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Urdbean web blight and its management strategies- A review

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ABSTRACT

The production and productivity of urdbean ranks lower in India as compared to the that of world average due to the biotic and abiotic constraints. Web blight of Urdbean is caused by *Rhizoctonia solani* Kuhn and is a soil borne pathogen and initial inoculums come mainly from soil splashes which fall on leaves during heavy rains. Respective optimum temperature and relative humidity i.e. 26–28°C and 90–100% favours disease development chronically. The pathogen can survive very longer in the absence of a host either as sclerotia or thick walled brown hyphae in plants debris. Currently the disease is prevalent in many countries across the globe. The pathogen is highly variable in terms of its pathogenicity, culture, morphology, biochemical and molecular characteristics. The severity of this disease is directly linked with the population of viable sclerotia in the soil. Yield losses due to this disease vary in between 20-30% depending upon prevailing conditions.

Key words: Management strategies, Pathogen biology, *Rhizoctonia solani*, Symptoms, Web blight.

Urdbean [*Vigna mungo* (L.) Hopper] also known as black gram, is one of the most valuable short duration pulse crops of India and cultivated in *kharif*, spring and summer seasons covering 3.77 million hectares area and production 1.52 million tonnes. (Purushottam and Singh, 2015). Despite being an important pulse crop its productivity has been quite low probably due to various biotic and abiotic constraints. Among many biotic constraints, Web blight disease of urdbean caused by *Rhizoctonia solani* Kuhn [Teleomorph: *Thanatephorous cucumeris* (Frank) Donk] is considered as an important constraints accountable for losses in production as well as productivity in India up to 20-30% depending on cultivars, environmental conditions, crop stages and cultivation practices (Dubey and Patel, 2001; Shailbala and Tripathi, 2007). Seed quality and grain yield are heavily affected in this disease. The disease had been reported in other countries; Pakistan, Sri Lanka, West Indies, Japan, Philippines, Myanmar, North America, South America, Argentina, Brazil, and Mexico too beside India. There are also reports of this disease from India including few major producing states; Punjab, Haryana, Bihar, Rajasthan, Uttarakhand, Uttar Pradesh, West Bengal, Himanchal Pradesh and Jammu & Kashmir. This review article incorporates etiology and biology of *Rhizoctonia solani* causing web blight diseases of urdbean and deals with disease management strategies in the light of present outlook.

PATHOGEN AND ITS BIOLOGY

The first report of occurrence of web blight on urdbean caused by *Rhizoctonia solani* Kuhn [Teleomorph: *Thanatephorous cucumeris* (Frank) Donk] in India was reported by Saksena and Dwivedi in 1973. This disease is known to occur in other leguminous crops like mungbean (Dwivedi and Saksena, 1975) pigeonpea (Dwivedi and Saksena, 1975), cowpea (Lakshman *et al.* 1979), soybean (Verma and Thapliyal, 1976), groundnut (Dwivedi and Dubey, 1986) and ricebean (Jalali, 1989).

Genus *Rhizoctonia* is considered as a heterogeneous assemblage of filamentous fungal taxa belonging to a group of fungi called the "Mycelia Sterilia." and share a number of common features in their anamorphic states. *R. solani* is a basidiomycete fungus that does not produce any asexual spore and occasionally produce sexual spores called basidiospores formed on specialized structures called basidia. This fungus grow by producing thin, vegetative strands called hyphae and large masses of this hyphae is referred as mycelium. The hyphae of *Rhizoctonia solani* shows many features: a) Each cell is multinucleate (many nuclei) rather than being binucleate; b) Branches at right (90°) and acute angles near the distal septum of cells in young vegetative hyphae; c) Forms septum in the branch near the point of origin; d) has special type of cross wall within the hyphae, called a dolipore septum. In general, the growth rate of *R. solani* is very rapid and can covers a 90 mm Petri plate within 72 hours. *Rhizoctonia* species also produces specialized hyphae composed of compact cells called monilioid cell. The monilioid cells fuse together to produce hard structures called sclerotia, which are brown to black and 3 to 5 mm long. Multinucleate hyphae consisting of many

