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Susceptibility of wild carrot (*Daucus carota* ssp. *carota*) to *Sclerotinia sclerotiorum*

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Abstract *Sclerotinia* soft rot, caused by *Sclerotinia sclerotiorum*, is a severe disease of cultivated carrots (*Daucus carota* ssp. *sativus*) in storage. It is not known whether *Sclerotinia* soft rot also affects wild carrots (*D. carota* ssp. *carota*), which hybridise and exchange genes, among them resistance genes, with the cultivated carrot. We investigated the susceptibility of wild carrots to *S. sclerotiorum* isolates from cultivated carrot under controlled and outdoor conditions. Inoculated roots from both wild and cultivated plants produced sclerotia and soft rot in a growth chamber test. Two isolates differed significantly in the ability to produce lesions and sclerotia on roots of both wild carrots and cv. Bolero. Flowering stems of wild carrots produced dry, pale lesions after inoculation with the pathogen, and above-ground plant weight was significantly reduced 4 weeks after inoculation in a greenhouse test. Wild and cultivar rosette plants died earlier and fewer plants survived when inoculated with the pathogen under outdoor test conditions. Cultivar plants died earlier than wild plants, but survived as frequently. Plants inoculated in the crown died earlier and at a lower frequency than plants inoculated on leaves. Wild carrots may thus serve as a host of *S. sclerotiorum* and thus eventually benefit from any uptake of resistance genes, among them transgenes, via introgression from cultivated carrots.

Keywords Pathogenicity · Risk assessment · Genetically modified crops · Wild plant–pathogen interactions

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Introduction

The cultivated carrot (*Daucus carota* ssp. *sativus*) has wild relatives belonging to the same species, among which the wild carrot *D. carota* ssp. *carota* is commonly found, for instance, along road verges in temperate regions (Brandenburg 1981; Holm et al. 1997), including Denmark (Hansen 1981). It is a common and sometimes serious weed (Mitich 1996;

Holm et al. 1997), in particular in no-tillage crop production systems (Stachler and Kells 1997). Cultivated and wild carrots are known to hybridize spontaneously, and wild carrots may therefore contaminate seed crops of cultivated carrots via its pollen (D'Antuono 1985; Hauser and Bjørn 2001), which is a major cause of genetic deterioration of seed stocks (D'Antuono 1985; Wijnheijmer et al. 1989). Also, there are strong indications that reciprocal hybridization and introgression from cultivated to wild carrot take place in nature (Wijnheijmer et al. 1989; Hauser and Bjørn 2001; Magnussen and Hauser 2007), and that hybrids are able to survive in natural habitats of wild carrots (Hauser and Shim 2007). Transfer of genes from cultivated crops to wild relatives may influence their evolution and ecological functioning. In the case of resistance genes to pathogens and pests, uptake of such genes may give wild plants a selective advantage and potentially make them more weedy in fields and wild populations. Increased and more continuous exposure of the resistance trait in wild populations, in addition to in the crop, may enhance the likelihood of resistance evolution in the pest, and deteriorate the possible role of wild relatives as resistance refuges to slow down resistance evolution (Conner et al. 2003; Andow and Zwahlen 2006). It is crucial to know to what extent important pathogens and pests affect wild relatives of our crop species to understand and manage these processes.

