Review Article

Current status of web blight of mung bean

JAI SINGH, K.K. MISHRA AND A.K. SINGH

MEMBERS OF RESEARCH FORUM:

Corresponding author:
JAI SINGH, Krishi Vigyan Kendra, SIDHI (M.P.) INDIA
Email: sidhi_jai01@rediffmail.com

Co-authors:
K.K. MISHRA, Regional Agricultural Research Station, POWARKHEDA HOSHANGABAD (M.P.) INDIA
A.K. SINGH, Krishi Vigyan Kendra, SIDHI (M.P.) INDIA

Key words: Web blight, Mung bean


Introduction

Pulses in India have been considered as the poor man’s only source of protein. Pulses are grown in 22-23 million hectares of area with an annual production of 13-15 million tonnes (mt). India accounts for 33 per cent of the world area and 22 per cent of the world production of pulses. Among the pulses mungbean [Vigna radiata (L.) Wilczek] also known as green gram or golden gram is one of the most important short duration pulse crops of India and grown in Kharif, spring and summer seasons. It is cultivated on 3.77 million hectares produced 1.52 million tonnes of grains (Anonymous, 2000). Mungbean mainly grown in Rajasthan, Maharashtra, Karnataka, Andhra Pradesh, Orissa, Bihar, Tamil Nadu, Madhya Pradesh and Uttar Pradesh.

Even with the best efforts, mungbean production and productivity has been stagnant due to mungbean is attacked by more than one disease and pest at a time. These are responsible for heavy yield reduction and contribute substantially in instability of production of the crop. Mungbean suffers seriously from several viral and fungal diseases. Among these Mungbean Yellow Mosaic Virus (MYMV), Cercospora leaf spot (CLS), Web blight and Anthracnose are the important diseases.

Geographical distribution:

Web blight is one of the major constraints in the production of many pulses in warm humid tropic zones of the world. On mungbean Rhizoctonia blight was reported for the first time from Philippines (Nacien, 1924) in 1924. Alam et al. (1985) reported occurrence of web blight of mungbean in Pakistan.

In India first report of its occurrence on mungbean was given by Dwivedi and Saksena (1974) from Kanpur, Uttar Pradesh subsequently; this disease has also been reported from Assam (Saikia, 1976), Punjab (Bains et al., 1988), Madhya Pradesh (Tiwari and Khare, 1998), Bihar, Rajasthan, Haryana, Himachal Pradesh and Jammu & Kashmir (Anonymous, 2004).

The disease has been known to occur in India on other leguminous crops like black gram (Saxena, 1973 and Sharma and Tripathi, 2001); pigeonpea (Dwivedi and Saksena, 1975); Cowpea (Dwivedi, 1977 and Lakshman et al., 1979) Soybean (Verma and Thapliyal, 1976); Groundnut (Dwivedi and Dubey, 1986) and ricebean (Jalali, 1989).

Economic importance:

In 1976 Saikia gave an account of the incidence and etiology of blight of Phaseolus aureus (Vigna radiata) resulting into about 30 per cent plant mortality. However,
status of web blight of mungbean in Eastern Uttar Pradesh was 1.0 to 69.0 per cent with an average of 12.7 per cent. The disease was observed to reduces 33 to 40 per cent grain yield and 28.6 per cent in 1000 grain weight at different level of disease severity and in different variety of mungbean (Singh, 2006 ; Gupta and Singh, 2002 and Gupta et al., 2010). Since then the web blight of mungbean has become one of the most serious problems of this crop in Northern India causing extensive damage to mungbean.

**Symptoms :**

Web blight of mungbean caused by *R. solani* (Kühn) is able to cause the disease both by soil borne and air borne mode of infections. The fungus infects all above ground parts of the plants *i.e.* leaves, petioles, stem and pod but is most destructive on foliage during second to third week of plant growth causing seedling mortality. If infection occurs on collar region, reddish brown lesion in the cortex of hypocotyle at or below the soil level develops. The seedling is killed when the lesions on hypocotyle; girdles the stem. The rapidly growing mycelium; hyaline when young but later turning brown; 4.5 to 13.25 µ and branching near the distal septum of hyphal cell, often nearly at right angles in older hyphae. Construction of branched hyphae at the point of origin and possess septum near its junction with the main axis. The cells of growing hyphae are always multinucleate and the septum is doliopore type. Sclerotia are white but later turning chestnut brown, globose to sub globose, oval or cushion shaped, often less than 1 mm in diameter to thin crusts several centimetres across (Saikia,1976; Saikia and Roy, 1976 and Singh, 2006). The Sclerotia are not differentiated into rind and medulla. Production of moniloid cells, often called barrel shaped cell or chlamydomspores, are 12-21 x 9-13 µ in size and formed in chains but some of these aggregating together to form masses up to 1.2 cm in diameter and formed superficially (Grewal et al., 1988).

