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CONTENTS

PRACTICAL SECTION FOR GROWERS

Objectives of the review

Cavity spot disease of carrot has been studied by scientists for 36 years, during which time almost 200 publications have been produced worldwide, in languages as diverse as Danish and Hebrew. Many publications give valuable information on control of the disease, but are inaccessible to the grower. Further, there has been no attempt to take an overview of the accumulated data to give a clear picture of what we really do know about control of the disease. This review summarises data from most of the publications.

The first part covers general aspects of the disease, its geographical distribution and the tortuous path taken by scientists to determine what causes cavities. Since work in the early 1980's demonstrated that slow-growing *Pythium* species initiate lesions, we have largely been able to control the disease. However, it is not likely that the current method of control will last for ever, or continue to be ecologically acceptable. It is therefore necessary to consider ways of minimising the disease by management of those agronomic factors which the grower can control.

The section on disease management considers four separate aspects; fungicides, cultivar resistance, biological control and cultural methods. Below we list the major findings from each.

FUNGICIDES

No fungicide has been found to be consistently more effective than metalaxyl. Over many trials, no application of metalaxyl has been found to improve on a single application at, or around drilling, of 1.2 kg/ha active ingredient.

CULTIVAR RESISTANCE

Particularly for maincrop carrots there are cultivars which show some field resistance. These are listed in NIAB publications and publicised by seed firms. There is no evidence that cultivar resistance will hold up when inoculum levels are high, so even the best cultivars may go down with severe disease in some fields.

BIOLOGICAL CONTROL

Work in this area has been extremely limited both in volume and success. Biological control agents have been identified in a number of countries, and these may hold out hope for the future.

CULTURAL METHODS

This is the largest part of the review, and covers the area where growers are best equipped to take action themselves. Aspects covered are soil factors such as moisture, temperature,

aeration and inoculum level. Crop rotation is a major consideration, and aspects which may be beneficial are listed. The effects on disease development of soil nutrients and pH are considered alongside effects on disease of plant age and density.

DISEASE PREDICTION

Work funded by HDC gave growers the only quantitative information on cavity spot risk which they currently may access. Use of the cavity spot diagnostic test is described.

FUTURE RESEARCH

The review gives considerable guidance on research work which has not been done, or which needs improvement. There are six main areas which need attention, and they have been incorporated into a major programme which will be considered for Horticulture Link funding in the summer of 1997. If accepted, the programme will run for four years.

ACTION POINT

This is the first time the cavity spot literature has been gathered together and its contents reviewed. It will be some years before it is done again. Take the trouble to read it, the key to reducing disease levels may be contained within it.

1. INTRODUCTION

At the point of writing, cavity spot of carrots has been recognised and studied for 36 years. For the first 22 years the cause of cavity spot was not known, and workers from a wide range of disciplines attempted to demonstrate causes most of which were thought not to involve a fungal pathogen. As late as 1983, UK research workers were able to present at a grower meeting three different potential causes of cavity spot, one nutritional, one bacterial and one associated with insect damage, although at the time it was known that researchers in Norway had shown control with different Oomycete fungicides. By the time Lyshol and co-workers published their findings in Plant Pathology it was well known that *Pythium violae* could cause cavity spot lesions, and could be re-isolated from those lesions, also that the disease could effectively be controlled by metalaxyl. The world literature on *P. violae* at that time was five papers (Plaats-Niterink, 1981), with one dating back to its isolation from *Viola* in 1939 (Chesters & Hickman, 1939). It is now apparent, using the carrot crop as bait, that the fungus can be isolated from many fields in many countries. A major reason for the lack of knowledge on the fungus is its slow growth rate which virtually precludes it from isolation by conventional soil-based assays.

Changes in specifications for carrots by retailers have increased the importance of the disease, and worldwide there are now many groups working specifically on cavity spot. *Pythium sulcatum* and *P. coloratum* have also been identified as major causal agents, with other species of *Pythium* being shown to be associated with lesions. Most other potential causes have been discarded, and to date no group has suggested that there may be cavity spot caused by fungi outside the genus *Pythium*. A positive element in this scenario is that growers now know what to target with fungicide, but there is effectively only one very good fungicide. Early ideas on the benefit to be gained from crop rotation now have a logical basis, and particularly in the UK, growers attempt to use land which has not had carrot crops for five or more years. It might be expected that the more carrot crops a field has carried, the higher will be the disease risk. This does not appear always to be the case, and it is now obvious that we must know far more about the biology of causal *Pythium* species. For the future, there is a worry about the length of time metalaxyl will remain effective, and available. The rate of metalaxyl required for disease control is high by modern standards, and retailers increasingly seek crops which have no, or few pesticides applied. Premium payments are offered to persuade growers down this pathway, so it is essential that other ways of reducing the impact of the disease are explored. There are few candidate methods in crop husbandry. Knowledge of crops which cause build-up of cavity spot pathogens should help to avoid cropping fields with high levels of inoculum. A serological diagnostic test is now available to give a pre-cropping risk assessment. Management of soil pH and water relations are known to be beneficial. With the benefit of recent information on the enzyme systems involved in the host/pathogen interaction it is likely that improved field resistance to the disease will become commercially available. Inevitably, growers will need to consider some or all of these factors before selecting fields for cropping with carrots.

This review summarises work since the first paper on cavity spot to the present day.

2. GEOGRAPHICAL DISTRIBUTION OF CAVITY SPOT

Cavity spot of carrots is a major disease of carrots worldwide. It was first described by Guba, Young and Ui (1961) on carrot and parsnip roots growing in Massachusetts, USA. It has since been found in the UK (Perry, 1967; Baker, 1972), Denmark (Jensen, 1973), Norway (Arsvoll, 1980a,b), Israel (Jacobsohn, Zutra, Dan & Kelman, 1984; Soroker, Bashan & Okon, 1984), France (Montfort & Rouxel, 1988), the Netherlands (Wagenvoort, Blok, Mombarg & Veldhuizen, 1989), California, USA (Guerard, 1988), Belgium and Spain (White, 1991). The disease has been reported from South-Australia (Walker, 1988) and Western Australia (Erceg, 1993; Galati & McKay, 1993), and in Canada from Ontario (McDonald, Knibbe & Edgington, 1987) and British Columbia (Punja, 1990). A similar disease, called brown-blotted root rot of carrots, has been reported from Japan (Nagai, Fukami, Murata & Watanabe, 1986).

In the UK symptoms similar to cavity spot were noted on carrots growing in peat and mineral soils in Eastern England from 1960 onwards (Baker, 1972). The symptoms were similar to those described as 'pit' or 'watermark' in the East-Midlands as early as 1933 (Anon., 1943). A *Pythium* sp. was recovered from these watersoaked lesions, but attempts to reproduce similar symptoms by inoculating healthy carrots failed (Anon., 1943). A species of *Pythium* was also found in affected tissue in 1961 (Baker, 1972).

Cavity spot disease was briefly described in the UK by Perry (1967) and since then it has been reported from all the main carrot-growing areas: Scotland (Perry & Rubens, 1967; Perry & Harrison, 1977; Rubens & Halford, 1983), Yorkshire (Tyler, 1971), East-England (Baker, 1972), Lincolnshire (Green & Makin, 1985) and Lancashire (Gladders & McPherson, 1986).

3. HISTORICAL PERSPECTIVE

In the first report of cavity spot on carrots and parsnips, Guba *et al.* (1961) failed to isolate a causal organism and concluded that the disease was a physiological disorder. Since then numerous hypotheses have been suggested concerning the cause of cavity spot. Initially, cavity spot was reported to be a symptom of calcium deficiency which could be induced by high levels of potassium (Maynard, Gersten, Vlach & Vernell, 1961; Maynard Gersten, Young & Vernell, 1963). However, further trials failed to relate calcium or potassium concentrations in the soil or calcium and potassium ratios to cavity spot (Perry & Harrison, 1979a; Scaife, Turner, Hunt & Barnes, 1981; Soroker *et al.* 1984; Vivoda, Davis, Nuñez & Guerard, 1991).

Research was also carried out on soil ammonia levels. DeKock, Hall & Inkson (1981) suggested that in waterlogged conditions the ammonium-form of nitrogen antagonised calcium uptake, which then led to cavity spot development. Scaife, Burton & Turner (1980a,b) found a significant positive correlation between cavity spot incidence of field-grown carrots and soil ammonium levels. However, further research led to a conclusion that the correlation between cavity spot and soil ammonium was not causal (Scaife *et al.*, 1981). Goh & Ali (1983) reported that high soil ammonium levels were conducive to cavity spot development.

Another series of studies implicated waterlogging and poor aeration of soils as factors in the aetiology of cavity spot. Guba *et al.* (1961) first noted that the incidence of cavity spot was greatest in wet seasons. Perry & Harrison (1977, 1979b) observed that cavity spot occured in poorly drained, compacted soil especially after heavy rainfall. They concluded that this could lead to anoxic conditions in which pectolytic anaerobic bacteria of the genus *Clostridium* could induce cavity spot symptoms. However, they were only able to isolate the bacteria from 22% of the cavity spot lesions of field-grown carrots. Later research suggested that poor soil aeration (Perry, 1983; Rubens & Halford, 1983; Jacobsohn *et al.*, 1984) or waterlogging resulting in reduced soil aeration (DeKock *et al.*, 1981; Goh & Ali, 1983) was a possible causative factor of cavity spot.

Studies carried out in Israel were unable to confirm the role of anaerobic bacteria in cavity spot development (Finkelstein, Bashan, Okon & Yaakobi, 1983; Soroker *et al.*, 1984). It was suggested that cavity spot was caused by environmental stress, which resulted from the combination of temperatures higher than 28°C and short periods of flooding.

There were also reports that ethylene or aliphatic acids could influence cavity spot development. In a glasshouse experiment the injection of ethrel to waterlogged pots increased the number of cavity spot lesions on carrots (Collier & Huntington, 1979). It was suggested that in anaerobic conditions in the soil, ethylene production increased and stimulated the synthesis of phenolics in carrot roots, which lead to formation of cavities. Perry (1983) suggested that in anaerobic conditions, exudation of carbohydrates from roots increased, resulting in increased anaerobic activity and formation of organic acids including aliphatic acids. *In vitro* tests showed that aliphatic acids could produce lesions similar to cavity spot when applied to carrot roots (Perry, 1982).

Larvae of the fungus gnat (*Bradysia impatiens* (Joh.)) were found in cavities of glasshouse and field-grown carrots. As an application of the systemic insecticide aldicarb controlled cavity spot, fungus gnats were implicated as the causal agent of cavity spot (Hafidh & Kelly, 1982).

The first attempts to isolate a causal organism from cavity spot lesions failed (Guba *et al.*, 1961; Perry & Horgan, 1983; Lyshol, Semb & Taksdal, 1984). However, the most significant discovery in the search for a causal agent of cavity spot was made by Lyshol *et al.* (1984), who found that disease could be reduced in glasshouse tests by the fungicides metalaxyl, fosetyl-Al and propamocarb which control Oomycetous fungi. The results for metalaxyl (White, 1984) and propamocarb (Green & Makin, 1985) were confirmed in pot experiments. Metalaxyl was subsequently shown to reduce cavity spot of field-grown carrots (Gladders & Crompton, 1984; Perry & Groom, 1984; Wheatley, Hardman & Edmonds, 1984a,b). Soon after this discovery *Pythium* spp. was identified as the causal agent of cavity spot (Groom & Perry, 1985a; White, 1986).

4. CAUSAL AGENTS OF CAVITY SPOT

The identification of the *Pythium* species implicated in causing cavity spot is important as they have different sensitivities to metalaxyl (White, Stanghellini & Ayoubi, 1988). Metalaxyl is the only active ingredient currently approved for control of cavity spot in the UK and some other countries. Several *Pythium* spp. have been reported as causal agents of cavity spot in different countries, however, the slow-growing *Pythium* spp., *P. violae* Chesters and Hickman and *P. sulcatum* Pratt & Mitchell are the species most often associated with cavity spot of carrots (Table 1). Before being implicated as a causal agent of cavity spot, *P. violae* had been isolated from *Viola* species in the UK (Chesters & Hickman, 1939, 1944), conifer seedlings in South-Australia (Vaartaja, 1967), hyacinths (Saaltink, 1969) and *Scilla* (Plaats-Niterink, 1975) in the Netherlands, feeder rootlets of alfalfa in California (Hancock, 1985) and roots of wheat and rye-grass in Western Australia (Dewan & Sivasithamparam, 1988). In all cases it was shown to be pathogenic to these plants. *P. sulcatum* had been shown to be the causal agent of the carrot disease, 'brown root', 'rusty root' or '*Pythium* root dieback' occurring on carrots grown in organic soils of the United States (Pratt & Mitchell, 1973; Howard, Pratt & Williams, 1978) and Canada (Barr & Kemp, 1975; Kalu, Sutton & Vaartaja, 1976; Wisbey, Copeman & Black, 1977).

In the UK the main causal agents of cavity spot are considered to be *P. violae* (Groom and Perry, 1985a,b; White, 1986) and *P. sulcatum* (White, 1986; 1988). Some of the fast-growing *Pythium* spp. such as *P. sylvaticum* Campbell & Hendrix, *P. intermedium* de Bary, *P. ultimum* Trow, *P. irregulare* Buisman, *P. aphanidermatum* (Edson) Fitzp., were also able to produce lesions on carrots (White, 1986; White, Dowker & Crowther, 1987), but they were not regarded as primary pathogens (Lyons & White, 1992).

