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# Effect of root injury on lesion development of caraway roots infected by *Mycocentrospora acerina*

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**Abstract**

The effect of injury on disease incidence, incubation period and lesion development rate on caraway roots by *Mycocentrospora acerina* was studied in three laboratory experiments. After inoculation with *M. acerina*, disease incidence of injured roots was significantly ($P < 0.001$) higher than of non-injured roots. The incubation period of *M. acerina* was significantly ($P < 0.001$) shorter on injured roots than on non-injured roots. The incubation period shortened with increasing root injury level. Younger injured roots tended to be more resistant to *M. acerina* infection than older injured roots, expressed by longer incubation periods. The lesion development rate was, on average, higher on heavily injured roots than on non- or slightly injured roots. The lesion development rate remained fairly constant after the first emergence of the symptoms on the caraway root, until the whole root was colonized. Caraway roots carefully dug up in autumn frequently showed injuries enabling *M. acerina* to penetrate the roots. However, the correlation between root injury and root rot after cold storage was weak. Injury of roots had a stimulating effect on infection and development of *M. acerina*, but roots without wounds could be infected too. Some relevant field observations are discussed.

## Introduction

Biennial caraway (*Carum carvi* L.) is an arable crop which, in the Netherlands, is grown mainly on clay soils. In the 1980's anthracnose (Westerdijk and van Luijk, 1924) caused by *Mycocentrospora acerina* (Hart.) Deighton was responsible for severe yield losses. The soil is probably a major inoculum source (Truscott, 1944; Newhall, 1944; Rader, 1945). In carrots (*Daucus carota* L.) the fungus enters the root mainly through wounds (Davies et al., 1981) and causes liquorice rot (Rader, 1945). Caraway roots frequently suffer mechanical injury by farm equipment and gnawing insects.

In stored carrots a positive correlation was found between the degree of root injury at harvest and the occurrence of liquorice rot after two months in storage (Davies et al., 1981). In agricultural practice, injury on caraway roots might be inflicted by mechanically weeding. Wounds can be caused by insects, mice and organisms of the soil fauna. Especially the caraway root aphid (*Pemphigus passeki* Börner) may injure caraway roots (Prinsen, 1991), and this insect possibly provides points of entry to *M. acerina*.

The aim of this study was to find out whether the development of lesions on caraway roots by *M. acerina* was enhanced by mechanical injury. The phenomenon was studied under controlled conditions in a climate chamber. Root injury under field conditions was considered. Root injury in autumn may be indicative for the risk of anthracnose development, and might be used to decide upon whether action is needed.

## Materials and methods

### *Treatments*

In experiments 1 and 2, caraway was sown early, in March 1991 and March 1992, respectively. Spring bar-

ley (*Hordeum vulgare* L.) was used as a cover crop. In experiment 3 no cover crop was used, therefore caraway was sown late, in June 1994. Caraway roots were collected from the field in spring 1992, autumn 1992 and autumn 1994. The ages of the caraway roots at lifting for use in the experiments 1, 2 and 3 were 373, 227 and about 100 days, respectively. The roots were carefully lifted, though the tap root inevitably broke off, thus creating a distal wound. The roots were gently washed to remove adhering soil. The leaves were removed by cutting, leaving the petiole bases. Roots with diameters between 8 and 11 mm and a length of approx. 15 cm were used in the experiments. The roots used had no apparent injury or infection, the distal wound excepted. The roots were artificially injured with a punching apparatus, creating one wound per root. A cylinder of tissue (diameter 5 mm) was removed to different depths, in order to create different levels of injury (Davies et al., 1981). Four levels of injury were applied.

0: no injury
1: only the epidermal layer removed
2: cortex cells were removed but the central cylinder was not injured
3: the central cylinder was injured

The roots thus prepared were placed on moist filter paper and incubated in covered plastic boxes.

