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Histological and Cytological Responses of Genotypes From *Capsicum* spp. to *Meloidogyne enterolobii*

Mônica Lau da Silva Marques[1] | Jéssica Maria Israel de Jesus[2] | Igor Araújo[3] | Renê Gonçalves Silva Carneiro[4] | Mara Rubia da Rocha[4]

[1]Instituto Federal Goiano Campus Ceres, Ceres, Brazil | [2]Universidade de São Paulo, Piracicaba, Brazil | [3]Programa de Pós-Graduação em Ecologia e Conservação, Universidade do Estado de Mato Grosso, Campus Nova Xavantina, Nova Xavantina, Brazil | [4]Universidade Federal de Goiás, Goiânia, Brazil

Correspondence: Jéssica Maria Israel de Jesus (jessicamariaisrael@gmail.com)

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ABSTRACT

Sweet and chilli peppers (*Capsicum* spp.) are widely cultivated horticultural crops, but they are highly susceptible to root-knot nematodes (*Meloidogyne* spp.). Among them, *Meloidogyne enterolobii* stands out for its aggressive parasitism and the severe damage it causes to economically important crops. This study aimed to describe and compare the anatomical and cytological alterations induced by *M. enterolobii* in the root system of susceptible (Cambuci, UFGCBA 3) and resistant (Bode Roxa A, UFGCCH 24) *Capsicum* genotypes, either inoculated (I) or non-inoculated (NI), and evaluated at five developmental stages. A completely randomised 2×2×5 factorial design was used, with assessments at 7, 14, 21, 28, and 35 days after inoculation (DAI). Nematode population density and reproduction factor were quantified, and histological analyses were performed on infected and healthy roots. *M. enterolobii* successfully penetrated and established in the roots of both genotypes, inducing giant cell formation and pronounced hypertrophy around feeding sites. However, the resistant genotype Bode Roxa A exhibited smaller giant cells accompanied by localised necrosis at 14 and 28 DAI, suggesting a delayed hypersensitive-like reaction. In contrast, enhanced cell proliferation and thickening of parenchymatic tissues were observed at 21 DAI in resistant plants, indicating active cellular reorganisation. These findings underscore the relevance of histopathological and cytometric analyses in understanding plant-nematode interactions and highlight Bode Roxa A as a valuable genetic resource for breeding resistant and sustainable *Capsicum* cultivars, contributing to crop resilience and food security.

1 | Introduction

Root-knot nematodes (*Meloidogyne* spp.) inflict extensive damage on plant roots, feeding on host tissues and profoundly altering their structure and function. Their parasitism causes both mechanical injury during root penetration and migration, as well as complex physiological and anatomical alterations associated with gall formation induced by nematode feeding (Vilela et al. 2019). Gall-forming root-knot nematodes feed on root tissues such as the phloem, pericycle, or endodermis. After establishing within the roots, they become sedentary and induce a cascade of cellular modifications, particularly disrupting vascular integrity through the formation of specialised feeding sites (Bartlem et al. 2014; Harris and Pitzschke 2020; Isaias et al. 2024).

Meloidogyne spp. are among the most destructive plant-parasitic nematodes, with an exceptionally wide host range and severe global economic impact. *M. enterolobii* is especially aggressive, causing severe root malformation that compromises water and nutrient uptake, leading to yield losses that may reach 100% (Vovlas, Mifsud, et al. 2005; Moens et al. 2009; Jones et al. 2013; Karssen et al. 2013). Although some cultivars exhibit genetic resistance to *M. javanica*, *M. arenaria*, and

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M. incognita, *M. enterolobii* can overcome these mechanisms (Carneiro et al. 2017; Marques, Jesus, et al. 2020). Therefore, a deeper understanding of plant resistance mechanisms and the development of integrated management strategies is essential to mitigate the damage caused by *M. enterolobii* (Philbrick et al. 2020).

The formation of giant cells, which function as specialised feeding sites essential for nematode nourishment, is a hallmark of root-knot nematode parasitism (Vovlas, Mifsud, et al. 2005). These cells arise through nematode-induced reprogramming of host gene expression. In resistant plants, this process is either inhibited, preventing nematode establishment (pre-infective resistance), or disrupted after initial induction, leading to degenerated or nonfunctional feeding sites (post-infective resistance) (Castañeda et al. 2017; El-Sappah et al. 2019). The life cycle of *M. enterolobii* begins when second-stage juveniles (J2) hatch and invade the roots. Females become sedentary, while males migrate out after sexual differentiation. Giant cells develop around sedentary females, exhibiting dense cytoplasm and multiple enlarged nuclei induced by nematode secretions. In resistant plants, however, defence mechanisms such as hypersensitive-like reactions, oxidative enzyme activation, and altered hormonal signalling may impair gall development and hinder nematode maturation (Vovlas, Rapoport, et al. 2005; Das et al. 2008; Marques et al. 2023).