P. violae (Montfort & Rouxel, 1988; Breton & Rouxel, 1993; Guerin, Briard & Rouxel, 1994) and *P. sulcatum* (Breton & Rouxel, 1993; Guerin *et al.*, 1994) are also regarded as the main pathogens associated with cavity spot in France. *P. violae* and *P. ultimum* were reported to be the major species involved in the disease in California, USA (Vivoda *et al.*, 1991), whereas *P. irregulare* (Shlevin, Ben-Nun, Tzror, Nachmias & Ohali, 1987) and *P. violae* (White, Wakeham & Shlevin, 1993) were shown to cause the disease in Israel. *P. sulcatum* was found to be the causal agent of brown-blotted root rot, a disease similar to cavity spot, in Japan (Watanabe, Nagai & Fukami, 1986). It was also associated with cavity spot in the Netherlands (Wagenvoort *et al.*, 1989). *P. coloratum* Vaartaja and *P. sulcatum* were implicated as causal agents of cavity spot in Western Australia (El-Tarabily, Hardy & Sivasithamparam, 1996a). Of these, the former was considered to be more important.

McDonald (1994) reported that *P. violae*, *P. ultimum* and *P. irregulare* caused cavity spot in Ontario, Canada, while Benard & Punja (1995) associated eight *Pythium* species with the disease in British Columbia, Canada. Of these species *P. violae* and *P. sulcatum* were regarded as the most important, *P. irregulare* and *P. ultimum* were considered less important and *P. sylvaticum*, *P. acanthicum* Drechsler, *P. paroecandrum* Drechsler and *P. mamillatum* Meurs were stated to be non-pathogenic. From our observations, it would appear likely that workers have not given sufficient weight to age and state of decay of the lesions from which

Table 1. Association of different *Pythium* species with cavity spot of carrots in different countries.

	Pythium species implicated in causing cavity spot					
Country	*Pv*	*Ps*	*Pc*	*Pu*	*Pi*	Source
Australia		+	+			El-Tarabily *et al.*(1996a)
Belgium	+					White (1991)
Canada	+	+		+	+	McDonald (1994) Benard & Punja (1995)
Denmark	+	+				White (1991)
France	+	+				Montfort & Rouxel (1988) Breton & Rouxel (1993) Guerin *et al.* (1994)
Holland	+	+				Wagenvoort *et al.* (1989) White (1991)
Israel	+				+	Shlevin *et al.* (1987) White *et al.* (1993)
Japan		+				Watanabe *et al.* (1986)
Spain	+					White (1991)
UK	+	+				Groom & Perry (1985a,b) White (1986,1988)
US	+			+		Vivoda *et al.* (1991)

Pv = P. violae, Ps = P. sulcatum, Pc = P. coloratum, Pu = P. ultimum, Pi = P. irregulare

they isolate. When lesions are first formed, it is common in the UK to isolate mono-species (Appendix 1), either *P. violae* or *P. sulcatum*, with few isolates of other species. In older cavities it is often the case that the first fungi to grow out are fast-growing species such as *P. intermedium*, *P. irregulare*, and *P. sylvaticum*, commonly found on asymptomatic periderm (White, 1988). After a few days, the slow-growing species may be seen growing from the tissue. It is likely that the fast-growing species are in the lesions only as secondary invaders taking advantage of what is basically high moisture content dead organic matter.

There has in the past been some confusion as to the identification and recognition of the symptoms of cavity spot. Symptoms on carrots caused by *Rhizoctonia solani* Kuhn were given the name cavity spot by Mildenhall & Williams (1970). In hot, dry periods in the UK the present authors have observed sunken lesions associated with *R. solani*. Characteristically they were deeply sunken and did not discolour in the way that cavity spot lesions do. It is assumed

that sinking of tissue following infection is a normal reaction of carrots, and that other interactions between host and pathogen define the final nature of lesions.

Different soil types, climatic conditions and soil microbial populations may be reasons for the variation between countries in *Pythium* species reported as causal agents of cavity spot. Other reasons could be different isolation media or techniques used such as incubation temperature, which may favour isolation of some species and at the same time exclude some others.

5. SYMPTOMS AND INFECTION PROCESS

Cavity spot disease is characterised by the appearance of sunken, elliptical lesions that are initially pale olive in colour, with intact periderm (Appendix 2). The lesions do not extend more than 10-12 layers of cells deep. They darken with time, the periderm ruptures and the lesions extend laterally and inwards (Appendix 3). Reports on the size and frequency of lesions vary. Guba *et al.* (1961) described the lesions as 3-4 mm deep with openings of 0.2-0.5 x 1.5-4 mm in diameter. He reported that some roots showed an abundance of lesions while others only had few or were free of lesions and that the lesions were usually more numerous on the upper than the lower part of the carrot. Perry & Harrison (1979a) noted that lesions were initially 2-15 mm long, but could extend up to 40 mm long and 7 mm deep radially. Nagai *et al.* (1986) reported different types of lesions in carrots grown in winter and summer. Lesions in summer-grown carrots were circular or elliptical, 3-5 mm in diameter, sometimes extending into irregular soft-rotted lesions over 3 cm diam. Those in winter-grown carrots were slightly sunken, small circular spots 2-3 mm in diameter and occasionally cracked vertically at the centre. More lesions were formed on the upper and middle than the bottom part of taproots.

Lesion formation was described by Perry & Harrison (1979a). The outer layer of cells of the secondary phloem collapsed. This effect spread to neighbouring cells until the periderm and pericycle cells disintegrated. A layer of wound periderm formed beneath the lesion. Lignin and suberin were present in the cell walls of the periderm and polyphenols were detected in healthy tissue surrounding the lesions.

Benard & Punja (1992, 1995) demonstrated that several *Pythium* spp. isolated from cavity spot lesions secreted pectolytic enzymes and often highly pathogenic isolates like *P. violae* and *P. sulcatum* also exhibited high enzyme activity.

The structural aspects of cavity spot pathogenesis were investigated by Zamski & Peretz (1995). Their findings agreed with those of White (1986) and Guerin *et al.* (1994) who associated lesions on carrot roots with slow-growing *Pythium* species; fast-growing species were associated with asymptomatic periderm. White (1986) demonstrated that some of the fast-growing species of *Pythium* were also able to cause cavities on carrots, but he did not consider them to be primary pathogens. Guerin *et al.* (1994) found that the isolates of various *Pythium* spp. which they tested induced lesions on carrot taproots *in vitro*, but the slow-growing isolates were highly aggressive, whereas the fast growing isolates were less so. Zamski & Peretz (1995) found that only the slow-growing *Pythium* spp. such as *P. violae* and *P. sulcatum* were able to cause cavity spot lesions. They suggested that the slow-growing species were able to penetrate the plant tissue and grow for several (3-4) days before the plant cells recognised the infection, by which time the hyphae had already established within the tissue. Host cells that were located several layers deep in the tissue died and produced the lesion. It was assumed that fast-growing species either provoked a hypersensitive reaction at the root surface, so lesions were not formed, or they lacked the ability to induce a response.

Zamski & Peretz (1995) suggested that the slow-growing *Pythium* spp. penetrated the cell walls and grew for several days during which time small amounts of wall-degrading enzymes

were secreted. Fragments of decomposed wall components decreased the solute and water potentials in the apoplast leading to water movement from the symplast into the apoplast. The turgor pressure dissipated gradually and some cells shrank and died. The living cells adjacent to the infection site secreted lignin and other phenols that halted the spread of the invader. Later, Zamski & Peretz (1996) demonstrated that *P. violae* secreted a wide spectrum of enzymes, such as cellulase, polygalacturonase, pectin lyase, pectate lyase and pectin methylesterase, that degraded the host wall components. The activity of cellulase and polygalacturonase was highest on the first day and the activity of the other enzymes was highest at 14-30 days post-inoculation. This pattern of activity enabled the penetration of the fungus through the walls of the host cells and the establishment of the hyphae. Several pathogen-induced plant enzymes such as peroxidase, chitinase, glucanase and polyphenol oxidase were produced in the infected tissue.

6. MANAGEMENT OF CAVITY SPOT

6.1 Fungicides

Metalaxyl

The work in Norway by Lyshol *et al.* (1984) showed that the fungicides metalaxyl, fosetyl-Al and propamocarb that selectively control fungi of the class Oomycetes reduced cavity spot. This finding was not only an important step to the implication of *Pythium* spp. as the causal agent of cavity spot (Groom & Perry, 1985a; White, 1986), but also a starting point for further research on the potential of metalaxyl and other fungicides for control of cavity spot. Metalaxyl is now used routinely in several countries to control cavity spot.

Lyshol *et al.* (1984) reported that metalaxyl sprayed after sowing reduced the incidence of cavity spot in field and pot experiments. In a field experiment on sandy soil, metalaxyl at a rate of 2.0 kg a.i./ha reduced cavity spot from 46% to 4%. Metalaxyl as a seed dressing (1.4 kg a.i./ha) was not as effective as the spray. These results were confirmed in a pot experiment by White (1984) who showed that the incidence of cavity spot was reduced from 42% to 3% by the combination of a metalaxyl seed treatment and a drench. In another experiment, metalaxyl applied in a fluid-sowing gel (0.2 kg a.i./ha) virtually eliminated cavity spot, although it was probably phytotoxic and reduced plant stand by 28% (Wheatley *et al.*, 1984a,b). Perry & Groom (1984) also reported a significant reduction in the incidence of cavity spot in the field following two spray applications of metalaxyl (2.5 kg a.i./ha).

Other workers have shown that metalaxyl reduced cavity spot when applied as a drench (Davis, Liddell, Guerard, Nuñez & Vivoda, 1988; McDonald & Edgington, 1988, 1989; Davis, Nuñez, Guerard & Vivoda, 1991) or in a granular formulation (Walker, 1988, 1991; McDonald & Edgington, 1989).

The efficacy of metalaxyl seed treatments in controlling cavity spot has been variable. Rates of 1.4 (Lyshol *et al.*, 1984; Gladders & Crompton, 1984), 1.5, 3 and 6 g a.i./kg seed (Walker, 1991) had no effect on cavity spot. White (1984, 1986) using 1.4 g a.i./kg seed found a reduction in the incidence of cavity spot, but it was not as great as when the seed treatment was combined with a metalaxyl soil drench. In a pot experiment using fluidized-bed film-coating of seed, a single layer of metalaxyl (10 g a.i./kg seed) was as effective in the control of cavity spot in infested soil as a commercial metalaxyl drench treatment (1.2 kg a.i./ha) (Petch, Maude & White, 1991). Metalaxyl seed dressings gave inconsistent results in three years of trials in Canada (3.5 and 17.5 g a.i./kg seed) (McDonald, 1994) and in the UK (rates thought to be 15-30 g a.i./kg seed) (McPherson, 1995).

The timing of metalaxyl treatment has been shown to be important. Early season application was generally found to be most effective. Applications of metalaxyl with mancozeb from sowing through to four weeks post-emergence gave best control in trials on mineral and organic soils in all the main carrot producting regions of England (Gladders & McPherson, 1986). Similar results were obtained in Israel using metalaxyl alone (Shlevin *et al.*, 1987). In Canada, McDonald (1994) found metalaxyl (+ mancozeb) applications to be most effective

when applied up to 6 weeks after sowing, whereas in South Australia applications of metalaxyl 4-14 weeks after sowing were effective (Walker, 1991). In California, Davis *et al.* (1991) found that metalaxyl applications 40-60 days after sowing were more effective than a pre-sowing application. They did not test earlier post-sowing applications. However, in British trials, applications of metalaxyl with mancozeb at 8 and 16 weeks post-emergence were less effective than earlier applications (Gladders & McPherson, 1986). In Western Australia when applied 6-17 weeks after sowing metalaxyl failed to control cavity spot in 5 out of 6 experiments (Galati & McKay, 1996).

No clear benefit from split applications of metalaxyl throughout the growing season has been demonstrated. In California multiple, dilute applications throughout the growing season were not more effective than a single drench application 40-60 days after sowing. They were more effective, however, than a single pre-sowing application at a comparable rate (Davis *et al.*, 1991). Multiple applications of metalaxyl with mancozeb (5 x 0.25 kg a.i./ha) in UK trials were not more effective than a single application (1.25 kg a.i./ha) four weeks post-emergence (Gladders & McPherson, 1986). Extra sprays ten weeks after sowing did not improve control of cavity spot (Gladders & McPherson, 1986). In South Australia a single application of metalaxyl at 1 kg a.i./ha three weeks after sowing was as effective as two split doses (1 kg a.i./ha/occasion) applied 9-12 weeks apart (Walker, 1988).

Gladders & McPherson (1986) and McPherson (1995) compared the efficacy of metalaxyl with the combination of metalaxyl and mancozeb (Fubol 58WP) for the control of cavity spot, but did not find any improvement with the latter. It was also observed that in *in vitro* tests, *P. violae* was not very sensitive to mancozeb (White, Wakeham & Petch, 1992); mancozeb had an ED_{50} value of 17.5 µg/ml for *P. violae* (White *et al.*, 1992), whereas that of metalaxyl was 0.4 µg/ml (White *et al.*, 1988).

The rates of metalaxyl that were effective in controlling cavity spot varied from region to region. In the UK, the metalaxyl rates of 0.6 and 1.2 kg a.i./ha applied as sprays, reduced the incidence of cavity spot on mineral and organic soils (Gladders & McPherson, 1986; White, 1988; Sweet, Beale & Wright, 1989). In South Australia, metalaxyl rates of 0.43-2.14 kg a.i./ha applied as granules consistently reduced the incidence of cavity spot on sandy loam (Walker, 1991). In California, two spraying regimes containing a total of 1.12 and 2.24 kg a.i./ha reduced cavity spot incidence on sandy loam (Davis *et al.*, 1991). In Canada metalaxyl was most effective when applied as a granular treatment at rates of 0.2-4.0 kg a.i./ha or as a drench at 0.5 or 2 kg a.i./ha (McDonald, 1994). Granular application of metalaxyl at 4 kg a.i./ha was phytotoxic.