*Inoculation*

An isolate of *M. acerina* originally obtained from a caraway root was used in all experiments. Chlamydospores were obtained by growing colonies for three weeks on sucrose agar (6g sucrose, 15 g agar and 200 mg streptomycin per litre medium, after Day et al., 1972). at 12°C. Inoculum was prepared by blending agar discs with *M. acerina* colonies in tap water. The suspension was sieved through double cheese cloth. The inoculum density was adjusted to $5x10^4$ chlamydospore chains of *M. acerina* per ml suspension. With a pipette a drop of 0.1 ml suspension was placed in the wound created on the root. Non-injured roots were inoculated too. As a control treatment some roots of each injury level were treated with tap water instead of a chlamydospore suspension. Three experiments were carried out. In experiment 1 four levels of root injury were distributed in 8 boxes in a completely randomized design with two replicates. Each box contained 4 inoculated roots and one root treated with water. Experiments 2 and 3 were carried out in the same manner but had 4 replicates, whereas each box contained 2 roots inoculated with *M. acerina* and two treated with water.

*Incubation and disease observations*

During the experiments the temperature was kept at 5°C. This temperature was chosen to simulate conditions in the field during winter. A high humidity was maintained by placing plastic covers over the boxes. A high humidity (RH $>$ 90%) is necessary for the development of *M. acerina*, as shown by Newhall (1944) who worked with stored celery. Experiments 1, 2 and 3 continued for eight, 13 and 32 weeks, respectively. The experiments were carried out in the dark. Lesion development was assessed weekly by measuring the length (mm) of the lesions caused by *M. acerina*. Isolations from the roots were made at the end of the experiments to confirm that *M. acerina* had caused the lesions.

*Field observations*

Caraway root injury in autumn was assessed in nine field experiments and in one commercial crop between 1992 and 1994. In another commercial crop, root injury was assessed in spring. The commercial crop was grown in the province of Groningen (Gr). The field experiments were situated in the provinces of Groningen (Gr), Flevoland (Fld) and Gelderland (Gld). Caraway was sown on clay soils, except for two field experiments in the vicinity of Wageningen (Gld) which had a sandy soil. Per field between 16 and 64 samples were taken. Each sample consisted of 10 to 20 caraway roots. The roots were carefully lifted in December when most leaves had already decayed. The root samples were washed and injury assessments were made. The degree of root injury was rated as follows:

0: no root injury
1: 1–10% of the root length covered with injuries
2: 11–30% " " " " " " "
3: 31–70% " " " " " " "
4: 71–100% " " " " " " "

A root injury index was calculated per sample by multiplying class ratings by class frequencies, adding the products, and dividing the sum by the number of roots per sample.

After injury rating the roots were placed in cold storage as described above for the root injury experiments. After six months in cold storage the roots were examined for the presence of *M. acerina*. The percent-

age of the roots rotted (disease incidence) was determined. The roots were rated according to the percentage decayed tissue, using the above mentioned root injury scale. A root rot index was calculated in the same way as the root injury index. The root rot ratings were based upon the classes used by Hermansen (1992) to classify liquorice rot (*M. acerina*) on the mid-root of carrots, but where Hermansen used lesion number and size, we used percentages of root surface rotted.

*Statistics*

Results of injury experiments were analyzed by linear regression and analysis of variance (ANOVA). Linear regression was conducted on the development of the lesion length (mm) with time, starting from lesion initiation. For proper comparison of the three experiments only data of the first eight weeks were used. The slope of the regression line represents the lesion development rate (mm/week). The incubation period (weeks) was estimated as the intercept of the regression line with the time-axis. The incubation period and the log-transformed lesion development rates were subjected to analysis of variance. Least significant differences (LSD) at the $P = 0.05$ level are used to indicate differences between injury levels.

The effect of root injury in the field, assessed in autumn, on root rot after cold storage was studied by linear regression analysis with Genstat 5, release 3.1.

