RICE BLAST DISEASE

Hajime Kato, Director of the Japan Plant Protection Association and former Head of the Department of Plant Protection, National Agricultural Research Centre, Ministry of Agriculture, Forestry and Fisheries, and Professor of Plant Pathology at Kobe University in Japan, introduces a series of articles on an important disease affecting rice growing worldwide.

Importance of the disease
Rice blast disease is distributed in about 85 countries in all continents where the rice plant is cultivated, in both paddy and upland conditions. Although the damage is very much influenced by environmental factors, this disease is recognized to be one of the most serious diseases of the rice plant worldwide.

Causal organism
Rice blast is caused by the Ascomycete fungus, Magnaporthe grisea Barr (anamorph Pyricularia grisea Sacc., synonym P. oryzae Cav.). Pyriform macroconidia, ca. 20 × 10 µm, (Figure 1) are produced on conidiophores which protrude from lesions on plants. These germinate and develop an appressorium (Figure 2) at the tip of the germ tube, which attaches to the surface of plant tissues; an infection-peg from the appresorium penetrates into plant tissues. The wall of conidiophores and appressorium are pigmented by melanin.

Symptoms
The fungus is able to infect and produce lesions on all organs of the rice plant except the root.

Leaf blast (Figure 3)
When the fungus attacks a young leaf, purple spots can be observed after an incubation period, changing into a spindle shape which has a gray centre with a purple-to-brown border, and then surrounded by a yellow zone as time passes. Brown spots appear only on the older leaves or leaves of resistant cultivars. In young or susceptible leaves, lesions coalesce and cause withering of the leaves themselves, especially at the seedling and tillering stages. Lesion formation on the n-leaf (where n is the top developing leaf), causes shortening of the n + 1 leaf sheath and the n + 2 leaf blade, with consequent stunting of the whole plant.

Neck rot and panicle blast (Figure 4)
Infection to the neck node produces triangular purplish lesions, followed by lesion elongation to both sides of the neck node – symptoms which are very serious for grain development. When young neck nodes are invaded, the panicles become white in colour – the so-called ‘white head’ that is sometimes misinterpreted as insect damage. Later infection causes incomplete grain filling, and poor grain quality. Panicle branches and glumes may also be infected. Spikelets attacked by the fungus change to white in colour from the top and produce many conidia, which become the inoculum source after heading.

Collar rot (Figure 4)
Infection at the junction of the leaf blade and the leaf sheath, i.e. the collar, readily occurs and causes browning of the tissues and withering of the leaves.

Node blast
During heading, the stem nodes which appeared from the leaf sheaths are attacked and sometimes cause lodging. Diseased nodes are brown or black in color.
**Disease cycle**

A disease cycle begins when a blast spore infects and produces a lesion on the rice plant and ends when the fungus sporulates repeatedly for about 20 days and disperses many new airborne spores. Under favourable moisture and temperature conditions (long periods of plant surface wetness, high humidity, little or no wind at night and night temperatures between 12–32 °C) the infection cycle can continue. In the canopy of rice plants, newly developed leaves act as receptors for the spores. The maximum number of spores produced was 20,000 on one lesion on leaves and 60,000 on one spikelet in one night. Lesions on leaves become an inoculum source for panicles.

**Overwintering**

The pathogen can continue to live in plants from one crop season to another in the tropics, or survive in the temperate zone on residues of diseased plants or seeds, or on ratoons of stubble. Weeds can act as alternative hosts for the disease in glasshouse tests, but their role in the field is unclear.

**Incubation period**

Incubation period, \( y \), can be expressed by \( y = -0.45 + 16.3 \) at seedling stage and \( y = -0.60 + 20.8 \) at tillering stage, where \( x \) is the average daily temperature. The incubation period is longer from spikelets (5–7 days), branch nodes and neck nodes (10–12 days) respectively.

**Lesion expansion**

Exposure of the diseased plants to higher temperatures, e.g. around 32 °C, causes lesions to expand rapidly in the first 8 days and level off shortly thereafter, then a swift cessation of lesion enlargement takes place. On the other hand, the rate of enlargement is slow and constant over the 20-day period at lower temperatures, e.g. 16 °C. Lesions expand slowly and cessation occurs gradually in the intermediate temperature regime, 20–25 °C.

**Yield loss**

Severe outbreak of leaf blast causes stunting and the development of small panicles. Early infection of neck nodes causes white head and yield loss, \( y \), expressed as \( y = 1.45x \) to \( y = 2.55x \) (where \( x \) is the percentage of white head), and \( y = 0.31x \) to \( y = 0.57x \) (where \( x \) is the percentage of diseased neck node); \( x \) is surveyed on the 30th day after heading. 15 years of data collected in Japan shows that \( y \) varies considerably under different circumstances from 1–100%.

**Control measures**

**Burning or composting of diseased tissues**

Diseased straw and stubble must be burned or composted, otherwise they can become inoculum sources for the next crop season.

**Healthy seed**

To obtain healthy seeds, the seeds must be collected from the field located under unfavorable conditions for the pathogen, and fungicide must be applied if necessary. Gravity separation methods for seeds are useful. Salt solution, 200 g l\(^{-1}\), or ammonium sulfate solution, 230 g l\(^{-1}\), is used to separate sufficiently matured seeds, followed by chemical treatment for seed disinfection against a range of pathogens.

**Fertilizer management**

Nitrogen and phosphorus content in the plants affects disease proneness. Excess nitrogen fertilizer encourage disease development, while silica application reduces disease development. Therefore the amount and type of fertilizer must be carefully decided upon according to the cultivar used, soil condition, climatic conditions and disease risk.

**Cultural systems**

Sowing into water eliminates disease transmission from seeds to seedlings because of the anaerobic condition that is unfavorable to the pathogen. On the contrary, sowing on wet soil allows seed transmission. Shade affects disease occurrence because of the longer wet condition.
Chemical control
Many fungicides are used against blast disease, including benomyl, fthalide, edifenphos, iprobenfos, tricyclazole, isoprothiolane, probenazole, pyroquilon, felimzone (= meferimzone), diclocymet, carpropamid, fenoxanil and metominostrobin, and antibiotics such as blasticidin and kasugamycin. Systemic fungicides are widely used to protect against leaf blast by seedling application and also to protect against panicle blast when applied more than 20 days before heading. The composition, amount, timing and application method of fungicide applied depends on the disease forecast or level of disease present.

To avoid pathogen resistance problems chemical control must involve the use of chemicals with different modes of action – see the three succeeding papers by Iwata, Uesugi and Kurahashi respectively which illustrate how well-established chemicals with different modes of action can continue to find a place in protection against rice blast from their contribution toward the avoidance of resistance problems.

Resistant cultivars
Race-specific and race-nonspecific resistant cultivars have been bred all over the world. Based on the information of distribution of races, these cultivars can be selected.

Forecasting systems
Forecasting systems have been developed in some countries and being used effectively.

Conclusion
Blast disease can be controlled by an integrated management system using a variety of methods – resistant cultivars, cultural practices and chemical application – based on the information from disease forecasting systems. Chemical methods of control will continue to have a role to play in fighting blast, and there follow papers on 3 classes of such chemicals—probenazole (a plant activator), choline biosynthesis inhibitors (CBIs), and melanin biosynthesis inhibitors (M BIs). Although many of these chemicals have been in use for some time they illustrate the value in retaining different classes of chemicals to help in the fight against resistance development.

Further reading