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Exploring Fusarium wilt in Castor Plant: A Comprehensive Overview

L. S. Chaudhary [a*], M. A. Chaudhari [a], H. K. Chaudhary [a] and S. S. Kansara [b]

[a] Department of Entomology, NMCA, NAU, Navsari- 396450, India.
[b] Department of Plant Pathology, NMCA, NAU, Navsari-396450, India.

Authors' contributions

This work was carried out in collaboration among all authors. Authors LSC, MAC and HKC prepared the original draft and designed the study and finally edited the manuscript. Author SSK gave useful guidance during preparation of the article. All authors read and approved the final manuscript.

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ABSTRACT

Wilt of castor (*Ricinus communis*) plant caused by xylem inhabiting fungus *Fusarium oxysporum* f. sp. *ricini* (Fusari), which gives way to opportunistic pathogens such as *R. reniformis*, a nematode, resulting in massive agricultural production loss and an increase in disease severity when both are involved. Disease prevalence varied greatly between crop stages and cultivars. The genotypes GP-640, JI-35, RG-3477, and SKI-341 were highly susceptible, while RG-1916, RG-155, RG-1647, AP-163, Ap-33, Ap-156, Ap56, Ap-42, Ap-200, Ap-180, and Ap-171 were resistant against castor wilt. In vitro evaluation of the castor crop as a component crop revealed a maximum and minimum wilt occurrence rate of 48% and 1-6% in Groundnut + Castor (5:1) and Pigeonpea + Castor (1:1) intercropping, respectively. In GCH-4, treatment of the seeds with carbendazim at 1g/kg soil was

**Corresponding author: E-mail: lalabhai261999@gmail.com;*

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shown to be the most efficient in lowering disease prevalence (24.4%) while also producing a high seed production of 1123 kg/h.

Keywords: Castor; Fusarium oxysporum f. sp. ricini; castor wilt; IDM.

1. INTRODUCTION

Castor, scientifically known as *Ricinus communis* L., is a Latin word that is also referred to as Palm of Christ. Castor is monospecific and belongs to family Euphorbiaceae (2n=20). The castor plant is grown all over the world, although it seems to have evolved in Eastern Africa, particularly in the Ethiopian region. India is the mainproducer of *R. communis* within the world [1]. It thrives in a range of environmental circumstances and grows throughout tropical and warm-temperate climates [2-5]. One of the significant non-edible oilseeds with significant industrial and economic potential is castor. This crop is ideally suited to the world's temperate zones and is widely dispersed across the tropics and sub-tropics. China, India, Brazil, and the USSR are the world's top producers of castor. In terms of castor cultivating area (10.02 lakhs ha), productivity (1815 kg per hectare), and production (19.67 lakhs tonnes), India leads the world [6]. In India, commercial cultivation of castor is taking place in 16 states. Among them Gujarat, Rajasthan, Haryana, Telanagana State and Andhra Pradesh are the major states contributing about 91% and 97% to the countries area and production respectively [7]. Due to the high economic return of monocropping, wilt became endemic and severely restricted castor cultivation in the state. [8]. According to Hegde [9], castor wilt reduces seed weight, yield and seed oil content by 8–14%, 10-40% and 1-2%, respectively. The yield reduction increased with each percentage of castor wilt disease incidence by 1.86 kg/ha [10].

