EPPO Datasheet: *Tilletia indica*

Last updated: 2022-03-21

IDENTITY

Preferred name: *Tilletia indica*
Authority: Mitra
Taxonomic position: Fungi: Basidiomycota: Ustilaginomycotina: Exobasidiomycetes: Tilletiales: Tilletiaceae
Other scientific names: *Neovossia indica* (Mitra) Mundkur
Common names: Indian bunt of wheat, Karnal bunt of wheat, partial bunt of wheat
view more common names online...
EPPO Categorization: A1 list
view more categorizations online...
EU Categorization: Quarantine pest ((EU) 2019/2072 Annex II A)
EPPO Code: NEOVIN



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HOSTS

The main host of *Tilletia indica* is bread wheat (*Triticum aestivum*). Durum wheat (*T. durum*), triticale (x *Triticosecale*) and rye (*Secale cereale*) can also be infected by *T. indica* (Aujla *et al.*, 1987, Fuentes-Davila *et al*., 1996). Although *Secale cereale* has been reported as a rare host, its potential to be an important natural host has been queried (Sansford *et al.*, 2008).

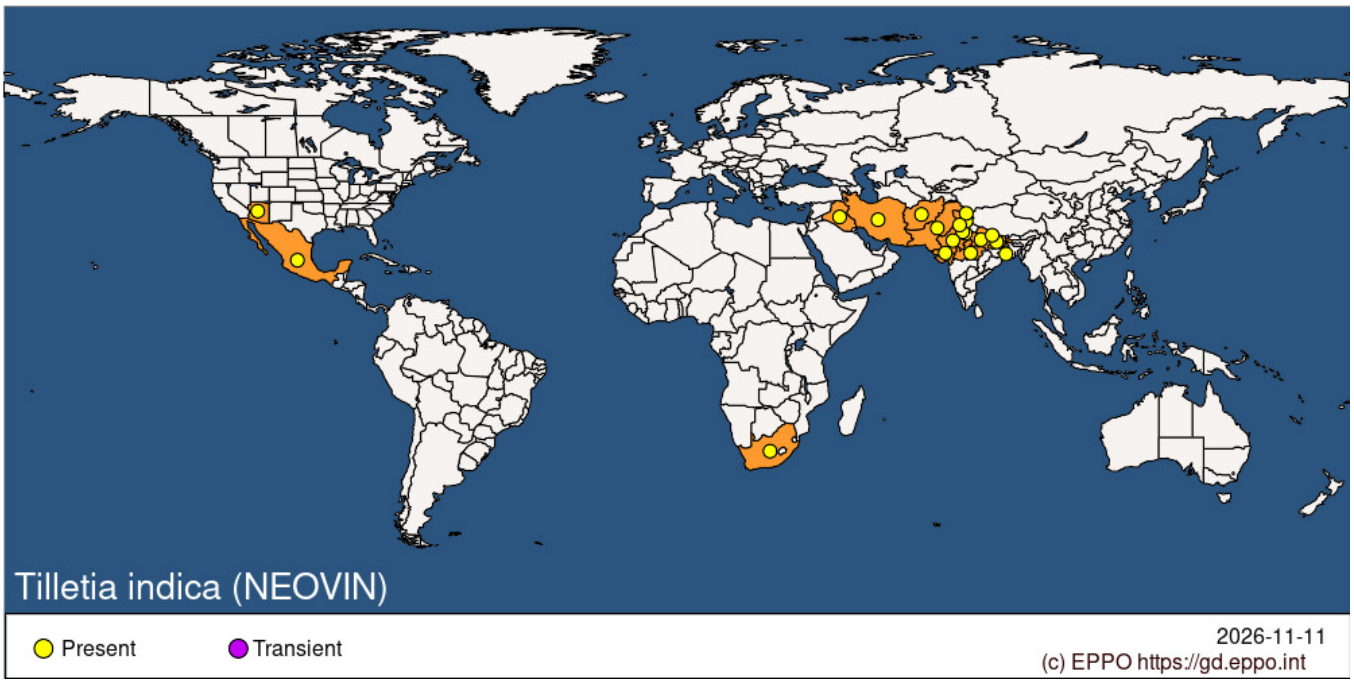
The wild relatives of wheat such as *Aegilops geniculata*, *A. sharonensis*, *A. peregrina*, *Triticum boeoticum* have been reported to be the hosts of *T. indica* by Aujla *et al.*, (1985) in vitro but this was not proved in natural conditions (Sansford *et al.*, 1998, Carris *et al.*, 2006). Numerous *Poaceae* family members (*Aegilops* spp., *Bromus* spp., *Lolium* spp., *Oloptum* sp., *Oryzopsis* sp.), a wild type of wheat (*Triticum dicoccon*) and a few cultivated wheat species (*T. timopheevii* and *T. monococcum*) are experimental hosts of *T. indica* (Mitra, 1931, Royer and Rytter, 1988, Inman *et al.*, 2003, Carris *et al.*, 2006, Kumar *et al.*, 2021).