## Results

*Injury experiments*

*Disease incidence*

Observations on disease incidence were made eight weeks after inoculation. Non-inoculated roots showed no anthracnose. Roots treated with water were omitted from further analysis, since no *M. acerina* infection occurred. With inoculation, disease incidence of injured roots was significantly ($P < 0.001$) higher than disease incidence of non-injured roots. Inoculated roots with injury had 100% disease incidence in all three experiments. Inoculated roots without injury had disease incidences of 50, 68 and 50% in the experiments 1, 2 and 3, respectively. On inoculated caraway roots lesions expanded with time. The lesions were caused by *M. acerina*, as was confirmed by re-isolation of the pathogen. On non-injured roots lesions were restricted, with hardly any expansion after eight weeks (2.8 mm), due to a longer incubation period (Table 1) and a tendency for a lower lesion development rate (Table 1) than found on injured roots (20.4 mm). On severely injured roots (classes 2 and 3) lesions became progressive, and finally covered whole roots.

*Incubation period*

The incubation period of *M. acerina* on non-injured roots was significantly ($P < 0.001$) longer than on injured roots. The incubation period shortened significantly with the level of root injury in experiments 1 and 3, but not in experiment 2 (Table 1). The mean incubation period of *M. acerina* on injured roots in experiment 3 (2.9 days) was significantly ($P < 0.001$) longer than in experiments 1 (1.8 days) and 2 (1.9 days). The roots used in experiment 1 and 2 were considerably older (> 200 days) at lifting than the roots used in experiment 3 (100 days). The result suggests that the incubation period decreased with increasing age of the roots at the time of inoculation, but the relationship may have been caused by other differences between the experiments.

*Lesion development*

A tendency ($0.05 < P < 0.10$) was found that the lesion development rate of *M. acerina* increased with the root injury level (Table 1). No significant differences between treatments were found in lesion development rate on roots in experiment 1 and 3. In experiment 2 the lesion development rate increased significantly with increasing root injury. The mean lesion development rate of the roots in experiment 2 (2.6 mm/week) was significantly lower ($P = 0.005$) than in experiments 1 and 3 (3.4 and 3.4 mm/week), but the difference could not be ascribed to root age. Experiments 2 and 3 continued for 13 and 32 weeks, respectively. During the experiments the lesion development rate remained fairly constant from first emergence of the symptoms on the caraway roots until the roots were colonized completely.

*Field observations*

*Injury*

The incidence of injury on caraway roots under field conditions in autumn was usually found to be less than 25% (Table 2). Injury was found on 85% of the roots in a commercial crop, where caraway was grown for a second seed harvest. In another commercial crop, which was also to be harvested for the second time,

*Table 1.* Effect of root injury levels (four classes) on incubation periods and lesion development rates of *M. acerina* on caraway roots in cold storage after inoculation. The experiments 1, 2, and 3 began in March and October 1992 and September 1994, respectively. Non-inoculated controls showed no infections

| Class | Root injury level | Incubation period (weeks) | | | | Lesion development rate (mm/week) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Exp. 1 | Exp. 2 | Exp. 3 | Mean | Exp. 1 | Exp. 2 | Exp. 3 | Mean |
| 0 | Non-injured control | 6.6 a[2] | 5.4 a | 6.7 a | 6.3 a | 2.1 | 1.0 a | 3.3 | 2.1 a |
| 1 | Epidermal layer | 2.7 b | 2.1 b | 4.5 b | 3.1 b | 4.2 | 1.6 a | 2.8 | 2.8 ab |
| 2 | Cortex parenchyma | 1.4 c | 1.6 b | 2.4 c | 1.8 c | 4.7 | 3.1 b | 2.9 | 3.6 bc |
| 3 | Central cylinder | 1.3 c | 1.8 b | 1.8 c | 1.6 c | 3.8 | 4.5 b | 4.4 | 4.2 c |
| LSD[1] | | 1.0 | 1.8 | 1.1 | 0.7 | n.s.[3] | [4] | n.s.[3] | [4] |

1: Least significant differences for treatments 1–3 at P = 0.05.
2: Different letters indicate significant differences between injury treatments within columns, based upon LSD values for incubation periods.
3: Not significantly different at P = 0.05, but significantly different at P = 0.10.
4: Different letters indicate significant differences between injury treatments within columns of log-transformed lesion development rates.

root injury in spring was 61%. The root injury index of a caraway crop to be harvested for the second time was approximately five times higher than of first year's caraway crops.

