ARTICLE

Evaluating common bean dual resistance to root-knot nematode and *Fusarium* wilt in recombinant inbred lines

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Abstract: *Common bean yield (Phaseolus vulgaris L.) is severely limited by diseases caused by root-knot nematode (Meloidogyne incognita) and Fusarium oxysporum f. sp. phaseoli (Fop). This study assessed co-infection effects by these phytopathogens in 73 F_8 recombinant inbred lines (RILs) derived from the 'IAC-Tybatã' × 'Branquinho' cross to identify genotypes with dual resistance. Plants received three treatments: control (water), Fop (root immersion in 1.0 × 10^6 conidia mL^{-1} suspension), and M. incognita (5,000 eggs/plant) + Fop. Co-infected plants developed significantly more pronounced vascular discoloration compared to plants inoculated with Fop alone. While 76.8% of Fop-inoculated RILs showed mild-to-moderate symptoms, 86.3% of co-infected RILs exhibited moderate-to-severe discoloration. Six RILs (1, 5, 8, 25, 31 and 47) showed dual resistance, displaying mild Fop symptoms and minimal nematode gall formation. The identification of these resistant genotypes is a critical breeding objective for developing common bean cultivars with enhanced protection against these damaging co-occurring pathogens.*

Keywords: Fusarium oxysporum *f. sp.* phaseoli, Meloidogyne incognita, *nematode-fungus synergism, genetic resistance, coinfection*

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is a staple crop in Brazil, the world's leading producer (FAOSTAT 2025). However, yields remain below potential due to biotic stresses (Junaid et al. 2014), including Fusarium wilt (*Fusarium oxysporum* f. sp. *phaseoli* - Fop) and root-knot nematodes (RKN). RKN induce root galls that disrupt water and nutrient uptake, leading to substantial crop yield losses. Juveniles penetrate roots, establish feeding sites, and manipulate host defenses through secretion of effectors that suppress resistance mechanisms (Khan and Sharma 2020). Control is hampered by persistence of nematodes in the soil and their high reproductive rate (Roberts 1992).

Mardani et al. (2024) evaluated the nematicidal efficacy of two abamectin formulations under commercial greenhouse conditions and reported that both treatments resulted in a significant suppression of nematode populations.

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However, continuous applications of nematicides may lead to the development of resistance in *Meloidogyne incognita* populations (Huang et al. 2016).

Fusarium wilt, intensified by successive cultivation, leads to chlorosis, vascular necrosis, defoliation, and plant death (Pereira et al. 2013, Batista et al. 2017, Quadros et al. 2021). Fop infects plants through lateral roots, colonizing the xylem and disrupting water transport. Resistant cultivars mitigate these symptoms through increased phenylpropanoid enzymatic activity, which strengthens vascular walls through lignin deposition, thereby limiting pathogen multiplication and spread (Quadros et al. 2019, Garcés-Fiallos et al. 2022). Fungicides are not effective in controlling the resistance structures of Fop (Batista et al. 2017), which it forms in the absence of a host plant.

Fusarium oxysporum f. sp. cepae triggers a strong antioxidant response in resistant onion genotypes, characterized by the transcriptional upregulation of catalase, peroxidase, and superoxide dismutase activities (Poursakhi et al. 2025). Complementarily, cluster analysis of Fusarium wilt resistance revealed a high degree of concordance between phenotypic classifications and molecular groupings, indicating a strong correlation between disease severity and genetic profiles associated with Fusarium wilt resistance (Sadeghpour et al. 2023).

Meloidogyne spp. and Fop often interact synergistically: nematode-induced galling creates physical entry points for the fungus, while suppressing jasmonate/ethylene defenses, thereby overcoming host resistance and increasing wilt severity (Hillocks and Marley 1995). This situation is further exacerbated by climate change, as elevated temperatures and altered water regimes amplify Fop proliferation and compromise plant health (Batista et al. 2017). These conditions make development of dually resistant cultivars a key management strategy.

In this context, the aim of this study was to evaluate a contrasting F_8 recombinant inbred line population derived from the 'IAC-Tybatã' × 'Branquinho' cross and to select genotypes with dual resistance to Fop and *M. incognita* for use in bean breeding programs.

