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COLLAR ROT OF CHICKPEA INCITED BY *SCLEROTIUM ROLFSII* AND THEIR INTEGRATED MANAGEMENT: A REVIEW

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ABSTRACT

Chickpea collar rot is caused by the soil-borne polyphagous pathogen *Sclerotium rolfsii* Sacc. It affects around 500 plant species, including tomato, chilli, lentil, brinjal, soybean, maize, groundnut, bean etc. It causes a variety of diseases, such as collar rot, stem rot, foot rot, charcoal rot, seedling blight, damping-off, stem blight, and root rot. According to reports, the mortality rate of chickpea seedlings caused by *Sclerotium rolfsii* ranges from 54.5% to 95.5%. The surface of the affected plant in the collar region exhibits obvious symptoms of the collar rot disease and it is covered in a white mycelium growth. *Sclerotium rolfsii* pathogen mycelium spreads quickly and can live in soil for long periods of time as sclerotia. *Sclerotium rolfsii* management is to limit crop losses, reduce the amount of Sclerotia, and minimize inoculum. Disease suppression by biocontrol agents such as *Trichoderma harzianum*, *Trichoderma viride*, FYM, and Vermicompost is a long-term manifestation of interactions between the plant, pathogen, biocontrol agent, and the microbial community on the plant and in the physical environment, which significantly inhibited the growth of the *Sclerotium rolfsii* pathogen. *Sclerotium rolfsii* can be effectively managed by chemical control when seeds are treated with 70% captan+5% hexaconazole, 5% propiconazole, 37.5% thiram and 70% propineb. Integrated control, which entails combining suitable systems of control measures for efficient disease management from profitability to food and environmental safety, is an increasingly popular strategy for preventing chickpea collar rot.

Keywords : Chickpea, Disease, *Sclerotium rolfsii*, Integrated, management

Introduction

Chickpea (*Cicer arietinum* L.) is the most important pulse crop broadly grown in India and accounts for almost 75 percent of the total pulse production in the world (Keote *et al.*, 2019). It is grown on all five continents in more than 45 countries. In poor nations, chickpeas offer people an excellent source of protein. In industrialised nations, many view it as a nutritious food. In South Asian nations, green chickpea leaves or twigs are utilised to prepare a nutrient-dense vegetable. Chickpea is used for both human consumption as well as animal feeds. Chickpea is the excellent source of energy, protein, minerals, vitamins, fiber, and also contains healthy

phytochemicals (Wood and Grusak, 2007). Total area of chickpea in India 9.85 million hectares (11.99 million tones production) in which Maharashtra have highest area 2.15 million hectares (2.37 million tones production) followed by Rajasthan 2.11 million hectares (2.32 million tones production), Madhya Pradesh 2.10 million tones (3.13 million tones production) and Uttar Pradesh 0.61 million tones (0.84 million tones production). The most important states growing chickpea are Maharashtra 21.78 % to all India, Rajasthan 21.45 %, Madhya Pradesh 21.35 %, Gujarat 8.29 % and Uttar Pradesh 6.20% to all India. (Average estimation 2020-21) (Source-Directorate of Economic and Statistics, DAC&FW). Chickpea crop