On the host, the sclerotia are superficial, variable in shape and size, dark brown to black, scab like and accompanied with dark short called, abundantly branched, stout mycelium without clamp connections. Inside the host tissues, the mycelium is slender, hyaline and with longer cells.

The perfect state *T. cucumeris* develops on healthy tissue adjacent to the lesion on black gram leaves as well as *in vitro* (Saksena, 1973; Tiwari and Khare, 1998). The sexual stage is induced on the host under warm and humid conditions with heavy dew formation during the night. Fructifications are resupinate, creamy to greyish white, loosely attached to the substratum, composed of arachnoid, repent hyphae giving rise to thin hypochroid or sub membranous fertile patches. Cluster of basidia are produced terminally in a discontinuous hymenium. Hyphae 4.5 to 17.0 µm wide without clamp connections, often anastomosed; basal hyphae widest with branching at wide angles while median and subhyaline hyphae are narrow, thin walled and hyaline. Basidia are mostly 9-25×5-12.4 µm in size but some times short, wider, barrel shaped to sub cylindrical without a median constr. Sterigmata are straight, attenuate towards the apex usually bearing 4 favourable conditions (Weber, 1939; Galvez et al., 1979).

**The pathogen :**

The pathogen has been reported on mungbean under the name *Thanatephorus cucumeris* (frank) Donk (Dwivedi and Saksena, 1974; Wang and Yang, 1976 and Singh, 2006). *T. cucumeris* the teleomorph of web blight pathogen of mungbean belongs to the family Ceratobasidiaceae of the order Tulasnellales, class Hymenomycetes, sub class Halobasidiomycetidae of the phylum Basidimycota. The anamorph; *Rhizoctonia solani* belongs to the phylum Ascomycota form class Deuteromycetes and order Agonomycetales (Alexopolus et al., 1996).

The colonies of *R. solani* are yellowish white to light yellowish brown and letter becoming pale brown to dark brown in colour. Sectoring of colony is frequent on agar medium. The rapidly growing mycelium; hyaline when young but later turning brown; 4.5 to 13.25 µ and branching near the distal septum of hyphal cell, often nearly at right angles in older hyphae. Construction of branched hyphae at the point of origin and possess septum near its junction with the main axis. The cells of growing hyphae are always multinucleate and the septum is doliopore type. Sclerotia are white but later turning chestnut brown, globose to sub globose, oval or cushion shaped, often less than 1 mm in diameter to thin crusts several centimetres across (Saikia,1976; Saikia and Roy, 1976 and Singh, 2006). The Sclerotia are not differentiated into rind and medulla. Production of moniloid cells, often called barrel shaped cell or chlamydomspores, are 12-21 x 9-13 µ in size and formed in chains but some of these aggregating together to form masses up to 1.2 cm in diameter and formed superficially (Grewal et al., 1988).

On the host, the sclerotia are superficial, variable in shape and size, dark brown to black, scab like and accompanied with dark short called, abundantly branched, stout mycelium without clamp connections. Inside the host tissues, the mycelium is slender, hyaline and with longer cells.

The perfect state *T. cucumeris* develops on healthy tissue adjacent to the lesion on black gram leaves as well as *in vitro* (Saksena, 1973; Tiwari and Khare, 1998). The sexual stage is induced on the host under warm and humid conditions with heavy dew formation during the night. Fructifications are resupinate, creamy to greyish white, loosely attached to the substratum, composed of arachnoid, repent hyphae giving rise to thin hypochroid or sub membranous fertile patches. Cluster of basidia are produced terminally in a discontinuous hymenium. Hyphae 4.5 to 17.0 µm wide without clamp connections, often anastomosed; basal hyphae widest with branching at wide angles while median and subhyaline hyphae are narrow, thin walled and hyaline. Basidia are mostly 9-25×5-12.4 µm in size but some times short, wider, barrel shaped to sub cylindrical without a median constr. Sterigmata are straight, attenuate towards the apex usually bearing 4
basidium, rarely 2,3 or up to 7 in number. Basiospore are hyaline smooth thin walled, oblong to ellipsoid and dorsally fattened or broad ovoid and commonly widest at the distal end form secondary basidiospores (Sakshena, 1973).