In some trials, metalaxyl at half of the recommended rate gave equal control to that of the full rate application. Gladders & McPherson (1986) reported that the high rates of metalaxyl (1.2 kg a.i./ha) gave the best control of cavity spot in their trials on mineral and organic soils, but half rates (0.6 kg a.i./ha) were effective at some sites. Similar results were obtained by Davies & Hembry (1993) who tested six carrot cultivars with different susceptibilities to cavity spot and found that all cultivars performed similarly with both half and full rate fungicide treatment. Sweet *et al.* (1989) and Beale & Sweet (1990) observed no differences in the efficacy of different doses of metalaxyl (0.6 and 1.2 kg a.i./ha). However, some variation in the responses of different varieties was seen; metalaxyl generally reduced the incidence of cavity spot more in susceptible than resistant varieties. McDonald (1994) also

found that there was an interaction between cultivar and metalaxyl (+ mancozeb) treatment; the fungicide treatment was more effective on susceptible cultivars than on a resistant cultivar.

There have been reports of metalaxyl failing to control cavity spot on carrots. Gladders & McPherson (1986) reported poor control of cavity spot by metalaxyl (+ mancozeb) in about 10% of their trials. White (1988) found that metalaxyl (+ mancozeb) was ineffective in one of the fields where *P. sulcatum* was identified as the primary pathogen involved in cavity spot development. In trials carried out over a three year period, McPherson (1995) found that disease control with metalaxyl (+ mancozeb) was only moderate at best and the efficacy varied from trial to trial. He estimated that the efficacy of metalaxyl has declined from nearly 100% to 50-75% control since early experiments carried out about ten years ago. Every year in the UK there are a small number of instances where metalaxyl fungicide applied at the full rate in accordance with the label recommendation appears to have failed to give disease control (White, unpublished information). In such cases, the causal organism has generally been shown to be *P. violae* which is highly sensitive to metalaxyl. Without further information it is not possible to fully account for such cases, but the possiblility of very high inoculum level effectively overcoming the fungicide must be considered. Because we still do not have an accurate quantitative measure of inoculum level as measured by live fungal material, this must remain a matter of conjecture.

In addition to the possibility that different inoculum levels may affect the success of metalaxyl, differences in efficacy may be due to the presence of different *Pythium* spp. that have different sensitivities to metalaxyl. White *et al.* (1988) found that *P. sulcatum* was less sensitive to metalaxyl than *P. violae*. One reason for the failure of metalaxyl to control cavity spot is late application (see page 10). Different levels of resistance in the cultivars used in the trials can also mask the efficacy of metalaxyl. Differences in the half-life or mobility of metalaxyl in organic and mineral soils could also contribute to differences in efficacy. Sharom & Edgington (1982) reported that in sandy loam the half-life of metalaxyl was shorter, and it was leached more rapidly than in muck soil. Subsequently, McDonald (1994) suggested that split applications of metalaxyl could be more effective on sandy soils because the combination of a shorter half-life and leaching or irrigation may reduce the length of time that metalaxyl remains effective in the root zone. However, experiments on split applications (see page 11) suggest that this would not be the case.

The reduced efficacy of metalaxyl could also be due to the development of resistance to the fungicide by the *Pythium* spp. responsible for causing cavity spot, or enhanced biodegradation of the fungicide with repeated use in fields where cavity spot frequently occurs. The failure of metalaxyl to control *P. aphanidermatum* has been documented in North America since 1983 (Sanders, 1984) and this was attributed to the development of metalaxyl resistance in the fungal populations.

Other fungicides

Relatively few fungicides other than metalaxyl have been studied for control of cavity spot of carrot. Fungicides such as propamocarb (Avigdori-Avidov, Jacobsohn, Zutra, Nachmias & Krikun, 1987; Gladders & Crompton, 1984; Gladders & McPherson, 1986; Green & Makin, 1985; Lyshol *et al.*, 1984), fosetyl-Al (Gladders & Crompton, 1984; Gladders & McPherson,

1986; Lyshol *et al.*, 1984; McDonald, 1994; Walker, 1988) and phosphorous acid (equivalent to phosphonate) (McDonald, 1994; Walker, 1988, 1991) have been tested against cavity spot, but the results were inconsistent. Fosetyl-Al is broken down in plant tissue to its active ingredient (a.i.) phosphorous acid (Cohen and Coffey, 1986).

Lyshol *et al.*(1984) found that fosetyl-Al reduced cavity spot incidence as effectively as metalaxyl when applied to carrots in pots. However, the effective rate of fosetyl-Al was greater (0.72 g a.i./pot) than that of metalaxyl (0.108 g a.i./pot). In contrast to this, Walker (1988) found that fosetyl-Al had no effect on the incidence of cavity spot when applied as a soil drench or foliar spray in two field trials. However, in one of the trials a foliar spray of phosphorous acid (4 g/l) applied to run-off at the five to six true leaf stage reduced the incidence of cavity spot. In another series of trials, Walker (1991) found that a foliar spray of phosphonate did not control cavity spot at rates of 10 or 16.5 kg a.i./ha when applied four, six, eight or ten weeks after sowing. However, an application of 25 kg a.i./ha twelve weeks after sowing was effective. Again the effective rate of phosphonate (25 kg a.i./ha) was much greater than that of metalaxyl (0.43-4.28 kg a.i./ha).

McDonald (1994) reported that fosetyl-Al and phosphonate were as effective as metalaxyl when applied as foliar sprays 12 or 17 weeks after sowing, but fosetyl-Al did not control cavity spot when applied as a drench. The effective rates of fosetyl-Al and phosphonate in these trials were much lower (1.6-4.8 kg a.i./ha) than those used by Lyshol *et al.* (1984) and Walker (1991). In field trials in Western Australia phosphonate (up to 14 kg a.i./ha) failed to reduce cavity spot in all experiments (Galati & McKay, 1996).

Walker (1991) suggested that the inconsistencies in results may be explained by the different half-lives of phosphonate in soil and in foliage, thus leading to different results from applications to soil and foliage, and differences in the fungal populations of soils between sites. McDonald (1994) suggested that fosetyl-Al could quickly leach out of soil and therefore, it may be more effective to apply fosetyl-Al later in the season when the incidence of infections is higher rather than early in the season. Fosetyl-Al is very soluble in water, although its half-life in soil is 16 weeks (Cohen and Coffey, 1986). Other possible explanations for inconsistent results could be different half-lives of phosphonate in different soil types, inoculum levels in soils and the type of experiment performed (experiments by Lyshol et al. (1984) were carried out in pots, whereas those by Walker (1988, 1991), McDonald (1994) and Galati & McKay (1996) were field experiments).

Propamocarb gave promising results in the control of cavity spot in pot tests (Lyshol *et al.*, 1984; Green and Makin, 1985), but the results from field experiments were not as convincing. Gladders and Crompton (1984) did not observe significant reduction in cavity spot incidence in their field trials. In field experiments reported by Gladders and McPherson (1986) the efficacy of propamocarb for control of cavity spot varied from failure to control in some experiments to efficacy equal to metalaxyl in others. In Israel propamocarb reduced the incidence of cavity spot in the field, but not as much as a combination of metalaxyl and mancozeb (Avigdori-Avidov *et al.*, 1987). In Western Australia propamocarb reduced the incidence of cavity spot in only one field experiment out of five carried out (Galati & McKay, 1996).

The efficacy of phenylamide fungicides for control of pathogens that cause cavity spot of carrots was tested *in vitro* (White & Wakeham, 1987). Only furalaxyl was as effective as metalaxyl against *P. violae*, but it had no effect on *P. sulcatum*. Benalaxyl, cyprofuram, ofurace and oxadixyl did not affect the *Pythium* spp. tested.

Soil sterilisation with methyl bromide (1 ml/kg soil) controlled cavity spot (White, 1986), whereas metham sodium (500, 700, 900 and 1000 l/ha) was ineffective (Jorgensen, 1976; Galati & McKay, 1996).

Nine potential fungicides were screened in laboratory tests and seven in glasshouse tests for control of cavity spot at Horticulture Research International in 1995-96 (White, Hiltunen & Petch, 1996). In laboratory tests only one fungicide (A9408B, a new metalaxyl formulation) gave a lower ED_{50} value for *P. violae* than metalaxyl. Two fungicides (ICI A5504 [a ß-methoxyacrylate derivative] and hymexazole) gave lower ED_{50} values with *P. sulcatum* than metalaxyl, but only ICI A5504 was regarded as worthy of further investigation. In a pot experiment, using field soil naturally infested by *P. violae* none of the new fungicides were as effective as metalaxyl. Many of them reduced the percentage of carrots with cavities, with the greatest reduction being achieved with A9408B. The efficacies of A9408B and ICI A5504 are currently being further evaluated in pot experiments. If any give promising results further trials will be carried out in the field.

6.2 Cultivar resistance

Varying levels of cultivar susceptibility to cavity spot were first reported by Guba *et al.* (1961). The National Institute of Agricultural Botany (NIAB) started assessing carrot cultivars for susceptibility to cavity spot in 1981 (Anon., 1986; Sweet & Beale, 1988). Varietal resistance was classified on a one to nine scale where a low rating denoted susceptibility. No immunity to cavity spot was found, but some varieties showed consistently lower levels of cavity spot than other varieties. Late harvested types generally had higher levels of cavity spot than earlier types and late harvested roots had higher levels of cavity spot than roots lifted earlier. NIAB now publishes a descriptive list of varieties of early and maincrop carrots that includes resistance ratings to cavity spot (Anon., 1994, 1995, 1996b) (see Appendix 4).

Soroker et al. (1984) did not find differences in susceptibility to cavity spot under different environmental stress situations between five carrot cultivars tested in pot trials in Israel. No useful genetic resistance was found during *in vitro* screening of 19 cultivars of five main groups (agronomic types) of carrots (White, Dowker & Crowther, 1987). However, significant differences in field tolerance to cavity spot between carrot varieties in field trials have been reported in Norway (Taksdal, 1990), Canada (McDonald & Sutton, 1992, 1993) and Western Australia (Galati and McKay, 1996).

Davies & Hembry (1993) carried out field trials on carrot cultivars treated with different rates of metalaxyl and mancozeb. There were differences between cultivars in their susceptibility to cavity spot; the cultivars Supreme and Nandor showed most resistance to cavity spot in the absence of fungicide, but all cultivars performed similarly in both half and full rate fungicide treatments.

Six carrot varieties with differing levels of putative cavity spot resistance were tested in the field with different rates of metalaxyl and thiram (Sweet *et al.*, 1989; Beale & Sweet, 1990). Fungicide-induced reduction of cavity spot was greater in the susceptible than in resistant varieties. Similar results were reported by McDonald (1994) who found that treatment with metalaxyl (+ mancozeb) was less effective on the resistant cultivar Six Pak than on more susceptible cultivars. It was suggested that under conditions of low to moderate disease pressure, cultivar resistance could substitute for fungicide use. Under high disease pressure use of susceptible cultivars should be avoided and resistant cultivars with fungicide should be used. McPherson (1995) compared two cultivars (Nanco and Nandor) with different susceptibilities to cavity spot in his three year trials. Disease levels in the cv. Nanco (NIAB rating 2) were consistently higher than those in cv. Nandor (NIAB rating 6). The reduction in disease incidence due to variety was greater than that achieved with the commercial application of metalaxyl with mancozeb on the susceptible cv. Nanco.

A test involving field or glasshouse-grown carrots inoculated by placing agar discs of *Pythium* spp. cultures onto the roots has been used for screening carrot cultivars for susceptibility to cavity spot. This method is quicker than field or glasshouse tests and can facilitate the testing of a large number of varieties at the same time. Sweet, Lake, Wright & Priestley (1986) reported a reasonable correlation between the disease levels found in the field and in *in vitro* tests. They suggested that the test procedure could give early information on resistance of varieties submitted for NIAB trials. However, White, Dowker, Crowther & Wakeham (1988) tested seven cultivars with known field tolerance to cavity spot in an *in vitro* test against three *Pythium* spp. (*P. violae*, *P. sulcatum*, *P. intermedium*), but did not find any relationship between the results from this test and those from field experiments. Vivoda *et al.* (1991) conducted a similar trial and concluded that inoculation of carrots with mycelial discs may not be a reliable technique for determining cultivar resistance. They observed that symptoms on carrots grown in potting mix artificially infested with *Pythium* spp. were typical of the cavity spot lesions seen in the field, whereas lesions on carrots inoculated with agar discs were untypical being superficial, discoloured areas with indistinct margins.

Benard & Punja (1992, 1995) evaluated 37 carrot cultivars for susceptibility to cavity spot in *in vitro* tests using *P. violae* inoculum. Differences between cultivars correlated well with results from field evaluations. The most resistant cultivars were Panther, Caropride, Fannia and Navajo. They regarded the average lesion diameter to be the most important criterion for determining whether a cultivar was resistant or susceptible, whereas percentage of infection was considered to be influenced by factors other than cultivar, such as moisture levels on the carrots. Galati & McKay (1996) also observed significant differences between varieties in their susceptibility to cavity spot in *in vitro* tests and found that the results correlated well with those from field experiments. In their tests, 95% of the agar discs of *Pythium* cultures produced lesions, but there were significant differences between the 12 varieties tested in average lesion diameter.

As with earlier comments on breakdown of control of cavity spot after the use of metalaxyl, although there should be benefit from the routine use of cultivars recognised to have field resistance, there are inevitably occasions when inoculum levels, or environmental conditions are so extreme that crops will express 100 % infection whatever the cultivar. Field resistance should therefore be regarded as just one component of several measures to be used in management of the disease.