suffer from various diseases like Wilt, Root rot, Collar rot, stem rot, Aschochyta blight etc. which cause serious losses in yields. Among them collar rot disease caused by *Sclerotium rolfsii* Sacc., have become more important in recent years due to drastic climate change which makes the pathogen more aggressive and increased with adaptability to the environment (Ghatak and Ansar, 2017; Kumar *et al.*, 2017; Savary *et al.*, 2011). The fungus placed in the form of genus *Sclerotium rolfsii* by Saccardo (1913). Collar rot is an emerging threat to chickpea production due to drastic climate change (Pandeet *et al.*, 2010). *S. rolfsii* is a soil borne pathogen generally found in tropical and subtropical regions of the world (Sumi *et al.*, 2018). This fungus is facultative parasite, and omnith pathogenic organism and has wide host range with prolific growth and ability to produce persistent sclerotia to inflict large economic losses (Ramesh *et al.*, 2014). They infect over 500 plant species including tomato, cucumber, brinjal, soybean, maize, peanut, bean, watermelon, and others. It causes a variety of diseases in many commercially valuable crops, including collar rot, stem rot, charcoal rot, seedling blight, damping-off, stem blight, and root rot. Mortality of chickpea seedling due to *S. rolfsii* has been reported to from 54.7 to 95.00% (Shrivastava *et al.*, 1984). It was initially noticed on tomato plants by Peter Henry Rolfs in 1892, with 70% losses. The hyphae developed upward on the surface of the infected plant and were dispersed inside and outside of the diseased stem at the soil level, covered with a cottony, white mass of mycelium. In the beginning, the fungus formed numerous little rounds, white sclerotia of uniform size, and when mature, it has a dark brown to black colour (Kwon and Park, 2002). The Pathogen *S. rolfsii* requires warm climates, occurs more frequently at high moistures and high temperatures (Al-Askar *et al.*, 2013). *Sclerotium rolfsii* is the cause of sclerotium blight in soybean lowers crop output, however in some cases, monoculture or brief rotation of soybean with another pathogen-susceptible plant can also result in notable yield losses. The wind, water, animals, and soil all disperse it. Considering the importance of commercial crops, management is required. Chemical, biological, sun therapy, and combined methods can all be used to control it. Other methods, aside from fungicidal management, are environmentally benign and safer.

Morphology of *Sclerotium rolfsii*

S. rolfsii produces a large amount of fluffy, white, septate mycelium that branches and spreads like a fan. The clamp connections are limited to the main hyphae. On the mycelium, little white tufts developed into hard,

smooth, dark brown sclerotia. When mature, sclerotia resembles a mustard seed and might have an irregular or rounded shape. (Taubenhaus, 1919; Barnett and Hunter 1972; Mahmood *et al.*, 1976; Boonthong and Sommart 1985; Harinath Naidu, 2000 and Mohan *et al.*, 2000). SEM was used by a number of researchers to examine the structure of sclerotia. The three layers that comprise each sclerotium are the inner medulla, middle cortex, and outer rind. Flora Zarani and Christias (1997) explored the sclerotial development stages, namely sclerotial beginning, development, and maturity. There have been reports of sclerotial sizes ranging from 0.1 mm to 3.0 mm. (Om Prakash and Singh, 1976; Ansari and Agnihotri, 2000 and Anahosur, 2001). The fungus was assigned the genus *Sclerotium* because to its small, round, tan to dark brown or black sclerotia those have separate rind cortex and medulla within (Punja and Rahe, 1992). Tripathi and Khare (2006) reported the PDA media showed the greatest radial development of *S. rolfsii* 4–7 days after inoculation, followed by chickpea meal agar, rice meal agar, and Richard's agar. Akram *et al.* (2007) reported variation in colony form, mycelial development rate, colony colour; sclerotial production, quantity and size of sclerotia were observed among *S. rolfsii* chickpea isolates taken from 12 distinct regions. Ansari and Agnihotri (2000) investigated the differences in morphology, physiology, and pathological between isolates of the soybean pathogen *S. rolfsii*. On the basis of the morphological characteristics of the sclerotia and their arrangement on semi-synthetic media, 44 isolates of *S. rolfsii* were divided into various groups.