Genetically modified or transgenic carrots with enhanced disease resistance against *Erysiphe heraclei* and *Alternaria dauci* (Takaichi and Oeda 2000), and *Sclerotinia sclerotiorum* (Chen and Punja 2002), have been produced, and several carrot lines with enhanced disease resistance have been tested (http://gophisb.biochem.vt.edu/cfdocs/fieldtests3.cfm, http://biotech.jrc.it/deliberate/dbplants.asp). Among these, genetically modified varieties with resistance to *Sclerotinia* soft rot, caused by *S. sclerotiorum* may be highly attractive, as no qualitative resistance genes are available from conventional breeding (Kora et al. 2003). *Sclerotinia* soft rot is one of the most severe diseases of carrots worldwide, particularly during storage (Lewis and Garrod 1983; Kora et al. 2003). The pathogen has a very wide host range. Based on a literature survey, Boland and Hall (1994) collated a list of 408 host species from 75 families of higher plants, the majority of which are dicots. Isolates from individual plant species may attack a wide range of host species (Price and Colhoun 1975). The susceptibility of wild host plants to *S. sclerotiorum* has been studied, e.g. *Circium arvense*, (Bourdôt et al. 1995), *Agropyrum spicatum* (Jacobs et al. 1996), *Ranunculus ficaria* (Kohn 1995; Kohli and Kohn 1996), *Ranunculus acris* (Cornwallis et al. 1999), *Chrysanthemoides monilifera* ssp. *rotundata* (Cother 2000) and *Heracleum mantegazzianum* (Erneberg et al. 2003). However, information on the susceptibility of wild host plant species to *S. sclerotiorum* is much more limited than for economically important cultivated crop plants, and to our knowledge no investigations of *S. sclerotiorum* on wild carrot have been done previously. The aim of this study was therefore to investigate whether and to what extent isolates of *S. sclerotiorum* from cultivated carrot could infect wild carrots under controlled and field conditions to generate results for future risk assessments related to the potential release of transgenic *Sclerotinia*-resistant carrots.

Materials and methods

Three different inoculation experiments were carried out on wild (*D. carota* ssp. *carota*) and cultivated carrots (*D. carota* ssp. *sativus*) from different Danish populations under controlled and outdoor conditions (experiments I–III). *Sclerotinia sclerotiorum* isolates for the experiments originated from two geographically isolated carrot fields in Denmark: Lumby, Funen (isolates car13A-1, car13A-3, and car14A-6), and Ejstrupholm, Jutland (isolate carE55-1). They were collected as sclerotia formed on infected carrot leaves. Two different inoculation methods were used in the experiments: inoculation with mycelium reared on agarose (experiments I and II), and inoculation with mycelium-infected wheat kernels (experiment III).

Root test in growth chamber with mycelial agarose plugs (experiment I)

Plants from two wild carrot populations were collected on Kalvebod Fælled, Amager (55°39′ N, 12°35′ E), and in Silkeborg, Jutland (56°10′ N, 9°35′ E) in July. The plants from Amager had branched and flat leaf rosettes, whereas the plants from Silkeborg had more upright foliage. Roots from both sites were divided into two groups: small and larger (possibly older) roots. All roots

consisted of a main taproot with many smaller lateral roots. All plants from Amager were flowering. The plants from Silkeborg with larger roots were all flowering; plants with small roots were not. The roots were left with 5 cm leaf petiole and rinsed in tap water. Roots of cultivated carrot cv. Bolero F.1 (Vilmorin, France) were from a commercial carrot field on Lammefjorden, Sealand (55°47′ N, 11°30′ E) and had been kept in a cold store (1–2°C) since November the previous year. Root weights were determined, and roots were placed horizontally in 30×60 cm plastic trays (PK060, Vefi, Larvik, Norway) on a double sheet of filter paper (Filtrak, 95 g m^{-2}, D-09471, Bärenstein), humidified with 100 ml distilled water. The test was a split-plot design with three replications: root origin/size was the main-plot factor, isolate the sub-plot factor. An experimental unit consisted of six roots that were inoculated with either *S. sclerotiorum* isolate car13A-3 or carE55-1 for each root origin/size.

For inoculum, agar plugs with mycelium were prepared for the two isolates by placing a single sclerotium of *S. sclerotiorum* on a 9 cm Petri dish on 20 ml 4% *w*/*v* Potato Dextrose Agar (PDA, Scharlau Chemie, S.A., Barcelona) after surface-sterilisation (3% NaOHCl for 5 min). After 2 days at 20–22°C, 5 mm diam agar plugs were cut from the edge of the growing mycelium and transferred to the centre of a new Petri dish. Two days later, agar plugs with growing mycelium was inoculated onto tap water-rinsed roots of carrot cv. Bolero, and placed on humidified filter paper in a tray in a plastic bag at 20–22°C. Three weeks later, a batch of sclerotia was collected from the roots, and was used for production of mycelium plugs on PDA as above. A 5 mm diam mycelial plug from a 2 day-old culture was placed on the centre of each root. The inoculated roots were covered with another tray, functioning as a lid, placed in a plastic bag, and incubated in a growth chamber at 15°C in darkness.

