

Review of Plant Pathology

Karnal bunt (*Tilletia indica*) of wheat – a review.

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INTRODUCTION

Wheat (*Triticum aestivum*, *T. durum* and *T. dicoccum*) is grown in India during the mild winter months of November to April. During 1995 nearly 65.2 m tonnes of wheat was harvested from 24.5 m ha. The widespread occurrence of Karnal bunt (KB) of wheat caused by *Tilletia indica* (Mitra) Mundkur, affects the quality of wheat grain. KB was first detected in 1931 at Karnal (Haryana), hence the name Karnal bunt (Mitra, 1931). The disease is known by various names such as kernel smut, Karnal bunt, partial bunt etc., and is native to south Asia. The disease is distributed all over north-west India in an endemic form and occurs in traces over a larger part of south Asia (Warham, 1986). Besides India and Pakistan, KB is reported from Syria (Williams, 1983), Afghanistan, Mexico (Joshi *et al.*, 1983), Nepal (Singh *et al.*, 1989), USA (Ykema *et al.*, 1996) and Iran (Torarbi *et al.*, 1996).

Since 1931, KB has appeared several times infecting land races of wheat grown over north western India. The disease remained less damaging until 1970 and subsequently severe epidemics started occurring, coinciding with the change to high yielding irrigated, semi dwarf and high fertilizer input farming. The change in wheat production technology, fall in organic matter content in soil and several other reasons are given for the increase in KB incidence. In India, KB occurs in the states of Punjab, Haryana, Jammu and Kashmir,

Himachal Pradesh, Uttar Pradesh, Delhi, Rajasthan and Bihar. The disease is endemic in Gurdaspur, Hoshiarpur, Jalandhar and Ropar districts (the submountainous tracts) of Punjab, India and along the rivers and canal network of Satluj and Beas (Gill *et al.*, 1993)

ECONOMIC IMPORTANCE

Brennan *et al.* (1990) estimated the economic losses from KB of wheat in Mexico. The direct quality and seed export losses were estimated at c. 0.12%/year in north-western Mexico. Indirect losses include those associated with the measures aimed at preventing the spread of KB and reducing its severity, for example quarantine restrictions and the cost of grain fumigation. Thus, the total losses (direct and indirect) due to KB in Mexico was estimated to be 7.02 million US dollars/year. No such information is available for India or Pakistan. More than the tonnage reduction and loss of grain quality, the quarantine related procedural wrangles create trade barriers.

TAXONOMIC POSITION

Tilletia indica, the causal agent of KB described by Mitra (1931), was transferred to the genus *Neovossia* by Mundkur (1940) since the pathogen produces numerous independent sporidia. Based on a detailed taxonomic study (Munjal, 1970) and

Krishna & Singh (1982a) justified the placement of *N. indica* under *Neovossia*. However, western scientific literature prefers to designate the causal agent of KB as *Tilletia indica* (Duran & Fischer, 1961; Duran, 1972). *N. indica* is pathogenic to *T. aestivum*, *T. durum*, *T. boeoticum*, *T. ovatum*, *T. variabilis*, *T. shareonensis* and triticale, and under artificial inoculation even *Aegilops* spp. are susceptible (Dhaliwal *et al.*, 1986; Royer *et al.*, 1986; Warham *et al.*, 1986).

SYMPTOMS AND HISTOPATHOLOGY

Karnal bunt is a disease of the seed, and symptoms become evident only when the grain fully develops. The pathogen converts the infected ovary into a sorus where a mass of dark brown coloured teliospores are produced. Small sori are generally formed in the embryo portion in the longitudinal furrow, leaving the rest of the seed unaffected. However, in severe cases the major part of the endosperm along the longitudinal furrow may get spoiled. If the host is vulnerable, the entire grain turns into a sorus if the environment is favourable. During threshing the teliospores are released from the severely infected grains and are dispersed. In partially infected grain, the embryonic tissue is not invaded and such KB infected seed do germinate and produce a weak plant that has poor survival chances. The infected grain emits a fishy odour due to trimethylamine and the wheat products from severely KB infected grain are unpalatable (Sekhon *et al.*, 1980; Singh & Bedi, 1985).

In a stand, all the earheads do not get infected, nor do all the grains in a spike. It seems as though fields, earheads and grain are infected randomly. In fact, partially infected grains occur in clusters of spikelets and many such aggregate clusters occur in a spike. In a standing wheat crop, the infected spike can be detected by the shiny silvery black spikelets, with glumes spread apart and swollen ovaries. The infected ears emit a foul smell of trimethylamine, a volatile compound, produced due to pathogenesis. Once the spikelet gets infected by the pathogen, the mycelium moves within the earhead infecting the adjoining spikelets and produces partially infected grains (Dhaliwal *et al.*, 1983).