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nuclei (commonly 4 to 8) per cell distinguishes this fungi from similar fungi that have only 2 nuclei per cell. The hyphae produced by germinating spores will fuse (anastamose), to form new hyphae with a mixture of different types of nuclei. Recently, anastomosis groups (AGs) have been able to elucidate the complexity related to the identification of large fungal genus and to define "*R. solani* isolates" with the help of few morphological characteristics. Further, when isolates pair with each other, only related mycelium fuse and thus anastomosis groups is used to define by a set of tester isolates.

R. solani has been categorised into sub-groups known as anastomosis groups (AGs) and on the basis of results obtained from hyphal fusion test, isolates of this fungus have been assigned to 14 anastomosis groups (AG-1 to AG-13 and AG-BI) according to the characters in culture medium, hosts, physiological as well as biochemical properties (Yong *et al*. 2008). Within each anastomosis groups, there is sub group which is distinguished with each other by a more or less narrow group of host plants. One of the fusion group AG-1 has been further divided into three subgroups designated as AG-1IA, AG-1IB and AG-1IC. Among these sub group, AG-1IB is the ascendant pathogenic flora which causes web blight in urdbean and mungbean (Jones and Belmer; 1989; Yang *et al.* 1990). Mentioned parameters of sub-grouping of isolates within the same AGs are not up to the mark, as no differences occur in their anastomosis reaction. Therefore, in order to determine AGs or subgroups in *R. solani*, genetic analysis using molecular approaches that employ multiple genetic loci is considered to be more effective.

Isolates of *R. solani* that exhibits DNA base sequence homology and affinities for hyphal anastomosis may represent a diverging evolutionary unit (Kuninaga and Yokosawa, 1980). This hypothesis has also been supported by restriction fragment length polymorphisms (RFLPs) and the sequences analysis of ribosomal RNA genes (rDNA) among different anastomosis groups of *R. solani* (Gonzalez *et al.* 2001; Carling *et al.* 2002).

VARIABILITY

Variability in *Rhizoctonia solani* has been reported by various authors with respect to its cultural, morphological, biochemical and molecular aspects (Tiwari and Khare, 1998; Lal *et al*., 2009; Susheela and Reddy, 2013; Prasad *et al*., 2015). This variability includes different virulence patterns, fungicide resistance, optimal growth temperature and epidemiology (disease development rate, fungal propagule type, dissemination and survival). Studies on variability provide information regarding pathogenicity and pathological behaviour under both controlled and field condition. Variations in *R. solani* isolates based on anastomosis groups (AGs) have also been recorded and it is 14 AGs so far (Yong *et al.* 2008). Although several AGs types has been characterized, but still there is reports that some of the isolates of *R. solani* from different AG generally do not anastomose with each other (Carling, 1996).

Cultural and morphological variability: Variation in colony colour ranges from creamy white, light whitish brown to dark brown in colour (Tiwari and Khare, 1998; Singh *et al*., 1999). This can be seen even in same anastomosis group (AG-1). Hyphal diameter vary widely both within and among the isolates. Diameter of hyphae within a colony also vary greatly depending upon the age of fungus, hyphae collected from different locations in the culture plate (periphery to centre), composition of culture medium and incubation temperature (Dhal, 1933). Nature of mycelium could be attributed to different geographical regions to which the isolates belonged and their adaptability to the new niche. Isolates of *R. solani* causing web blight showed variation in number of nuclei per cell, varied from 3-15 and in the tip cell it ranged from 2-5 nuclei per cell (Tiwari and Khare, 1998). Sclerotia characters within and among isolates, such as; colour, pigmentation, number of sclerotia per plate, pattern and position of sclerotia formation was also variable. The Sclerotia develop either individually or aggregated (Singh, 2006). Singh (2001) identified variation in pigmentation, intensity, pattern and position of sclerotia formation among different isolates of *R. solani*, belonging to same anastomosis group.

