Review Article

Wilt (*Fusarium oxysporum* f. sp. *ricini*) disease of castor: A review

ABSTRACT

Castor (*Ricinus communis* L.) is one of the most oilseed crops in India. It is cultivated around the world and is endemic in many parts of the world. The castor wilt is caused by xylem inhabiting fungus, *Fusarium oxysporum* f. sp. *ricini* (Fusari) is most destructive disease and pathogen is both seed and soil borne. The disease is characterized by the presence of spores in the castor germplasm which is highly virulent. The morphological, cultural and pathogenic characteristics of castor isolates and their management practices. Fungicides like thiram, carbendazim and mancozebwere evaluated for their ability to inhibit the growth of pathogen.The information regarding the geographic distribution, symptoms, nematode-fungus interaction, disease cycle, epidemiology, variability of pathogen, host–pathogen interaction, screening of castor germplasm for identification of resistant sources and management practices of castor wilt disease has been reviewed in this article.

Key word: Castor, *Fusarium oxysporum* f. sp. *ricini*, Castor wilt, Management, IDM

Introduction

Castor is a Latin word, also known as Palm of Christ and scientifically known as *Ricinus communis* L. Castor is monospecific and belongs to family Euphorbiaceae (2n=20). The castor plant appears to have originated in Eastern Africa, especially around Ethiopia and cultivated around the world. Asian country (India) is the main producer of castor within the world (Sudha *et al.*, 2016). It grows throughout the warm-temperate and tropical regions and flourishes under a variety of climatic conditions. Castor is one of the important non-edible oilseeds with immense industrial and commercial values. This crop is widely distributed throughout the tropics and sub-tropics and is well adapted to the temperate regions of the world. The major castor producing countries in the world are India, Brazil, USSR and China. India is the world leader with regards to castor area (10.02 lakh ha), production (19.67 lakh

tons) and productivity (1815 kg/ha) (Anon., 2022). The most important castor manufacturing states in this country are Gujarat, Rajasthan and Andhra Pradesh. Together, these States account for quite 90 per cent of total domestic production with Gujarat being the most important physical seed manufacturing State. Monocropping followed due to its high economic return resulted in wilt endemic which seriously hampered castor cultivation in the state (Dange *et al*., 1997). The wilt disease is endemic to all castor growing states of India causing a significant yield loss of around 80-100 per cent per year (Anjani *et al*., 2004). Chattopadhyay (2000) reported yield reduction of 1.86 kg/ha with each per cent incidence of wilt disease.

Geographical Distribution

The disease was first detected in Morocco in 1953 (Reiuf, 1953) and it is also prevalent in Russia, Brazil, Taiwan, and Nepal. It was first identified in India in 1974 from the Rajasthan regions of Udaipur and Sirohi (Nanda and Prasad, 1974), followed by Gujarat, Andhra Pradesh, and Karnataka. The disease is prevalent in Rajasthan, Gujarat, Telangana, Karnataka, Haryana, Maharashtra, Tamil Nadu, Andhra Pradesh, Odisha, Madhya Pradesh, and Bihar. Wilt disease was a significant problem under irrigated conditions, whereas root rot disease was prevalent under rainfed conditions (Anon., 1985). Disease occurrence is higher in irrigated conditions due to the longer crop duration in Gujarat than in the southern regions. Wilt was found to be rooted in dark soil (Anon., 1990). The incidence of wilt varies with hybrid and crop rotation. Disease incidence varied significantly across all stages of crop development and between cultivars. The disease emerges on young crop seedlings sown between July and August, but the largest mortality occurs during flowering and at various phases of spike production. Disease incidence in Russia reached up to 80 per cent (Moshkin, 1986). The amount of seed production loss is determined by the stage of the crop at which plants wilt: 77 per cent at the flowering stage, 63 per cent at 90 days old, and 39 per cent at later stages on secondary branches (Pushpavathi, 1995). All cultivated castor hybrids in Gujarat experienced yield losses (Dange *et al*., 1997) and wilt incidences of up to 85 per cent were reported under North Gujarat conditions (Dange, 2003). Wilt incidence in Andhra Pradesh ranged from 5 to 60 per cent, resulting in a 1.86 kg/ha loss for each percent incidence of wilt disease (Chattopadhyay, 2000). More than 95 per cent of Gujarat's castor growing regions are occupied by castor hybrids that are tolerant to wilt disease, and productivity has increased dramatically from 350 to 1970 kg/ha (Damodaram and Hegde, 2010). Andreeva (1979) reported the occurrence of wilt in the USSR.

