Data Sheets on Quarantine Pests

# Tilletia indica

## **IDENTITY**

Name: *Tilletia indica* Mitra Synonyms: *Neovossia indica* (Mitra) Mundkur Taxonomic position: Fungi: Basidiomycetes: Ustilaginales Common names: Karnal or partial bunt of wheat (English) Carie de Karnal (French) Indischer Weizenbrand (German) Bayer computer code: NEOVIN EPPO A1 list: No. 23

## HOSTS

The main host of *T. indica* is wheat (*Triticum* spp.), but it has also been found on rye (Aujla *et al.*, 1987). In inoculation experiments *Aegilops* spp., *Bromus* spp., *Lolium* spp. and *Oryzopsis* showed varying degrees of susceptibility (Royer & Rytter, 1988). Within the EPPO region, wheat and rye are the main potential hosts.

## **GEOGRAPHICAL DISTRIBUTION**

The first report of a new bunt disease in wheat came from the region of Faizalabad (Pakistan) in 1909. This was presumably Karnal bunt, which was first formally recorded in 1930 near the north Indian city of Karnal (Mitra, 1931). Within India the pathogen spread and can now be considered widespread in northern and central India (in regions where low temperatures and high humidity prevail during anthesis, viz. Delhi, Uttar Pradesh, Haryana, Punjab, Himachal Pradesh, Rajasthan, Madhya Pradesh, Jammu and Kashmir, West Bengal and Gujarat) (Singh *et al.*, 1985).

The first report of Karnal bunt from a non-Asian country came from Mexico in 1972, but the disease has been confined to localized areas (500 000 ha) within the state of Sonora (EPPO Reporting Service 513/06, 1991). Several very dubious distribution records have been published by various authors and institutions, giving Lebanon, Sweden, Syria and Turkey as countries where the fungus occurs. However, these records were based on intercepted wheat consignments and have not been confirmed by the countries or by a screening survey of the International Center for Agricultural Research in the Dry Areas (ICARDA) on wheat germplasm from the Middle East (Diekmann, 1987). For more information, see Locke & Watson (1955), Warham (1986). Very recently, isolated outbreaks have been found in southwestern USA.

#### EPPO region: Absent.

Asia: Afghanistan, India (Jammu and Kashmir, Punjab, Uttar Pradesh), Iraq, Nepal, Pakistan.

North America: Mexico, USA (Arizona, New Mexico, Texas; under eradication). South America: Brazil (limited outbreak in Rio Grande do Sul; under eradication).

#### EU: Absent. Distribution map: CMI (1989, No. 173).

## BIOLOGY

T. indica survives in the soil. In certain areas a 2-year period free from wheat reduces, but does not eliminate, the disease. However, survival and spread of the fungus usually occur on seed. Teliospores germinate in the soil, around the time of flowering of the wheat crop and normally at temperatures between 20 and 25°C (Krishna & Singh, 1982), producing a promycelium bearing many sickle-shaped primary sporidia. The primary sporidia give rise to protuberances which develop into secondary sporidia. Dhaliwal & Singh (1989) found that two types of secondary sporidia are produced: allantoid sporidia and filiform-like sporidia, of which only the allantoid sporidia are thought to be able to infect and cause the disease. Primary and secondary sporidia are dispersed by wind or rainsplash to the wheat ears and act as the primary source of infection. Germ tubes arise from secondary sporidia and grow towards stomatal openings of the glume, lemma or palea where they enter. The hyphae grow intercellularly within the glume, lemma, palea and possibly rachis, entering the base of the ovary from these tissues and leading to infection of the seed, which is normally limited to the pericarp (Goates, 1988). Temperatures of 8-20°C and high humidity associated with light rain showers and cloudy weather are most favourable for infection of the ears at flowering. Environmental conditions are considered to play a decisive role in infection, dry weather, high temperatures (20-25°C) and bright sunlight being unfavourable.

