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FUNGAL DISEASES

Lateral roots of carrot have a low impact on alloinfections in cavity spot epidemic caused by *Pythium violae*

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Abstract Carrot cavity spot, caused by a complex of *Pythium* species, is characterized by sunken elliptical lesions on the taproot. Recent epidemiological studies of *P. violae* have demonstrated the occurrence of both primary and secondary infections, with two types of secondary infection, autoinfection and alloinfection. Investigating the mechanisms underlying alloinfection and the role of carrot lateral roots, we asked whether direct physical root contact plays a role in alloinfection and whether root exudates enhance mycelial growth in soil alone. A rhizobox system was designed to differentiate the effects of each mechanism: a buffer zone created by nylon mesh was used to test the first mechanism, and young carrots with a root system similar to lateral roots were used to test the second. Alloinfections were generated in rhizoboxes via diseased taproots transplanted close to healthy, mature carrots. The nylon mesh had no significant effect on disease intensity (reflecting alloinfection), providing evidence that mycelial growth in soil contributed more to disease spread than did physical contact among roots. Nor did young carrots significantly affect alloinfection; thus root exudates had little effect on mycelial growth.

Keywords Alloinfections · Carrot cavity spot · *Pythium violae* · Rhizobox system · Root-to-root contamination · Soilborne pathogen

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Introduction

Carrot cavity spot (CCS), characterized by sunken elliptical lesions on the taproot, is one of the most damaging diseases caused by a soilborne pathogen that affects carrot (*Daucus carota*) (Hiltunen and White 2002). CCS is caused by a complex of *Pythium* sp., predominantly *P. violae* and *P. sulcatum* in France (Guerin et al. 1994) and *P. sulcatum* in Japan (Nagai et al. 1986; Watanabe et al. 1986).

Soilborne plant pathogens are transmitted to the plant host by various means, including mycelial growth, organic debris in soil, infected host plants, spore dispersion in soil water, and the root growth that brings infected and healthy roots into direct physical contact. Inoculum in the soil is responsible for primary infections. The pathogen then produces secondary inoculum, which spreads from primary foci to neighbouring plants, in a process that is called alloinfection. Such alloinfections have been described for some soilborne pathogens, such as *Sclerotium cepivorum* causing white rot of onion and garlic (Scott 1956), *Fusarium oxysporum* f. sp. *radicis-lycopersici* causing fusarium crown and root rot of tomato (Rekah et al. 1999), and *Pythium irregulare* (Burdon and Chilvers 1975) and *P. ultimum* var. *ultimum* (Green and Jensen 2000) causing cress and cucumber damping off, respectively.

Although CCS was suggested to resemble other soilborne pathogens with regard to secondary infections (Phelps et al. 1991) nearly two decades ago, alloinfections by *P. violae* have only recently been investigated (Suffert 2006). Three complementary methods were used. The first approach was a deductive one to look at the decrease over time of a time-dependent parameter involved in the relation between incidence and severity (Suffert and Montfort 2008). On the basis of the results, CCS epidemics seemed to be driven successively by (1) the mobilisation of soil inoculum, leading to primary infection; (2) the spread of disease to neighbouring taproots (alloinfection); and (3) the intensification of disease in the taproot (autoinfection). The second approach was a modeling one to describe CCS epidemics in a field experiment after the soil was artificially infested with *P. violae*. Three mathematical models that support the hypothesis of both primary and secondary infections (the logistic model, the bilogistic model of Hau et al. (1993), and the model of Brassett and Gilligan (1988)) were correctly fitted to the disease incidence progress curves (Suffert 2007). For the third approach, a purely experimental approach, diseased carrot taproots were transplanted close to healthy roots in a greenhouse environment. *P. violae* spread from taproot to taproot, firmly establishing the occurrence of alloinfection and the polycyclic nature of a CCS epidemic (Suffert and Montfort 2007).