Generally the endosperm and the dorsal side of the seed remain unaffected. Recently, Cashion & Luttrell (1988) have demonstrated that the pathogen does not invade the embryo and that mycelial growth is limited to the pericarp. Transmission electron microscopy shows that the mycelium proliferates in the pericarp by disintegrating the middle layers of parenchymatous cells and prevents the fusion of the outer and inner layers of pericarp with the seed coat. The mycelial mat forms a compact hymenium-like structure and gives rise to short, septate stalks that bear single teliospores (Roberson & Luttrell, 1987). Mycelial growth ruptures the connection between the pericarp tissue surrounding the vascular bundle in the bottom of the adaxial groove in the pericarp and the nuclear projection along the length of the developing seed. The consequence is atrophy of the seed through disruption of normal flow of nutrients from the pericarp and it starves first the endosperm and then the embryo. The endosperm is shrunken to varying degrees and normally the embryo is not infected or damaged except under very severe infection.

SOIL BANK

During harvesting and threshing the KB-infected grains release teliospores and contaminate the soil and seed. Since soilborne primary inoculum is the source for the annual recurrence of the disease, a procedure was developed to quantify the teliospore population in soil (Datnoff, 1986). It has been observed that in many fields of the Indian Punjab the teliospore density is 5×10^3 to 16×10^3 / 250 g of soil. This indicates that teliospore availability is not a limiting factor in the recurrence of KB. The soilborne teliospores have dormancy and retain their viability for more than eighteen

months when buried in soil at a depth of 5 cm. One-year-old teliospores germinate in compost extract, grain extract medium and even in plain water in 2-3 weeks. Nagarajan (1991) was of the view that the teliospores lose their viability at extremely low (below freezing) and high temperatures. The argument stems from the fact that the disease rarely occurs in the irrigated areas bordering the Indian Thar desert. The fringes of the desert, being dry and hot, reduce teliospore viability. Similarly KB does not occur at elevations above the snow line in the Himalayas, where thawing and snowing occur several times during the winter months. Vivek *et al.* (1995) demonstrated the effect of snowing and thawing and found that chilling reduced the viability of teliospores. The chances of the disease occurring where there is snow are therefore much reduced. Such extreme conditions are in close proximity to the KB endemic areas, and have remained free from the disease.

TELIOSPORE NATURE

Teliospores are globose to subglobose in shape, 22-49 μm in size, reticulate with projections, and are surrounded by a thick covering sheath (2-4 μm). Morphological studies show that there are three distinct layers: the perisporium or sheath, a fragile, fractured structure, easily identifiable under the electron microscope; the epispore, which is reticulated and has numerous curved projections; and the endosporium (Khanna *et al.*, 1966). The mature teliospore is a diploid (2n) and at germination the nucleus divides meiotically and then mitotically to produce haploid primary sporidia. According to Fuentes & Duran (1986) meiosis occurs at germination followed by a series of mitosis in teliospores and promycelia or both, producing numerous haploid nuclei in the promycelia. In these studies the mean F-DNA content of filiform primary sporidia was 0.115 arbitrary units (a.u.). The mean F-DNA content of the binucleate teliospore initials was 0.217 a.u., that of post fusion nuclei 0.473 a.u. Apparently DNA replication in the teliospores occurred initially before nuclear fusion.

TELIOSPORE GERMINATION

The teliospore enables the pathogen to survive during the hot dry summer months of May and June when the maximum temperature exceeds 45°C. Fresh teliospores have dormancy and this can be broken by exposing them to 40-43°C under direct sunlight for 18 days or more (Krishna & Singh, 1982b; 1983). Soaking teliospores in peptone or wheat straw extract, benzaldehyde, furfuraldehyde or butyric acid are all reported to influence dormancy and the germinability of teliospores. Even after this, only 50% of the teliospores germinate in plain water, compared with just 2% germination of fresh teliospores kept in darkness at 20°C (Aujla *et al.*, 1986). If teliospores are buried deep in top soil they retain germinability for two years. By keeping fresh teliospores at 15-20°C for 10-15 days and by subjecting the spores to various treatments germination can be enhanced. Thus teliospore dormancy gets broken, permitting a free and better germination (Smilanick *et al.*, 1985a, b). The stout promycelium measures 10-190 μm long and 6-13 mm broad and produces a cluster of 60-185 primary sporidia at the tip. These sensitive, short lived sporidia germinate in free water and produce a thick mycelial mat. Subsequently, from a cushion-like structure, crescent-shaped secondary sporidia or allantoid spores are produced. The secondary sporidia occasionally exhibit yeast-like tendencies to bud and produce another crop of allantoid spores on a wet leaf surface. Depending on temperature and availability of free water, the pathogen follows different pathways to produce crops of spores (Dhaliwal, 1989; Dhaliwal & Singh, 1989; Smilanick *et al.*, 1989).