Host list: *Aegilops peregrina*, *Aegilops sharonensis*, *Secale cereale*, *Triticum aestivum subsp. aestivum*, *Triticum boeoticum*, *Triticum durum*, *x Triticosecale*

GEOGRAPHICAL DISTRIBUTION

Typical Karnal bunt symptoms caused by *T. indica* were formally recorded for the first time in 1931 near the city of Karnal in the Indian state of Haryana in an experimental field at the Indian Agricultural Research Institute (Mitra, 1931). However, a wheat disease resembling Karnal bunt had been observed in the region of Faisalabad (Pakistan) in 1909. The disease remained restricted to the Jammu and Kashmir, Punjab, and Tarai areas of Uttar Pradesh (Agarwal *et al.*, 1977) until 1974-75. Subsequently, it became widespread in other areas of India due to the introduction of the disease into new areas when using contaminated wheat seed in the field (Joshi *et al.*, 1983, Singh *et al.*, 1985).

Karnal bunt caused by *T. indica* subsequently spread and was recorded in other parts of Asia (Afghanistan, Iran, Iraq, Nepal, and Pakistan) (Locke and Watson, 1955; Williams, 1983; Singh *et al.*, 1989; Torabi *et al.*, 1996) as well as in non-Asian countries such as Mexico (Duran, 1972), the USA (Ykema *et al.*, 1996) and South Africa (Crous *et al.*, 2001).



Africa: South Africa
Asia: Afghanistan, India (Bihar, Delhi, Gujarat, Haryana, Himachal Pradesh, Jammu & Kashmir, Madhya Pradesh, Punjab, Rajasthan, Uttar Pradesh, West Bengal), Iran, Islamic Republic of, Iraq, Nepal, Pakistan
North America: Mexico, United States of America (Arizona)

BIOLOGY

Karnal bunt is a seed-borne disease (Mitra, 1931) and contaminated wheat seeds are the major source of inoculum. Teliospores of the pathogen have an important role in the life cycle of disease and are the primary source of inoculum. Teliospores can survive in the soil for 4 years (Agarwal *et al*., 1993; Bonde *et al.* 2004) and can be spread over long distances by wind during harvest. Ingestion of infected crops with Karnal bunt by livestock does not destroy teliospores; therefore, animals' feces are also considered as a possible source of inoculum. Contaminated seeds are accepted to be the main mode of introducing and spreading Karnal bunt disease (Duveiller & Mezzalama, 2009).

The life cycle of Karnal bunt starts with harvesting of infected crops. The teliospores are released from contaminated seeds and straw during the harvest. When the soil temperature is between 20°C and 25°C, teliospores germinate and this usually corresponds to the flowering time of the wheat (Krishna & Singh, 1982). During germination, each teliospore produces a promycelium which bears sickle-shaped primary sporidia at its tip. The primary sporidia give rise to protuberances which develop into secondary sporidia (allantoid and filiform) that play an important role in the disease cycle. While the allantoid sporidia are able to infect the host plants, filiform sporidia increase the inoculum on both the host and soil surface (Dhaliwal & Singh, 1989). Primary and secondary sporidia are dispersed by wind or rain splash. 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 this leads 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 the most favorable conditions for infection of the ears at flowering. On the contrary, dry weather, high temperatures (20-25°C) and bright sunlight are unfavorable for the pathogen. Therefore, environmental conditions play a decisive role in infection; seed- or soil-borne teliospores and their subsequent germination are believed to only play 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*. For more information, see Mitra (1931; 1935; 1937), Mundkur (1943a; 1943b), Warham (1986), Goates (1988).

