VG96015
An investigation of carrot diseases in north-western Tasmania and their control

Hoong Pung and Pam Cox
Serve-Ag Research

Know-how for Horticulture™

VG96015

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An investigation of carrot diseases in north-western Tasmania and their control

Final Report

Conducted on behalf of

Horticultural Research and Development Corporation

Project VG96015
(Project completion 30/06/00)

by

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January 31, 2001

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Industry Summary

Concomitant with the rapid expansion in the production of fresh market carrots in Tasmania for domestic consumption in Australia and export to Asia, there has been an increase in the incidence of a range of diseases that affect not only marketable yield but also post-harvest storage. Little is known about these diseases or their causal agents, making it difficult to devise any disease management program.

The aim of this project was to obtain a better understanding of carrot diseases in Tasmania, their associated causal organisms, and their effects on marketable carrots.

As a result of these studies, a carrot disease guide, **"Description of Typical Carrot Diseases & Disorders in Tasmania"**, was published and distributed to carrot growers throughout Australia.

Information on violet root rot disease due to *Rhizoctonia crocorum*, which was first recorded on carrots in Australia through observations made in this project, was compiled in an information flier, **"Violet Root Rot of Carrots"**, and distributed to the carrot industry.

The most common diseases on carrots produced in Tasmania are crown rot, cavity spot, forking and scab. Crown rot is the most common and important carrot disease in Tasmania, and is the most frequent cause of severe reduction in carrot packout rate.

There are two distinct types of crown rot symptoms on carrots, corky crown rot and smooth crown rot. Two types of organisms, *Fusarium* spp. and *Streptomyces* spp., were found to be associated with corky crown rot, whereas three types of organisms, *Fusarium* spp., *R. solani* and *S. sclerotiorum,* were found in association with smooth crown rot.

Although forking and cavity spot are also common, their level is usually low. *Pythium* spp. appears to be the most common cause of carrot forking, and this can be reduced with the application of Ridomil. Root-knot nematode, which also causes carrot forking, tends to be rare. Cavity spot in Tasmania is caused by *Pythium sulcatum*, and although common in Tasmania, the disease incidence is usually low (less than 2% carrots infected).

Other diseases found in Tasmania, *Sclerotinia* rot, tiger stripe and tip rot, are pre-harvest as well as post-harvest diseases. Although their symptoms can develop in the field, they often become evident only after storage and shipping. Despite occurring only on a few crops in some years, these diseases can be devastating since they cannot always be detected in pre-harvest assessments or in the grading process.

Technical Summary

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Information on violet root rot disease due to *Rhizoctonia crocorum*, which was first recorded on carrots in Australia in this project, was compiled in an information flier, **"Violet Root Rot of Carrots"**, and distributed to the carrot industry. It contains information on the pathogen, disease symptoms, and strategies for managing the disease and preventing its spread.

The most common diseases on carrots produced in Tasmania are crown rot, cavity spot, forking and scab. These are pre-harvest diseases, with symptoms that develop in the field. Crown rot, the most common and important carrot disease in Tasmania, is the most frequent cause of severe reduction in carrot packout rate.

There appear to be two distinct types of crown rot symptoms on carrots, corky crown rot and smooth crown rot. Two types of organisms, *Fusarium* spp. and *Streptomyces* spp., were found to be associated with corky crown rot, whereas three types of organisms, *Fusarium* spp., *R. solani* and *S. sclerotiorum,* were found in association with smooth crown rot.

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Cavity spot in Tasmania is caused by *Pythium sulcatum*. Even though cavity spot is common in Tasmania, the disease incidence is usually low (less than 2% carrots infected). Its disease severity on affected carrots in Tasmania is considered mild, with few cavity spot lesions and small lesions. As a result of the mild disease severity, carrots with cavity spots are often still marketable.

Other diseases found in Tasmania, *Sclerotinia* rot, tiger stripe and tip rot, are pre-harvest as well as post-harvest diseases. Although their symptoms can develop in the field, they often become evident only after storage and shipping. Despite occurring only on a few crops in some years, these diseases can be devastating since they cannot always be detected in pre-harvest assessments or in the grading process.

Recommendations

- Long term management strategies are needed for soilborne pathogens such as *Fusarium, Rhizoctonia, Sclerotinia, Streptomyces*, and *Pythium*, which affect carrots as well as many other horticultural and field crops.
- Further studies are required to establish how crown rot disease develops, and to identify strategies for managing it.
- Investigation of methods for early detection of diseases that develop in the field, but mainly become apparent only at post-harvest, is recommended.

Introduction

In Tasmania, carrots have previously been grown mainly for processing into frozen produce by Simplot Australia Pty Ltd and McCain Foods (Australia) Ltd. In recent years, there has been a rapid expansion in the production of fresh market carrots for domestic consumption in Australia and export to Asia.

Unlike carrots produced for processing, the quality of fresh market carrots, as measured by appearance and good post-harvest storage, both in cool storage and in transit, is very important.

Concomitant with the rapid expansion in fresh market carrot production, there has been an increase in the incidence of a range of diseases and disorders that affect not only marketable yield but also post-harvest storage. With the increasing utilisation of soil for root crops it is anticipated that the problem will only increase in severity as the industry expands. Little is known about the different types of diseases or their causal agents, making it difficult to devise any disease management program.

This project, which was initiated by Vecon Pty Ltd (now part of Field Fresh Tasmania Pty Ltd) and Harvest Moon Pty Ltd, was aimed at identifying the different types of carrot diseases that occur, their causes, and their impact on the marketable yield of fresh market carrots.

Crown rot disease was initially identified by Vecon Pty Ltd as a serious disease concern, which often reduced packout of carrots cv Kuroda that were exported to Japan. This disease was subsequently found on other fresh market carrot varieties produced in Tasmania. Studies were therefore conducted in this project to investigate the cause of this disease, of which little is known, and its management.

1. Impact of diseases on marketability

Introduction

Little is known about the types of carrot diseases that occur in Tasmania, or their impact on carrot packout. This study was, therefore, conducted to identify the types of carrot diseases and to quantify their effects on carrot packout.

Materials & Methods

Over a two-year period, field sampling of carrot crops was conducted to assess for diseased carrots prior to harvest. These assessments were conducted on 17 Kuroda crops in 1996-97, and 24 crops in 1997-98 (11 Kuroda and 13 HiPak). Three sub-samples of carrots in one-square metre per hectare were sampled.

The sampled carrots were washed and examined for disease and other defects that can affect carrot marketability. The percentages of carrots affected were tabulated and results are shown in Figures 1.1 – 1.3.

Post-harvest assessments were conducted on 30 Kuroda crops produced in 1999-2000.
The data was based on post-harvest grading assessment of carrots that were rejected for export by Field Fresh Tasmania. A total of 100 carrots were taken from each half tonne bin (after sorting for first-grade export quality carrots), and assessed for cause of rejection.

1. Impact of diseases on marketability

Results & Discussions

Table 1.1: The percentage of carrot crops affected by various diseases

Disease	% Crops affected			Economic impact
	Pre-harvest assessments		Post-harvest & post-grading assessment	
	1996-1997 (17 crops assessed)	1997-1998 (24 crops assessed)	1999-2000 (30 crops assessed)	
Crown rot	100	54	100	A
Corky crown rot	N/a	38	97	A
Smooth crown rot	N/a	46	100	A
Scab	82	50	97	A
Cavity spot	65	13	30	A
Sclerotinia	65	13	47	A, B
Forking	N/a	100	100	A
Tiger stripe rot	0	0	10	A, B
Tip rot	N/a	0	23	A, B

A = pre-harvest disease; B = post-harvest disease

Crown rot, cavity spot, forking and scab

The most common diseases of carrots produced in Tasmania are crown rot, *Sclerotinia* rot, cavity spot, forking and scab (Table 1.1). These are pre-harvest diseases, with symptoms that develop in the field. The field survey study shows that crown rot is a frequent cause of significant losses in carrot production in Tasmania.

In 1997-98, carrots with crown rot were further separated into corky crown rot and smooth crown rot, based on their distinct symptoms (Table 1.1). In crops with high levels of crown rot, symptoms of both corky and smooth crown rot can be present in the same crop (Figures 1.2 & 1.3).

Scab disease occurs on most fresh market carrot crops sown in Tasmania (Table 1.1, Figures 1.4 & 1.5). Its frequent occurrence is not surprising, as most of the ground where carrots are sown have been previously been planted with potatoes. Common scab is a common disease problem on potatoes grown in Tasmania.

Although forking of carrot roots is also common, the disease level is usually low. In the survey conducted in 1997-98, 92% of crops had less than 5% forked carrots (Figure 1.4). Similarly, the post-harvest quality assessment of crops sown in 1999-2000 showed that although forking occurred on 100% of the crops assessed, it accounted for less than 3% of the rejected carrots (Figure 1.5).

1. Impact of diseases on marketability (Cont.)

In the 1997-98 survey, only two crops had very high levels of forked carrots (Figure 1.4). A hard pan caused one occurrence by adversely affecting root elongation (crop no. 20), and the other was due to root-knot nematodes (crop no. 24).

Even though cavity spot is common, the disease level is usually low (less than 2% carrots infected). The disease severity on affected carrots in Tasmania is usually low compared to those grown in Western Australia, with fewer cavity spot lesions and smaller lesions. As a result of the low disease severity, carrots with cavity spots are often still marketable.

The frequent use of crop rotation in Tasmania may explain the low cavity spot disease incidence and severity. Unlike carrot production in other Australian States, where multiple carrot crops are sown consecutively, carrots in Tasmania are typically produced in a minimum of three to four years' rotation with a wide range of horticultural and field crops.

Sclerotinia rot, tiger stripe and tip rot

Sclerotinia rot, tiger stripe and tip rot are pre-harvest as well as post-harvest diseases, where symptoms can develop in the field or after harvest (Table 1.1), although they often become evident only after storage and shipment. Although these diseases occur only on a few crops in some years, they can be devastating, since they cannot be detected in pre-harvest assessments or in the grading process.

Sclerotinia disease usually occurs in the field close to harvest, when dense plant foliage and dead leaves on the ground create ideal warm and wet conditions. *Sclerotinia* rot can occur after harvest on carrots with no previously obvious symptoms, if warm and humid conditions prevail in storage or shipment. In order to minimize potential loss of shipments and reputation, some processors will not export carrots from a crop with obvious *Sclerotinia* infection in the field.

Carrot disorders

Other major causes of reduced carrot pack-out are small sizes, cracks, splits, and misshapen carrots. These factors have been examined in another HRDC project, VG97019, which was conducted in Tasmania.