*Disease incidence*

In cold storage pathogenic micro-organisms colonized the roots. After six months in cold storage approx. 50% of the roots had rotten. Root rot was caused by *M. acerina*, *Fusarium* spp., bacteria and other unidentified micro-organisms.

*Wounding and root rot*

When all fields were taken together the rotting of the roots was not significantly correlated with the level of injury assessed in autumn. However, in six out of ten fields a positive correlation between root injury index per sample and root rot index per sample was found (Table 2).

## Discussion

*Inoculum source*

In celery, chlamydospores in the soil were shown to be the main inoculum source of *M. acerina* (Day et al., 1972). In the present experiments, chlamydospores were chosen as the inoculum, since caraway root infection in soil is probably due to chlamydospores too. It is unlikely that a natural inoculum source interfered with the results, since no infections were found on control roots treated with water.

*Cold storage*

Roots of caraway remain in the field during autumn and winter. Circumstances in the field in winter are to some extent comparable to cold storage of carrots, although circumstances in the field are much more variable than in storage. The development of carrot plants slows down and growth almost ceases as the temperature falls during autumn (Lewis and Garrod, 1983), as will happen to caraway. But where carrots are lifted, caraway stays in the soil to flower next spring. The physiological activity of both carrot and caraway roots in winter is low. In such a period the defense of the roots probably weakens, so that *M. acerina* might penetrate and colonize the roots. If the physiological activity of caraway roots decreases relatively more than that of *M. acerina*, the fungus gains an advantage over the host in winter time.

*Wounding and disease incidence*

Davies et al. (1981) stated that liquorice rot was more often found in stored carrots severely injured at harvest than in carrots only slightly injured. They also found that liquorice rot was almost always connected with root injury, be it severe damages or ones invisible to the naked eye. They concluded that *M. acerina* was predominantly a wound pathogen. In parsnips (*Pastinaca sativa* L.), however, lesion development was not

*Table 2*. Effect of root injury in the field (9 trials and one commercial crop (G93a)) on root rot after cold storage. Linear regressions of the root injury index in autumn on the root rot index after cold storage were positive, $r^2$-values and P-values are given

| Experiment | Year of sowing/ harvest | Number of samples examined[1] | Root injury percentage [%] | Root injury index [–] | Percentage infected roots [%] | Root rot index [–] | Coefficient[2] of determination ($r^2$) | P-value |
|---|---|---|---|---|---|---|---|---|
| G93a | 1991/93 | 50 | 85 | 1.16 | 57 | 1.85 | –[3] | 0.73 |
| EH659 | 1992/93 | 50 | 19 | 0.27 | 52 | 1.43 | 0.13 | 0.006 |
| EH699 | 1992/93 | 50 | 17 | 0.22 | 35 | 0.87 | 0.14 | 0.006 |
| EH705 | 1993/94 | 48 | 19 | 0.21 | 55 | 1.07 | – | 0.25 |
| EH707 | 1993/94 | 16 | 10 | 0.12 | 50 | 1.02 | – | 0.43 |
| PAGV3433 | 1992/93 | 24 | 25 | 0.34 | 49 | 0.79 | 0.18 | 0.022 |
| PAGV3737 | 1993/94 | 36 | 8 | 0.08 | 51 | 1.53 | 0.16 | 0.010 |
| DR93B | 1992/93 | 24 | 5 | 0.06 | 47 | 1.41 | – | 0.078 |
| DR94B | 1993/94 | 24 | 4 | 0.06 | 44 | 0.70 | – | 0.37 |
| LN94B | 1993/94 | 64 | 5 | 0.05 | 50 | 1.87 | 0.14 | 0.003 |

1: Assessments were made on 10 to 20 roots per sample. 2: Coefficient of determination for linear correlation of root injury index in autumn with root rot index after cold storage. 3: Residual variance exceeds the variance of the Y variate.

increased by wounding the roots before inoculation (Channon, 1965). Artificial wounding had no influence on disease incidence of celery roots in storage (Gündel, 1976). In contrast, severe damage to the petiole of celery (*Apium graveolens* L.) plants considerably increased the rate of *M. acerina* infection (Day et al., 1972).