New sources of resistance to cavity spot in cultivated carrot germplasm from HRI's Genetic Resources Unit have been sought (Smith & Crowther, 1991). Resistance to cavity spot was identified in purple coloured carrots from Turkey and pink carrots from Afghanistan (Anon., 1996a). The possibility of transferring these sources of resistance to varieties suitable for UK production has been investigated.

6.3 Biological control

Literature on the biological control of *Pythium* species is extensive, but few biological control agents have been commercialised and few are in the development and registration stage for use in the control of *Pythium* spp. (Whipps & Lumsden, 1991). McDonald & Edgington (1989) and McDonald (1994) investigated possibilities for biological control of cavity spot. They evaluated some growth-promoting rhizobacteria (*Pseudomonas fluorescens*, *P. putida* and *Serratia proteamaculans*) seed treatments for control of cavity spot. The efficacy of seed treatments varied between bacteria and cultivar; *P. putida* and *S. proteamaculans* were effective, but only on the susceptible cultivar Chanton (McDonald, 1994). Rhizobacteria did not reduce the numbers of *Pythium* spp. recovered from carrot roots. El-Tarabily, Hardy, Sivasithamparam and Kurtböke (1996b) found that many actinomycetes isolated from field soil were able to produce inhibitory compounds *in vitro* which were active against *P. coloratum*, a causal agent of cavity spot in Western Australia. These organisms could have potential for biological/integrated control.

In the first report on observations on *P. violae* (Chesters & Hickman, 1944) *Pythium oligandrum* Drechsler was found to be associated with the cavity spot pathogen. This was the first report of *P. oligandrum* isolation in the UK, and the authors did no more than describe the fungus. Drechsler (1946), however, described the mycoparasitic habit of *P. oligandrum* and speculated that when it is found in *Pythium* damaged plant tissue, it may be there in the role of parasite of the *Pythium* causing the damage. It is as a potent pathogen of *P. violae* (Appendix 5) that the fungus currently is of interest. The potential for the use of *P. oligandrum* to regulate cavity spot is therefore considered below.

6.4 Cultural methods

Soil moisture

Soil moisture is one of the most important factors which favours activity of *Pythium* spp. (Stanghellini, 1974). In the first report describing cavity spot of carrots, Guba *et al.* (1961) observed that high soil moisture was associated with cavity spot. Other observations from commercial fields indicated that cavity spot was common in wet seasons and in wet patches in fields (Norman, 1981; Long, 1985).

Cavity spot was more common on flat badly drained fields and those with poor soil structure than on other soil types. Records from a canning factory have also revealed an association between a high incidence of lesions with greater than average rainfall in July and August (Perry & Harrison, 1979b). In one field experiment an increase in soil moisture content from 10% to 23% induced by irrigation in combination with rolling, increased the incidence of

cavity spot from 1.7 to 29.9%. The cavity spot incidence in rolled plots without irrigation was 2.4%. In another experiment, irrigation during July or August but not October increased cavity spot from 3.0 to 14.4%. In pot experiments, the percentage of roots with cavity spot lesions was higher in treatments that were watered frequently or waterlogged as compared to carrots from treatments that were watered infrequently; sealing the soil surface with wax and flooding also induced cavity spot lesions (Perry & Harrison, 1979b). Similar results were obtained by Soroker *et al.* (1984) and Vivoda *et al.* (1991).

McDonald (1994) studied the effect of rainfall and soil moisture content on the incidence of cavity spot during six years of field trials. She found that increases in cavity spot incidence followed within 9-39 days of rainfall exceeding 20 mm when rainfall occured before mid-October and soil moisture content was below field capacity. The incubation period between rainfall events and an increase in incidence of cavity spot varied with cultivar and may have been affected by soil temperature. Early growing season rainfall (up to eight weeks after sowing) was not closely related to a high level of cavity spot on susceptible cultivars. The incidence of cavity spot was low during growing seasons when total rainfall was low (200-400 mm), higher in seasons with moderate to high rainfall (550 mm) and lower when rainfall was very high (720 mm).

As neither *P. violae* nor *P. sulcatum* appear to have an asexual reproductive stage in their life cycle (Lyons & White, 1992), they do not require saturated soil conditions to stimulate infection *via* zoospores. Stanghellini (1974) stated that high soil moisture content and accompanying poor soil aeration indirectly favour *Pythium* spp. by decreasing host vigour, increasing host exudation and by providing a suitable environment for the rapid diffusion and subsequent increased availability of host exudates necessary for germination and/or vegetative growth of dormant propagules; vegetative growth is apparently tolerant of, but not necessarily favoured by saturated soil conditions. Dormant resting structures of *Pythium* spp. are capable of rapid germination, once they have been stimulated by exogenous nutrients (Stanghellini, 1974). Carrot roots exude sugars and other nutrients into water (Perry, 1983; Soroker *et al.*, 1984). Perry (1983) found that the quantity of soluble carbohydrates exuded into water by carrot roots increased when the roots were in anaerobic conditions. Soroker et al. (1984) reported that the leakage of electrolytes was enhanced in flooded carrots at temperatures of 30°C and above. The leaking substances were mainly composed of sugars (70%), but there were also proteins, amino acids, lipids and minerals.

McDonald (1994) suggested that an exact determination of soil moisture content may not be necessary to predict cavity spot disease. A disease forecasting system based on rainfall rather than soil moisture measurement would be cheaper and easier to implement. The moisture content of muck soil increased in conjunction with rainfall and decreased during periods of no rain (McDonald, 1994). However, details of the soil moisture prior to rainfall along with the rainfall data would be necessary.

Soil temperature

Temperature does not appear to be as important as soil moisture in cavity spot development.

Van der Plaats-Niterink (1981) reported that the optimum temperature for growth of *P. violae in vitro* was 25°C. However, White *et al.* (1993) compared *P. violae* isolates from the UK, France and Israel and found that they had similar temperature optima for growth *in vitro* (20°C or less). Schrandt, Davis & Nunez (1994) also found 20°C to be the optimum temperature for growth of *P. violae in vitro*. These results were supported by those from pot and laboratory experiments (Vivoda *et al.*, 1991; Montfort & Rouxel, 1988). Carrots that were transplanted into growing media artificially infested with *P. violae* or *P. ultimum* developed more lesions when incubated at 15°C than at 20° or 25°C (Vivoda *et al.*, 1991). Similar results were reported by Montfort & Rouxel (1988) who found that the optimum temperature for lesion expansion on mature carrots inoculated with mycelial plugs of *P. violae* was 15°C.

Many field observations suggest that cavity spot is favoured by cool soil temperatures (Guerard, 1988; Vivoda *et al.*, 1991; White *et al.*, 1993; McDonald, 1994). In the San Joaquin Valley of California, average soil temperatures were 15°C or below at 15 cm depth during November to March when cavity spot is most often observed (Vivoda *et al.*, 1991). In Israel, cavity spot is a problem in spring, autumn and winter, when mean temperatures at 10 cm depth are normally 20°C or below (White *et al.*, 1993). McDonald (1994) found that in Ontario, cavity spot developed over a range of temperatures (3-22°C). In the six to eight weeks after sowing, cavity spot incidence was higher at low soil temperatures (16-17.5°C) than at high soil temperatures (20-22°C). In conflict with these observations, Jacobsohn, Dan, Yaakobi & Sander (1973) reported severe cavity spot from Israel from irrigated fields during the hot season. The findings in another study in Israel that short periods of flooding and temperatures above 28°C caused cavity spot in controlled environment supported these field observations (Soroker *et al.*, 1984) and possibly indicate that seasonal fluctuations in cavity spot are more strongly associated with soil moisture than temperature.

Soil aeration

Poor soil aeration caused by poor soil structure, soil type or waterlogging has been associated with the development of cavity spot.

The observations described earlier that cavity spot was more common on flat imperfectly drained fields with poor soil structure than on other soil types and the fact that high soil bulk density was the only characteristic which was related to the disease in a survey of carrot crops (Perry & Harrison, 1979a) indicates that soil aeration could be important in the development of cavity spot. Cavities were induced in carrots on land where the disease was not normally found by trickle irrigating to field capacity for 14 days during the growing season. Perry & Harrison (1979b) concluded that poor aeration was a predisposing factor to lesion development. In a later experiment, Perry (1983) found that improving soil aeration by cultivating between rows and beds reduced the symptoms of cavity spot compared to rolled plots. There were no differences in cavity spot incidence between cultivated and ridged plots. Jacobsohn *et al.* (1984) found that cavity spot incidence was reduced from 26.7% to 16.5% (average from several years experiments) by growing carrots in ridges. However, they pointed

out that it was possible to accomodate only three ridges within the width occupied by a flat bed of four rows. Consequently, the marketable yield from ridges was 10-15% less than that from an equal area of flat beds.

Perry & Harrison (1977, 1979b) found anaerobic pectolytic bacteria of the genus *Clostridium* more frequently associated with cavity spot lesions than with healthy roots, and showed that the bacteria were pathogenic to roots in anaerobic conditions. However, Soroker *et al.* (1984) did not find an association between anaerobic bacteria and cavity spot.

Rubens & Halford (1983) reported that cultivating between rows during the growing season reduced the severity of cavity spot and rolling immediately after sowing caused a 40% increase in the incidence of the disease. To avoid poor soil aeration, they advised the farmers to avoid heavy rolling of the land before and after sowing, to cultivate in July or August, if possible and avoid harvesting when the soil was very wet.

Soil inoculum level

Oospores are important survival structures of *Pythium* spp. in soil and they are known to remain viable for over 12 years (Plaats-Niterink, 1981). *P. violae* readily forms oogonia and it is assumed that this stage is the means of survival and infection in soil (Phelps, White & Henn, 1991). Both *P. violae* and *P. sulcatum* appeared to lack an asexual reproductive stage in their life cycle (Lyons & White, 1992).

Quantification of the initial inoculum of the *Pythium* spp. that cause cavity spot and correlation between soil inoculum levels and disease severity are important, but have proved difficult. There is only one report of direct isolation of *P. violae* from soil and the frequency of isolation was low (Dick & Ali-Shtayeh, 1986). Isolations on soil dilution plates have been dominated by fast-growing species, which precluded the isolation of slow-growing species (Phelps *et al.*, 1991). El-Tarabily *et al.* (1996a) examined the symptoms and the severity of cavity spot resulting from varying inoculum levels of *P. sulcatum* and *P. coloratum* on glasshouse trials. *P.coloratum* produced few lesions at an inoculum density 0.1% (w/w, weight of millet seed based inoculum/weight of soil) and substantial and numerous lesions at 0.5% (w/w). *P. sulcatum* produced few and small lesions at densities of 0.8 and 1% (w/w), but none at 0.5% (w/w).

Studies of the frequency distribution of cavity spot symptoms in carrots suggested that there were low levels of randomly-distributed inoculum in the fields tested (Phelps et al., 1991).

As discussed later in Section 7, inoculum levels/potentials can be estimated by serological means. However, it has to be borne in mind that the actual severity of the disease, would also be affected by environmental factors such as soil moisture, temperature and atmosphere (Stanghellini, 1974), and agronomic practices like irrigation, carrot variety and use of fungicide (Petch & White, 1995).

Alternative hosts and crop rotation

Dormant resting structures of *Pythium* species formed during pathogenic and/or saprophytic colonisation of plant tissues have long been considered the primary sources of inoculum for succeeding crops, but the non-pathogenic colonisation of other crops and weeds may provide an alternate or initial source of inoculum (Stanghellini, 1974).

Apart from carrots, *P. violae* has been isolated from and shown to be pathogenic on violas, conifer seedlings, hyacinths, Scillas, alfalfa, wheat and rye-grass (Chesters & Hickman, 1944; Vaartaja, 1967; Saaltink, 1969; Plaats-Niterink, 1975; Hancock, 1985; Dewan & Sivasithamparam, 1988). In addition to these, *P. violae* was isolated from six symptomless hosts in a glasshouse experiment in California, i.e. cowpea, broccoli, celery, cucumber, sugarbeet and watermelon, and in a field experiment also from cauliflower (Schrandt *et al.* 1994). If such susceptible hosts occur in rotations with carrot, they may maintain or increase populations of *P. violae* in commercial fields.

Lyshol *et al.* (1984) reported that in fields with a history of frequent carrot cropping, there was a tendency for increased incidence of cavity spot. Intervals of 1-3 years between carrot crops did not reduce the incidence of the disease. Jacobsohn *et al.* (1984) did not find any differences in the cavity spot incidence associated with the following preceeding crops: wheat, potatoes, onions and cotton; the interval between carrot crops was not given. Long term crop rotation was considered by a number of authors to be necessary for affected fields to reduce the risk of cavity spot (Rubens & Halford, 1983; Lyshol *et al.*, 1984; Guerard, 1988).

In a survey of commercial carrot fields in Western Australia, Galati & McKay (1996) found that cavity spot was more severe with more intensive carrot cropping. Losses due to cavity spot were higher (34% of crops had losses of >10% marketable yield) for harvest intervals of less than 12 months whereas for harvest intervals of greater than 12 months losses were lower (only 10% of crops had losses of >10% marketable yield).

Pythium oligandrum

P. oligandrum has been known for many years to be mycoparasitic (Drechsler, 1946) and it has been shown to be pathogenic to *P. violae* (White *et al.*, 1992). It was found to be present in almost all fields in a survey where carrots were grown in the UK (White, 1993). In plate tests it overgrew cultures of *P. violae* and *P. sulcatum* killing the mycelium and preventing the formation of oogonia. However, *P. oligandrum* populations were reduced or eliminated in soil by applications of metalaxyl with mancozeb (White, 1991), and it was found to be sensitive to both metalaxyl and mancozeb *in vitro* (White *et al.*, 1992). The levels of *P. oligandrum* in fields varied depending upon crop rotations, but the information on previous cropping did not give a clear picture of which crops enhanced the fungus (White, 1992). It was suggested that this may be due to the numbers of crops in rotations which would have received sprays of metalaxyl with mancozeb.

