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REVIEW ARTICLE

Comprehensive overview of host plant resistance and its progress in wilt resistance in castor bean (*Ricinus communis* L.)

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Abstract

Castor bean, a non-edible monotypic species known for its unique hydroxy fatty acid, is susceptible to a range of pathogens. Wilt caused by *Fusarium oxysporum f. sp. ricini* is the most complex and destructive disease. Chemical control of wilt has proven ineffective due to the systemic nature of the disease and the seed and soil borne nature of the pathogen. Physical, chemical, biological and integrated management methods have shown only limited success in controlling wilt. Host plant resistance, however, stands out as the most promising strategy, offering a viable pathway for the genetic improvement of castor for wilt resistance. Screening techniques are well established and several resistant donors have been identified. While significant progress has been made in understanding the inheritance of wilt resistance, a complete understanding of its genetic mechanisms still requires further research. Single Nucleotide Polymorphism (SNP) markers and genomic regions linked to wilt resistance have been successfully identified. Reniform nematode (*Rotylenchulus reniformis*) has been identified as a predisposing factor for wilt and genomic regions linked to nematode resistance have also been identified. However, Marker-assisted selection has not yet been utilized to develop wilt-resistant castor varieties. Further research is required to explore the pathogen diversity, host-pathogen interaction and mechanisms underlying wilt resistance including the metabolites responsible, for preventing the emergence of new pathogenic races and ensuring long-term protection against wilt.

Keywords

castor; *Fusarium* wilt; reniform nematode; host plant resistance; SNP markers

Introduction

Castor bean (*Ricinus communis* L., 2n=2x=20,) a member of the mono-specific genus *Ricinus*, is one of the oldest and most important non-edible oilseeds within the spurge family (Euphorbiaceae). It is an unventured oilseed crop with immense agricultural, industrial and pharmaceutical applications (1). It thrives well in hardy conditions and is widely cultivated in tropical regions of the world (2). India leads global castor production, contributing 1.96 million tons from 6,27,000 hectares, with an average productivity of 1,937 kg/ha (3). The country dominates the international market, exporting 83% of its castor oil to countries such as China, USA, Japan and Thailand (4), generating approximately Rs.7,000 crores in foreign exchange annually (5).

Castor seeds contain 50-55% oil with a high concentration of ricinoleic acid, which makes up 80-90% of the total fatty acids. Ricinoleic acid is an 18-carbon monounsaturated hydroxy fatty acid. The unique physical and chemical properties of castor oil, such as the presence of a unique trihydroxy functional group, and its renewable and biodegradable nature make it a valuable raw material in various industrial products (6). It is widely used in the production of paints, coatings, polishes, textile dyes, resins, polymers, waxes, soaps, medications, cosmetics and fragrances (7). Additionally, due to its high viscosity, castor oil serves as a lubricant in aircraft engines (8). Castor leaves are also used as feed for the Eri silkworm (9). It is cultivated as a pure crop, border crop and intercrop in rainfed situations (10, 11).

As a monotypic species, castor has seen significant exploitation of its naturally occurring genetic variability for improvement. One notable genetic trait is the pistillate condition-a rare recessive mutant in which the inflorescence consists entirely of female flowers, without male flowers (12). This trait holds great potential for use in heterosis breeding programs. In castor, hybrids are more commonly cultivated than traditional varieties (13). However, large-scale hybrid cultivation under intensive input systems, often without appropriate scientific management, has made the crop susceptible to various biotic stresses. A range of pests and diseases affect castor throughout its growing season. Major diseases include wilt, grey mold, root rot and seedling blight (14, 15). Among them, Wilt, a seed and soil-borne disease that can infect castor at any phenological stage, is particularly devastating and can result in significant economic losses (16).

Wilt is a systemic pathogen that is challenging to control once infection sets in. It can also persist in the soil for extended periods (17), making chemical control measures largely ineffective (18). While bio-control and integrated disease management strategies have been attempted, they have not yielded satisfactory results. Therefore, utilizing host plant resistance is considered the most effective approach for managing wilt. This method is simple, safe, cost-effective and significantly reduces environmental pollution, maintains ecological balance and minimizes health risks to humans.

The success of host plant resistance depends on several factors: evaluating different screening methodologies to identify the most effective one, identifying resistant donor lines, understanding the genetics of wilt resistance, studying host-pathogen interactions, examining any associations between wilt and other pathogens, exploring the role of molecular markers in screening cultivars, constructing linkage maps and locating the quantitative trait loci (QTL) for wilt resistance, selecting appropriate breeding techniques and ultimately developing high-yielding hybrids or varieties with integrated disease resistance (19). This review aims to provide a comprehensive overview of all aspects of host plant resistance to support the genetic improvement of castor for wilt resistance.

Wilt and its economic importance

Fusarium wilt was first identified in Brazil in 1937 and reported in India in 1974, in Udaipur, Rajasthan (16). The disease persists throughout the crop's growing season, with symptoms typically appearing in patches. Yield loss varies depending on the crop stage when wilt symptoms manifest. The disease has been shown to cause a 77% yield loss during the flowering stage, 63% loss at 90 days after sowing, and 39% loss in the final stages of crop development (20). Overall, *Fusarium* wilt can reduce yield by up to 40%, lower oil content by 1-2% and decrease seed weight by 8-14% (21). Additionally, (22) reported that each 1% incidence of wilt results in a yield reduction of approximately 1.86 kg/ha. Since *Fusarium* wilt is a soil-borne disease, managing it through conventional physical or chemical methods is extremely challenging (15, 23).

