SATSA Mukhapatra - Annual Technical Issue ***28*** *: 2024* ISSN 0971-975X

Major Diseases of Green Gram, Black Gram and Their Integrated Management Strategies

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(Received : January 11, 2024; Revised : January 14, 2024; Accepted : January 15, 2024)

ABSTRACT

Green gram (*Vigna radiata*) and black gram (*Vigna mungo*) are prominent pulse crops crucial for global food security and agricultural sustainability. However, these crops face substantial yield losses due to various diseases. This chaptersummarizes an extensive exploration of the diseases affecting green gram and black gram, detailing their causal agents, symptomatology, epidemiology, and integrated disease management (IDM) strategies. It aims to serve as a concise guide for stakeholders, offering insights into effective disease control measures, fostering sustainable practices, and enhancing the productivity of these essential pulse crops.

The exploration of diseases impacting green gram and black gram encompasses several key pathogens *viz.*Anthracnose (*Colletotrichum* spp.), Powdery Mildew (*Erysiphe polygoni*), Mungbean Yellow Mosaic Virus (MYMV), Cercospora Leaf Spot (*Cercosporacanescens*). The chapter further explores the epidemiology of these diseases, elucidating the environmental factors influencing their prevalence. It highlights the significance of accurate symptom diagnosis for effective disease management and emphasizes the implementation of integrated approaches.

Keywords : *Vigna radiata*, *Vigna mungo*, diseases, IDM, pulses.

Introduction

Green gram (*Vigna radiata* (L.) R. Wilczek var. *radiata*) is the third most important pulse crop after chickpea and pigeonpea, which is cultivated across more than 7 million hectares, primarily in Asia, and increasingly expanding to various other regions globally. Mungbean seeds are a rich source of nutrients, including approximately 24% easily digestible proteins, fiber, antioxidants, and various phytonutrients (Itoh *et al.*, 2006). This versatile crop is consumed in multiple forms such as whole seeds, split cooking, flour, or as sprouts, serving as a crucial dietary protein source. Green gram sprouts, particularly, contain significant quantities of thiamine, niacin, and ascorbic acid. India is the largest mung bean producer, yielding 2.17 million tons of grains from 4.32 m ha area. Still, the average productivity of mungbean in India

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is somewhat low (~502 kg/ha). This low productivity is attributed to various factors, among them biotic constraints are major. Especially, among the biotic challenges, mungbean is susceptible to major diseases such as yellow mosaic, anthracnose, powdery mildew, Cercospora leaf spot (CLS), dry root rot, halo blight, and tan spot (Nair *et al.*, 2019).

Blackgram (*Vigna mungo* (L.) Hepper), commonly known as Urdbean, holds significant agricultural importance in the Indian sub-continent. In India, it's primarily cultivated during the kharif (rainy) season in the north and central regions of the country. Furthermore, it serves as a rice fallow crop after paddy in large areas of the southern states of India during the rabi (winter) season and is also grown as a catch crop in the spring season in the northern plains of India. Black gram grown in an area of 4.63 million hectares, resulting in a total production of 2.78 million tonnes, averaging a productivity of 600 kg per hectare in India during 2020–2021, (AICRP Kharif Pulses; PC Report 2022). Urdbean is a valuable source of protein and carbohydrates and is consumed in various forms. Although it has a potential yield of more than 1.5 tonnes, its average productivity remains around 0.6 tonnes. Biotic stress poses a significant challenge affecting the crop at different growth stages.

Both mungbean and urdbean yields are influenced by various biotic factors. Among the biotic factors, some common diseases *viz.*Mungbean yellow mosaic virus (MYMV), powdery mildew, Cercospora leaf spot (CLS), anthracnose, dry root rot, leaf crinkle virus, web blight, and others are of main concern as they significantly reduce crop yields. The following sections provide details about some major diseases and their management strategies.