Variation in the *P. oligandrum* populations in soil are likely to affect its ability to suppress the activity of *P. violae*. Martin & Hancock (1986) indicated that different soil populations of *P. oligandrum* had different abilities to suppress the activity of *P. ultimum* in crop residues.

Therefore, any reduction in the soil population of *P. oligandrum* as a result of fungicide usage could potentially deleteriously affect crop health (White *et al.*, 1992). Conversely, if it were possible to enhance the population of *P. oligandrum* in fields, natural control of cavity spot might be possible.

Soil nutrition

The first description of cavity spot (Guba *et al.*, 1961) suggested a link between the disease and low nutrient levels in soil. Since then research has concentrated on studying soil calcium and potassium levels and the effect of these on cavity spot. Maynard *et al.* (1961) reported a relationship between cavity spot and low levels of calcium in carrot roots and petioles and suggested that the disease was the result of potassium-induced calcium deficiency (Maynard *et al.*, 1963).

Excess potassium was linked with cavity spot also in other reports (Tyler, 1971; Perry, 1972; DeKock *et al.*, 1980; Jakobsen & Jorgensen, 1986). High levels of potassium in soil were assumed to lead to a build-up of potassium in the plants, which affected the uptake of calcium which in turn was thought to be a factor in cavity spot development (Jakobsen & Jorgensen, 1986). Roots and leaves of carrots with cavity spot had elevated K/Ca ratios (DeKock *et al.*, 1980) and it was concluded that this may have been induced by over fertilisation with potassium. Jakobsen & Jorgensen (1986) found that cavity spot incidence was highest when crops had been fertilised with high levels of potassium; increased levels of nitrogen also increased cavity spot. Wagenvoort, Babik & Findenegg (1985) reported that in hydroculture the highest incidence of cavity spot occurred at low concentrations of calcium in the nutrient solution and was associated with low calcium levels in leaves and roots.

There have been many reports that have not found an association between cavity spot, nutrients and various soil factors (Perry & Harrison, 1979a; Scaife *et al.*, 1981, 1983; Jacobsohn *et al.*, 1984; Soroker *et al.*, 1984; White, 1986; Vivoda *et al.*, 1991). No relationship was found between cavity spot and the concentrations of magnesium, manganese, copper, boron, calcium or potassium in the field soil or ratios of the latter two (Perry & Harrison, 1979a). Similarly, no association was found between cavity spot and K/Ca ratios in leaves, peel or core of carrots (Scaife *et al.*, 1981) or between the disease and nitrogen, potassium, phosphorous, calcium, magnesium or sodium content of carrot roots (White, 1986). The application of nutrient solutions containing nitrogen, potassium, phosphorous, calcium, magnesium or sodium did not affect the incidence of cavity spot and no differences were found in the nutrient element content of affected and symptomless carrot roots or foliage (Jacobsohn *et al.*, 1984). Soroker *et al.* (1984) reported that in Israel, carrots with cavity spot are found in fields where the soil has high levels of both available and unavailable calcium. However, it was possible that a temporary lack of available calcium occurred as a result of flooding which would have disturbed the balance of ion absorption from the soil solution by roots. Vivoda *et al.* (1991) did not find a correlation between cavity spot incidence and a number of soil factors including soil electrical conductivity, moisture holding capacity, organic matter, total and exchangeable calcium or particle size distribution.

In some reports high soil ammonium levels were associated with cavity spot of carrots (Scaife *et al.*, 1980a,b; DeKock *et al.*, 1981; Goh & Ali, 1983).

Soil pH

Reports on the effect of soil pH on cavity spot give conflicting evidence. It was first indicated that cavity spot was reduced when soil pH was lowered to below pH 6.6 (Perry & Harrison, 1979a). Later studies found that cavity spot incidence was lowest at pH's above 7.0 (Scaife, Turner, Barnes & Hunt, 1983; Perry & Groom, 1984) and 8.0 (White, 1988) and highest at pH's below 6.5 (Scaife *et al.*, 1983) and 5.5 (Perry & Groom, 1984). However, Soroker *et al.* (1984) and Jacobsohn *et al.* (1984) reported that cavity spot disease was found in carrots grown in highly calcareous soils (pH 7.8-8.3) in Israel. A survey of commercial carrot fields in California did not reveal a correlation between the incidence of cavity spot and pH in the range 5.7-7.7 (Vivoda *et al.*, 1991).

In Western Australia, a survey of carrot fields showed that the incidence of cavity spot was lower in soils where the pH was above 7 (Galati & McKay, 1996). These authors tested the efficacy of lime in controlling cavity spot in several field trials. In one of the trials it reduced the severity of cavity spot at a rate of 5 t/ha, but did not reduce the disease incidence. The pH was increased from 6.4 (untreated plot) to 7.3 by treating plots with lime. In another experiment, hydrated lime (3 and 12 t/ha) and lime sand (8, 16 and 32 t/ha) reduced the incidence and severity of cavity spot in three carrot crops sown over 18 months and increased the total and marketable yield of Nantes carrots. After 12 months, the pH of soil was increased from 5.9 (untreated plot) to 7.0 and 8.0 in plots treated with hydrated lime and 7.3, 7.5 and 7.6 in those treated with lime sand.

The mode of action of lime/calcium is not clear. In addition to the above studies, in which the effect of soil pH on disease severity was examined, there have been a number of studies on the effect of pH on the growth of *P. violae* and *P. sulcatum*. Schrandt *et al.* (1994) found that *P. violae* grew over the pH range of 5.5-8.0, but growth was decreased rapidly below pH 5. Galati & McKay (1996) observed that isolates of *Pythium* spp. responded differently to changes in pH. A *P. violae* isolate from the UK grew optimally at pH 5.8-7.8, whereas one from the USA grew best at pH 5.8-6.8. Isolates of *P. sulcatum* (from the UK and the Netherlands) and *Pythium* spp. from Western Australia grew optimally between pH 6.8 and 7.4.

In studies in Western Australia the application of lime (4 tn/ha) to a field soil (pH 6.9) used for commercial carrot production reduced the incidence of cavity spot compared to unlimed soil (pH 5.1) (El-Tarabily *et al.*, 1996b). In the limed soil, microbial activity as measured by the hydrolysis of fluorescein diacetate and arginine ammonification was increased. There was also an increase in the total numbers of colony forming units (cfu) of aerobic bacteria, fluorescent pseudomonads, Gram negative bacteria, actinomycetes and a decrease in the cfu of filamentous fungi and yeasts compared to unlimed soil. The numbers of actinomycetes antagonistic to *P. coloratum*, a causal agent of cavity spot, increased in soil amended with lime.

Plant age

As the time a crop is in the field increases, severity of cavity spot on carrots appears to increase (Maynard *et al.*, 1963; Montfort & Rouxel, 1988; Sweet *et al.*, 1989). Maynard *et al.* (1963) found that the number of lesions per root increased from 1.19 to 9.95 on field grown carrots during ten weeks from August to November. In commercial carrot fields in France, lesions were found on young carrots less than 5mm in diameter and the incidence of cavity spot increased progressively during the four month growing season (Montfort & Rouxel, 1988). In NIAB trials in the UK, cultivars often had more susceptible ratings when harvested late rather than if harvested early (Anon., 1986; Sweet *et al.*, 1986). Similar observations were made in commercial fields (Rubens & Halford, 1983; Long, 1985). Perry (1983) found that the percentage of roots with lesions was higher (37.4%) in late harvested carrots (November) than in carrots harvested earlier (October) (28.2%). In another study, cavity spot levels increased by up to 16.8% between October and January (Sweet *et al.*, 1989). However, no indication was found in field trials in Canada that the disease incidence was higher in old than young plants (McDonald, 1994). The number of lesions per carrot root was not assessed.

Carrots inoculated *in vitro* using discs of cultures of *Pythium* spp. as inoculum and carrots grown in pots, demonstrated differing susceptibility with age. Groom & Perry (1985b) found that the disease incidence increased with carrot age when field-grown carrots sown in May were inoculated *in vitro* using discs of *P. violae* cultures; lesions formed at less than 20% of the inoculation sites on samples collected before August, but in September this increased to 87% and remained between 67-95% on carrots from all successive harvests until December. They also found differences between cultivars during *in vitro* screening of glasshouse-grown carrot roots that were 18 and 23 weeks old. However, when the roots were 31 weeks old lesion incidence increased on those cultivars which had appeared to be resistant at 18 and 23 weeks. Transplanting glasshouse-grown carrots that were three, four or five months old into artificially infested soil also demonstrated that older carrots were more susceptible to infection by *P. violae* and *P. ultimum* (Vivoda *et al.*, 1991). The five month old roots had approximately twice as many lesions as the three or four month old roots. There were no significant differences in the numbers of carrots with lesions.

The increased level of disease with time could result from increased susceptibility as carrots mature, an accumulation of lesions over time or an expansion of lesions as the diameter of the root increases (Vivoda *et al.*, 1991). Another possible explanation is that the chance of infection increases as the carrot root surface increases with growth (Wagenvoort *et al.*, 1989).

Early harvesting and sampling of carrots has been recommended for growers. In the UK, the growers were advised to sample their crops in late September or early October, especially if there had been one or more periods of heavy rainfall in the preceding months. If lesions were present in any quantity, it was recommended that the roots should be lifted as soon as possible (Rubens & Halford, 1983). Galati & McKay (1995) advised Western Australian growers to harvest carrots as soon as they reached marketable size, and on sites with a history of cavity spot, to monitor disease development over the life of the crop.

Plant density

Some reports have indicated that high plant density may increase the incidence of cavity spot (Tyler, 1971; Perry, 1972; Norman 1981). Perry & Harrison (1979b) suggested that a high plant density may increase cavity spot by causing a localised depletion of oxygen in the soil, whereas White (1988) suggested that at the high plant densities there was a greater potential for plant-to-plant spread than at the low densities. However, in two recent studies no correlation was found between cavity spot incidence and plant densities of 57, 115 or 230 plants/m (Vivoda *et al.*, 1991) and 22-120 plants/m^2 (Galati & McKay, 1996).

7. DISEASE PREDICTION

Following the demonstration of control of cavity spot using metalaxyl, a large percentage of the UK carrot crop was treated prophylactically with 1.2 kg/ha of the fungicide in the product Fubol 58WP. The treatment was considered by growers to be expensive, and as it was known that relatively few fields in any one year would be seriously affected by cavity spot, for many fields it would be applied unnecesarily. Work was therefore initiated at HRI, Wellesbourne to predict the disease risk of fields to be cropped with carrot (White, Lyons & Petch, 1996). A serological method based on competition ELISA (enzyme-linked immunosorbent assay) for detecting *P. violae* and *P. sulcatum* in soil was developed (Petch & White, 1995; White *et al.*, 1996). Serological methods rely on the recognition of solid or soluble antigenic materials by antibodies raised against the organism and use of an enzymic labelling system. Competition ELISA can detect small antigens at far lower concentrations than the more conventional direct and indirect methods and increases the specificity of antisera (Kitagawa, Sakamoto, Furumi & Ogura, 1989). A competition ELISA process was developed with polyclonal antisera, using the supernatant from soil slurries as test material. It is likely that the antigen detected is a mixture of extracellular enzymes and cytosolic components of the fungi released by abrasion during shaking of the slurry. By comparing ELISA data from unconcentrated and freeze-dried samples an absorbance ratio was derived which over a number of years was related to disease development in the field. Of importance to the grower was the identification of high risk fields which could be avoided. At the other extreme, many fields gave ratios around 1.0, indicating lack of detection, and correlated with minimal disease development. For these the grower could reliably crop without the use of fungicide. Between the extremes, the relevant course of action was to treat the field with metalaxyl fungicide in accordance with the label instructions.

Success in the test relies on assaying soil in the winter before carrots are to be sown, also on the ability particularly of *P. violae* to grow vigorously at low temperature. To understand why the fungus should grow well in some fields and not others remains to be explained, but the role of previous crop and/or amounts of organic matter incorporated are likely to be important. Hancock (1985) observed that *P. violae* was the most common species isolated from alfalfa rootlets in California in early part of growing season.

The process went fully commercial in the UK in 1993 and was licensed for use in other countries in 1996.

8. RECOMMENDATIONS FOR FUTURE RESEARCH AND DEVELOPMENT

This review is the prelude to a concerted programme of work to be carried out over a period of four years with support from the HDC, MAFF, the seed and agrochemical and allied horticultural industries and commercial growers. The main objectives of the work fall into six areas which in the first instance require separate consideration, but which will be integrated in the course of the project:

1. Studies on the host range and biology of *P. violae*, integrating serological and molecular methods for detection and quantification.

2. Infection processes, fungal enzymes and pathogenesis related proteins.

3. Water management.

4. Soil pH optimisation.

5. Studies on the fate of metalaxyl.

6. Work on alternative fungicides to metalaxyl.

The programme has essentially been agreed by all parties, and will have the aim of producing grower advice to permit growing carrots to 'blueprint' standard with respect to minimising the risk of cavity spot.

9. GLOSSARY

a.i.	active ingredient
ED_{50} -value	the concentration of fungicide which reduces colony growth to half of that in the untreated control
ELISA	enzyme-linked immunosorbent assay; a serological method that relies on the recognition of solid or soluble antigen materials by antibodies raised against the organism and use of an enzymic labelling system
ppm	parts per million = µg/ml

Should you require clarification of botanical terms please contact the project leader.

10. REFERENCES

ANON., 1943. Diseases of crop plants. A ten years' review (1933-1942). *MAFF Bulletin No. 126.* 101pp.

ANON., 1986. NIAB cavity spot research pays off. *Grower, 1986,* November 29, 10.