Studies by Marques et al. (2019, 2023); Marques, Oliveira, et al. (2020) have significantly expanded current knowledge on *M. enterolobii* resistance in *Capsicum* spp. A total of 31 resistant genotypes were identified across species: 17 *C. chinense* with reproduction factors (RF) below 0.85, ten *C. annuum* with RF below 0.75, three *C. frutescens* with RF below 0.87, and one *C. baccatum* with RF of 0.6. Fourteen genotypes remained resistant under increased inoculum pressure, confirming stable resistance (Marques et al. 2019). Subsequent comparative studies using two resistant *C. chinense* and three susceptible *C. baccatum* genotypes demonstrated that J2 juveniles penetrated both genotypes; however, resistant plants harboured fewer nematodes, exhibited delayed development, produced fewer galls, and had lower RF values (Marques, Oliveira, et al. 2020). Further biochemical analyses revealed enhanced activity of defence-related enzymes, including β-1,3-glucanase (GLU), chitinase (CHI), and lipoxygenase (LOX), particularly in the resistant genotype Bode Roxa A, reinforcing their involvement in defence signalling and structural reinforcement (Marques et al. 2023).

Additional resistance screenings in Mexico evaluated 90 *C. annuum* landraces inoculated with 2000 *M. enterolobii* eggs, identifying 11 resistant varieties. At 75 days after inoculation (DAI), resistance in accessions UTC66, UTC90, UTC67, UTC88, and UTC81 remained consistent across independent trials (Carrillo-Fasio et al. 2020). Moreover, studies with multiple *M. enterolobii* populations revealed that gall formation begins at 21 DAI, with egg masses observed by 28 DAI on all tested vegetable cultivars; no significant variation in pathogenicity or reproduction rates was detected among the eight nematode populations. However, at 35 DAI, cucumber (*Cucumis sativus*) cultivars exhibited nearly double the gall and egg mass counts compared to *C. annuum* cultivars (Salazar-Mesta et al. 2024).

Given that factors influencing nematode infection and gall morphogenesis are highly variable, the objective of this study was to characterise the anatomical responses of resistant and susceptible *Capsicum* spp. genotypes to *M. enterolobii* at multiple time points, thereby improving the understanding of how host resistance mechanisms influence the nematode's life cycle and pathogenic progression.

2 | Materials and Methods

2.1 | Experimental Design, Inoculum and Inoculation of *M. enterolobii*

The study was conducted in a greenhouse and laboratory at 16°40′22″ S, 49°15′19″ W; at an altitude of approximately 730 m. A completely randomised design (CRD) was used for histological and cytometric analyses, following a 2×2×5 factorial arrangement, comprising two *Capsicum* genotypes (Cambuci, UFGCBA 3—susceptible; Bode Roxa A, UFGCCH 24—resistant), two inoculation conditions (inoculated and non-inoculated), and five evaluation periods [7, 14, 21, 28, and 35 days after inoculation (DAI)]. Three biological replicates per treatment were used for histological and cytometric evaluations while nematological assessments were conducted at 35 DAI, using ten replicates per genotype. Additionally, ten tomato plants [*Solanum lycopersicum* L. cv. Santa Cruz Kadá] were grown in parallel as a positive control to verify inoculum viability.

The *M. enterolobii* inoculum was obtained from naturally infested roots of guava (*Psidium guajava* L. var. Paluma) collected in São João da Barra, RJ, Brazil, and subsequently multiplied on *S. lycopersicum* (cv. Santa Cruz Kadá) under greenhouse conditions. A protein extract from *M. javanica* females was included as a reference phenotype. The identification of *M. enterolobii* females was confirmed by esterase (EST) phenotype analysis, following Carneiro and Almeida (2001). Adult females were manually extracted from infected roots and subjected to polyacrylamide gel electrophoresis (PAGE), followed by isoenzyme staining using α-naphthyl acetate as substrate and Fast Blue RR salt as dye. Relative mobility (Rm) was calculated using the slowest *M. javanica* band (Rm = 1.0) as a reference. Samples identified as *M. enterolobii* exhibited diagnostic bands at approximately Rm = 0.90, allowing clear and reliable distinction from other *Meloidogyne* species.

2.2 | Development, Histopathology and Cytometry of *M. enterolobii*

Seeds of *Capsicum* spp. were obtained from the Universidade Federal de Goiás germplasm collection. Genotypes were selected based on their known resistance or susceptibility to *M. enterolobii*: resistant (*C. chinense*, Bode Roxa A, UFGCCH 24; RF < 1) and susceptible (*C. baccatum*, Cambuci, UFGCBA 3; RF > 1) (Marques et al. 2019). Seedlings were produced by a certified nursery (Vivati, GO) and transplanted at the four-true-leaf

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stage into 500-mL polyethylene bags filled with sterilised 1:1 (v/v) soil: sand mixture. Fifteen days after transplanting, seedlings were inoculated with 5 mL of aqueous suspension containing approximately 5000 eggs and J2 per plant, considered as the initial nematode population (Pi). Plants were irrigated daily, avoiding waterlogging; average minimum and maximum temperatures were 21°C and 35.5°C, respectively.