1. Yellow Mosaic Disease

Introduction

Yellow Mosaic Disease poses a significant challenge to mungbean and urdbean cultivation worldwide, causing substantial economic losses and impacting food security. Understanding the etiology, symptoms, disease cycle, and implementing effective management strategies are crucial in mitigating the impact of this devastating viral disease on these important leguminous crops. Continued research and concerted efforts towards developing resistant varieties and integrated disease management strategies are essential for sustainable control of YMD and ensuring stable yields of mungbean and urdbean. The yellow mosaic disease (YMD) is caused by four different begomoviruses*viz.,* (i) mungbean yellow mosaic India virus (MYMIV), (ii) mungbean yellow mosaic virus (MYMV), (iii) horsegram yellow mosaic virus (HgYMV) and (iv) dolichos yellow mosaic virus (DoYMV) which are collectively known as yellow mosaic viruses (YMVs) (Malathi and John, 2009; Naimuddin et al., 2016). The yellow mosaic disease is transmitted by a sucking insect whitefly *Bemisiatabaci* Genn (Nariani 1960). The average yield loss varies from 10 to 100%, depending on the stage at which crop is infected and the genotype cultivated (Singh, 1980; Bashir et al., 2006). The disease occurs within 2 weeks of sowing. The epidemic of the disease is increasing every year in all urdbean&mungbean cultivating areas because of its vector whitefly (*Bemisiatabaci).*

It is predominantly prevalent in states like Chhattisgarh, Delhi, Uttar Pradesh, Bihar, Haryana, Himachal Pradesh, Punjab, Odisha, Rajasthan, Tamil Nadu, Karnataka, Andhra Pradesh, and Madhya Pradesh.

Symptoms

The first symptoms include small yellow spots or patches on the green lamina of young leaves. These patches gradually develop into golden yellow mosaic or bright yellow mosaic symptoms. As the infection progresses, the yellow discoloration increases, ultimately causing complete yellowing of the leaves. This disease significantly delays the plant's maturity, decreases flower production, and hampers pod formation. Severely affected plants produce undersized and immature seeds. In some varieties, a necrotic centre can develop within the yellow spots. Pods may become small and distorted, and in severe cases, early infection can lead to plant death before seed set.

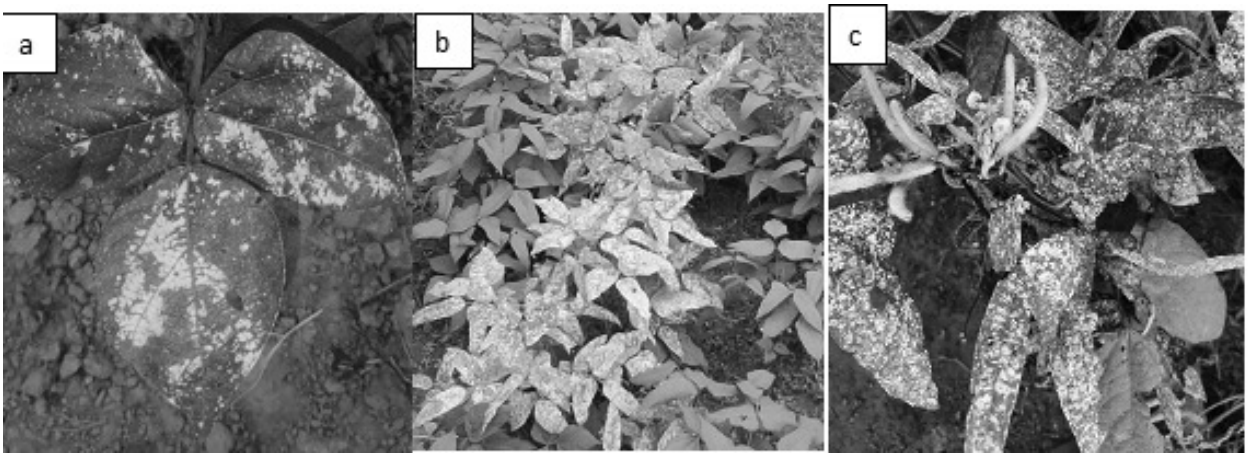

Fig. 1 : Yellow mosaic disease symptom on greengram (a) and blackgram (b,c)

Pathogen

The yellow mosaic disease of moongbean and urdbean is caused by four different viruses namely (i) mungbean yellow mosaic India virus (MYMIV), (ii) mungbean yellow mosaic virus (MYMV), (iii) horsegram yellow mosaic virus (HgYMV) and (iv) dolichos yellow mosaic virus (DoYMV) which are jointly called as yellow mosaic viruses (YMVs) belong to the genus begomoviruses and family *Geminiviridae*. They are characterized by geminate virus particles, which are paired and isometric, with a size of 15 x 30 µm. These viruses have a bipartite genome comprising of DNA-A and DNA-B, primarily found within the phloem-associated elements of infected plants.

Predisposing Conditions

Yellow mosaic disease is more prevalent in summer crops compared to rainy or winter crops. Weeds acts as a reservoir for the virus, serving as the primary source of inoculum. The disease spreads during India's monsoon period, with temperatures ranging from 31 to 35°C and relative humidity around 70%, facilitating disease progression and the multiplication of the whitefly vector, *Bemisiatabaci*.