DETECTION AND IDENTIFICATION

Symptoms

Appearing on the spikelet of the grains, symptoms depend on climate and manifest themselves most clearly when cool or warm and humid conditions prevail at flowering. The initial symptoms are quite difficult to recognize due to the small number of grains in the spikelet that is infected by *T. indica*. Infection progresses through the germination end of the kernel and develops within the pericarps. Due to the infection, the number of spikelets and the length of ears are reduced; in addition, the infected plant's height may be shorter than healthy plants. Infected grains are generally empty and filled with dusty brown or black spore masses (i.e. oblong or oval sori of 1-3 mm in diameter) and emit a characteristic decaying fish-like smell owing to the presence of trimethylamine.

The grain is partially destroyed by *T. indica*. The attack of the pathogen starts at the hilum and runs along the suture until it covers the whole or partly ruptured seed coat, but leaves the endosperm intact. 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 and expose the infected grains. Ultimately, both glumes and grains may fall to the ground. For more information, see Holton (1949), Duran & Fischer (1961).

Morphology

Teliospores of *Tilletia indica* are globose to subglobose. Generally immature teliospores have a small hyphal fragment, which is rarely observed in mature teliospores. The teliospore diameter usually ranges between 22 to 47 µm, but in some cases, it may be 35-51 µm. The colours of immature teliospores can be rather different and vary from pale orange-brown to dark, reddish-brown and can even be black and opaque (Turgay *et al*., 2020). Mature teliospores are densely ornamented with sharply pointed to truncate spines, rarely with curved tips, 1.4–5.0 (up to 7.0) µm high, which may appear as either individual spines or closely spaced, narrow ridges in surface view. A thin hyaline membrane covers the spines (Carris *et al.*, 2006; CMI, 1983). Sterile cells of *T. indica* can be spherical, spheroidal or tear-shaped, yellowish brown, 10–28 µm × 48 µm, with or without an apiculus, with smooth walls up to 7 µm thick and laminated (Carris *et al*., 2006; CMI, 1983). The height and width of primary sporidia and secondary sporidia can be 64-79 x 1.6-1.8 µm and 11.9-13 x 2 µm, respectively. For more information, see Duran & Fischer (1961), Khanna *et al.* (1968), CMI (1983), Carris *et al.*, 2006.

Detection and inspection methods

Detection of Karnal bunt in the field is quite difficult, as usually not all the seeds on an ear are infected, and seeds are only partially colonized. Since symptoms start at the bottom of the spikelet with low disease intensity, they may easily go unnoticed during a visual inspection of the field. However, symptoms in susceptible cultivars may be apparent, especially at the end of the growing season. In the laboratory, the identification of *T. indica* may be based on the morphology of teliospores alone or in combination with molecular tests. It may also be carried out directly on suspect teliospores (EPPO, 2018).

PATHWAYS FOR MOVEMENT

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 global spread, however, is via infected wheat seeds.

PEST SIGNIFICANCE

Economic impact

The economic importance of Karnal bunt in the areas of the world where it occurs has been well-reviewed (Sansford *et al.*, 1998, Kumar *et al*., 2021). Karnal bunt infection does not lead to considerable yield losses, but it is a threat to international wheat trade since contamination levels of more than 1% affect the wheat quality and more than 3%

infected seed in consignment is considered to be not acceptable (Warham, 1990).

The disease appeared near the city of Karnal in the Indian state of Haryana and it was first reported in 1931 (Mitra, 1931). The epidemic there occurred between 1953 and 1954 (Agarwal *et al.*, 1976). Until 1970, sporadic outbreaks occurred every 2-3 years in Punjab, Haryana and Uttar Pradesh, with disease incidences of 0.1-10% and annual yield losses of about 0.2% (Munjal, 1976). In 1974 and 1975, the disease became epidemic in other regions (Himachal Pradesh, Tarai areas of Uttar Pradesh, and the Gurdaspur area of Punjab) with 50% infection level 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. During the last decade, Karnal bunt has been re-emerging in wheat growing areas of the northwestern plain zone of India and the infection rate was recorded to be up to 14.25% (Gurjar *et al*., 2016, Bishnoi *et al.,* 2020).