Diversity within the Population of Pathogens

A lot of research has been conducted to clarify the variations in *Sclerotium rolfsii* appearance, physiology, and pathogenicity. Hernandez and Ysla (1997) evaluated cultural and morphological characteristics in eight *Sclerotium rolfsii* isolate and found variability in their mycelial growth rate, number and diameter of the sclerotia, mycelial density, the presence of rhizomorphs and duration of sclerotial formation. Almeida *et al.* (2001) revealed differences in the quantity, size, and positioning of Sclerotia on the medium surface between the *S. rolfsii* isolates that were isolated from Brazil. Sarma *et al.* (2002) found that there were differences in the colony morphology, mycelial growth, sclerotial formation, sclerotial size and colour, and other characteristics among the 26 isolates of *S. rolfsii* that were obtained from different hosts, soil samples, and locations. Bagwan (2011) When 59 *S. rolfsii* isolates were examined for variability, the findings showed that, of the 59 isolates,

the colonies of 35 isolates were fluffy and the colonies of 24 isolates were compact. Naresh *et al.* (2017) the variability of ten *S. rolfii* isolates collected from different region of chilli host. These isolates differed in terms of colony diameter, number of sclerotia, colony character (colony appearance and colony colour) and sclerotial behaviour. Out of 10 isolates of *S. rolfii*, the SR1 isolation had the highest mycelial growth of 88.0 mm, while the SR6 isolate had the lowest mycelial growth of 79.0 mm.

Symptomatology of *Sclerotium rolfii*

A drying of plants whose foliage turns half yellow before dying and spreads throughout the field is indicative of an infestation of chickpea collar rot. Affected seedlings turn yellow; younger seedlings may collapse, but older plants may dry out without doing so. The seedlings exhibit rotting in the collar area and downward when they are uprooted. Mycelial filaments with a yellowish tint cover the degraded area. Nearly all of the diseased and wilted plants with collar rot exhibit white to black mustard-colored sclerotia and white mycelial development. (Kumar and Venkatesh, 2013). Rapeseed-like sclerotia can be visible in the

early stages of infection when afflicted seedlings are plucked from moist soil (Nene *et al.* 2012). Koike *et al.* (2004) observed the pathogen *S. rolfii* was to produce damping off or stem rot in infected plants by sclerotial germination, which measured 1–3 mm and produced a mustard-like look on the surface of damaged plant sections, as well as white cottony mycelium. Prasad *et al.* (2008) *Sclerotium rolfii* was shown to develop numerous tiny sclerotia on all affected tissues and even on adjacent soil. Affected seedlings turn yellow and can be readily uprooted. Saccardo (1913) classified the fungus as belonging to the genus *Sclerotium rolfii* because it produces sterile mycelia and differentiated sclerotia. Kalmesh and Gurjar (2001) described the symptom of chilli root rot caused by *S. rolfii*. In chilli growing locations, high mortality of chilli plants was recorded between March and April. Mature chilli plants from a standing crop collapsed and dried out suddenly. A close investigation of the sick plants revealed significant cracks around the collar region. On the surface of the newly infected area, the roots were torn and sickly and covered in a thick layer of white mycelium growth.



Fig. 1: Symptom and growth of Mycelium

Disease Cycle

Hyphalite proliferation from infected tissues and germinating sclerotia resumes throughout the rainy season. Infection is facilitated by wounds, but hyphae and germinated sclerotia directly penetrate susceptible hosts. If environmental factors such as temperature, humidity, and others are favourable, the virus may infect a vulnerable host plant (Mishra *et al.*, 2021). The *Sclerotia rolfii* fungus can proliferate readily in dirt that is adhered to shoes, hand tools, car tyres, machinery, or splattering water. During transportation,

sclerotic in plant material or soil is used for long-distance transfer (Mullen J, 2001). When the weather is favourable, sclerotia can continue their activity by hyphal germination or eruptive processes. Aggregates of white mycelium emerge out of the sclerotial rind when germination occurs erratically. This kind of germination can occur without the need for an outside food source. Sclerotic must be stimulated by dry circumstances or volatile substances in order to germinate eruptively (Punja and Grogan 1981). Hyphalization of sclerotia can occur more than once. Individual hyphae from sclerotia grow in response to

exogenous nutrition availability (Punja and Damiani, 1996).