Disease Cycle

The disease is primarily transmitted by a sucking insect i.e. whitefly, *Bemisiatabaci*, and can also be transmitted through grafting. It is not transmitted through seed or soil or sap. Disease spreads when viruliferous whiteflies feed on plants. Summer-sown crops are mostly susceptible. Weed hosts such as *Croton sparsiflorus*, *Acalypha indica*, *Eclipta alba*, and other legume hosts acts as reservoirs for the virus.

Management Strategies

- Several management strategies can be employed to control Yellow Mosaic Disease.
- Resistant Varieties : Breeding efforts have been focused on developing resistant cultivars of mungbean and urdbean against YMD. Resistant varieties play a crucial role in minimizing disease incidence and mitigating yield losses. Grow mungbean(table 1) and urdbean varieties/genotypes that exhibit resistance to the disease, such as IPM 205-7 (Virat), IPM 99-125 (Meha), PDM 139 (Samrat), IPM 2-14, IPM 312-20 (Vasudha), IPM 409-4 (Heera), IPM 410-3 (Shikha), IPM 02-3, IPM 2014-9 (Varsha), IPM 302-2 (Kanika), and urdbean varieties like IPU 02-43, IPU 94-1 (Uttara), IPU 10-26, IPU 11-02, VBN 7, VBN 4, VBN 6 among others.
- Vector Control : Implementing whitefly control measures is vital in managing YMD. Strategies such as the use of insecticidal sprays, reflective mulches, and introducing natural predators of whiteflies can help reduce vector populations. Use Yellow Sticky Traps @ 12 traps per hectare to attract and trap whiteflies.
- Crop Rotation and Sanitation : Practicing crop rotation with non-host plants and maintaining good field hygiene by removing infected plant debris can limit the buildup of viral reservoirs and reduce disease spread.
- Seed Treatment : Seed treatment with antiviral agents or hot water treatment can help in reducing the initial inoculum of the virus and prevent primary infection or with Thiamethoxam or Dimethoate/ Imidacloprid.
- Sowing Manipulation and Plant Density : Adjust sowing dates and plant population, and consider intercropping with sorghum or maize to create an environment less conducive to the vector's activity and reduce its movement.
- Rogueing Infected Plants : Remove infected plants up to 45 days to prevent further spread.
- Foliar Sprays : Apply neem formulation (3 ml/lit) at 30 days after sowing to manage the disease.

2. Cercospora Leaf Spot (Cls)

Introduction

Cercospora Leaf Spot (CLS) is a prevalent fungal disease affecting mungbean (*Vigna radiata*) and urdbean (*Vigna mungo*) which was initially reported in Delhi, India (Munjal *et al.*, 1960), and it has since become widespread throughout the country, leading to significant leaf spotting and defoliation. This disease

Table 1 Resistant Genotype(S) of mung bean against mungbean yellow mosaic disease.

Genotype(S)	Resistant level*	Country	References
NM-10-12-01	R	Thailand	Akhtar *et al.* (2009)
NM-2,VC-3960(A-88), 98-CMH-016,VC-3960(A-89),BRM-195 014043,014133, 014249, 014250	R R	Pakistan Pakistan	Bashir *et al.*(2005) Iqbal *et al.*(2011)
ML1265,ML1229	R	India	Kooner and Cheema (2007)
SML1815,MH421	R	India	Mahalingam *et al.* (2018)
BPMR-145,Vaibhav,PhuleM-2003-3,TARM-18,PhuleM-2002-13, PhuleM-2001-3,PhuleM-2002-17,PhuleM-2001	R	India	Mandhare and Suryawanshi (2008)
EC300072,K141	R	India	Manivannan *et al.* (2001)
LGG424B,LM108B	I		
VC-6960-88,VC-6773(B-G),VC-3960-89,ACC-12840014,VC-1089A	R	Bangladesh	Mondol *et al.* (2013)
NCM-15-11,AZRI-1,AZRI-06,NCM-21,14063,NCM-11-8	R	Pakistan	Munawar *et al.* (2011)
NM94	T-Odisha and Andhra Pradesh	India	Nair et al. (2017)
ML1628	T		
VRMG(g)1,LM235(GY),K851,T44,Nelambur,SonaMoong,	R	India	Pandiyan *et al.* (2007)
ML267,LGG407	R	India	Panduranga *et al.* (2011)
ML- 5, ML405, ML408, ML337, MUM2, VGG345,Pusa8773	R	India	Patel and Srivastava (1990)
ML-818	R	India	Paul *et al.* (2013)
ML-9	MR		
GG-89 andGG-39,R:TM-98-50,TM-97-55,Co-5	I	India	Salam *et al.* (2009)
IPM2-14,PDM139	R		Suman *et al.* (2015)
HUM1,HUM12,DMS03-17-2,PantMung4,Pusa9531,HUM	MR	India	