Pathogen

The disease was characterized, and its etiology was identified as *Fusarium oxysporum* f. sp. *ricini* (16). On semi-synthetic media, the fungus produces abundant white mycelium, which turns pink when incubated under fluorescent light. The fungus produces microconidia, macroconidia and chlamydospores. Despite being host-specific, the pathogen exhibits variability in its pathogenicity, with differences in cultural and morphological characteristics. *In vitro* studies investigating the production of pectinolytic and cellulolytic enzymes by the pathogen showed no correlation between pectic enzymes and the virulence of the fungus on castor plants. Furthermore, culture filtrates of *F. oxysporum* f. sp. *ricini* were found to significantly reduce seed germination, root and shoot elongation, fresh root and shoot weight, dry shoot weight and overall plant height, indicating the detrimental effects of pathogen infection (21).

Disease symptoms

The disease affects castor plants throughout the growing season, with the most significant damage occurring during the flowering, spike formation and capsule maturation stages. In wilt-endemic areas, seedling infection is common, with young seedlings exhibiting discolored hypocotyls, loss of leaf turgidity and symptoms of leaf blight. Infected plants often droop and eventually die (21).

During the pre-flowering stage, symptoms include yellowing of the leaves, marginal and interveinal necrosis, and senescence of lower leaves, leading to permanent wilting. Infected plants typically fail to produce inflorescences. At the flowering, spike formation and capsule maturation stages, symptoms worsen, with leaves turning yellow, developing marginal necrosis, becoming completely necrotic and eventually shriveling, resulting in plant death (24). Figure 1 illustrates these disease symptoms.

Additional signs of infection include browning and blackening of the xylem tissues, visible upon splitting the stem (25). Sick plants may either fail to produce seeds or yield dull, wrinkled seeds. Since this is also a seed-borne disease, seeds can transmit the infection up to 20%, particularly at the micropylar end, in 2-19% of cases (21).