Anderson (1982) stated that hyphal anastomosis in *R. solani* is a manifestation of somatic or vegetative incompatibility between hyphae. Yokoyama and Ogoshi (1984) conducted an experiment to that when two isolates of *R. solani* paired 2-3 cm apart on a medium (usually 2% water agar) in a Petridish, their mycelia grow and overlap each other which can be observed under light microscope at low magnification. Concluded that if hyphal fusion occurs (C-3 type reaction or perfect fusion), these isolates are assigned to the same anastomosis group and often, attraction of hyphae and death of fused cells (C-2 type reaction or killing reaction) are observed in such a situation. If fusion, attraction and hyphal death do not occur, the isolates are placed in different anastomosis groups (AGs). The AGs system is now considered as the most useful grouping method for *R. solani*. Based on hyphal anastomosis reaction *R. solani* has been divided into 12 anastomosis groups (AG-1 to AG-11 and AG-B1) and additional AG probably exists (Carling, 1995). Recent protein and DNA – based studies support the separation of *R. solani* into genetically distinct groupings, but has also revealed considerable genetic diversity within one anastomosis group. Hyphal anastomosis and molecular methods are currently being used to further examine the taxonomy, ecology and pathology of *R. solani*. Many of these are sub divided (using cultural, virulence, molecular, biochemical, immunological or other characteristics) into intra-specific groups (IGs) (Table 1) (Ogoshi, 1987). Ogoshi (1987) suggested that based on pathogenicity, Ag-1 can be divided into at least three groups IA,IB and IC. Isolates of AG-1IB (the Web blight fungus) are also virulent on rice but cause different type of symptoms on it than do isolates of AG-1IA (Rice sheath blight).

*Rhizoctonia solani* casual agent of web blight of mungbean belongs to anastomosis group 1, intraspecific group IB (Singh, 2006). Aerial blight pathogen (*R. solani*) of soybean belongs to AG-11A group (Yang and Chen, 1989) while both IA and IB intraspecific groups and are responsible for causing foliar blight of legume crops specially in soybean (Harvillae et al., 1996).

**Variability :**

**Cultural and morphological :**

Growth rate of different web blight isolates of *R. solani* from mungbean showed wide variation ranging from 1.25 mm per hour to less than 1 mm per hour (Singh, 2006; Tiwari and Khare, 1998). Variation in colony colour ranged from creamy white, whitish brown to dark brown in colour (Singh, 2006; Tiwari and Khare, 1998). Even in same anastomosis group (AG-1) Colony colour of different isolates were varied from light to dark brown (Li and Xia, 1988; Singh et al., 1999). Variation in hyphal width within an anastomosis group was observed, range of hyphal width was found 4.68 to 13.25 μm (Li and Xia, 1988; Singh et al., 1999; Tiwari and Khare, 1998; Das Gupta, 1992). Hyphal diameter varied widely both within and among the isolates. Diameter of hyphae within a colony also varied greatly depending upon the age of fungus, hyphae collected from different locations in the culture plate (periphery to centre), composition of culture medium and temperature of incubation (Dhal, 1933; Palo, 1926). Nature of mycelium could be attributed to different geographical regions to which the isolates belonged and their adoptability to the new niche. The type mycelial behaviour was recorded *i.e.* slight, moderate and abundant in 48 web blight isolates of mungbean. 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; Singh, 2006). According to Ogoshi (1987) isolates of *R. solani* having more than two nuclei per cell, come under teleomorph *T. cucumeris* however, isolates having two nuclei per cell have been grouped under teleomorph in the genus Ceratobasidium Rogers. Sclerotal characters of 48 isolates of *R. solani* grown at 28± 2°C yielded interesting results. Pigmentation of sclerotia varied from reddish yellow to light reddish brown. In some isolates sclerotia were light brown to dark brown in colour. Beside 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* which belonged to same anastomosis group. Size of sclerotia of web blight isolates varied from 0.2 to 5.0mm in diameter (Burpee et al., 1980; Yang et al., 1989; Singh, 2006). Papvizas (1965) suggested that variation among single isolates elicit sclerotia of sibling isolates which may show enormous differences in size, shape, colour, texture and distribution on culture medium.

**Pathogen variability :**

In general *R. Solani* is considered as a non- specialized plant pathogen; however, host specificity has been recognised at various levels. For example, isolates AG-1 and AG-2 could be divided into sub group 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 were pathogenic to rice, 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. Different isolates of *R. solani* showed wide variation in their host range and were classified according to their host specificity.
Isolates of *R. solani* are known to show greater variability in virulence. Isolates of *R. Solani* of mungbean were grouped into three different categories i.e. poorly virulent, moderately virulent and highly virulent on the basis of the degree of their virulence (Tiwari and Khare, 1998; Singh, 2006).