2. GEOGRAPHICAL DISTRIBUTION

First identified in Morocco in 1953 [11], this condition is also common in Brazil, Taiwan, Nepal, and Russia. It was first found in India in the Udaipur and Sirohi districts of Rajasthan in 1974 [12]. Andhra Pradesh, Gujarat, and Karnataka were the following states to have it noticed. Rajasthan, Gujarat, Telangana, Karnataka, Haryana, Maharashtra, Tamil Nadu, Andhra Pradesh, Odisha, Madhya Pradesh, and Bihar are among the states where the disease is common. Under irrigated conditions, castor wilt disease was a major issue, while under rainy conditions, root rot disease was more common [13]. The greater incidence of diseases in irrigated areas can be attributed to Gujarat's longer castor crop duration compared to the southern regions. It was discovered that Fusarium wilt had settled in black soil [14]. The prevalence of disease differed greatly between cultivars and at every stage of crop development. The disease first appears on early crop seedlings sowed in July and August, although flowering and different stages of spike formation are when greatest losses occur. Disease incidence in Russia has risen to 80% [15]. The crop's wilting point determines the amount of seed production loss: 77% occurs during the flowering stage, 63% occurs at 90 days, and 39% occurs later on secondary growth of castor plant [16]. In Gujarat, all cultivated castor hybrids had losses in production [8], and in North Gujarat, wilt incidences as high as 85% were reported by Dange [17]. More than 95% of Gujarat's castor growing regions are inhabited by wilt disease-tolerant castor hybrids, and productivity has risen drastically from 350 to 1970 kg per ha [18].

3. SYMPTOMS OF FUSARIUM WILT OF CASTOR

"R. Communis seedlings are vulnerable to wilt at all growth phases, but disease generally appear during blooming and spike formation, and it becomes more noticeable later in the growing season of castor crop. Young seedlings at the two- to three-leaf stage exhibit hypocotyl discoloration and lack of turgidity, with or without color change. Sick plants produce either no capsules or very little seeds" [15]. "Young plants in the budding stage are also severely damaged, resulting in yellowing of apical leaves, shriveling with marginal necrosis, which and drying altogether. The mycelium penetrates the vascular system of the roots, stems, and leaves, causing necrosis, wilting, and eventually death of the plant" [19]. " Infected stems reveal blackish lesions above the collar region, which spread up to 15 to 20 cm above ground level" [11]. During the flowering and spike production stages, the disease causes yellowing and shriveling of the leaves, as well as marginal and inter-veinal necrosis. Finally, the petioles dried up and fall down [11,20]. "Wilted plant roots show blackening and tissue necrosis, but partial wilting only affects one side of the root system, leaving the other side unharmed. When the stem of a wilting plant is splattered open, white

cottony fungi growth emerges in the pith region, and the pith turns blackish. Transverse and longitudinal sections of the damaged roots show the fungus in the vascular tissue and the xylem tissue. Infected roots also produce tyloses" [12]. In some situations, a dark stripe may cover the entire stem and extend to the affected leaves [21]. "Pre-flowering leaves turn yellow, and marginal and inter-veneal necrosis begins with total senescence of the lower leaves, eventually leading to terminal wilting with bent apices. Infected plants generate no inflorescences. Plants infected during blooming, spike production, and capsule development appear unwell, and their leaves turn yellow with marginal necrosis, eventually becoming necrotic and shriveling. Senescence causes lower leaves to drop away, with the exception of a few top leaves, followed by irreversible plant wilting" [22]. When the damaged roots were sectioned in both transverse and longitudinal orientations, fungus was found in vascular tissue and xylem parenchyma. Tyloses are also seen in the xylem channels of diseased roots. When the stem was split open, the xylem tissues turned brown and black. Infected stem tissue showed intercellular fungal growth in arteries as well as enlargement of xylem parenchymatous cells [23]. The mature sensitive plants had typical wilt signs such as slowed development, progressive yellowing, shriveling with marginal disintegration and full withering of branches and leaves, vascular discoloration, and plant death. These plants developed dark brown lesions above the collar region of the stem, which eventually engulfed the entire stem. The pith of the afflicted stem turned dark and became infected with white cottony Fusarium fungus. The roots of wilted plants displayed blackening and necrotic [24].

3.1 Causal Organism

Fusarium wilt of castor *(R. communis*) is caused by the xylem-dwelling fungus *Fusarium oxysporum* f. sp. *ricini* (Fusari). The pathogen spreads through both seeds and soil. White fluffy mycelial development of a fungus seen on potato dextrose agar medium, which turns pinkish when cultured under a fluorescent lamp. The fungus showed both macro- and microconidia. The micro conidia are single or two celled, spherical to ovoid, hyaline, and numerous in number. They measure 6.31-15.29×3.66-3.76 µ. Desai and Dange [25] describe macroconidia as straight, spindle, and sickle-shaped, with 2-6 septates (usually 3). They measure 17.50-70.00×3.50-5.25 µ. The terminal and intercalary chlamydospores are 8.7×4.44 µ. Sporodochia typically develops in two-week-old cultures [26].

