Udbatta disease or false ergot of rice - *Balansia oryzae-sativae*

*Balansia oryzae-sativae* is an ascomycete related to the ergot fungi that is pathogenic on a number of grasses but is of particular concern when it occurs on rice. Infection of the plant is systemic and results in the loss of most or total yield. Although the wider range of hosts among both wild species and cultivated forage grasses can provide a source of inoculum, the pathogen is also shown to be seed-borne in rice. A recent introduction of the pathogen or a very closely related fungus to the USA on an ornamental grass suggests an alternative means of introduction. The pathogen is reported primarily from southern and eastern Asia and a few West African and Pacific island countries. There is evidence for ecologically limitations on its spread and, where it is already endemic, occurrence is sporadic.

*Balansia oryzae-sativae* Hashioka, 1971

**Stromata** on mummified spikelets, hemispherical, capitulate, gregarious, 0.67-1.2 µm diam with a black, coarsely papillate surface, yellowish-brown to white interior, arising from a mummified spikelet upon which the conidiomata developed. **Ascomata** with perithecia embedded in periphery stromatal head, perithecia rounded, ovoid to pyriform, 125-200 × 85-100 µm diam. **Asci** cylindrical, 92-120 × 6 µm with a rounded, thickened apex, an attenuated base, eight-spored. **Ascospores** filiform, non-septate, straight or curved, 12-27 × ca. 1 µm in width.

**Acervuli** on surface of mummified inflorescence emerging from leaf sheath; when wet, appearing gelatinous, cupulate or convex fructification, 1-1.5 mm diam bearing a palisade of conidiophores. **Conidiophores** terminating in narrow conidiogenous cells that proliferate percurrently to form a mass of filiform to acicular, hyaline **conidia**, 13-35 × 1-2 µm.

For additional details, see Tai and Siang, 1948; Hashioka, 1971; Booth, 1979.

**SIMILARITIES TO OTHER SPECIES/CONDITIONS**

Tsukiboshi et al. (2008) observed no differences in morphology between isolates of two molecularly distinguishable groups of *E. japonica*; these groups were related to those which Tanaka et al. (2001) linked to two different species of *Balansia*, the Asian *B. andropogonis* and the American *B. discoidea*. Some identifications of *E. oryzae* on grasses have been based primarily on conidium size and thus may be erroneous (Mohanty, 1975b; Reddy and Channamma, 1976; Hiremath et al., 1982). *Ephelis oryzae* and *B. andropogonis* share hosts in several grass genera: *Chrysopogon, Cynodon, Digitaria, Echinochloa, Microstegium, Panicum, Paspalum and Pennisetum* (USDA/SMML, 2010). Therefore, although *B. andropogonis* may produce the teleomorph on infected heads (Govindu and Thirumalachar, 1961), distinguishing between the species in the absence of the teleomorph is not easy.

**NOTES ON TAXONOMY/NOMENCLATURE**

The fungus causing Udbatta disease was described from India in its anamorphic (spermatial) stage as *Ephelis oryzae* by Sydow (1914). Narasimhan and Thirumalachar (1943) incubated infected rice grains on sand to obtain immature teleomorphic stromata. They referred to the fungus as *Balansia oryzae*, but did not formally describe the sexual stage. Hashioka (1971) described the teleomorph from herbarium specimens under the name *B. oryzae-sativae*.

Deighton (1937) reported a similar disease from Sierra Leone and referred to the causal organism as *E. pallida* Pat. distinguished from *E. oryzae* by its smaller spores. Deighton (1956) also found the Balansia state on rice and several wild grasses. Hashioka (1971) considered the fungus from Sierra Leone to be *E. oryzae* (Ou, 1985).

Phylogenetic studies (Kuldau et al., 1997; White., 2000; Bischoff et al., 2004) have placed the group of fungi with an *Ephelis* anamorph as a discrete group within the family *Clavicipitaceae*, which includes the ergot fungi.