Symptoms

Castor plants are susceptible to wilt at all growth stages but disease generally appears at flowering and spike formation stage and becomes more prominent in later stage of the crop. Young seedling at two-three leaf's stage exhibit discolouration of hypocotyl and loss of turgidity with or without change in colour. Sick plants either do not bear capsules or give puny seeds (Moshkin, 1986). Young plants at budding stage are also severely attacked, which exhibit gradual yellowing of apical leaves, shrivelling with marginal necrosis and dry completely. The mycelium penetrates the vascular system of the roots, stems and leaves causing necrosis which lead to wilting and finally death of the plant (Sviridov, 1989). Infected stem shows blackish lesions above the collar region and these lesions further spread up to a distance of 15 to 20 cm above the ground level (Reiuf, 1953). At the time of flowering and spike formation stages, the disease is characterized by gradual yellowing, shrivelling with marginal and inter-venial necrosis of leaves. Finally, the leaves with petiole dry up and hang down (Reiuf, 1953; Nanda and Prasad, 1974). Roots of wilted plant show blackening and necrosis, while in case of partial wilting only one side of root system is observed blackish and necrotic and the other side root system remains healthy. When the stem of wilted plant is splatted open, white cottony fungal growth is observed in the pith region and the pith become blackish. Transverse and longitudinal sections of the affected roots reveal the presence of the fungus in vascular tissue and the xylem parenchyma. Formation of tyloses is also observed in infected roots (Nanda and Prasad, 1974). In some cases, dark stripe may cover the whole stem and is formed up to the infected leaves (Raoof and Rao, 1999). At pre-flowering stage, leaves turn yellow, marginal and inter venial necrosis starts with complete senescence of lower leaves, ultimately leads to irreversible wilting with bend apices. Infected plants do not produce any inflorescences. Plants infected at flowering, spike formation and capsule development stages appear sick and leaves become yellow with marginal necrosis and later, the entire leaf becomes necrotic and shrivels. Lower leaves drop away due to senescence except few top leaves and followed by irreversible wilting of plant (Raoof and Nageshwar Rao, 1997). When the affected roots were sectioned in transverse and longitudinal directions, the presence of fungus was noticed in vascular tissue and in xylem parenchyma. Tyloses formation is also observed in xylem vessels of the infected roots. The browning and blackening of xylem tissues was observed, when the stem is split open. Intercellular fungal mycelium in vessels and hypertrophy of xylem parenchymatous cells were observed in the infected stem tissue (Nanda, 1975). Anjani *et al.* (2014) noticed that grownup susceptible plants exhibited typical wilt symptoms like stunted growth, gradual yellowing, shrivelling with marginal necrosis and complete drying of leaves and branches, vascular discolouration

and death of entire plant. These plants showed blackish lesions above the collar region of stem which further covered the entire stem. Pith of the infected stem became black and covered with white cottony Fusarium fungus. Roots of wilted plants showed blackening and necrosis.

Causal Organism

Fusarium wilt of castor (*R. communis*) is caused by xylem inhabiting fungus, *Fusarium oxysporum* f. sp. *ricini*(Fusari). The pathogen is both seed and soil borne. White fluffy mycelial growth of fungus observed on potato dextrose agar medium, that changes to pinkish when incubated under fluorescent light. The fungus produces both macro and micro conidia. The micro conidia are single or two celled, round to ovoid, hyaline, many in number which measures 6.31–15.29×3.66–3.76 µ in size. Macroconidia are straight, spindle and sickle shaped, few in number, 2–6 septate (mostly 3) and measure 17.50–70.00×3.50–5.25 µ (Desai and Dange, 2003). Both terminal and intercalary chlamydospores appear which measures 8.7×4.44 µ. Generally, sporodochia develops in two-week old cultures (Kolte, 1995).