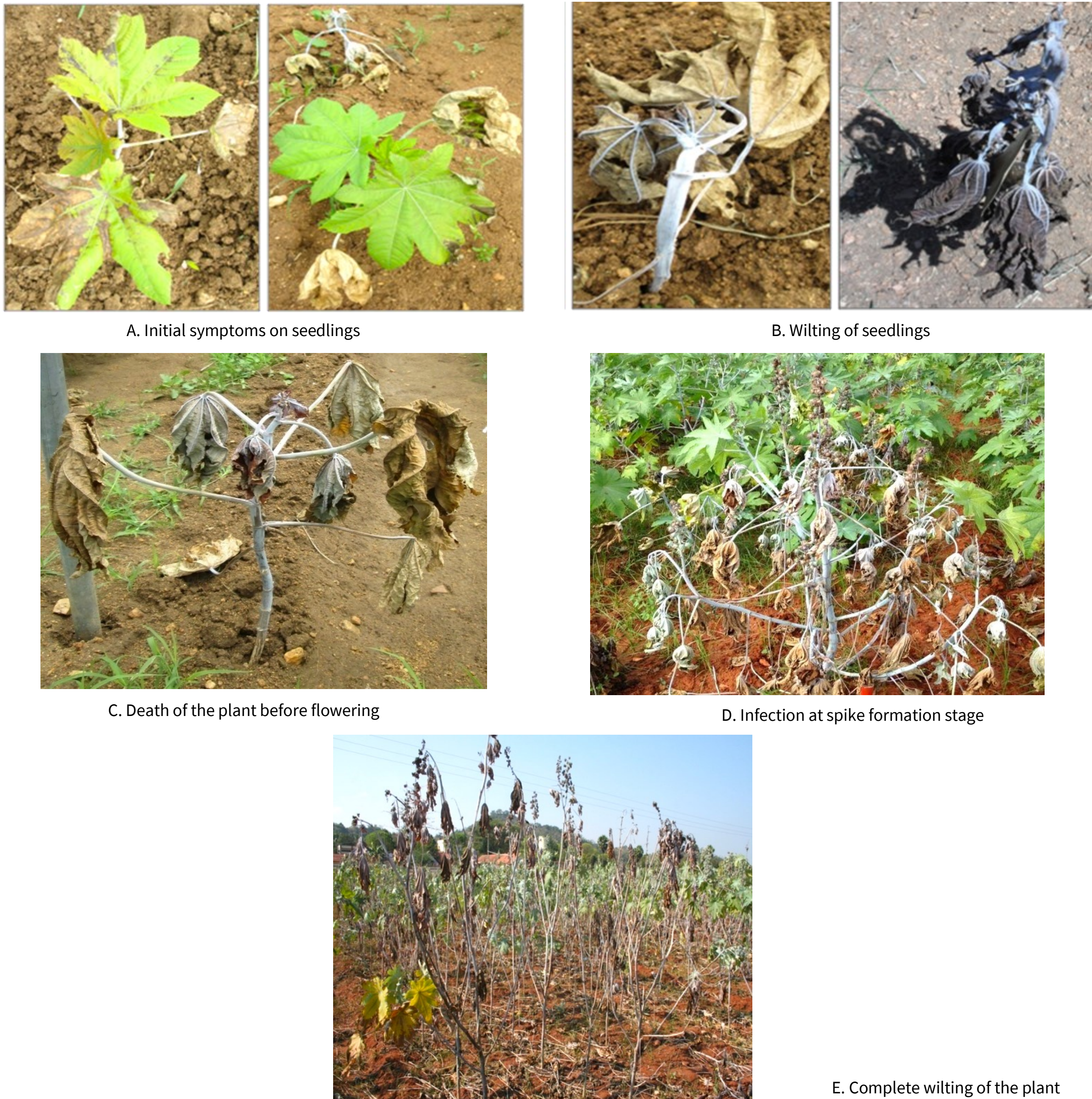

Fig. 1 (A-E): Symptoms of fusarium wilt observed at various stages

Epidemiology and Disease cycle

The fungus primarily spreads to new regions through seeds from infected areas (26). It produces thick-walled resting structures called chlamydospores, which can survive in the soil or crop debris for extended periods. When conditions become favorable, these chlamydospores germinate and produce two types of spores: microconidia and macroconidia. These spores germinate in moist soil and once they come into contact with plant roots, they infect the host. The fungus can also enter roots through natural wounds or punctures caused by nematode feeding. After penetrating the roots, the fungus spreads into the plant's xylem vessels, moving to other parts of the plant, where it blocks the vessels, ultimately causing wilting. Continuous monocropping over several years can create wilt-endemic areas (21). The most favorable temperatures for plant infection are between 13 and 15°C, with symptoms becoming more prominent at temperatures between 22 and 25°C (27).

Genetic diversity of the pathogen

Numerous wilt-resistant varieties and hybrids have been developed, but none have demonstrated consistent resistance across diverse geographical regions (28). This inconsistency highlights the dynamic nature of the wilt pathogen, which evolves continuously through mutations and recombination events. The variability of the wilt pathogen is a major factor in the breakdown of resistance in castor cultivars. Monitoring genetic diversity allows breeders to track pathogen evolution and anticipate potential shifts in disease dynamics. Understanding the genetic diversity of the wilt pathogen is crucial for developing strategies aimed at achieving durable and stable resistance against evolving pathogen strains (29). Additionally, screening castor genotypes against different isolates of *Fusarium oxysporum* f. sp. *ricini* helps to identify the differential responses of castor genotypes, enabling the categorization of race-specific resistant cultivars. These selected differential lines can also serve as valuable tools for studying the dynamics of the castor wilt pathogen.

Using the root dip inoculation method, twelve isolates were screened against seven castor genotypes, revealing varied pathogenic reactions among the test isolates. The genotypes SKI-293 and SKI-295 were identified as differential hosts for discerning pathogen variants (29). Five pathotypes were categorized based on the reactions of four distinct castor cultivars to the twenty-nine isolates. Additionally, genetic diversity assessment using RAPD markers grouped these twenty-nine isolates into five clusters. Interestingly, there was limited or no correlation between the categorization of isolates based on pathogenic variation and their clustering based on RAPD analysis (30). (28) noted significant diversity in the cultural and morphological traits among the twenty-two isolates of *F. oxysporum f.sp. ricini*. Screening of these isolates against nine castor genotypes revealed varying reactions to different isolates. Moreover, (31) also observed significant variation in the morphology, cultural traits and pathogenicity of fifteen isolates of *F. oxysporum* f. sp. *ricini*. A relationship was identified between virulence, sporulation and mycelial growth. More virulent isolates exhibited higher mycelial growth but lower sporulation, while less virulent isolates showed higher sporulation and lower mycelial growth.

Analysis of 146 isolates of *F. oxysporum* f. sp. *ricini* revealed 28-54% diversity using RAPD markers. Specific SCAR primers were designed based on two RAPD markers (OPJ-14 and OPK-12), which will be highly useful for the identification and separation of this pathogen from other species (32). Ten pathogenic races were identified among the 146 isolates; races 2, 3, 4, 5, 7, 8 and 10 were more prevalent in Andhra Pradesh, while races 1, 2, 4, 6 and 9 were more common in Gujarat (33). Comparative proteomic analysis demonstrated significant variation in protein patterns between the highly virulent isolate *Palem* and the less virulent isolate For *13-52*, *GJ*. A total of 200 protein spots were expressed, with 12 statistically significant spots showing more than a 1.5-fold increase in upregulation, while 8 spots were differentially present in *Palem*. The upregulated protein spots include 129, 3, 55, 48, 102, 84, 231 and 105. These proteins exhibited similarities to fungal proteins essential for pathogen entry, colonization, invasion, plant cuticle breakdown, detoxification of reactive oxygen species produced by the host and toxin synthesis. The differentially expressed protein spots include 175, 222, 208 and 164. The functions of two of these proteins remain unknown, while the roles of the other two were discovered to be involved in scavenging host-generated reactive oxygen species, breaking down host cell wall proteins and exhibiting antifungal activity (34).

Host-Pathogen interaction

Knowledge of host-pathogen interactions provides valuable insights into the mechanisms governing disease resistance and susceptibility. A comprehensive understanding of the molecular and biochemical mechanisms underlying these interactions enables breeders to identify specific genes or gene networks associated with resistance. This knowledge significantly enhances the ability to develop resistant crop varieties. Plants exhibiting disease resistance often produce various secondary metabolites, including phenolic acids, which are synthesized through phenylpropanoid metabolism. Expression analysis of key genes in the phenylpropanoid pathway revealed a significant increase in the expression of Phenylalanine Ammonia Lyase (PAL) and Cinnamate-4-Hydroxylase-2 (C4H-2) genes in both resistant and susceptible genotypes, regardless of wilt infection. Notably, the Cinnamate-4-Hydroxylase -1 (C4H-1) gene was upregulated in resistant genotypes but downregulated in susceptible ones. Furthermore, phenolic acid profiling using High-Performance Thin-Layer Chromatography (HPTLC) at regular intervals after wilt infection showed an increase in caffeic and ferulic acid levels in resistant genotypes, while caffeic acid was absent in susceptible genotypes. These findings clearly illustrate the crucial role of phenolic compounds in conferring wilt resistance in castor (35).