Lesion lengths on the main root (mycelial growth) were recorded 1 week after inoculation (wai). Three wai, the number of sclerotia on the roots, the weight of the sclerotia, and the root weight after scraping off the rot were determined. Root decay during the experimental period was determined as the difference between start and final root weights. Means were calculated for each plant type (origin/size) and used for analyses of variance (ANOVA:general linear model). SAS/STAT version 6.12, SAS Institute Inc., Cary, NC, USA was used for all statistical analyses. This programme was also used for statistical analyses in experiments II and III.

Whole plant greenhouse test with mycelial plugs (experiment II)

In the period May–July, 44 plants of flowering wild carrots were transferred from the site at Amager to 3.5 l plastic pots with fertilized peat (Weibulls Enhetsjord, K-Jord, Svalöf Weibull Trädgärd AB, Hammenhög, Sweden) and placed under greenhouse conditions. In July, when the plants were well established in the pots, they were divided into 22 pairs similar in size: one plant for inoculation, and one control plant. A 10 mm diam mycelial plug of isolate car13A-3, produced as for experiment I, was placed at the base of a petiole on each of the 22 plants. Control plants were mock-inoculated with the same size PDA discs. A humidified piece of foam rubber was placed on top of the inoculum, and the pot surface was wrapped with plastic for 3 days to maintain high humidity. Thereafter the plastic and the foam rubber were removed and the plants were kept under greenhouse conditions at 20–30°C.

Lesion lengths on stems were recorded 3 days after inoculation, and 2 and 3 wai. The above-ground plant weight and root weight was determined 4 wai, and the stems were cut open to check for sclerotia. Above-ground plant weight and root weight were analysed in a paired *t*-test, with each plant pair representing a replicate. The analysis was performed on log-transformed data to achieve homogeneity of variance.

Whole plant outdoor test with infected wheat kernels (experiments IIIa and IIIb)

Seeds from two wild carrot populations on Sealand: Hundested (55°58′ N, 11°55′ E) and Karlstrup (55°33′ N,12°12′ E), and two open-pollinated carrot cvs Flakkeer ‘Regol’ and Nantes 6 ‘Fancy’, (L. Dæhnfeldt A/S), were sown in 12 cm pots with a peat-soil mixture in April, and thinned to seven plants per pot after 1 month. Ten week-old plants (in the rosette stage) were inoculated with wheat kernels of isolate car13A-1 and car14A-6. Infested wheat kernels of the two isolates were prepared as follows: Fifty ml of wheat kernels were soaked in water overnight in a 250 ml Erlenmeyer flask. The following day, the

water was drained off, the flask plugged with cotton and sterilized three times. Then, actively-growing mycelium from a 9 cm PDA plate was added, and the flask kept at room temperature for 7 days, until the grains were colonised and sclerotia started to form. Flasks were shaken daily to keep the wheat kernels separate. One kernel was placed either between the bases of the leaf stems of the crown (crown inoculation) or midway on a leaf stem (leaf inoculation). For leaf inoculations, a bent metal paper clip was used to hold the kernel in place on the leaf by bending the leaf horizontally to the soil surface. After inoculation, the pots were humidified, using a hand-held atomizer, and kept in plastic bags on water-containing trays at a shaded site. Two days later, the plastic bags and clips were removed and the state of the kernels and infection noted.

Two pots were treated for each combination of isolate, incubation method, and population/cultivar, and one pot per population/cultivar and incubation method served as the mock-treated control (i.e., 32 treated and 8 controls; 280 plants in total; experiment IIIa). The experiment was repeated 3 weeks later, but this time only with plants/pots from the two wild plant populations (*i.e.* 16 treated and 4 controls; 140 plants in total; experiment IIIb).

Development of disease symptoms and survival/death of the plants were assessed approximately once a week for six (experiment IIIa) and 3 (IIIb) weeks, and then at 14 weeks. The final survival rate was registered at the end of the experiment. The time to plant death (determined as the assessment (number) at which a plant was first recorded dead) was tested by ANOVA (proc. Anova, SAS). Data on final survival rates were analysed by logistic regression (proc. Genmod, SAS).