**Host parasite interaction:**

It was thought that *R. Solani* penetrates the host through stomata by means of growing hyphal tips without formation of any swelling, mycelial cushion or asppersorium but now it has been shown (Jhooty and Bains, 1976) that the pathogenic isolates of *R. solani* forms the infection cushion on green gram hypocotyls. Non pathogenic isolates do not form any infection cushion. Not only the host, the infection cushion formation differs on different parts of the same plant according to their susceptibility. It has been reported that in mungbean and urdbean crops, *R. solani* penetrates by hyphal tips developed beneath the infection cushion formed by the mycelial growth on the host surface and through the stomatal openings without the formation of appressorium above the stomata (Dubey and Dwivedi, 1989). After penetration, the hyphal branches grow rapidly both inter and intra cellularly in the host tissue. The invaded mesophyll cells collapse and become discoloured after some time.

After infection the physiology of the host is influenced. It is reported to increase the rate of leakage of electrolyte from the cells of green gram hypocotyls. It is due to increase in the cell membrane permeability caused by a heat- labile substance detected in droplets formed on the infected host surface (Lal et al., 1980). It seems that the toxins produced by the pathogen play important role in the establishment of the infection of *R. solani* in green gram, Wu (1965) showed a direct correlation between the growth, pathogenicity and toxin production in an isolates of *R. solani*. The giberellic acid probably reverses the process of the disease development in greengram due to *R. solani*.

The infection by *R. solani* increases the reducing sugars and amino acid contents but decreases the nucleic acid and protein contents of greengram (Wu, 1967). The protein contents decreases in the early stages of infection in cotyledons but increases in axil part with the development of the lesion (Wu, 1969). At this stage, flow of arginine and methionine from cotyledons to axil part has been found to increase. It is due to the fact that the pathogen is able to synthesize at least 15 amino acids of which DL-threonine stimulates the growth of greengram while DL-alanine, L-serine, L-cystine and DL-methionine are inhibitory (Wu, 1969).

Exposures of seed to *R. solani* increases activity of phosphorylase and glutamate dehydrogenase but later it comes to the level of control (Wu, 1973). The aldolase activity is declined while glucose 6-phosphate dehydrogenase decreases first but increases later until symptoms appear. Amylase activity is increased. It is suggested that in the early stages of the pathogenesis, an increase in reducing sugar is caused by reduction in carbohydrates metabolism while later the hexose monophosphate pathway plays a major role in carbohydrate metabolism.

The total phenols contents was reported more in healthy tissues of greengram as compared to those inoculated by *R. solani* (Arora and Bajaj, 1978). However, it resulted in appearance of two new substances while one disappeared after 24 hours of inoculation. The total phenolic contents of the infected hypocotyls of the greengram can be increased by treating it with 50 ppm ethrol (Arora and Bajaj, 1977). In the infected hypocotyl, the peroxidase activity increases at 24 hours after inoculation, decreases at 48 hours and 72 hours and again increases at 120 hours after inoculation. Polyphenoloxidase activity increases after inoculation and is correlated with the appearance of the symptoms (Arora and Bajaj, 1985). The treatment of the greengram seedlings/ hypocotyl with ethaphon, an ethylene releasing compound, is reported to enhance the peroxidase and polyphenoloxidase activity increasing resistance against the pathogen.

**Disease cycle and epidemiology:**

The epidemiology of Rhizoctonia blight may be divided into two phases, one before and the after canopy closures. The first phase is soil borne and the second is leaf borne (Yang, 1990). Though, the pathogen *R. solani* has been isolated from seeds of mungbean and urdbean, it is primarily soil borne and can survive for many years by producing sclerotia in soil and on plant tissue/seed coat. *R. solani* also survive as mycelium by colonizing soil organic matter as saprophytes, particularly as a result of plant pathogenic activity. The highest inoculum potential is noted in the top 10 cm soil and no inoculum is found below 40 cm (Kaiser et al., 1970). It germinates to produce vegetative threads (hyphae) of the fungus that can attack mungbean on to the leaves during heavy rains. The fungus is attracted to the plant by chemical stimulants released by actively growing plant cells and/or decomposing plant residues. As the attraction process proceeds, the fungal hyphae will come in contact with the plants and become attached to its external surface. After attachment, the fungus continues to grow on the external surface of the plant and will cause disease by producing a specialized infection structure that the plant cell releases nutrients for continued fungal growth and development. The infection process is promoted by the production of many different extracellular enzymes that degrade various components of plant cell wall. As the fungus kill the plant cells, the hyphae continues to grow and colonize dead tissue, often forming sclerotia. Once the floral parts are get infected, the diseases become seed borne which can lead to spread of the pathogen to infested areas. Comporata (1982) evolved a technique to measure the infectious potential of soils infested.
by *R. solani* and calculated the linear regression of disease index against log soil concentration.