Lipoxygenases (LOX) are a class of physiologically active compounds that play a crucial role in plant defence mechanisms. They facilitate the oxygenation of polyunsaturated fatty acids, leading to the production of phyto-oxylipins. In castor, twelve candidate LOX genes have been identified and primers have been designed and amplified for six of these genes. The results indicated that all Rc-LOX genes, except for LOX 5, contain five conserved iron-binding sites within their LOX domains. In contrast, LOX 5 has unique consensus histidine residues at positions 547, 556 and 715, suggesting a distinct functional role. Notably, during wilt infection in castor, the expression of LOX 5 significantly increased in resistant genotypes (48-1 and SKP-84), further emphasizing its importance. Therefore, the Rc-LOX 5 gene could serve as a valuable marker for evaluating the response of castor genotypes to wilt infection (36). Enhanced activities of superoxide dismutase (SOD) and peroxidase (POX) were observed in the roots of susceptible cultivars, while higher catalase activity was noted in the roots of resistant cultivars. Following infection, there were significant increases in the activities of PAL, β-1,3-glucanase, and thiobarbituric acid reactive substances (TBARS). Ascorbate peroxidase (APX) and polyphenol oxidase (PPO) consistently exhibited higher expression levels. Specifically, SOD 3 and POX 5 were induced 24 and 48 hours after infection in resistant genotypes, respectively (37). In resistant cultivars, the activities of SOD, glutathione reductase (GR) and β-1,3-glucanase were elevated, whereas susceptible cultivars showed higher APX activity. Antioxidant enzymes such as SOD, GR and β-1,3-glucanase were released into the roots of resistant cultivars, effectively restricting the browning of xylem vessels. This indicates that resistant cultivars often display elevated activities of certain enzymes associated with defence responses (38).

The proteomic analysis aimed at identifying proteins released during *Fusarium* infection revealed the presence of 18 unique peptides in resistant genotypes compared to 8 unique peptides in susceptible ones. Notably, five genes-CCR1, Germin-like protein 5-1, RPP8, Laccase 4 and Chitinase -like 6 were significantly upregulated during wilt infection. Genes such as CCR1 and Laccase 4, which were involved in lignin production, were also upregulated, providing mechanical strength that may inhibit fungal mycelial entry. The upregulation of Germin-like protein 5-1 aids in

neutralizing reactive oxygen species through superoxide dismutase activity. Endpoint PCR analysis of cDNA indicated selective amplification of three genes-Chitinase-like 6, RPP8 and β-glucanase in resistant genotypes. This suggests that these genes play a crucial role in conferring resistance in castor. Functional genomics further supports the distinct roles of these genes in enhancing resistance and in the development of transgenic crops resistant to wilt (39).

Interaction of the pathogen with reniform nematode

Numerous nematode species infect castor plants, with the reniform nematode (*Rotylenchulus reniformis*) causing significant economic losses (40). This obligate, sedentary semi-endoparasite affects over 300 plant species (41), ranking second to root-knot nematodes (*Meloidogyne spp.*) in the host range. The reniform nematode interacts with pathogens that cause root rot and wilt diseases, leading to considerable losses. Unlike root-knot nematodes, it does not induce extensive tissue modifications, often remaining undetected despite high population densities (42). Reports indicate symptoms such as die-back, stunting and reduced growth in castor plants heavily infested with reniform nematodes (43). In India, yield loss estimates indicate financial losses of approximately Rs. 180 million, translating to a 13.93% reduction in yield (44).

Research on the interactions between reniform nematodes and *Fusarium* wilt demonstrates that the nematode can render even the wilt-resistant hybrid GCH-4 susceptible to wilt due to its predisposing nature. Earlier onset of disease was observed in various combinations of nematode and fungal treatments compared to fungal treatment alone. This synergistic effect of the nematode-fungal complex diminishes the growth of both wilt-susceptible and resistant hybrids (45). Interaction studies between *Rotylenchulus reniformis,* and *Macrophomina phaseolina* revealed that the maximum plant mortality of 4.7% occurred with combined inoculation of the reniform nematode and root rot fungus, compared to inoculation with either pathogen alone or with sequential treatment of *Macrophomina* followed by the nematode or the nematode followed by *Macrophomina* (46). It is evident from the studies that *R. reniformis* has been implicated in the wilt complex, indicating its role as a predisposing factor for wilt incidence and a significant contributor to the progression of wilt disease (41).

Host plant resistance and its importance

Wilt is a systemic and vascular disease caused by a soil-borne pathogen, making physical and chemical management approaches difficult (18). Chemical control methods are often short-term and can result in significant environmental hazards. Therefore, the most effective and sustainable strategy for managing wilt is the host plant resistance (47). This approach provides long-term benefits by reducing environmental pollution, maintaining ecological balance and eliminating health risks to humans. The success of host-plant resistance hinges on several key factors, such as evaluating different screening methodologies to identify the most effective one, identifying resistant donors, understanding the genetics of wilt resistance, deciphering host-pathogen interactions, utilizing molecular markers to accelerate the screening process, constructing linkage maps and conducting QTL analysis to identify genomic regions associated with resistance to both wilt and reniform nematodes, selecting appropriate breeding techniques to incorporate resistance into elite varieties. By addressing these factors, it becomes possible to select resistant and desirable genotypes for the development of high-yielding, wilt-resistant hybrids or varieties (48).