Seed- or soil-borne teliospores and their subsequent germination are, however, believed to play only a starting role in Karnal bunt epidemics (Dhaliwal, 1989). According to Bains & Dhaliwal (1989), repeated cycles of sporidial production in the ears provide more inoculum than soil-borne teliospores of *T. indica*. Secondary sporidia were also able to germinate and multiply on surface-sterilized leaves and in sterile soil as well as on glumes and leaves of resistant wheats, thus providing a large inoculum for airborne infection (Dhaliwal, 1989).

For more information, see Mitra (1931; 1935; 1937), Mundkur (1943a; 1943b), Warham (1986), Goates (1988).

## **DETECTION AND IDENTIFICATION**

#### **Symptoms**

Symptoms depend on climate, and are manifested most clearly when cool/warm, humid conditions prevail at flowering. The fungus causes a reduction in the length of ears as well as in the number of spikelets of the bunted ears. Infected plants may be dwarfed. In general, *T. indica* rarely infects more than a few spikelets per ear, and then the affected grains are not swollen. Oblong or ovoid sori, 1-3 mm in diameter, develop, containing dusty, brown to black spore masses. These characteristically smell of decaying fish (trimethylamine) as do those of *T. tritici*, *T. foetida* and *T. controversa* (EPPO/CABI, 1996). The grain is partially destroyed, the attack starting at the hilum and running along the suture, leaving the endosperm intact and covered by the whole or partly ruptured seed coat. In the case of mild infection, only a black point just below the embryo towards the suture is apparent. In advanced attack, tissues along the suture and adjacent endosperm are replaced by spores. The glumes spread apart, exposing the infected grains, and both glumes and grains may fall to the ground. For more information, see Holton (1949), Duran & Fischer (1961).

## Morphology

Teliospores are dark-reddish to coppery, dull- or dark-brown, globose to subglobose, occasionally with a mycelial fragment (apiculus) attached; 24-47  $\mu$ m in diameter (about twice the size of *T. caries* spores); exospore with thick, truncate, compact projections, 1.4-4.9  $\mu$ m high, seen in median view. Sterile cells are intermingled with teliospores in the sori; very variable, globose, subglobose, frequently lacrymiform, yellowish-brown, 10-28  $\mu$ m at their widest point to 48  $\mu$ m in overall length, with a well-developed stalk; walls laminated, up to 7  $\mu$ m thick.

Primary sporidia on average 64-79 x 1.6-1.8  $\mu$ m. Secondary sporidia on average 11.9-13 x 2  $\mu$ m.

For more information, see Duran & Fischer (1961), Khanna *et al.* (1968), Waller & Mordue (1983).

#### **Detection and inspection methods**

A quarantine procedure for testing seeds of *Triticum* spp. for *T. indica* has been described by EPPO (OEPP/EPPO, 1991b). Crops for seed should be inspected during the growing season. Field inspection should take place between heading and harvest. Any bunted seeds detected during the field inspections should be examined under the microscope for the characteristic teliospores of *T. indica*. For quarantine purposes, seed should be tested for the presence of the fungus by the washing test: about 400 seeds (eight replicates of 50) are placed in test tubes with sufficient water to submerge the seed; the test tubes are placed on a mechanical shaker for 10 min to obtain a spore suspension, then centrifuged for 20 min at 3000 revolutions per min; the sediment is examined under a compound microscope.

Direct visual observation for Karnal bunt (dry seed inspection) is regarded as insufficient for quarantine purposes since low levels of infection might pass undetected (Agrawal *et al.*, 1986) and even minimal seed infections can substantially contaminate healthy seed lots (Aujla *et al.*, 1988).

## MEANS OF MOVEMENTAND DISPERSAL

Natural spread can be substantial since teliospores can be carried over long distances by wind. Teliospores can pass through the digestive tracts of animals undamaged (Smilanick *et al.*, 1986), thus making it possible that the pathogen is distributed with farm manure. The main mode of international spread, however, is on infected wheat seeds.