Epidemiology

When there are no living plants present, the pathogen survives as Sclerotia or mycelium on infected plant detritus. The pathogen causes black, round or oval-shaped sclerotia on affected plants. The presence of dead organic debris near plants that are sensitive to fungal infection increases that likelihood. Between 25 and 30 degrees Celsius is when the fungus grows and produces sclerotia at its best. A water-saturated soil is necessary for hyphal growth and sclerotia germination in addition to warmth; high humidity also promotes fungal development. Soil pH below 7 and bright, well-aerated, well-drained soil are favourable for sclerotia formation and survival. According to Zapeet *et al.* (2013), the pathogen could not develop and create sclerotia at temperatures as low as 10°C and as high as 40°C. The ideal temperature range for *S. rolfsii* superior radial growth was 25 to 35°C, while the ideal temperature range for sclerotial formation was 20 to 30°C.

Disease Management Strategies

The majority of the control strategies discussed seek to eliminate plant waste, lower the amount of sclerotia in the soil, and lessen the amount of time the inoculum comes into contact with the host.

Management through Host Plant Resistance

The least expensive, safest, and most efficient way to manage illness is to utilise resistant cultivars. Amule *et al.* (2014) 88 Desi chickpea genotypes were investigated among them GNG 1958 was found resistant to disease whereas, 13 entries viz., NDG 9-21, PG 97030, BG 3004, JG 14-11, H 04-68, PG 054, BGD 1058, GJG 0724, RSG 931, JG1307, GJG 0504, JG 14-110, H05-24 were found moderately resistant. Akram *et al.* (2008) 98 chickpea germplasm accessions were tested for chickpea collar rot. Out of the 98 germplasm samples, only 5 genotypes (FLIP 97-132C, FLIP 97-85C, FLIP 98-53C, ILC-5263 and NSC 9905) demonstrated a highly resistant response to the disease. The remaining 9 genotypes (FLIP 96-153C, FLIP 97-129C, FLIP 97-172C, FLIP 98-185C, FLIP 98-227C, FLIP 98-107C, FLIP 98-230C, ILC-182 and NCS 9903) displayed a moderately resistant to tolerant response, and the remaining genotypes were very susceptible to this disease. Shirsole *et al.* (2017) He tested 112 desi and kabuli chickpea entries and reported that all of the chickpea entries were prone to very susceptible to collar rot disease. None of the entries were either resistant or slightly resistant to

collar rot. Singh *et al.* (2012) screened 50 chickpea germplasm against collar rot caused by *Sclerotium rolfsii* and they found that only 3 germplasm lines viz., KG-1226, KG-8, B-321 and B-311 were showed moderately resistant reaction. Gupta and Anita (2006) 233 desi and Kabuli chickpea genotypes and 38 promising line susceptible checks JG 62 were screened against dry root rot, vascular wilt and collar rot in a multiple disease sick plot at Jabalpur, M.P. They found that two genotypes, HK 00297 and PG 97-313, and three genotypes, H 99-264, PG 9425-5, and PG-9425-9, showed resistance against all three pathogens. Hussain *et al.* (2005) investigated 57 varieties of chickpea germplasm in a greenhouse to find the source of resistance to chickpea collar rot. One genotype, FLIP 97-174C, was found to be very resistant, whereas five genotypes, FLIP 00-55C, ICC-4936, ICC-13051, ICC-12961, and ICC-14911, were found to be resistant. Twenty genotypes were found to be moderately resistant, with the other genotypes highly sensitive to susceptible.

Management Through Bioagents

Using disease-suppressive microorganisms to enhance crop health is known as bio-agent. The persistent manifestation of interactions between the plant, pathogen, biocontrol agent, and the surrounding microbial population is known as disease suppression by bioagents. Simply put, "Biological Control" refers to the application of any organism by humans for the control of pathogens. Any method of disease control or pathogen reduction that depends on biological systems or organisms other than humans is referred to as biological control in its broadest sense (Campbell, 1989). The following definitions have been advanced to illustrate the concept of biological control: "Biological control is the reduction of inoculum density or disease producing activities of a pathogen or parasite in its active or dormant state, by one or more organism, accomplished naturally or through manipulation of environment, host or antagonist or by introduction of one or more antagonists" (Baker and Cook, 1974).