R-resistance, MR-Moderately Resistance, I-Immune, T-Tolerant
Source : Nair *et al.*, 2019

significantly impacts the yield and quality of these leguminous crops, causing economic losses to farmers worldwide. The AVRDC in Taiwan identified CLS as one of the most important diseases since its appearance therefrom 1973 (Mew *et al.*, 1975). Yield losses due to this disease varies from 50–70% (Lal *et al.*, 2001; Chand *et al.*, 2012). The disease has also been reported in other parts of the world namelyBangladesh, Indonesia, Taiwan, Thailand, Philippines, and West Malaysia (Pandey *et al.*, 2009). In India, CLS affects most mungbean-growing states, including Uttar Pradesh, Madhya Pradesh, Punjab, Haryana, Himachal Pradesh, Rajasthan, Assam, Odisha, and Maharashtra.

Symptoms

Initially, CLS appears on the lower leaves as water-soaked spots and then spreads to other aerial parts of the plant. Characteristic symptoms include numerous small leaf spots with grey centres and reddish-brown margins. Similar spots can also occur on branches, petioles, and pods. Under favourable conditions, the spots grow larger and can lead to defoliation, especially if the infection occurs during flowering and pod formation. Infected leaves may become malformed and wrinkled, leading to delayed maturity and reduced pod formation and yield. On the pods, symptoms range from a few spots to complete darkening and death of the entire pod, with seeds within the infected pod becoming shrivelled, small, and darker in colour.

Fig. 2 : Cercospora Leaf Spot disease symptom on greengram

Pathogen

Among these five species of *Cercospora, C. cruenta* and *C. canescens* Ell. & Martin is commonly observed on mungbean. The pathogen produces clusters of dark brown, septate conidiophores. The conidia are linear, hyaline, and have septa (5–6).

Predisposing Conditions

CLS mostly predominant during the rainy season and it can cause severe defoliation on urdbean and mungbean particularly at temperatures of 25 to 30°C and a high relative humidity of 90–100%. The disease is mainlyseen on leaves but it

may also infectbranches, stems, and pods. The severity of the disease depends on the temperature and humidity conditions, exclusively when coinciding with a virulent isolate of the pathogen.

Disease Cycle

The fungus survives on infected plant debris which are affected by disease and on seeds. When infected seeds grow into seedlings, they show lesions on their cotyledons. These lesions on the cotyledons serve as the primary source of inoculum for younger leaves. Under humid conditions, there is a substantial amount of sporulation. The spores are spread by the wind and rain, landing on other leaves and stems, where they start secondary infections. These secondary infections, in turn, produce more spores, which can infect additional leaves, stems, and pods during warm and wet weather. The highest dispersal of spores occurs when the dew evaporates in the morning and when rainfall begins.

Management

- Crop Rotation: Rotate mungbean and urdbean with non-host crops to break the disease cycle and reduce pathogen build-up in the soil.
- Agronomic practices such as intercropping mungbean with tall-growing cereals and millets and mulching can reduce disease incidence and increase yields.
- Sanitation: Remove and destroy infected plant debris to minimize the inoculum source.
- Proper Plant Spacing: Ensure adequate spacing between plants to promote air circulation and reduce humidity levels, creating an unfavourable environment for fungal growth.
- Fungicides: Application of fungicides, such as azoxystrobin, chlorothalonil, or mancozeb, during the early stages of infection can help manage CLS. However, rotating different classes of fungicides is essential to prevent resistance development in the pathogen population.
- Seeds treatment the with captan or thiram @ 2.5g/kg of seed.
- Two foliar sprays of Difenconazole (0.0125%), Carbendazim (12%) + Mancozeb (63%)–75WP (0.02%), Hexaconazole (5%) + Captan (70%) – 75 WP (0.05%), Radomil Gold (0.1%), or Propiconazole (0.1%) are effective in minimizing CLS incidence and severity, thereby increasing mungbean and urdbean yields. Additionally, neem leaf extract has shown promise in reducing CLS.
- On the appearance of the disease symptoms spray with mancozeb 45 WP @ 2.0 g/l or carbendazim 50 WP @1.0 g/l. Next spray should be done 10 to 15 days after the first spray, if required. Spraying with copper oxychloride @ 3 to 4 g /literof water hasalso been found effective in this disease management.
- Host plant resistance can also be effective, with mungbean varieties like IPM 312-20 (Vasudha), IPM 409-4 (Heera), IPM 205-7 (Virat), IPM 512-1 (Soorya), (Shikha), IPM 2014-9 (Varsha) and urdbean varieties IPU 11-02, IPU 10-26 showing resistance to CLS.

