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Review Article

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Eco-friendly Management of Karnal Bunt (*Neovossia indica*) of Wheat

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ABSTRACT

Karnal bunt incited by Neovossia indica is one of the most important disease of wheat crop. To develop an eco-friendly management practice against Karnal bunt of wheat, integration of fungicidal seed treatment with foliar sprays of phytoextracts, bio-control agent and fungicide revealed. Uses of Thiram 75DS or Kavach 75WP @2g/Kg, Dithane M-45 or Captan 50WP@2.5g/Kg, Vitavax 75WP@2.5g/Kg, Tilt 25EC or Raxil 2DS@1mL/Kg or Pseudomonas fluorescens@5 mL/Kg or Trichoderma viride (Ecoderma) or T. harzianum@5 mL/Kg seed treatment for eliminating primary inoculum (teliospores). 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 also. Application foliar spray of Baycor 25WP or Bavistin 50WP or F-100 or Moximate 72WP@2.5g/Kg, Tilt 25EC or Folicur 25EC or Contaf 25EC@1mL/Kg at boot leaf stage and 50% emergence flowering heads against the secondary air-borne inoculum (Allantoides sporidia). This is concerning integration of fungicide seed treatment with foliar spray of bio-control agent and phyto-extract. It is cheaper and eco-friendly practice for the control of Karnal bunt of wheat.

Key words: *Karnal bunt, Neovossia indica, Triticum aestivum, Phytoextracts and Bio-control agents*

INTRODUCTION

Wheat (*Triticum aestivum* L.) is the most popular grain of the world and is the staple food of millions of people. The geographical concentration of wheat is found between 30-55ºN latitude in the Northern hemisphere and between 20-40ºS in Southern hemisphere[3]. The Karnal bunt, also referred to as 'Partial bunt' caused by *Neovossia indica* (Mitra) Mundkur was reported first by Mitra in 1931 on wheat (*Triticum aestivum* L.) from an experimental seed material grown at Botanical Station, Karnal (Haryana) in India and hence the name Karnal bunt. Approximately 70 countries place quarantine restriction on movement of wheat from countries where Karnal bunt is known to occur[51]. Its widespread prevalence and effects on the quality and viability of seed has caused concern to agroscientists. Karnal bunt causes heavy losses in wheat production mainly in Northern and Central India.

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All efforts to control the disease through cultural practices and chemical treatment have been futile. The only alternative and long term control measures to avert this disease. Some antagonistic agents such as, *Trichoderma viride, T. harzianum, Bacillus subtilis* and *Pseudomonas fluorescens* have been found effective against *Neovossia indica*.

The world wheat market is enormous. Wheat is dominant grain of the world commerce and is the staple food of millions of people. It is also an important part of the daily diet of many millions more. Annual global wheat consumption is in excess of 550 million tonnes. Approximately two-thirds of the wheat produced in the world is used for human food and about one-sixth is used for livestock feed. Industrial uses, seed requirements, and postharvest losses account for the remaining withdrawal from the world wheat granaries[3].

2. The Disease

Karnal bunt is visible on wheat grains, which are partially or completely converted into black powdery masses enclosed by the pericarp[55,58].

In the spikelets with bunted grains, the glumes open apart exposing the bunted grains, which later fall off on the ground with a little jerk. Freshly collected infected grains emit a foul smell, like rotten fish, due to production of trimethylamine (C_3H_9N) by the fungus[56].

2. Host range

Karnal bunt affects hosts which include *Triticum aestivum* (wheat), *T. durum* (Durum wheat), rye (*Secale cereale*) *T. boeticum, T. ovatum, T. variabilis* and *T. shareonensis* and under artificial inoculations even *Aegilops* spp. and triticale (*Triticum aestivum* x *Secale cereale*). It has been shown to infect other grass species under glasshouse conditions but has never been detected in the field in these alternative hosts[34].