For histological analyses, root tips (0.5–1.0 cm radicels) were excised under a stereomicroscope, fixed in FAA 70 (formaldehyde: acetic acid: ethanol 70%, 1:1:18, v/v/v) for 24–48 h, and stored in 70% ethanol. Samples were dehydrated through graded ethanol (70%–100%), ethanol: butyl acetate series (3:1, 1:1, 1:3, and pure acetate), then embedded in Paraplast (Johansen 1940). Longitudinal and transverse sections (8–10 μm) were prepared using a rotary microtome and mounted on slides coated with Haupt's adhesive (Haupt 1930). Sections were dewaxed in butyl acetate: ethanol series, rehydrated through descending ethanol series (100–30%), stained with 0.3% Astra blue and 0.1% Safranin (9:1, v/v), and mounted in colourless glass varnish 500 (Paiva et al. 2006). Observations were performed using a Leica EZ4 microscope (40×) coupled to an ICC50 digital camera (LAS EZ v1.8.1).

Cytometric analyses were conducted using AxioVision SE64 software (Zeiss), version 4.9.1. Fifteen vascular parenchyma cells, including giant cells when present (five per replicate), were measured for each genotype (resistant or susceptible) and inoculation status (inoculated or non-inoculated) across all five time points. The total root section area and cell dimensions were quantified according to Souza et al. (2016). Anatomical and cytometric data were jointly analysed to identify structural indicators of nematode resistance.

At 35 DAI, root systems were harvested, washed, weighed (fresh root mass; precision 0.001 g), and stained with 0.015% Floxin B for 15–20 min, then rinsed to visualise egg masses under a stereomicroscope (Taylor and Sasser 1978).

The gall index (GI) and egg mass index (EMI) were determined using the Taylor and Sasser (1978) scale, ranging from 0–5: 0 = no galls or egg masses; 1 = 1–2; 2 = 3–10; 3 = 11–30; 4 = 31–100; and 5 = > 100. Scores of 0–2 indicate resistance, while 3–5 indicate susceptibility.

Roots were macerated and nematodes were extracted following Hussey and Barker (1973). Eggs and J2 were counted using a Peters' chamber under optical microscopy (40×). Population density (Pd; per 10 g roots) and reproduction factor (RF = Pf/Pi) were calculated according to Oostenbrink (1966).

2.3 | Statistical Analysis

Data were subjected to Lilliefors and Bartlett's tests to verify normality and homoscedasticity. Subsequently, analysis of variance (ANOVA, F-test) was performed, and means were compared using Tukey's HSD test ($p < 0.05$) in Assistat v7.7. For the interaction among genotype, inoculation, and evaluation time, regression models were fitted using Sisvar software (Ferreira 2011).

3 | Results

The histopathological observations revealed that *M. enterolobii* penetrated the roots of both genotypes, indicating the absence of a pre-infective resistance mechanism. At 7 DAI, multinucleated giant cells were not evident in the susceptible genotype (Cambuci) (Figure 1A) or the resistant genotype (Bode Roxa A) (Figure 1B).

At 14 DAI, well-developed giant cells containing numerous nuclei were observed in the susceptible genotype (Figure 1C). Marked disorganisation of the vascular cylinder, including xylem and phloem, and phloem and irregularly shaped hypertrophied cells were evident in both genotypes (Figure 1C,D).

At 21 DAI, the susceptible genotype exhibited typical giant cells with dense cytoplasm and increased volume associated with feeding sites. Adult females with large bodies filled with eggs were observed adjacent to giant cells, confirming the efficiency of these feeding sites in supporting nematode development (Figure 1E). In contrast, the resistant genotype showed atypical giant cells, characterised by smaller size and intense staining compatible with localised degeneration/necrosis, suggesting a post-infective or hypersensitive-like response. Nematodes associated with these cells were smaller, and no eggs were observed inside females (Figure 1F).

At 28 DAI, the susceptible genotype still displayed numerous intact giant cells with dense cytoplasm and multiple nuclei near well-developed females. The combination of large females and extensive hypertrophy frequently led to cortical disruptions and severe deformation of vascular tissues (Figure 1G). In the resistant genotype, giant cells were rarely observed; when present, they were smaller, irregularly shaped, and frequently surrounded by cells showing an HR-like response at the periphery of the feeding site. Females at different developmental stages and with reduced sizes were recorded in these unstructured feeding sites (Figure 1H).