Exploring biocontrol agents, such as microbial antagonists or beneficial fungi, holds promise in suppressing CLS by competitively excluding or inhibiting the growth of Cercospora species.

Table 2 : Resistant Genotype(S) of mungbean against Cercospora leaf spot (CLS)

Genotype(S)	Resistant level*	Country	References
ML5,453, 443, 515,611, 610, 613,682, 713,688,735,728,746,759 and769	R	India	Singh *et al.*(2004)
PANTM103,PUSA105,PANTM3,PANTM2, ML613,ML173,ML561,ML347,PDM11 andPANTM4	R	India	Marappa (2008)
ML1464,ML1486,ML1194andML1349 GM-02-08,GM-03-03andGM-02-13 LGG-460	R R HR	India India	Kaur *et al.* (2011) Yadav *et al.* (2014b)
ML-5,HUM-9,ML-4,HUM-4,SM-9-124, HUM-1,LGG-450,andSM-9-107	R	India	Singh and Singh(2014)
1224-52and 12404	HR	India	Zhimo *et al.*(2013)
AKM9910,ML1299,IPM02-5,andSML668 (2014)	R	India	Akhtar *et al.*
KMP-13	MR	India	Bhaskar (2017)
Powderymildew			
BPMR-145, TARM-18, Vaibhav, Phule M-2002-13,PhuleM-2003-3,PhuleM-2001-3, PhuleM-2001-5 and PhuleM-2002-17	R	India	Mandhare and Suryawanshi (2008)
TARM-18	R	India	Sujatha *et al.* (2011)
LGG-460	R	India	Yadav *et al.* (2014a)
BL849,BL865,LM1668,PMB63 andAKM8803	HR	India	Ramakrishnanand Savithramma (2014)
KGS83,Pusa572,MH96-1,GS33-5,GS21-5, AKM99-4,COGG936,TMB47,ML1299, MH429,HUM1,MH429andMH530	HR	India	Akhtar *et al.* (2014)
C1-34-23,C1-32-22,C1-37-23,C1-28-20, C1-38-27,C1-44-31,C1-175-111,C1-41-28, C1-246-159,C1-236-152,C1-275-177	HR	India	Kumar *et al.* (2017)
KMP-36,KMP39andKMP41	HR	India	Bhaskar (2017)

R-resistance, HR- Highly Resistance MR-Moderately Resistance, I-Immune, T-Tolerant
Source : Nair et al., 2019

3. Powdery Mildew

Introduction

Powdery mildew poses an important challenge to urdbean and mung bean cultivation, and it has recently become a limiting factor in Rabi-sown urdbean and mung bean crops in Odisha, Karnataka, Andhra Pradesh, and Tamil Nadu. This disease has been reported in various countries, including India, Sri Lanka, Philippines, Australia, Colombia, Taiwan Korea, Ethiopia, Thailand, and the USA. It causes ayield lossvaries from 9% to 50% in farmers' fields (AVRDC, 1982).

Symptoms

Powdery mildew infects on all above-ground plant parts. Initially, white powdery patches appear on leaves and other green areas, which later become dull in colour. These patches steadily grow larger and circular, ultimately covering the lower leaf surfaces. Under severe infections, both the leaf surfaces are completely covered by a whitish powdery growth. The powdery mass contains conidia and mycelium, which in due course turn dirty white. Heavilyinfectedplant parts become distorted and shrivelled. Severe infections lead to yellowing of the foliage, causing premature defoliation. The disease also forces the premature maturity of infected plants, resulting in substantial yield losses.

Pathogen

It is caused by *Erysiphae polygoni*, which is an obligate parasite ectophytic fungus that spreads on the leaf's surface, sending haustoria into the epidermal cells to obtain nutrients. Conidiophores emerge vertically from the leaf surface, carrying conidia in short chains. Conidia are thin-walled, hyaline, and typically elliptical, barrel-shaped, consisting of a single cell. Later in the season, cleistothecia appear as small, black, globose structures with myceloid appendages.