Interaction between reniform nematode, *Rotylenchusreniformis*Linford and Oliveira. and *F. oxysporium* f. sp. ricini

Rotylenchulusreniformis Linford is a stationary semi-endoparasite. It is mostly found in tropical, subtropical and warm temperate zones over South America, North America, The Caribbean Basin, Africa, Southern Europe, The Middle East, Asia, Australia and the Pacific (Ayala and Ramirez, 1964). It has a wide host range, including cereals (sorghum, wheat, maize, and rice), oilseed crops (sunflower, groundnut, and castor), legumes (soybean, pigeon pea, common bean, chickpea, and black gram), vegetables (tomato, potato, cucumber, and eggplant) and fruits (grape vine, citrus, tropical fruits, and banana) (Castillo *et al*., 2008).*R. reniformis*, a nematode, has been documented to be implicated in the castor wilt disease complex, which gives way to opportunistic pathogens such as Fusarium, resulting in massive agricultural production loss and an increase in disease severity when both are involved. Wilt fungus, *F. oxysporum* f. sp. *ricini*, and reniform nematode, *R. reniformis*, are soil-borne organisms that may live in the soil for a long period of time without a host. According to Patel *et al*. (2000), *F. oxysporum* f. sp. *ricini*alone could not cause wilt in the wilt resistant castor hybrid GCH-4, but when combined with the reniform nematode *R. reniformis*, wilt incidence increased from 25.0 to 33.3 per cent. This implies that nematodes play an important role in the breakdown of wilt resistance in castor hybrid GCH-4, hence enhancing the severity of castor wilt (Pathak, 2003). Nematodes play vital role in the breakdown of wilt

resistance in castor hybrid and thus, increasing the severity of the castor wilts (Jangir *et al*., 2018). Reniform nematode damage has been estimated to cause yield losses of up to Rs. 18 crores per year (13.93% yield loss) in castor (Jain *et al*., 2007). GCH 4, a popular wilt resistant castor hybrid of erst while Andhra Pradesh became susceptible to wilt in the presence of reniform nematode and wilt appeared earlier in this synergistic interaction (Patel *et al*., 2000).

Fungus Disease Cycle and Epidemiology

Disease cycle

Macroconidia are hyaline, few in number, having 2- 6 septa, straight, spindle as well as sickle-shaped and measure 17.5-70 x 3.50-5.25 µm (Desai *et al*., 2003). Generally, chlamydospores both terminal and intercalary are developed in later stages of growth after in two weeks old of inoculation (Kolte, 1995). The favourable temperature for infection by wilt pathogen is 13-15°C and for symptom expression is 22-25°C (Andreeva, 1979).

Epidemiology

The fungus found to be externally as well as internally seed borne to an extent of 10–20 per cent (Chattopadhyay, 2000, Raoof *et al*. 2006). Plants are susceptible at all growth stages, but the disease generally appears in months of October–November when the crop is about 3–4 months old and becomes more prominent during February- March when the crop is in seed formation stage (Nanda and Prasad 1974). Plants were most susceptible to infection when the temperature is between 13-15 °C, and between 22-25 °C when the entire range of symptoms appears (Andreeva, 1979). In the perpetuation and spread of the pathogen infected seeds play an important role upto a depth of 60 cm (Sviridov, 1988; Dange, 2003). For prolonged durations, the fungus survives in the infected crop residue as thick-walled resting structures as chlamydospores.

Variability in Wilt Pathogen, *F. oxysporum* f. sp. *ricini*

Pathogenic variability and aggressiveness in the pathogen are the main cause of breakdown of resistance in host cultivars. The diferential reaction of varieties and breeding lines over the years and across the locations indicated prevalence of variants in the pathogen. Wide variation observed in morphological, cultural and pathogenic characteristics among various isolates of *F. oxysporum* f. sp. *ricini* (Nanda and Prasad 1974; Desai *et al*., 2003) and isolates which were highly virulent produced abundant spores compared to moderately virulent isolates. The disease incidence varied between 49.9 and 100 per cent in pistillate lines VP-1 and VI-9 respectively but variety 48-1 remained completely free from wilt. Santhalakshmi Prasad *et al.* (2008) grouped five pathotypes based on wilt reaction on castor

cultivars and five clusters among isolates of *F. oxysporum* f. sp. *ricini* using RAPD *i.e.*, random amplifed polymorphic DNA analysis and no correlation of pathogenic variability of castor wilt isolates with genetic variability observed based on RAPD. Ten pathogenic races were identifed among *F. oxysporum*f. sp. *ricini*146 isolates, of them, seven races (races 2, 3, 4, 5, 7, 8 and 10) were predominant in Andhra Pradesh and five races (races 1, 2, 4, 6 and 9) in Gujarat (Reddy, 2010). Mulekar *et al.* (2017) recorded morphological variability in 24 isolates of *F. oxysporum* f. sp. *ricini* representing various castor growing regions of India in Andhra Pradesh, Gujarat, Rajasthan, Tamil Nadu and Telangana states.