3. Distribution

Karnal bunt also called as partial bunt was first detected by Mitra in1930 within the experimental seed material at the Botanical Research Station, Karnal (Haryana), hence named as Karnal bunt and was reported by him in 1931. Since then, the disease has spread to north western parts of Madhya Pradesh, Rajasthan and Junagarh district of Gujarat[31]. It is widespread in northwest India and in adjacent areas of Pakistan and Afghanistan[82]. It has also been detected in Mexico, the South-western United States of America and South Africa[19]. In 1996, the disease was detected in Arizona in certified durum wheat seed. The USDA also detected Karnal bunt in grain shipments from Lebanon and Syria[79]. Sharma *et al.*[72], reported that 68.9 per cent wheat seed samples from Himachal Pradesh were infected with Karnal bunt (*Tilletia indica*); however, disease incidence was highest in Sirmour district (76.8%).

4. Disease Symptoms

Due to seed borne nature, the symptoms of the Karnal bunt disease become evident only after threshing. However, Duran and Cromarty[25] detected the infection of grains in an ear head by their swollen appearance and slightly wider opening of the glumes (Plate -1). The glumes open apart exposing the bunted grains which later fall off on the ground with a little jerk in severely infected spikelets.

There is reduction in length of the spike and number of spikelets in the infected plant[57]. All the ears are not affected and in an ear head all the spikelets are not bunted[56]. 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[55,59]. 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[36].

Plate 1. Typical symptoms of Karnal bunt of wheat

Plate 2. Karnal bunt infected grains of wheat

Histopathology

Cashion and Luttrell[14] have demonstrated that the pathogen does not invade the embryo and the mycelial growth is limited to the pericarp. Transmission electron microscope (TEM) study 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[69]. Mycelial growth ruptures the connection between the pericarp tissue surrounding the vascular bundle in the bottom of adaxil 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.

6. Incidence and Losses

Normally, the disease appears sporadically in isolated areas as a minor disease causing insignificant loss in yield, but in certain years it assumes epiphytotic proportion and causes substantial losses in yield[35,62,2]. Loss in yield

up to 20 per cent was reported by Mitra[55]. During 1969-70, 7.5 per cent infected grains were recorded in cultivar S-331 and 10.03 per cent in Kalyansona[1,62]. Bunted grains upto 50% were recorded in the Tarai of Nainital during 1974-75[2]. Karnal bunt infection ranged between 0.27-51.57 per cent and resulted in significant reduction in weight of infected grains[67]. Singh *et al.*[76], observed 15-23% infection of Karnal bunt in cultivar HD 2009. The loss in yield caused by the disease was calculated as 1/3 X yield X per cent infection, as the disease covers one third area under wheat cultivation which comes approximately to 40,000 metric tonnes of grain in India[62]. Extent of loss in weight of wheat grain lots due to Karnal bunt infection ranged between 0.27 and 51.57 per cent depending upon the grade and per cent or roughly one fourth reductions in weight of cent per cent infected grain lots[67].

The total damage in terms of quantitative losses due to Karnal bunt ranged between 0.2 to 0.5 per cent of total value of wheat crop in India during epiphytotic years[62,37]. Singh[75] estimated that 1.0 per cent of total value of wheat crop was lost due to Karnal bunt during epidemic year of 1987 in Uttar Pradesh. However, this did not include the value of the losses to seed growers due to non-certification of their infected seeds.

7. Economic Importance

The nutrient composition in bunted wheat grains differs from that of healthy grains. Bhat *et al.*[11], found the total ash and phosphorus content of diseased grains increases with increased levels of infection while the thiamin and lysine content decreases. Bedi and Meeta[9] found that diseased grains have a greater percentage of crude fiber, crude protein and free amino acids, and a lower percentage of soluble sugars and starch as compared to healthy grains. Sekhon *et al.*[71], however, found no appreciable effect on protein content of diseased grains, although arginine and aspartic acid levels tend to increase in diseased grains.

The infected grains are somewhat swollen and lighter in weight and get blown off during threshing. Rai and Singh[67] estimated loss in grain yield as actual yield X per cent infected grains X 0.256/100. This indicates a low impact of Karnal bunt on grain yield.

