A Look at Worldwide Rice Blast Disease Control

Rice blast is not only one of the earliest plant diseases known but also one of the most widely distributed, occurring in all major rice-growing regions. The Commonwealth Mycological Institute has recorded the disease in more than 80 countries (Fig. 1). Rice blast disease was described in China in 1637. In Japan, M. Tsuchiya wrote about the disease in his book published in 1704, according to K. Goto, and S. Miyanaga recorded rice blast in 1788. In Italy, a disease called “brusone,” considered by many authors to be blast, was reported by Astolfi in 1828 and by Brugnatelli in 1838. Metcalf, perhaps the first to call the disease “blast” in the English language (7), indicated that rice blast had been causing damage in South Carolina as early as 1876. Cavara first described the rice blast fungus (Pyricularia oryzae) in 1891.

Blast is considered the most important disease of rice. The disease strikes whenever and wherever climatic factors are favorable, and many countries have had repeated devastating epidemics. Although the epidemics are startling, the aggregated annual toll from light infections may cause even greater production losses; a 5% panicle blast often goes unnoticed.

Symptoms and Disease Cycle

The fungus attacks all aboveground parts of the rice plant. The most conspicuous symptom is leaf spot (Fig. 2). Typical spots are elliptic and about 10–15 × 3–5 mm, with a brown margin and gray center. Spots also may be smaller and round or, on resistant cultivars, be just brown specks. The number of spots ranges from a few to so many that all leaves are killed and the plant dies.

After the flowering stage, the fungus attacks all parts of the panicle, producing brownish lesions. Infection of the panicle base (Fig. 3) is particularly devastating and has been called “neck blast,” “neckrot,” and “rotten neck.” The time of infection is important—the earlier in the development of panicles, the more damage. When infection occurs early in the flowering stage, all spikelets will be empty (Fig. 4) and crop loss may be complete. The fungus also attacks the nodes, which turn black and break apart while drying up (Fig. 5).

The conidiophores of the fungus are produced on lesions in 4–5 hr under favorable conditions. The conidia are pear-shaped and usually three-celled and form on the tips of simple, denticulate conidiophores in succession, one every
30–40 min (Fig. 6). A conidiophore may bear 20 or more conidia, and a typical leaf lesion produces 4,000–6,000 conidia every night for 2 wk or longer (Fig. 7). Thus, a single diseased plant produces many millions of conidia. Recently, the perfect stage of the fungus was found in culture by mating isolates from distant countries (2).

Conidia are released from the conidiophores by dew or rain and disseminated by air currents. Most conidia travel only 1 or 2 m from their source before lodging on the rice plant. When the temperature is right, conidia germinate in water in 3–4 hr, and the germ tubes usually soon form appressoria from which infection pegs penetrate host cells. Germination and penetration may be accomplished in 7–8 hr. Lesions may appear 4 days after spore germination, and a new crop of conidia is produced in 6–7 days.

Factors Favoring Infection
The rice blast fungus is primarily a night organism. All the vital processes of the disease cycle—spore release, spore germination, infection, and spore production—require free water, and nighttime dew provides it. The longer the dew remains on the leaves, the more lesions are produced. In the tropics, the temperature is always optimal at night, and the disease is practically determined by the presence and duration of dew. Relative humidity does not seem to be as important.

In temperate regions, the fungus overwinters in straw piles and seeds. In the tropics, conidia are airborne all year because susceptible hosts are always present. Some workers found the fungus infects several grasses and plants other than rice, but other workers found such fungi to be different species of Pyricularia.

Rice is grown in all types of water regimes (Fig. 8), and upland rice is particularly prone to blast disease. Why this is so is not completely clear, but at least two factors favoring blast infection and development are involved: 1) protein and sugar contents are higher in upland than in lowland rice and 2) the dew period is longer in the uplands than the lowlands. In the tropics, lowland rice fields flooded with water have shorter dew periods than upland fields. This is because the sun heats the water in the fields to 42°C. During the night, the warm water releases the absorbed heat slowly, delaying dew formation; the closer to the water surface, the shorter the dew period.

Measures for Control of Blast
Ever since rice blast became known, control has been attempted. The early Chinese recommended washing the seeds at planting time with snow water, which had to be collected 9 days after the "Day of Winter." Some Egyptian farmers reportedly carried long poles horizontally over the rice plants at night to remove the dew.