Although some *Pythium* species rarely produce zoospores (Van der Plaats-Niterink 1981), they spread from primary foci to adjacent plants. Alloinfections by *Pythium* may be caused by active mycelial growth through the soil from CCS lesions. Zoospores may be involved in root-to-root contaminations but are not crucial.

Until now, CCS has been defined only on the basis of symptoms on the taproot; however, infection of lateral roots cannot be totally excluded. Lateral roots, as defined by Harvis (1939), may be involved in another mechanism for disease spread if there is direct physical contact between lateral roots and adjacent taproots. Some soilborne pathogens, such as *Gaeumannomyces graminis* var. *tritici* (Gilligan 1980; Holden 1976) and *Rhizoctonia* sp. (Hofman and Jongebloed 1988), colonize the surface of their host roots with superficial, long, unbranched hyphae, called runner hyphae, and spread from plant to plant. While runner hyphae have never been observed in *Pythium* sp., intermingling lateral roots of carrot may be regarded as an ideal medium for mycelial growth and an efficient route for inoculum dispersal.

Another mechanism may involve biochemical effects of root exudates. Root exudates in moist soil stimulate the sporangial germination and the mycelial growth of *Pythium* sp. (Huisman 1982; Schroth and Hildebrand 1964). Similarly, exudates from carrot lateral roots might enhance mycelial growth of *P. violae* through a rhizosphere effect.

Few papers have addressed the importance of active mycelial growth in root-to-root contamination; more precisely, we lack knowledge about the dynamics of CCS. In this context, we designed a rhizobox system to investigate the mechanisms involved in potential alloinfection via diseased lateral roots of carrot and to look specifically for a link between direct physical contact among roots and alloinfection. We also asked whether root exudates alone enhance mycelial growth in the soil.

Materials and methods

Daucus carota (cv. Nanco, Vilmorin & Cie, Paris, France) grown in a steam-sterilised soil mixture (50% sand, 25% compost, and 25% organic soil) was used. *P. violae* (strain Pv490, CBS 102.609, Baarn, The Netherlands) was grown at 20°C on carrot juice agar.

Following Green and Jensen (2000) and Rekah et al. (1999), using Petri dishes and a water agar medium, we verified that a 200-µm nylon mesh does not prevent the spread of mycelium of *P. violae*. Then, rectangular rhizoboxes were divided into two identical compartments (the donor and the receptor compartments) using that nylon mesh, which created a 30-mm wide central buffer zone designed to prevent contact between the carrot root systems without preventing movement of *Pythium* through the nylon mesh (Fig. 1a). Four polyvinyl chloride (PVC) tubes were placed in the donor compartment in a row 30 mm from the central buffer zone (the distance between the centre of the PVC tube and the nearest nylon mesh) (Fig. 1b). The two compartments and the buffer zone were filled with the soil mixture.

Experiments were done in a greenhouse maintained between 17 and 27°C, compatible with growth of both *D. carota* and *P. violae* (Suffert and Guibert 2007). Eleven carrots (the receptor plants) were sown in the receptor compartment, in two staggered rows 30 mm (first row) and 50 mm (second row) from the nearest nylon mesh. Fresh store-bought Nanco carrots were used as donor taproots. They were wounded at two spots (each 1–2 cm^2) with abrasive tissue, and a mycelial plug (5 mm in diameter), extracted from a 7-day-old *P. violae* culture, was placed on each wound (Suffert and Montfort 2007). Plugs were held in place with sterile pins. Inoculated taproots were incubated for 48 h in hermetically sealed plastic boxes. The agar plugs were then removed. Alloinfections by *P. violae* were generated in rhizoboxes by replacing the PVC tubes with these donor roots 12 weeks after the sowing of the receptor plants.