Javaid and Ali (2016) used the dual culture method to investigate the effects of four species of *Trichoderma* (*T. viride*, *T. harzianum*, *T. koningii*, and *T. pseudokoningii*) on *Sclerotium rolfsii*-caused chickpea collar rot. *Trichoderma* isolates exhibited a 40–68% growth reduction against pathogens in an antagonistic manner. *T. viride* demonstrated the most antagonistic behaviour, followed by *T. harzianum*, producing a 68% and 57% reduction in pathogen development, respectively. *T. viride*, *T. harzianum*, *T.*

virens, *T. asperellum*, and *T. atroviride* were tested against collar rot of chickpea using a dual culture method, and *T. asperellum* inhibited the most mycelial growth while *T. harzianum* inhibited the most sclerotial production. Amin *et al.* (2010) six isolates of *Trichoderma* spp. were investigated for their ability to cause tomato collar rot caused by *S. rolfsii*. When compared to the control, *T. viride* (Tv-1) exhibited the greatest reduction of *Sclerotium rolfsii* mycelial growth (67.91%). Singh *et al.* (2016) *Trichoderma harzianum*-1432 (42.2%) and *Trichoderma atroviride* (40.3%) were shown to be potent antagonists when the antagonistic capacity of the *Trichoderma* strain against chickpea collar rot was examined by the cultural filtrate. Bhuvaneswari *et al.* (2019) examined six *Trichoderma* spp. isolates. In order to combat the collar rot of chillies produced by *S. rolfsii*, viz., *T. ovalisporum*, *T. harzianum*, *T. koningiopsis*, *asperellum*, *T. atroviride*, and *T. hypocrea* were employed. It was found that *Trichoderma harzianum* inhibited 16.08% mycelia growth at a concentration of 7.5% and 41.57% mycelia growth at a concentration of 15%. Using a non-volatile method, *T. hypocrea* and *T. atroviride* are ineffective against the pathogen. Kushwaha *et al.* (2018) The effectiveness of the three biocontrol agents *T. viride*, *T. virens*, and *T. harzianum* against *S. rolfsii* caused lentil collar rot was assessed. Using the culture filtrate method, it was discovered that these drugs reduced the pathogen's radial growth by 57.46 and 49.62% at 15% concentration as well as sclerotial formation by 98.20 and 99.83%. Radwan *et al.* (2006) found that *Trichoderma hamatum* and *Trichoderma harzianum* were the most effective in suppressing the growth of *Sclerotium rolfsii* mycelial by 79%. Rekha (2012) revealed that *S. rolfsii* mycelial development and sclerotia production were suppressed by *Trichoderma* sp. Samsuzzaman *et al.* (2012) findings showed that *Trichoderma harzianum* increased tomato height and production and decreased mortality in tomato plants artificially inoculated with *S. rolfsii* in soil, suggesting that bio agents, as opposed to chemical control, inhibit the growth of *S. rolfsii*, which causes tomato collar rot disease, with no risk of environmental pollution.

Chemical Management

In agricultural crops, fungicide or chemical management is an efficient way to manage certain soil-borne illnesses. Since soil-borne plant pathogens might be more difficult to eradicate with non-chemical alternatives, they can also take longer. Different kinds of chemicals have proved effective in managing the main illnesses that affect crops grown for commercial markets. Prabhu (2003) examined various fungicides in