Results

Root test (experiment I)

Sclerotinia sclerotiorum established on all inoculated roots. Whitish mycelia were observed and resulted in soft rot lesions on both the cultivated and the wild roots (Fig. 1a,b). Analysis of lesion lengths 1 wai showed a significant difference among carrot origin/size (P=0.0009), and between the two isolates (P=0.0001). Lesions were significantly longer on the wild carrots than on the cultivated roots, and tended to be longer on the small roots than on the larger and thicker roots (Table 1), indicating that mycelial growth was directed outbound towards non-infected tissue, in particular when growth in depth was limited by the root thickness. Isolate car13A-3 was more aggressive than isolate carE55-1 (Table 1). Two wai the lengths of the roots clearly limited the growth of the fungus (data not shown).

Sclerotial initiation was observed 1 wai, and many dark, melanized sclerotia were seen from 2 wai and onwards both on the main taproot and on the lateral roots of the wild roots as well as on the cultivated roots (Fig. 1a,b). Sclerotia number was significantly different among carrot origin/size (P=0.0001) with many more formed on the larger roots of Bolero than the wild carrots. A significant difference was also found between the two isolates (P=0.0001) with car13A-3 producing the highest number. The weight of sclerotia also differed significantly among carrot origins/sizes (P=0.0055) with a higher mean sclerotium weight on Bolero than on the wild roots (Table 1).

Three wai, the cortex of the wild roots was decayed. The stele was intact in the large roots, but decayed in the small roots. In the cultivated roots, both stele and cortex were decayed. Root origin/size showed a significant effect in terms of root decay (P=0.0001) and was more pronounced for cv. Bolero than for the wild carrots; larger wild roots tended to result in more root decay than the smaller roots, indicating that the initial root weight was a limiting factor for the decay.

Whole plant greenhouse test (experiment II)

Twenty-one out of the 22 inoculated plants became infected and exhibited symptoms. Pale to light brown dry lesions up to 2 cm long were visible near the inoculation site on flower-bearing stems as early as 3 days after inoculation, and the lesions expanded along the stem during the following weeks (Fig. 1c, Table 2). Two wai, the inoculated plants showed wilting symptoms, and 3 wai they were visibly necrotic, and the first sclerotia were produced. Four wai, sclerotia had formed on 10 out of 22 inoculated plants. None of the 22 agar-only control inoculated plants showed symptoms of disease (Fig. 1d).

Four wai, there was a significant effect of disease on the above-ground plant weight (P=0.002), which