8. Karnal bunt reduces seed germination

Mildly infected grains do not suffer any significant loss in germination, but severely infected grains show considerable reduction in viability of seeds and produce weak seedlings[68]. Mildly infected seeds germinated freely up to 89 per cent[56] but abnormal germination producing poor seedlings have been recorded[61].

9. Karnal bunt is an important issue in wheat trade

The disease affects both the quality and quantity of wheat grains, seriously reducing its market value[62]. Chapaties made out of wheat lots with 3.0 per cent or more infected grains have fishy odour and perceptible discoloration and products made out of 5 per cent Karnal bunt infection are not acceptable for human consumption[54]. Wheat importing countries have also imposed strict quarantine measures and insisted on a zero tolerance limit shipment of wheat from Karnal bunt prone regions[12]. Exporting countries are, thus, required to meet the challenge posed by Karnal bunt. USA is concerned about the possibility of export losses due to recent introduction of Karnal bunt in Arizona, Texas and New Mexico[83].

10. Toxicology

Various short-term toxicological studies have been carried out with bunted wheat grains using rats, chicks, monkeys and goats. Feeding albino rats with Karnal bunt infected grains lead to liver and renal insufficiency[11]. Feeding bunted grains to goats decreased the bacterial and protozoan population in the rumen and depressed the formation of ammonia and volatile fatty acids[73].

11. The pathogen

Karnal Bunt fungus was originally classified as *T. indica*[55], in that it had no greasy spore mass, no smell of decaying fish and had much larger spores. However, it was later renamed by Mundkur[58] into *Neovossia indica* because of its unbranched rather long promycelium with a 33-123 or more whorl of non-fusing conidia (Buller's primary sterigmata) at the

apex. However, others now consider *Tilletia* to be the appropriate genus[28,26].

12. Classification

The fungus belongs to:
Kingdom : Fungi
Division : Eumycota
Subdivision: Basidiomycotina
Class: Teliomycetes
Order: Ustilaginales
Family: Tilletiaceae

13. Taxonomic position

Tilletia indica, the causal agent of Karnal bunt as described by Mitra[55] was transferred to the genus *Neovossia* by Mundkur[59] since the pathogen produces numerous independent sporidia. Based on detailed taxonomic study Munjal[63] and Krishna and Singh[41] justified the placement of *N. indica* under *Neovossia.* However, as is characteristics of *Tilletia*, sterile cells are found intermixed with teliospores of *T. indica and* trimethylamine is produced and therefore, it was again transferred to *T. indica* Mitra by Fischer. However, western science literature prefers to designate the Karnal bunt causal agent as *Tilletia indica*[26,27]. The *N. indica* is pathogenic to *T. aestivum, T. durum, T. boeticum, T. ovatum, T. variabilis* and *T. shareonensis*, triticale and under artificial inoculations even *Aegilops* spp. are susceptible[23,70,81]. The important differences between the two genera are given below.

***Tilletia* Tul.**	***Neovossia* Korn.**
1. Produce systemic infection.	Localized host infection.
2. Production of few to many primary sporidia which often fuse and produce secondary sporidia of one or two types.	Production of large number of primary sporidia which do not fuse.
3. Produce sterile cells.	The absence of sterile cells.
4. Production of trimethylamine.	No production of trimethylamine.

The T. indica genome was sequenced employing hybrid approach of PacBio Single Molecule Real Time (SMRT) and Illumina HiSEQ 2000 sequencing platforms. The genome was assembled into 10,957 contigs (N50 contig length 3 kb) with total size of 26.7 Mb and GC content of 53.99%. The number of predicted putative genes were 11,535, which were annotated with Gene Ontology databases. Functional annotation of Karnal bunt pathogen genome and classification of identified effectors into protein families revealed interesting functions related to pathogenesis. Search for effectors' genes using pathogen host interaction database identified 135 genes[46,45].

14. Teliospore nature

Mature teliospores are brown to dark brown, spherical or sub spherical to oval, 22-49 µm (average 35 µm) in diameter (exceptionally upto 55 µm), intermingled with them are numerous large yellowish or subhyaline sterile cells[55]. These are rounded or angular and smaller in size than the teliopores, with comparatively thinner walls[55]. Teliospore with curved tips, 1.5-5.0 µm high, narrow ridges (finely cerebriform) spines are covered by a thin hyaline membrane[18,13]. Electron microscope studies have revealed that the teliospore has three walls or layers of the endosporium, episporium and perisporium[38]. The endosporium is thick and lamellate, while the episporium is adorned with thick truncate projections[39].