Planting time has been shown to be important. Early plantings in Japan usually have less disease than later ones. A similar relationship has been reported in India, where severe blast is correlated with low temperature, high humidity, and heavy dew. Because of low silicon content of epidermal cells and increased nitrogen, amino acids, and amine, seedlings raised in upland nurseries are more susceptible to blast even after being transplanted. Nitrogenous fertilizers increase the protein content of rice tissues, and heavy applications are conducive to blast infection. The time at which irrigation water is drained affects disease incidence, and seedlings transplanted deep in the field are more susceptible to blast infection.

At present, the three primary control measures are cultural practices, fungicides, and cultivar resistance. Although P. oryzae has been reported to be prone to infection by some viruses and other microorganisms, the possibility of biologic control of the fungus is remote.

Use of Chemicals for Control
Numerous chemicals have been used to control rice blast. Copper compounds in the early days were followed by organomercural compounds in the 1950s and 1960s, then antibiotics and organophosphorous compounds, and, most recently, systemic fungicides.

Japan has been a leader in research, development, and use of fungicides for rice blast control. According to Okamoto (5), Bokura in 1914 found that Bordeaux mixture controlled blast in the field. Copper fungicides, however, are phytotoxic to rice plants and not only are ineffective during a severe epidemic but may reduce rather than increase yields.

In 1950, Ogawa in Japan discovered that a mixture of phenylmercuric acetate and slaked lime was surprisingly effective, low in toxicity, and inexpensive. This stimulated research on the mode of action, formulation, and methods of application for various mercury compounds, and 16 organomercural compounds were developed and used in Japan. Okamoto (5) and his associates found that organomercural compounds could generally be expressed by the formula R-Hg-X and that the most effective compounds against blast had the R represented by phenyl [i.e., phenyl-Hg], with X only slightly influencing effectiveness. The mode of action on the fungus; activities against penetration, lesion development, and sporulation; and residual effects were all studied. For about 15 yr, organomercural compounds were the fungicides most commonly used to control rice blast in Japan and other countries. The compounds helped reduce losses but were highly toxic to animals and polluted the environment. These hazardous effects eventually became obvious, and organomercural compounds have been banned in Japan since 1968.

Since 1949, when Yoshii reported that cephalothin inhibited growth of P. oryzae, many antibiotics have been tested in Japan, including antiblasticin, antimycin, blasticmycin, and blasticidin A. In 1955, blasticidin S, produced from Streptomyces griseochromogenes, was found to inhibit spore germination and mycelial growth at 1 ppm as well as or better than phenylmercuric acetate. Since then, several other excellent antibiotics, including the widely used kasugamycin, have been discovered.

Several organophosphorus compounds, such as kitazin P (IBP) and Hinosan (edifenphos), have been used successfully in Japan. Nonmercurial compounds developed and used in Japan include Blastin (pentachlorobenzyl alcohol), Oryzoxon (pentaachloromandel nitrate), and Rabecn (pentachlorophenol acetate). The seven fungicides used most commonly in Japan at present are listed in Table 1.

Fungicides for blast control have also
been tested in other countries. The International Rice Research Institute (IRRI) has been evaluating fungicides for blast control for several years and has found benomyl, thiophanate-methyl, and the experimental compounds NF-48, Hoe 22843, Hoe 22845, Hoe 22985, CGA 49104, and PP 389 to have strong systemic action and to be very effective.

Formulations and methods of application have changed gradually over the years—from powder or liquid to granules and from dusting or spraying to ultralow volume and application of granules into field water.

Chemical control of rice blast disease has succeeded in Japan for several socioeconomic and technical reasons.

The government maintains a high supporting price for rice (usually two or three times higher than the world market price) and provides extensive disease forecasting and agricultural extension services. The average yield of rice per hectare has been high. Relatively cheap chemicals and equipment are produced by well-developed industries. Even though individual farm holdings are small, aircraft can be used for large-scale application when needed because communities are well organized. The cost of application is about 2–3% of the value of the product. Therefore, chemical control of blast is economically feasible even when the disease causes only slight damage to the rice crop. Fungicides are also used in quantity on large farms in Latin America but not on small, poor farms in Asia, Africa, and Latin America, where the ingredients for successful chemical control are not available.

Drawbacks to the use of chemicals to control blast include increased cost of rice production, addition of pollutants to the environment, and development of resistance to the fungicides.