At 35 DAI, the susceptible genotype exhibited few remaining giant cells, but females persisted at feeding sites, and egg masses occupied residual cavities within the roots (Figure 1I). In contrast, the resistant genotype no longer displayed typical giant cells; only a few females were present, and egg masses were rarely observed (Figure 1J), indicating restriction of feeding site maintenance and nematode reproduction.

At 35 DAI, no significant differences were detected between the resistant (Bode Roxa A) and susceptible (Cambuci) genotypes for root fresh mass (RFM) or population density (Dp), although Dp was 47.55% lower on the resistant genotype (Bode Roxa A) (Table 1). The reproduction factor (RF) was < 1.0 in Bode Roxa A (RF = 0.67), confirming its resistant status, whereas Cambuci showed RF = 1.82, consistent with susceptibility. The gall index (GI) and egg mass index (EMI) were also lower in Bode Roxa A than in Cambuci (Table 1). The tomato control ('Santa Cruz Kadá') confirmed inoculum viability, with RF = 16, GI = 5, and EMI = 5.

Anatomical and cytometrical analyses showed that cellular hypertrophy was strongly associated with nematode infection. In inoculated treatments (SI and RI), giant cell areas increased markedly over time, with contrasting magnitudes between

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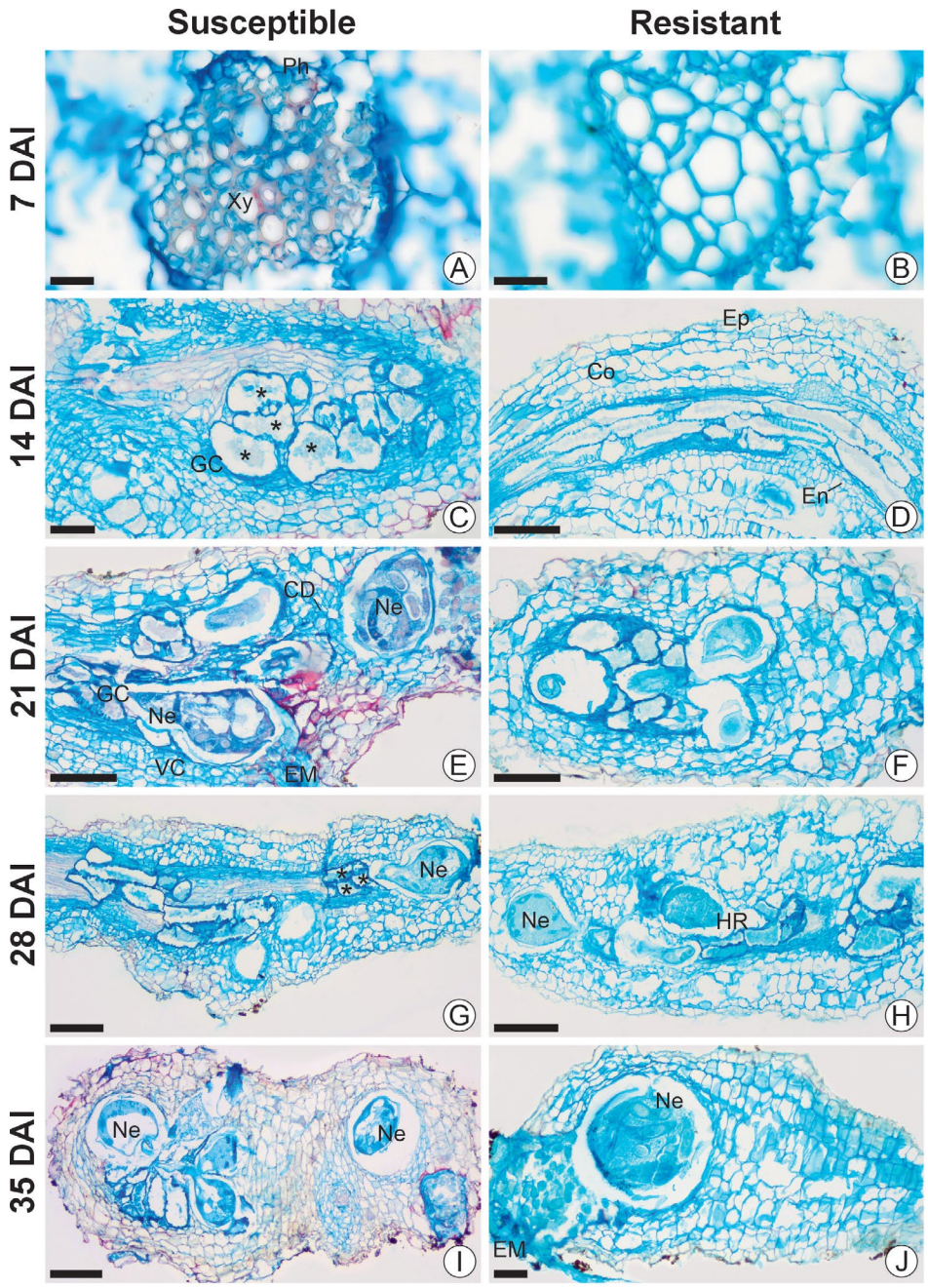

