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Karnal bunt disease a major threatening to wheat crop: A review

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Abstract
Wheat has been a source of staple food to mankind since ancient times. Decreased production of wheat in the major wheat growing countries may be attributed to prevalence of Karnal bunt disease. The major impact of Karnal bunt is yield reduction and a decrease in quality of grains by imparting a fishy odour and taste to the wheat. The disease has gained significant importance due to the fact that it is prevalent only in a few countries around the world. The pathogen *Tilletia indica* is soil and seed borne which pose a serious quarantine problem and thus interferes with wheat trade. Early recognition of the pathogen is a critical step in analysis and its management. The present review highlights a brief outline of the pathogen, symptoms and various methods like seed treatment, crop rotation, fungicide application etc. for the control of Karnal bunt disease.

Keywords: Bunt, trimethylamine, bunt ear, teliospore, seed treatment

Introduction
Karnal bunt of wheat (*Triticum aestivum* L.), caused by the smut fungus *Tilletia indica* Mitra (*Neovossia indica* (Mitra) Mundkur), was first discovered in 1930 at the Botanical Research Station, Karnal, Haryana, in northwest India (Mitra, M. 1931) [32, 33] and now is considered common in the Punjab region of India. The disease has been reported from Pakistan, Iraq, and Nepal, and is found in wheat from Afghanistan (Bonde, M. R. *et al.*, 1996) [6]. It was first reported in Mexico in 1972 (Duran, R. 1972) [15], and since then it has occurred sporadically in localized areas in the states of Sonora and Sinaloa, northwest Mexico. Because the disease was not known in major wheat producing countries, trade of Karnal bunt– infested wheat grain became highly regulated internationally, and the Mexican government in 1984 placed an internal quarantine on Karnal bunt to prevent disease spread within the country (Mathur, S. B. *et al.*, 1993) [30]. Karnal bunt pathogen is seed, soil and air-borne. It affects mainly common wheat, durum wheat, triticale and other related species. The disease reduces seed quality, changes the chemical composition of infected grains, and makes seed inedible. Wheat containing 3% bunted grains is unfit for human consumption. Highly infected grains show remarkable decrease in the seed viability. Increase in grades of infection leads to continuous decrease in fertility. The disease is characterized by a fishy odour, which is due to the presence of trimethylamine secreted by teliospores. (Ullah *et al.*, 2012) [48].
The teliospores of the Karnal bunt fungus can survive in soil for more than 5 years (Krishna and Singh, 1983; Babadoost *et al.*, 2004; Bonde *et al.*, 2004) [27, 3, 5]. Although many control strategies have been suggested for the management of Karnal bunt disease and the strategies include biological treatments, soil fumigation and seed treatment with fungicides and soil drenching with fungicides (Anonymous, 2005) [2]. However, the results were not convincing, the cheapest and the most feasible method of Karnal bunt control is the use of resistant varieties to Karnal bunt disease. This paper reports on the screening of advanced lines and commercial varieties of wheat for the sources of resistance against Karnal bunt disease of wheat.

Losses due to karnal bunt
Karnal bunt can reduce wheat yields. There is no estimate of losses, due to this disease, occurring in Pakistan (Shakoor *et al.*, 2014) [40], However, survey in India conducted during the years of heavy disease revealed a total loss of almost 0.5 percent, but in some fields

where 89 percent of the kernels were infected, the yield losses ranged from 20-40 percent in highly susceptible varieties (Anonymous, 2004) [1]. Brennan *et al.* (1990) [9] estimated the economic losses from Karnal bunt of wheat in Mexico to be US $ 7.02 million per year. Besides yields losses, Karnal bunt can reduce wheat flour quality due to fishy, unpalatable odour and taste, if a grain lot contains 1-4 percent infected seed (Bonde *et al.*, 1997; Hussain *et al.*, 1988; Mehdi *et al.*, 1973; Sekhon *et al.*, 1980) [8, 20, 31, 39]. If in a grain lot 5 percent of the grain is infested, the quality of the flour recovery and chemical changes in composition of flour and gluten contents cause poor dough strength (Sekhon *et al.*, 1980; Gopal & Sekhon, 1988) [39, 18]. Karnal bunt is also a disease of quarantine interest and it affects the international trade of commercial wheat grain and movement of wheat germplasm throughout the world. Thus presence of diseased grain in wheat lots can cause economic loss to wheat exporting countries (Bonde *et al.*, 1997, Babadoost, 2000; Butler, 1990) [8, 3, 11].