The collateral weed hosts play an important role in initiation and early spread of the disease to the main host (Saksena, 1985). Because of their proximity to the soil carrying primary inoculum and their special microclimate, the weed hosts are first to take the infection and facilitate the production of the basidiospores of the pathogen. Thereafter, the pathogen become air borne to cause leaf, stem or pod infection and produces fresh crops of basidiospores on the main host. The maximum basidiospores production and their discharge occurs during dark night and early morning before sunrise (Saksena and Dwivedi, 1973). The spore production is favoured by night temperature below 24 °C, RH above 95% and rate of evaporation below 1.5 mm per day. In nature, basidiospores production starts in the mid August in north India. The basidiospores germinate in two hours and penetrate leaf through the intact surface by formation of infection cushion or direct penetration through stomatal openings. They lose their viability rapidly in dry storage and after exposure to direct sun light. In *R. solani* secondary spread via sclerotic through wind and rainsplash is also reported. Cob web like mycelial growth of pathogen also gets transmitted from diseased to healthy leaves in the web blight of legumes (Saxena, 1979; Verma and Thapliyal, 1976).

Environmental factors play a vital role in the development of web blight disease caused by *R. solani*. Higher aerial temperature (26 to 32 °C) relative humidity near 100% and soil temperature 30-33°C favoured the development of high disease severity. Rainfall (91-97 mm) had a significant role in severe development of web blight during early stage of crop in urdbean (Sharma and Thripathi, 2001; Saksena, 1985).

Host susceptibility is also important in disease incidence and severity. In case of web blight of legumes, the plant are susceptible seedling stage until maturity and the severity is maximum in 30-70 days old plants probably because of dense canopy of the crop which facilitates the early spread of the pathogen through contact of plants and leaves with one another, forming mycelial bridges.

**Disease management:**

**Host resistance:**

Plant resistance is one of the most attractive approaches in suppressing plant disease. It is not only compatible with other disease management techniques but ecofriendly also.

Resistance is a characteristic of a plant which suppresses pathogen development. The magnitude of resistance can range from very small (pathogen development is suppressed only slightly) to very large (the pathogen does not complete pathogenesis). Even resistances that do not completely prevent pathogenesis can suppress disease adequately in populations of plants. If resistance has a effect large enough to slow pathogen reproduction rates to replacement levels (new individuals produced at the same rate that old ones are removed), pathogen populations will not increase. If resistance is of small magnitude, disease can increase in the resistant plant population, and other disease management techniques are also needed along with it to suppress epidemic development adequately.

Very meagre information is available on the screening of mungbean against web blight. This is despite of the fact that a high disease incidence (85-90%) coupled with 25-30% plant mortality due to *R. solani* had been observed earlier (Saikia, 1976). No resistance could be identified in mungbean cultivars till the findings of Singh *et al.* (1989) from Faizabad, Uttar Pradesh in which of the 458 *Vigna radiata* lines tested, three lines were found immune and 67 resistant to the pathogen. Out of 85 lines of mungbean screened for resistance source, only seven lines, of mungbean showed resistance reaction regarding disease severity below 20%, 18 were moderately resistant. 28 were moderately susceptible and 32 lines showed susceptible reaction (Singh *et al.*, 2003).

**Cultural modifications to suppress the rate of disease development:**

Cultural practices, which include all manipulations necessary in crop production and that, can frequently be modified to help suppress the rate of epidemic development. Environment is closely associated not only with the plants in the field but also with the pathogen, its survival, infection and disease development. Suitable adjustment in cultural practices are modified the environment in such a way that its physical and biological component changes and become unfavourable for the pathogen and disease development. Management of plant disease through cultural practices involves the principal of avoidance, exclusion and eradication of the pathogen.

Alteration in dates of sowing and manipulation in plant spacing are such techniques to reduce the crop losses from plant diseases. The effect of dates of sowing on web blight have been studied and higher disease incidence is reported in early sown plants (July 1<sup>st</sup> and July 16<sup>th</sup>) where as least disease severity was recorded on late sown crop (16<sup>th</sup> Aug.). However, maximum grain yield and comparatively lower disease severity was recorded in plants sown on August 1<sup>st</sup> as compared to those which were sown on July 1<sup>st</sup> and July 16<sup>th</sup>, respectively (Singh, 2006). Singh (2006) reported that wider row spacing of 50 cm or more resulted in decreased web blight infection and increased yield of mungbean. Burying the infected leaves immediately after the harvest will reduce the primary infection. For proper management of disease, the diseased material should be destroyed and crop rotation be practiced. Weed hosts should be removed, soil drainage should good and soil should be well aerated. Shallow planting will also help in minimizing the infection by *R. solani* (PANS, 1981).
Biological management:

Biological control is the reduction of inoculum or disease producing activity of a pathogen accomplished by or through one or more living organism other than man (Cook and Baker, 1983). It comprises use of living agents to control plant pathogens (Mukhopadhayay, 1996). Biocontrol can be achieved by either promoting the native antagonists to reach a density sufficient to suppress a pathogen(s) or by introducing alien, antagonists. Although some of the earlier work was related to promoting indigenous antagonists by using organic amendments or other cultural practices, the recent trends is to isolate, multiply and introduce the antagonists to soil or specific court of infection to achieve a successful biological suppression of disease (Mukhopadhyay, 1996).