MICROSPORIDIA

The primary sporidia or the macro (filiform) conidia are splash dispersed and in turn produce a large quantity of

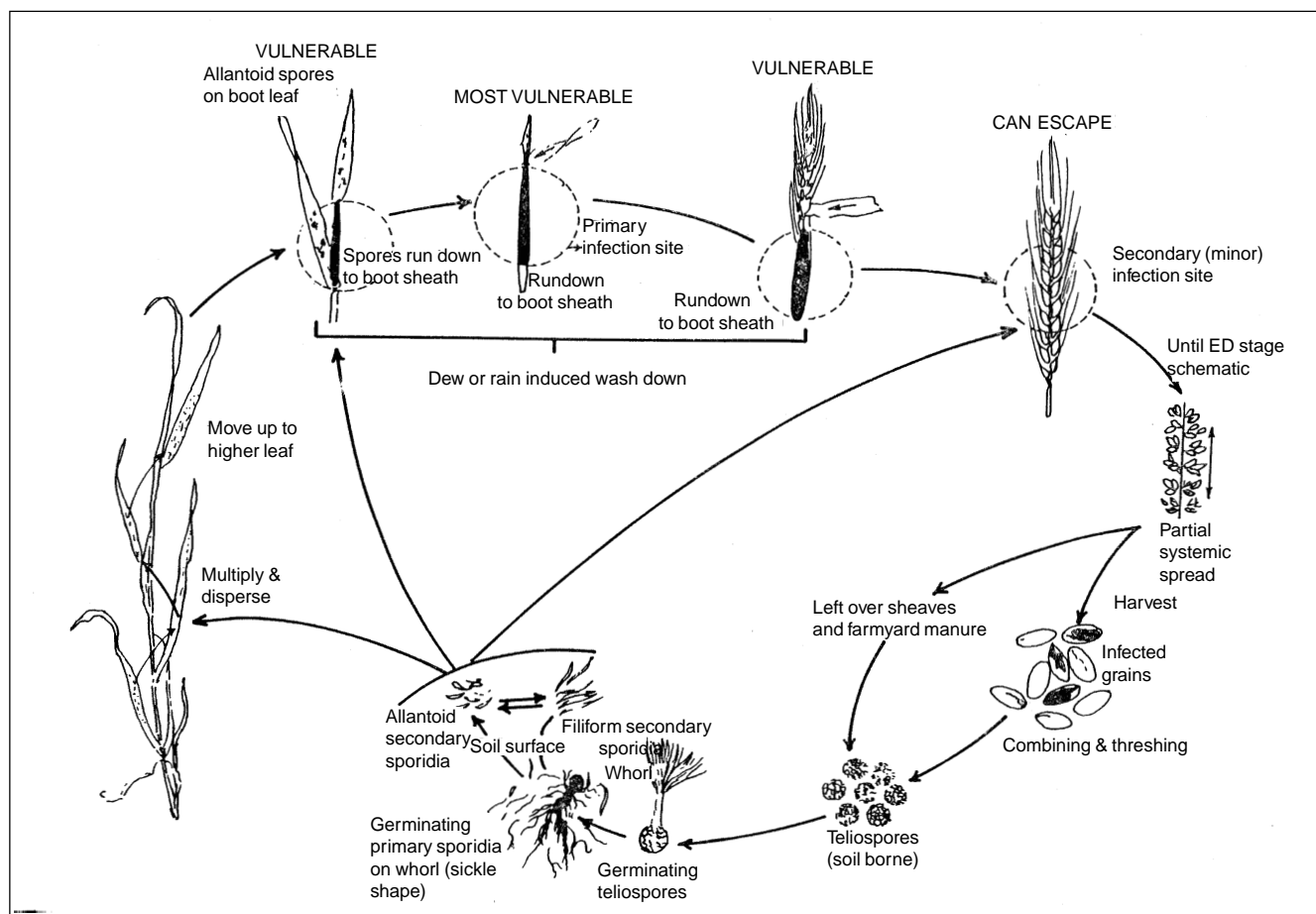


Figure 1. Pathogenicity cycle of *Tilletia indica*.