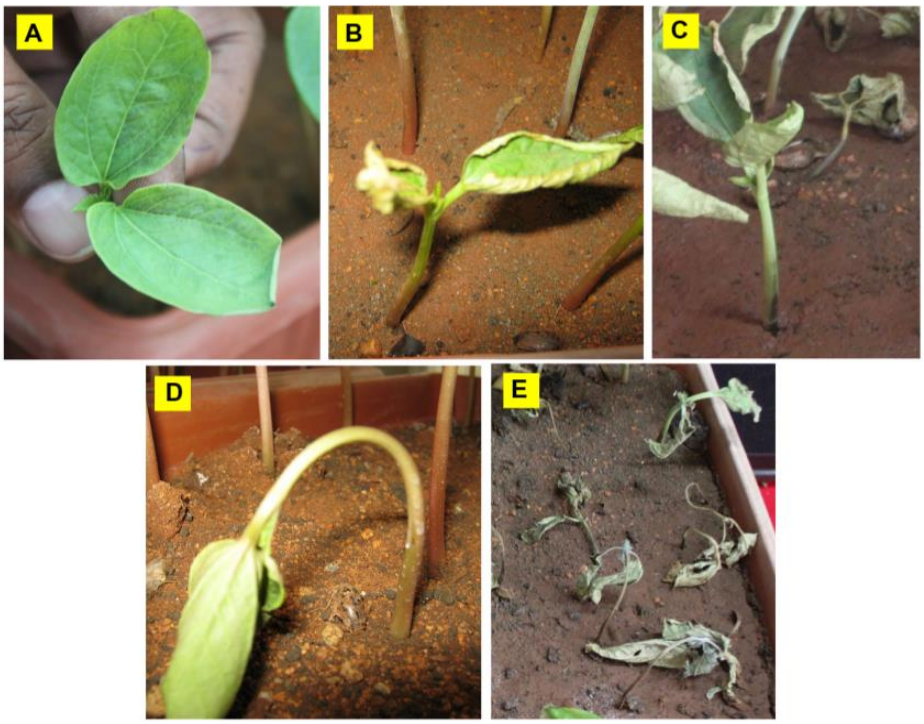

Fig. 1. Symptoms of Castor wilt infection caused by *F. Oxysporium* [A. Tissue necrosis, B. Leaves discouloration and drying, C. Blackstreak from the collar region, D. Seedling drooping, E. Seedling mortality]

3.2 Relationship between the Reniform Nematode, *Rotylenchus Reniformis* Linford and Oliveira and Castor wilt Fungus, *F. oxysporium* f. sp. *ricini*

"Semi-endoparasite *R. reniformis* Linford is a stationary parasite. Across South America, North America, the Caribbean Basin, Africa, Southern Europe, the Middle East, Asia, Australia, and the Pacific, it is primarily found in tropical, subtropical, and warm temperate zones" [27]. Its host range is broad and includes the following: vegetables (tomato, potato, cucumber, and eggplant); fruits (grape vine, citrus, tropical fruits, and banana); oilseed crops (sunflower, groundnut, and castor); legumes (soybean, pigeon pea, bean, chickpea, and black gram); and cereals (sorghum, wheat, maize, and rice) [28]. *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. Two soil-borne organisms that can persist in the soil for extended periods of time without a host are wilt fungus and reniform nematodes. Wilt in the wilt resistant castor hybrid GCH-4 was not caused by *F. oxysporum* f. sp*. ricini* alone, but wilt incidence raised from 25.0 to 33.3% when associated with the reniform nematode *R. reniformis* [29]. According to Jangir et al*.* [30], nematodes are essential for the loss of wilt resistance in castor hybrids, which increases the severity of wilts. Castor yield losses due to reniform nematode damage have been projected to reach 15% [31]. When reniform nematodes were present, Andhra Pradesh became vulnerable to wilt, even though GCH 4, a well-known wilt-resistant hybrid of castor, had already manifested itself earlier in this beneficial interaction [29].