Figure 1.1: Major carrot disease levels in pre-harvest disease assessment of 17 crops surveyed in the 1996-97 season

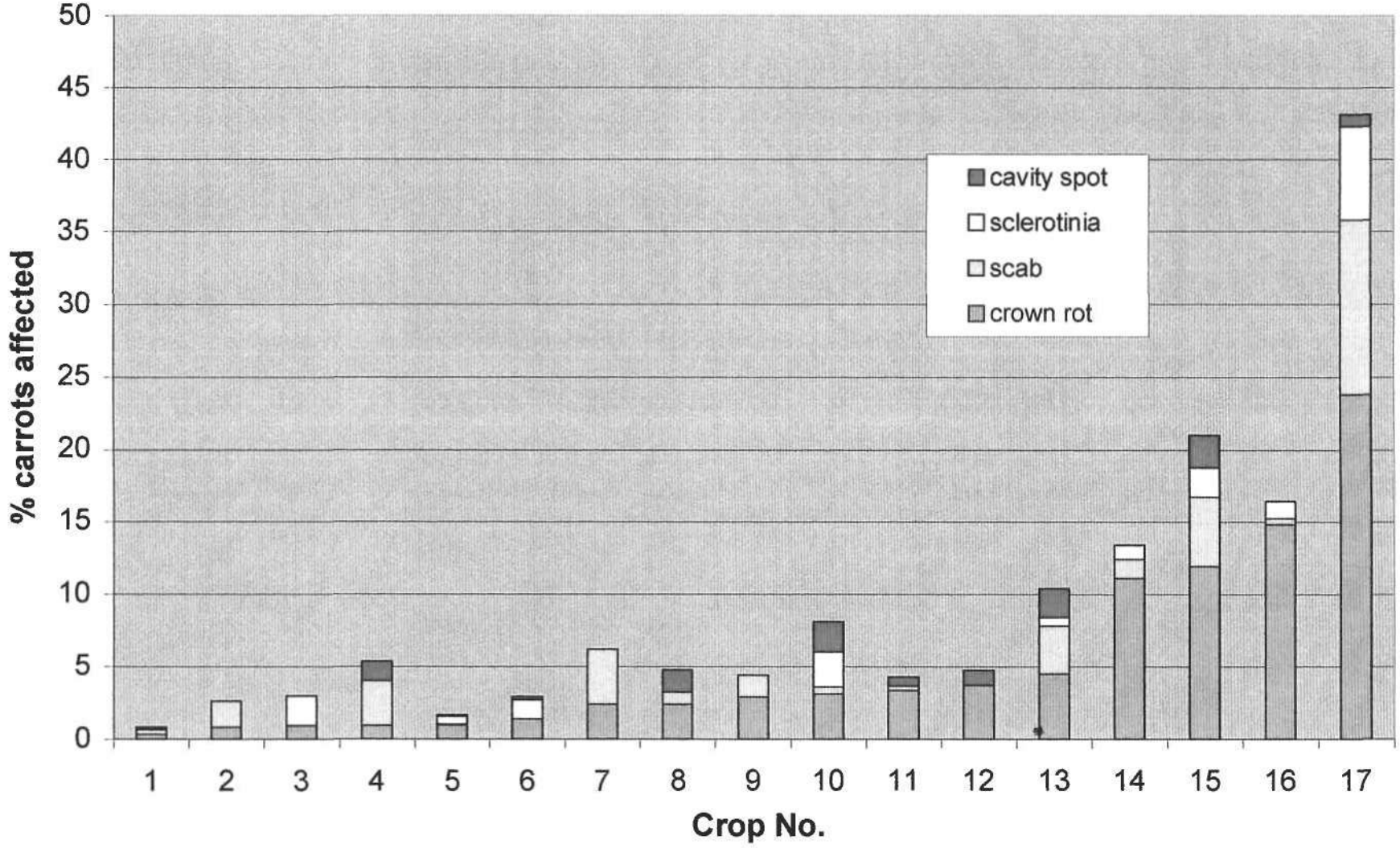


Figure 1.2: Major carrot disease levels in pre-harvest disease assessment of 24 crops surveyed in the 1997-98 season

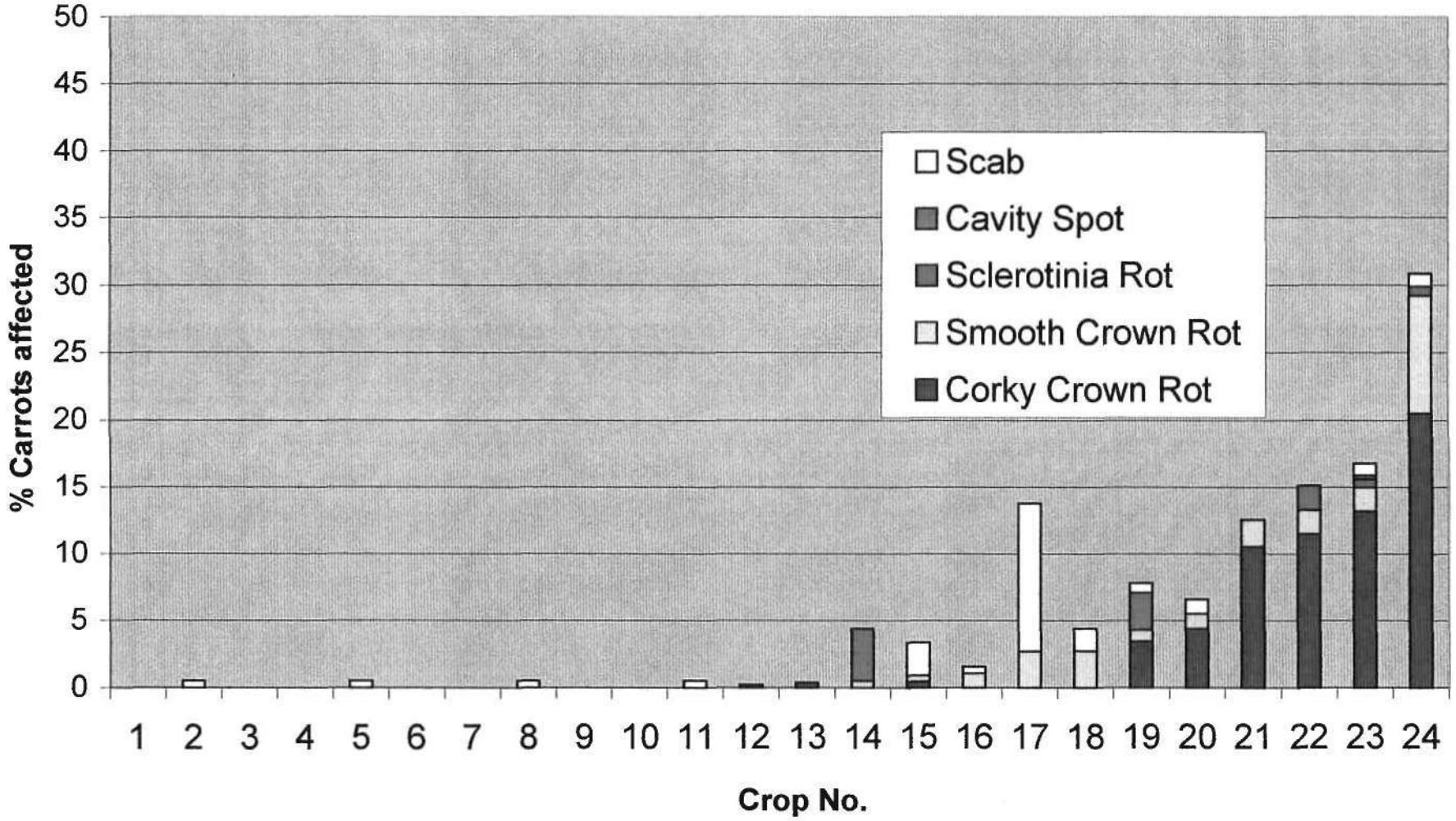
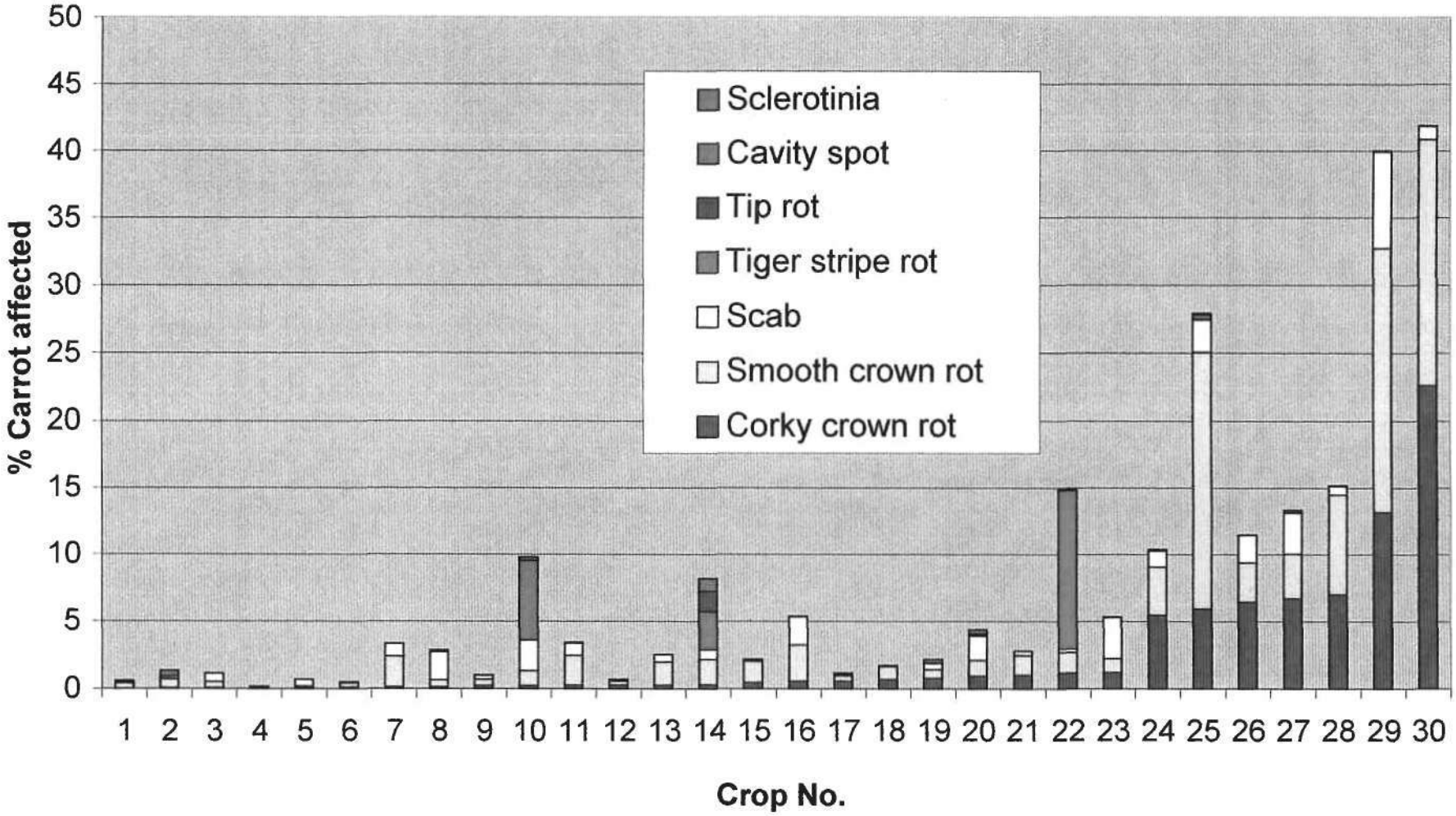