secondary or micro or allantoid spores. The secondary sporidia are released forcibly and are produced in enormous quantities when the leaf wetness tends to dry. These spores are the only form that infect the wheat earhead (Dhaliwal & Singh, 1988; 1989). In *T. indica* heterothallism and bipolar incompatibility have been recorded (Duran & Cromaty, 1977; Krishna & Singh, 1983). The heterothallism demands fusion between secondary sporidia that are compatible. The rapid vegetative multiplication of the allantoid spores and the heavy sporulation increases the probability of successful fusion of opposite mating types.

DISPERSAL FROM SOIL LEVEL TO EARHEAD

The secondary sporidia or the crescent-shaped allantoid spores are a product of the primary sporidia produced by

Table 1. Impact of detaching the flag leaf on severity of Karnal bunt (Unpublished data, Nagarajan *et al.*)

S. No.	Variety	Percentage Severity*		% reduction due to clipping
		I	II	
1.	HD2009 (Arjun)	26.0	5.0	80.76
2.	WL 711	28.8	6.81	76.70
3.	Sonalika	17.85	5.37	64.31
4.	HD 2329	13.04	2.17	83.35
5.	HD 2285	14.43	3.38	69.64
6.	WH 147	17.94	8.33	53.56
	Mean	19.67	5.17	71.38

* Mean of 3 replications I = Unclipped flag leaf II = Clipped flag leaf

the teliospores present in soil. The secondary sporidia get deposited on the lowermost leaves by wind and splash (Prescott, 1986). The maximum trapping of the secondary sporidia occurs in air samples during early morning hours when 100% RH prevails. The secondary sporidia in the presence of leaf wetness produces a secondary crop of spores and as the leaf surface dries, these spores get dispersed to higher leaves. Having climbed up the leaves through monkey jumps, they reach the flag leaf, some also get wind dispersed and if the earhead emergence stage coincides with a mild drizzle or rain, the secondary sporidia get washed down into the sheath (Nagarajan, 1991). With free water, once again crops of microsporidia are produced, and here the requirements of heterothallism also get fulfilled. Before complete ear emergence, if more favourable weather occurs causing run down, then more severe KB develops. Occasionally microconidia get lodged on the floral parts at anthesis and, if conditions are favourable, they infect to produce a KB sorus on individual grains (Bedi *et al.*, 1949). (Figure 1).

An experiment was conducted at Dhaulakuan, in the foothills of Himachal Pradesh, a hot spot for KB under natural conditions during the epidemic year 1995-96. Clipping the flag leaf at the auricle before earhead emergence, reduced the severity of KB infection by 80% in the most susceptible variety HD 2009 (Arjun) (Table 1).

This confirms that the flag leaf acts as the receptor of the secondary sporidia and suitable modification of it will result in a reduction of KB severity. In the same trial at Dhaulakuan, eighteen earheads of three varieties were covered with their flag leaf by a thin paper bag at crop growth stage 37. From growth stage 43 onwards three earheads were opened up until harvest to identify the physiological stages during which *T. indica* causes infection. From Figure 2, it may be seen that growth stages 49-52 are the most vulnerable.