Epidemiology

Karnal bunt initiation and development is dependent on suitable weather conditions during the period wheat plants are flowering and most susceptible to infection (Singh, A. 1994) [46]. According to Singh the optimum temperature range for teliospore germination is 15 to 25 °C (Singh, A. 1994) [46]. Smilanick *et al.*, 1985 [47] reported that the optimum after 3-week incubation in continuous light was 15 to 20 °C over a pH range of 6.0 to 9.5.Moisture is a critical element in determining whether there will be a disease outbreak (Gill, K. S., 1993, Smilanick, J. L. *et al.*, 1985) [17, 47]. Teliospore germination requires at least 82% relative humidity (RH) and preferably, free water (Singh, A. 1994) [46].

Symptoms

Due to seed borne nature, the symptoms of the Karnal bunt disease become evident only after threshing. However, Duran and Cromarty detected the infection of grains in an ear head by their swollen appearance and slightly wider opening of the glumes. The glumes open apart exposing the bunted grains which later fall off on the ground with a little jerk in severely infected spikelets (Duran, R. and Cromarty, R., 1977) [16].

There is reduction in length of the spike and number of spikelets in the infected plant (Mitra M., 1937) [35]. All the ears are not affected and in an ear head all the spikelets are not bunted (Mitra, M., 1935) [34]. The infected grains are partially or completely converted into bunt sori (Plate -2). The sori are oblong to ovoid, 1-3 mm long, brown to black in colour and contain black powdery spores mass enclosed in the pericarp (Mitra, M., 1931;, Mundkur, B.B., 1940) [32, 33, 36]. In severely affected kernels, most of the endosperm along with longitudinal furrow, together with the scutellum, is destroyed leaving only the pericarp and the aleuronic layer (Joshi, L. M., *et al.*, 1980) [24].

Disease cycle

Karnal bunt was reported as a soil borne disease by Mitra (1931) [32, 33], but now it is considered as an air borne disease. The fungal spores are also transferred by means of equipment, tools or by man moving from milling places. The spores remain viable for several years in soil (Bonde *et al.*, 2004) [5], wheat straw and farm yard manure. Soil or seeds are primary sources of inoculum (Fig. 1). Environment plays a key role in disease progression. Teliospores germinate at suitable temperature (15–25 ºC) and humidity in the soil. This condition generally dominates during February to March in North Indian plains (Dumalasová & Bartoš 2009, Rush *et al.*, 2005) [14, 38].