Figure 1.3: Carrot disease levels in carrots rejected for first grade export from crops harvested in 1999-2000 season



1. Impact of diseases on marketability (Cont.)

Figure 1.4: Forked carrot incidence in pre-harvest disease assessment of 24 crops surveyed in the 1997-98 season

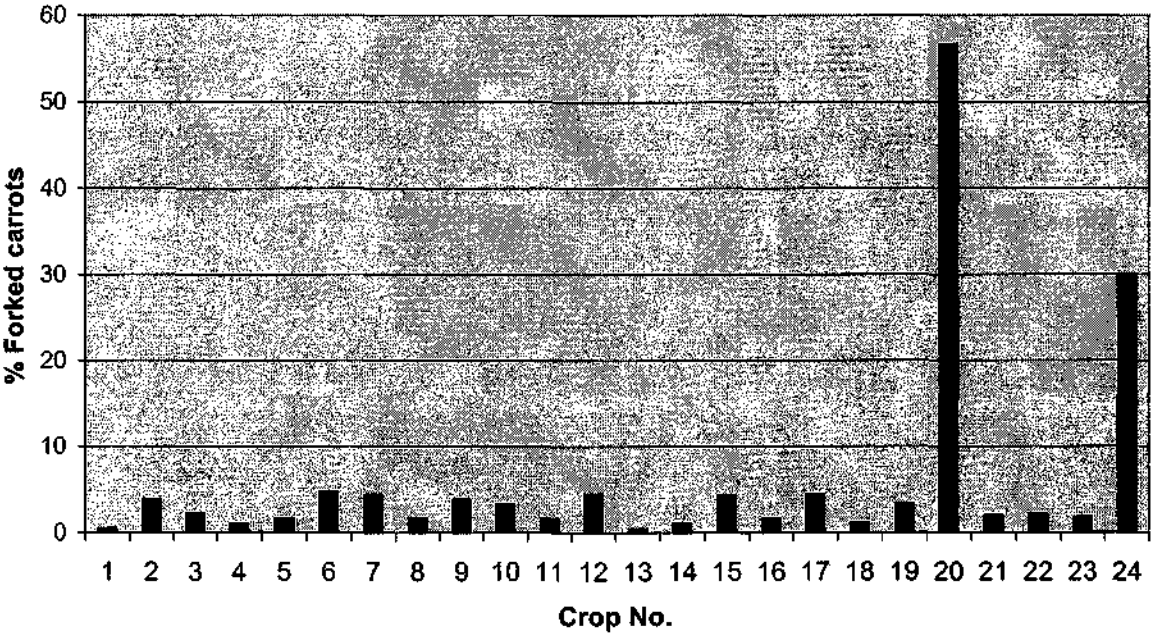
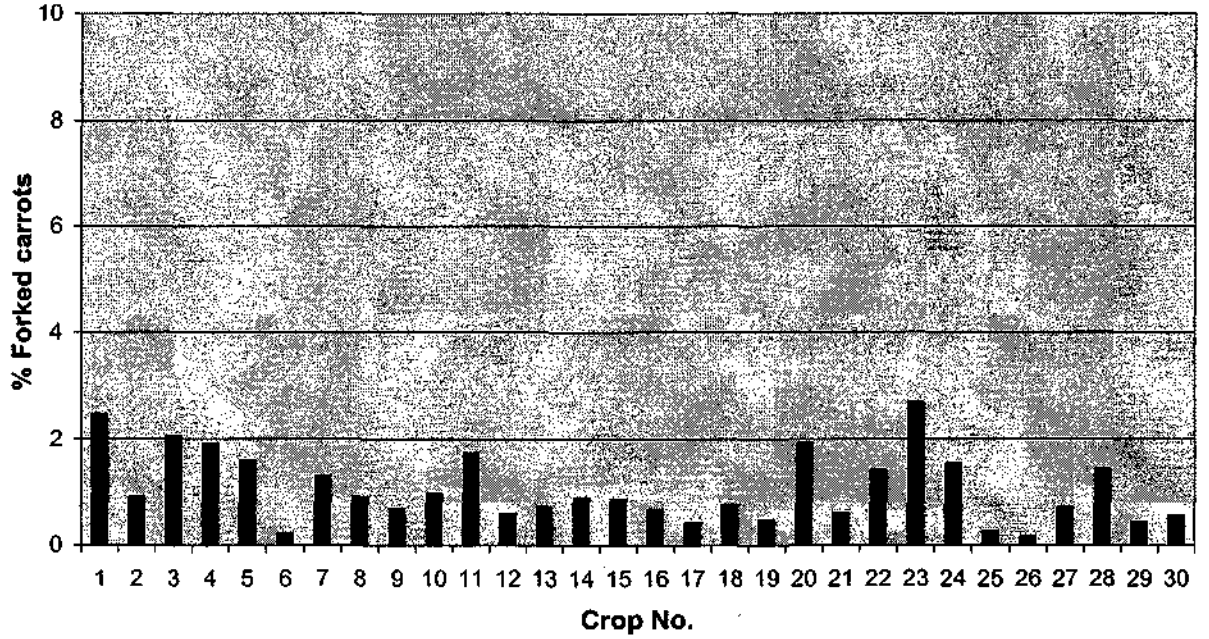


Figure 1.5: Forked carrot incidence of carrots rejected for first grade in crops harvested in 1999-2000 season



2. Causal agents and field conditions

Introduction

This study was conducted to identify the cause of carrot diseases and, where appropriate, associated field conditions and cultural practices.

Diseased carrots from the survey studies conducted from 1996 to 1998 (Section 1), including those rejected due to disease from packing lines or after storage, were also collected for isolations of causal organisms. Where possible, field conditions and practices that may be associated with a disease were recorded. Photographic records of each of the diseases were also taken.

Materials & Methods

Fungal isolations

Sections of diseased tissues from affected carrots were examined. Fungal isolation was carried out by cutting 1cm pieces from diseased areas, which were surface-sterilized for 2 minutes in 1% sodium hypochloride solution and rinsed in sterile water. These samples were blot-dried on sterile tissue paper, plated out onto different agar media (potato dextrose agar, water agar, Coon's agar and P10 agar), and incubated at 23°C.

Fungi isolated from diseased specimens were identified to fungal types, and pure cultures were sent to the appropriate specialist fungal taxonomists for species identification.

Field conditions and cultural practices

When high disease incidence was found in pre-harvest assessments, field inspections were conducted to identify field conditions that may be associated with the disease. This included further sub-sampling from different parts of the crop according to the topography of the paddock (eg. well-drained area vs. poorly drained area, differences in soil types and major weeds).

In addition to carrot sampling and disease assessments, information on field conditions and cultural practices was compiled with the help of a questionnaire. This included information on soil conditions, seed and planting information, plant density, micro-climatic conditions, weeds, pesticide applications, irrigation and crop rotation.

Results & Discussion

Crown rot

Crown rot disease is discussed in Section 4.

Scab

Scab is caused by *Streptomyces* (Janse, 1988, Hanson & Lacy 1990). A pot trial conducted as part of this project (Section 4.4) showed that *S. scabies* isolated from potato scab lesions could cause corky scab lesions on carrots.

Slightly raised corky lesions usually develop at the point where lateral roots emerge from the tap rot. Many lesions can join to cause a continuous scabby band across the tap root.

In field survey studies and inspections conducted in Tasmania, high scab incidence was often found to be associated with potatoes as previous crops, or with a high incidence of volunteer potatoes.

Sclerotinia rot

This disease is caused by *Sclerotinia sclerotiorum*. In the field, soft rot caused by *S. sclerotiorum* tends to occur on the crowns of infected carrots. Field observations indicate that the disease is usually initiated by the spread of *Sclerotinia* mycelium from dead plant matter on the ground, which is in contact with healthy carrot foliage and crowns.

Foliar infections by *S. sclerotiorum* can reduce yield by weakening carrot tops, making mechanical harvesting difficult. Infected roots can be symptomless at harvest, but develop soft watery rot in storage. Poor temperature management after harvest increases the risk of *Sclerotinia* rot in storage.

Carrots produced in sheltered or low-lying areas are prone to *Sclerotinia* infection, and damaged plants in irrigation tracks are highly susceptible. High planting density, especially in wide beds, also increases disease incidence.

As the fungal pathogen has a wide host range, this disease could not be related to any previous crop rotation. However, sclerotia (long-term surviving fungal structures) of *S. sclerotiorum* can increase in residues of bean, poppy and potato crops.

Tiger Stripe or *Phytophthora* Root Rot

Phytophthora spp. is widely believed to be the cause of this disease (Ho 1983, Howard et al. 1994), which is commonly known as tiger stripe in Tasmania, while elsewhere it has been called ring rot, rubbery brown rot, black rot, and *Phytophthora* root rot. The name, *Phytophthora* root rot, has been proposed by the American Phytopathogical Society (APS) as the official APS designated common name for this disease (Strandberg 1999).

In this project, both *Phytophthora* and *Pythium* species have been isolated from tiger stripe lesions. Two species of *Phytophthora*, identified as *P. megasperma* and *P. citricola*, were isolated in this study. *P. megasperma* has previously been isolated from diseased carrots in Tasmania (Dowson 1934). Warm and wet conditions pre-dispose carrots to tiger stripe.

The role of the *Pythium* species is believed to be that of secondary invader. *Pythium ultimum*, which was consistently isolated from diseased carrots from a crop, did not cause disease symptoms when carrot roots were inoculated with pure culture of the fungus in a pathogenicity test. It is possible that *Pythium* is often isolated due to the use of P10 as a selective medium for *Phytophthora* isolations. *Pythium* is ubiquitous in soil.

Infected roots can be symptomless at harvest, but can develop a firm brown rot that occurs in a band across the carrot root in storage. In time, the firm rot becomes sunken and soft due to secondary decay by bacteria. Disease symptoms on affected carrots may develop in cold storage, in transit, or when displayed in stores. Incidence and severity of the disease increases with in-ground storage after maturity.

This disease is always associated with carrots produced in level or low-lying ground that is prone to long periods of wetness, caused by temporary flooding from irrigation or rainfall. Heavy soil type also increases the risk of this disease.

Cavity Spot

This disease is caused by *Pythium sulcatum* (Davison et *al.* 2000). In Tasmania, cavity spot appears first as gray lesions that become small elliptical hollow cavities after washing. *P. sulcatum* was isolated from typical cavity spot lesions on carrots from different crops in Tasmania. The cause and management of cavity spot was investigated in the HRDC projects, VG95010 and VG98011, by researchers in Western Australia.