ANON., 1994. Descriptive list of early and maincrop carrots grown in NIAB trials. *Vegetable Growers Leaflet, National Institute of Agricultural Botany No. 5, 1994,* 16pp.

ANON., 1995. Vegetable variety handbook. NIAB descriptive lists of vegetables 1995. *National Institute of Agricultural Botany 1995.*

ANON., 1996a. Asian carrots resistant to cavity spot. *Grower, 1996,* June 13, 11.

ANON., 1996b. Vegetable variety handbook. NIAB descriptive lists of vegetables 1996. *National Institute of Agricultural Botany 1996.* 139pp.

ARSVOLL, K., 1980a. Gropflekk i gulrot - problemet loyst. *Aktuelt fra Landbruksdepartementets opplysningstjeneste 1980, No. 2,* 65-69.

ARSVOLL, K., 1980b. Gropflekk i gulrot - problemet loyst. *Gartneryrket* **70**, 559-560.

AVIGDORI-AVIDOV, H., JACOBSOHN, R., ZUTRA, D. NACHMIAS, A. and KRIKUN, J., 1987. [Chemical control of cavity spot of carrots]. *Hassadeh* **67**, 1556-1559.

BAKER, J. J., 1972. Report on diseases of cultivated plants in England and Wales for the years 1957-1968. *Ministry of Agriculture, Fisheries and Food, Technical Bulletin 25,* 86, 236-237.

BARR, D. J. S. and KEMP, W. G., 1976. *Olpidium brassicae,* tobacco necrosis virus and *Pythium* species in relation to rusty root rot of carrots in Ontario and Quebec. *Canadian Plant Disease Survey* **55,** 77-82.

BEALE, R. E. and SWEET, J. B., 1990. Control of diseases in field vegetables using resistant cultivars and their integration with biological and chemical methods. *Monograph - British Crop Protection Council No. 45,* 131-136.

BENARD, D. and PUNJA, Z. K., 1992. Role of Pythium spp. in the developement of cavity spot on carrots in British Columbia. *Phytopathology* **82**, 1091. (Abstr.)

BENARD, D. and PUNJA, Z. K., 1995. Role of *Pythium* species in cavity spot developement on carrots in British Columbia. *Canadian Journal of Plant Pathology* **17**, 31-45.

BRETON, D. and ROUXEL, F., 1994. Donnees recentes sur le cavity-spot de la carotte en France. *Acta Horticulturae* **354**, 159-170.

CHESTERS, C. G. C. and HICKMAN, C. J., 1939. Preliminary report on stem and root rot of Viola and Pansy. *National Viola and Pansy Society Yearbook 1938*, 1-8.

CHESTERS, C. G. C. and HICKMAN, C. J., 1944. On *Pythium violae* n.sp. and *P. oligandrum* Drechsler from cultivated *Viola*. *Transactions of the British Mycological Society* **27**, 55-62.

COHEN, Y. and COFFEY, M. D., 1986. Systemic fungicides and the control of *Oomycetes*. *Annual Review of Phytopathology* **24**, 311-338.

COLLIER, G. F. and HUNTINGTON, V. C., 1979. Cavity spot in carrots. *National Vegetable Research Station Annual Report for 1978*, 109.

DAVIES, J. S. and HEMBRY, J. K., 1993. Novel production systems to minimise pesticide use in intensive field vegetable crops. Crop Protection in Northern Britain 1993: *Proceedings of a Conference, Dundee University, 23-25 March 1993*, 195-200.

DAVIS, R. M., LIDDELL, C. M., GUERARD, J. P., NUÑEZ, J. and VIVODA, E., 1988. *Pythium* diseases of carrots in California. *Phytopathology* **78**, 1578. (Abstr.)

DAVIS, R. M., NUÑEZ, J. J., GUERARD, J. P. and VIVODA, E., 1991. If registered, fungicide could reduce cavity spot of carrots. *Californian Agriculture* **45** (2), 29-30.

DEKOCK, P. C. HALL, A. and INKSON, R. H. E., 1981. Cavity spot of carrots. *Anales de Edafologia y Agrobiologia* **40**, 307-316.

DEWAN, M. M. and SIVASITHAMPARAM, K., 1988. *Pythium* spp in roots of wheat and rye-grass in Western Australia and their effect on root rot caused by *Gaeumannomyces graminis* var. *tritici*. *Soil Biology and Biochem*istry **20**, 801-808.

DICK, M. W. and ALI-SHTAYEH, M. S., 1986. Distribution and frequency of *Pythium* species in parkland and farmland soils. *Transactions of the British Mycological Society* **86**, 49-62.

DRECHSLER, C., 1946. Several species of *Pythium* peculiar in their sexual development. *Phytopathology* **36**, 781-864.

EL-TARABILY, K. A., HARDY, G. E. ST. J. and SIVASITHAMPARAM, K., 1996a. Association of *Pythium coloratum* and *Pythium sulcatum* with cavity spot disease in Western Australia. *Plant Pathology* **45**, 727-735.

EL-TARABILY, K. A., HARDY, G. E. ST. J., SIVASITHAMPARAM, K. and KURTBÖKE, I. D., 1996b. Microbiological differences between limed and unlimed soils and their relationship with cavity spot disease of carrots (*Daucus carota* L.) caused by *Pythium coloratum* in Western Australia. *Plant and Soil* **183**, 279-290.

ERCEG D., SIVASITHAMPARAM, K. and O'BRIEN, P., 1993. Identification of *Pythium* isolates associated with cavity spot disease of carrots in Western Australia. *Proceedings of the 6th International Congress of Plant Pathology, Montreal, Canada,* p. 149.(Abstr.)

FINKELSTEIN, E., BASHAN, Y., OKON, Y. and YAAKOBI, C., 1983. Induction of, and crop loss due to, cavity spot of carrots. *Phytoparasitica* **11**, 210-211.

GALATI, A. and MCKAY, A., 1993. The export carrot industry. *Western Australian Journal of Agriculture* **34**, 48-51.

GALATI, A. and MCKAY, A. G., 1995. Cavity spot disease of carrots. *Farmnote No. 74/95. Agriculture Western Australia.*

GALATI, A. and MCKAY, A. G., 1996. Carrot yield decline. *Final report for Horticultural Research and Development Corporation. Western Australia, 1996.* 268pp.

GLADDERS, P. and CROMPTON, J. G, 1984. Comparison of fungicides for control of cavity spot of carrots. *Tests of Agrochemicals and Cultivars 1984, No. 5 (Annals of Applied Biology* **104,** *Supplement)* 36-37.

GLADDERS, P. and MCPHERSON, G. M., 1986. Control of cavity spot in carrots with fungicides. *Aspects of Applied Biology* **12,** 223-233.

GOH, K. M. and ALI, N. S., 1983. Effects of nitrogen fertilisers, calcium and water regime on the incidence of cavity spot in carrot. *Fertilizer Research* **4,** 223-230.

GREEN, C. D. and MAKIN, T., 1985. Soil-borne transmission of cavity spot of carrots, grown in north Lincolnshire for processing. *Crop Protection* **4,** 351-358.

GROOM, M. R. and PERRY, D. A., 1985a. Induction of 'cavityspot-like' lesions on roots of *Daucus carota* by *Pythium violae. Transactions of the British Mycological Society* **84,** 755-757.

GROOM, M. R. and PERRY, D. A., 1985b. The epidemiology and control of cavity spot of carrots. *Annual Report, Scottish Crop Research Institute, 1984*, 132.

GUBA, E. F., YOUNG, R. E. and UI, T., 1961. Cavity spot disease of carrot and parsnip roots. *Plant Disease Reporter* **45,** 102-105.

GUERARD, J., 1988. Fungal disease threaten carrot harvests. *American Vegetable Grower, 1988*, August, 17.

GUERIN, L., BRIARD, M. and ROUXEL, F., 1994. Biochemical characterisation of *Pythium* spp. involved in cavity spot of carrots in France. *Annals of Applied Biology* **125**, 255-265.

HAFIDH, F. T. and KELLY, W. C., 1982. Cavity spot of carrots caused by feeding of fungus gnat larvae. *Journal of the American Society for Horticultural Science* **107,** 1177-1181.

HANCOCK, J. G., 1985. Fungal infection of feeder rootlets of alfalfa. *Phytopathology* **75,** 1112-1120.

HOWARD, R. J., PRATT, R. G. and WILLIAMS, P. H., 1978. Pathogenicity to carrots of *Pythium* species from organic soils of North America. *Phytopathology* **68,** 1293-1296.

JACOBSOHN, R., DAN, C., YAAKOBI, C. and SANDER, N., 1973. [Unidentified spots on carrots]. *Hassedeh* **53,** 1168-1171.

JACOBSOHN, R., ZUTRA, D., DAN, H. and KELMAN, Y., 1984. Studies of carrot cavity spot. *Journal of Horticultural Science* **59,** 529-535.

JAKOBSEN, S. T. and JORGENSEN, I., 1986. Virkning af godninger pa Cavity Spot i gulerodder. [Effect of fertiliser treatment on cavity spot in carrots]. *Gartner-Tidende* **102,** 748-749.

JENSEN, A., 1973. Syddomme hos gulerodder. *Soertryk of landbonyt* **27,** 75-82.

JORGENSEN, I., 1986. Jorddesinfektion med metam-Na til gulerodder. [Soil sterilisation with metam-sodium for carrot growing]. *Gartner-Tidende* **102,** 38-39.

KALU, N. N, SUTTON, J. C. and VAARTAJA, O., 1976. *Pythium* species associated with root dieback of carrot in Ontario. *Canadian Journal of Plant Science* **56,** 555-561.

KITAGAWA, T., SAKAMOTO, Y., FURUMI, K. and OGURA, H., 1989. Novel enzyme immunoassays for specific detection of *Fusarium oxysporum* f.sp. *cucumerinum* and for general detection of various *Fusarium* species. *Phytopathology* **79,** 162-165.

LONG, E., 1985. Cavity spot - the culprits are Pythium fungi. *Grower, 1985,* March 14, 15-16.

LYONS, N. F and WHITE, J. G., 1992. Detection of *Pythium violae* and *Pythium sulcatum* in carrots with cavity spot. *Annals of Applied Biology* **120,** 235-244.

LYSHOL, A. J., SEMB, L. and TAKSDAL, G., 1984. Reduction of cavity spot and root dieback in carrots by fungicide applications. *Plant Pathology* **33,** 193-198.

MARTIN, F. N. & HANCOCK, J. G., 1986. Association of chemical and biological factors in soils suppressive to Pythium ultimum. *Phytopathology* **76,** 1221-1231.

MAYNARD, D. N., GERSTEN, B., VLACH, E. F. and VERNELL, H. F., 1961. The effects of nutrient concentration and calcium levels on the occurrence of carrot cavity spot. *Proceedings of American Society for Horticultural Science* **78,** 339-342.

MAYNARD, D. N., GERSTEN, B., YOUNG, R. E. and VERNELL, H. F., 1963. The influence of plant maturity and calcium level on the occurrence of carrot cavity spot. *Proceedings of American Society for Horticultural Science* **83,** 506-510.

MCDONALD, M. R., 1994. Cavity spot of carrot (*Pythium* spp.): Etiology, epidemiology and control. *Ph.D. Thesis. University of Guelph.* 399pp.

MCDONALD, M. R. and EDGINGTON, L. V., 1988. Development of control of cavity spot of carrot. *Canadian Journal of Plant Pathology* **10**, 369-370. (Abstr.)

MCDONALD, M. R. and EDGINGTON, L. V., 1989. Evaluation of metalaxyl and plant growth promoting rhizobacteria for the control of cavity spot of carrot. *Canadian Journal of Plant Pathology* **11**, 194. (Abstr.)

MCDONALD, M. R., KNIBBE, E. N. and EDGINGTON, L. V., 1987. Evaluation of phenylamides for the control of cavity spot of carrots. *Phytopathology* **77**, 120. (Abstr.)

MCDONALD, M. R. and SUTTON, J. C., 1992. Horizontal resistance to cavity spot in commercial carrot cultivars. *Canadian Journal of Plant Pathology* **14**, 245-246. (Abstr.)

MCDONALD, M. R. and SUTTON, J. C., 1993. Influence of cultivar, plant age, rainfall and soil temperature on the development of cavity spot of carrot. *Proceedings of the 6th International Congress of Plant Pathology, Montreal, Canada*, p. 106. (Abstr.)

MCPHERSON, G. M., 1995. Evaluation of fungicides applied as seed treatments, HV sprays or granules for the control of cavity spot in susceptible and tolerant carrot cultivars. *HDC report (FV 5b).* 94 pp.

MILDENHALL, J. P. and WILLIAMS, P. H., 1970. *Rhizoctonia* crown rot and cavity spot of muck-grown carrots. *Phytopathology* **60**, 887-889.

MONTFORT. F. and ROUXEL, F., 1988. La maladie de la "tache" de la carotte due á *Pythium violae* Chesters et Hickman: donnees symptomatologiques et etiologiques. *Agronomie* **8**, 701-706.

NAGAI, Y., FUKAMI, M., MURATA, A. and WATANABE, T., 1986. Brown-blotted root rot of carrots in Japan (1). Occurrence, symptoms and isolation. *Annals of the Phytopathological Society of Japan* **52**, 278-286.

NORMAN, B., 1981. Cavity spot conundrum. *Supplement to Grower, 1981*, November 26, 20.

PERRY, D. A., 1967. Carrot root disorders. *Agriculture* (London) **74**, 222-225.

PERRY, D. A., 1972. Excess potash linked to cavity spot. *Grower, 1972*, April 22, 19.

PERRY, D. A., 1982 Induction of carrot lesions. *Annual Report of the Scottish Crop Research Institute for 1981*, 82-83.