The basis of biological control is exploitation of the antagonistic potential of biocontrol agents. Such bioagents are known as bioagents. An antagonist is a microorganism that adversely affects another organism (e.g. target pathogen) growing in association with it (Baker and cook, 1974). In biological control of plant pathogens, antagonists or biological agents, with the potential to interfere in the life process of plant pathogen are used (Cook and Baker, 1983). The mechanism of action of an antagonist can be called as antagonism. Antagonism is the balanced wheel of nature and is fundamental to biological control (Snyder, 1961).

Antagonism (Wood and Treit, 1955) includes:

- Antibiosis, which results from the liberation of an antibiotic or other chemical by one microorganism and is harmful to the pathogen.
- Competition for some nutrients or other commodities in limited supply but needed by the pathogen.
- Predation, hyperparasitism, mycoparasitism or other forms of direct exploitation of a pathogen by other microorganism.

Mechanisms of antagonism between antagonists and R. solani:

Farnawany and Shama (1996) reported hyphal interactions between Trichoderma viride and four anastomosis groups (AG) of R. solani using a bright field microscope. Trichoderma got attached to Rhizoctonia by different means including surface colonization, growing in contact, direct penetration and formation of appressorium and node like structures. Under greenhouse conditions, incorporation of a wheat bran inoculum preparation of T. viride in pathogen infested soil at different concentrations controlled bean (Phaseolus vulgaris) damping off induced by the 4 isolates (AGs) of R. Solani. Slight infection was obtained in the case of AG 3 at all test concentration of T. viride. Good disease control was obtained at 5 g of T. viride / kg soil. Singh (2006) evaluated four isolates of T. harzianum and T. virens against Thanatephorous cucumeris (Rhizoctonia solani) causing web blight of green gram. Pantnagar isolates of T. virens and T. harzianum were superior over other.

Soil application of T. viride , T. harzianum and Gvirens significantly inhibited the mycelial growth and sclerotia production in T. cucumeris (R. solani) causal agent of web blight of horsegram (Dubey, 1998) and Groundnut (Dubey, 2000).

In vitro studies showed inhibitory effect of culture filtrate of T. viride against R. solani (Khan and Husain, 1991). Foliar application of T. virens significantly reduced web blight of greengram in glass house (Singh, 2006).

Chemical management:

Undeniably chemical application is one of the most effective and economic means to manage crop losses incurred by plant diseases. To maintain the health of crop raised from pathogen free seed on a pathogen free land, chemical protection is unavoidable in the standing crop unless true resistance in the crop is assured. Further, for certain diseases, chemical protection is inseparable from an integrated approach.
Table 1: Host range of *Rhizoctonia solani* and Rhizoctonia diseases arranged by anastomosis groups (Sneh et al., 1991)

