Krishi Netra
ISSN : 3048-5207

Rice Blast- A Primitive Threat To Rice

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Manuscript No: KN-V2-08/006

Blast Disease of Rice

Blast disease of rice is one of the earliest known plant diseases. Now it is of worldwide occurrence wherever rice is grown. In India, blast disease occurs in rice crop of all the states where rice is commercially grown. The disease is quite common in areas of high rainfall and in irrigated areas where the crop is overdosed with nitrogen. Several epidemics have been experienced in different parts of the world due to blast resulting in yield losses ranging from 50 to 90 percent. In India, the yield loss during the year 1960-61 was estimated to be around 2,66,340 tonnes.

Symptoms: The blast symptoms appear on leaves and other above ground parts of rice plants although leaves and the neck of the panicle are more commonly affected. Water soaked, spindle/boat shaped, whitish to greyish lesions develop on the infected leaves. The lesions may range from 0.5 to several centimeters in size and are surrounded byyellow halo. The lesions enlarge and coalesce with each other covering the whole leaf surface and the leaves subsequently fall off. Other parts of the plant such as stem, leaf collar, stem nodes and occasionally the internodes are also affected. On the stem nodes, brown to black lesions is formed and cover about 1 to 2 cm on both sides. After the emergence of heads, the fungus attacks and girdles the peduncle neck node. This stage is known as neck infection which is the most destructive. If neck infection is observed at an early stage, the paniclebecomes erect and do not fill with grains, but if the infection occurred late, the grains are partially filled. However, in such situation, the panicle base is broken due to grain weight and neck tissue is weakened. Such panicles collapse and droop down and are visible in the rice field even from far distances.

A).Spindle shaped lesions on leaves B). Nodal blast

Pathogen: Pyricularia oryzae Cavara (It is not distinguishable from P. grisea (Cooke) Sacc.) The pathogen belongs to family Moniliaceae, order Moniliales and sub-division Deuteromycotina, Phylum Ascomycota (Kirk et al., 2008)

Perfect or Teleomorph stage is Magnaporthe grisea Kato and Yamaguchi (This stage has not been found in nature but developed experimentally in the laboratory). In nature, most of the pathogen isolates are of the same mating type (male) and do not cross with each other. The teleomorphs were produced in the laboratory by crossing appropriate isolates. As it is of higher fungi, it produces septate, branchedand hyaline to olivaceous mycelium in localized lesions. The conidiophores emerge from the leaf through stomata or by rupturing the cuticle. The conidiophores bear terminal pear shaped or pyriform conidia having 1 to 3 septa, but majority arebiseptate. The conidia develop in successions and each cell of the conidium is uninucleate. Conidia measure approximately 20-25 X 8.5-9.5 micron. The pathogen produces several toxins such as pyricularin, and alpha

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picolinic acid etc.The blast pathogen has several physiological races. In India, about 32 races have been reported but races 3(1C3) and 1(1D1) are more virulent.

Etiology: The perfect stage of the pathogen is Magnaporthe grisea (Hebert.) Barr. which belongs to phylum Ascomycota. Perithecia of this fungus occur singly or in groups without apparent stroma. The asci are beak-shaped, cylindrical to subclavate containing fusiform hyaline four celled ascospores. The asci arose from the base of perithecium and measure 7-10 X 55-90 micron. Ascospores measure about 4-7 X 17-24 micron.

Several physiological races are recognized in P. oryzae based on infection types developing on different hosts by artificial inoculation. Cytological studies have shown that the chromosome number in nuclei varies from 2-12. The pathogenicity of individual conidia continues to segregate in each generation. Asynchronous division, nondisjunction and lagging chromosome appear to be the cause of differences in chromosome numbers. The unusual nuclear behaviour explains the pathogenic variability in blast fungus.

The nature of resistance in rice varieties due to blast has been studied over the past 50 years. Silicification of the epidermal cells of rice plant is considered to confer resistance against blast as it prevents the entry of the pathogen. Heavy nitrogenous manuring decreases silicate accumulation in rice plants which makes the plants susceptible to the pathogen. Rice plants have several prohibitins which are phenolic in nature. The nitrogen nutrition greatly influences the concentration of prohibitins and their oxidation. In a resistant variety browning reaction or necrotic response is due to irreversible oxidation of polyphenols of the host tissue. There is an increase in the hydrogen peroxide level of the infected tissue leading the rapid death of infected tissue. Toxins such as alpha picolinic acid, pyriculol and pyricularin will be produced by Pyricularia. Phenolic compounds detoxify alpha picolinic acid while the resistant tissues detoxify pyricularin to non-toxic substances.

Practically, manger genes conferresistance to blastin rice varieties. The higher, the degree of resistance of a variety, the fewer are the races of the pathogen that would be virulent to that variety. The rice varieties fall under the botanical group of japonica, indica, javanica and glaberrina differ in their genetic behaviour and can provide potential for breeding new blast resistant varieties of rice.

Disease Cycle: The pathogen over seasons as mycelium and conidia on rice straw and seed from contaminated crops. The inoculum may also survive on gramineous weeds near the crop or on overlapping crops of rice. The pathogen produces and releases conidia in abundance at a favorable temperature and relative humidity of 90% or more. The conidia can be formed at temperatures between 15 and 32oC but the optimum is between 20 and 28oC. The mature conidia are released by wind and reach to the healthy plants. On landing to the healthy plants, the conidia adhere by producing mucilage at their tips. Conidia germinate in the presence of free water through a germ tube. The germ tube produces an appressorium which produces and accumulate melaninsthat help the appressorium to penetrate the host through stomata. Seedlings and young leaf tissues are comparatively more susceptible to infection. Under favourable conditions, new lesions may develop within 4 to 5 days after infection. Newly formed conidia are released within hours and continue for several days. Low temperature (about 20C) and excessive dew during night may increase the intensity of the disease. Most of the conidia are released after midnight to sunrise. Reoccurrence of the chain will be seen several times till the crop is harvested.

Control: Growing of resistant varieties is the best option. The resistant genes have been identified in more than 13 rice cultivars. However, the resistance break down occurs due to appearance of new races. Therefore, resistant varieties need to be developed more frequently.

Rice cultivars like Jaya, Vani, Akashi, IR-8, IR-36 etc. are some of the promising resistant cultivars. Other methods like use of certified seed, burning of rice remains from the field, early sowing, avoid excessive doses of nitrogen and use of fungicides are also helpful in controlling the blast disease. Several fungicides are being used for controlling rice blast viz; ediphenfos, benomyl, carbendazim and antibiotics such as blasticidin -S and kasugamycin at 20 ppm.Systemic fungicides, pyroquilone and tricyclozole were found to provide better control as these fungicides interfere in melanin production by the appressorium thereby inhibiting the entry of the pathogen into the host plant. Rabcide 20% solution @ 1.5 kg/ha spray can control both leaf and neck blast.