*Wounding of caraway roots*

*M. acerina* does not need visible wounds to infect caraway roots under the conditions provided in the study. Though care was taken not to injure the roots at lifting, some minute wounds might have been inflicted, especially on or near small lateral roots and root hairs. These minute injuries might have been the points of entry for *M. acerina* in the inoculated non-injured roots (class zero treatment). Both disease incidence and disease severity increased when roots were artificially injured. Minute wounds of caraway roots can easily occur during the formation of lateral roots. Organisms of the soil fauna can cause minute to medium sized wounds. Some agricultural practices cause injuries, such as weeding. Under cool conditions wound healing will be slow, as indicated by the fact that a period of high temperatures before cold storage accelerated wound repair of harvested carrot roots and diminished infection by *M. acerina* (Lewis et al., 1981). Especially in winter, conditions are relatively favourable for *M. acerina* development since the fungus is able to grow at low temperatures (Newhall, 1944; Neergaard and Newhall, 1951; Channon, 1965; Gündel, 1976). Any injury during such a period might lead to successful penetration and colonization of the caraway root. Roots of caraway to be harvested for a second year in succession tend to be more injured than roots of a first year caraway crop. This finding indicates that a second harvest caraway crop runs a higher risk to become seriously infected by *M. acerina* than a first harvest crop.

*Incubation period and lesion development rate*

The incubation period of *M. acerina* in stored carrots was up to four months when the roots were not injured (Lewis and Garrod, 1983). In carrots, progressive lesions developed rapidly in wounds which reached into the xylem parenchyma (Lewis et al., 1981). The incubation period of *M. acerina* on stored celery heads was seven weeks (Truscott, 1944). In celery plants, infection did occur at an early stage when the petioles were severely damaged (Day et al., 1972). In our experiments the incubation period was longer than six weeks when the caraway roots were not injured, which is in accordance with the observations in carrots. By damaging the caraway roots the incubation period for

*M. acerina* decreased drastically, as also found in carrots. On celery heads in storage *M. acerina* seems to behave similarly. No data on lesion development rates were found in the literature.

*Incubation period and root age*

The longer incubation period in experiment 3 as compared to experiments 1 and 2 may be caused by the age of the caraway roots at lifting. In carrot, wound infection increased with age of roots at harvest. Injured carrot roots harvested 127 days after sowing were more resistant to *M. acerina* infection than carrot roots harvested 162 to 211 days after sowing (Davies and Lewis, 1980). The same was found for colonization of caraway roots. In experiment 3 caraway roots were lifted about 100 days after sowing, and in experiments 1 and 2 after 373 and 227 days, respectively. The incubation period in experiment 3 was much longer than in experiments 1 and 2. The effect of root age on susceptibility of caraway to root colonization has an indicative value only since the results were obtained from three experiments not designed for this purpose. Nevertheless, the results suggest that serious caraway root infection might be expected at the end of winter rather than during the autumn. Furthermore, a caraway crop which will be harvested for the second time might run more risk of root infection, as observations in a commercial crop seem to confirm. In Germany, root infection of biennial caraway, probably caused by *Fusarium* spp. increased 2.5 times between September and June the following year (Plescher and Herold, 1983).

*Lesion types*

Davies et al. (1981) described two lesion types on carrot roots. The first type is a restricted lesion, which expands very slowly. The second type is a progressive lesion which expands rapidly. The first stage is often followed by the second when the carrot roots have been in storage for some months. Le Cam et al (1993) studied lesion development on carrots under various environmental conditions. On caraway roots these two lesion types were found too. Restricted lesions were prevalent on non-injured and slightly injured roots, whereas progressive lesions were prevalent on roots with medium and severe injuries. Caraway roots were relatively thin. As lesions rapidly ringed the roots, lesion development was adequately assessed by measuring lesion length.