Tanaka et al. (2001) found two subgroups within the clade of *Ephelis*-producing fungi, one including *B. andropogonis* and some *Ephelis* isolates from Asia and another comprised of isolates identified as *E. japonica* Henne. from grasses in Asia, the one available isolate of *E. oryzae* from India, and isolates from the American species *Balansia discoidea* ( = *B. strangulans* forma *discoidea*). Because isolates from some host plants occurred in both groups, and only one *E. oryzae* isolate was available for sequencing, it could not be concluded that all of the *Ephelis* reported from rice and other grasses in India necessarily belonged to the second subgroup (Tanaka et al., 2001), that is, some of what was reported as “udbatta” might have been caused by isolates related to *B. andropogonis*. According to Tai and Siang (1948), *Ephelis oryzae* has conidiomata that are cupulate or convex; *B. andropogonis* is another species in the Ephelis-producing clade that has cupulate conidiomata (Reddy et al., 1998).
Because Tanaka et al. (2001) noted that the name Ephelis japonica predated *E. oryzae*, some subsequent work has applied the earlier name to the udbatta pathogen (Zhou et al., 2003; Tsukiboshi et al., 2008), synonymizing that disease with “black choke” of grasses. Examining twenty isolates from grasses in Japan, Tsukuboshi et al. (2008) found two RFLP patterns of DNA among them, although the conidia could not be distinguished and the observed isolate morphology fit the descriptions of both *E. japonica* and *E. oryzae*. The more widespread type A RFLP subgroup was identified with the *B. discoidea/E. japonica* subgroup of Tanaka et al. (2001); the type A RFLP subgroup, found only in Okinawa, the southernmost part of Japan, was identified with the *B. andropogonis* subgroup. The lack of distinguishing morphology in the anamorph and the greater distribution of the isolates corresponding to *E. oryzae*, the udbatta pathogen, indicate even further that accurate identification of Ephelis species on grasses in southern Asia, at least, and probably worldwide, will require examination by molecular techniques.

**DISTRIBUTION**

As Balansia oryzae-sativae, with the anamorph *Ephelis oryzae* or *E. pallida*, this pathogen is reported primarily from southern and eastern Asia and a few West African and Pacific island countries. If the suggested identity with *E. japonica* (Tanaka et al., 2001) is accepted, then the fungus occurs in the Western Hemisphere as well, having been reported from Puerto Rico and the Virgin Islands (USDA/SMML, 2010). There is one record from rice in Louisiana, USA (Anon., 1960).

**RISK OF INTRODUCTION**

Historically, “udbatta” disease has caused significant yield losses in areas where it is endemic, but its occurrence has generally been sporadic and of minor importance (Lee and Gunnell, 1992). Use of resistant varieties and improved cultural practices eventually reduced the incidence to a very low level in both India and China (Tanaka et al., 2001). Therefore, the risk of an introduction to a rice-growing area depends on the existing level of resistance in the predominant varieties grown and the cultural practices, including seed treatment, that are already in use. Some effect of the local environment was also considered to be involved with incidence of the disease (Tai and Siang, 1948; Mohanty, 1964) but the particular factors involved were not determined.

**DETECTION AND INSPECTION METHODS**

While still within the sheaths, the panicles of infected rice plants become matted together by the mycelium of the fungus. They emerge as single, small, cylindrical rods, covered by white mycelium. In time, they become hard and sclerotium-like, bearing the conidiomata which appear as small black masses. Infected plants are usually stunted and occasionally, the white mycelium and conidia form narrow stripes along the veins on the flag leaves prior to panicle emergence. The flag leaf and sheath of infected tillers are sometimes slightly distorted and the upper leaves (including the flag leaf) may appear silvery (Booth, 1979).

**DIAGNOSTIC METHOD**

The symptoms described for this disease make it readily recognizable in mature rice plants. A means to diagnose seedlings or stunted immature plants or to test asymptomatic seed rapidly and accurately is needed. No DNA sequences for *B. oryzae-sativae* or *Ephelis oryzae* are available in GenBank for use in molecular assays, but a few sequences for several DNA regions of *E. japonica* have been deposited (NCBI, 2010). Zhou et al. (2003) report a sensitive nested-PCR technique, based on sequences in the ITS regions, for detection of fungi in either of the *E. japonica* subgroups of Tanaka et al. (2001). Species-specific primers enabled detection in asymptomatic plant tissue of grasses.

**NOTES ON CROPS/OTHER PLANTS AFFECTED**

The fungus has many grass hosts including *Isachne elegans*, *Arthronax ciliaris* var. *coloratus*, *Sacciolepis indica*, *Cynodon dactylon*, *Secale cereale* and *Oryza brachyantha* (Rao et al., 1959; Venkatateyan, 1937; Deighton, 1956). Other hosts include *Sehima nervosum* (Hiremath et al., 1991) and *Sorghum vulgare* (Gowda et al., 1981; Hiremath et al., 1982). *Paspalum orbiculare*, *Eragrostis nigra* and *Alloteropsis cimicina* were reported as newly observed weed hosts by Indrasenan and Mammen (1983). Cotton grass, *Imperata cylindrica*, was found infected in the vicinity of rice fields in Mysore, Maharashta, India (Govindu, 1969). A fungus found on *Microstegium nudum* and *Leptochloa chinensis* was tentatively assigned to *E. oryzae* by Mohanty (1975b).