Screening methodologies

Root dip inoculation technique

Screening germplasm under controlled conditions is faster and more accurate than open-field screening, as it ensures favorable conditions for disease infection. The root dip inoculation technique involves germinating castor seeds in sterile sand, trimming the roots of seedlings, dipping them into a pathogen spore suspension and planting them in pots. The process starts with surface sterilization of the seeds, which are then sown in autoclaved riverbed sand. The wilt pathogen is isolated from the roots of already infected plants and purified through single spore isolation. Sterilized sorghum grains are inoculated with the fungal culture and incubated for 10-14 days at 27 ± 2 °C, during which the grains become colonized by the fungal mycelium. To prepare the spore suspension, a few infected sorghum grains are placed into distilled water, achieving a concentration of 1×10^6 spores/ml. Seedlings that are 10 days old are carefully uprooted, their root tips and trimmed and immersed in the spore suspension for 1-2 minutes before being transplanted into pots containing autoclaved soil. Seedlings dipped in sterile distilled water serve as the control group. For up to 30 days post-transplantation, the seedlings are closely monitored for signs of wilt, and wilt incidence is regularly recorded. This method is used alongside open-field screening (sick plots) to validate wilt resistance in the accessions (25). This method enables the screening of a large number of entries in a shorter timeframe and allows for the identification of even low levels of resistance, which can be leveraged in breeding programs to develop high-yielding, resistant varieties (24). Furthermore, this approach is highly valuable for studying the pathogenic diversity among *Fusarium oxysporum f. sp. ricini* isolates (49).

Sick plot technique

It is the most widely used, accepted and effective method for screening castor genotypes for wilt resistance. It offers clear differentiation between susceptible and resistant genotypes, enabling the screening of large numbers of genotypes efficiently (49). In this method, wilt-affected plants collected from existing fields are incorporated into the soil during ploughing. Before sowing, the inoculum load must be carefully checked and adjusted to 2×10^3 colony-forming units (cfu) per gram of soil. To ensure the accuracy of the screening, susceptible and resistant genotypes are sown every four to five rows. After 60 days, the inoculum load is rechecked and maintained at the optimal level. Germination percentage is recorded between 7-10 days after sowing and wilt incidence is observed at 30-day intervals from sowing up to 150 days, with the number of wilted plants recorded for each entry (15).

The effectiveness of this method was highlighted by researchers who screened genotypes based on the number of days to wilt in sick plots. Their results demonstrated that this kind of scoring is more efficient, reproducible and thorough, with no disease escape-a common limitation of normal screening procedures. The scoring system they used categorized plant resistance into four scales based on the number of days to wilt post-sowing (Table 1). Furthermore, they emphasized that inconsistent results in inheritance studies can often be attributed to variations in the screening method employed when using different resistant and susceptible parents (50).

Table 1. Grading of genotypes to wilt based on the number of days to wilt in sick plot (50)

Scale	Category of resistance	Wilt reaction
1	susceptible	plants wilted before 30 days of sowing
2	moderate	plants wilted from 31 to 50 days after sowing
3	resistant	plants wilted from 51 to 65 days after sowing
4	highly resistant	plants survived beyond 65 days after sowing

Sick pot technique

The sick pot technique is another approach used for screening castor genotypes for wilt resistance, which involves cultivating *Fusarium* on sterilized sorghum grains for 10-14 days (25). To prepare the medium, sorghum grains are soaked overnight in a solution containing 5% sucrose per litre and 30 mg/l of chloramphenicol, followed by autoclaving at 15 psi for 20 minutes at 121°C. Fungal mycelium discs (4-5) are then added to inoculate the grains, which are incubated for 15 days at 28 ± 2°C. After the incubation period, 3 grams of the fungal culture are mixed with every kilogram of potting soil, ensuring an initial mycelial load of 1×10^3 cfu per gram of soil. The inoculum load is then adjusted to 2×10^3 cfu per gram. Pots with sterile soil serve as controls for comparison (15).

Technique for reniform nematode - wilt complex screening

As reniform nematode is a predisposing factor for wilt, the technique for screening castor genotypes against the reniform nematode-wilt complex is crucial for identifying genotypes that exhibit combined resistance to both wilt and nematodes. In this approach, sterilized seeds are sown in earthen pots and after seedling emergence, *Fusarium* culture is mixed with the upper surface of the soil. Additionally, around 1,000 immature females of reniform nematode are inoculated into the root zone of the seedlings by gently removing the soil around the collar region. Wilt incidence is monitored periodically, starting one week after nematode inoculation and continuing up to 30 days after inoculation. Genotypes are classified based on wilt incidence: genotypes with 0% wilt incidence are classified as highly resistant, those with 20% wilt incidence as resistant and those with >20% incidence as susceptible. This screening method is highly effective for evaluating large numbers of castor genotypes for nematode-wilt complex resistance under greenhouse conditions in a relatively short time (21).

Identification of resistant sources

Since wilt is a soil-borne and vascular disease, controlling it with fungicides is highly challenging. Effective control can only be achieved through the use of resistant varieties. Therefore, it is essential to screen vast germplasm collections using standard protocols to identify resistant sources that can be used as donors in breeding programs aimed at developing wilt-resistant varieties (15). Numerous resistant donors have already been identified in castor, which are listed in Table 2.