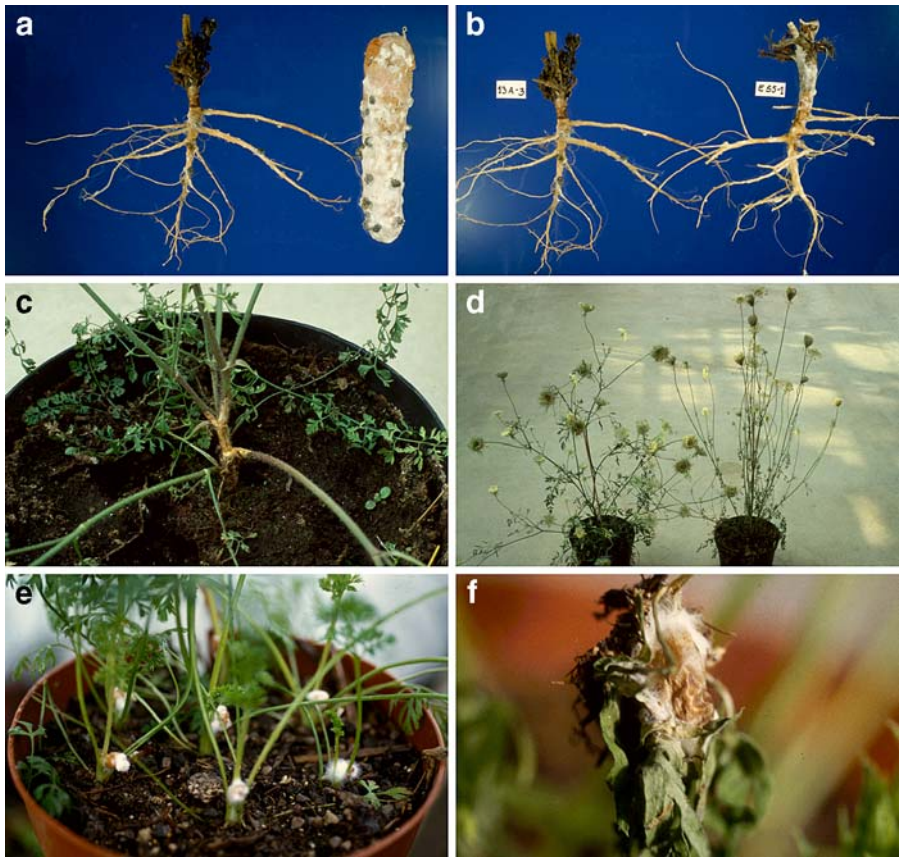

Fig. 1 Symptoms caused by *Sclerotinia sclerotiorum* on cultivated and wild carrot under controlled conditions (**a**–**d**) and kernel inoculations and symptoms on wild carrots under outdoor conditions (**e**–**f**). **a** Mycelial and sclerotial development 3 weeks after inoculation (wai) on roots of cultivated and wild carrot. **b** Mycelial and sclerotial development on wild carrot roots with two different isolates 3 wai. **c** Lesion development on petiole and inflorescence bearing stem of wild carrot plant 1 wai. **d** Control plant (*left*) and infected wilting wild carrot plant (*right*) 2 wai. **e** Inoculation of the crown of wild carrot plants in outdoor experiments with mycelium-infected kernels. **f** The resulting wilting symptoms from leaf inoculation

was reduced to approximately 50% in comparison to control plants, but there was no significant effect on root weight (P=0.0532; Table 2). Nevertheless, the root tissue of the inoculated plants had decayed, except for the stele, and exhibited a brownish colour in comparison to the roots of the agar-only control inoculated plants, which were white-beige.

Whole plant outdoor test (experiment III)

Both wild and cultivated carrot plants were infected by *S. sclerotiorum*. Leaves developed pale, green to light brown, water-soaked symptoms and decayed gradually (Fig. 1e,f). These symptoms were not seen on any of the control plants.

In experiment IIIa, wild carrots and leaf-inoculated plants survived significantly longer after inoculation than cultivated and crown infected, respectively (P= 0.0045 and P=0.0001; Table 3). The final survival rate at the end of the experiment was significantly lower for inoculated than for control plants (P=0.003; Table 3). Significantly fewer plants survived of those inoculated in the crown compared to those inoculated on the leaves (P=0.0001); there was no difference in survival between wild and cultivated carrots (P=0.10) or between plants inoculated with the two isolates (P=0.9; Table 3).

In experiment IIIb, leaf-inoculated plants also survived for a longer time than crown-inoculated plants (P=0.01; Table 3), whereas there was no difference in their final survival rate which was significantly lower for inoculated plants than for control plants (P=0.012); overall survival was much lower in experiment IIIb than in IIIa, probably due to an aphid attack during the summer. No sclerotia developed during the two experiments.

Discussion

In three different experiments, *S. sclerotiorum* isolates from cultivated carrot were pathogenic on both

Table 1 Root characteristics, lesion development, formation of sclerotia and root decay of cultivated and wild carrot roots after inoculation with either of two isolates of *Sclerotinia sclerotiorum* from cultivated carrot (car13A-3, and carE55-1) (experiment I)

	Root assessments		Disease assessments			
	Root length (cm)	Root weight (g)	Lesion length (cm) 1 wai	Formation of sclerotia (number)	Sclerotia weight (g) 3 wai	Root decay (g)
Wild carrots						
Amager-small roots, F[a]	9.72	3.04 (1.93)[b]	6.61	4.08	0.012	1.29 (1.23)
Silkeborg-small roots	10.00	2.33 (1.65)	6.37	3.83	0.016	2.32 (1.28)
Amager-larger roots, F	11.03	7.45 (3.32)	6.00	8.11	0.018	4.67 (1.58)
Silkeborg-larger roots, F	13.73	7.44 (3.33)	5.04	4.94	0.017	3.31 (1.43)
Cultivar						
Bolero F.1 =	15.46	93.79 (15.24)	3.99	30.39	0.043	65.12 (3.48)
$LSD_{0.05}$	1.31	(0.41)	1.06	2.44	0.007	(0.16)
Isolate						
car13A-3	11.60	21.83 (1.57)	7.07	12.70	0.241	19.16 (1.88)
carE55-1	12.38	23.79 (1.59)	4.14	7.84	0.198	13.54 (1.85)
$LSD^{c}_{0.05}$	ns[d]	(ns)	0.67	1.54	ns	(ns)

wai Weeks after inoculation

[a] *F* Plants from which the roots originated were in flower.