Infection occurred on *Pennisetum typhoideum* (=*P. glaucum*, pearl millet) growing next to a crop of *Setaria italica* (foxtail millet). The florets in the lower half of the affected head were greyish, stuck together and pressed towards the rachis (Reddy and Channanma, 1976). *Ephelis* species, recorded as *E. oryzae*, *E. japonica* or *E. pallida*, have also been observed on, or isolated from, grass species in many additional genera, including *Acroceras*, *Andropogon*, *Brachiaria*, *Chloris*, *Chrysopogon*, *Digitaria*, *Echinochloa*, *Eragrostis*, *Eriochloa*, *Eulalia*, and *Panicum* genera, including *E. japonica* or *E. pallida*, and pressed towards the rachis (Reddy and Channamma, 1976). *Setaria italica*
Eragrostis, Eriochloa, Eulalia, and Panicum (Venkatakrishniah, 1952; Deighton, 1956; Tanaka et al., 2001; Tsukuboshi et al., 2008; SMML/USDA, 2010). In the USA, the pathogen was discovered on an imported ornamental grass, Pennisetum purpureum (Roberts and White, 2006).

SYMPTOMS

Infection is systemic and most or all of the tillers are involved. Infected plants are usually stunted and, occasionally, white mycelium and conidia form narrow stripes along the veins on the flag leaves prior to panicle emergence (Tai and Siang, 1948). The flag leaf and sheath of infected tillers are sometimes slightly distorted and the upper leaves (including the flag leaf) may appear silvery (Mohanty, 1971; Booth, 1979). Occasionally the head may fail to emerge (Narasimhan and Thirumalachar, 1943). Infected plants often produce fewer heads (Mohanty, 1976; Shivandanappa and Govindu, 1976).

Symptoms become most characteristic at the time of panicle emergence. While still within the sheaths, panicles become matted together by the mycelium of the fungus. They emerge as single, small, cylindrical rods, covered with white mycelium. Spikes infected by E. oryzae are somewhat dry and mumified with partially formed buds and, become darker in colour and more stromatic as conidial acervuli, appearing as small black masses, develop on the surface. When wet, these conidial acervuli appear gelatinous; they consist of a saucer-shaped fructification bearing a palisade of conidiophores and a mass of conidia (Booth, 1979).

BIOLOGY AND ECOLOGY

The systemic infection, results in the emergence of an erect, greyish-white, cylindrical axis from the leaf sheath instead of a normal inflorescence (Booth, 1979). The primary source of inoculum is considered to be infected seeds. There is no evidence that the pathogen is soilborne (Mohanty, 1964). Inoculation of heads before flowering resulted in a significant increase in infection in the succeeding crop (Mohanty, 1964). Inoculation of dry seeds with a spore suspension of E. oryzae, or treating dry or pre-soaked seeds with spore dust, induced the disease more effectively than inoculation of germinated seeds. Seedling infection appears to be the principal mode of infection; inoculation of plants with spore suspensions at various growth stages, including flowering, failed to produce the disease (Mohanty, 1977). Mycelium and conidia were observed in and on the leaf blades and sheathes of infected tillers (Mohanty, 1979).

The disease is prevalent at higher elevations in India, indicating that the eco-climatic conditions prevailing in these areas are more favourable to the completion of the life cycle of the fungus than those on the plains (Mohanty, 1964).

An average soil temperature of 28°C and abundant soil moisture in the nursery beds during the first week after sowing, followed by average soil temperatures of 28-38°C and adequate soil moisture in the subsequent period of growth up to flowering, is conducive to the development of the disease. Air temperature appears to have no direct effect, but may indirectly influence the soil temperature. Consequently, disease development is greater during seasons of higher air temperature (about 25°C) in the first week after sowing (Mohanty, 1976).

Conidia of E. oryzae germinate at temperatures between 18 and 30°C, with an optimum of 26°C. Cultures grow slowly, but produce abundant conidia on most common laboratory media. They are resistant to drying; about one-third of spores which were air-dried on glass slides at 26°C for 162 days were still capable of germinating in water. Approximately the same proportion of spores survived for five months on infected rice panicles exposed to winter air temperature and humidity in Yunnan, China (Tai and Siang, 1948).

The life cycle of the fungus is not well known (Lee and Gunnell, 1992). Tai and Siang (1948) suggest that initial inoculum may come from overwintering infected heads or from surviving stools of harvested plants left in damp parts of the field. That inoculation of rice heads in one season could result in infection of plants growing from the seed the next year was demonstrated by Mohanty (1964), but natural plant-to-plant inoculation has not been observed or demonstrated in the field. Inoculum for infection of rice might also come from infected wild grasses (Padwick, 1950). The role of the sexual stage is not known; it was not observed to occur in areas of India where the pathogen was endemic (Narasimhan and Thirumalachar, 1943; Mohanty, 1964, 1979).

