diseases but *Magnaporthe grisea* is one most destructive one. So, controlling this disease is very important. This paper present review on rice blast disease of rice with an emphasis on *M. grisea*.*

**Keywords:** Rice, *Magnaporthe grisea*, Blast disease.

### **Introduction**

Rice (*Oryza sativa L.*) is a major staple food crop for half of the world's population. The International Rice Research Institute, Philippines, estimates that in order to feed the growing global population, rice production must increase by another one-third by the year 2020. Rice blast is a widespread and damaging disease of cultivated rice caused by the fungus *M. grisea* (Rossmann et al.,1990). It is the most destructive pathogen of rice worldwide; around 50% of production may be lost in a field moderately affected by infection. Each year the fungus destroys rice enough to feed an estimated 60 million people (Zeigler et al.,1994). The disease is currently managed using resistant cultivars, fungicides and cultural practices. Strains of this fungus have also been reported to infect wheat, barley and turf grass. Most of the rice cultivars are susceptible to some strain of this fungus and since the pathogen is highly variable, breeding for durable resistance to blast remains a major challenge (Subhankar, 2005).

*M. grisea* is noted for expressing a large number of virulence forms or pathotypes, especially in rainfed, upland areas where the environments most favourable for epidemic development (Ou,1980).

For most rice growing areas, rice blast disease management relies on the frequent introduction of resistant rice cultivars. However, blast resistance is rarely effective for more than 2-3 years (Bonman et al.,). It is not known whether the ability of the pathogen to overcome resistant the frequency of formerly rare pathotypes or the frequent occurrence of genetic changes to new virulence forms or a combination of both phenomena. Consequently, detailed genetic information on population structure is essential for understanding strategies to reduce the impact of rice blast disease.

### **Mechanism of Pathogenesis**

To bring about rice blast disease, *M. grisea* has evolved a remarkable mechanism involving production of a cell that is required for attachment to the rice leaf surface and for generation of mechanical force to penetrate the rice leaf cuticle. To bring about rice blast disease, *M. grisea* undergoes a series of defined morphogenetic developmental steps, leading to the production of a specialized infection structure called the appressorium (Talbot, 2003). These cells are

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produced on the surface of rice leaves, and bring about plant infection primarily by physical breakage of the leaf cuticle. Experiments performed in the early 1990s demonstrated that appressoria of the rice blast fungus generate substantial turgor (Howard et al., 1991). Incipient cytorrhysis experiments were performed by applying increasing concentrations of polyethylene glycol to appressoria of *M. grisea*, and then determining the rate of cell collapse (Howard et al., 1991). In this way, the equivalent turgor within appressoria

was estimated. These experiments provided evidence that appressoria of *M. grisea* generate up to 8MPa of pressure during plant infection. As a result of this enormous turgor, the appressorium produces a narrow penetration hypha at the base of the cell, which is forced through the underlying cuticle and later develops into invasive hyphae that fill the epidermal cells of the leaf (Talbot, 2003; Valent, et al.,1991). Rice blast symptoms become apparent 4–5 days after initial infection (Valent, et al.,1991).

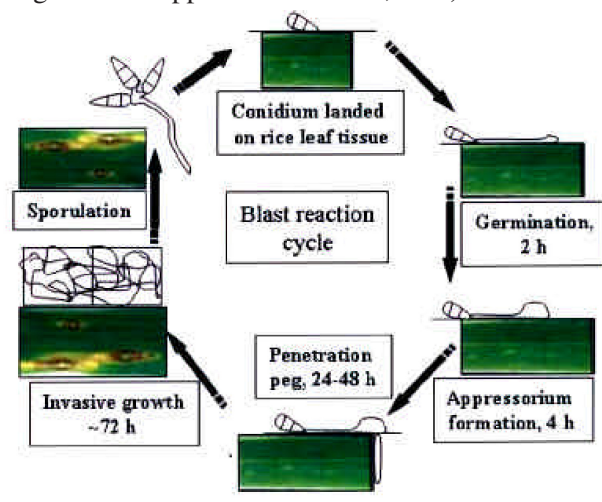


Fig: Infection process

Rice infection by *M. grisea* is initiated when three-celled, teardrop-shaped conidia land on the surface of a rice leaf. These spores germinate immediately on contact with the rice leaf, and adhere tightly to the hydrophobic surface by means of a spore tip mucilage that is released from the apex of the spore (Hamer, 1988). Germination proceeds by extension of a narrow germ tube that emerges from the conidium within an hour of its landing on the leaf surface (Talbot, 2003; Hamer, 1988). Within 4 h, the germ tube starts to swell at its apex, and flattens against the surface of the rice leaf. The germ tube apex then develops into a swollen dome-shaped cell, called the appressorium (Talbot, 2003; Valent, et al.,1991; Howard et al., 1991).