**Disease-Resistant Cultivars and Pathogen Variability**

The ideal way to control rice blast would be use of cultivars resistant to the disease. Many countries, including India, Japan, and Taiwan, have been developing
blast-resistant cultivars for 50 yr or more. Each country has developed several such cultivars. The fungus is extremely variable in pathogenicity, however, and each cultivar is useful for a few years, then becomes susceptible as new races of 

Pyricularia develop.

As early as 1922, Sasaki in Japan ascertained that rice cultivars resistant to strain A of the blast fungus were susceptible to strain B in hill regions of the same prefecture. Studies on physiologic races conducted in many countries during the 1950s and 1960s identified numerous races. Each country, however, used a different set of cultivars as race differentials. A cooperative study between Japan and the United States in the early 1960s resulted in a set of eight differentials recommended for international use so that races identified in one country could be compared with those in another (7).

In the mid-1960s, a new dimension of fungal variability was discovered. Not only were conventionally understood races present, but conidia from a single lesion or from a culture started with a single spore consisted of more than one race. The conidia continued to change in virulence pattern (race) from generation to generation (8). This genetic heterogeneity among conidia originating from single spores, although unusual, was demonstrated repeatedly and confirmed by other experiments.

The reaction of rice leaves resistant to the blast fungus is tiny brown specks. Lesions with an intermediate reaction are medium-size brown spots with small, necrotic gray centers. All reactions result from interactions between virulence gene(s) of the pathogen and resistance gene(s) of the host. When spore suspensions from single conidial cultures were sprayed on plants, all three reactions often appeared on the same leaf, suggesting that different genotypes for virulence (races) existed among the spores in the inoculum, even though all originated from the same conidium. Virulence tests of individual conidia in the spore suspension confirmed the presence of different virulence types (8). This unusual variability had been observed by earlier workers, but the significance had not been recognized.

Because the majority of conidial and mycelial cells are uninucleate, this pathogenic variability is difficult to explain genetically, although heterokaryosis and paramecricity have been suggested. A recent report (10) confirmed an earlier one that the number of chromosomes in the nuclei varies from two to 12. Furthermore, frequent lagging chromosomes, one or two per nuclear division, were noted in meiosis as well as mitosis during sexual spore development (9). K. V. S. R. Row and J. P. Crill (personal communication) studied the nuclei and chromosomes in both mycelial and conidial cells and found that hyphal cell size correlated with nuclei size, which correlated with the number of chromosomes. Nuclear configuration varied in each cell of the three-celled conidium, and the number of chromosomes ranged from two to 12, with three to six in most cases. Asynchronous division, non-disjunction, and lagging chromosomes caused the differences in chromosome numbers. These unusual nuclear divisions and the varying numbers of chromosomes appear to offer the best genetic explanation of pathogenic variability. The frequency and magnitude of chromosome variation match those of pathogenic variability.

Tests for Host Resistance

How to identify the best sources of resistance and how to screen for resistant lines are the problems involved in testing for host resistance. During the past 15 yr, the International Uniform Blast Nurseries (IUBN) has tested thousands of rice cultivars and lines at about 60 stations in 30 countries (Fig. 9). Of the 300 sets of results that have been compiled, the following are among the most significant:

1. Rice cultivars reacted differently to blast from country to country and from region to region within a country, i.e., a cultivar resistant in one area was sometimes susceptible in another. Although the fungus produced many different races, the prevailing ones at any location depended on the host cultivars being grown there. Japonica types were mostly susceptible in the temperate zone of Asia and mostly resistant in the

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*Fig. 7. Clusters of conidia developed on leaf tissue infected with rice blast fungus.*

*Fig. 8. Conidia of rice blast fungus in culture.*
tropics, whereas the reverse was true for indica types. So far, no area has been located where all races are present (such as the Toluca Valley in Mexico where tests for potato late blight are conducted).

2. Rice cultivars also reacted differently to blast from season to season in the same locality. Of 10,000 cultivars tested in the blast nursery at the IRRI, about 1,400 were initially highly resistant. Five tests and 2 yr later, however, only 400 were still resistant. The composition and frequency of fungus races in the nursery changed from month to month, although some races prevailed most of the time.

3. Cultivars showed great differences in the resistance spectrum, and this was consistent in all tests. At one end of the spectrum were resistant cultivars such as Tetep, which was resistant in 98% of tests, and at the other end were susceptible ones such as Fanny, which was resistant in 20% of tests; in between were all gradations. The spectrum was also evident when large numbers of fungus isolates were artificially inoculated on many rice cultivars.