PHYSIOLOGY AND PHENOLOGY

Tsukiboshi et al. (2008) observed subgroups among E. japonica (considered to include E.oryzae) isolates in Japan that appeared to correspond to either Balansia discoides or B. andropogonis. Both of these species were described as “epibiotic” by Reddy et al. (1998) rather than as “endophytic”. Nevertheless, the fungus does exist systemically within the plant until heading, after infecting the seed or seedling (Mohanty, 1964). Further evidence for the systemic nature of infection exists in the observation of Mohanty (1979) that approximately the same number of tillers were infected in the “ratoon” regrowth of diseased plants whose stems were cut back after the harvest.
MOVEMENT AND DISPERSAL

Natural dispersal: Conidia produced on the diseased heads or on the leaf blades (Mohanty, 1964, 1975a) are the only propagules known for this fungus in most of its range. These are likely to be wind-disseminated between plants, although plant-to-plant infection has not been demonstrated or observed. Wild grasses probably serve as a source of inoculum for rice (Padwick, 1950); the inoculum would have to be transported into the field, and wind is the likely carrier for dry spores. Vector transmission: Not reported. Accidental introduction: The pathogen can be carried on or in seed (Mohanty, 1964; Misra et al., 1994b).

SEEDBORNE ASPECTS OF DISEASE

Incidence There is no information available on incidence of natural seed infection. Mew and Gonzales (2002) do not address *E. oryzae* as a seedborne pathogen. Effect on Seed Quality Infected heads are a 100% loss (Mohanty, 1971). Seeds infected by the fungus appear discolored, abnormally small, deformed and contain whitish masses of conidia (Misra et al., 1994b). As the disease develops, the entire endosperm is destroyed, leaving a black hyphal mass covered by persistent glumes (Narasimhan and Thirumalachar, 1943). Mohanty (1964) did not report symptoms on seeds of artificially inoculated heads; damaged seed would, however, generate a poor host for systemic infection the following season. Pathogen Transmission Mohanty (1964) demonstrated that inoculation of rice heads before flowering causes a significant increase in the infection of the succeeding crop. Inoculation of dry seeds with a spore suspension of *Ephelis oryzae*, or soaking seeds with spore dust, induced the disease more effectively than inoculating germinated seeds (Mohanty, 1977). Seedling infection appears to be the principal mode of infection; inoculation of plants with sprays of spore suspensions at various other growth stages, including flowering failed to produce the disease in the plants (Mohanty, 1977). The primary source of inoculum is therefore considered to be infected seeds. There is no evidence that the pathogen is soilborne (Mohanty, 1964). Seed Treatment The fungus is seedborne and can be controlled by treatment of presoaked seeds with hot water at 54°C for 10 minutes (Mohanty, 1979). Other methods include “solar treatment” – the exposure of presoaked seeds to sunlight on a hot paved surface - and the use of ethylmercury chloride, carboxin or oxycarboxin, or thiabendazole (Mohanty, 1971). In tests of infested seed, more infection developed in untreated unsprouted seed than in untreated sprouted seed, possibly due to the faster growth of the plant. Carbendazim performed best in reducing disease incidence and increasing yields, followed by mancozeb and iprobenfos, (Sanne Gowda and Pandurangegowda, 1986). Gowda (1980) reported that a hot-water seed treatment of 54°C for 20 minutes was highly effective in controlling the disease; this is a departure from the 10 minute period suggested by other researchers. For further information on physico-chemical methods of seed treatment for control of this disease, see Mohanty (1975b). Seed Health Tests Blotter method (Misra et al., 1994a). 1. Place two or three layers of good-quality white or coloured blotting paper, moistened with distilled water, into Petri dishes (9.5 cm) of Pyrex glass or clear plastic (to allow the penetration of NUV light). 2. Distribute seeds from the sample to be tested (with or without pretreatment) evenly on the blotting paper at 25 seeds/plate. 3. Incubate the seeds at 22°C under a 12-h light and 12-h dark cycle with NUV light for 6-8 days. 4. Express the number of infected seed as a percentage of the total number of seeds. Washing test method (Misra et al., 1994a). 1. Place the seed sample to be tested in a beaker or flask and add water, with or without a wetting agent or alcohol. 2. Shake the container vigorously to remove any organisms adhering to the seed surface. 3. Transfer the washings into centrifuge tubes and centrifuge for about 5 minutes at 3000-5000 rpm. 4. Decant excess liquid from each tube and examine the extracted material under a compound microscope for fungal spores, hyphae and nematodes. 5. Stain with lactophenol blue to colour the fungal spores and hyphae. 6. Count the number of fungal spores using a haemocytometer.