#### Structure of *Magnaporthe grisea*

Hyaline, pyriform to obclavate, 1-3 septate, conidia with rounded base and a narrow apex, provided with a small basal appendage, having slightly

constricted at septa, measuring 14-40 µm broad. Mycelium is septate, branched with multinucleate cells. From the mycelium conidiophores arise, which are simple, rarely branched, grayish, septate and slender, producing conidia at their apex. Conidia are produced singly, are ovate, 3-celled when mature with pointed blunt apex and multinucleate. Conidiophores are produced in clusters from each stoma. They are rarely solitary with 2-4 septa. The basal area of the conidiophores is swollen and tapers toward the lighter apex. The conidia of the fungus measure 20-22 x 10-12 µm. The conidia are 2-septate, translucent, and slightly darkened. They are obclavate and tapering at the apex. They are truncate or extended into a short tooth at the base.

#### Disease Cycle and Epidemiology

Plant diseases are often severe during periods of warm temperatures and high moisture. Spores do not germinate in direct sun light. Cloudy overcast

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weather, dew drops encourage blast spread. Conidia could remain viable under snow to over winter period. *M. grisea* overwinters as dormant mycelium in death plants debris. Weed hosts have been reported primarily from studies in the green house, but little is known of the importance of inoculums from weeds hosts in the field. Infected residue is probably the most important source of inoculums under the culture conditions practice in California.

The blast pathogen may go through several disease cycles in a single season. Each begins when a blast spores (conidium) infects and produces a lesions on the rice plant, produces new spores on the lesions and they are dispersed to another area on that plants or other plants. Most dispersions and spread of spores is through the air. As a result, rice in a field without leaf blast may develop collar and neck blast from air borne spores.

When environmental conditions are favorable for sporulation and infection, a single cycle of disease may occur within 7-10 days with individual lesions producing thousand of spores. This may result in several disease cycles providing exponential amount of air borne conidia to cause the various stages (leaf, collar and neck of blast) throughout the season.

Harmon and Latin 2001, found that survival of *M. grisea* was greatly reduced during the winter, but they successfully induced sporulation of the fungus from infested plant debris in the spring. Conidia produced from the leaf debris apparently serve as the primary inoculums for leaf infections early in the growing seasons, although details of this early infection process need to be determined. (Webster, 2000).

### **Symptoms**

Initial symptoms of disease are white to gray-green lesions or spots with darker borders produced on all parts of shoot. Older lesions are elliptical or spindle-shaped and whitish to grey with necrotic borders lesions wide in 22 cm in center and pointed toward either end lesions may enlarge and coalesce to kill the entire leaves symptoms also observed on leaf collar, culm, culm nodes, and panicle neck node internodal infection of the

culm occurs in a banded pattern nodal infection causes the culm to break at the infected node few, no seeds, or whiteheads when neck is infected or rotten. The disease appears on leaves, leaf sheaths, and rachis and even on the glumes. On foliage, it appears as small, bluish flecks, which on younger leaves may enlarge considerably to several centimeters long. In large lesions, the central part becomes pale green or dull greenish green and the outer rim is of dark brown colour. At later stage, the spots become grey or straw colour in the center. The most characteristic symptoms appear on clumps. The neck becomes covered with grey fluffy mycelium and becomes shriveled due to necrosis of tissues of neck, the ear breaks down and collapses (Sharma, 1998) The first symptoms of *M. grisea* infection, whitish or green-grayish spots with darker greenish edges, appeared on the leaves in all cultivars when they were in the mid tillering stage. As the damage developed, the spots gradually became whitish-greenish in colour with brown-reddish necrotic edges. The spots were of different shapes although frequently oval, elliptical with the extreme more or less pointed and facing the same way as the veins. When sporulation occurred, the centre of the spots turned ash grey due to the presence of conidia and hyphae. On average, the lesions were less than 1 cm long when the crop was in the plantlet stage, and reached 3 or 4 cm by the jointing stage. In the culm (nodes and internodes) and panicle (base, axis, branches and glumes), necrotic dark brown lesions appeared that sometimes reduced or cut off the circulation of the sap. Node damage was much more common and harmful than at the internodes; this sometimes broke the culm either partially or completely. The damage caused by *M. grisea* was frequently occasioned by dewdrops carrying the fungus sliding down the flag leaf and collecting at the collar and affecting the ligules was usually the most destructive form of infection. As with node damage, this sometimes caused a reduction in the weight grain and even led to white, erect panicles with empty grains when the attack was early and severe. Infection of the panicle base and branches (axis, primary and secondary branches)