Genetics of Wilt resistance

Several resistant donors have been identified in castor germplasm collections, and understanding the inheritance of wilt resistance in these accessions is crucial for identifying diverse resistance genes and establishing their allelic relationships. This knowledge aids in developing durable wilt-resistant hybrids (19, 63). Different studies have reported various modes of inheritance for wilt resistance based on the parental materials used, as summarized in Table 3. Notably, distinct inheritance patterns have been observed when the same resistant parent is crossed with different susceptible parents. For example, when the resistant parent 48-1 was crossed with JI 35, it exhibited monogenic inheritance. However, when crossed with JC12, the inheritance pattern shifted to digenic with complementary gene action. The common locus, r1, confers wilt resistance in 48-1 in both crosses. In the JC12 × 48-1 cross, another locus, r2, from 48-1 interacted with r1, resulting in a 9:7 segregation ratio in the background of the susceptible parent JC12. The inconsistency in the inheritance patterns observed in different studies is largely attributed to variations in the screening methods employed (50).

Mutation breeding for wilt resistance in castor

Castor is a monotypic species, which results in narrow genetic diversity within the species. To enhance this diversity, controlled mutations can be induced to introduce novel genetic traits, including resistance to wilt. Mutation breeding exploits physical and chemical mutagens to cause random changes in nucleotide sequences, thereby increasing genetic diversity and improving the overall genetic potential of the castor. Selection for wilt resistance was done by the irradiation of the wilt-susceptible pistillate line VP 1, leading to the development of five wilt-resistant pistillate lines: M 619, M 571, M 568, M 574 and M 584 (70). Additionally, a popular pistillate line, DPC 9, which is highly susceptible to leafhoppers linked with the zero-bloom trait, was also mutated. In the M_5 generation, segregation for bloom character was observed and intense selection pressure for bloom resulted in the identification of nine DPC 9 mutants with diverse morphological traits. Among these, IPC 23 emerged as an outstanding pistillate line, demonstrating high resistance to both leafhoppers and wilt compared to their respective checks (71).

Chemical mutagen Ethyl Methane Sulphonate (EMS) has also been utilized to develop wilt-resistant parental lines in castor, specifically in the pistillate line DPC 9 and the landrace Rasipuram Local. The pistillate

Table 2. Resistant sources for Fusarium wilt

S.No.	Resistant sources	References
1.	DPC 9, DPC 16, Geeta, M 574, JP 96, SKP 84, and M 619–1.	(15)
2.	48-1, ANDCI-10-1, ANDCI-10-2, ANDCI-10-3, ANDCI-10-5, ANDCI-10-7, ANDCI-10-12, ANDCI-12-1, DCS-107, DCS109, DCS-119, JI244, JI342, JI402, JI403, MI35, MI41, RG1673, RG1941, RG1954, RG2561, RG2800, RG3160, RG3749, GAC11, SKI92, SKI255, SKI31	(51)
3.	IPC 46, BCS 3, ICS 253, ICS 309	(52)
4.	ICI-RG-2774-1, RG 4007, RG 4011, RG 4014, RG 4017, RG 4025, RG 4026, RG 4139 and RG 3390	(53)
5.	ICS-303, ICS-304, ICS-305, ICS-312, ICS-314, ICS-315, ICS-316, ICS-318, ICS-319 ICS-320 ICS-321	(54)
6.	RG 1624	(55)
7.	HCG-1, HCG-6, MI-68, MI-71, MI-73, MI-83, MI-86, MI-88, MI-93, 48-1, HCG-20, HCG-35, HCG-36, HCG-37, HCG-38, HCG-43, HCG-45, HCG-48, HCG-50, K. LOCAL, HCG-1, HCG-6,	(56)
8.	RG 2430, RG 2818, RG 111, RG 224, RG 297, RG 558 and RG 28	(57)
9.	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	(58)
10.	DCS-86, DCS-105, DCS-107, DCS-118, DPC-23, M571	(59)
11.	RG 1221, RG 1624, RG 2741, RG 2781, RG 2787, RG 2800, RG 3105 and RG 3093	(60)
12.	RG 2800	(61)
13.	RG 815, RG 844, RG 1146, RG 1221, RG 1577, RG 1697, RG 1766, RG 2100, RG 2720, RG 2759, RG 2924, RG 3225, RG 3242, RG 3253, RG 3292, RG 3296, RG 3336, RG 3338, RG 3352, RG 3352, RG 3359, RG 3361, RG 3378, RG 3383, RG 3386, RG 1714, RG 2093, RG 2145, RG 2161 and RG 2254	(62)
14.	RG43, RG111, RG109, RG297, RG1608, RG1624, RG2758, RG2787, RG2800, RG2818, RG2822, RG3016 RG3105 and RG3322	(63)
15.	RG-13, RG 21, RG 38, RG 425, RG 430, RG 441, RG 445, RG 453, RG 457, RG 534, RG 572, RG 587, RG 709, RG 743, RG 788, RG 789, RG 811, RG 819, RG 831, RG 848, RG 876, RG 903, RG 920, RG 937, RG 957, RG 969, RG 972	(64)
16.	RG 1930 and RG 2008	(65)
17.	RG71, RGS8, RG 231-3, RG232, RG234, RG297, RG318, RG319, RG354, RG913, RG948, RG971, RG845, RG982-1, RG40-1, KA13, KA16, KA34, KA5, KAll, KA40, KA41, KA26, KA29, KA77, KA6, KAJ, RGl441, RG656, RG64I, RG776, RG777, RG764, RG772, RG489, RG508, RG73, RG63, RG784, RG786	(24)