IMPACTS

Economic impact: Udbatta disease, caused by *E. oryzae*, infected 9-11% of rice panicles in Bombay, India, and 5-20% in Yunnan, China (Ou, 1985). The disease was considered important in some areas of Bangalore, India, causing direct and indirect losses of between 1.75 and 3.69% for different rice cultivars (Shivandanappa and Govindu, 1976). Govindu (1969) reported 10% infection on the rice cultivar IR-8. Levels of earhead infection are usually 2-3%, but in years when the disease is severe, losses of up to 11% are common in susceptible varieties (Mohanty, 1964). Kamat and Patel (1951) reported that between 9 and 11% of the plants in northern areas of Kanara district, Bombay State, were infected. Tai and Siang (1948) recorded 5-30% earhead infection in damp fields in the Kunming Lake area of China. Preliminary disease surveys conducted in the mangrove swamp rice fields of northern Sierra Leone during the 1977 cropping season indicated that this was a potentially major disease (Fomba and Raymundo, 1978). Overall, Udbatta disease causes significant yield losses in areas where it is endemic, but its occurrence is generally sporadic and of minor importance (Gowda and Janardhan, 1980; Lee and Gunnell, 1992). Use of resistant varieties and improved cultural practices eventually reduced the incidence to a very low level in both India and China (Tanaka et al., 2001).

MANAGEMENT
SPS measures Because seed and seedling infection is apparently symptomless, uncertified rice seed, and seed and immature plants of warm-season grasses, may need to be quarantined before entry to rice or millet-growing countries while testing, either by slower cultural methods or a rapid and sensitive molecular technique (Zhou et al., 2003) is done. Cultural control and sanitary measures Sowing either very early (last week in May) or very late (first or second week of July) rather than at the usual time (second or third week of June) reduced the incidence of Udbatta disease in rice variety J1 in Jeypore, Orissa, India (Mohanty, 1964). Chemical control In field plots containing inoculated plants, carbendazim reduced disease intensity the most on rice cv. IET 1444, followed by aureofungin (a fungicidal antibiotic), iprobenfos and mancozeb. Aureofungin and benomyl increased grain yield of rice cv. Kalinga, and benomyl, more than carbendazim and aureofungin, increased that of cultivar IET 1444 (Indrasenan et al., 1981). For further information on chemical control of Udbatta disease, see Mohanty (1971) and Pandurangegowda et al. (1986). Soil treatments with carbendazim + thiram with and without seed treatment with the same material or an organic mercurial provided the best disease control and increase in yield. Under conditions of natural infection in Orissa, India. Soil treatment with carbendazim + thiram or with quintozene, without any seed treatment, was better than seed treatment alone, without any soil treatment (Padhi and Mohanty, 1984). Host resistance Some resistance to Udbatta disease was found in pathology experiments and screening trials for multiple rice diseases in India (Mohanty, 1964; Sanne-Gowda et al., 1973; Shivandanappa and Govindu, 1976; Indrasenan et al., 1982; Gangopadhyay and Padmanabhan, 1987). According to Tanaka et al. (2001), improved cultivars of rice has contributed to the near-elimination of the disease in India and China. GAPS IN KNOWLEDGE/RESEARCH NEEDS The relationship of this species to Balansia andropogonis should be clarified, and the true host ranges of both investigated. If both species occur on the same host species of other grasses, perhaps both can infect rice or sorghum. When the species distinction is clear, then the true distribution should be determined, particularly whether the E. japonica reported from the Americas is identical, or closely related, to the rice pathogen in Asia. The biology of this species requires more investigation. How does seed of rice become infected in the field - by plant-to-plant dispersal of conidia from other rice plants or from grasses, or as the result of infected and clean seeds and heads being harvested together? The pathogen has been shown to persist in the bases of rice plants and infect the "ratoon" crop. Does it persist in a similar manner in perennial grasses? The teleomorph was observed in Sierra Leone - does it also occur in Asia, and what role, if any, does it have in the cycle of the rice pathogen? A PCR technique should be tested for the detection of seed infection by E. oryzae.

References


Use this link to revisit SMML website
Diseased heads of rice - detail, 7.5x BPI 0393473

Conidiomata on rice spikelets, 22x BPI 0393473

Ephelis conidia, 400x BPI 0393473.