vitro against *Sclerotium rolfsii*-caused soybean collar rot and found that carboxin inhibited mycelium development by 100%, followed by carbendazim (63%) + mancozeb (12%), and propiconazole at all dosages. Carbamazepine showed the least amount of inhibition at 0.05%. Thakur *et al.* (2002) Under pot culture conditions, the efficacy of many fungicides, including carbendazim, thiram, benomyl, captan, prochloraz, and mancozeb, against chickpea collar rot was examined. The colony diameter decreased to 1.09 cm, 2.14 cm, and 2.77 cm, respectively, with the use of carbendazim, benomyl, and captan, which was found to be significantly more effective than the other fungicides (compared to 8.60 cm in the control). The lowest rates of collar rot infection were likewise observed with these fungicides (11.0, 22.0, and 27.6%, respectively). Shirsole *et al.* (2019) Using the poisoned food approach, the effectiveness of seven systemic, four non-systemic, and six combination fungicides against *S. rolfsii* was investigated in vitro at different concentrations of 20, 50, 100, 200 and 500 ppm. The non-systemic fungicides mancozeb 75% WP, thiram 75% WS and propineb 70% WP were found to be inhibitive only at higher concentrations (100 ppm) against *S. rolfsii*, while systemic fungicides like hexaconazole 5% EC, propiconazole 25% EC, and combo products like tubaconazole 50%+crifloxystrobin 25% WG, Captan 70%+hexaconazole 5% WP, propiconazole 13%+difenoconazol, and carboxin 37.5%+thiram 37.5% were found to be completely inhibitive at all concentrations tested. Ahsan *et al.* (2018) Propiconazole, hexaconazole, bavistin, topsin M, and vitavax were the five fungicides whose efficacy against *S. rolfsii* was examined in vitro at concentrations of 100, 250, and 500 ppm. They demonstrated that *S. rolfsii* growth was completely repressed in vitro by propiconazole, hexaconazole, and vitavax, while growth was reduced by 79.52 and 71.78%, respectively, at 500 ppm by bavistin and topsin M. Kumar *et al.* (2011) evaluated four fungicides through poison food technique under *in vitro* condition @500, 1000, and 2000 ppm against collar rot of Chilli caused by *S. rolfsii*. They reported that all fungicides tested, inhibited mycelia growth of pathogen. However, average mycelia growth inhibition recorded in carboxin (98.14%), propineb (66.94%), copper oxychloride (50.50%) and carbendazim (43.94%). Yaqub and Shahzad (2006) shown that *Sclerotium rolfsii* was successfully suppressed by six fungicides: benomyl, sancozeb, thiovit, dithane M-45, carbendazim, and topsin-M. At low concentrations, no fungicide was able to stop *Sclerotium rolfsii* from growing, but at high concentrations, sancozeb and

dithane M-45 greatly slowed down the fungus's ability to proliferate. Khan and Javaid (2015) found that, when used in vitro, the four fungicides tegula (tebuconazole), thiophanate methyl, ridomil gold (metalaxyl + mancozeb), and mancozeb considerably impeded the radial growth of *S. rolfsii*. In addition, two fungicides that significantly inhibit the growth of *S. rolfsii* in vivo and cause collar rot disease in chickpeas are thiophanate methyl and mancozeb. Wanget al. (2015) revealed that the strong antifungal activity of fluazinam against the stem-rot-causing *Sclerotium rolfsii*. Methane sodium, methyl bromide, and chloropicrin are examples of soil fumigants that stop *S. rolfsii* mycelium growth. Kondeet al. (2008) found that treating soybean seeds with Thiram + Carbendazim ($2+1\text{g kg}^{-1}$) significantly prevented collar rot. Rahman et al. (2020) examined the effectiveness of specific fungicides in treating soybean collar rot. They came to the conclusion that Dithane M-45 showed the lowest percentage of soybean plant death (27.28%) at 0.2% concentration in vitro and totally prevented the mycelial growth of the collar rot pathogen *Sclerotium rolfsii*.

Compatibility of Bioagents with Chemical Fungicides

Sub lethal dose of fungicide along with the antagonist tolerant to the fungicide to enhance the suppressive effect is desired in the IDM programme. Sensitivity of four fungal antagonists *Chaetomium globosum*, *T. harzianum*, *T. viride* to six fungicides were evaluated by Kayand Stewart (1994) they fungi observed that antagonistic fungi were insensitive to captan, mancozeb and thiram but were sensitive to benomyl ($\text{EC } 50 < 0.3 \text{ ugml}^{-1}$) iprodione and procymidone ($\text{EC } 50 < 3.3 \text{ mgml}^{-1}$).