Fig. 1 Rhizobox apparatus and treatment setup to study the effect of lateral roots on alloinfections by *Pythium violae*. **a–b** Dimensions and composition of rhizobox. $w = 250$ mm, $l = 360$ mm, $h = 320$ mm. Material: 26-L polypropylene container; 200-µm nylon mesh; 32-mm diameter polyvinyl chloride (PVC) tube; 6-mm thick PVC strip; 5-mm diameter inox screw and wing bolt. **c–f** Infected carrots were transplanted into rows in the rhizobox at one of two distances from carrots later sown from seed. Distances: $i = 30$ mm, $j = 90$ mm (first row of receptor taproots) or $j = 110$ mm (second row of receptor taproots). Treatments: **c** N−/R−. **d** N−/R+. **e** N+/R−. **f** N+/R+ (*N*+ with nylon mesh, *N*− no nylon mesh, *R*+ with added roots, *R*− no added roots)

Sixty carrots were sown in the central buffer zone in treatments N+/R+ and N−/R+ (see below) 3 days after the transplantation of the donor taproots, so that the young roots provided a root system similar to lateral roots and as a way to enhance root exudates and any potential effect on mycelial growth. The leaves were cut 1 cm above the soil surface 18 days after. The receptor taproots were harvested 2 weeks later, i.e., 5 weeks after the transplantation of the donor taproots because in prior experiments several alloinfections occurred within 4 weeks after transplantation (Suffert and Montfort 2007).

The experiment had a two-factor, completely randomized factorial design (N and R) with eight replications, i.e., a total of 32 rhizoboxes. The treatments were (1) rhizoboxes without nylon mesh or added roots (N−/R−) (Fig. 1c), (2) rhizoboxes without nylon mesh and with added roots (N−/R+) (Figs. 1d and 2), (3) rhizoboxes with nylon mesh and without added roots (N+/R−) (Fig. 1e), and (4) rhizoboxes with nylon mesh and with added roots (N+/R+) (Fig. 1f). In treatment N−/R−, the mycelium of *P. violae* could grow from CCS lesions on donor taproots to receptor taproots, and the lateral roots of the donor and the receptor root systems could contact each other as in the field. In treatment N−/R+, mycelial growth was potentially enhanced by exudation from the presence of added roots. In treatment N+/R−, the mycelium of *P. violae* could grow from lesions on the donor taproots to the receptor taproots through the nylon mesh; however, the amount of contact between lateral roots of the donor and of the receptor root systems was greatly reduced, and the root exudation in the central buffer zone was also reduced (not formally estimated). In treatment N+/R+, the mycelium of *P. violae* could grow through the nylon mesh; however, contact between lateral roots of the donor and of the receptor root systems was greatly reduced, and mycelial growth was potentially enhanced by exudation from the presence of additional roots.

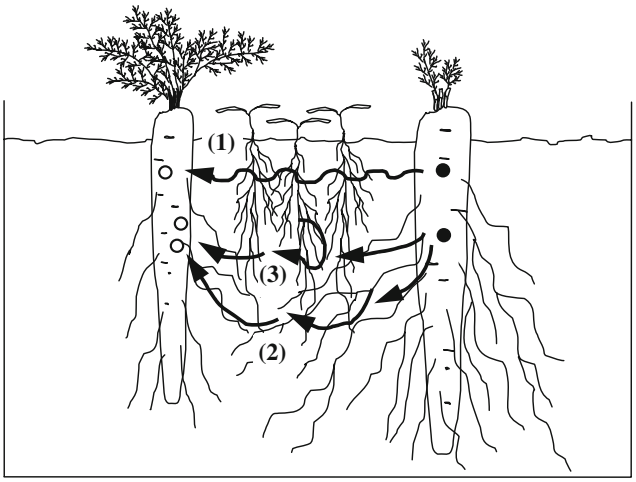

Fig. 2 Diagram of processes involved in alloinfections by *Pythium violae* and potentially occurring in the treatment N−/R+. (1) Active mycelial growth in the soil alone. (2) Infections enhanced by direct physical contact between lateral roots of donor and receptor root systems. (3) Mycelial growth enhanced by exudation from added roots. Black circles represent CCS lesions on the donor taproot and *white circles* represent CCS lesions on the receptor taproot