Host Pathogen Interaction

To our knowledge, only two research have been conducted on the molecular expression of fusarium wilt resistance in castor. mRNA expression study of the lipoxygenase (LOX) gene, LOX5 (Rc9-LOX), exhibited a higher expression level in resistant genotypes (48-1 and SKP-84) compared to susceptible genotypes (VP-1 and VI-9), indicating its involvement in a defence response in an incompatible host-pathogen relationship. Thus, the RcLOX5 gene can be utilized to identify wilt-resistant genotypes (Somnath *et al.*, 2013). Expression study of major phenylpropanoid pathway genes revealed the role of phenylalanine ammonia lyase (PAL) and cinnamate 4-hydoxylase 2 (C4H2) genes, as evidenced by enhanced expression in resistant genotypes (Jadav *et al.*, 2013).

Identification of Resistant Source

The wilt disease is soil borne; hence it is difficult to manage through chemicals. Developing a resistant variety is imperative to combat the disease under field conditions. Screening and identification of resistant sources are the prerequisite in any breeding program for developing a resistant variety. Castor being a monotypic genus, several workers identified resistant sources to castor wilt by evaluating a greater number of diverse germplasm accessions, breeding lines, varieties and hybrids in sick plot and artificial inoculation conditions in pot culture in AICRP (Castor) system (Raoof and Nageshwar Rao, 1996; Pushpavathi *et al.*, 1997; Pathak, 2003). The identified resistant sources should be used in crossing in the breeding programme. Breeding for disease resistance requires an efficient screening technique, genetic sources of resistance and appropriate transfer of resistance genes into improved genetic backgrounds. Different techniques *i.e.* root dip has been employed to screen castor germplasm for wilt resistance.

Management Practices

Wilt disease is both seed-borne and soil-borne, making control difficult with a single strategy. Therefore, an integrated approach is required. The management may be based on the

integration of the host's genetic resistance, cultural methods that create an environment unfavourable for disease development, such as seed treatment with fungicides or bio-control agents, crop rotation with non-fungus hosts, continuous rouging of wilted plants, and sanitation. Cultivation of resistant cultivars is the best way to manage wilt. This method is without a doubt the most economical, conceptually simple, and safest way to handle wilt. It also decreases environmental pollutants, eliminates risks to human health, and preserves biological equilibrium in the ecosystem.

Cultural methods

Crop rotation: Continuous cultivation of castor crops promotes the pathogen population, hence crop rotation with nonhost crops like finger millet and pearl millet reduced the frequency of wilt (Raoof and Raju, 2005).

Resistant variety: Nagesh *et al.* (2020) recorded the three resistant accessions (RG-43, RG-111, RG-109, RG-297, RG-1608, RG-1624, RG-2758, RG-2787, RG-2800, RG-2818, RG-2822, RG-3016 and RG-3105) would be of great value as donors of resistance. Patel *et al.* (2020) reported that GCH 8 exhibited resistant reaction (≤ 20 % disease incidence) against wilt and root rot under irrigated condition and moderately resistant reaction (20-40 % disease incidence) to wilt under rainfed condition. Prasad *et al.* (2016) observed that 83 parental lines evaluated against wilt in wilt sick plot, 44 lines were susceptible to wilt with more than 20.5 per cent wilt and wilt incidence was not observed in three lines *i.e.*, PMC 40, DCS 86 and DCS 118. Thirty-six lines showed resistant reaction with less than 20 per cent wilt incidence. Bhati *et al.* (2018) noticed that 16 genotypes (JI-422, JI-384, JI-416, JI-402, JI-258, SKP-84, GEETA, JP-86, JI-368, JI-403, JI-423, JI-424, SKP-72, SKP-106, RG-43 and 48-1) showed resistant reaction for wilt disease. Rajput *et al.* (2023) revealed that a total of 28, 8, 4, 2, 4 and 4 genotypes fell into various disease reaction categories *viz.*, highly resistant, resistant, moderately resistant, moderately susceptible and susceptible, respectively. The castor genotypes *i.e.*, GP-640, JI-35, RG-3477 and SKI-341 were found highly susceptible and ANDCI-10-8, MI-27, RG-3938 and SKI-284 found susceptible. The castor genotypes viz., RG-1916, RG-155, RG-1647, AP-163, Ap-33, Ap-156, Ap56, Ap-42, Ap-200, Ap-180, Ap-171 were resistant to wilt pathogen isolates of Palem, S. K. Nagar, Hyderabad (Anon., 2016)