Pathogenic variability: Isolates of *R. solani* are known to show greater variability in pathogenecity and categorized into poorly, moderately and highly pathogenic on the basis of the degree of their pathogenicity (Tiwari and Khare, 1998; Singh, 2006). Generally *R. solani* is considered as a non-specialized plant pathogen; however, host specificity has been recognized at various levels. For example, isolates AG-1 and AG-2 could be divided into subgroup based on pathogenicity (Anderson, 1982; Liu *et al.* 1991) even with a wide host range, where as isolates of AG-3 were pathogenic primarily on potato only (Anderson ,1982). Isolates of web blight of urdbean were pathogenic to rice and maize but do not produce typical sheath blight and banded blight symptoms on rice and maize, respectively (Ogoshi, 1987; Singh, 2006). Parmeter *et al.* (1969) also reported that mode of pathogenicity appears to be quite variable within the groups.

IDENTIFICATION

Cultural and microscopic identifications: *R. solani* does not produce spores; hence it is identified only from mycelial characteristics or DNA analysis. It produces cottony white to deep brown colored mycelium when grown on artificial culture medium. It produces small number of globose sclerotia, which are initially white, later turn to brown or black in colour. The hyphae are 4-15 μm wide and tend to branch at right (90°) angles and usually possess more than three nuclei per (multinucleate) hyphal cell. A septum near

each hyphal branch and a slight constriction at the branch are diagnostic (Fig. 1).

Molecular/biochemical identifications: Molecular methods are generally more accurate, sensitive, specific, quantitative, and can be performed by personnel with no specialized taxonomic expertise (Capote *et al.* 2012). Traditional technique is able to identify *Rhizoctonia* up to the genus level only and it takes at least one week for times or more. Several biochemical techniques *viz.*, soluble protein patterns, isoenzymes, pectic zymograms, fatty acids profiles, lectins and serological have been used for identification/ detection of *Rhizoctonia* species complex (Jabaji-Hare, 1996). Among these techniques, pectic zymograms and isoenzyme profiles have been employed to identify anastomosis groups or the study of genetic relationships in members of the form genus *Rhizoctonia*, especially in *R. solani* and binucleate species (Damaj *et al.* 1993; Masuhara *et al.* 1994). After the introduction of PCR, a revolution has occurred in the area of pathogen diagnostics. Recent approaches utilizing molecular markers, genome finger printing, and ITS sequencing have increased the speed, precision, and practicality of accurately identifying or grouping *R. solani* isolates (Lakshman and Amaradasa, 2014). The most common region used for these purposes has been the internal transcribed spacer (ITS) region of ribosomal RNA genes. rDNA region consists of multiple copies (up to 200 copies per haploid genome) arranged in tandem repeats comprising the 18S small subunit, the 5.8S, and the 28S large subunit genes separated by internal transcribed spacer regions (ITS1 and ITS2) (Liew *et al.* 1998; Aggarwal *et al.* 2013). This region contains highly conserved areas and high copy numbers of rRNA genes in the fungal genome which can be used for sensitive detection of *R. solani*. This region was used for defining sub group of anastamosis of *Rhizoctonia solani* (Budg, 2009). Furthermore, a large numbers of ribosomal RNA sequences are publicly available in databases, facilitating the validation and the reliability of the detection assays. Accurate detection of genetic variation among strains/isolates within a species has been reported to be very successful in *R. solani* using molecular markers like Random Amplified Polymorphic DNA (RAPD) (Sharma *et al.* 2005; Lal *et al.* 2014). *Rhizoctonia solani* can be identified using a set of universal primers ITS1 (5'-TCCGTAGGTGAACCT GCGG-3') and ITS4 (5'-TCCTCCGCTTATTGA TATG C-3') based on ITS region (White *et al.* 1990).