Carrot roots have some degree of resistance to *M. acerina* but this resistance is easily overcome by mechanical injury. In carrot root tissues, a decrease in the concentration of falcarindiol was found from the epidermic layer to the central cylinder. Falcarindiol is believed to be the resistance factor since the concentration measured in the outer tissues is toxic to *M. acerina* (Davies & Lewis 1981; Garrod & Lewis 1982). Such a chemical component may exist in caraway roots too. A decrease in the concentration of such a component in the inner tissues would explain why injury of caraway roots leads to the formation of progressive lesions, as in carrots. For breeding purposes, identification and measurement of the chemical involved could be interesting. An alternative explanation might be that the mechanical resistance to penetration is much higher in the epidermal layer than in tissues beneath.

*Caraway root aphid*

The caraway root aphid injures the roots of caraway while feeding on phloem cells. The insect migrates to caraway from mid-June to mid-August and prevails during dry periods. Most insects leave the crop before winter, and only a few are able to overwinter on caraway (Prinsen, 1991). During the period when the majority of the caraway root aphids are present on caraway, conditions are unfavourable to *M. acerina* but favourable for wound healing. Considering the life cycles of the organisms involved, it is unlikely that the caraway root aphid causes root injury through which the fungus could penetrate the roots. In a small survey among some fields nor in field experiments any relation was observed between the presence of caraway root aphids and the incidence of anthracnose.

*Field observations*

*Root injury and root rot*

Caraway roots in commercial crops were frequently injured at the end of autumn. Root injury was especially marked in caraway crops to be harvested a second time. These injuries increase the probability of roots becoming infected by *M. acerina*, as found in the root injury experiments. Because disease assessments were hard to make on roots dug up in autumn, the roots were placed in cold storage in order to allow the pathogenic micro-organisms present to colonize the roots. In carrots, *M. acerina* does not spread from root to root in storage (Davies et al., 1981) and it is unlikely the fun-

gus will do so in caraway in the field. Approximately 50 percent of the caraway roots from commercial crops were rotten after cold storage. *M. acerina* infection was found occasionally but anthracnose was hard to distinguish from infections by other micro-organisms.

In several fields positive relationships were found between root injury and root rot, though correlations were poor (Table 2). Taking the fields together, linear regression showed no significant relation between mean root injury index in autumn and mean root rot index after six months of cold storage. Possibly the correlation was masked because the mean root injury levels did not vary much per field. Another explanation might be that *M. acerina* is not strictly a wound pathogen in caraway, so that even roots not injured at the time of lifting could have been infected by *M. acerina*. Because of the long duration of storage all the infected roots could become colonized entirely, despite a lower lesion development rate of *M. acerina* on non-injured roots. A third explanation might be that injuries not visible to the naked eye played a part in the infection of caraway roots by *M. acerina*. A fourth explanation might be that the occurrence of other pathogenic and saprophytic micro-organisms might have caused root rot irrespective of the injury level of the caraway roots at autumn, thus obscuring any correlation between root injury and root infection by *M. acerina*.

It is not known whether a positive relation between root injury and *M. acerina* infection under field conditions exists. From the results presented in the laboratory experiments such a relationship is plausible, but observation of root rot after cold storage could not confirm the relationship. The coefficients of determination were low (0.13–0.18), but the number of samples per field were high (24–64), so that (r from 0.36–0.43) the significance of the regression was high ($P \leq 0.01$). Apparently root injury can play a role in the loss of caraway plants through root rot, but that role is not very prominent. Root injury assessment in autumn cannot be used as an indication for anthracnose development on roots. Many micro-organisms were found in infected roots. It is not known whether these are secondary pathogens following *M. acerina* infection or whether they are primary pathogens to caraway roots.

*Further research*

The relative importance of the different pathogens involved in root infection must be investigated further. The importance of root infection on caraway growth and yield, relative to the effects of stem infection, has to be established. Data gathered sofar indicate that infection of the stems is more important than infection of the roots in respect to yield loss (Evenhuis and Verdam, 1995).

## Conclusions

This study shows that injury on roots increases the risk of root infection by *M. acerina*. An indication was found that a caraway crop which is to be harvested a second time runs a higher risk of becoming seriously infected by *M. acerina* than a first harvest crop.

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