CCS lesions were scored using standardised measurements defined by Suffert and Montfort (2008): *i* is the disease incidence, tda is the total diseased area, *d* is the lesion density, and si is the symptom intensity. Statistical analyses were carried out using the SAS statistical package, version 8.1 (SAS Institute 2000). Treatment effects were tested on the average CCS measurements (*i*, tda, *d*, and si) of the 11 receptor taproots in the first and second rows in each rhizobox using ANOVA (SAS PROC GLM) including main effects (N and R) and interactions (N × R) for the two-factorial model (*F*-test, $P < 0.05$):

$$Y_{ijk} = \mu + \alpha_i + \beta_j + (\alpha\beta)_{ij} + \varepsilon_{ijk},$$

where *Y* is a CCS measurement (*i*, tda, *d*, or si), $i =$ N−, N+ and $j =$ R−, R+ are the levels of factors N and R, and $1 < k < n/2$ (with $n = 16$) denotes replications per factor level combination.

Results

Alloinfections from donor to receptor taproots occurred in the rhizoboxes in the absence ($Y_{N-} > 0$) and the presence of the nylon mesh ($Y_{N+} > 0$), and the null hypothesis H_0: $Y_{N+} = Y_{N-}$ was not rejected. This confirmed that mycelium of *Pythium* could grow through the nylon mesh in soil.

Extremely few (0–8) fragments of lateral roots were found in the buffer zone in treatments N+/R+ and N+/R−. The central buffer zone prevented the growth and the intermingling of lateral roots from one compartment to the other. The differences in disease incidence *i* and symptom intensity si between treatments N+ and N− in the absence (R−) and presence of additional roots (R+) were not statistically significant (*F*-test, $P < 0.05$) (Table 1; Fig. 3). The differences in the occurrence of alloinfection between treatments N+ and N− for lesion density *d* and for total diseased area tda were higher, but also not statistically significant (*F*-test, $P < 0.05$), implying that root-to-root contact did not increase the contamination between donor and receptor root systems. Thus, the spread of *P. violae* through the soil may be the predominant mode of spatial spread of CCS.

Alloinfections from donor to receptor taproots occurred in the rhizoboxes in the absence ($Y_{R-} > 0$) and in the presence of added roots ($Y_{R+} > 0$), and the null hypothesis H_0: $Y_{R+} = Y_{R-}$ was not rejected. The added roots in treatment R+ had little impact on the spread of CCS from donor to receptor plants, especially for *i* and si (Fig. 3). Only low, nonsignificant differences between treatments R+ and R− were observed for *d* and tda (*F*-test, $P < 0.05$). Despite high standard errors for *i* and tolerable ones for *d* and si, this treatment established that root exudation potentially involved in root-to-root contamination is negligible in sterilized sandy soil.

Discussion

The two-compartment rhizobox system used in the present study confined carrot lateral roots to their respective compartment and thus prevented the carrot root system of each compartment from intermingling.

Although differences in the lesion density *d* between treatments N+ and N− were observed ($d = 2.3$ in N+/R+ compared to 4.3 in N−/R+, $d = 1.5$ in N+/R− compared to 3.5 in N−/R−), they were not statistically significant, and the hypothesis $Y_{N+} = Y_{N-}$ was not rejected: root-to-root contamination was not a major mechanism of the alloinfection. The hypothesis $Y_{R+} = Y_{R-}$ was not rejected

Table 1 Results of ANOVA to test the effects of each factor (N, R) and interaction (N × R) on the occurrence of alloinfections by *Pythium violae*