FIGURE 1 | Anatomical sections of roots susceptible (*C. baccatum*—Cambuci: A, C, E, G, I) and resistant (*C. chinense*—Bode Roxa A: B, D, F, H, J) genotypes inoculated with *Meloidogyne enterolobii* at 7–35 days after inoculation (DAI). (A, B) Roots without differentiated giant cells (7 DAI); (C, D) Hypertrophy, hyperplasia, and giant cells (14 DAI); (E, F) Adult females, egg masses, and giant cells (21 DAI); (G, H) Adult females and host response with hypersensitivity-like reaction (28 DAI); (I, J) Females and egg masses (35 DAI). (A, B) Cross sections; (C–J) Longitudinal sections. EM = egg mass; GC = giant cells; HR = hypersensitive-like response; Ne = nematode; Ph = phloem; Xy = xylem; Ep = epidermis; Co = cortex; En = endodermis. Scale bars = 50 μm.

genotypes (Table 2). In non-inoculated plants (SNI and RNI), cells of the vascular cylinder and conductive tissues (xylem and phloem) remained structurally normal, with no significant differences across evaluation times.

In the SI treatment, cell area increased sharply at 14 DAI, followed by a reduction at 21 DAI, consistent with intensive cell division, and then a second expansion peak at 28 DAI, when the highest mean areas were recorded (Table 2). At 35 DAI, cell area decreased, in line with the completion of the nematode life cycle and partial degeneration of feeding sites. In the RI treatment, cell area increased progressively from 7 to 21 DAI and then remained relatively stable from 28 to 35 DAI, resulting in values comparable to or exceeding those of SI at later stages (Table 2), but associated with atypical and degenerating feeding structures.

Cytometric data (Figure 2) support these patterns, showing that inoculated treatments (SI and RI) exhibited the largest cellular areas at 14–28 DAI, whereas non-inoculated controls (SNI and

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TABLE 1 | Reactions of susceptible (*Capsicum baccatum*—Cambuci) and resistant (*Capsicum chinense*—Bode Roxa A) *Capsicum* genotypes to *Meloidogyne enterolobii* at 35 days after inoculation (DAI), UFG, Brazil.

Capsicum spp.	RFM[1]	Dp[2]	RF[3]	GI[4]	EMI[5]
Cambuci	1.94a*	55,329a	1.82a	4.0a	4.0a
Bode Roxa A	1.37a	29,019a	0.67b	3.0b	3.0b
Mean	1.66	42,174	1.25	3.5	3.5
CV[6] (%)	16.83	43.76	34.76	17.84	18.28

Note: Analyses performed in Sisvar; data were transformed using $\sqrt{(x+1)}$ where appropriate.
[1]Fresh root mass (g).
[2]Population density (eggs + J2 per 10 g roots).
[3]Reproduction factor (Pf/Pi).
[4]Gall index (Taylor and Sasser 1978).
[5]Egg-mass index (Taylor and Sasser 1978).
[6]Coefficient of variation.
*Means followed by the same letter in the column do not differ at the 5% level.

TABLE 2 | Cellular area (μm^2) in roots of susceptible (*Capsicum baccatum*—Cambuci) and resistant (*Capsicum chinense*—Bode Roxa A) *Capsicum* genotypes, inoculated (I) or non-inoculated (NI) with *Meloidogyne enterolobii,* at different evaluation times (DAI), UFG, Brazil.

Treatments	7	14	21	28	35
SNI	115a*	525c	87c	314c	160b
SI	688a	4501a	2621b	7297a	3531a
RNI	105a	358c	1046c	254c	284b
RI	474a	2370b	6577a	5549b	4580a

Note: Means of 15 vascular parenchyma cells per treatment (5 cells in three replicates), including giant cells when present.
Abbreviations: RI, resistant inoculated; RNI, resistant non-inoculated; SI, suscetptible inoculated; SNI, susceptible non-inoculated.
*Means followed by the same letter in the column do not differ by Tukey's test at 5%. CV (Coefficient of variation) = 32.56%.

RNI) maintained low and oscillating values. Although both inoculated genotypes showed hypertrophy, the architecture and stability of feeding sites differed, with RI displaying structurally disorganised and degenerating giant cells, consistent with restricted nematode development. These anatomical differences at 35 DAI are illustrated in Figure 3, which shows pronounced hypertrophy and tissue disorganisation in inoculated roots, in contrast with the normal vascular architecture observed in non-inoculated controls.