Table 3. Genetics of wilt resistance

S.No.	Genetics	References
1.	monogenic recessive gene or duplicate gene action	(26)
2.	Monogenic recessive	(66)
3.	Monogenic dominant and digenic complementary gene action	(67)
4.	Complementary gene action	(19)
5.	Polygenes	(48,68)
6.	Monogenic dominant	(32,33,69)
7.	Digenic duplicate, complementary and Inhibitory gene action	(63)
8.	monogenic recessive, digenic dominant and digenic recessive complementary gene action	(50)
9.	monogenic recessive, digenic complementary, duplicate dominant and duplicate recessive gene action	(15)

progeny YRCP 2 and the monoecious progeny YRCS 1904 exhibited maximum seed yield combined with wilt resistance, making them suitable candidates for developing high-yielding wilt-resistant hybrids or varieties (72). In a separate study, researchers standardized the dosage of EMS by testing three concentrations (0.5%, 1% and 1.5%) for durations of 4, 8 and 12 hours, following two pre-soaking times (12 and 24 hours) in water. The findings suggested that the ideal lethal dose for inducing mutations involved pre-soaking the seeds in water for 12 hours, followed by 1.0% EMS treatment for either 8 or 12 hours, resulting in a germination rate of 52% (73).

Molecular markers for wilt resistance in castor

Screening for wilt resistance in castor using molecular markers is crucial, as it offers a more accurate and effective approach compared to traditional methods. Given that castor is a long-duration perennial crop, molecular markers enable the early selection of resistant individuals, even at the seedling stage, before the expression of wilt symptoms.

To identify molecular markers associated with wilt resistance, mapping populations were developed from two crosses: Kranthi x Haritha and Kranthi x Jwala. A total of 160 RAPD primers were utilized to assess parental polymorphism, revealing 56 polymorphic primers for the Kranthi x Haritha cross and 48 for the Kranthi x Jwala cross. Screening of these polymorphic primers in bulked segregant populations indicated that primers H12 and J15 were polymorphic between resistant and susceptible bulks for the Kranthi × Haritha cross, while J15 was identified in the Kranthi × Jwala cross. These markers are closely associated with *Fusarium* wilt resistance genes in castor (33). Similarly, for the crosses Haritha × Kranthi and 48-1 × Kranthi, F_2 and BC_1F_1 mapping populations were generated, and parental polymorphism was assessed using 186 RAPD primers. This led to the identification of 16 and 21 polymorphic primers for the respective crosses. Among these, two RAPD markers, OPJ-154268 and OPH-124973, were closely associated with the resistant lines 48-1 and Haritha, located at distances of 7 cM and 5 cM, respectively (32).

In another study, three markers-RKC 231375, RKC 211080 and OPBE 18900 were found to flank the wilt resistance gene in a bulked segregant analysis of 200 F_2 individuals from the cross 48-1 x VP-1. Linkage analysis placed these markers at distances of 5 cM, 10.7 cM and 7.6 cM from the wilt-resistant gene FOR 1 (69). Further investigations involved the F_2 mapping populations of crosses RG-27 x JI-35 and RG-2944 x JI-35 to identify SNP markers linked to wilt resistance. Scoring and genotyping using SNP markers revealed two SNP markers, Rc_30146-1221543 and Rc_29706-482910, co-segregating with the phenotypic data in RG-27 x JI-35. Additionally, four SNP markers-Rc_30152-1185440, Rc_30152-1283827, Rc_29852-1074057 and Rc_30061_63432- were found to co-segregate with phenotypic data in RG-2944 x JI-35 (74). In the cross 48-1 x JI-35, an F_2 population was also screened for wilt using the Kompetitive Allele Specific Polymorphism (KASP) assay. Two polymorphic markers, Rc_30146-1103419 and Rc_28694-84511, that were physically closer to the QTL-associated marker, Rc_30146-1221543, were utilized to genotype the F_2 plants. Both markers co-segregated with the observed phenotypes, allowing for the prediction of resistant phenotypes with greater than 90% accuracy (75). Additionally, eight wilt-resistant lines were crossed with the susceptible line JI 35 and their F_2 populations were screened for wilt in a sick plot. SNP markers were employed to locate the resistance loci, identifying two SNP markers, Rc_29706-482910 and Rc_29609-103709, on chromosome 7 and one marker, Rc_43141-440, on chromosome 8, linked to wilt resistance (76).

Linkage map construction, Quantitative Trait Loci mapping and Association mapping for wilt resistance

Linkage mapping and QTL mapping are essential techniques for identifying the genetic factors associated with wilt resistance in castor. While linkage mapping helps physically locate the genes or genetic markers linked to wilt resistance on chromosomes, QTL mapping dissects the exact genomic locations associated with the trait. These methods greatly facilitate Marker Assisted Selection (MAS) for improving wilt resistance in castor. In one study, (61) screened parental lines for *Fusarium* wilt using 786 markers, including 520 RAPD, 100 ISSR and 166 SSR markers, identifying 141 polymorphic markers for the mapping population. A QTL associated with wilt resistance was found in linkage group 6 at a 90% threshold value, along with a putative QTL in linkage group 8. However, when the threshold value was raised to 95%, only the QTL on linkage group 6 was confirmed. This QTL was located near the markers CST 73 and R 83, with a LOD score of 13.5, indicating its significance.