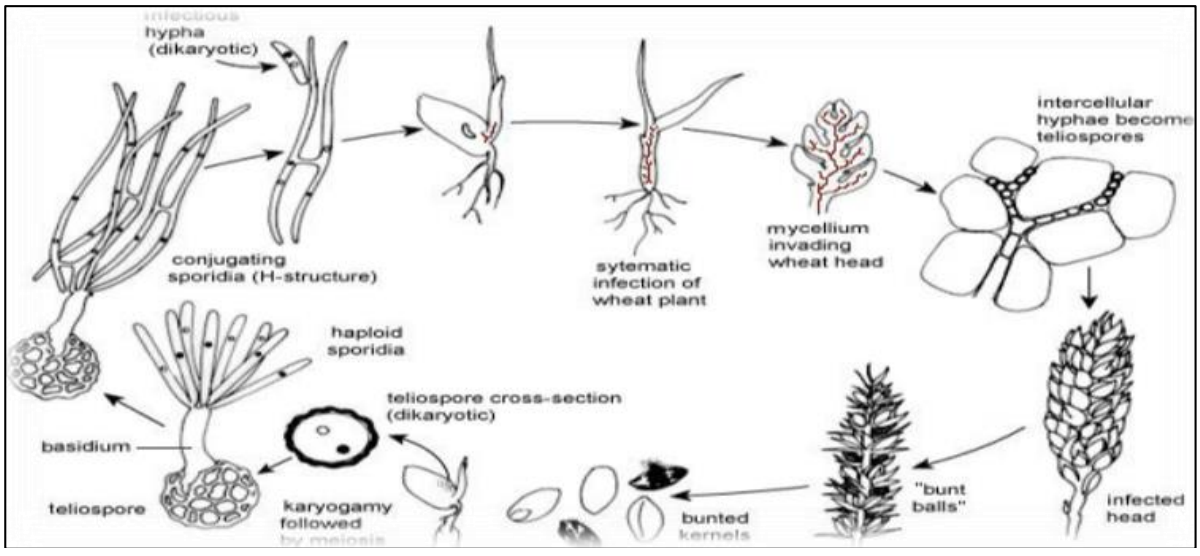

Fig 1: Disease cycle of the pathogen

On germination, each teliospore produces promycelium which bears 110–185 primary sporidia at its tip. The primary sporidia are sickle shaped and were regarded as infective entities. Now it is well known that secondary sporidia (allantoid and filiform) play an important role in the disease cycle of the pathogen. The allantoid sporidia are pathogenic while filiform sporidia increase the inoculum by division on host/soil surface. The sporidia are mostly binucleate and on 169 germination produce a germ-tube that penetrates the developing grain through stigma or ovary wall. Infection takes place mainly at the time of anthesis. Generally, the grains are moderately affected but in severe conditions whole grain may be infected (Carris *et al.*, 2006, Castlebury *et al.*, 2005, Inman *et al.*, 2008) [13, 21].

Management practices

Resistant varieties

Use of resistant varieties is the most effective method for controlling the disease. Among recent wheat varieties, PBW 502 is resistant while the cultivars Pastour, N-75-3 and N-75-5 are partially resistant. Some lines of wheat and allied genera (*Aegilops*) were found to be resistant to bunt and this resistance can be manipulated for transferring into bread wheat (Jafari *et al.*, 2000, Ullah *et al.*, 2012) [22, 48].

Seed treatment with fungicides

This method has been used to reduce the spread of inoculum via seed, however, there are only a few fungicides currently registered for use against bunts. Control of Karnal bunt has now become a major concern in India due to lack of desired resistance in popular bread wheat cultivars in Northern plain and Central zones coupled with favourable weather conditions at flowering stage favour the high incidence of Karnal bunt. Fungicides namely corboxin (Vitavax 75WP), Carbendazim (Bavistin 50WP), Subeej (Bavistin 25SD), Propiconozole (Tilt 20EC), Vitavax power (Cromp. Uni. Royal) along with one untreated control. All these five fungicides were tested at three concentrations *i.e.* 0.1%, 1.0% and 2.5%. The radial growth was measured after 15 days and per cent inhibition radial growth was calculated and propiconazole (Tilt 20EC) was found most inhibitory against the radial growth (Shukla, D. N. *et al.*, 2018; Shukla, D. N. *et al.*, 2018) [44, 45]

Seed soaking in Lantana (*L. camara*) or Eucalyptus (*E. globulus*) or Akh (*Calotropis procera*) or Kali basuti (*Eupatorium adenophorum*) @ 250 mL/L for 60 min and dry in shad are effective in eradicating the seed infection (Kumar S., 2011; Kumar S., *et al.*, 2014) [28, 29].

The main problem of using current fungicides is that Karnal bunt spores may germinate when the chemical is washed off the spore (Sharma & Basandrai 2000) [41].

Crop rotation

Crop rotation is also useful in managing the disease. The number of viable teliospores in the soil can be reduced by increasing the time between wheat crops. Rotation with non-host crops may reduce viable spores sufficiently to control the disease (Sharma *et al.*, 2004) [43]. Crop rotations should always be considered when cropping decisions are made as it provides many benefits in addition to disease control (Porter *et al.*, 2003, Brooks 2011) [37, 10].

Soil fumigation

Fumigation of soil with chemicals such as methyl bromide, metham-sodium and formaldehyde has been partially successful for killing teliospores, but is not likely to be cost effective except for eradicating a new introduction of fungus (Sharma & Kumar, 2017) [42].

Biological control

There are several reports on the biological control of common bunt. As early as 1975, Kollmorgen and Jones (Kollmorgen, J. F. and Jones, L. C., 1975) [26] demonstrated that isolates of *Streptomyces* and *Bacillus* species can cause marked reductions in teliospore germination of *T. caries* and *T. laevis in vitro*. In a follow up study, Kollmorgen (Kollmorgen, J. F., 1976) [25] observed that *Bacillus* species reduced disease incidence of common bunt under field conditions. McManus *et al.*, reported that some strains of *Pseudomonas fluorescens* inhibited the germination of *T. laevis* teliospores and reduced bunt incidence by 65% when wheat seeds were inoculated with these strains (McManus, P. S. *et al.*, 1993). Hokeberg *et al.* and Johnsson *et al.* found that one *P. chlororaphis* isolate, MA 342, is a potent inhibitor of *T. caries* in the greenhouse and in the field (Hokeberg, M. *et al.*, 1997; Johnsson, L., 1998) [19, 23].

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