PERRY, D. A., 1983. Effect of soil cultivation and anaerobiosis on cavity spot of carrots. *Annals of Applied Biology* **103**, 541-547.

PERRY, D. A. and GROOM, M. R., 1984. Epidemiology and etiology of root disorders of carrots. *Report of the Scottish Crop Research Institute, 1983*. 128.

PERRY, D. A. and HARRISON, J. G., 1977. Pectolytic anaerobic bacteria cause symptoms of cavity spot in carrots. *Nature* **269**, 509.

PERRY, D. A. and HARRISON, J. G., 1979a. Cavity spot of carrots. I. Symptomology and calcium involvement. *Annals of Applied Biology* **93**, 101-108.

PERRY, D. A. and HARRISON, J. G., 1979b. Cavity spot of carrots. II. The effect of soil conditions and the role of pectolytic anaerobic bacteria. *Annals of Applied Biology* **93**, 109-115.

PERRY, D. A. and HORGAN, A., 1983. Cavity spot of carrots. Field experiment. *Annual Report of the Scottish Crop Research Institute Annual Report for 1982*, 110-111.

PERRY, D. A. and RUBENS, T. G., 1967. Cavity spot of carrots. *Annual Report of the Scottish Horticultural Research Institute for 1966*, 37.

PETCH, G. M., MAUDE, R. B. and WHITE, J. G., 1991. Effect of film coat layering of metalaxyl on the germination of carrot seeds, their emergence and the control of cavity spot. *Crop Protection* **10**, 117-120.

PETCH, G. M. and WHITE, J. G., 1995. Coping with cavity spot. *Grower* **123**, 25-28.

PHELPS, K., WHITE, J. G. and HENN, A. J., 1991. Studies on the spatial distribution of *Pythium*-induced cavity spot of carrots. *Annals of Applied Biology* **119**, 21-30.

PLAATS-NITERINK, A. J., van der, 1975. Species of *Pythium* in the Netherlands. *Netherlands Journal of Plant Pathology* **81**, 22-57.

PLAATS-NITERINK, A. J., 1981. Monograph of the genus Pythium. *Studies in Mycology No. 21. Centraalbureau voor Schimmelcultures, Baarn*, 242 p.

PRATT, R. G. and MITCHELL, J. E., 1973. A new species of *Pythium* from Wisconsin and Florida isolated from carrots. *Canadian Journal of Botany* **51**, 333-339.

PUNJA, Z. K., 1990. Association of *Pythium* spp. and other soilborne fungi with cavity spot lesions on fresh market carrots grown in muck soils. *Canadian Journal of Plant Pathology* **12**, 338. (Abstr.)

RUBENS, T. G. and HALFORD, M. 1983. Seeking the causes of cavity spot in the field. *Grower, 1983*, February 24, 49, 51, 53.

SAALTINK, G. J., 1969. Root rot of hyacinth caused by species of *Pythium*. *Netherlands Journal of Plant Pathology* **75**, 343-354.

SANDERS, P. L., 1984. Failure of metalaxyl to control Pythium blight on tufgrass in Pennsylvania. *Plant Disease* **68**, 776-777.

SCAIFE, M. A., BURTON, A. K. and TURNER, M. K., 1980a. Cavity spot of carrots - an association with soil ammonium. *Communications in Soil Science and Plant Analysis* **11**, 621-628.

SCAIFE, M. A., BURTON, A. K. and TURNER, M. K., 1980b. Cavity spot of carrots. *National Vegetable Research Station Annual Report 30*, 110.

SCAIFE, M. A., TURNER, M. K., BARNES, A. and HUNT, J., 1983. Cavity spot of carrots - observations on a commercial crop. *Annals of Applied Biology* **102**, 567-575.

SCAIFE, M. A., TURNER, M. K., HUNT, J. and BARNES, A., 1981. Cavity spot of carrots - A pot experiment on cation effects. *Communications in Soil Science and Plant Analysis* **12**, 897-908.

SCHRANDT, J. K., DAVIS, R. M. and NUÑEZ, J. J., 1994. Host range and influence of nutrition, temperature and pH on growth of *Pythium violae* from carrot. *Plant Disease* **78**, 335-338.

SHAROM, M. S. and EDGINGTON, L. V., 1982. The adsorption, mobility, and persistence of metalaxyl in soil and aqueous systems. *Canadian Journal of Plant Pathology* **4**, 334-340.

SHLEVIN, E., BEN-NUN, R., TZROR, L., NACHMIAS, A. and OHALI, I., 1987. [Reduction of "cavity spots" in carrots by Ridomil applications]. *Hassadeh* **67**, 2272-2274.

SMITH, B. M. and CROWTHER, T. C., 1991. Carrot breeding work at HRI-Wellesbourne. *Umbelliferae Improvement Newsletter 1*, 7.

SOROKER, E., BASHAN, Y. and OKON, Y., 1984. Reproducible induction of cavity spot in carrots and physiological and microbial changes occurring during cavity formation. *Soil Biology and Biochemistry*. **16**, 541-548.

STANGHELLINI, M. E., 1974. Spore germination, growth and survival of *Pythium* in soil. *Proceedings of American Phytopathological Society* **1**, 211-214.

SWEET, J. and BEALE, R., 1988. It's the big screen for cavity spot. *Grower*, September 8th, 1988, 16-17.

SWEET, J. B., BEALE, R. E. and WRIGHT, I. R., 1989. Cavity spot disease in six carrot cultivars treated with a metalaxyl and thiram fungicide. *Tests of Agrochemicals and Cultivars* **10** (*Annals of Applied Biology* **114**, *Supplement*) 38-39.

SWEET, J. B., LAKE, S. E., WRIGHT, I. R. and PRIESTLEY, R. H., 1986. Resistance of carrot varieties to cavity spot disease. *Aspects of Applied Biology* **12**, 235-245.

TAKSDAL, G., 1990. Verdiproving i gronsaker. Seine gulrotsortar til friskkonsum. [Performance trials of vegetables. Maincrop carrots for the fresh market]. *Aktuelt fra Statens Fagtjeneste for Landbrucket No. 9*, 19pp.

TYLER, F., 1971. High potash levels could encourage cavity spot. *Grower, 1971*, November 13, 958.

VAARTAJA, O., 1967. Damping-off pathogens in S. Australia. *Phytopathology* **57**, 765-768.

VIVODA, E., DAVIS, R.M., NUÑEZ, J.J. and GUERARD, J.P., 1991. Factors affecting the development of cavity spot of carrot. *Plant Disease* **75**, 519-522.

WAGENVOORT, W. A., BATIK, I. and FINDENEGG, G. R., 1985. The effect of oxygen supplySoil and calcium levels in hydroponic culture on the occurrence of cavity spot. *Soilless Culture* **1**, 67-72.

WAGENVOORT, W. A., BLOK, I. MOMBERG, H. F. M. and VELHUIZEN, T., 1989. Cavity spot of carrots in relation to a Pythium sp. *Gartenbauwissenschaft* **54**, 70-73.

WALKER, G. E., 1988. Control of cavity spot of carrot with metalaxyl and phosphorous acid. *Australasian Plant Pathology* **17**, 41-44.

WALKER, G. E., 1991. Effect of metalaxyl and phosphonate on incidence of cavity spot in carrots. *Australasian Plant Pathology* **20**, 21-26.

WATANABE, T., NAGAI, Y. and FUKAMI, M., 1986. Brown-blotted root rot of carrots in Japan (2). Culture and identification. *Annals of the Phytopathological Society of Japan* **52**, 287-291.

WHEATLEY, G. A., HARDMAN, J. A. and EDMONDS, G. H., 1984a. Development of cavity spot in carrots and effect of pesticides. *Report of the National Vegetable Research Station for 1983*, 40-41. UK, Wellesbourne.

WHEATLEY, G. A., HARDMAN, J. A. and EDMONDS, G. H., 1984b. Effects of metalaxyl and carbofuran in a fluid-drilling gel on cavity spot incidence and the yield of carrots. *Tests of Agrochemicals and Cultivars 1984 No. 5* (*Annals of Applied Biology* **104**, *Supplement*), 38-39.

WHIPPS, J. M. & LUMSDEN, R. D., 1991. Biological control of *Pythium* species. *Biocontrol Science and Technology* **1**, 75-90.

WHITE, J. G., 1984. The association of *Pythium* and *Phytophthora* spp. with cavity spot. *Report of the National Vegetable Research Station for 1983*, 80-81.

WHITE, J. G., 1986. The association of *Pythium* spp. with cavity spot and root dieback of carrots. *Annals of Applied Biology* **108**, 265-273.

WHITE, J. G., 1988. Studies on the biology and control of cavity spot of carrots. *Annals of Applied biology* **113,** 259-268.

WHITE, J. G., 1991. Curing spotty carrots. *Grower* **115,** 9-10.

WHITE, J. G., 1992. The effects of previous cropping and fungicides on field populations of *Pythium* oligandrum. *Phytoparasitica* **20**, Supplement 117s-120s.

WHITE, J. G., 1993. A natural approach to cavity spot. *Grower* **120,** pp. 9-10.

WHITE, J. G., DOWKER, B. D. and CROWTHER, T. C., 1987. Screening carrot cultivars against *Pythium* spp. associated with cavity spot. *Tests of Agrochemicals and Cultivars* **8**, (*Annals of Applied Biology* **110**, *Supplement*) 138-139.

WHITE, J. G., DOWKER, B. D., CROWTHER, T. C. and WAKEHAM, A. J., 1988. Laboratory screening of carrot cultivars with reported differential field performance for cavity spot to three *Pythium* spp. *Tests of Agrochemicals and Cultivars* **9**, (*Annals of Applied Biology* **112**, *Supplement*) 76-77.

WHITE, J. G, HILTUNEN, L. H. and PETCH, G. M., 1996. Screening of fungicides against the pathogens which cause cavity spot of carrot. *HDC report (FV 5d).* 25pp.

WHITE, J. G., LYONS, N. F. and PETCH, G. M., 1996. Development of a commercial diagnostic test for the pathogens which cause cavity spot of carrot. 1996 *BCPC Symposium Proceedings No 65: Diagnostics in Crop Production*, 343-348.

WHITE, J. G., STANGHELLINI, M. E. and AYOUBI, L. M., 1988. Variation in the sensitivity to metalaxyl of *Pythium* spp. isolated from carrot and other sources. *Annals of Applied Biology* **113,** 269-277.

WHITE, J. G. and WAKEHAM, A. J., 1987. Responses of Pythium spp. associated with cavity spot of carrots to metalaxyl and related fungicides. *Tests of Agrochemicals and Cultivars 1987, No. 8* (*Annals of Applied Biology* **110**, *Supplement*) 52-53.

WHITE, J. G., WAKEHAM, A. J. & PETCH, G. M., 1992. Deleterious effect of soil-applied metalaxyl and mancozeb on the mycoparasite *Pythium oligandrum. Biocontrol Science and Technology* **2**, 335-340.

WHITE, J. G., WAKEHAM, A. J. and SHLEVIN, E., 1993. *Pythium violae* isolated from cavity spot lesions on carrots in Israel. *Phytoparasitica* **21,** 230-243.

WISBEY, B. D., COPEMAN, R. J. and BLACK, T. A., 1977. Other incitants associated with *Pythium* root dieback of muck-grown carrots in British Columbia. *Canadian Journal of Plant Science* **57,** 235-242.

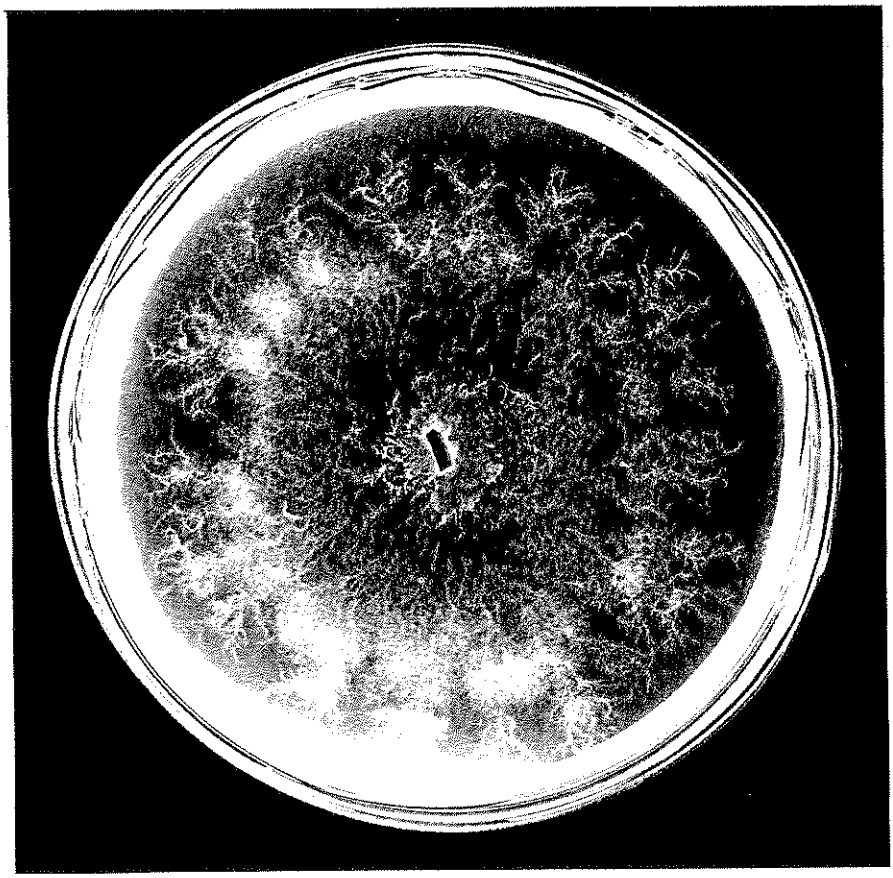
ZAMSKI, E. and PERETZ, I., 1995. Cavity spot of carrots: Interactions between the host and pathogen, related to the cell wall. *Annals of Applied Biology* **127**, 23-32.

