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Karnal Bunt Disease of Wheat Study from Jhunjhunu, Rajasthan

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Abstract: The disease was first time reported in (1931) form Karnal. The disease cause by fungi. Karnal bunt disease is soil borne as well as air borne. The soil borne & seed borne teliospore germinate when the host is in the flowering stage, both quantity & quality of wheat grains are adversely affected. The spore-mass smells strongly of rottesh fish.

Keywords: Fungi, Karnal Bunt, Disease, Wheat, Fields, Teliospre, Soil.

INTRODUCTION

Fungi exhibit greater diversity in form, function and life-history than other pathogens. They are the most important agent of plant disease. More than 70% of the literature in plant diseases is devoted to fungal infection. (Hawks worth et al., 1995). Smut and bunt disease are characterized by the sooty– black powdery mass of spores formed on the infected plants. The term smut in German language means “dirty”, while “bunt” is a distortion and contraction of “burnt ear”. At an international symposium on Bunts Smuts of wheat (Malik & Mathre 1998) the three major Smuts discussed were Karnal bunt, dwarf bunt, flag smut.

KARNAL BUNT OF WHEAT (partial bunt or new bunt)

Pathogen *Tilletia indica* (syn. *Neovossia indica*)

Distribution: The disease was first time reported in (1931) form Karnal (then in the Punjab, now in Haryana.) by Mitra and since then this has been named as “Karnal bunt of wheat. In Uttar Pradesh this disease was first reported in 1942. it now occurs endemically in India, Pakistan, Nepal, Mexico & U.S.A Karnal bunt is included among “new & emerging disease” at the international level. Though the disease, is of less importance, yet in certain years it caused heavy damage.

The disease in fact becomes a major problem since (1966) with the introduction of Mexican wheat and concurrent new technology for wheat cultivation. Both quantity & quality of wheat grains are adversely affected. The pathogen appears to have very little effect on seed viability. Seeds are likely to have lower survival rate and storage as compared to disease-free seeds.

Pathogen *Tilletia indica* Mitra or *Neovossia indica* (Mitra) & Mundkur

The teliospore are large & darker than those of the hill bunt (*T. caries*) (*T. foetida*). They are spherical and sub-spherical to oval, brown to dark brown, 22-42 micrometer in diameter. Teliospore size is influenced by environment factors.

Their wall is three layered-Endosporium, episporium & perisporium. The endosporium is thick and lamellate whereas the episporium has thick truncate projections. The perisporium is delicate and fragile. Prior to germination the spore require a long resting period. Teliospores germinate after a period of dormancy. Each teliospore germinates to produce a whorl of 32 to 128 filiform primary sporidia at its tip. Occasionally, the promycelium is unusually long or so much suppressed that the whorl of sporidia appears to arise from the teliospore itself. Promycelium can also be branched (false branching) or occasionally two or even three promycelia arise from the some teliospore each bearing primary sporidia. Primary sporidia are long, sickle shaped and do not fuse in pairs to form H shaped structure.

The secondary sporidia produce hyphae or other sporidia. H shaped structures are not formed by *T. indica*. They germinate either directly to produce lateral and terminal monokaryotic hyphae, or indirectly to from monokaryotic falcate secondary sporidia.

Disease cycle: The disease is soil borne as well as air borne. The disease is not systemic. The teliospores are the source of primary

inoculums. The soil borne & seed borne teliospores germinate when the host is in the flowering stage. The disease perennates through soil borne spores which fall down on the soil and survive there. The spores are also introduced into the soil through external seed contaminants. They germinate towards the middle of February to mid-March when suitable soil temperature and moisture are available.

Environment conditions: High humidity & low temperature at flowering time are condition to infection. In Punjab the disease was severe when temperature from February onward were 18°C to 22°C and relative humidity above 70%. The factor most likely to be limiting in the field, is moisture both on the soil surface for teliospore germination and for infection on the spike by secondary sporidia.

Air borne secondary sporidia germinate on the glumes and the fungus then grows in rachis and rachilla & bunt spores are wind-borne to fresh flowers. Infection takes place only in the flower buds soon after the ears have emerged out of the boot leaves and pollination take place. The infection hyphae are incapable of infecting the ears at a later stage. The environment conditions are same in Jhunjhunu for the disease. Germ tubes of dikaryotic sporidia infect actively growing meristem of floral buds of young shoots in the field. As a result of air borne infection, the ovaries of infected spikelet's become converted to bunt balls composed of teliospores.

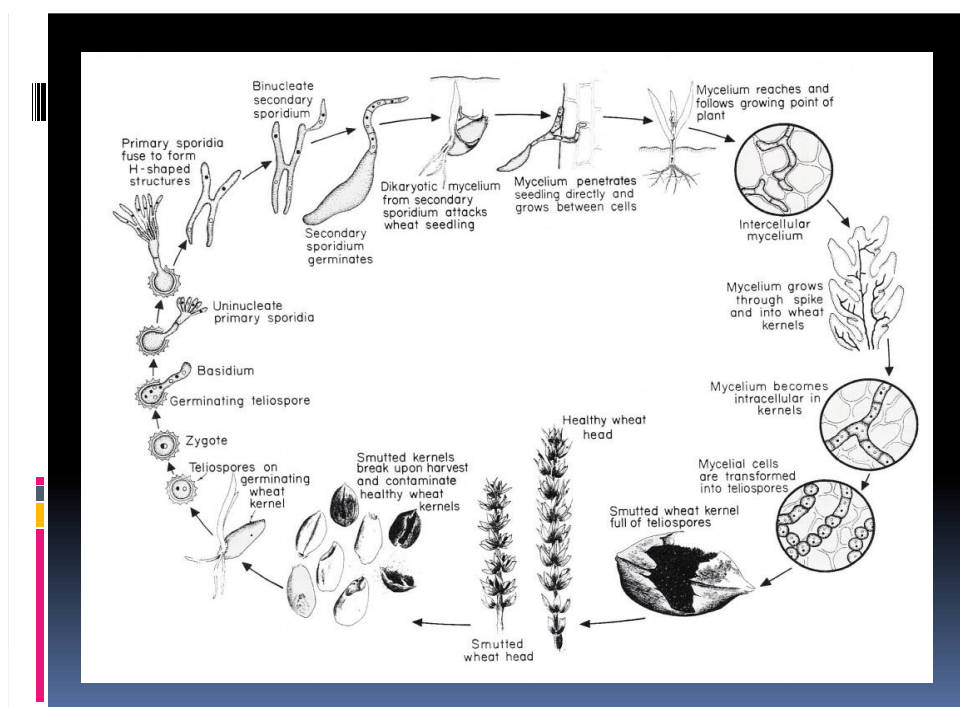


Fig: Disease cycle of Karnal bunt of wheat.

Disease management culture practices - destruction of infected ears, avoiding threshing a diseased crop in the field, avoiding use of excessive nitrogen fertilizers. Dense cropping, greater use of manures, good irrigation, and rotation with non-hosts are preferable. Seeds treatment by hot water, solar energy or fungicides are good, but are not much effective because the primary source of the infection are the teliospores in the soil.

Chemical control by foliar application of fungicides gives good results (Singh et al., 1985). Fumigation of wet soils by Methyl bromide, under plastic cover, reduces teliospores germination by 100%.

Resistant cultivars are available e. g. HD-29, HD-30, HP-1531, HD-1907, HP-743, L-176, M-137A, X-6A. Soil solarization, plastic mulching.

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