SYMPTOMATOLOGY

The first symptom appears as small circular brown spot on the leaves. These spot enlarge often show concentric banding and surrounded by irregular shaped water soaked areas (Singh, 2006). The lesions expend and coalease and white fungal growth is observed on lower surface of the infected including petioles and young branches of infected plants. The spot become greenish to reddish brown and later turn brown to black. The coloured, spider web like mycelial growth and white microsclerotia develop in abundance on the affected plant parts. The mycelium on infected leaves appear as spider web thus suggested the name web blight disease (Fig. 2). If infection occurs on collar region, reddish brown lesion occurs and causes collar rot. It soon girdles the basal portion of stem and this point seedling wilt and collapses and lies flat on the ground. The pods are also vulnerable to infection at all stages of their development. In the initial stage, light irregular specks appeared on the young pods, the spots were dark brown, more or less circular, sunken with darker margin in mature pods. Gradually the spots increased in number and size to cover the entire pods. The grains in the infected pods become small, shrivel and light in colour.

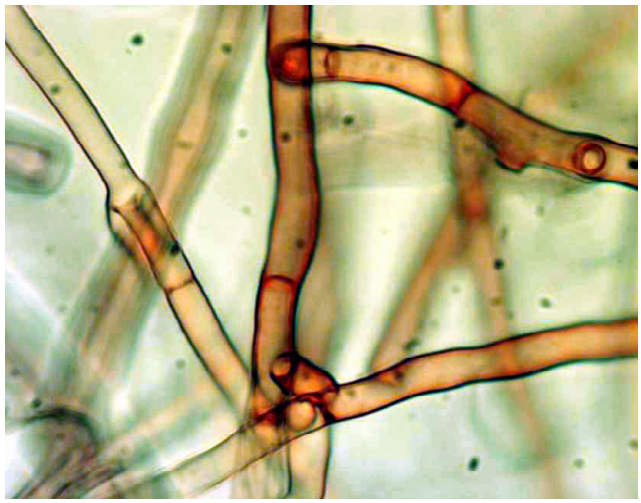

Fig 1: ***R:*** *solani* mycelium showing constriction and septa at the branching at right angle.



Fig 2: Leaves showing typical web blight symptom.

HOST RANGE

Strains of *Rhizoctonia solani* may differ in the hosts they infect to, the virulence of infection, selectivity for a given host ranges from non-pathogenic to highly virulent. Scanty information is available in perspective of the host range of *R. solani* infecting urdbean and crops. *R. solani* can infect about 32 plant families. The strain of *R. solani* from weed host has been reported to be pathogenic to several crop plants too (Bains and Dhaliwal, 1993). Kumar *et al*., (2013) evaluated 21 host species belonging to different families (Leguminosae-11, Gramineae-4, Solanaceae-3 and Malvaceae-3) and found that the *R. solani* was able to infect all crop plants. However, typical web blight symptoms were observed on plants belonging to family Leguminosae followed by Gramineae only. Minimum disease infection was observed on plants belonging to family Solanaceae. Sharma and Tripathi (2001a) have also revealed host range of urdbean isolates of *R. solani* and the wide host range belongs to family *Leguminoseae, Solanaceae, Brassicaceae, Malvaceae* and *Cucurbitaceae*. In mungbean field, several weed spp. infected with *R. solani* transmit the disease in the mungbean also (Bains and Dhaliwal, 1993).

EPIDEMIOLOGY

The pathogen is known to prefer warm wet weather, and its outbreaks occur typically during an early summer months in tropics and subtropics region. A combination of environmental factors has been linked to the prevalence of the pathogen such as: presence of host plant, frequent rainfall/ irrigation and increased temperatures and relative humidity in spring and summer. In addition, soil type, reaction, moisture and temperature are also known to create favourable environments. Higher aerial temperature (26 to 32°C), relative humidity near 100% and soil temperature 30-33°C favour the development of high disease severity. Rainfall (91-97 mm) has a significant role in severe development of web blight during early stage of crop in urdbean (Sharma and Thripathi, 2001a). Optimal hyphal growth was measured at temperature range of 20 to 30°C with an optimum at 25°C, when pH range was 5 to 8 (Kumar *et al.* 2014b). Dubey (1997) reported that 26-28°C temperature and 90-100% relative humidity favour development of severe disease.