[b] Values in parentheses are means after transformation of data in SAS (**0.6 for root weight and **0.3 for root decay).

[c] $LSD_{0.05}$ Least significant difference at $P=0.05$.

[d] Not significant

cultivated and wild carrot, and the disease cycle was completed with the formation of sclerotia on both subspecies under the controlled conditions. This is in accordance with previous reports stating that the pathogen is plurivorous and that it may be pathogenic not only to the host plant from which it is isolated, but also to a range of other host plant species (Price and Colhoun 1975), and therefore also to wild and cultivated subspecies of carrot. The isolates used in the root test exhibited a difference in aggressiveness determined on the basis of the ability to cause lesions and form sclerotia. Such differences in the aggressiveness of *S. sclerotiorum* isolates have previously been reported in other studies of *S. sclerotiorum* interactions with tomato and dahlia (Price and Colhoun 1975), alfalfa (Pratt and Rowe 1995) and

Table 2 Lesion lengths, weight of aerial plant parts and root weight of wild carrot plants, control, and inoculated with *Sclerotinia sclerotiorum* on the petiole at the stem base during the flowering phase (experiment II)

Treatment	Lesion length (cm)[a]			Above-ground plant weight (g)[b]	Root weight (g)[b]
	1 wai	2 wai	3 wai	4 wai	
Inoculated	2.42 (0.16)	4.44 (0.73)	5.32 (0.75)	26.43 [2.96]	3.02 [1.20]
Control	0.00	0.00	0.00	51.77 [3.67]	4.07 [0.93]
$LSD^{c}_{0.05}$	–	–	–	[0.30]	[ns][d]

wai Weeks after inoculation

[a] Values in parentheses are standard errors, $n=22$.

[b] Values in brackets are means after log transformation of data.

[c] $LSD_{0.05}$ Least significant difference at $P=0.05$.

[d] Not significant

Table 3 Average time to death[a] and final plant survival rate after inoculation of wild and cultivated carrots in the rosette stage with two isolates of *Sclerotinia sclerotiorum*, either on the leaves or in the crown (experiments IIIa and b)

Assessment	Time to death[a]		Final plant survival rate[b]	
	Wild carrot	Cultivated carrot	Wild carrot	Cultivated carrot
Experiment IIIa				
Infection method				
Leaf inoculation	6.3[a] (0.3)	6.5[b] (0.2)	0.66[a] (0.12)	0.72[a] (0.11)
Crown inoculation	5.2[c] (0.3)	3.5[d] (0.1)	0.44[b] (0.07)	0.10[b] (0.04)
Isolate				
Isolate car13A-1	5.4 (0.4)	4.0 (0.3)	0.64[ab] (0.09)	0.40[ab] (0.15)
Isolate car14A-6	5.8 (0.3)	4.3 (0.3)	0.46[ab] (0.12)	0.42[ab] (0.13)
Control	–	–	0.93[c] (0.07)	1.00[c] (0.00)
Experiment IIIb				
Infection method				
Leaf inoculation	3.4[a] (0.1)	–	0.04[a] (0.03)	–
Crown inoculation	1.8[b] (0.1)	–	0.06[a] (0.04)	–
Isolate				
Isolate car13A-1	2.6 (0.2)	–	0.02[a] (0.02)	–
Isolate car14A-6	2.6 (0.2)	–	0.08[a] (0.04)	–
Control	–	–	0.29[b] (0.00)	–

[a] Time to death was measured as the first assessment at which a plant was recorded as dead, out of seven assessments in experiment IIIa and four in experiment IIIb; standard errors in parentheses.