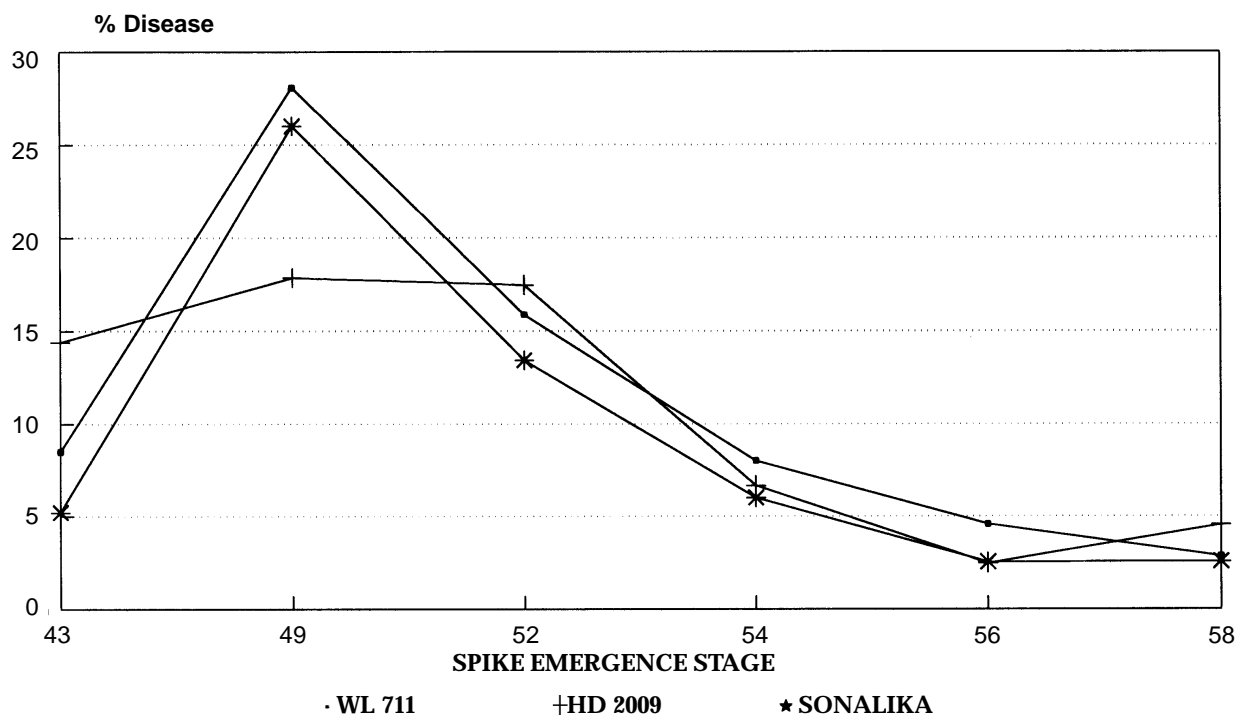


Figure 2. Effect of spike emergence on growth stage and severity of KB.

PROLIFERATION INSIDE THE EARHEAD/OVARY

There is a great likelihood of the germinating secondary sporidia directly invading the ovary and causing infection. The mycelium makes its way to the ovary after passing through the spaces of lemma and palea (Dhaliwal & Singh, 1989). The mycelium spreads systemically from one spikelet to another through the rachis and from one floret to another through the rachilla. Such a movement results in KB infected grain, often from adjacent florets and oppositely located spikelet (Bedi & Dhiman, 1984). In an earhead 3-4 such sites can establish, depending upon the site of infection.

On invasion of the ovary either through single or multiple site entry, the mycelium proliferates and switches from a vegetative to a sporophytic stage. The mycelium/hyphae proliferate in the space formed by the disintegration of the middle lamella of the parenchymatous cells of the pericarp. On short sporophores, the hyaline mycelial cushion produces the dark coloured teliospores, characteristic of KB.

LEVELS OF HOST - PATHOGEN INTERACTION

Host resistance operates by: (a) restricting primary infection; (b) restricting systemic movement between spikelets/florets; (c) arresting mycelial proliferation inside the karyopsis; and (d) arresting the switch from mycelial to sporophytic stages. Since measuring each component in breeding for KB resistance is very difficult, an integrated selection index is followed taking: (1) the number of KB infected grains/unit sample; and (2) the average coefficient of infection value derived by sorting the KB infected grains into various grades based on sorus size (Aujla *et al.*, 1989a).

EVALUATING HOST RESISTANCE

After several years of research and after evaluating different procedures, it is concluded that inoculation into the sheath of the boot leaf at the pre-earhead emergence stage is the most reliable method of initiating KB. (Table 2).

Inoculum density of 50,000 secondary sporidia/ml, suspended in water, is the inoculum potential needed to create reliable artificial epidemics (Warham & Cashion, 1984). Success is further enhanced when humidity is high and moisture is adequate during the post inoculation period. In the field using a spray system such conditions can be created. It was further noted that inoculations carried out between the second half of January to March, preferably in the evening at 6 pm, followed by misting, result in 100% of the inoculated heads producing KB and 60% of the grains becoming infected (Gill *et al.*, 1993).

PHYSIOLOGICAL SPECIALIZATION

Variability is a pre-requisite for better survival and co-evolution of organisms. Mitra (1935) reported two collections of *T. indica* to differ in teliospore size and this was subsequently shown to be due to environmental variation (Mundkur, 1943). Spore size then cannot be taken as a criterion for differentiating physiological races of *T. indica*.