Black Ring

Black ring rot occurs on the carrot crown just below the decaying stems. A range of fungi and bacteria have been found in association with this disease. Foliage and stem base decay may be caused by fungal infections (*Alternaria, Sclerotinia, Fusarium* and *Rhizoctonia*) and bacterial rot.

Carrot crops sown for processing into frozen produce tend to be prone to this disease, because these crops are usually stored in-ground and harvested long after carrot maturity. The practice of slashing foliage and leaving it to rot over carrot crowns also increases the disease incidence.

2. Causal agents and field conditions (Cont.)

Forking

Forking develops as a result of damage to the root tips at the seedling and root elongation stage. A range of organisms or factors, e.g. fungal pathogens, plant parasitic nematodes, soil compaction, and herbicide residues, could cause the early root tip damage.

Pythium spp. appears to be a common cause of forking in Tasmania. Early application of metalaxyl (Ridomil ™) just after seedling emergence has been shown to reduce forking incidence in studies conducted in this project (Section 3). A drastic reduction in carrot forking was observed in a commercial crop at Forth in rows where metalaxyl was applied, compared to adjacent rows where metalaxyl was not applied. Studies conducted elsewhere have shown that metalaxyl also reduces tap root dieback due to *Pythium* spp. on young plants (Walker 1988, Lyshol et *al.* 1984)

One or more pathogenic species of *Pythium* is believed to be involved in *Pythium* root dieback, which can result in carrot forking (Howard et *al.* 1978, Mildenhall et *al.* 1971, Howard et *al.* 1994). In Tasmania, *P. ultimum* and *P. irregulare*, which have been isolated and identified in this project, are capable of causing carrot root dieback at the seedling stage (Howard et *al.* 1994, Ribeiro 1999). Infected seedlings may wilt and die, while those that survive usually have a forked tap root.

Forking of carrots due to plant parasitic nematodes appears to be relatively rare in Tasmania. The combination of relatively cool soil conditions and the use of cultivation in Tasmania, is likely to assist in preventing a build up of plant parasitic nematodes to population levels that can cause damage to plants. Carrots are highly susceptible to root-knot nematode damage (Belair 1987, Howard et al 1994). In field inspections conducted in this study, root-knot nematode was found to be the cause of carrot forking in a few crops. These crops were sown after long-term pasture. Clover in pastures was found to be highly susceptible to root-knot nematode, helping to increase the nematode population. Root lesion nematodes are known to increase in pyrethrum, a long-term perennial crop.

Tip Rot and/or Sour rot

Soft and slimy tip rot develops on the lower parts of carrot roots in the field or after harvest and storage. *Geotrichum* was consistently isolated from tip rot occurring in the field. Severely affected tip rot in the field has a sour vinegar like odour and hence the name sour rot. Wright et al (1964) has identified *Geotrichum candidum* as the cause of sour rot on carrots. Bacteria associated with tip rots are likely to be secondary invaders on decaying tissues.

Carrots produced in ground where water tended to pool for long periods of time after heavy rainfall or irrigation were prone to tip rot. *G. candidum* is believed to be widespread in nature, and is pathogenic to a wide range of plants (Lewis & Sinclair 1966).

2. Causal agents and field conditions (Cont.)

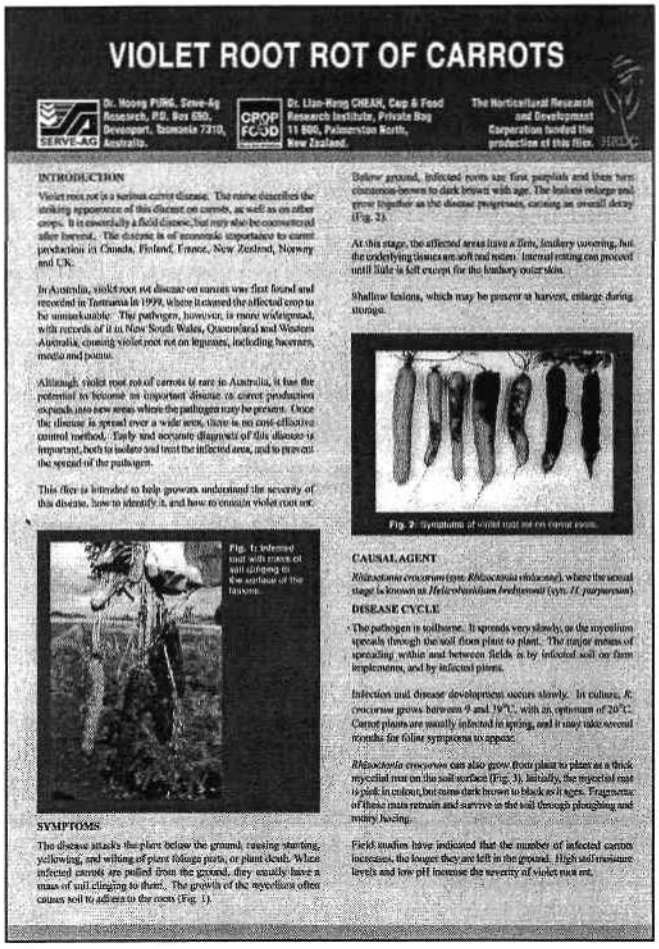
Violet Root Rot

This disease is caused by *Rhizoctonia crocorum* (Whitney 1954). Michael Priest, a fungal taxonomist, confirmed the identification of the disease organism on carrots with violet root rot from an infected crop in Tasmania, in 1999. This is the first record of its occurrence on carrots in Australia. Violet root rot is a serious disease, and the infected crop was not harvested due to the severe rot. This disease occurs in low-lying areas, where water drains from surrounding sloping ground.

As a result of this new discovery, information on violet root rot has been collated and produced into a flier, which was printed and distributed in July 2000.

The flier includes a description of the disease symptoms, alternative hosts, favourable conditions, disease prevention, early detection and appropriate management strategy to prevent spread of the pathogen.

A copy of the violet root rot flier is included with this report (Appendix i).



VIOLET ROOT ROT OF CARROTS

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The Horticultural Research and Development Corporation funded the production of this flier.

INTRODUCTION

Violet root rot is a serious carrot disease. The name describes the striking appearance of this disease on carrots, as well as on other crops. It is essentially a field disease, but may also be encountered after harvest. The disease is of economic importance to carrot production in Canada, Finland, France, New Zealand, Norway and UK.

In Australia, violet root rot disease on carrots was first found and recorded in Tasmania in 1999, where it caused the affected crop to be unmarketable. The pathogen, however, is more widespread, with records of it in New South Wales, Queensland and Western Australia, causing violet root rot on legumes, including lucerne, medic and pasna.

Although violet root rot of carrots is rare in Australia, it has the potential to become an important disease as carrot production expands into new areas where the pathogen may be present. Once the disease is spread over a wide area, there is no cost-effective control method. Early and accurate diagnosis of this disease is important, both to isolate and treat the infected area, and to prevent the spread of the pathogen.

This flier is intended to help growers understand the severity of this disease, how to identify it, and how to contain violet root rot.

Below ground, infected roots are first purplish and then turn cinnamon-brown to dark brown with age. The lesions enlarge and grow together as the disease progresses, causing an overall decay (Fig. 2).

At this stage, the affected areas have a firm, leathery covering, but the underlying tissues are soft and rotten. Internal rotting can proceed until little is left except for the leathery outer skin.

Shallow lesions, which may be present at harvest, enlarge during storage.

Fig. 2 Symptoms of violet root rot on carrot roots.

Fig. 1: Infected root with traces of soil clinging to the surface of the lesions.

CAUSAL AGENT

Rhizoctonia crocorum (syn. *Rhizoctonia violacea*), where the sexual stage is known as *Helicobasidium brebissonii* (syn. *H. purpureum*).

DISEASE CYCLE

The pathogen is soilborne. It spreads very slowly, as the mycelium spreads through the soil from plant to plant. The major means of spreading within and between fields is by infested soil on farm implements, and by infected plants.

Infection and disease development occurs slowly. In culture, *R. crocorum* grows between 9 and 19°C, with an optimum of 20°C. Carrot plants are usually infected in spring, and it may take several months for foliar symptoms to appear.

Rhizoctonia crocorum can also grow from plant to plant as a thick mycelial mat on the soil surface (Fig. 3). Initially, the mycelial mat is pink in colour, but turns dark brown to black as it ages. Fragments of these mats remain and survive in the soil through ploughing and many hosts.

Field studies have indicated that the number of infected carrots increases, the longer they are left in the ground. High soil moisture levels and low pH increase the severity of violet root rot.

SYMPTOMS

The disease attacks the plant below the ground, causing stunting, yellowing, and wilting of plant foliage parts, or plant death. When infected carrots are pulled from the ground, they usually have a mass of soil clinging to them. The growth of the mycelium often causes soil to adhere to the roots (Fig. 1).

2. Causal agents and field conditions (Cont.)

Current knowledge on carrot diseases and disorders in Tasmania

Based on fungal isolations and field survey studies, a list of current knowledge on the types of diseases and disorders and the associated field conditions and practices, and causal organisms, are summarised in Table 2.1.

Table 2.1: Current knowledge on carrot diseases and disorders in Tasmania

Disease or Disorder	Associated Causal Organism	Conducive Field Conditions and/or Practices
Corky crown & side rot	*Streptomyces* spp. & *Fusarium* spp.	Tends to occur in well-drained soil. High incidence can be associated with high numbers of volunteer potatoes. Incidence and severity increase with ground storage after maturity.
Smooth crown & side rot	*Fusarium* spp., *Rhizoctonia solani*, & *Sclerotinia sclerotiorum*	Tends to be worse in wet areas. Incidence and severity increase with ground storage after maturity.
Scab	*Streptomyces* spp.	Tends to be worse in crops sown after potatoes.
Sclerotinia	*Sclerotinia sclerotiorum*	Usually worse with wet and warm conditions in sheltered or low-lying areas. Poor temperature management after harvest increases risk of *Sclerotinia* rot.
Tiger stripe or Phytophthora root rot	*Phytophthora citricola* and *P. megasperma*.	Associated with wet areas or temporary water-logging. Heavier soils increase risk of this disease. Incidence and severity increases with ground storage after maturity.
Cavity spot	*Pythium sulcatum*	Worse in poorly drained soils or in areas with an overlap of irrigation. Incidence increases with ground storage after maturity.
Black ring rot	No specific pathogen. A range of fungi or bacteria has been found in association with this rot.	Foliage senescence associated with over-maturity, in-ground storage, and damaged tops, increase risk of foliage infection and decay, which spreads to the crown.
Forking	*Pythium* spp., root-knot nematode, and other factors that will damage root tips.	Seedling root infections by fungal pathogens, eg. *Pythium,* and parasitic nematodes, or damage by other soil related factors such as soil compaction or herbicide residue.
Sour rot or Tip rot	*Geotrichum candidum* A range of secondary fungi and bacterial invaders hasten the decay.	Carrots sown in areas where water tends to pool are prone to this disease. Tip damage can also develop in the field or storage.
Violet root rot	*Rhizoctonia crocorum*	Soils with poor drainage are conducive to this disease.