4. DISEASE CYCLE AND EPIDEMIOLOGY

4.1 Disease Cycle

According to Desai et al*.* (2003), macroconidia are hyaline, a few in number, straight, spindle-like and sickle-shaped, with two to six septa. They range 17.5-70 x 3.50-5.25 µm. Even terminal and intercalary chlamydospores typically form later in the development cycle following an inoculation two weeks ago [26]. 13–15°C is the ideal temperature for wilt pathogen infection, while 22–25°C is the ideal temperature for symptom expression [32].

4.2 Epidemiology

It was discovered that the fungus had 10–20% seed borne both internally and externally [10,33]. All growth phases of plants are vulnerable, although the disease usually manifests in October and November when the crop is approximately 3.4 months old, and it intensifies in February and March when the crop is in the seed formation stage [12]. Plants are most vulnerable to infection when the temperature is between 13 and 15 °C, and between 22 and 25 °C when all of the symptoms appears [32]. Up to a depth of 60 cm, infected seeds are crucial to the pathogen's continuation and dissemination (55; Dange, 2003). The fungus lives for extended periods of time in the contaminated agricultural residue as chlamydospores, which are thick-walled resting structures.

4.3 Variability in *F. oxysporum* f. sp. *Ricini*, the Wilt Pathogen

The pathogen's aggression and diversity are primarily responsible for the breakdown of host cultivar resistance. Variants in the germ were prevalent, as evidenced by the different responses of breeding lines and cultivars throughout time and in different places. Numerous *F. oxysporum* f. sp. *Ricini* isolates varied greatly in their morphological, cultural, and pathogenic traits (32; Desai et al., 2003). Moreover, extremely virulent isolates produced a greater number of spores than moderately virulent isolates. In pistillate lines VP-1 and VI-9, the illness incidence ranged from 49.9 to 100%, respectively, although variety 48-1 showed no signs of wilt at all. Using random 860mplified polymorphic DNA analysis, or RAPD, Santhalakshmi Prasad et al. [34] grouped five pathotypes based on wilt reaction on castor cultivars and five clusters among isolates of *F. oxysporum* f. sp. *Ricini*. They found no correlation between the genetic variability observed by RAPD and the pathogenic variability of castor wilt isolates. *F. oxysporum* f. sp*. Ricini* 146 isolates were found to have ten pathogenic races; of these, seven (races 2, 3, 4, 5, 7, 8, and 10) were more common in Andhra Pradesh and five (races 1, 2, 4, 6 and 9) in Gujarat [35]. Mulekar et al. [36] observed morphological variation in 24 isolates of *F. oxysporum* f. sp*. Ricini* from states in Andhra

Pradesh, Gujarat, Rajasthan, Tamil Nadu, and Telangana, which are among the castor-growing regions of India.

4.4 Interaction in Host Pathogen

"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" [37]. "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" [38].

4.5 Resistant Source

Since the wilt disease is spread by soil, chemical management is a challenging solution. To tackle the disease in the field, developing a resistant variety is essential. Any breeding operation that aims to produce a resistant variety must first screen and identify resistant sources. Since Castor is a monotypic genus, researchers have utilized a larger number of diverse germplasm accessions, breeding lines, varieties, and hybrids in sick plot and artificial inoculation conditions in pot culture in the AICRP (Castor) system to identify resistant sources to Castor wilt [39,40,41]. In the breeding program, crossings should be made using the sources of resistance that have been found. An effective screening method, genetic sources of resistance, and suitable introduction of genes for resistance into improved genetic lines are necessary for breeding for disease resistance. Various methods, such as root dip, have been used to check for wilt resistance in castor germplasm.