Roberson and Luttrell[69] studied ultra structure of teliospore ontogeny and found that teliospores of *T. indica* arose directly from a thin hymenial layer of hyphae and covered the surface of the cavities formed by the separation of the inner and outer layers of the pericarp and by the separation of the inner pericarp from the seed coat.

15. Teliospore Dormancy

There are three types of teliospores dormancy in *T. indica*. The first is postharvest dormancy. Teliospores taken from freshly harvested grain commonly germinate poorly as compared with germination after bunted grains are stored for several months to a year or longer[6]. The second type of dormancy, which is long-term and likely, contributes to teliospore survival under field conditions. The third type of dormancy is induced by cold temperature. In a study of *T. indica*, dry teliospores kept at -18ºC progressively lost the ability to germinate over a period of 12 weeks of treatment[84].

16. Teliospore germination

Freshly collected teliospores are incapable of germination and are considered to have a period of dormancy[53,55,6]. Five to nine month old spores also showed poor germination[53]. One or two year old teliospores show the highest germination[52,40,6]. Smilanick *et al.*[78], found the highest germination (55-60%) of teliospores after 3 weeks of incubation at 15-20ºC in continuous light at pH 6.0-9.5.

Singh *et al.*[77], found that dilute solutions of aldehydes and fatty acids are better soaking media than water for germination of teliospores. The organic acids and phenolic compounds (citric acid, acetic acid, oxalic acid and formic acid) inhibited germination and adversely affected the formation of sporidia[75]. Bansal *et al.*[6], found that liquid nitrogen treatment for 15 minutes increased teliospore germination on 1.5% water agar.

On normal germination, each teliospore gives rise to a stout promycelium, bearing 32-128 or more sickle shaped primary sporidia at its tip[59]. Occasionally, the promycelium is unusually long so that the cluster of primary sporidia appears to arise from the teliospore itself[33]. Sometime the promycelium can be branched[56,43]. When a teliospore germinates, the single diploid nucleus undergoes meiosis and subsequently rapid mitosis to produce a large number of haploid nuclei. These nuclei migrate from the hypobasidium into the promycelium and then to sporidia formed at the promycelial tip but sometimes mitosis produce septate sporidia with two or four nuclei[29].

17. Macro and Microsporidia

Primary sporidia (Macrosporidia) have mean length and width ranging from 64.4 to 78.8 µm and 1.6 to 1.8 µm, respectively and the secondary sporidia (Microsporidia) has mean length and width ranging from 11.9 to 13.0 µm and 2.0 µm respectively[65]. The primary sporidia are sickle shaped and are attached on the tip of the promycelium[59]. The allantoid spores are released forcibly and are produced in enormous amounts when the leaf wetness tends to dry (Plate -3 and 4). These spores are the only type that infects the wheat head[20,21].

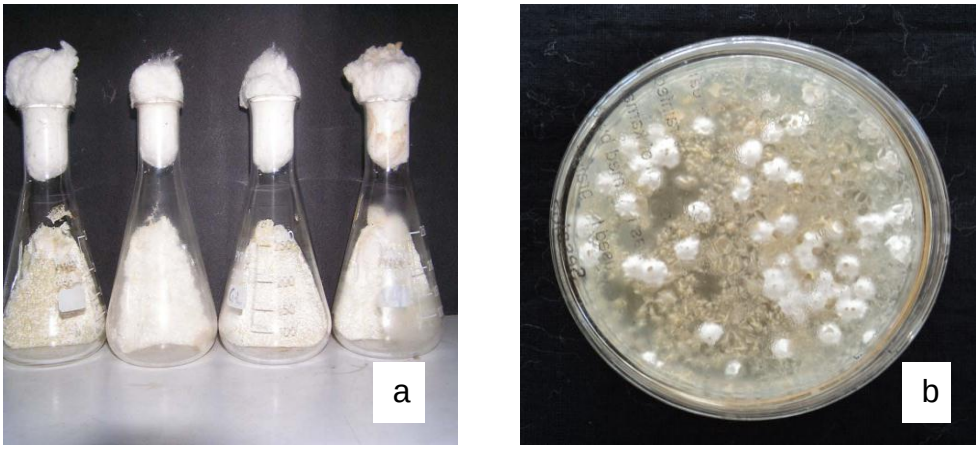

Plate 3. Culture of *N. indica* on PDA (a and b)