MATERIAL AND METHODS

A F_8 recombinant inbred line (RIL) population (n = 73), derived from crosses between the nematode-resistant cultivar 'IAC-Tybatã' and the susceptible cultivar 'Branquinho' (Giordani et al. 2021), was evaluated for resistance responses to Fop and *Meloidogyne incognita*. The following treatments were used: 1. control (water), 2. Fop inoculation, and 3. *M. incognita* + Fop co-inoculation. A randomized block design was used with three treatments and three replicates. The experimental unit was one plant per 0.8 L pot. The Fop isolate UFV01 from the 'Meia Noite' cultivar in Coimbra, MG, Brazil (Pereira et al. 2013), and *M. incognita* race 3 from west-central Brazil (lat 15° 33' 60" S, long 55° 10' 08" W) were used (Orsi et al. 2025).

Seeds were germinated in a BOD chamber at 25 °C and transplanted two days later into a sterilized 1:1:1 (soil:sand:substrate) mixture in 123-cell trays. The seedlings were kept in a greenhouse for 10 days.

Meloidogyne incognita eggs were initially extracted from infected tomato roots (*Solanum lycopersicum* 'Santa Clara VF5600') at 60 days after sowing tomato. The inoculum was then prepared according to the method of Hussey and Baker (1973), as modified by Bonetti and Ferraz (1981). To establish the nematode-infection treatments, 5,000 eggs per plant were pipetted into holes made in the soil within the root zone. Fop inoculation was performed using the root immersion method (Pastor-Corrales and Abawi 1987, Paulino et al. 2020). Seedlings were carefully removed from their tray cells, and the roots were washed to remove adhering substrate. Then, 1/3 of each root system was excised, and the roots were immersed in a conidial suspension (1×10^6 conidia mL^{-1}) for 20 minutes. For the co-infection treatment, plants were first inoculated with *Meloidogyne incognita*. One week later, they were inoculated with Fop, applying the conidial suspension (1×10^6 conidia mL^{-1}) through the same pipetting procedure as for the nematode inoculation, without removing the plants from their pots.

The incidence of Fusarium wilt was evaluated at 14, 21, and 28 DAI (days after inoculation). For the final assessment, plants were removed from the pots. Disease severity was quantified using a scoring scale reflecting the degree of vascular discoloration in the xylem, a symptom of infection by *Fusarium oxysporum* (Pastor-Corrales and Abawi 1987). The following scale was used: 1 for healthy tissue, 3 for mild discoloration, 5 for moderate discoloration, 7 for severe discoloration, and 9 for a dead plant. Genotypes were then classified as resistant (R) when obtaining scores from 1.0 to

3.0, and susceptible (S) when obtaining scores from 3.1 to 9.0 (Salgado et al. 1995).

The roots were washed and stained to allow evaluation of gall formation caused by *Meloidogyne incognita* (Taylor and Sasser 1978). Root gall severity was assessed using a scale adapted from Hussey and Janssen (2002), where 0 = no galls in the root system, 1 = signs of infection, with a small number of galls, 2 = < 25% of roots with galls, 3 = 25-50% of roots with galls, 4 = 51-75% of roots with galls, and 5 = > 75% of roots with galls. Based on these scores, genotypes were classified as resistant (R) with scores of 0.0–1.0, or susceptible (S) with scores of 2.0–5.0.

Data from the final assessment of disease severity caused by *Fusarium oxysporum* were analyzed using R software (R Core Team 2025). Treatment means were compared using the Scott-Knott test at a 5% level of significance.

RESULTS AND DISCUSSION

Plants under co-infection exhibited significantly more pronounced vascular discoloration from the onset of the evaluation compared to plants inoculated with Fop alone (Table 1). Vascular discoloration is the main symptom of Fusarium wilt (Borba et al. 2017). At 28 DAI, the severity of vascular discoloration in the RILs differed: 76.8% of plants inoculated with Fop alone exhibited mild-to-moderate vascular discoloration, whereas 86.3% of co-infected plants showed moderate-to-severe vascular discoloration (Table 2). This accelerated vascular discoloration progression in co-infected plants (Table 2) is consistent with findings from a previous study in bean tissues, where nematode penetration and establishment facilitated subsequent Fop colonization (Francl and Wheeler 1993).

In the study conducted by Yaseen et al. (2024), okra cultivars showed increased susceptibility to *Fusarium oxysporum* f. sp. *vasinfectum* wilt when inoculated sequentially with *Meloidogyne incognita* followed by the fungal pathogen. Conversely, resistance to Fusarium wilt was observed when both pathogens were inoculated concurrently or when *F. oxysporum* f. sp. *vasinfectum* was introduced prior to *M. incognita*. Similar to that experimental framework, the present study employed a sequential inoculation approach in which the F_8 RIL population was first inoculated with the nematode and subsequently with *F. oxysporum* f. sp. *phaseoli* (Fop), following the methodology of Yaseen et al. (2024).