4 | Discussion

The two *Capsicum* spp. genotypes, Bode Roxa A (resistant, RF = 0.67) and Cambuci (susceptible, RF = 1.82), were selected based on previous work by Marques et al. (2019), who evaluated 73 *Capsicum* genotypes for parasitism by *M. enterolobii* under different inoculum concentrations. In the present study, these resistance and susceptibility patterns were confirmed, and additional mechanistic aspects can be inferred from the anatomical traits observed.

The presence of giant cells within the vascular cylinder of both *Capsicum* genotypes at 14 days after inoculation (DAI) indicates a functional impairment of roots due to structural changes in vascular tissues. Such changes likely disrupt water and nutrient transport through the xylem, as reported for *C. annuum* infected by *M. incognita* at 14 DAI (Pegard et al. 2005). Several second-stage juveniles (J2) were observed in the subapical meristem and cortex, apparently migrating toward the stele; cell wall damage was frequently detected at this stage. Our findings reinforce that vascular parenchyma is the primary feeding site of *Meloidogyne* spp., as widely discussed in the literature (Bartlem et al. 2014; Vovlas, Mifsud, et al. 2005; Mitkowski and Abawi 2003). Both *Capsicum* genotypes were parasitized by *M. enterolobii*, but the extent of root structural damage at feeding sites differed, differentially compromising hydraulic and nutritional homeostasis and eliciting metabolic stress responses of varying intensity. Altogether, these patterns enhance the pathophysiological understanding of plant-nematode interactions, particularly the anatomical and physiological facets of this pathosystem (Jindapunnapat et al. 2025; Shi et al. 2025).

Typical cellular modifications induced by sedentary phytonematodes include increased cytoplasmic density, enlarged nuclei and nucleoli, and a reduction in the central vacuole. Giant cells originate from vascular parenchyma, and their induction begins with repeated nuclear divisions unaccompanied by cytokinesis within the initial feeding cell (Bartlem et al. 2014). Each root-knot nematode typically induces five to seven giant cells that may contain up to approximately 100 nuclei due to extensive endoreduplication (Abad et al. 2003). The induction of these structures depends on substances secreted from the oesophageal glands of J2 during migration through epidermal and cortical tissues toward the vascular cylinder (Abad et al. 2003; Azlay et al. 2023).

Overall, the anatomical features observed in galls of both resistant and susceptible plants are consistent with previous descriptions of giant-cell induction and morphogenesis. Comparable processes were described by Mitkowski and Abawi (2003), wherein plants respond to enzymatic/chemical actions of *Meloidogyne* spp. with successive nuclear replications without cell division, generating enlarged, multinucleated, metabolically active cells with relatively thick walls and dense cytoplasm. Remarkably, clear signs of degradation in giant cells were evident in resistant plants (Bode Roxa A) by 21 DAI, rendering them unsuitable nutritional sources. This supports a post-infection resistance mechanism that limits pathogen progression after initial establishment. Such delayed, non-hypersensitive (non-HR) resistance has been associated with premature feeding-site disintegration roughly two weeks post-infection (Bakker et al. 2006).

Capsicum spp. deploy multiple defences against phytonematodes, including secondary metabolites and resistance genes. Several *Capsicum* species harbour *Me*-family resistance (R) genes conferring resistance to *Meloidogyne* spp. (Djian-Caporalino et al. 2007; Abd-Elgawad 2022). Pinheiro et al. (2020) identified new resistance sources to root-knot nematodes in pepper, highlighting their breeding potential. Delayed resistance responses similar to those observed in this study were also reported in other crops: tobacco (*Nicotiana tabacum*) presenting HR in

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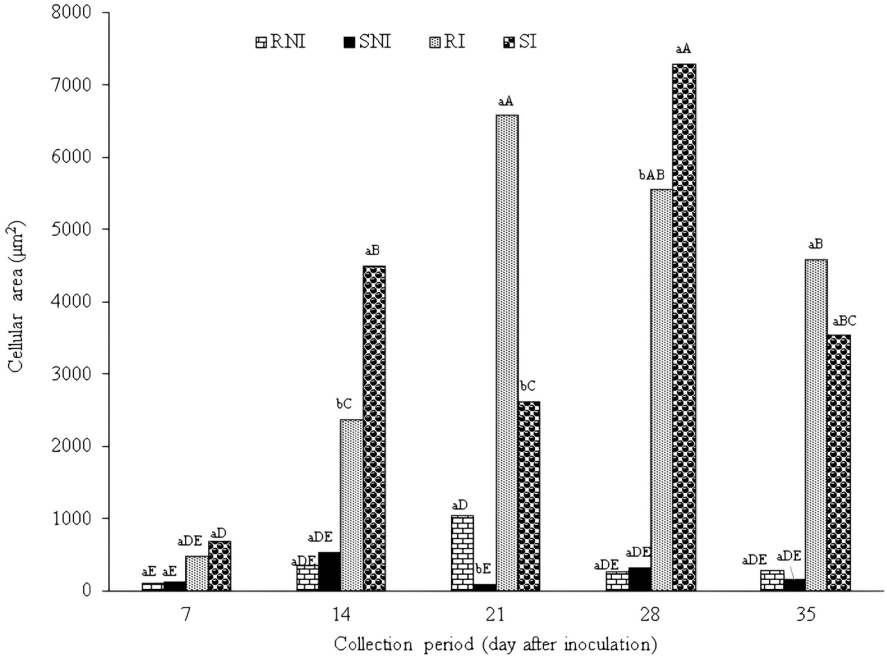