Tiwari and Singh (2004) investigated the non-target effects of several fungicides on *Trichoderma harzianum* and *Rhizobium leguminosarum* as well as their effectiveness against *Rhizoctonia solani* and *Sclerotium rolfsii*. When it came to stopping the mycelial growth of *Sclerotium rolfsii* and *Rhizoctonia solani*, as well as their non-target impacts on *Rhizobium leguminosarum* and *Trichoderma harzianum*, all fungicides performed much better than controls. However, the growth of *Trichoderma harzianum* and *Rhizobium leguminosarum* was least affected or unaffected in Ediphenphos, Isoprothiolane, Kitazin, Triadimefon and Validamycin amended medium. Gupta (2004) found that *Trichoderma harzianum* had good compatibility with pesticides such as captan and monocrotophos, followed by thimet and 2-4-D, even at higher concentrations (1000 ppm). Only

at concentrations between 1 and 100 ppm were pesticides like vitavax and chloropyriphos compatible with the growth of *Trichoderma harzianum*, but carbendazim was not compatible with the growth of the fungus at any of the concentrations tested between 1 and 1000 ppm. Kumar et al. (2005) *T. harzianum* compatibility with three fungicides (mancozeb, carbendazim, and wettable sulphur) revealed a significant reduction in *T. harzianum* growth with increasing concentrations of the toxicants in both solid and liquid medium. The fungitoxic activity of mancozeb was tolerated by the antagonists. Carbendazim had the highest rate of reduction in *T. harzianum* growth. In comparison to the control, wettable sulphur (Sulfex) inhibited the antagonist the least in both liquid and solid media. They also stated that *T. harzianum* can be combined with all toxins except carbendazim.

Integrated Management

Integrated management was first formulated by scientists of California University in 1959 (Stern et al., 1959). This concept was later introduced as integrated pest management (which also included integrated disease management). Integrated Disease Management is a most appealing strategy in which biological, cultural, and gential disease management with adequate but limited pesticide use. Panand Das (2011) A field study was done to prevent cowpea root and collar rot with two organic formulations of *Trichoderma harzianum* as well as various combinations. It was found that seed priming with the antagonist *Trichoderma harzianum* mycelia preparation at 4gkg^{-1} of seed and organic formulation of the antagonist in vermicompost in combination with 20% neem cake (w/w) provided the best disease control. *Trichoderma* in vermicompost + 20% neem cakes has given better disease control than the others. Lahreet al. (2012) They carried out a field experiment to test the effectiveness of *Trichoderma* by treating seeds along with applying neem cake, mustard cake and karanj cake to the soil in order to combat *S. rolfsii*. They discovered that the most effective method was to treat seeds with neem cake as soil application, which resulted in the highest seed germination rate (93.05%) and lowest seed mortality (8.32%). This was followed by treating seeds with mustard cake and karanj cake. Singh et al. (2017) conducted a study to investigate the combination of fungicides, *Pseudomonas*, and *Trichoderma* for the treatment of chickpea collar rot. Their findings showed that the most effective therapy was a soil application of *Trichoderma harzianum* enhanced FYM @ 8qha^{-1} + seed treatment with hexaconazole @ 3mlkg^{-1} , with the lowest mortality

(4.30 and 2.25%) and the highest increase in grain production (5.80 and 2.59%) compared to the control.

Conclusion

The biggest problem in the production of chickpeas is collar rot, and controlling diseases is crucial to the process. The initial density of the inoculum, the pace of infection, and the duration and stage of the plant host all influence the development of the disease. Consequently, therefore, the goal of control measures to stop a disease epidemic should be to lower the initial inoculum density, as well as the inoculum's survival and spread, infection rate, and duration of crop exposure. Control techniques to avoid a disease epidemic should be directed at minimizing the initial inoculum density the survival and spreading of inoculum, the rate of infection and the duration the crop is exposed to infection.

Conflict of Interests

The authors have declared that no conflict of interest exists

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