SURVIVAL AND DISEASE CYCLE

R. solani is a soil-borne fungus primarily and can survive for longer either as sclerotia or thick walled brown hyphae in plants debris in soil and on plant tissues (Boosalis and Scnaren, 1959). Most of sclerotia occur on the tap root of diseased plants and in the soil adjacent to the diseased root. Over 80% sclerotia occurred on the top 10 cm of the soil. The pathogen can be disseminated by irrigated water, infected/ infested or contaminated seeds, transplanted material, rain splashed sclerotia and farm equipments. In the tropics where the teleomorph of *R. solani* develops regularly, the pathogen spreads rapidly by the production and dissemination of airborne basidiospores. The fungus is attracted to the plant by chemical stimuli released from actively growing plant and/or decomposing plant residues. Entry of pathogen can occur through direct penetration of the plant cuticle/epidermis or by means of natural openings in the plant. During this process, the hyphae of fungus will come in contact with the plant and attach to its external surface. Fungi begin to produce an appressorium or infection cushion as a result of mycelial growth. Later on appressorium penetrates the plant cell and allows pathogen to obtain nutrients from the plant cell. The pathogen can also release enzymes that break down the various components of plant cell walls (e.g. cellulose, cutin and pectin), and continue to colonize inside dead tissue, often forming sclerotia. These sclerotia act as a inoculum for new host tissue, and repeats new cycle. The secondary spread of the disease may results due to growth of filamentous hyphae from either one leaf to another leaf of the same plant or from the leaves of one plant to another plant. The maximum diseases severity occurs in 30-60 days old plant probably because of dense canopy of the crop which facilitates the early spread of the pathogen through contact of plants and leaves, forming mycelial bridges. Kumar *et al.* (2013) reported that, maximum web blight severity was recorded in the plants inoculated at 60 days (68.8%) after sowing followed by 45 days (65.1%) and 30 days (61.5%). It has been observed in field condition that plots heavily infested with weeds possess high severity of web blight.

DISEASE MANAGEMENT STRATEGIES

The *Rhizoctonia solani* Kuhn is a soil-borne fungal pathogen, polyphagous, necrotrophic and adaptability to diversified ecosystems have made the management of the disease caused by this pathogen a challenging job. Even no single method is available currently for effective and economic management of web blight disease. However few of the important control measures which are applicable either as in single or integration have been dealt below in detail.

Cultural management: Cultural practices mostly affect the environmental conditions favorable for the growth, building up of inoculums, survival and their spread. Manipulation in sowing date with proper row spacing may be used to cope with the losses incurred due to web blight. Efficacy of this manipulation would differ from place to place, as it is the temperature and relative humidity in an area that determine reduction or increase in plant mortality during the crop season. A wider row spacing of 50 cm or more resulted in decreased aerial blight infection and increased yield of soybean (Joye, 1990). Certain practices *viz.*, proper sanitation, avoiding of planting at the time of rainy season during the susceptible crop stage, avoiding the thick canopy of the crop using proper seed rate and adopted proper rotation are helpful in the control of this disease to a greater extent. Burying of the infected leaves immediately after harvest will reduce the concentration of primary inoculums.