Carrot disease guide

Following discussions with growers and field officers, it became apparent that the same disease symptom was called by many different names. This initially created much confusion between growers, field officers from packers and processors, consultants and researchers.

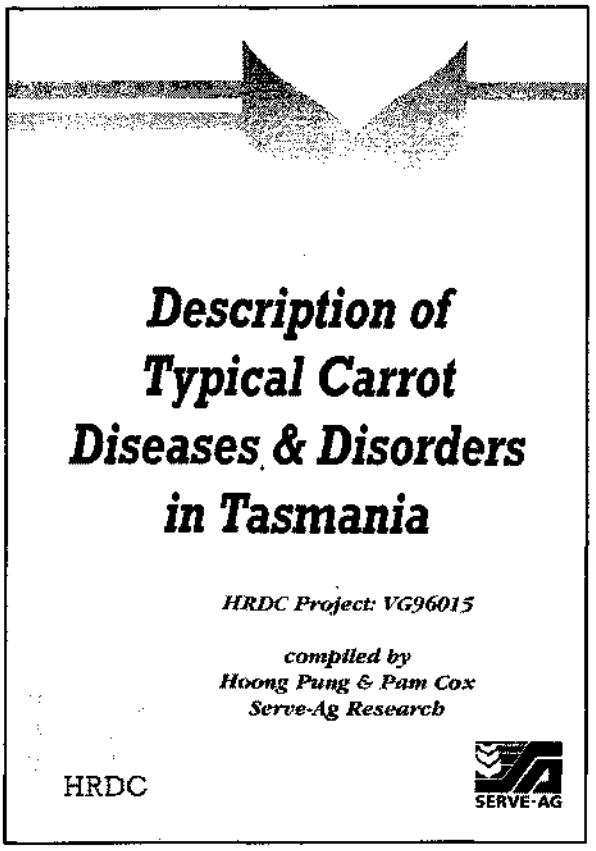
A carrot disease guide, "Descriptions of typical carrot diseases and disorders in Tasmania", was compiled, published and distributed to the carrot industry in Tasmania as well as other states in Australia.

The pictorial guide, with descriptions of typical carrot diseases and disorders, was put together to help growers and others to put a common name to a disease that they are likely to encounter in carrot production.

Photographic records of all carrot diseases and disorders that were encountered in this project were taken and used in the guide.

The use of a common terminology will assist both growers and processors in determining the relative importance of the various diseases/disorders in crop losses, and assist in taking the appropriate actions to prevent further losses.

A copy of the disease guide is included with this report (Appendix ii).



3. Disease development in the field

Introduction

Three field studies were set up in commercial crops in areas that may be prone to carrot diseases, to investigate disease development in the field.

Fungicides that have activity against different groups of fungi were used at the sites to alter the balance of certain groups of soilborne fungi, and enable evaluation of fungal pathogens involved with the major types of carrot diseases, in particular crown rot disease.

Materials and Methods

Trial details

	SITE 1	SITE 2	SITE 3
LOCATION	Forth (BH)	Deloraine (LE)	Kindred (AJ)
VARIETY	HiPak	Ringo	Kuroda (plot 1) HiPak (plots 2-3)
PLOT SIZE	1.6m x 5m	1.1m x 5m	1.6m x 5m
BEDS	Narrow beds	Wide beds	Narrow beds
PLANT DENSITY	65 (1m x 2 rows)	278 (1m x 8 rows)	65 (1m x 2 rows)
REPLICATES	3	3	3
PLANTING DATE	6/11/98	13/11/98	2/10/98
ASSESSMENT DATE	16/3/99	11/5/99	9/3/99

Product Formulations

Product	Active Ingredient (a.i.)	Rate of a.i.
Mancozeb	mancozeb	750g/kg
Ridomil Gold MZ	metalaxyl	50g/kg
Shirlan	fluazinam	500g/L

Treatment List

Site/s	No.	Product	Product Rate/Ha	Application Method
1 & 2	1	Mancozeb	40 kg	Broadcast just after seedling emergence, then drenched in with water at 8L/m^2
1 & 2	2	Ridomil	40 kg	
1 & 2	3	Untreated control	N/a	N/a
3	1	Mancozeb	40 kg	Broadcast just after seedling emergence, then drenched in with water at 8L/m^2
3	2	Shirlan	3 L	Sprayed onto surface just after seedling emergence, then drenched in with water at 8L/m^2
3	3	Untreated control	N/a	N/a

Assessments

Plants in the treatment plots were inspected at 5, 10 and 20 weeks after sowing for signs of above and below ground infections. At close to harvest, disease assessments were conducted on carrots in 1m^2 harvested from each treatment plot. The percentage of carrots affected by disease was tabulated, and the mean values of three replicate plots are given in Tables 3.1 – 3.5.

Results

The major disease at each of the three sites was different.

Site 1

At Site 1, the crop had a high level of smooth crown rot and forked carrots at 130 days after sowing (Table 3.2). The levels of other carrot root diseases (corky crown rot, cavity spot, scab and *Sclerotinia*) were very low and insignificant.

The perfect stage of *R. solani*, teleomorph *Thanatephorus cucumeris*, was noted at the stem base of 60% of carrots in the crop at Site 1. The fungus was recorded on 90% of carrots that had smooth crown rot. The fungus was also found on many volunteer potato plants within the crop.

At harvest, many plants infected by *R. solani* had decaying foliage. The fungus was also isolated from the smooth crown rot tissue, indicating that it was likely to be the primary cause of smooth crown rot at this site.

3. Disease development in the field (Cont.)

Table 3.1: Carrot disease levels at Site 1

No.	Treatment	% Smooth crown rot	% Corky crown rot	% Cavity spot	% Scab	% *Sclerotinia*	% Forked carrots
1	Mancozeb	7.2	0.2	2	0.2	0	9.1
2	Ridomil	3.2	0	1.9	0.3	0.1	0.6
3	Untreated control	4.9	0	0.8	1	0	2.4

Both Mancozeb and Ridomil applications had little or no effect in reducing smooth crown rot (Table 3.1). It is interesting that the percentage of forked carrots appeared to be reduced with Ridomil application, but increased with Mancozeb application. This indicates that the forked carrots were likely to be caused by Oomycetes fungi (eg. *Pythium*), which are controlled by Ridomil.

Mancozeb is a broad-spectrum fungicide that has no effect against Oomycetes fungi. The increase in the percentage of forked carrots in plots treated with Mancozeb is not surprising. Research conducted elsewhere has shown that Mancozeb soil application tends to suppress non-Oomycetes fungi, and results in severe root rot by *Pythium*.

Site 2

At Site 2, the crop had little or negligible levels of corky crown rot (Table 3.2). There was some smooth crown rot, which appeared to be reduced by Mancozeb application.

At this site, both *R. solani* and *S. sclerotiorum* were observed on the foliage of some carrots in the crop at 91 days after sowing. At close to harvest, 178 days after sowing, the foliage of carrots infected by *R. solani* and *S. sclerotiorum* had deteriorated and senesced (Table 3.3). Both fungi were consistently associated with smooth crown rot at this site. In field observations, the spread of foliage infection onto carrot crowns, as well as direct mycelium infection from sclerotia or from infected plant debris onto crown areas was noted.

Soft and light brown coloured rot that was typical of *S. sclerotiorum* and classified as *Sclerotinia* infection was present in levels ranging from 4.5 to 8.3% (Table 3.2). Field observations indicated that dry conditions may halt the spread of *Sclerotinia* infections on carrot crowns, which can then be colonised by secondary invaders, causing a darkening of the infected crown tissues.

In contrast to Site 1, there appears to have been a reduction in the levels of smooth crown rot with Mancozeb application at Site 2.

Pythium sulcatum was isolated from cavity spot, which was present at high levels at this site. The Ridomil drenched application reduced the level of carrots affected by this disease.

3. Disease development in the field (Cont.)

The level of forked carrots at this site was very low, ranging from 0.9 to 1.8% (Table 3.2). Damaged root tips at the seedling stage led to forked carrots. *P. sulcatum*, which causes cavity spot and is also known to cause tip damage on seedlings and hence carrot forking, was present at the site, however it is possible that soil conditions were not conducive to *Pythium* infection at the seedling stage.

The high scab incidence at this site did not appear to be affected by Mancozeb application, but seemed to be increased by Ridomil application.

Table 3.2: Carrot disease levels at Site 2

No.	Treatment	% Cavity spot	% Scab	% Corky crown rot	% Smooth crown rot	% Tiger stripe	% *Sclero-tinia*	% Forked
1	Mancozeb	18.4	9.6	0.2	0.5	0	4.7	0.9
2	Ridomil	6.1	12.5	0	2.4	0	8.3	1.0
3	Untreated control	13.7	8.8	0.2	3.8	0.9	4.5	1.8

Black sclerotia of *R. solani* were found attached to the surface of carrot roots. Fungal cultures of *R. solani* were re-isolated from the black sclerotia. This is the first record of *R. solani* sclerotia attached to carrots, similar to black scurf on potatoes. Unlike black scurf on potatoes however, the sclerotia on carrots are removed during the rigorous washing process in commercial operations.

Table 3.3: Effects of *R. solani* and *S. sclerotiorum* foliage infections on senescing tops and sclerotia formation at Site 2

No.	Treatment	% Senescing tops	% Black sclerotia
1	Mancozeb	60.8	3.1
2	Ridomil	62.6	1.4
3	Untreated control	60.1	4.6

At Site 2, *R. solani* was isolated from the foliage of infected carrots, and pure culture of the fungal isolate was grown on potato dextrose agar. The fungus (10mm diameter agar discs of the fungal culture) was then inoculated onto twenty carrot crowns in the field at 137 days after sowing. At 178 days after sowing, a relatively high percentage of the inoculated carrot crowns had smooth crown rot and black sclerotia (Table 3.4).

Table 3.4: Disease incidence on *Rhizoctonia* inoculated carrots at Site 2

No.	Treatment	% Smooth crown rot	% Black sclerotia
1	Inoculated carrots	25	10
2	Non-inoculated carrots	4	4.6

3. Disease development in the field (Cont.)

Site 3

At Site 3, the crop had a high percentage of carrots with scab (Table 3.5). Corky crown rot and forked carrots were also present in significant levels.

As in Site 1, the percentage of forked carrots at Site 3 tended to be higher after Mancozeb application. Mancozeb and Shirlan application did not appear to reduce scab or corky crown rot.

Table 3.5: Carrot disease levels at Site 3

No.	Treatment	% Scab	% Corky crown rot	% Smooth crown rot	% Cavity spot	% Forked
1	Mancozeb	12.8	8.6	2.7	3.6	13.9
2	Shirlan	18.4	4.5	0.3	1.3	3.5
3	Untreated control	13.9	3.0	1.4	1.2	2.2

Discussion

This study indicates an association between smooth crown rot and *R. solani* and *S. sclerotiorum*. *R. solani* has also been recorded as a cause of crown rot in Canada (Howard et al 1994).