ZAMSKI, E. and PERETZ, I., 1996. Cavity spot of carrots: II. Cell-wall-degrading enzymes secreted by *Pythium* and pathogen related proteins produced by the root cells. *Annals of Applied Biology* **128**, 195-207.

There are number of references that are not cited as they either duplicate the information given or are not considered to contribute significantly to current knowledge. A list of these references can be obtained from the project leader.

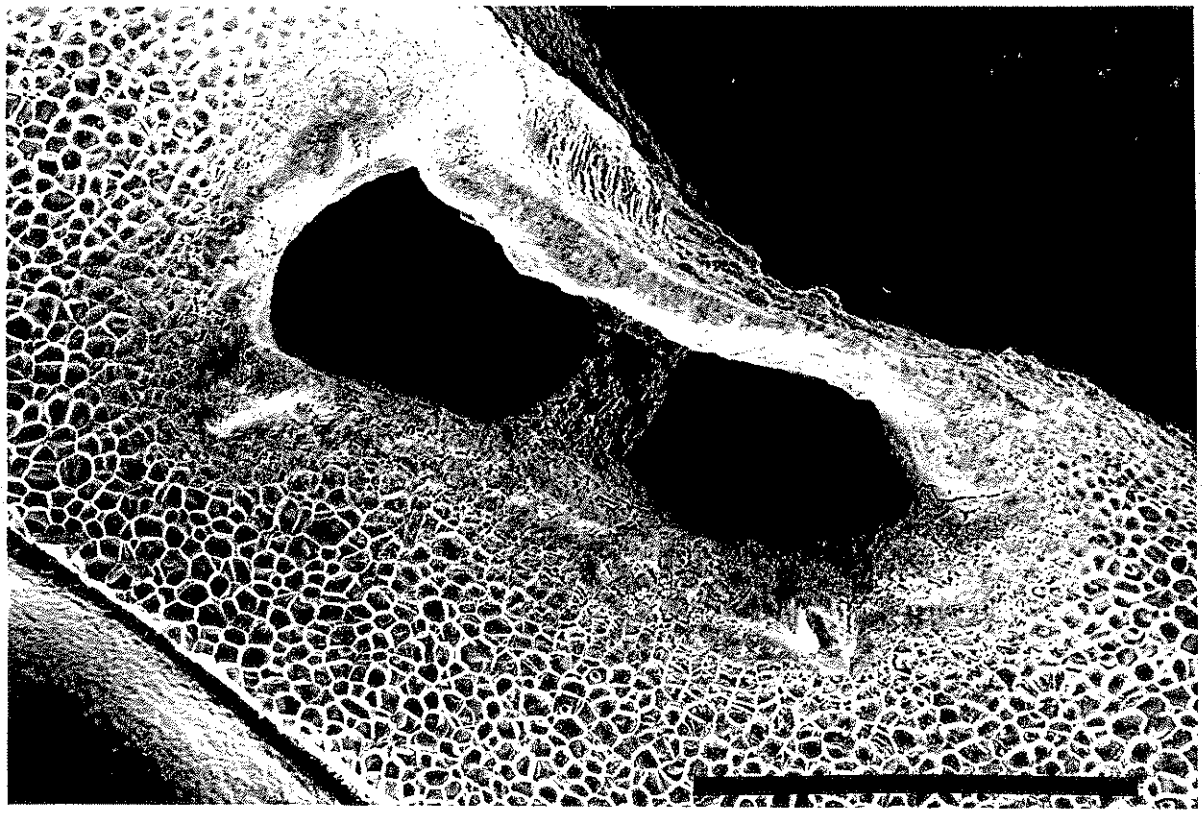
APPENDIX 1

Direct isolation of *Pythium violae* from carrot tissue using a highly selective isolation medium.



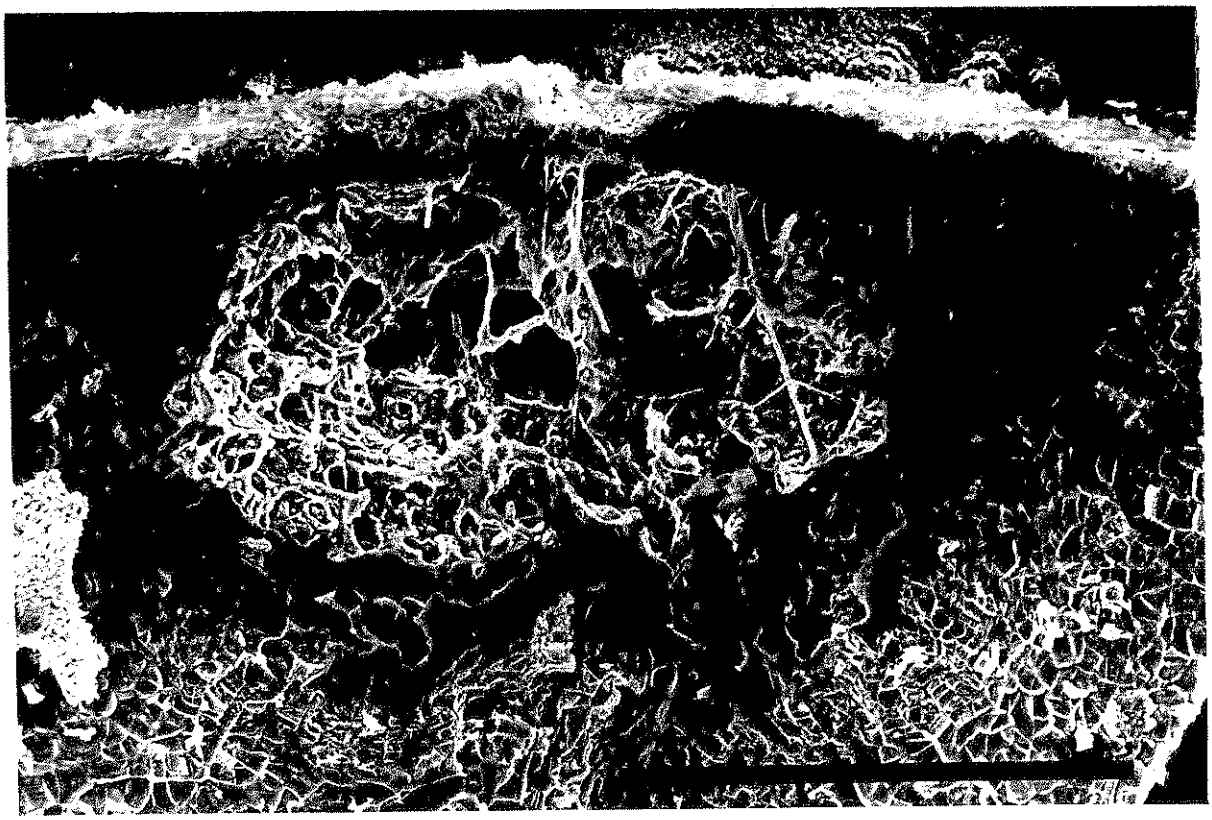
APPENDIX 2

Freeze fracture preparation of a cavity recently induced. There is no evidence of the presence of a fungal pathogen or discoloration of tissue showing reaction of carrot to *Pythium*. Bar = 1 mm.



APPENDIX 3

Freeze fracture preparation of an old, secondarily infected cavity showing fungal mycelium, bacterial cells, deposition of polyphenols. Bar = 1 mm.



APPENDIX 4

Table 1. NIAB resistance ratings to cavity spot of early and maincrop carrot varieties (Anon., 1994, 1995, 1996b).

Variety	Cavity spot resistance raiting		
	1994 1=low; 9=high	1995 A=high; D=low 1=low; 9=high	1996 A=high; D=low
Early maincrop			
Almaro	(4)	-	-
Anglia	2	C	D
Cosmos	(5)	(B)	(C)
Favor	5	B	C
Futuro	(4)	-	-
Meteor	(6)	(B)	(B)
Moreno	(6)	-	-
Nabora	(4)	(C)	(C)
Nairobi	5	B	C
Nandor	5	B	C
Nansen	(5)	(B)	(C)
Nantura	6	-	-
Narbonne	7	B	B
Navarre	7	B	B
Nerac	-	(A)	(A)
Newburg	(6)	-	-
Newmarket	-	(B)	(B)
Panther	6	B	C
Punta	(7)	(B)	-
Valor	(7)	(B)	(B)
Canning			
Chantenay Red Cored 2 - Cluseed New Model	(3)	(3)	-
Chantenay Red Cored 2 - Comet	5	5	-
Chantenay Red Cored 2 - Redco	4	4	-
Chantenay Red Cored 3 - Supreme	1	1	-

- Not tested
( ) Limited data

.../continued

APPENDIX 4

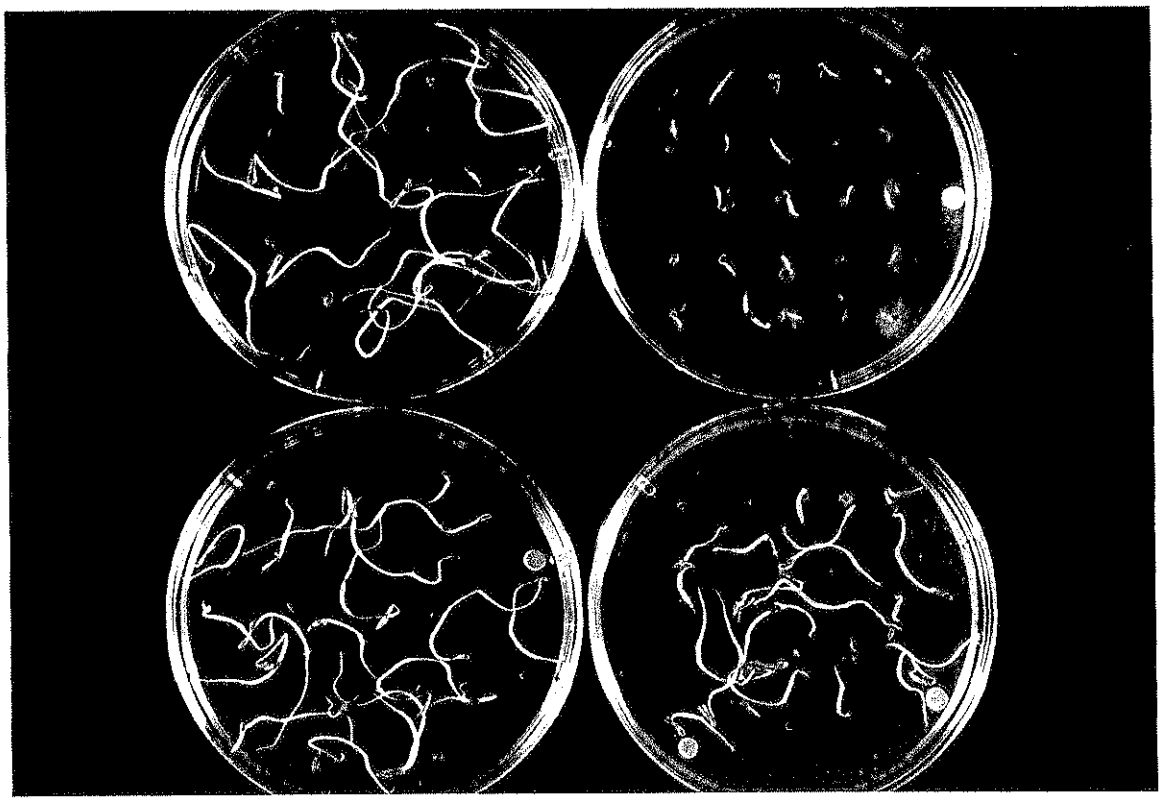
Table 1. - continued

Variety	Cavity spot resistance raiting		
	1994 1=low; 9=high	1995 A=high; D=low 1=low; 9=high	1996 A=high; D=low
Late maincrop			
Bangor	(4)	(C)	(C)
Bergamo	(2)	(D)	-
Bertan	(1)	(D)	(D)
Bolero	(7)	(B)	(B)
Boston	(6)	(B)	(B)
Camberley	3	D	(C)
Camden	5	B	(B)
Campestra	4	C	(C)
Carlo	-	-	(B)
Cordia	(1)	(D)	(C)
Flacino	(6)	(B)	-
Futuro	(2)	-	-
Invictor	(5)	-	-
Lagor	-	-	(D)
Magno	-	-	(D)
Major	2	D	(D)
Moreno	(4)	-	-
Nairobi	-	-	(C)
Nantes 2-Titan	(2)	(D)	-
Narbonne	(5)	C	B
Narman	4	C	C
Navarre	-	-	C
Nerac	-	(B)	(B)
Newmarket	-	(C)	(C)
Punta	(5)	(C)	-
Senior	(3)	(D)	(C)
Sheila	(1)	(D)	(D)

- Not tested
( ) Limited data

APPENDIX 5

Petri dish cultures of *Pythium violae* show kill of carrot seedlings within 5 days. When plates are co-inoculated with *Pythium oligandrum* seedling germination and growth are almost equivalent to that on uninoculated plates and those inoculated only with *P. oligandrum*.



Key

Top left:	uninoculated control - complete germination with healthy seedlings
Top right:	inoculated with *Pythium violae* - complete kill of seedlings
Bottom left:	inoculated with *Pythium oligandrum* - complete germination with healthy seedlings
Bottom right:	co-inoculated with *Pythium violae* and *Pythium oligandrum* - suppression of pathogenic effect of *Pythium violae*