Predisposing Conditions

The pathogen thrives in dry weather with a temperature range (22 to 26°C) and a relative humidity (80–85%). The disease is more severe in older plants than in those that are 40 days old.

Disease Cycle

The fungus is an obligate parasite and survives as cleistothecia in infected plant debris. Primary infection usually occurs from ascospores that develop in cleistothecia. The pathogen has a wide host range and it can survive in oidial form on various hosts, including *Lathyrus sativa, Vicia faba, Vigna ungulate*, etc., during the offseason, it may act as reservoirs of inoculum. The disease is also spread by air-borne conidia, and rain splash eases its spread.

Management

- Crop rotation: Avoid planting green gram or black gram in the same field consecutively to reduce the build-up of fungal spores in the soil.
- Timely planting: Optimal sowing time can help avoid the peak period of disease incidence.
- Weed management: Remove weeds that may act as alternative hosts for the powdery mildew fungi.
- Developing and cultivating resistant varieties through breeding programs is an effective long-term strategy to combat powdery mildew. These varieties possess genetic traits that make them less susceptible to the disease.

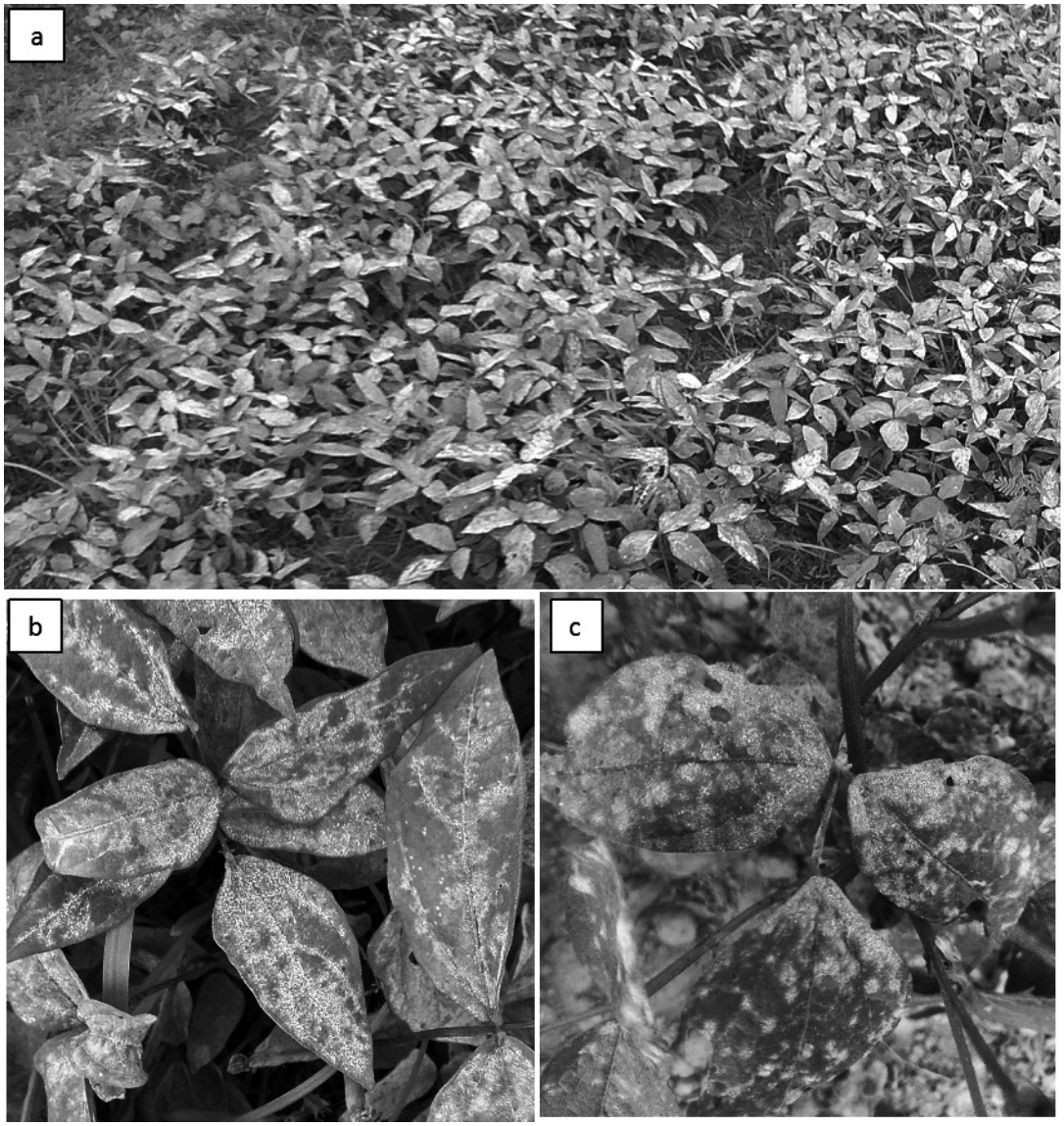

Fig. 3 : Powdery mildew disease symptom on blackgram (a,b) and greengram (c)