Based on the reaction of various *T. indica* isolates on a set of 17 differentials, four pathotypes (K1, K2, K3 and K4) have been reported (Aujla *et al.*, 1987). Since in each annual cycle of the pathogen, nuclear fusion takes place between heterothallic secondary sporidia, in *T. indica* the race/

Table 2. Percentage severity of KB induced by inoculating at different growth stages. (Gill *et al.*, 1993)

Growth Stage	Percentage infection of KB*
Boot stage	6.97
Earhead just peeping out at the tip or from centre	22.16
Earhead half outside boot leaf	6.93
Earhead completely out from boot leaf	3.33
Anthesis	1.74
Post-anthesis	0.00
Slight grain formation	0.00
Standard method of syringe inoculation	58.63

*on the basis of 20 earheads

Table 3. Promising Karnal bunt resistant strains (artificially inoculated for up to 10 years)

Bread Wheat strains	Parentage
W 485	WL923/HD2160/UP368
W1786*	POLK/UP301
WL6975*	WL1535/UP291/HD2116/HD2177
HD29	HD2160-HD1977/HD7449-HD1944/HD2136
HD30	HD2160-HD1977/HD7949-HD1944/HD2136
HP1531	HD1275/C306
ISWRN191	WGA434
ISD227-5	498 ´ RAJ 911

*Also resistant to loose smut

pathotype approach is not valid. As the basis of classification of the isolates is on the KB sorus produced by *T. indica* and not on the basis of the host pathogen interaction (production of necrotic, chlorotic or mesothetic reaction), it is appropriate to classify the variation in isolates as aggressiveness. Following this argument and using a set of overlapping differentials proposed by Aujla *et al.*, (1987), five aggressive (KB Ag) isolates have been identified (Singh *et al.*, 1995) and there is a likelihood for the presence of many more. Furthermore, isoenzyme analysis for single spore cultures from Indian and Mexican collections for 48 enzymes has provided fairly good evidence in favour of the existence of genetic variability. Isoenzyme banding patterns obtained with 40 cultures were interpreted as representing 31 presumed loci, of which 16 were polymorphic (genetic variation present) and 15 were monomorphic having no variation (Bonde *et al.*, 1985; Bonde, 1986; Bonde & Peterson, 1990).

Polyacrylamide gel electrophoresis (PAGE) employed on mycelial proteins of isolates collected from various geographical locations can demonstrate the presence of three varying banding patterns in a population of *T. indica* prevalent in north west India (Anonymous, 1996). Resolving genetic differences among populations by using random amplified polymorphic DNA (RAPD) and DNA markers may become suitable approaches for variability analysis in *T. indica*. These molecular tools have already been employed successfully to elucidate genetic relationships among different species of *Tilletia*, except *T. indica* (Shi *et al.*, 1996).

IPM APPROACHES

Biological mulches like chickpea, intercropped with wheat reduced KB incidence by 60% and covering the inter-row space with transparent polythene sheets from the tillering stage onwards, reduced KB incidence by 75% (Singh *et al.*, 1991). The plastic mulch treatment increases soil temperature and also restricts the liberation of primary sporidia from soil and hence reduces disease severity.

Soil inhabiting *Trichoderma* spp., such as *T. viride*, when applied to soil or earheads, reduced the incidence of KB infected grains from 1.63% in the control to 0.8% and 0.65%, respectively, under natural conditions. Preliminary studies showed that there is a potential for biocontrol of KB under an overall IPM strategy (Singh *et al.*, 1991).

TEMPORAL HURDLE

In north west India, wheat is sown between the end of October and the first week of January. Since a variety of crop sequences are followed, fields get vacated at different times. The differing seeding time, staggers the earhead emergence stage and thus creates a temporal hurdle for *T. indica*. Coincidence of intermittent rainy days and ideal temperature conditions do not prevail over such a long heading period. At best, isolated fields with severe KB infection appear. The currently popular wheat varieties possess a high degree of field

tolerance to KB. This multi pronged management strategy has reduced the extent of KB damage, which otherwise was causing severe crop damage.

HOST RESISTANCE TO KB

A large collection of wheat germplasm were field inoculated and were rated for resistance based on the number of spikes infected and the type of sorus produced. Sorus size was broadly grouped into four grades and a numerical rating of 0.25, 0.5, 0.75 and 1.00 was assigned to each grade (Aujla *et al.*, 1989a). By multiplying the percentage of grain showing KB infection with the numerical rating, the partial coefficient of infection (CI) value was obtained. Genotypes possessing a CI value of >10 were considered susceptible and were rejected and such entries were discouraged for inclusion in varietal improvement activities. As a result of this rigorous evaluation done at several locations over several years, a number of bread wheats could be released/identified as resistant to KB. (Table 3).