4.6 Different Management Procedures Against Castor Wilt

Because wilt disease can spread through seeds as well as the soil, controlling it with a single method is challenging. Consequently, an integrated strategy is needed. Incorporating the host's genetic resistance, cultural practices that prevent the spread of disease (e.g., treating seeds with fungicides or biocontrol agents), crop rotation with non-fungus hosts, ongoing rouging of wilted plants, and sanitation are examples of management strategies. The best method of controlling wilt is to cultivate resistant cultivars. This is without a question the safest, most theoretically straightforward, and most cost-effective technique to deal with wilt. Additionally, it reduces pollution to the environment, removes hazards to human health, and maintains the ecosystem's biological balance.

4.7 Cultural Control

Crop rotation: Crop rotation with nonhost crops such as finger millet and pearl millet decreased the frequency of wilt since continued cultivation of castor crops encourages the pathogen proliferation [42].

Resistant variety: Nagesh et al. [43] "identified 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) that might be valuable as resistance donors". According to Patel et al. [20], "GCH 8 was resistant to wilt and root rot (≤ 20% disease incidence) under irrigated conditions and moderately resistant to wilt (20-40% disease incidence) in rainfed conditions". Prasad et al. (2016) "observed that of the 83 parental lines tested against wilt in a wilt sick plot, 44 were susceptible to wilt with more than 20.5% wilt, while wilt incidence was not found in three lines, PMC 40, DCS 86, and DCS 118. Thirty-six lines exhibited a resistant reaction, with less than 20% wilt incidence". Bhati et al. [44] "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) were resistant to wilt disease". According to Rajput et al. [45], "a total of 28, 8, 4, 2, 4 and 4 genotypes were classified as highly resistant, resistant, moderately resistant, moderately susceptible, and susceptible". "Castor genotypes GP-640, JI-35, RG-3477, and SKI-341 were very vulnerable, whereas ANDCI-10-8, MI-27, RG-3938, and SKI-284 were shown to be susceptible. Castor genotypes RG-1916, RG-155, RG-1647, AP-163, Ap-33, Ap-156, Ap56, Ap-42, Ap-200, Ap-180, and Ap-171 were resistant to wilt pathogen isolates of Palem, S. K. Nagar, Hyderabad" [46].

Sr. No.	Reaction	Varieties	Reference
1.	Highly Resistant	• 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; • GCH 8; • PMC 40, DCS 86 and DCS 118; • 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; • RG-1916, RG-155, RG-1647, AP-163, Ap-33, Ap-156, Ap56, Ap-42, Ap-200, Ap-180, Ap-171	• Nagesh et al.; [43] • Patel et al.[20]; • Prasad et al. [4]; • Bhati et al. [44]; • Rajput et al. [45]
2.	Suscetible	GP-640, JI-35, RG-3477 and SKI-341	Rajput et al. [45]

4.8 Intercropping

"The accumulation of a large inoculum load as a result of continuous castor cultivation may be the primary cause of severe wilt. Groundnut + Castor (5:1) intercropping had the highest wilt incidence (48%), while Pigeonpea + Castor (1:1) intercropping had the lowest (1–6%). Wilt incidence in the Sunflower + Castor (2: l) intercrop was 32%. Under rainfed conditions, the castor crop was assessed as a component crop in several non-leguminous crops and legumes in a 10:1 and 6:1 row proportion, respectively. Intercropping urd bean with castor in a 6:1 row proportion produced the highest land equivalent ratio value of 1.85, as well as the maximum seed production. Wilt incidence was reduced in a castor-pigeonpea intercropping system" [47].

4.9 Physical Methods

"Raoof and Rao [22] investigated the effect of soil solarization on castor wilt in a wilt sick plot by covering it with a low-density transparent polyethylene sheet (200 gauge) and recorded that plots solarized for six weeks in summer had the greatest reduction in wilt incidence, *F. oxysporum* f. sp. *ricini* population, and nematode population (50%, 35%, and 78%, respectively) and the highest castor seed yield. Solarizing a wilt sick plot for three weeks during the summer season with transparent linear low-density polyethylene (LLDPE) sheets (25 µm) reduced wilt incidence by 38%, castor wilt pathogen population by 67%, and increased castor seed yield by 125% compared to non-solarized plots" [25].