Variable	*i*		*d*		si		tda	
	F-ratio	*P*	*F*-ratio	*P*	*F*-ratio	*P*	*F*-ratio	*P*
N	1.07	0.310	3.10	0.089	0.77	0.389	2.48	0.126
R	0.12	0.729	0.53	0.474	0.13	0.723	0.36	0.555
N × R	0.04	0.844	0.01	0.978	0.96	0.335	0.03	0.876

The *F*-ratios measure the contribution of each factor (N, R) and interaction (N × R) on the variance of the response. When the *p*-value was lower than 0.05, the corresponding factor had a statistically significant 95% confidence level

i disease incidence, *tda* total diseased area, *d* lesion density, *si* symptom intensity

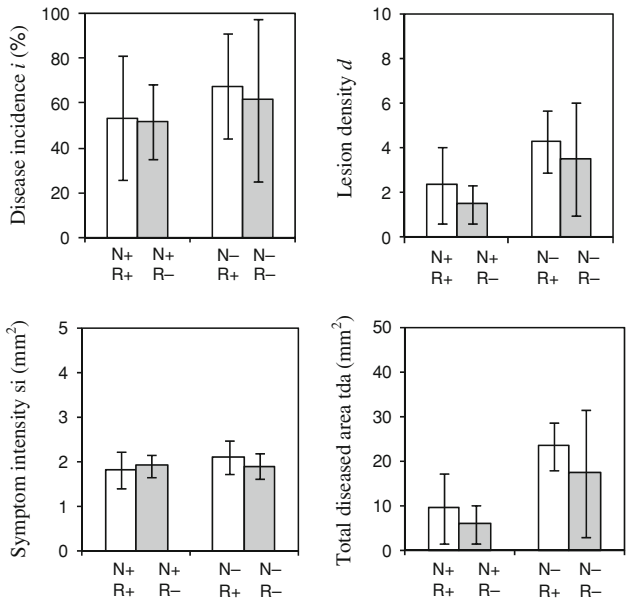

Fig. 3 Effect of carrot lateral roots on the occurrence of alloinfections by *Pythium violae*. *N+* with nylon mesh, *N−* no nylon mesh, *R+* with added roots, *R−* no added roots. Data are from 11 donor roots × 32 rhizoboxes. *Bars* represent the standard error. No differences between means were significant (ANOVA, *F*-test, $P < 0.05$)

either: root exudates did not enhance alloinfection. Consequently, the experiment established that active mycelial growth from CCS lesions is probably the most prevalent mechanism involved in alloinfections by *P. violae* because zoospores are of little importance (Van der Plaats-Niterink 1981). Similarly, Green and Jensen (2000) showed in controlled conditions that growth of mycelium from primary foci within the potting mix to roots of adjoining plants can be an important means of the spread of cucumber damping-off and root rot caused by *P. ultimum* var. *ultimum*.

Our results showed that *P. violae* can spread more than 90 mm throughout a sterilized sandy soil after 5 weeks. This distance is compatible with previous results obtained by Suffert et al. (2008) using taproot transplantation as a method of soil infestation by *P. violae* and with results obtained by Green and Jensen (2000) for *P. ultimum* var. *ultimum* (in steamed potting mix, hyphae on average grew 96 mm from diseased root tissue). The intensity of CCS on the receptor taproots in the second row was not significantly different from the intensity on taproots in the first row (data not shown). Thus, the mycelium of *P. violae* could grow equally through 90 or 110 mm in the given rhizobox system, perhaps enhanced by the first row of receptor taproots, which may support further disease spread.

The given rhizobox system could be improved, but constraints that contributed to heterogeneous results (high variance of disease intensities) are inherent to diseases caused by soilborne pathogens and cannot be easily taken into account. For example, the occurrence of alloinfection depends on edaphic conditions: low soil moistures and temperatures over 30°C prevent the development of *Pythium* sp. The duration of exposure between donor and receptor taproots cannot exceed 5 weeks in sandy soil or the transplanted taproots will rot.