In crops where *R. solani* infection occurs, black sclerotia of *R. solani* were also found attached to the surface of carrot roots. These are not a concern, however, as they are removed during the rigorous washing process in commercial operations.

Ridomil (metalaxyl) application reduced cavity spot and carrot forking in this study, indicating that these diseases are likely to be caused by *Pythium* spp. Studies conducted elsewhere have shown that metalaxyl also reduces tap-root dieback due to *Pythium* spp. on young plants (Walker 1988, Lyshol et *al.* 1984).

In contrast, Mancozeb application tends to increase both diseases. These differences show that certain fungicide applications into soil may alter the balance of soilborne fungi, and promote one group of fungi over another.

This study showed that *Pythium* is likely to be a common cause of carrot forking. No root-knot nematodes were found at any of the sites, and root lesion nematode population was very low.

None of the fungicides used in this study showed sufficient efficacy for the control of both types of crown rot.

4. Crown rot disease

Summary

There appear to be two distinct types of crown rot symptoms on carrots, corky crown rot and smooth crown rot. Two types of organisms, *Fusarium* spp. and *Streptomyces* spp., were associated with corky crown rot, whereas three types of organisms, *Fusarium* spp., *R. solani* and *S. sclerotiorum,* were found in association with smooth crown rot. *S. scabies* was shown in a pot trial to cause corky lesions on carrot crowns under relatively dry soil condition, which resembled corky crown rot. In a pathogenicity test conducted in the laboratory, three species of *Fusarium* frequently isolated from crown rot of carrots, *F. culmorum, F. compactum and F. semitectum,* caused crown rot lesions on inoculated carrot crowns. Crown rot, however, did not develop in pot and field trials inoculated with *Fusarium* spp., *R. solani* and *S. sclerotiorum*, yet their roles in crown rot development should not be discounted. Field observations and fungal isolations conducted in this project indicate that in some crops, *R. solani* and S. *sclerotiorum* infections of lower stems spread directly onto the carrot crown, causing smooth crown lesion. Studies conducted elsewhere indicate that pathogenic *Fusarium* species cause crown rot.

4.1. Associated organisms and field conditions

Introduction

This study includes observations on crown rot disease from crop survey studies conducted from 1996-98 (Section 1). Field inspections were also made on crops where the disease incidence was high.

Crown rot

In this project, two distinct types of crown rot symptoms were identified on carrots, corky crown rot and smooth crown rot. The characteristics of the two types of crown rot are described pictorially in the "Description of typical carrot diseases and disorders in Tasmania" (Appendix ii). Both types of crown rot lesions are firm rots, which do not spread or deteriorate in storage, and are rejected due to their appearance. Fortunately, crown rot affected carrots are obvious to the naked eye and can be sorted out in the grading line after washing.

Smooth crown rot

Some crops that have a high incidence of smooth crown rot also have high levels of *S. sclerotiorum and R. solani* on plant foliage. In these crops, smooth crown lesions appear to develop as a result of the spread of infected senescing or dead plant matter, spread directly from lower stem infections, and direct mycelium infection from sclerotia.

Fusarium spp. were always isolated from crown rot lesions in this project, even when initial infection by *R. solani* and *S. sclerotiorum* was observed. It is unclear whether the *Fusarium* species isolated are crown rot pathogens, interact with other pathogens, or are secondary invaders.

In 1999, due to wet and warm conditions following a period of frequent rainfall, stem rot and crown rot due to *R. solani* or *S. sclerotiorum* was found in 20 crops out of the 26 examined. *R. solani* and *S. sclerotiorum* were isolated from newly formed crown rot lesions. In a field test, carrot crowns inoculated with agar blocks of *R. solani* showed initial symptoms of smooth crown rot (Section 3).

It was noted that a high incidence of smooth crown rot was not always related to the presence of *R. solani* or *S. sclerotiorum* foliage infections. This indicates that, apart from *R. solani* or *S. sclerotiorum*, smooth crown rot may be caused by other pathogens or groups of pathogens in some crops.

Carrots sown in low-lying areas and ground with poor drainage are prone to severe infection by *S. sclerotiorum*. High levels of trash on the ground also promote *Sclerotinia* infection. This includes trash that originates not only from a previous crop, but also from plant residues of herbicide sprayed weeds that become hosts for *Sclerotinia* infections.

Corky crown rot

Fusarium spp. were always isolated from corky crown rot lesions in this project, and *Streptomyces* has been isolated from some corky crown rot lesions. A pot trial conducted as part of this project (Section 4.3) showed that *Streptomyces scabies* isolated from potato common scab lesions caused corky lesions on carrot crowns.

Corky crown rot incidence tends to be high in crops that are sown after potatoes, or in fields where volunteer potatoes are a problem. However, not all carrot crops sown after potatoes, have high crown rot incidence. There was also no correlation between the incidence of scab and corky crown rot ($p>0.05$). Corky crown rot appears to be common in well-drained soil. High incidence of corky crown rot could not be associated to other field conditions or cultural practices.

Fusarium species isolated from crown rots

Fusarium spores were often found on crown rot lesions, and *Fusarium* spp. was always isolated from surface sterilised tissues from crown rot lesions. Various species of *Fusarium* have been isolated from specimens collected from different properties. *Fusarium* species isolated included *F. acuminatum, F. avenaceum, F. compactum, F. culmorum, F. reticulatum, F. semitectum* and *F. solani*. *Fusarium* is ubiquitous in soil and there appears to be no consistency in the type of the fungal species found in association with crown rot disease.

Crown and side rot have been attributed to *Fusarium* dry rot caused by various species of *Fusarium,* and different species cause different disease symptoms (Howard et *al* 1994). A humid and warm environment, at 16 to 20°C appears to encourage disease symptoms (Howard et *al* 1994). In a pathogenicity test conducted, three species of *Fusarium* frequently isolated from crown rot of carrots, *F. culmorum, F. compactum and F. semitectum,* caused crown rot lesions on inoculated carrot crowns (Section 4.2). The incidence and severity of the lesions varied according to presence or absence of physical wounding and species of *Fusarium*. Studies conducted elsewhere indicate that pathogenic *Fusarium* spp. causes crown rot of carrots (Howard et *al* 1994).

4.2. Pathogenicity test of Fusarium *spp. frequently isolated from crown rot*

Introduction

This study was conducted to determine the pathogenicity of three species of *Fusarium* that were frequently isolated from crown rot lesions.

Materials & Methods

The Fusarium species, *F. compactum, F. culmorum and F. semitectum*, were frequently isolated from crown rot lesions in 1995 and 1996. *Gliocladium roseum* was also included in the study as it was also frequently isolated from crown rot lesions. Dr. Micheal Priest, a fungal taxonomist, identified the fungal species.

Fresh carrots (cv. Kuroda, 15 week old) were used in this study. The carrots were washed thoroughly and the carrot crowns were cut across with a clean knife (from stem base to about 3cm below the crown). The cut crowns were surface sterilised in 1% sodium hypochlorite solution for 5 minutes, rinsed twice in tap water, dried on absorbent paper and then placed on a tray lined with a layer of moist blotting paper. One side of the crown top was physically damaged with a sharp pin probe (5 holes on each carrot top; hole size: 0.5mm wide x 1.5mm deep).

The fungi were grown for 14 days on potato dextrose agar (PDA). Fungal spore suspensions were then prepared by scraping each fungal colony on PDA, into 15ml of sterile water and mixing vigorously.

Carrot roots with both damaged and undamaged sides were inoculated with spore suspensions of the selected fungi. Only sterile water was used for the control treatment. The trays containing the inoculated carrot tops were placed in clean plastic bags, and sealed to maintain a moist atmosphere.

The carrot roots were assessed for incidence and severity of necrosis or crown rot at 7 and 14 days after incubation at 17-20°C. The disease severity was rated according to the following scale:

0 = no necrosis
1 = necrotic area 1mm or less
2 = necrotic area between 1 to 2mm
3 = necrotic area between 2 to 5mm
4 = necrotic area greater than 5mm

The disease rating scale was then converted to a crown rot disease indices according to the methodology of McKinney (1923) using the formula below:

Crown rot disease index = Mean of disease severity scale * 100 /4

Results & Discussion

Smooth crown rot developed on the crowns that were inoculated with *F. compactum, F. culmorum* and *F. semitectum*. The incidence and severity of crown rot varied according to the species of *Fusarium* used. *F. culmorum* caused the highest disease incidence and severity, followed by *F. compactum*.

G. roseum did not cause crown rot. This fungus is widely regarded as a non-pathogen and is believed to be a potential biocontrol agent against fungal pathogens.

Figure 4.2.1: The incidence of crown rot caused by *Fusarium* spp. and *G. roseum* on damaged and undamaged sides of carrots, at 7 days after incubation

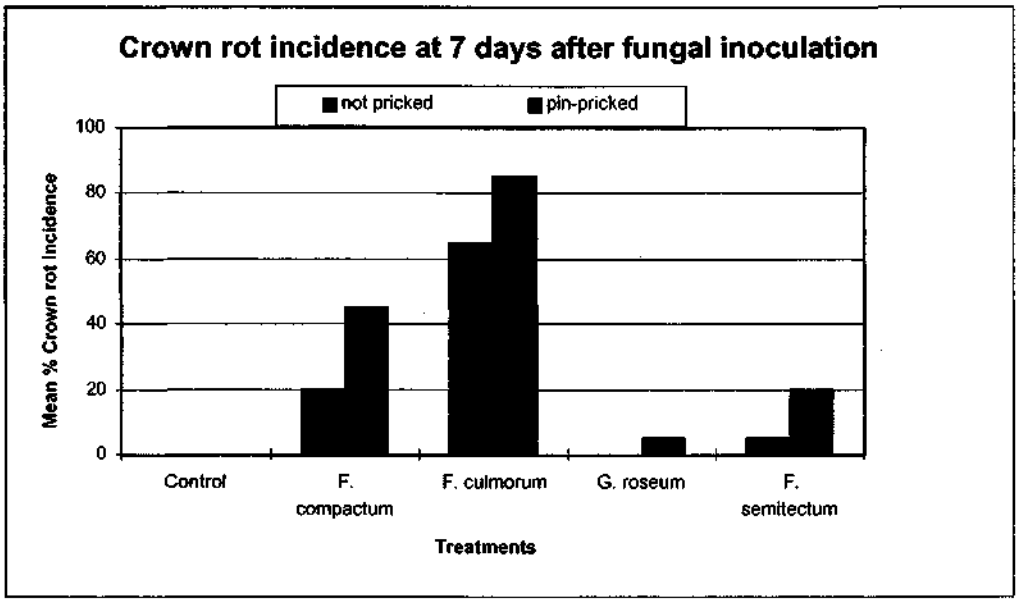


Figure 4.2.2: The incidence of crown rot caused by *Fusarium* spp. and *G. roseum* on damaged and undamaged sides of carrots, at 14 days after incubation