4.10 Biological Control

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%) [48]. Janga et al. [49] found 42 antagonistic isolates from 500 bacterial isolates recovered from castor rhizosphere soil samples and chose four isolates: E37, P37, P40, and P46, which demonstrated disease suppression of 65-70%. Apurva et al. [50] investigated the antagonistic activity of various bioagents in vitro throughout 2019-20. Eight fungal and eight bacterial bioagents were tested using the dual culture approach. The Th14 strain of *Trichoderma harzianum* inhibited the mycelial growth of *F. oxysporum* f. sp. *ricini* the most (80.47%), whereas the remaining strains of both *T. harzianum* and *T. viride* were able to suppress the pathogen by at least 72% in vitro. In the case of bacterial antagonists, *Bacillus velezensis* (P42) shown superior inhibition of 58.89% over the others.

In greenhouse testing with castor cultivar GCH 4, seed germination was significantly higher in bioagent treatments compared to the pathogen control group. *P. fluorescens* Pf2 had the lowest wilt incidence of any bioagent (30%), being followed by *T. harzianum* ThN2 (45%) and *T. harzianum Th4d (55%).*

4.11 Chemical Control

Shalini et al. [51] "noticed that the fungicide carbendazim inhibited radial growth of the test pathogen *F. oxysporum* f.sp. *ricini* by 100% at both the recommended and half the recommended doses, while metalaxyl inhibited radial growth by only 77.86% at both the recommended and half the recommended doses". According to Jadav et al. [52], "laboratory screening of various fungicides revealed that thiram 75% WP (87.24%) was quite effective in inhibiting the radial growth of the test pathogen among non-systemic fungicides, whereas carbendazim 50% WP

(100%) and carbendazim 12% + mancozeb 63% WP (88.33%) significantly inhibited the growth of *F. oxysporum* f. sp. *ricini in vitro".*

4.12 Compatibility of the Seed Coat Biopolymer with the 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%) (Annonymous, 2016)

4.13 IDM

Dange et al. (2006) "revealed that using bio-agents such as *Trichoderma* spp. and chemicals such as carbendazim as seed treatments, as well as the use of resistant cultivars and correct cultural techniques such as soil solarization, results in excellent disease management". Shalini et al. [53] "examined the efficiency of biocontrol agents and chemicals against castor wilt, *F. oxysporum* f. sp. *ricini* in vitro and found that all treatments considerably reduced wilt incidence and increased plant growth when compared to the untreated inoculation control. The combination treatment of carbofuran at 2g/kg soil and carbendazim at 1g/kg soil was shown to be the most efficient in lowering wilt incidences and reniform nematode population, followed by *T. viride* at 4g/kg seed and P. fluorescens at 10g/kg seed".

Ghante et al. [54] "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 resulted in the lowest wilt incidence (7.12%) and the highest yield (458.38 kg/ha), respectively". "Seed treatment with carbendazim (2 g/kg seed), soil application of T. viride @ 2.5 kg mixed with 10 t FYM/ha, and intercropping of castor (4:1) with pigeon pea (PRG-100) were found to be more effective than farmers' practices of only applying chemical pesticides" [55]. "Carbendazim seed treatment resulted in a significantly lower wilt incidence (24.4%) and a higher seed production of 1123 kg/h in GCH-4. Seed treatment and soil application of *T. harzianum* Th4d WP resulted in a low wilt incidence (26%) and seed yield of 1016 kg/ha, whereas pathogen control resulted in a wilt incidence of 60.6% and a seed yield of 905 kg/h" [46]. Rajpurohit et al. [56] "found that seed treatment with *T. viride* at 10g/kg, followed by soil application at 2.5kg/ha, reduced percent wilt incidence from 41.6 to 7.2". Sudhakar et al. [57] "found that treating seeds with *T. viride* at 10g/kg and applying neem cake to the soil at 1 ton/ha were beneficial for wilt disease reduction".

Shalini et al. [53] "noted that 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 significantly effective, with a wilt incidence of around" 30% [58-60].

5. CONCLUSION

F. oxysporum is a worldwide pathogen that causes brownish or obstructing xylem. Castor plants are sensitive to wilt at all phases of growth; however, the disease most commonly arises during flowering and spike formation.

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

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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