Diseases resistance source: Progress in breeding for Web blight resistance in urdbean is limited due to considerable variability in varietal reaction and the less understanding of pathogen variation and disease resistance mechanisms. Presently, high levels of WB resistance are not available in commercial varieties. Basandari *et al*. (1999) found genotypes HPBU 67, KU 304, TEU 95-1 as a highly resistant, while COBG 627, HPBD 47, HPBU 51, HPBU 70, HPU 153, HPBU 216, P 44, UPG 89-7, VPU 91-7 and 'HPBU 5, HPBU 7, HPBU 38, HPBU 64, HPBU90, HPBU94, HPBU 108, HPBU 122, P 38, Pant U 19, Pant U 30, PDU 89-8, PU 23, Pusa 3, UG 367, VPU 20, WHG 57, LEG 626 as a moderately resistant. In one of the experiment, Kumar *et al*. (2014a) evaluated 45 genotypes against web blight, where six genotypes *viz.*, NDU 7-24, COBG 653, IPU 02-33, KU 99, OBG 31 and NM 57 showed highly resistant reaction while nine KKB 20055, UH 04-6, IPU 02-43, SB 27-3, Pant U 30, KUG 222, LBG 623, LBG 20 and BS 23-5 showed resistant reaction only. In other experiment Neelam *et al*. (2014) evaluated 42 genotypes and they found that fourteen genotypes i.e. IPU-2-43, KPU-1-10, KU-1106, CoBG-10-5, LBG 752, VBG-10-024, NUL-7, ACM 05- 007, Uttara-3, UH-08-05, UH-07-06, CoBG-761, NDU-11-202 and KUG-58 were moderately resistant to web blight. Genotypes with multiple resistances against mungbean yellow mosaic and *geminivirus* and Rhizoctonia web blight have already been reported (Kaushal and Singh, 1989).

Biological management: Sufficient augmentation in agriculture produce can also be brought through biological control instead of using pesticides. Seed treatment with *T. viride*, carboxin and *Rhizobium* sp. provides additive effect for better management of disease due to variation in the mode of action of the fungicide and bioagent. *Rhizobium* is not inhibited by *Trichoderma* but contributes greater root nodulation during seed treatment. Dubey and Patel (2001) evaluated six isolates of *Trichoderma viride, T. harzianum* and *T. virens* against *R. solani* causing web blight of urdbean and were found superior results. Shailbala and Tripathi (2010) screened *T. virens* for its antagonistic potential against *R. solani* causing web blight of urdbean and showed that it grew fast and better potential after 6 days incubation, which attained 78.2 mm growth showing 59.1% inhibition of the radial growth of pathogen. Kumar and Tripathi (2011) evaluated six strain of *T. harzianum* against *R. solani* causing web blight of urdbean and reported that all strains of *Trichoderma* suppressed the growth of the pathogen significantly. Maximum inhibition 88.8 mm after 72 hrs of incubation was recorded in strain Th5 followed by strain Th1, where inhibition was 85.0 mm. Ray *et al*. (2007) tested efficacy of two fungal bioagents, *T. harzianum, T. viride* and one bacterial bio-agent, *Pseudomonas fluorescens* under *in vitro* conditions against *R. solani*. *T. harzianum* inhibited mycelial growth of *R. solani* after 96 hrs of incubation followed by *T. viride* and *Ps. fluorescens*. However, after 120 hrs of dual culture with *R. solani*, both bio-agents covered the inoculated plates completely. The bacterial bio-agent *Ps. fluorescens* produced clear inhibition zone and no fungal growth was observed at the site of inhibition zone. After 120 hrs of incubation *Ps. fluorescens* overlapped the test pathogen. Sharma and Tripathi, (2001b) reported that *G. virens* was found most effective with respect to low disease severity (47%) and more grain yield (791-851 kg/ha) in comparison to *T. harzianum* under field conditions.