(77) developed two linkage maps using crosses JC 12 × 48-1 and DCS 9 × RG 1139, incorporating 1090 and 1273 SNP markers, respectively. The JC 12 × 48-1 map spans 1139.8 cM with 82 to 207 markers per linkage group, yielding an average inter-marker distance of 1.12 cM, indicating a high marker density. The DCS 9 × RG 1139 map covers 904.8 cM with 45 to 183 markers per linkage group, resulting in an average inter-marker distance of 0.81 cM, showcasing dense mapping. Additionally, a consensus map was constructed using 1978 SNP markers, with 392 markers shared between the two crosses. This consensus map spans 995.8 cM, with an average distance of 0.55 cM between markers and 9 to 75 common markers per linkage group. QTL mapping was conducted using the linkage map generated from 1090 SNP markers in 185 F_6 Recombinant inbred lines (RILs) of the cross JC 12 × 48-1. A significant QTL was identified on chromosome 7, exhibiting a LOD score of 18.7 and explaining 44% of the total phenotypic variance. This QTL also displayed an additive effect of 8.11, emphasizing its potential value in breeding programs aimed at enhancing wilt resistance in castor (47).

Association mapping is a powerful population mapping approach that utilizes a diverse array of individuals from natural populations as the mapping population. This method capitalizes on the linkage disequilibrium (LD) between genetic markers and target genes to identify associations between markers and traits. One of the key advantages of association mapping is its ability to detect markers positioned much closer to the genes of interest than conventional linkage mapping allows. In a study analysing 3465 SNP loci from 300 genetically diverse castor germplasm accessions, 69 significant marker-trait associations for wilt resistance were uncovered. These associations were distributed across chromosomes 1, 2, 3, 4, 5, 8 and 9, with a significant concentration on chromosome 8, which harbored 46 associated SNPs. Notably, many significant SNPs clustered on chromosomes 4, 5 and 8. Interestingly, the QTL identified in the RIL population of the cross JC 12 × 48-1 did not coincide with the findings from the association analysis. This discrepancy suggests that there is a broad genetic diversity in castor regarding wilt resistance, indicating the potential for selecting diverse genetic sources to enhance breeding efforts aimed at developing wilt-resistant castor varieties (47).

Molecular markers and QTL mapping for reniform nematode resistance

As reniform nematode is a predisposing factor for wilt, identification of QTLs linked to nematode resistance is very crucial. Combined selection for both wilt and nematode resistance can be effectively achieved using molecular markers. In a study involving a recombinant inbred line (RIL) population derived from the cross JC-12 × 48-1, genotypic analysis using 957 SNP markers identified four genomic regions associated with reniform nematode resistance. The flanking markers linked to these regions were Rc_29949-26898, Rc_29647-244577, Rc_28151-12413 and Rc_29666-381712, located on linkage groups 3, 5, 6 and 8, respectively (74). Another study using the same RIL population and a linkage map constructed with 1,090 SNP markers identified additional QTLs. One QTL, linked to resistance, was found at the 79th position on chromosome 6, flanked by markers Rc_29666-381712 and Rc_29666-471509. A second QTL was mapped to the 66th position on chromosome 8, flanked by markers Rc_28037-33296 and Rc_28151-12413 (41). These flanking markers hold significant potential for use in MAS for reniform nematode resistance in castor.

Conclusion

This review comprehensively covers all aspects of host plant resistance to wilt in castor. The use of susceptible hybrids or varieties lacking durable wilt resistance poses significant

risks, as it not only causes substantial yield losses and economic setbacks for farmers but also increases the inoculum load in the soil. Given that wilt is a soil-borne vascular disease, host plant resistance remains the most effective and sustainable strategy for managing the disease, as chemical control measures are neither environmentally nor financially viable. Since nematodes are a predisposing factor for wilt, emphasis should be placed on screening for combined resistance to both wilt and nematodes to identify donors with dual resistance. The genetics of wilt resistance still require further investigation due to the variability caused by differences in parental materials and screening techniques.

The varieties/hybrids developed that are exhibiting resistance to wilt alone are considered and notified for release. Castor, being a monotypic species, exhibits narrow genetic diversity. The development of wilt-resistant hybrids often results in reduced yield and lower heterotic levels of the hybrids. Therefore, it is crucial to develop wilt-resistant hybrids without compromising yield or heterosis. Controlled mutations could be employed to introduce novel genetic traits that enhance resistance to wilt while preserving or improving the yield and heterotic potential of castor hybrids. Although SNP markers and genomic regions associated with both wilt and nematode resistance have been identified, there are no reported cases of MAS being used to develop cultivars that are resistant to wilt and nematode.

Fine mapping of wilt resistance genes will further enable the precise identification of genomic regions responsible for resistance. The introgression of genomic loci linked to wilt resistance into agronomically superior, high-yielding varieties or hybrids is essential for developing durable wilt-resistant hybrids. The integration of MAS has the potential to significantly enhance breeding efficiency and precision, enabling faster and more accurate selection for wilt resistance. As *Fusarium* is a highly dynamic pathogen, addressing pathogen diversity is critical for preventing the emergence of new races and the subsequent breakdown of resistance in current cultivars. To achieve long-term resistance, race-specific resistance genes should be identified and pyramided into a single genotype to develop stable and durable cultivars. Understanding the host-pathogen interactions and the mechanisms underlying wilt resistance, including identifying key metabolites and genes, is also crucial for advancing disease resistance research. This review provides valuable insights for future genetic studies on wilt resistance in castor and contributes significantly to the genetic improvement of the crop.

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Authors' contributions

RHH and SRV conceived the idea for this manuscript. RHH conducted the literature review and drafted the initial manuscript. PA, PAS and SKN provided critical feedback to the manuscript. RHH and SRV prepared the final version of the manuscript. All authors read and approved the final manuscript for submission.

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