## PEST SIGNIFICANCE

#### **Economic impact**

The disease appeared in the Punjab (India) around 1930. It was epidemic there in 1953-1954 (Agarwal *et al.*, 1976). Until 1970, sporadic outbreaks occurred every 2-3 years in the Punjab, Haryana and Uttar Pradesh regions, with disease incidence of 0.1-10% and annual yield losses of about 0.2% (Munjal, 1976). In 1974 and 1975, the disease was epidemic in other regions (Himachal Pradesh, Tarai areas of Uttar Pradesh and the Gurudaspur area of the Punjab) with 50% infection on the cultivar HD-2000. In 1976-1977, low levels of infection (up to 3%) were observed on cultivars HD-1553 and HD-1593 in Uttar Pradesh, Punjab, Haryana, Rajasthan and Madhya Pradesh.

When infection is severe, yield, seed quality and germination are adversely affected. Food grain is unacceptable when infection exceeds 3%.

In Mexico, where Karnal bunt appears regularly, direct losses are not very significant and do not exceed 1%. However, indirect costs to the Mexican economy are more significant due to quarantine measures which have to be applied for grain exports (OEPP/EPPO, 1991a; Brennan *et al.*, 1992). In addition, the presence of Karnal bunt in Mexico has created a need for considerable extra precautions in the dispatch of cereal germplasm material by the International Maize and Wheat Improvement Center (CIMMYT).

#### Control

High nitrogen applications and excessive irrigation favour the disease (Warham, 1986). Crop rotation may help to control the pathogen, but its value is questionable since *T. indica* can survive up to 4 years in the soil.

Chemical seed treatments have proved to be ineffective in killing the teliospores of T. indica on seeds of wheat, with the exception of mercurial compounds (Warham et *al.*, 1989) which are, however, banned in most countries.

Foliar sprays of fungicides may be used to control the airborne inoculum of primary and secondary sporidia. Propiconazole was shown to be effective against natural infection in India (Singh *et al.*, 1989). In Pakistan, propiconazole and bitertanol reduced the disease by 79 and 67%, respectively (Chandhry & Khan, 1990).

Intensive research has been carried out to breed resistant cultivars of bread wheat. It has been reported (Warham, 1986) that all commercially available cultivars in India are susceptible to the pathogen. However, Gill *et al.* (1986a; 1986b) reported two cultivars to be either tolerant or field resistant. Cultivars of bread wheat, durum wheat and triticale tested by Warham (1988) were all equally susceptible to inoculation at the boot stage. In Mexico, a few lines of bread wheat showed moderate resistance (Singh & Dhaliwal, 1989).

#### **Phytosanitary risk**

*T. indica* is an A1 quarantine organism for EPPO (OEPP/EPPO, 1980), and is also of quarantine significance for IAPSC and NAPPO. Once introduced, it would be almost impossible to eradicate the fungus since spores can remain viable in the soil for a considerable time. *T. indica* presents a hazard to both bread wheat and durum wheat in areas with favourable climatic conditions. A huge area within the European and Mediterranean region which is devoted to the cultivation of wheat and other Poaceae would be at risk to infection by *T. indica*, especially in the temperate and cool areas of western and northern Europe. Losses due to infection could have a severe economic impact, and restrictions on the export of grain wheat would certainly be applied by other continents.

## PHYTOSANITARY MEASURES

EPPO currently recommends (OEPP/EPPO, 1990) that seeds from non-EPPO countries should come frome a crop found free from *T. indica* and seeds of *Triticum* spp. from countries where Karnal bunt occurs should be tested according to EPPO Quarantine Procedure No. 37 (OEPP/EPPO, 1991b) and found free from *T. indica*. CIMMYT (1989) uses the following procedures for germplasm material sent to other continents: production in areas free from *T. indica*; propiconazole sprays of seed-production plots; treatment of seed batches in a sodium hypochlorite bath; seed treatment with carboxin, captan and chlorothalonil.

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