Over north west India, apart from KB, brown rust (*Puccinia recondita* f.sp. *tritici*) and stripe rust (*P. striiformis* f.sp. *tritici*) are also important. Therefore, to have multiple disease resistance, segregating materials are being alternatively tested in the Karnal bunt screening nursery (KBSN) and rust screening nursery (RSN). The Karnal bunt resistant plants selected from the KBSN (Step 1) are screened during the succeeding season for stripe and leaf rust in the RSN (Step II). The rust resistant materials in RSN are again screened for Karnal bunt (Step III) resistance once. In this process, several bread wheat lines possessing high yield, acceptable agronomic requirements and multiple disease resistance have been identified and released for cultivation.

Genetic analysis shows that HD 2329, when used as the recurrent parent in a back cross programme, gives more KB resistant progenies. Monosomic analysis using three resistant lines HD 29, WL 6975 and WL 2348 and the susceptible WL 711 shows that the gene governing resistance to KB is located on chromosomes 1D, 2D, 3B, 3D, 5B and 7A. The backcross involving some alien sources and cultivar Kalyansona (recurrent parent) yielded derivatives which show resistance to the three rusts and KB (Sawhney & Sharma, 1996).

Triticum araraticum (wild form of *Triticum timopheevi*, 2n=4x=28 AAGG) has been considered a valuable source of resistance to a number of wheat pathogens including *Tilletia* spp. (Bijral & Sharma, 1995). Studies on the genetics of resistance to KB in eight donor lines indicate that dominant alleles are involved in according low infection scores (Nanda *et al.*, 1995). Also partial dominance of resistance has been observed in a cross involving four resistant synthetic hexaploid wheats (Villareal *et al.*, 1995). Epistatic variance was observed as an integral part of inheritance of KB resistance (Sharma *et al.*, 1995). Resistance to KB has been found to be due to two genes in cultivars Luan, Attila, Vee7/Bow, Star, Weaver, Milan, Sasia and Taracio/Chil., and monogenic in Cettia, Irena, Turacio, Opata, Picus and Yaco. Digenic genotypes had a higher level of resistance to KB than those lines with a single gene (Singh *et al.*, 1995).

VARIETAL RESISTANCE

During the 1970s many of the widely cultivated semidwarf wheat varieties such as HD 2009, WL 711, UP 262 etc. were highly susceptible to KB as they were not selected for resistance to the disease which was then unimportant. With KB becoming endemic over north west India, varietal screening was intensified and soon tolerant genotypes WL 1562, PBW 154 and HD 2281 were released (Gill & Aujla, 1986). Also durum wheats PBW 34, DWL 5023, PDW 215 and PDW 233 were released since durums in general are resistant to KB. This management strategy executed from the mid 1980s onwards enabled the national programme to contain the KB severity over north west India (Gill *et al.*, 1993).

PREDICTING KB SEVERITY

Reinvestigating the life cycle of *T. indica*, to develop an efficient prediction system, indicated that the allantoid spores produced by the pathogen multiply on the wheat leaf surface and are deposited from the bottom leaf to boot leaf. Between crop stages of the decimal scale (Zadoks *et al.*, 1974), if spells of drizzle occur, the secondary sporidia get washed into the leaf sheath. It is here that these spores germinate, get diploidised and infect the glumes of the emerging earhead (Figure 1). Frequent run-down into the sheaths promotes the establishment of multiple infection sites and severe disease development (Nagarajan, 1991). The following linear prediction model developed for KB using mean weekly weather parameters had an R² value of 0.89 indicating a high degree of fitness for the situations of north west India:

$$Y = 0.4381 + 2.97a - 2.77b - 0.09c + 0.13d$$

Where Y is the predicted KB severity on bread wheat and the variables a to d are rainy days during 15-21 February, rainy days during 22-28 February, amount of rainfall during 15-21 February and the amount of rain during 22-28 February, respectively.

When a similar exercise was done for Mexico where KB occurs in Sinaloa and Sonora states, the R² value was 0.91 and the model, when validated, was found to give a reliable prediction:

$$Y = -0.71 + 1.67a + 0.95b + 1.07c + 0.20d$$

Where a to d represent the number of rainy days during 1-7 February, 8-14 February, 22-28 February and amount of rainfall during 21-28 February, respectively. Though there are minor differences between the models for the partial coefficient values, the common denominator is the relationship between KB and the number of rainy days during a particular period of February when the earhead emerges.