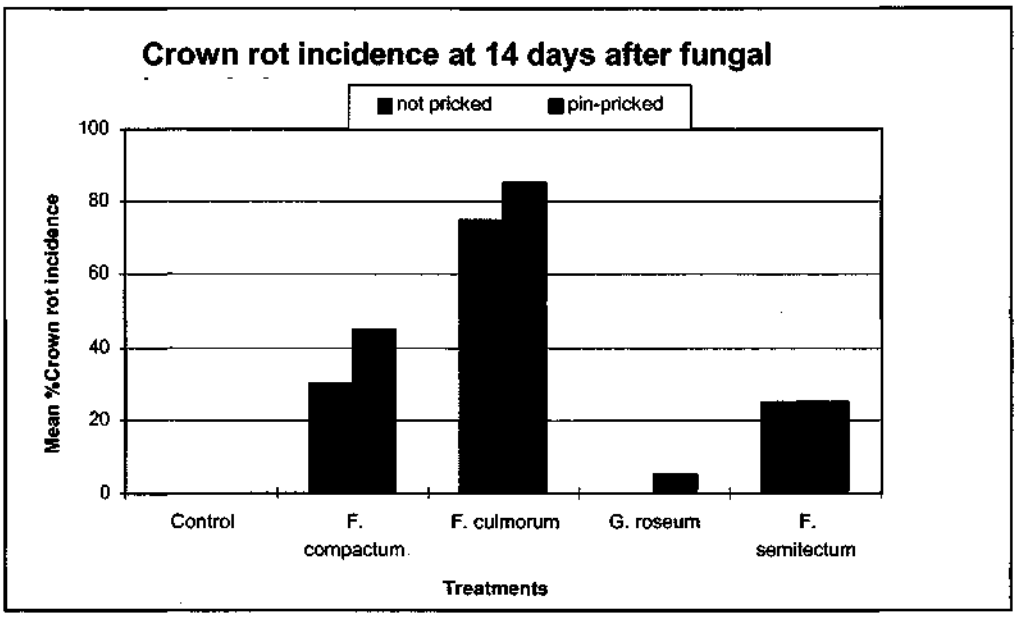


Figure 4.2.3: Crown rot severity caused by *Fusarium* spp. and *G. roseum* on damaged and undamaged sides of carrots, at 7 days after incubation

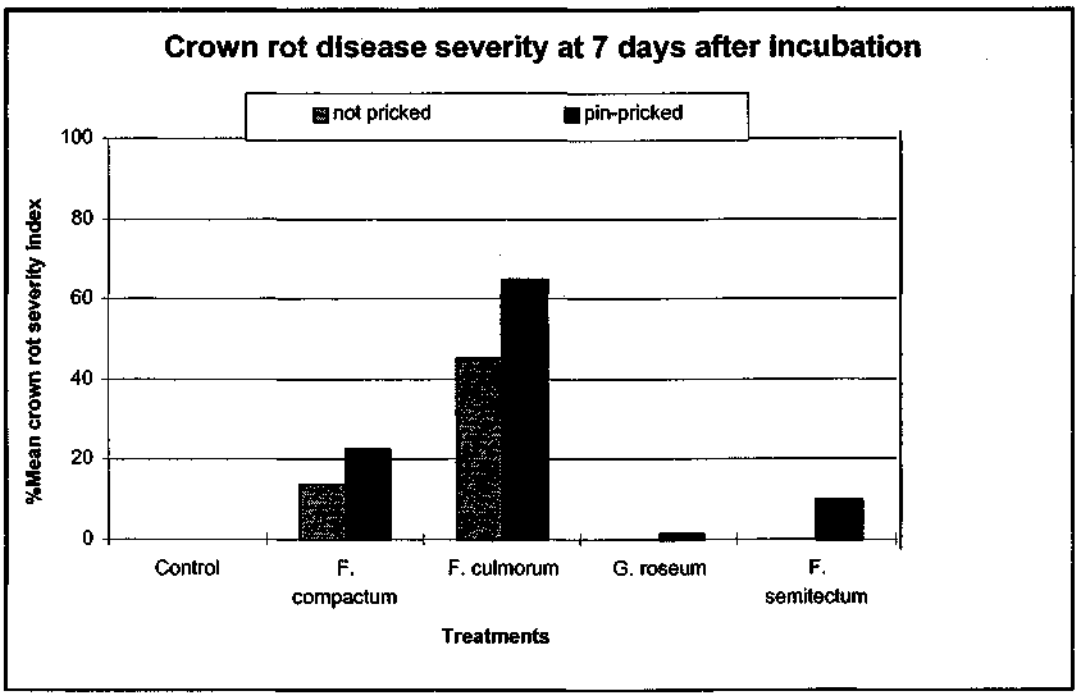
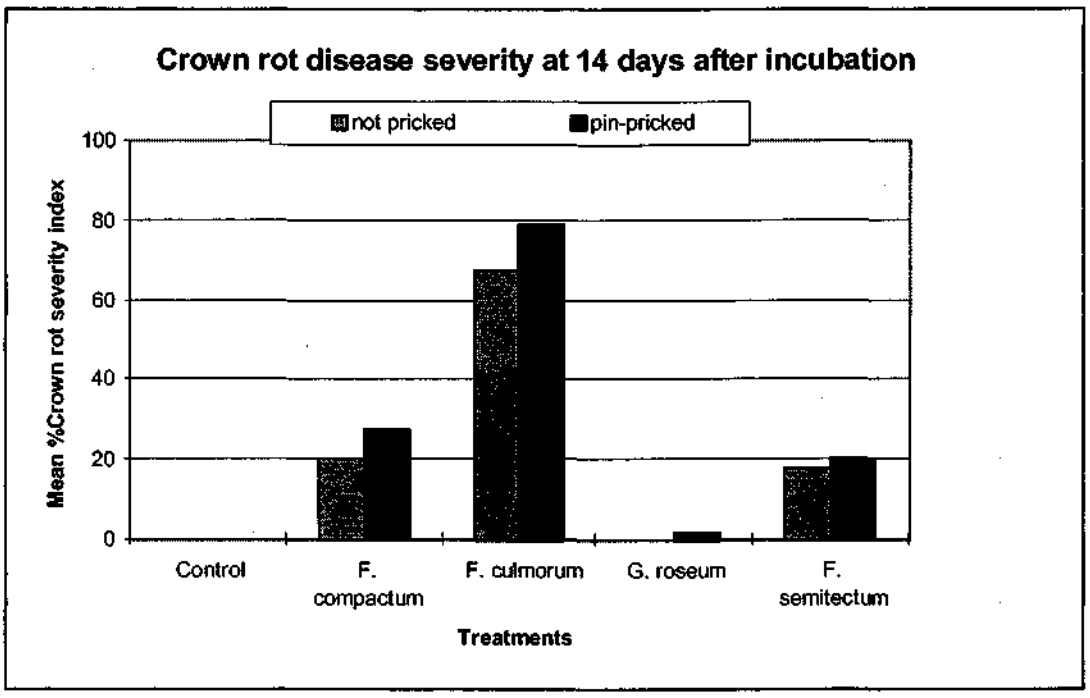


Figure 4.2.4: Crown rot severity caused by *Fusarium* spp. and *G. roseum* on damaged and undamaged sides of carrots, at 14 days after incubation



4.3. An investigation of the effects of Fusarium *spp. on crown rot disease*

Introduction

Two field trials were set up at Forth in the 1996/97 season to determine the ability of *Fusarium* spp. isolated from crown rot lesions on carrots to cause crown rot under field conditions.

Materials & Methods

The two trials were set up in the same area, adjacent to one another, and sown with the two main carrot varieties (HiPak and Kuroda). Trial design used was a complete randomised block, with 5 replicates per treatment, and each replicate plot was 4m x 1 bed.

Three species of *Fusarium* used in this trial were *F. compactum, F. culmorum and F. semitectum.* The pathogenicity of the three *Fusarium* isolates and the combined *Fusarium* species were examined under field conditions on two varieties of carrots (HiPak & Kuroda), in a factorial design on damaged versus undamaged plants. The two varieties were planted on separate blocks.

Fusarium inoculum was grown on autoclaved millet seeds for 4 weeks at approximately 20°C. Prior to the sowing of carrot seeds, the *Fusarium* inoculum was broadcast onto field plots and then mixed into the top 5cm soil with a rake.

Both carrot varieties were sown on 19 November 1996. Hipak carrot seed was sown in double plant rows on 0.8m narrow soil beds, while Kuroda was sown in three double plant rows on 1.2m wide soil beds.

Wounding or damage of 30 carrots in the appropriate treatment plots was conducted by either removing the two oldest leaves (leaf damage) or superficial scratching of carrot crowns with a blunt probe (crown damage), when seedlings had reached the 4-5th leaf stage.

Assessment for seedling emergence was made at 48 days after planting (6-Jan-97). Twenty carrots per plot were then sampled at 2-3 week intervals for crown rot incidence from 29-Jan-97 until final harvest, at 139 days after sowing for HiPak carrots and 143 days after sowing for Kuroda carrots.

Results and Discussions

The lack of crown rot disease in the two trials (Tables 4.3.1 & 4.3.2) is likely to be related to field conditions, which were not conducive to disease. The trial site was located on a high, exposed ground, and the top soil in both the carrot beds and moulds tended to be dry most of the time. Carrot density and shoot growth were also generally low, possibly due to dry conditions and irregular seedling emergence.

Table 4.3.1: Pathogenicity of *Fusarium* spp. on HiPak carrots in Trial 1

	No. seedling emerged in 1/2m row	%Carrots with crown rot*				
Treatment	06-Jan-97 48	29-Jan-97 71	12-Feb-97 85	26-Feb-97 99	19-Mar-97 120	10-Apr-97 139
non-leaf damaged						
F. compactum	48	0	0	n/a**	0	0
F. culmorum	47	0	0	n/a	0	0
F. semitectum	52	0	0	n/a	0	0
Combined spp	50	0	n/a	n/a	0	0.5
Control	51	0	0	0	0	0
leaf damaged						
F. compactum	56	0	0	n/a	n/a	0
F. culmorum	39	0	0	0	n/a	0
F. semitectum	56	0	0	n/a	n/a	0
Combined spp	61	0	0	1	n/a	0.5
Control	66	0	0	0	n/a	0
crown damaged						
Combined spp	n/a	n/a	0	0	n/a	1

* %Carrots with crown rot during the growth period and at harvest time; **data not availbale as no assessment was made

Under relatively dry conditions, there were negligible levels of crown rot disease in the two trials (Tables 4.3.1 & 4.3.2). Physical damage on the leaf or crown damage did not increase disease incidence in either of the varieties tested. Superficial scratch marks made on the crown developed into deep wounds, but no evidence of rots was observed. Similarly with leaf damage, the damaged area appeared to heal with no sign of stem rot.