The present experiment confirmed that a carrot taproot quickly produces new lateral roots and leaves regrow 2 weeks after transplantation (Suffert and Montfort 2007). Concomitantly with CCS lesions on taproots, brown to rust-colored microlesions were sometimes observed on carrot lateral roots, but *Pythium* species were rarely isolated from them (Suffert and Guibert 2007). Consequently, while lateral roots have appeared to enhance disease spread, this enhancement was probably due to an asymptomatic colonization of the root surface rather than to infection *sensu stricto*. Previous studies have shown that CCS lesions occasionally coincide with lenticels, but the lesions are not consistently associated with anatomical features such as sites of lateral roots origin (Perry and Harrison 1979).

The separation of two soil compartments by a selective barrier (e.g., a nylon mesh) was also used by Scott (1956), Rekah et al. (1999), Crowe and Hall (1980) and Green and Jensen (2000) to test the effects of infections from active mycelia in the soil alone and from direct physical root contact on the spread of soilborne pathogens and resulting disease, but their conclusions varied. Scott (1956) studied the spread of white rot from *S. cepivorum* in field onion crops and reported that the pathogen spread from inoculated onion bulbs to adjacent plants but did not spread when root systems were enclosed in small bags made of a nylon fabric even though mycelia from infected roots penetrated the mesh and came into contact with roots of neighbouring plants. To study the spread of *F. oxysporum* f. sp. *radici-lycopersici* from roots of an inoculated plant to roots of adjacent plants, Rekah et al. (1999) used containers divided by a 2.5-cm-wide wooden barrier, with an opening of 10 cm × 10 cm in the center. The openings were sealed with a 50-µm nylon mesh, and the space between the nets was filled with noninfested fumigated soil. When no barrier existed, 70% of plants adjacent to the focus plants were infected compared to less than 10% in containers with a barrier. The pathogen spread preferentially via root-to-root contact, unlike *P. violae*.

Two weeks after sowing, the added carrot roots were still thin enough to have a biochemical impact similar to lateral roots. Although differences in lesion density *d* between treatments R+ and R− were observed ($d = 2.3$ in N+/R+ compared to 1.5 in N+/R− and $d = 4.3$ in N−/R+ compared to 3.5 in N−/R−), they were not statistically significant, and the hypothesis $Y_{R+} = Y_{R-}$ was not rejected: the presence of additional roots in treatment R+ had a very limited impact on the CCS propagation from donor to receptor plants. The plant density is important for

Pythium-induced disease spread; for example, the increase in disease severity caused by increasing cucumber density was greatest at a lower concentration of inoculum of *P. ultimum* var. *ultimum* (Green and Jensen 2000), and a reduction in mean planting density was also effective in limiting CCS development by alloinfection (0.5, 1.6 and 2.0 lesions per root in microcosms containing 8, 16 and 31 roots, respectively) (Suffert et al. 2008). Very few studies have dealt with the effect of root exudates (exogenous carbohydrates and amino acids diffusing from host plants) on alloinfections. Zoospores of *P. ultimum* are attracted to roots of a number of plant species; accumulation typically occurs in the root hair region and the zone of cell elongation just behind the root cap (Nelson 1990). The production of secondary inoculum of *Pythium* sp. has already been established (Burdon and Chilvers 1975; Green and Jensen 2000; Suffert and Montfort 2007); however, no study has firmly differentiated between spread by mycelia from that by zoospores. Microscopic examinations of *P. ultimum* in the bulk potting mix confirmed that the spread was due to active mycelial growth because no zoospores were observed in the soil (Green and Jensen 2000). Van der Plaats-Niterink (1981) stated that *P. violae*, like *P. ultimum* var. *ultimum*, rarely produces sporangia and zoospores; however, this hypothesis should be tested for other *Pythium* sp. known to cause CSS (Hiltunen and White 2002). Direct microscopic observation of zoospores and mycelium growing either along the lateral roots or directly through soil may help to confirm this.

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