FIGURE 2 | Mean cellular area (μm^2) in roots of susceptible (*C. baccatum*—Cambuci) and resistant (*C. chinense*—Bode Roxa A) Capsicum genotypes, non-inoculated (SNI, RNI) or inoculated (SI, RI) with *Meloidogyne enterolobii*, at 7, 14, 21, 28, and 35 DAI. Different letters indicate significant differences (lowercase, within columns; uppercase, within rows) according to Tukey's test ($p < 0.05$).

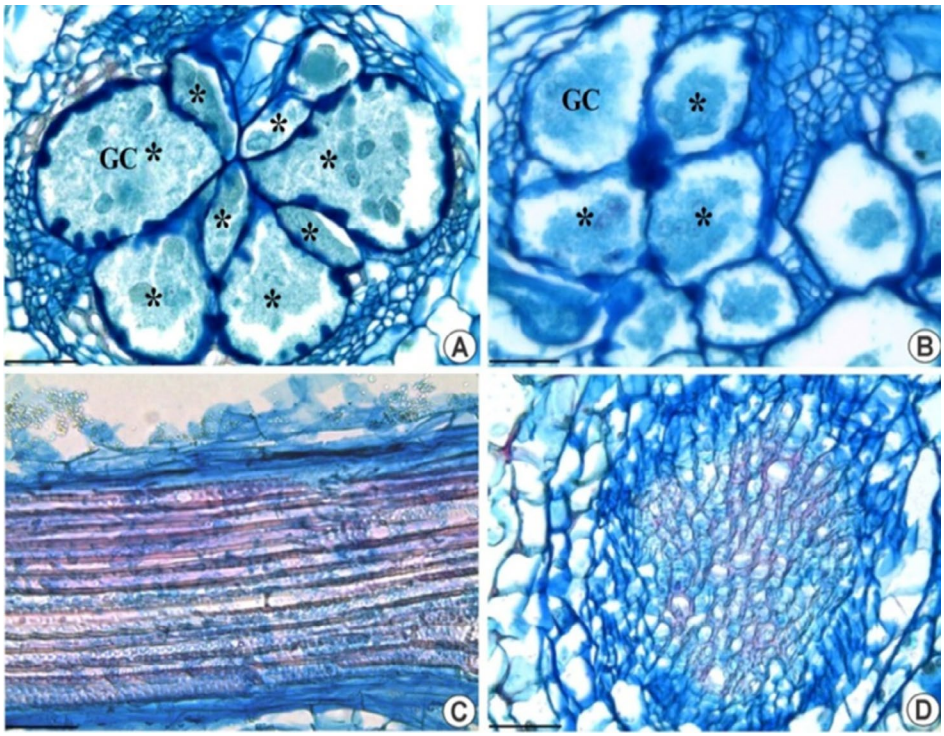

FIGURE 3 | Transverse and longitudinal sections of parasitized and non-parasitized roots of *Capsicum* genotypes at 35 DAI. (A) Transverse section, susceptible inoculated (SI); (B) Transverse section, resistant inoculated (RI); (C) Longitudinal section, susceptible non-inoculated (SNI); (D) Transverse section, resistant non-inoculated (RN). *Hypertrophied and hyperplasic cells; GC = giant cells. Scale bars = 50 µm.

fully developed giant cells (Ng'Ambi et al. 1999) and cowpea (*Vigna unguiculata*) carrying the *Rk* gene, where typical HR was not evident in resistant roots at 9–21 DAI (Das et al. 2008). Alternatively, delayed responses may be mediated by *Me-1* (Abd-Elgawad 2022).

Siddiqui and Taylor (1970) described galls induced by *M. naasi* on wheat roots (*Triticum aestivum* L.), noting conspicuous giant cells near the anterior nematode region from 4 to 5 DAI and complete dissolution of giant cells by around 40 DAI (protoplast and walls disintegrated). In our susceptible *Capsicum*

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(Cambuci), comparable cellular outcomes were evident by 35 DAI, suggesting that the 35–40 DAI window is critical for evaluating the nematode life cycle and the full trajectory of giant-cell development and decay.