4. Disease intensity varied from locality to locality, probably because the level of inoculum was lower or the number of races fewer in one locality than the other. Similarly, some artificially inoculated isolates infected many cultivars while others infected only a few. The general reaction pattern observed in international and national blast nurseries as well as in large-scale artificial inoculation tests indicates that many resistance genes in the host and many virulent genes in the fungus are involved.

5. A number of rice cultivars with a broad spectrum of resistance were identified, including Tetep, Carreon, Mamoria, C46-15, Nany-chet-cue, Ram Tulasi (sel), Dissi Hatif, and Huangsen-go. These cultivars have been tested for 15 yr, with consistent results, and are the best sources of resistance known.

**Stable Resistance**

Occasionally, a few typical blast lesions developed on the highly resistant cultivars identified by IUBN studies. Presumably, a new race had developed and the cultivar would eventually become susceptible. When 37 isolates from lesions on Tetep were reisolated to Tetep, however, some could not infect the cultivar, others produced a few lesions, and only two produced many lesions. The average number of lesions per seedling was two, whereas the average number of lesions on a susceptible control plant inoculated at the same time was 33. When the isolates that had produced the most lesions were then used in field inoculations, the average number of lesions on Tetep was three and that on the susceptible control was 40. Examination of each isolate showed that the conidia consisted of many races, most of which could not infect Tetep even though they had originated from that cultivar. The fungus was continually changing.

Other resistant cultivars were studied in the same manner, with similar results. The expected build up of virulent fungus strains on cultivars with a broad
spectrum of resistance did not develop. Perhaps the extreme variability of the organism prevented such a buildup, thus establishing stable resistance.

The concept that rice cultivars had fewer lesions (quantitative) in the field because they were resistant to more races (qualitative) has been confirmed in supplementary experiments (8). The apparent quantitative resistance is the total expression of qualitative resistance. Cultivars with a broad spectrum of resistance always have fewer lesions than susceptible ones, regardless of the isolate inoculated. The broader the spectrum, the fewer the lesions.

Breeding for Blast Resistance

All evidence seems to indicate that the resistance to blast of such rice cultivars as T etep is stable. How to incorporate such resistance into new cultivars, however, needs further study. The number and nature of the genes of resistant cultivars

<table>
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<tr>
<th>Chemical</th>
<th>Application</th>
<th>Preventive activity</th>
<th>Inhibitive activity</th>
<th>Residual activity</th>
<th>Remarks</th>
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<tr>
<td>Blasticidin S (antibiotic)</td>
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<td>Fuji-one (isoprothiolane)</td>
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<td>Hinosan (edifenphos)</td>
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<tr>
<td>Kasugamycin (antibiotic)</td>
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<td>-</td>
<td>++++</td>
<td>+++</td>
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<tr>
<td>Kitazin P (IBP)</td>
<td>Dust, spray, or granules</td>
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<tr>
<td>Oryzemate (probenazole)</td>
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<tr>
<td>Rabclide (tetrachloro- rophthalide)</td>
<td>Dust or spray</td>
<td>-</td>
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^a Adapted from Moji (4).
^b Activity ranging from no (−) to slight (+) to strong (++++).
are not known, but judging from the broad spectrum of resistance, many genes apparently are involved. Genetic studies are difficult because of the great pathogenic variability among spores from pure cultures. Nevertheless, Kiyosawa (3) was able to identify 13 genes for resistance to the rice blast fungus.

In most breeding programs, simple crosses between donors and commercial cultivars are made and the progenies screened for resistance. Transferring the whole complement of resistance genes from donor to progeny is difficult unless very large numbers are tested under a wide spectrum of virulence. Tetep has been used as a donor in many resistance breeding programs. The selections were more resistant than the susceptible parent, but this resistance broke down under severe epidemic conditions, while Tetep remained resistant. Progenies should be selected in at least several key test stations to allow exposure to a broad range of fungus races; this requires international cooperation. The breeding procedure also must permit pyramiding resistance genes by multiple crosses involving several donors with stable resistance.

With the recent understanding of pathogen variability and host resistance, development of new rice cultivars with more stable resistance to blast should be possible. The numerous crosses made at the IRRI during the past several years have been followed by extensive testing and selecting to find such cultivars. Results of tests at the international blast nurseries have shown that many lines from these crosses have essentially the same level of resistance as that of Tetep (1). Until a broad spectrum of resistance is incorporated into many new cultivars, blast will remain a major disease of rice.

**Literature Cited**


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