- The most effective method to reduce disease incidence is to cultivate powdery mildew-resistant varieties/ genotypes, such as TARM-1, TARM-2, TARM-18, COG-4, Pusa-105, Kamdev ML-131, ML-1299, and LGG-460.
- Application of fungicides, especially during the early stages of infection, can help manage the disease. However, their use should be strategic to prevent the development of resistance in the fungal population and minimize environmental impact.
- Foliar sprays of Carbendazim (12%) + Mancozeb (63%) –75WP @ 0.2% or other recommended fungicides at the onset of the disease and repeated 15 days later have been found effective in

controlling the disease under field conditions.

- Additionally, spraying with neem-based products i.e. Neem oil 3% or NSKE 5% twice at 10 days interval from initial disease appearance.
- Spray wettable sulphur 1500g/ha or Propiconazole 500 ml/ha or Carbendazim 500 g/ha or Karathane at initiation of the disease and 10 days later.
- Employing biological control agents, such as certain beneficial fungi or bacteria, can aid in suppressing the growth and spread of powdery mildew.
- Regular scouting and monitoring of crops for early detection of powdery mildew symptoms.
- Proper sanitation practices, including the removal and destruction of infected plant debris, to reduce the overwintering sources of the pathogen.
- Improving air circulation and reducing plant crowding to create an environment less conducive to powdery mildew development.

4. Anthracnose

Introduction

Anthracnose, caused by *Colletotricum truncatum* (Schw.) Andrus and Moore, is a significant seed-borne fungal disease in mungbean and urdbean leading to considerable reductions in yield and quality of seed. It causes a yield lossvariesfrom 24% and 67%. The severity of disease depends on the cultivar, infection stage, and environmental factors.

Symptoms

Anthracnose can appear on all above-ground plant parts and at any stage of growth. It often appears on the leaves ventral surface and is characterized by circular, black, sunken spots with dark centres and bright red-orange margins on leaves and pods. In case of severe infestation, affected plant parts wither. Seedlings can be blighted if infection occurs just after emergence of seedling.

Pathogen

Infected mungbean seeds are associated with different species of Colletotrichum, including *C. lindemuthianum, C. truncatum, C. dematium,* and *C. graminicola.* The pathogen mycelium is branched, septate, and hyaline. Conidia are produced in acervuli, arising from the stroma under the epidermis, which later rupture to become erumpent. The conidiophores are short, hyaline and bearing cylindrical or oblong, thin-walled, hyaline, single-celled conidia with oil globules. The sexual stage of the fungus produces perithecia with a limited number of asci, each containing characteristically 8 ascospores, which are either one/two-celled with a central oil globule.

Predisposing Conditions

The disease incidence is negatively correlated with minimum and maximum temperatures and positively correlated with rainfall and RH. The highest disease scores are observed at RH of 91–100% and temperature of 26–29°C, with low rainfall. Early sowing during June is more susceptible to the disease compare to late sowing.

Disease Cycle

The fungus is seed-borne in nature and it causes primary infection through seed. It also survives in infected plant debris in the soil. Secondary spread occurs through air-borne conidia produced on infected plant parts. Spattering rains, aided by wind currents, are the main sources of local pathogen dissemination. The pathogen can infect various leguminous hosts, which may also act as reservoirs of the primary inoculum.

Management

- Remove crop debris from the field after harvesting.
- Treat the seed with hot water at 58°C for 15 minutes to minimize seed-borne infection.
- Cultivation of mungbeanvariety such as IPM 512-1 (Soorya) and urdbeanvarieties *viz.*IPU 11-02, IPU 10-26 that show high resistance to anthracnose.
- Treat the seed with fungicides such as Carbendazim or Thiram+Carbendazim@ 2g/kg of seed.
- Foliar sprays of Carbendazim 0.5g/litor Mancozeb 2g/litat the onset of symptoms and repeated 15 days later can help in reduction of disease incidence.