<table>
<thead>
<tr>
<th>Anastomosis group</th>
<th>Diseases</th>
<th>Host</th>
</tr>
</thead>
<tbody>
<tr>
<td>AG1-IA</td>
<td>Sheath blight, Sheath spot</td>
<td>Rice</td>
</tr>
<tr>
<td></td>
<td>Sclerotial disease, leaf blight, banded leaf</td>
<td>Corn</td>
</tr>
<tr>
<td></td>
<td>Leaf blight, Banded leaf</td>
<td>Sorghum</td>
</tr>
<tr>
<td></td>
<td>Leaf blight</td>
<td>Bean and Soybean</td>
</tr>
<tr>
<td></td>
<td>Summer blight</td>
<td>Crimson clover</td>
</tr>
<tr>
<td></td>
<td>Southern blight</td>
<td>Camphor seedlings</td>
</tr>
<tr>
<td></td>
<td>Brown patch</td>
<td>Turfgrass</td>
</tr>
<tr>
<td>AG-1IB</td>
<td>Web blight Rot</td>
<td>Bean, Mungbean, Rice, Soybean, Figs and Leguminous woody Plant</td>
</tr>
<tr>
<td></td>
<td>Bottom rot</td>
<td>Cabbage</td>
</tr>
<tr>
<td>AG-1IC</td>
<td>Damping off</td>
<td>Buckwheat</td>
</tr>
<tr>
<td></td>
<td>Damping off and crown root rot</td>
<td>Carrot</td>
</tr>
<tr>
<td></td>
<td>Damping off</td>
<td>Soybean, Flax and Pine</td>
</tr>
<tr>
<td>AG-2-1</td>
<td>Damping off</td>
<td>Crucifers</td>
</tr>
<tr>
<td></td>
<td>Bud rot</td>
<td>Strawberry</td>
</tr>
<tr>
<td></td>
<td>Leaf blight</td>
<td>Tulip</td>
</tr>
<tr>
<td></td>
<td>Root rot</td>
<td>Japanese radish</td>
</tr>
<tr>
<td></td>
<td>Root rot</td>
<td>Subterranean clover</td>
</tr>
<tr>
<td>AG-2-2 IIB</td>
<td>False sheath blight</td>
<td>Rice</td>
</tr>
<tr>
<td></td>
<td>Sheath blight</td>
<td>Mat rush</td>
</tr>
<tr>
<td></td>
<td>Black scurf</td>
<td>Edible burdock</td>
</tr>
<tr>
<td></td>
<td>Brown patch</td>
<td>Turf grass</td>
</tr>
<tr>
<td></td>
<td>Crown and brace rot</td>
<td>Corn</td>
</tr>
<tr>
<td></td>
<td>Damping off</td>
<td>Sugar beet</td>
</tr>
<tr>
<td>AG-2-2 IV</td>
<td>Root rot and leaf blight</td>
<td>Sugar beet</td>
</tr>
<tr>
<td></td>
<td>Large patch</td>
<td>Turf grass</td>
</tr>
<tr>
<td>AG-3</td>
<td>Black scurf and stem/ stolon cankers</td>
<td>Potato</td>
</tr>
<tr>
<td></td>
<td>Target spot</td>
<td>Tobacco</td>
</tr>
<tr>
<td></td>
<td>Leaf blight</td>
<td>Tomato</td>
</tr>
<tr>
<td></td>
<td>Brown spot</td>
<td>Egg plant</td>
</tr>
<tr>
<td>AG-4(HG I, HG II and HG III)</td>
<td>Fruit rot</td>
<td>Tomato</td>
</tr>
<tr>
<td></td>
<td>Stem rot</td>
<td>Pea</td>
</tr>
<tr>
<td></td>
<td>Damping off and Stem canker</td>
<td>Potato</td>
</tr>
<tr>
<td></td>
<td>Damping off and root rot</td>
<td>Soybean, Onion, Pea, Cotton, Stevia</td>
</tr>
<tr>
<td></td>
<td>Pod rot</td>
<td>Snap bean</td>
</tr>
<tr>
<td>AG-5</td>
<td>Black scurf</td>
<td>Potato</td>
</tr>
<tr>
<td></td>
<td>Brown patch</td>
<td>Turf grass</td>
</tr>
<tr>
<td></td>
<td>Root rot</td>
<td>Beans and Soybean</td>
</tr>
<tr>
<td>AG-6</td>
<td>Non pathogenic group</td>
<td></td>
</tr>
<tr>
<td>AG-7</td>
<td>Non pathogenic group</td>
<td></td>
</tr>
<tr>
<td>AG-8</td>
<td>Bare Patches</td>
<td>Cereals</td>
</tr>
<tr>
<td>AG-9</td>
<td>Week pathogen</td>
<td></td>
</tr>
<tr>
<td>AG-10</td>
<td>Non pathogenic group</td>
<td></td>
</tr>
<tr>
<td>AG-11</td>
<td>-</td>
<td>Wheat</td>
</tr>
<tr>
<td>AG-BI</td>
<td>Non pathogenic group</td>
<td></td>
</tr>
</tbody>
</table>
of disease control.

Use of chemicals in plant disease control is aimed to (i) Create a toxic barrier between the host surface or tissue and the pathogen, and (ii) eradicate the pathogen present at a particular site on the host such as seed, foliage, roots, etc. The chemicals used in disease control oppose the germination, growth and multiplication of the pathogen or directly destroy it by toxicity.

The effect of bavistin or benlate @ 2.5 g/kg was found effective in eliminating the seed borne infection of *Trichoderma cucumeris* causing web blight of rice bean (Tiwari, 1993) and dolichos bean (Dwivedi and Dwivedi, 1999). Carbendazim and thiophanate methyl were found best controlling seedling mortality of mung bean caused by *R. solani* (Singh et al., 1995). Some fungicide viz., flutonil, penicycuran, carbendazim, tolcloks-methyl, thiophanate methyl used as seed dressing (2x3 g ai/kg seed) or as soil drench (200 and 300 p.g/ml) were found highly effective in reducing the disease incidence (Reddy et al., 1992).