SEED CERTIFICATION

For limiting the entry of the pathogen to disease free areas within India through either infected or contaminated seeds, stringent seed health standards have been established. The seed certification programme lays down a maximum of 0.05 and 0.25% level of KB infection for foundation and certified seeds, respectively. The seed washing test is the most efficient method for quantifying the externally seedborne teliospores. If teliospore contamination is above 25 spores/grain, the seed lot is rated as contaminated. (Agarwal *et al.*, 1973; Agarwal & Verma, 1983)

QUARANTINE

Since occurrence of KB is limited to certain states of Asia and north America only, there is a general quarantine imposed against KB in most countries. In Mexico there is a domestic quarantine restricting the movement of wheat planting material from one zone to another. Since KB quarantine gets in the way of free exchange of germplasm from the International Maize and Wheat Research Center (CIMMYT), salvage techniques have been developed to clean contaminated seed before shipment (Zhang *et al.*, 1984; Prescott, 1984). With the globalization of trade, there is a need to rationalise quarantine restrictions which hamper the free market. The FAO and other global organisations are of the view that quarantine should be based on risk analysis.

CHEMICAL CONTROL

SEED DRESSING

A number of chemicals have been found to restrict seed contamination, but have shown only limited success. None of the fungicides could completely eliminate all the seedborne spores on the surface or under the pericarp (Aujla *et al.*, 1989b). Another implication of seed treatment lies in the adverse effects on seed germination. Treatment with formaldehyde, ethanol, hot water (54°C), common bleach, chlorine dioxide, quaternary ammonium solution, fumigation with methyl bromide, sulfur dioxide and chloropicrin interfere with seed germination and hence are not all that effective (Smilanick *et al.*, 1988).

SPRAYING

Among several fungicides evaluated for spray application, Tilt (propiconazole) at the heading stage gave 71.4-100% disease control (Aujla *et al.*, 1989b), and resulted in greater yield (Rattan, 1988). No detectable residues of propiconazole were found in wheat grain or straw (Singh *et al.*, 1991) and may be regarded as a possible chemical for the control of KB. Systemic fungicides like Tilt, Folicur (tebuconazole) or SAN 619 have potential for commercial exploitation at times of dire need (Nagarajan, 1991).

CONCLUSIONS

Karnal bunt of wheat, first detected in 1931 in India, is caused by a fungus, *T. indica* Mitra (Mund.). The pathogen infects the ovary and converts the grain into a sorus of dark coloured teliospores. Economic losses occur in terms of quality, germination loss and trade due to the fishy odour the grain emits and for reasons of quarantine concern. KB occurs in several parts of south Asia, Syria, Afghanistan, Mexico and the USA. The teliospores of *T. indica* get released and dispersed by falling on the soil and contaminate the seed during threshing. After dormancy, soilborne teliospores germinate to produce the primary sporidia which in turn produce several crops of secondary sporidia under ideal conditions. These infective propagules become wind or (rain) splash-borne, get deposited on the boot leaf from where they get drained into the leaf sheath to infect the emerging spike. The pathogen is heterothallic and inside the leaf sheath compatible secondary sporidia fuse prior to infection. On infecting the spike, the mycelium moves systemically within the rachis infecting several florets on the way. So in an infected head two or three patches or aggregates of KB infected kernels can occur under ideal conditions.

The diseased seeds emit a rotting fish smell due to the production of trimethylamine. Biological mulching, such as intercropping wheat with chickpea, covering the inter row space with transparent polythene and application of *Trichoderma* spp. as biocontrol agents, reduce the disease severity. Cultivation of KB resistant varieties forms the major element in the IPM strategy. Forecasting the occurrence of KB can be done based on the amount of rain received during February or at the earhead emergence stage. The extent of KB also depends on the number of spells of rain received at this stage. The disease can be effectively checked by spraying systemic fungicides like Tilt (500 ml/ha), Folicur (500 ml/ha) or SAN 619 (500 ml/ha) at decimal growth stages 50-56 (Zadoks scale) of crop growth. Since KB of wheat is limited to Asia and north America, there is a general quarantine embargo imposed by most of the countries. With globalization of trade, there is a need to rationalize quarantine restrictions as KB is being increasingly used as an instrument of a non-tariff barrier to dampen the spirit of free grain trade.

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