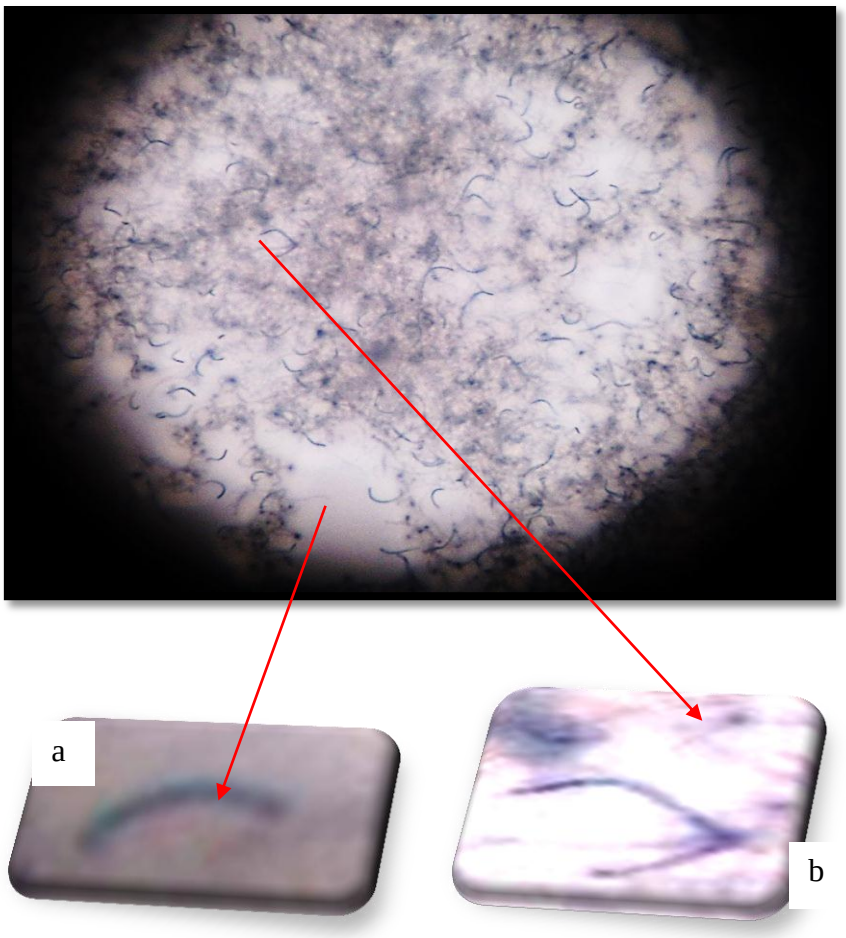

Plate 4. Allantoid (a) and filiform (b) secondary sporidia of *N. indica*

In *N. indica* heterothallism and bipolar incompatibility has been recorded[25,42]. The heterothallism demands fusion between allantoid spores that are compatible. The rapid vegetative multiplication of the allantoid spores and the heavy sporulation increases the probability of successful fusion of oppositely charged spores. On potato-dextrose agar medium, the primary sporidia arise in while still on the promycelium and germinate to produce hyphae which in turn produce two types of sporidia falcate (allantoid), which are released forcibly and filiform like the primary sporidia[17]. Holton[33], using staining techniques, found primary sporidia to be mono or binucleate and secondary sporidia to be mostly mononucleate. The primary sporidia germinate either directly, giving rise to lateral and terminal mononucleate hyphae from various cells, or indirectly to form sterigmata from which mononucleate, falcate secondary sporidia are forcibly discharged. The secondary sporidia also germinate directly or by repetition[30]. The release of sporidia from inoculated soil exhibited diurnal periodicity, number being greatest during the night and early hours of the morning[5].