In a pot study, during 1984-85, Dubey and Dwivedi (1988) demonstrated bavistin was the most effective chemical against *Thanatephorus cucumeris* on Vigna mungo, followed by dithane Z-78 (Zinb) at 0.25% and benelate (Benomyl) at 0.2%. Nineteen fungicides were evaluated in pot experiments in the greenhouse conditions and benomyl, quintazone, zinb, oxycorboxin and carboxin gave effective control of the disease. (Singh and Malhotra, 1994).

Benomyl (0.25-0.5 kg/ha) helps manage the pathogen when it is applied at first-symptoms appearance and then every 15 days. Fentin acetate (0.16 kg/ha) or fentin hydroxide (0.20 kg/ha) applied after benomyl, gives good control. Thiophanatemethyl (0.5 kg/ha), carbendazim (0.5-1.0 kg/ha), and captato1 (1.0-3.5 kg/ha) are also useful (Gálvez et al., 1989).

Captato1 @ 1.64 kg a.i./ha or oxycarboxin @ 0.16 kg a.i./ha as foliar spray has been reported to be very effective for the control of web blight of cowpea (Oyeken, 1979). Oladiran (1980) evaluated some fungicide and insecticide combinations for controlling web blight of cowpea and reported significant reduction in disease severity and an increase in grain yield occurred by spraying brestan (Triphenyltin acetate fentin acetate), benomyl + gammalin 20 and fentin acetate + gammalin 20. The web blight phase of disease could be controlled to an extent of 96% by spraying the benomyl or carbendazim 0.05% (Saikia and Phookan, 1983) Three foliar spray of bavistin (0.1%) at 15 days interval has been reported to control web blight of ricebean (Tiwari,1993) and dolichos bean (Dwivedi and Dwivedi, 1999) under field condition. Dubey and Prasad (1997) reported that bavistin and topsin-M gave the best control of the disease along with highest yield under field conditions. However, bavistin + indofil – M-45 was more economical (Rs. 3.2/Rupee return ) than the other treatments. In another study foliar spray of bavistin (0.05%) along with seed treatment with celest (0.2%), raxil (0.2%) or bavistin (0.2%) was highly effective in reducing web blight severity of French bean followed by topsin-M and baycor, significantly increase in yield of the crop was also observed (Mathew and Gupta, 1996).

The treatment combinations based on bio-agent *Gliocladium virens* (*Trichoderma virens*), karanj (*Pongania glabra*) cake, carboxin and *Rhizobium* were evaluated in integration of three methods as soil application, foliar spray and seed treatment for the management of web blight of urd (*Vigna radiata*) and mung (*Vigna radiata*) bean caused by *Thanatephorus cucumeris* (*Rhizoctonia solani*) under sick field conditions. The integration of soil application of karanj cake (2 q ha–1), seed treatment with G. *virens* (106 spores/ml/10g seed) + carboxin (1 g kg–1) + *Rhizobium* sp. (25 g kg–1 seed) and foliar spray of karanj leaf extract (10%) increased 35.7-36 % seed germination, reduced 92.4-93.7 % disease intensity and considerably enhanced plant vigour and grain yield of urd and mung bean. However, soil application of karanj cake or G. *virens* multiplied on pulse bran-saw dust-tap water (3:1:4 w/w/v), seed treatment with G *virens* + carboxin+ *Rhizobium*, and foliar spray of karanj leaf extract separately also proved effective for the management of disease, but integration of any two was more effective than any one alone (Dubey, 2003). Dubey (2007) reported that the efficacy of *Trichoderma viride* (idarip-21), *Ponganzia glabra* vent cake and leaf extract and carboxin in different combinations and modes of application in field trials for the management of web blight (*Rhizoctonia solani* Kuhn). The resulting yield of mungbean [*Vigna radiata* (L.) Wilczek] was measured. Fifty-four combinations of different treatments were applied through soil, seed and foliar spray. Integration of soil application of *P. glabra* cake (200 kg/ha), seed treatment with *T. viride* (2 g/kg seed) + carboxin (1 g kg−1) + *Rhizobium* sp (25 g/kg seed) and foliar spray of *P. glabra* leaf extract (10 %) suppressed disease severity to a significant extent (92.7 %). This treatment also increased seed germination (32.4 %), improved plant vigour and enhanced production (49.2 %). The same combination excluding carboxin was also effective and could be an option for organic production of mungbean. The integration of any two modes of applications of treatments was superior to any single mode of application.

**Literature Cited**