5. Urd bean Leaf Crinkle Disease (ULCD)

Introduction

Leaf crinkle, which is induced by the urdbean leaf crinkle virus (ULCV), is a significant constraint in the successful cultivation of urdbean in India.

ULCD was for the first time reported from Delhi during 1966 by Williams et al. (1968) and in the Tarai region of the then Uttar Pradesh in 1967 by Nene et al. 1968(Kamaal *et al.*, 2023). It has the potential to cause up to 100% yield losses depending upon the variety and the season (Malathi *et al.* 2008, Reddy *et al.*, 2005) and it can even result in complete seed production failure in plants infected at an early stage.

Symptoms

Typical symptoms of ULCD encompass abnormal enlargement of leaves, often described as curling, crinkling, or rugosity (Williams *et al.*, 1968, Nene *et al.*, 1968). This disease affects both the reproductive and vegetative plant parts, frequently leading to a complete loss of productivity. In field conditions, the initial noticeable symptom usually appears in the third trifoliate, exhibiting a light green colour with slight expansion of leaflets and raised inter-venal areas. Subsequent leaves display malformations, characterized by increased size and a crinkled lamina surface. The leaf lamina may grow to one and a half times the size of healthy ones. Shortened internodes often result in the clumping of apical growth. Crinkled plants experience delayed flowering, and their inflorescences appear bushy due to clumping. Malformations of floral parts are not uncommon, and reduced pod setting in affected plants is linked to ULCD's adverse impact on pollen fertility(Kamaal et al 2023). Flowering is delayed, and when inflorescences do form, they often contain malformed, small flower buds that fail to open. Pollen fertility and pod formation are significantly reduced in infected plants, leading to a substantial decrease in yield.

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Fig. 4 : Leaf Crinkle disease symptom on blackgram

Pathogen

The 'Urdbean leaf Crinkle disease is caused by the Leaf Crinkle Virus. The virus particles are roughly spherical and measure approximately 25-30 nanometers in size. Studies have reported that ULCV particles purified from urdbean were 32 nanometers in diameter. In sections of urdbean cells, particles typically measured between 25 and 30 nanometers in diameter. The A260 280 /A ratio was found to be 1.51.

Transmission

ULCV can be spreadviaseveral means, *viz.*grafting, insect vectors, sap, and seeds. Studies have reported that it can be seed-transmitted to some extent, up to 15% in mungbean. Several insects, such as aphids (*Aphis gossypii* and *A. craccivora*), whiteflies (*Bemisiatabaci*), and beetles (*Henosepilachna dodecastigma*) have been identified as vectors for the virus.

Predisposing Conditions

Several factors can predispose plants to the disease. The presence of weed hosts like *Aristolochia bracteata* and *Digera arvensis*can facilitate the spread of the virus. Kharif season crops are highly susceptible. Continuous cropping of other legume crops that also harbour the virus contributes to its prevalence. The maximum disease incidence (DI) was found during kharif season when the minimum and maximum temperatures were around

25 æ%C ± 2 æ%C, and 35 æ%C ± 2 æ%C respectively, and with a morning and evening RH of >70% and >50%, respectively (Kamaal *et al.*, 2023).

Disease Cycle

The virus is seed-borne in nature and primary infection occurs by infected seeds and presence of weed hosts like *Aristolochia bracteolata* and *Digera arvensis.* Kharif season crop and continuous cropping of other legumes, i.e., Cowpea, Pigeon pea, Taparybean (*Vigna aconitifolicus*) (Kolte and Nene, 1975; Beniwal, 1983) serve as source of inoculum. White fly, beetle, and aphids help in the secondary spread of this virus.

Management Diseases

1. Use of disease-free seeds is recommended to prevent early infection
2. Removing of all collateral weed hosts from the field, and rouging out the infected plants to reduce the further spread of viruses.
3. Treat the seed with hot water at 52°C for 20–30 minutes is effective in reducing seed-borne infection.
4. Cultivating urdbean varieties which are resistant to the virus has also been successful in decreasing disease incidence.
5. Foliar spray of Thiomethaxam@ 0.02% at 15 days after sowing can reduce whitefly populations and the incidence of leaf crinkle (Ganapathy and Karuppiah, 2004).
6. Additionally, using NSKE@ 2% has been reported to reduce the severity of ULCV infection (Binyamin et al., 2011), and the application of nitrogen, potash,phosphorus, boron, zinc, and naphthalene acetic acid (NAA) has shown effectiveness in reducing ULCV infection in different urdbean varieties (Zeshan *et al.*, 2012).

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