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 that have to be applied for grain exports (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

Seed treatments can reduce the teliospore's viability on the seed, but they have been proven to be ineffective in killing teliospores with the exception of mercurial compounds (Warham *et al.*, 1989) which are, however, banned in most countries. In the cases where treated seeds are planted in contaminated soil, as teliospores can survive up to 4 years in the soil, infections may be minimalized but cannot be suppressed (El-Naimi *et al.,* 2000).

Fungicide foliar treatments may be used to control the airborne inoculum of primary and secondary sporidia. Although these are costly, applications in late boot and flowering stages can effectively reduce the incidence of the disease (Shakoor *et al.*, 2015, Kumar & Singh, 2014).

Fumigation of soil using certain chemicals can be effective in killing teliospores; however, it is costly and not environmentally friendly (Peterson *et al.*, 1997; Sharma & Kumar, 2017). Soil solarization is also an effective strategy to eliminate Karnal bunt teliospores from the soil (Katan, 1981; Stapleton and DeVay, 1986; Sarraf and Farah, 1989; Phillips, 1990; Goates & Mercier, 2009).

It has been reported that many antagonistic fungi and bacteria are able to decrease teliospore germination in in vitro conditions (Asthana *et al.*, 2016; Singh, 1994; Sharma *et al.*, 1998; Vajpayee *et al.*, 2015); however, this still needs to be further evaluated in vivo. Sharma and Basandrai (2000) also reported that Karnal bunt could be completely controlled by the application of the biocontrol agent, *Trichoderma viride*, and fungicides.

The use of resistant varieties is the most effective control strategy for controlling Karnal bunt. Some lines of durum wheat and triticale were found to be resistant (Sharma *et al.*, 2011). These lines were involved in crossing programs in India and many resistance lines such as KBRL 10 (HD 29/HP 1531), KBRL 13 (HD 29/W 485) and KBRL 22 (HD 29/W 485) were registered (Sharma *et al.*, 2001). Karnal bunt resistance wheat lines were developed by introgression of Karnal bunt resistance from KBRL 22 into the genetic background of high yielding PBW343 (Sharma *et al.*, 2004).

Good agricultural practices (appropriate sowing time, reducing plant density, intercropping, appropriate fertilization, controlled irrigation, mulching) and crop rotation are very important and will help to control the disease and reduce its spread (Stansbury *et al.*, 2002, Sharma and Nanda, 2002; Porter *et al.*, 2002; Brooks *et al.*, 2006; Wright *et al.*, 2006).

Phytosanitary risk

Tilletia indica is absent from the EPPO region, and PRA studies have concluded that it has the potential to enter, establish and cause socio-economic damage in the wheat-growing areas of Europe (Sansford, 1998; Sansford *et al.*, 2008).

PHYTOSANITARY MEASURES

To avoid the introduction of *T. indica*, it can be recommended that seeds (for sowing) of host plants should come from areas which are known to be free from the fungus. For grain, it can be recommended that consignments should come from areas or places of production that are free from *T. indica*. Place of production freedom can be evaluated by inspection of the crop during the growing season followed by testing samples at the time of harvest and before shipment. Further guidance can be found in the EPPO Standard on consignment inspection of seed and grain of cereals (EPPO, 2021).

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ACKNOWLEDGEMENTS

This datasheet was extensively revised in 2022 by Emine Burcu Turgay, Republic of Turkey Ministry of Agriculture and Forestry, Field Crops Central Research Institute, Ankara, Turkey. Her valuable contribution is gratefully acknowledged.

How to cite this datasheet?

EPPO (2026) *Tilletia indica*. EPPO datasheets on pests recommended for regulation. Available online. https://gd.eppo.int

Datasheet history

This datasheet was first published in the EPPO Bulletin in 1980 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2022. It is now maintained in an electronic format in the EPPO Global Database. The sections on 'Identity', 'Hosts', and 'Geographical distribution' are automatically updated from the database. For other sections, the date of last revision is indicated on the right.

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Co-funded by the European Union