Table 1. Disease severity progression caused by *Fusarium oxysporum* f. sp. *phaseoli* (Fop) in a F8 recombinant inbred line (RIL) population inoculated with Fop alone and with *Meloidogyne incognita* + Fop

Treatments	**Vascular discoloration** (scores)		
	14th day	**21st day**	**28th day**
Fusarium oxysporum f. sp. *phaseoli* (Fop)[1]	1.6 a[3] A[4]	2.7 a B	4.4 a C
Meloidogyne incognita + Fop[2]	2.4 b A	3.7 b B	5.5 b C

[1,2] Average of 73 RILs with three replications. [3,4] Lowercase letters compare treatments within the same evaluation day (column), while uppercase letters compare evaluation days within the same treatment (row), according to the Scott-Knott test at a 5% significance level. CV = 21.5%. The scores indicate the degree of vascular discoloration in the xylem caused by *Fusarium oxysporum* (Pastor-Corrales and Abawi 1987), where 1 = healthy tissue, 3 = mild discoloration, 5 = moderate discoloration, 7 = severe discoloration, and 9 = dead plant.

Table 2. Absolute and relative frequency in a F8 recombinant inbred line (RIL) population by level of vascular discoloration caused by inoculation with *Fusarium oxysporum* f. sp. *phaseoli* (Fop) and with *Meloidogyne incognita* + Fop (Mi + Fop) on different evaluation days

Vascular discoloration level[2]	**Days evaluated and percentage of plants with vascular discoloration caused by *Fusarium oxysporum* for each score**											
	14th day				**21st day**				**28th day**			
	Fop		**Mi + Fop**		**Fop**		**Mi + Fop**		**Fop**		**Mi + Fop**	
	Absolute frequency and relative frequency (%)											
Score 1	177[1]	80.8	99	45.2	87	80.8	24	11.0	18	8.2	0	0.0
Score 3	33	15.1	90	41.1	99	15.1	102	46.6	84	38.4	30	13.7
Score 5	3	1.4	30	13.7	18	1.4	81	37.0	84	38.4	108	49.3
Score 7	0	0.0	0	0.0	9	0.0	12	5.5	12	5.5	81	37.0
Score 9	6	2.7	0	0.0	6	2.7	0	0.0	21	9.6	0	0.0
Total	219	100	219	100	219	100	219	100	219	100	219	100

[1] Average of 73 RILs with three replications. [2] The scores indicate the degree of vascular discoloration in the xylem caused by *Fusarium oxysporum* (Pastor-Corrales and Abawi 1987), where 1 = healthy tissue, 3 = mild discoloration, 5 = moderate discoloration, 7 = severe discoloration, and 9 = dead plant.

This inoculation sequence likely enhanced fungal colonization, thereby contributing to the increased severity of Fusarium wilt symptoms observed in co-infected plants.

The interaction between *M. incognita* and *F. oxysporum* is similar to earlier reports of nematode-facilitated fungal pathogenesis when roots were damaged through physical wounding (Yaseen et al. 2024). Physiological shifts also increased host vulnerability through breakdown of resistance, suppression of defense mechanisms through effector proteins, reduction in vascular occlusion, and a delayed phytoalexin response (Hillocks and Marley 1995, Yaseen et al. 2024). This phenomenon has been observed in beans and tomatoes, where virulent *M. javanica* disrupts Fop resistance and exacerbates Fusarium wilt, particularly in susceptible cultivars (Simão et al. 2010, Hajji-Hedfi et al. 2017). *Fusarium oxysporum* has a differential effect on the oxidative metabolism in susceptible versus resistant bean roots (Quadros et al. 2020), leading to vascular collapse from fungal proliferation and toxin deposition (Garcés-Fiallos et al. 2022).

Regarding biochemical and structural resistance mechanisms, resistant plants activate defense enzymes (PAL, GPX) and produce phenolic compounds that reinforce xylem walls. Resistant plants also form papilla-like structures and cellular deposits that restrict the spread of fungi (Benchimol-Reis et al. 2023).