Botanical management: The presence of antifungal compounds in higher plants has long been recognized as an important factor to control disease (Mahadevan, 1982). Ansari (1995) reported fungistatic activity of extracts of *Ocimum sanctum, Mentha arvensis* and *Eucalyptus* sp. against *R. solani*. Foliar spray of *Pongamia glabra* leaf extract successfully reduce web blight of urd and mungbean and can be further exploited in organic farming (Dubey 2002). Chalavaria (2005) tested nine botanicals namely *Calotropis*, eucalyptus, neem, *Vinca rosea*, mentha, onion, garlic, datura and tulsi against *R. solani* and showed that the garlic extract was found effective in inhibiting cent-percent of mycelial growth which was followed by Onion (50%) neem (48.21%), datura and eucalyptus (41.07%). Jhamaria and Sharma (2002) reported that leaf pep (an organic liquid based on natural clove oil) was found effective for controlling web blight of mungbean. Aqueous plant extracts (*Calotropis gigantea, Vinca rosea, Ocimum sanctum, Azadirachata indica, Eucalyptus citriodora, Allium cepa* and *Zingiber officinale*) against *R. solani* causing web blight in moongbean in *in vitro* and found that highest inhibitory action (86.11%) was recorded with *Zingiber officinale* (Mishra *et al*., 2005). Kumar and Tripathi (2012) tested six botanicals namely onion, garlic, neem, bhang, ginger, and datura against web blight of urdbean and showed that the onion extract was found effective in inhibiting mycelia under *in-vitro* condition and therefore in, reduction in disease severity, increase in grain yield kg/ha and 1000 grains weight.

Chemical management: Fungicides play a very important and significant role in disease management because of fact that they are easy to apply, economical, quick acting and effective. The effect of bavistin or benlate @ 2.5 g/kg seed was found effective in eliminating the seed-borne infection of *T. cucumeris* causing web blight of ricebean (Tiwari, 1993) and dolichos bean (Dwivedi and Dwivedi, 1999). Sharma and Tripathi, (2001b) observed that seed treatment and two foliar sprays of tilt (0.1%) at 15 days interval was most effective in reducing disease severity (30-32%) and increased grain yield (950-1012 kg/ha) as well as 1000-grain weight followed by bavistin, caftaf and indofil M-45 sprayed plots. Shailbala and Tripathi (2004) revealed that tilt (0.1%) gave lowest disease severity, highest grain yield and maximum thousand grain weight followed by bavistin (0.1%), dithane

M-45 (0.25%). Shailbala and Tripathi (2010) screened eight fungicides namely propiconazole, hexaconazole, carbendazim, tebuconazole, epoxiconazole, mancozeb, propineb, thiophanate-methyl were tested at different concentration against web blight of urdbean. Epoxiconazole was found the most effective followed by propiconazole and carbendazim. Epoxiconazole completely inhibited radial mycelia growth at 2.5 µg/ml concentration while propiconazole at 5 µg / ml and hexaconazole and carbendazim at 10 µg / ml concentrations. Kumar *et al.* 2015 evaluated six systemic and non- systemic fungicides at different concentration under field condition. Carbendazim as systemic fungicide was found the most effective followed by propiconazole and hexaconazole, while captaf non-systemic fungicide as was found most effective followed by dithane M-45 and sulphur. Foliar spray of carbendazim 0.05% along with seed treatment with celest (0.2%), raxil (0.2%) or carbendazim (0.2%) was highly effective in reducing web blight severity of frenchbean followed by topsin-M and baycor, significant increase in yield was also observed (Mathew and Gupta, 1996). Five fungicide namely carbendazim (0.2%), propiconazole (0.1%), hexaconazole (0.1%), propineb (0.1%), mancozeb + thiophanate-methyl (0.25%) as seed treatment and foliar sprays were tested at different concentration against web blight of urdbean (*Vigna mungo* cv. PU-19). Propiconazole (0.1%) at 15 days interval resulted highest reduction in disease severity (30-32%), grain yield (950-1012 kg/ha) and 1000-grain weight (35 g) (Sharma and Tripathi, 2000). Dubey and Dwivedi (1988) found that bavistin at 0.2% was the most effective of 6 spray treatments against *Thanatephorus cucumeris* on urdbean followed by dithane Z-78 at 0.25% and benlate (benomyle) at 0.2% under glasshouse conditions.

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