18. Teliospore: An entity to spread disease

19. Seed and soil-borne inoculum

Threshing and harvesting of the infected crop cause the dispersal of teliospores into the soil and over the seeds. Seed- borne inoculums can spread the disease to wheat varieties, planted in non-infested soil of an isolated plot. Quantification of teliospores in soil shows contamination of many fields in Punjab, where teliospores number range between 5 x $l0^3$ and 16 x 10^3 per 250 g of soil. The soil borne teliospores have dormancy and retain their viability upto 42 and 24 months buried in soil up to a depth of 8 and 34 cm, respectively. The teliospores can also survive in wheat straw and farm yard manure up to 2 and 1 year, respectively[7,42].

In infected or bunted seeds, the teliospores are present inside the seed as a black powdery mass whereas in the infested seeds, the teliospores are adhered to the seed surface. During threshing, the infected seeds get ruptured to release the teliospores causing infection in the fresh and healthy seeds.

20. Sporidia: An entity to cause disease

21. Dispersal from soil level to ear head

On germination the teliospores present in soil or on seed surface produce primary sporidia. These sporidia get deposited on the lower leaves by wind and splashes. The maximum trapping of secondary sporidia occurs in air samples during early morning hours, when 100% relative humidity prevails[66]. As the leaf surface dries, these spores get dispersed to higher leaves and to flag leaves. If the boot emergence stage coincides with a mild drizzle or rain, the secondary sporidia get washed down into the sheath[21,64].

22. Proliferation inside the ear head and ovary

There is however, a greater likelihood of the mycelium produced by the germinating sporidia directly invading the ovary. The mycelium finds its way to the ovary after passing through the spaces of lemma and palea[22]. It also moves systemically from one spikelet to another through the rachis and from one floret to another through the rachilla. Such a movement results in Karnal bunt infected grain, often from adjacent florets and oppositely located spikelets[8]. In an ear head 3-4 such sites can get established depending upon the site of infection. On invasion of the ovary either through single or multiple site entry, the mycelium proliferates and switches from vegetative to sporophytic stage. The mycelium/ hyphae proliferate in the space formed by the disintegration of middle lamella of the parenchymatous cells of the pericarp. On short sporophores, the hyaline mycelial cushion produces dark colored teliospores that are so characteristic of the Karnal bunt.

23. Levels of host-pathogen interaction

Host resistance possibly operates by: (a) restricting primary infection, (b) restricting systemic movement between spikelets/florets, (c) arresting mycelial proliferation inside the karyopsis, (d) arresting the switch over from mycelial to sporophytic stages. Since measuring each component in breeding for Karnal bunt resistance is very difficult an integrated selection index is followed taking (i) the number of Karnal bunt infected grains/unit sample and (ii) the average coefficient of infection value derived by grading the Karnal bunt infected grains into various grades based on sorus size[4].

24. Disease Cycle

The Karnal bunt pathogen perpetuates as teliospores which may be deposited in or on the soil at the time of harvesting and threshing, or they may contaminate the surface of seed[10,16]. The teliospores of *T. indica* are known to retain their viability for 7 years in storage.

Sidhartha *et al.*[74], m revealed that the density and the viability of teliospores were reduced with the increase of soil depth. Krishna and Singh[44] found that the maximum length of teliospore survival was 45, 39 and 27 months at 0, 3 and 6 inches depth of the soil, respectively. Nagarajan[64] speculated that the teliospores may not survive the snowing and thawing conditions prevailing in mid altitudes of Himalayas since the disease does not occur at places above the snow line.

Primary infection occurs between heading and anthesis when the sporidia germinate on the glumes and the fungal hyphae enter the epidermal cells of the glumes to penetrate the ovaries directly[61]. The disease progresses systemically to other florets within the spikelet initially infected and then to adjacent spikelets including those on the alternate side of the rachis[24,8,80].