Studies aimed at elucidating the genetic mechanisms of resistance in plants have identified defense components. The defense-related genes *R1*, *PR5*, *RGA29*, *Lectin*, *LOX*, and *Osmotin* were significantly upregulated in resistant onion cultivars exhibiting tolerance to Fusarium basal rot (FBR) (Poursakhi et al. 2024). The activation of pathogenesis-related genes, particularly *R1* and *PR5*, following *Fusarium* infection represents a typical defense response associated with FBR resistance. Similarly, in resistant melon genotypes challenged with *Fusarium oxysporum*, the antioxidant enzymes superoxide dismutase (SOD), polyphenol oxidase (PPO), and peroxidase (POX) were significantly upregulated, suggesting that activation of enzymatic antioxidant defenses complements gene-mediated resistance mechanisms (Sadeghpour et al. 2022). The expression of WRKY transcription factors, lectin receptor kinase, pathogenesis-related protein, lipoxygenase, and ribosome-inactivating protein genes were analyzed in *Iris* plants using quantitative polymerase chain reaction (qPCR). All five defense-related genes exhibited significant transcriptional upregulation in samples infected with *Fusarium oxysporum* f. sp. *gladioli* (FOG) (Tehrani et al. 2020).

Genetic resistance can remain stable under co-infection, as demonstrated in cucurbit rootstocks that retained *Fusarium* immunity despite *M. incognita* challenge when the material was not susceptible to nematode gall formation (Keinath and Agudelo 2018). These findings suggest that this strategy of nematode resistance should be prioritized in breeding programs. Specifically, the initial selection of common bean materials resistant to root-knot nematodes (*Meloidogyne* spp.) followed by selection for resistance to *Fusarium oxysporum* may facilitate the development of genotypes with stable resistance to co-infection by both pathogens. Alternatively, backcrosses with genotypes previously identified as having dual resistance could accelerate the incorporation of combined resistance into elite lines.

The genetic resistance of common bean to Fusarium wilt and to root-knot nematodes (*Meloidogyne* spp.) has been widely investigated in various studies. Resistance to Fusarium wilt in common bean is a dominant trait controlled by a few major genes (VC 13 and VC 25) or through polygenic inheritance involving multiple genes of minor effect (Batista et al. 2017, Benchimol-Reis et al. 2023). Resistance to RKN in common bean is primarily through inheritance of two genes: *Me*1, and *Me*2me3, which exhibit dominance up to 26 °C and semi-dominance up to 28 °C (Omwega and Roberts 1992). Recently, resistance to RKN (specifically to *M. incognita* and *M. javanica*) in common bean was also shown to have a duplicate recessive epistasis inheritance pattern, exemplified by the resistance provided by the Ouro Negro cultivar (Pesqueira et al. 2025). In the F_2 generation of the cross between Branquinho (susceptible) × IAC-Tybatã (moderately resistant) cultivars, the genetic architecture of resistance to *M. incognita* race 3 proved to be polygenic (Orsi et al. 2025). Sadeghpour et al. (2023) reported 11 gene markers implicated in polygenic resistance during infection by *Fusarium oxysporum* f. sp. *melonis*.

In the present study, six RILs (1, 5, 8, 25, 31, and 47) exhibited low infection levels by both *Fusarium oxysporum* f. sp. *phaseoli* (Fop) and root-knot nematodes under co-infection. Genotypes 1, 5, and 25 showed no root galls, whereas 8, 31, and 47 developed only a few (Note 1). All genotypes received a vascular discoloration score of 3, indicating mild symptoms, in both the Fop-only and Fop + nematode treatments (data not displayed). These results demonstrate genuine dual resistance that effectively mitigates the synergistic effects of the two pathogens. Furthermore, these

findings underscore the importance of prioritizing resistance to root-knot nematodes (*Meloidogyne* sp.) first, followed by selection for resistance to *F. oxysporum*, in common bean breeding programs.

Studies have shown that the presence of nematodes can intensify the development of *Fusarium* wilt (Bell et al. 2017, Kumar et al. 2017, Hua et al. 2019), although this effect was not observed in all cases (Keinath and Agudelo 2018). Specifically in common bean, the combined infection by *Meloidogyne incognita* and *F. oxysporum* f. sp. *phaseoli* (Fop) has demonstrated that the nematode can exacerbate symptoms caused by Fop in susceptible genotypes and can even reverse resistance responses (France and Abawi 1994, Simão et al. 2010). Nevertheless, uncertainties remain regarding the mechanisms underlying this process–whether genetic factors are involved in the interaction or whether the increased susceptibility to Fop results merely from the nematode-induced root injuries and the consequent reduction in nutrient uptake.

The six RILs identified in this study represent valuable genetic resources for common bean breeding programs. Particularly resistance to root-know nematodes, together with resistance to Fop, responds to the actual conditions of cultivated fields, which are frequently infested by both root-knot nematodes and *F. oxysporum* f. sp. *phaseoli*. Therefore, the development of genotypes with simultaneous resistance is essential for sustainable disease management in the crop.

DATA AVAILABILITY

The datasets generated and/or analyzed during the current research are available from the corresponding author upon reasonable request.

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