Intercropping

Build-up of high inoculum load due to continuous cultivation of castor could be the main cause for severe wilt incidence. Maximum wilt incidence 48 per cent was recorded in Groundnut + Castor (5:1) inter cropping and minimum incidence (1–6%) in Pigeonpea + Castor (1:1) intercropping. Wilt incidence in Sunflower + Castor (2: l) intercrop was 32 per

cent. For inter cropping, the castor crop was evaluated under rainfed conditions as a component crop in different non-leguminous crops and legumes in 10:1 and 6:1 row proportion. Inter cropping urd bean with castor in 6:1 row proportion resulted in the highest land equivalent ratio value of 1.85 coupled with highest seed yield. There was a reduction in the wilt incidence in castor and pigeonpea intercropping system (Chand *et al.*, 2000).

Physical methods

Raoof and Rao (1997) studied on effect of soil solarization on castor wilt in a wilt sick plot by covering the low density transparent polyethylene sheet (200 gauge) and recorded that maximum reduction in wilt incidence (50%), *F. oxysporum* f. sp. *ricini* population (35%), nematode population (78%) and highest castor seed yield in plots solarized for six weeks in summer. Three weeks soil solarization of wilt sick plot during summer season by covering with transparent linear low-density polyethylene (LLDPE) sheets (25 µm) reduced wilt incidence by 38 per cent, population of castor wilt pathogen by 67 and increased castor seed yield by 125 per cent as compared to non-solarized plots (Desai and Dange, 2003).

Biocontrol control

Ajay and Naik (2015) noted the *in-vitro* efficacy of biocontrol agents against an isolated castor wilt pathogen and found that *T. viride* had the highest inhibition of 92.35 percent, followed by the bacterial bioagent *Bacillus subtilis* (88.75%). Janga *et al.* (2017) identified 42 antagonistic isolates from 500 bacterial isolates that were isolated from the castor rhizosphere soil samples and selected four isolates E37, P37, P40 and P46 which showed 65–70 per cent of disease suppression. Apurva *et al.*, (2020) conducted to know the antagonistic activity of different bioagents *in-vitro* during 2019-20. Eight fungal and eight bacterial bio-agents were evaluated using dual culture method. Th14 strain of *Trichoderma harzianum* exhibited highest (80.47%) inhibition of mycelial growth of *F. oxysporum* f. sp. *ricini* while the remaining strains of both *T. harzianum*and *T. viride* were able to control the pathogen at least by 72 per cent under *in vitro* conditions. In case of bacterial antagonists *Bacillus velezensis* (P42) showed a good inhibition of 58.89 per cent over the rest.

In greenhouse trials with castor cultivar GCH 4, seed germination was found to be significantly high in bioagent treatments when compared to the pathogen check. Among all the bioagents low wilt incidence was recorded with *P. fluorescens* Pf2 (30%) followed by *T. harzianum* ThN2 (45%) and *T. harzianum* Th4d (55%) (Anon., 2016)

Chemical control

Shalini *et al.* (2021) noticed that high inhibition (100%) of radial growth of test *pathogen F. oxysporum*f.sp. *ricini*was recorded by the fungicide carbendazim at both the

recommended and half the recommended doses, while minimum inhibition was recorded by metalaxyl at the recommended (82.86 percent) and half the recommended doses (77.86 percent). Jadav *et al.* (2022) studied on laboratory screening of different fungicides, thiram 75 per cent WP (87.24%) was found to be quite effective in inhibiting the radial growth of test pathogen among non-systemic group of fungicides, while in systemic group of fungicides, carbendazim 50% WP (100%) and in case of ready-mixed fungicides carbendazim 12 per cent + mancozeb 63 per cent WP (88.33%) were significantly inhibited the growth of *F. oxysporum* f. sp. *ricini* under in vitro.