Dhaliwal and Singh[21] proposed an update life cycle of *N. indica.* The primary sporidia germinate while still attached to the soil surface producing allantoid and filiform secondary sporidia. The allantoid or banana shaped sporidia are released forcibly and cause infection in nature. The filiform sporidia serve as the reproductive entities to raise more allantoid sporidia in successive germinations. The allantoid sporidia also multiply on leaf

surface of wheat and other graminaceous hosts and produce superficial colonies which generate crops of allantoid secondary sporidia. The air-borne allantoids secondary sporidia germinate on the glume surface and the fungus becomes partially systemic in rachis and the rachilla. The disease spreads to adjacent florets and spikelets around the primary infection site. Hyphae grow through the base of the glume into the subovarian tissue and enter the pericarp through the funiculus[32]. Pathogen is restricted to the pericarp where it proliferates and sporophores produce single solitary teliospore. The embryo is free from infection, even in severely infested grains[14]. The embryo and endosperm are not colonized[14,32].

25. Integrated management:

- To grow resistant verities i.e. bread wheat: Highly resistant- DBW-52, VL-829, VL-616, TL-2942 (I), HS-375, HS-513, DDW-12, HPW-251, RAJ-3777, RAJ-3765, HPW-211 and HPW-236; resistant- AKAW-4627(I), HI-8627(d), HS-490, WH-1061, HD-4719 (d) and VL-944; moderately resistant- MACS-2971(I), DBW-51, WHD-943, VL-925, HI-1544(C) and PDW-314(1), Durum wheat: Altar C84, Jupare C2001, Aconchi C89, Atil C2000, Banamichi C2004[49].
- Use of disease-free seed is essential.
- Rotate to crops other than wheat, durum wheat, and triticale for up to 5 years.
- Proper irrigation and drainage at the time of heading and flowering.
- Summer deep plowing.
- Avoid dense cropping.
- Seeds treatment by hot water, solar energy are good.
- Proper application of nitrogenic fertilizers.
- The movement of farm machinery and soil from contaminated fields may also be restricted.
- Mulching with polyethylene can be used to raise soil temperature and reduce teliospore germination.
- Planting dates can also be adjusted so that heading does not occur under weather conditions conducive to infection.
- To treat seeds with Thiram 75DS or Kavach 75WP @2g/Kg, Dithane M-45 or Captan 50WP@2.5g/Kg, Vitavax 75WP@2.5g/Kg, Tilt 25EC or Raxil 2DS@1mL/Kg, seed for eliminating seed-borne infection.[48].
- Application foliar spray of Baycor 25WP or Bavistin 50WP or F-100 or Moximate 72WP@2.5g/Kg, Tilt 25EC or Folicur 25EC or Contaf 25EC@1mL/Kg at boot leaf stage and 50% emergence flowering heads[48,49].
- 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[48,49].
- Application of *Pseudomonas fluorescens*@5 mL/Kg or *Trichoderma viride* (Ecoderma) or *T. harzianum*@5 mL/Kg for seed treatment[48,49].

CONCLUSIONS

The major challenge in production /cultivation from Biotic point of view is imposed by Karnal bunt disease. To overcome these challenge Farmers and Growers started indiscriminate use of chemical pesticide. It has not only destroyed the balance of natural system but also imposed health risk to the humans and animals. Resistance against such chemicals has also been reported. Complete dependence on Biological control is also not practical. Hence considering all above challenges we tried to combine maximum possible minimum/non chemical approaches in one plate form which earlier was scattered or confined to the only Research. This review suggests some eco-friendly management approach for significantly important and serious Karnal bunt of wheat. Eco-friendly approaches not only will reduce chemical usage but also will insure good yield and low input cost. As an ecological point of view it will also be useful in bringing harmony between natural and artificial Ecosystem. If it is adopted for long term then there will be less chance for development of Biotype and less frequent depletion of diversity. It will be more effective when all this recommendations are

considered starting from pre sowing to harvesting and storage to insure low chances of disease development (or) at least to maintain it to below Economic Injury Level/ Economic Threshold Level.

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