Shigueoka et al. (2019) showed that J2 of *M. paranaensis* penetrate both resistant and susceptible coffee genotypes, paralleling our observations. However, in coffee the resistant reaction hinders giant-cell establishment—distinct from *Capsicum-M. enterolobii* here. Although giant cells occurred in resistant Bode Roxa A, they were atypical and presumably nutritionally deficient, with fewer/smaller cells. Because giant-cell formation was not fully blocked in Bode Roxa A, some females still deposited eggs by 35 DAI, albeit in much smaller numbers than in the susceptible genotype. These results highlight the efficiency of resistance while illustrating contrasting histological dynamics in susceptible versus resistant hosts.

Cytometric data further corroborated these interpretations, revealing distinct cell-growth dynamics between resistant (Bode Roxa A) and susceptible (Cambuci) genotypes inoculated with *M. enterolobii*. Susceptible plants supported higher nematode populations, consistent with comparative studies of susceptible versus resistant plants (Bartlem et al. 2014; Vovlas, Mifsud, et al. 2005), owing to greater differentiation and sustained enlargement of giant cells. The measured cellular areas corroborate how resistance in Bode Roxa A restricts nematode development.

The partial, quantitative resistance (QR) in Bode Roxa A (*C. chinense*, UFGCCH 24) against *M. enterolobii* appears multifactorial, with histological manifestations closely linked to biochemical responses. Reduced cytometric area and delayed nematode development (Marques, Oliveira, et al. 2020) reflect a complex and durable defence mechanism in Solanaceae. Recent studies indicate that QR is mediated by a coordinated activation of defence pathways and key enzymes. Resistant guava (*Psidium guineense*) likewise increases phenolics and other secondary metabolites as an effective strategy against *M. enterolobii* (Costa et al. 2025). In Bode Roxa A, phenylpropanoid-related peroxidase (POX) activity likely diverts resources to build physical/chemical barriers around infection sites, limiting giant-cell expansion. This metabolic redirection and localised cell confinement produce the observed physical restriction, supporting QR as a promising, durable strategy for breeding *Capsicum* against aggressive *M. enterolobii* (Sánchez-Solana et al. 2016; Sato et al. 2021; Marques et al. 2023; de Barros Souza et al. 2025).

Population density was 47.55% lower in Bode Roxa A than in Cambuci, with RF values of 0.67 and 1.82, respectively, confirming resistant and susceptible statuses to *M. enterolobii*. Plants are classified as resistant when they do not permit adequate nematode reproduction (Oostenbrink 1966; Trudgill 1991), as observed for Bode Roxa A. In Cambuci, development proceeded normally, aligning with prior reports in *Capsicum-M. enterolobii* (Marques et al. 2019). The presence of females in resistant roots, though at much lower numbers, indicates resistance is partial rather than absolute. A similar pattern, juvenile penetration in both genotypes but restricted development in resistant hosts, was noted for coffee-*M. paranaensis* (Shigueoka et al. 2019).

Altogether, the study of giant cells under *M. enterolobii* parasitism clarifies host-pathogen relationships, highlighting root alterations and damage gradients among *Capsicum* genotypes. Resistance in Bode Roxa A likely involves a delayed, non-HR disintegration of giant cells, reducing nematode development within the vascular cylinder. Fewer females at feeding sites at 28–35 DAI, together with cytometric evidence, indicate reduced stimuli for giant-cell differentiation/maintenance in resistant plants. Such understanding supports targeted breeding and integrated nematode management strategies to enhance crop resilience and sustainability. In practical terms, elucidating resistance in Bode Roxa A can inform the development of new cultivars and potential rootstocks for other solanaceous crops (e.g., bell pepper), decreasing dependence on chemical nematicides and contributing to food security by mitigating nematode impacts on *Capsicum*.

Author Contributions

Conceptualization: Mônica Lau da Silva Marques, Renê Gonçalves Silva Carneiro, Igor Araújo, Mara Rubia da Rocha. Formal analysis: Mônica Lau da Silva Marques, Renê Gonçalves Silva Carneiro, Mara Rubia da Rocha. Investigation: Mônica Lau da Silva Marques, Renê Gonçalves Silva Carneiro, Igor Araújo, Mara Rubia da Rocha, Jéssica Maria Israel de Jesus. Methodology: Mônica Lau da Silva Marques, Renê Gonçalves Silva Carneiro, Mara Rubia da Rocha. Writing – original draft: Mônica Lau da Silva Marques, Renê Gonçalves Silva Carneiro, Mara Rubia da Rocha. Writing – review and editing: Jéssica Maria Israel de Jesus, Igor Araújo.

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Disclosure

Declaration of Use of AI Technologies: We hereby declare that in our activities, practices, and in the processes of development and presentation of the products provided, we do not use Artificial Intelligence (AI) technologies. All results and conclusions are obtained solely through conventional methods and resources, without the intervention or assistance of automated data processing systems or artificial intelligence.

Ethics Statement

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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