Compatibility of seed coat biopolymer with biocontrol agent *T. harzianum*

In the compatibility studies of seed coat polymers with biocontrol agent *T. harzianum*, the combination of chitosan with biocontrol agent gave the highest germination percentage (95%) and vigour index in GCH-4 than the polymers and biocontrol agent used alone. *F. oxysporum*f.sp. *ricini* incited seed and seedling root rot was significantly low in combination of chitosan with biocontrol agent (20%) compared to pathogen check (70%) (Annon., 2016)

IDM

Dange *et al.* (2006) revealed that application of bio-agents like *Trichoderma* spp. and chemical like carbendazim as seed treatment as well as use of resistant cultivars along with proper cultural practices like soil solarization provides an effective control of the disease. Shalini *et al.* (2014) evaluated that efficacy of biocontrol agents and chemicals under *in vitro* condition against *F. oxysporum* f. sp. *ricini* and recorded that all treatments in significantly decreased wilt incidence and improved plant growth compared to untreated inoculated control. Combination treatment of carbofuran @ 2g/kg soil + carbendazim @ 1g/kg soil was found most effective in reducing wilt incidence and reniform nematode population followed by *T. viride* @ 4g/kg seed + *P. fluorescens* @ 10g/kg seed.Ghante*et al.* (2018) revealed that soil application of (*T. viride* + neem seed cake) + seed treatment of (carbendazim 25 WP + mancozeb 50 WP + *T. viride*) + soil drenching of azoxystrobin 23 EC showed minimum wilt incidence *i.e.* 7.12 per cent and 45.42 per cent with maximum yield *i.e.* 1368.73 kg/ha and 458.38 kg/ha, respectively. The seed treatment with carbendazim (2 g/kg seed), soil application of *T. viride* @ 2.5 kg mixed with 10 t FYM/ha, intercropping of castor (4:1) with pigeon pea (PRG-100) proved to be effective in comparison to farmers' practices in which the application of chemical pesticides only (Singh *et al*., 2008). The seed treatment with carbendazim recorded significantly low wilt incidence (24.4 %) with high seed yield of 1123 kg/h in GCH-4. Seed treatment and soil application of *T. harzianum* Th4d WP also recorded

low wilt incidence (26%) and seed yield of 1016 kg/ha, whereas in pathogen control the wilt incidence was 60.6 per cent with seed yield of 905 kg/h (Anon., 2016).

Rajpurohit *et al.,* (2009) reported that seed treatment with *T. viride* @10g/kg followed by soil application @ 2.5kg/ha reduced per cent wilt incidence from 41.6 to 7.2. Sudhakar *et al.* (2009) noted that seed treatment with *T. viride* @ 10g/kg and soil application of neem cake @ 1ton/ha effective for wilt management.

Shalini *et al.* (2014) noted that treatments of soil application of carbofuran 3G@ 2g/kg soil + seed treatment with carbendazim 50WP @ 1g/kg soil, Seed treatment with carbendazim 50WP @ 1g/kg seed and seed treatment with *T. viride* Trichogen-T Tv@ 4g/kg seed + seed treatment with *P. fluorescens*Florozen-P @ 10g/kg seed were found significantly effective as the recorded around 30 per cent wilt incidence.

Conclusion

The distribution of pathogen, *F. oxysporum* is known to be cosmopolitan and caused xylem brownish or blocking. Castor plants are susceptible to wilt at all growth stages but disease generally appears at flowering and spike formation stage.

- The crop rotation with non-host crops like finger millet and pearl millet reduced the wilt disease.
- The castor genotypes/ verities *viz.*, GCH 8, RG-1916, RG-155, RG-1647, AP-163, Ap-33, Ap-156, Ap56, Ap-42, Ap-200, Ap-180, Ap-171 were resistant to wilt.
- The intercropping with Groundnut + Castor (5:1), Pigeonpea + Castor (1:1) and Sunflower + Castor (2:1) to reduce the wilt disease.
- The biological agent of *Trichoderma harzianum*, *T. viride*. *Pseudomonas fluorescens* effective against castor wilt and reniform nematode.
- Most effective fungicides like Mancozeb, Carbendazim 50 WP, Carbendazim 12% + Mancozeb 63%.

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