[b] Means with standard errors in parentheses. Statistically different effects are indicated by superscript letters.

soybean (Kull et al. 2004). Leaf crown infections resulted in lower plant survival rates than leaf infections, an observation also reported by Finlayson et al. (1989), suggesting that the infection route for the pathogen to enter roots under field conditions may be via the crown.

The overall effect of any pathogenic fungus on a host plant depends on properties of the pathogen, the host, the pathogen–host interaction, and environmental factors. We have shown that *S. sclerotiorum* affects plant growth and survival under controlled conditions, which are conducive for pathogen attack and disease development. When young plants in the rosette stage were infected, this resulted in plant death. Also, when plants were attacked during the flowering stage, the disease reduced the aerial weight of the plant parts, and caused premature wilting resulting in plant death. These results indicate that *S. sclerotiorum* has a negative effect on the fitness of wild carrots when conditions are favourable to the pathogen. Two different inoculation techniques, both based on mycelial inoculum, were used. Both infected wheat kernels and mycelial agar disks resulted in disease attack. The production and inoculation with mycelial agar disks seemed easy to work with, and generally produced severe symptoms on the plants.

Our results thus corroborate the view that plant diseases may have a large impact on plant populations (Burdon et al. 2006). Diseases of crops may affect the surrounding plant communities, and diseases in susceptible wild populations may serve as a reservoir of inoculum for disease to spread to nearby cultivated crops (Wang et al. 2004). In the case of *Sclerotinia* soft rot in carrots, disease dynamics may be further influenced by the possible introgression of resistance genes, among them transgenes, from cultivated into wild carrots (Wijnheijmer et al. 1989; Magnussen and Hauser 2007; Hauser and Shim 2007). Cultivar resistance genes may enter wild populations directly from seed and root-production fields; some individuals often flower in root-production fields, especially after a cold spring, despite being bred to be biennial (Hauser and Bjørn 2001). Cultivar genes may also reach wild populations indirectly: seed plants in seed-production fields are sometimes pollinated by surrounding wild carrots, and the resulting hybrid seeds are transported with the seed lots to other cultivation regions, e.g. in Denmark. Here, the hybrids may

flower in carrot fields and pollinate each other and probably neighbouring wild plants (Hauser and Bjørn 2001).

If resistance genes are transferred, they may confer a selective advantage to the wild carrots in areas with occurrence of the pathogen. For instance, it has been shown that resistance to *Hyaloperonospora* (formerly *Peronospora*) *parasitica* in *Arabidopsis thaliana* may enhance the competitiveness and the predicted probability of long-term ecological success when resistant plants in a plant community are exposed to the disease (Damgaard and Jensen 2002). Increased plant fitness may result in changed weed dynamics (Pascher and Gollmann 1999); in our case, wild carrots may become a more problematic weed in carrot seed and production areas. It may also enhance the chance for survival of wild carrots in *Sclerotinia*-infested areas, as has been shown for wild gooseberries with resistance to American gooseberry mildew (*Podosphaera mors-uvae*, formerly *Sphaerotheca mors-uvae*), which is introgressed from the cultivated type (Warren and James 2006).

Increased exposure of the crop resistance gene, both in time and space, may decrease the time period before the fungal disease evolves a counter-resistance, enabling it to again utilize the host plant. Spontaneous transfer of resistance genes to related wild plants may thus complicate resistance management in the crop. However, whether a particular crop resistance gene will actually spread within a wild host population depends strongly on cost–benefit relationships, which vary substantially according to the nature of allelic variability at the locus in question, developmental responses to the pathogen, nutrient availability and environmental conditions (McDowell and Simon 2006). Cost of resistance in terms of plant fitness has been shown only in extremely few cases. For instance, a study of R-gene-mediated disease resistance in *A. thaliana* isogenic lines revealed a fitness cost of resistance with no pathogen pressure (Tian et al. 2003), showing that the fate of such genes depends on a balance between costs of resistance and infection.

Our study has revealed that wild carrots are clearly susceptible to *S. sclerotiorum* under conducive environments and that infection affects the fitness of the plants. This needs to be taken into consideration when assessing the ecological and agronomic impacts of releasing genetically modified *Sclerotinia*-resistant carrots.

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