Table 4.3.2: Pathogenicity of *Fusarium* spp. on Kuroda carrots in Trial 2

	No. seedling emerged in 1/2m row	%Carrots with crown rot*				
Treatment	06-Jan-97 48	05-Feb-97 78	19-Feb-97 92	05-Mar-97 106	26-Mar-97 127	14-Apr-97 143
non-leaf damaged						
F. compactum	36	0	0	n/a	n/a	0
F. culmorum	29	0	0	n/a	0	0
F. semitectum	23	0	0	n/a	n/a	0
Combined spp	34	0	0	n/a	0	0
Control	30	0	0	n/a	0	0
leaf damaged						
F. compactum	n/a	0	0	2	n/a	0
F. culmorum	n/a	0	0	0	n/a	0
F. semitectum	n/a	0	0	0	n/a	0
Combined spp	n/a	0	0	0	n/a	0
Control	n/a	0	0	2	n/a	0
crown damaged						
Combined spp	n/a	n/a	n/a	n/a	n/a	0

* %Carrots with crown rot during the growth period and at harvest

4.4. Relationships between Streptomyces scabies, Fusarium culmorum *and crown rot*

Introduction

This study was conducted to investigate the association of *S. sclerotiorum, S. scabies* and *F. culmorum,* with corky and smooth crown rot incidence.

Materials & Methods

Two pot trials were conducted in this study. Each pot (10.5L capacity) was first filled with 9.5L of pasteurised potting mix (containing complete fertiliser and lime super), and then covered with 2L of topsoil inoculated with the appropriate pathogen.

The carrot variety, Kuroda, was sown with 12 seeds per pot, at 2cm depth, in a free-draining pot. After seedling emergence, the number of plants per pot was reduced to 8 plants per pot. The pots were maintained in an open environment subject to rainfall or irrigated when required. Trial design used in both trials was a complete randomised block, with 10 replicates in Trial 1, and 5 replicates in Trial 2.

F. culmorum and *S. sclerotiorum* stock inoculum for the trials were produced on autoclaved millet seeds. *S. scabies* inoculum was produced on sterile vermiculite, sucrose and yeast extract mixture. Both inocula were incubated at 25°C for 14 days before use. *F. culmorum* and *S. sclerotiorum* cultures were isolated from carrot, while the *S. scabies* was from common scab lesion on potato.

Pot Trial 1 was conducted to investigate the relationship between crown rot, *F. culmorum* and *S. scabies* (Table 4.4.1). In treatments with *F. culmorum* or *S. scabies*, stock inoculum was mixed into the topsoil per pot. At 135 days after sowing, the scab and corky rots were assessed. The corky lesions due to scab and corky rot are indistinguishable; hence both types of lesions were classified as corky lesions.

Pot Trial 2 was conducted to investigate the relationship between crown rot, *F. culmorum* and *S. sclerotiorum* (Table 4.4.2). The topsoil for the appropriate treatment was inoculated with *F. culmorum*. At 98 days after sowing, *S. sclerotiorum* mycelia suspension was sprayed onto carrot foliage until run-off (20ml per pot) in the appropriate treatments. The mycelia suspension was prepared by macerating the fungus on potato dextrose agar (2 plate cultures) in 200ml sterile water. At 149 days after sowing, carrots were examined for either corky or smooth crown or side rots.

Results & Discussion

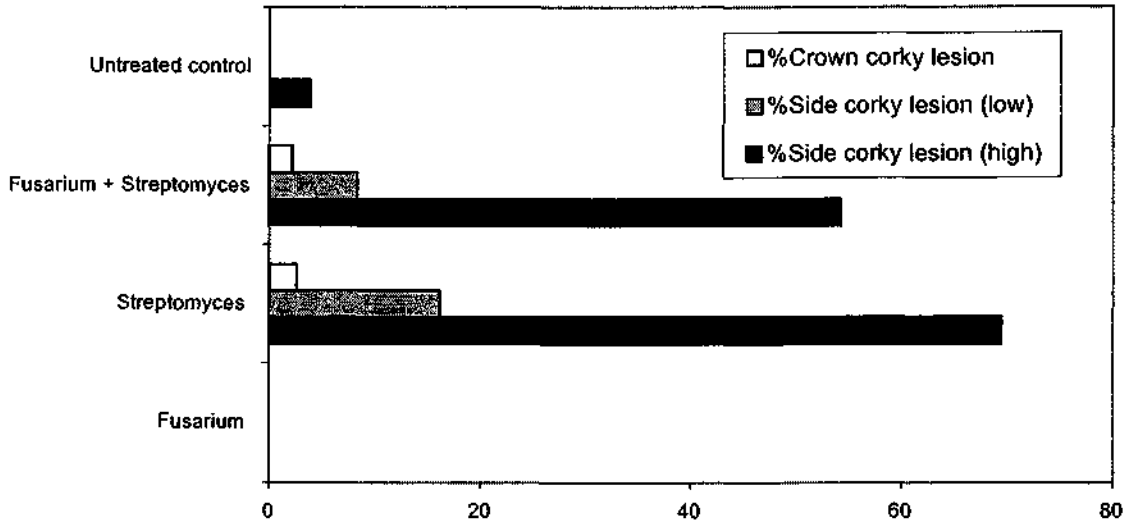
In Trial 1, *S. scabies* isolated from common scab lesions on potato caused corky lesions that resembled scab on carrots (Table 4.4.1, Figure 4.4.1). Both *S. scabies* and *F. culmorum* did not cause smooth crown rot. Corky lesions that occurred on the carrot crowns resembled corky crown rot found in commercial crops.

Table 4.4.1: The effects of *Fusarium culmorum* and *Streptomyces scabies* on the incidence of corky and smooth rot of carrots

No.	Treatment	Inoculum Rate	% Corky lesions	% Smooth crown rot
1	*Fusarium*	50g	0 a	0
2	*Streptomyces*	50g	88 b	0
3	*Fusarium + Streptomyces*	25g + 25g	65 b	1
4	Untreated control	N/a	4 a	0

* Within the same column, means followed by the same letter are not significantly different according to Duncan multiple range test at the 95% confidence level.

Figure 4.4.1: The effects of *Fusarium* and *Streptomyces* species, alone and when combined, on corky lesion incidence according to their location



low = corky lesion on lower half of carrot; high = corky lesion on upper half of carrot

In Trial 2, no significant levels of smooth rot or corky rot were noted (Table 4.4.2). Although the plants in the pots were irrigated frequently, the plant foliage mostly remained dry due to hot, dry and windy conditions. The wet conditions believed to be necessary for *Sclerotinia* and *Fusarium* infection could not be attained in this study.

Table 4.4.2: The effects of *Fusarium culmorum* and *Sclerotinia sclerotiorum* on smooth and corky rot

No.	Treatment	Inoculum Rate	% Smooth crown rot*	% Corky crown rot*
1	*F. culmorum*	50g	0	0
2	*S. sclerotiorum*	20ml	2.5	0
3	*F. culmorum + S. sclerotiorum*	50g + 20ml	0	0
4	Untreated control	N/a	0	0

*No significant differences between treatments according to analysis of variance (P.0.005).

4.5. Evaluation of fungicides for Sclerotinia & Rhizoctonia *control on carrots*

Introduction

Field observations indicated that infections by *S. sclerotiorum* and *R. solani* may cause crown rot disease on carrots. Two field trials were, therefore, conducted to evaluate products that may be used to prevent crown rot caused by these pathogens.

Typically, no foliar fungicide is applied with current production, and reduced chemical application is also encouraged for market advantage. There is also a high cost associated with multiple fungicide applications. Growers are, therefore, unlikely to apply fungicides to prevent disease. Instead, growers are more likely to apply fungicides at the onset of infection to prevent a severe loss in yield. The following trials were, therefore, designed to evaluate the efficacies of systemic fungicide products, to be applied at the onset of *Rhizoctonia* and *Sclerotinia* infections for curative activities, preventing the disease from spreading, and minimising losses.

Materials & Methods

Trial Details

The two field trials were conducted on ferrosol soil, within a commercial crop (cv. Stephano) at Forth. The trial design was a complete randomised block, with 4 replicates on wide beds (6 plant rows) x 6m. The crop was sown on 28/12/1999 and harvested on 10/05/00.

Disease inoculation

In **Trial 1**, *R. solani* inoculum was applied in the 2 middle plant rows along all the treatment plots. In **Trial 2**, *S. sclerotiorum* inoculum was applied instead. The appropriate fungal inoculum was applied at 79 days after sowing, when the average carrot diameter was 15mm and spaces between plant rows were beginning to be covered by plant foliage. Fungal inoculum was prepared by culturing the appropriate fungus on autoclaved barley seeds.

Product details

Product	Active Ingredient (a.i.)	Rate of a.i.	Formulation
Amistar	azoxystrobin	500g/kg	Water Dispersable Granules
Aliette	fosetyl	800g/kg	Water Dispersable Granules
Bavistin	carbendazim	500g/L	Suspension Concentrate
Benlate	benomyl	500g/kg	Wettable Powder
Folicur	tebuconazole	430g/L	Suspension Concentrate
Fortress	procymidone	500g/L	Suspension Concentrate
Fungaflor	Imazalil	750g/kg	Wettable Soluble Powder

Application Methods

Two fungicide-sprays were applied in all fungicide treatments, with the first spray at 6 weeks before maturity, then a second spray 10 days later. Fungicide sprays were applied using a knapsack precision sprayer fitted with 1.5 metre boom, and fan-jet air-induction spray nozzles AI11005 at 300kPa and 443.3 L/ha. At maturity, 30 carrot plants in the centre of each plot were harvested, washed and assessed for disease.

Treatment details for Trial 1 & 2.

No.	Fungicide	Fungicide Rates	Application Schedule
1	Amistar	0.4 kg/ha	1st spray at 24th March 2000, and 2nd spray at 4th April 2000.
2	Aliette	2.0 kg/ha	
3	Bavistin	1.5 L/ha	
4	Benlate	2.0 kg/ha	
5	Folicur	0.5 L/ha	
6	Fortress	1.5 L/ha	
7	Fungaflor	0.5 kg/ha	
8	Untreated control		N/a

Results and Discussion

Crown rot disease incidence in both trial areas was low, and no significant differences could be found between treatments in the trials (Tables 4.5.1 & 4.5.2). No foliage infections by *R. solani* or *S. sclerotiorum* were noted in the trials. This may be due to the lack of high humidity and wet soil conditions, which are necessary for *R. solani* and *S. sclerotiorum* infection. At the beginning to the end of the trials, the topsoil (5cm deep) and plant foliage were usually dry in both trial areas.

The incidence of forked carrots was high in the trials. None of the fungicides applied were expected to have any effect on the incidence of forked carrots, as the treatments were applied at 79 days after sowing. The forking of carrots occurred much earlier, at the seedling stage. No significant differences were found in the incidence of forked carrots between treatments in both trials (Tables 4.5.1 & 4.5.2).

Tiger stripe was also noted on carrots in the field, albeit at a low level, where water drained into low-lying areas and deeper soil beneath the topsoil tended to stay wet for long periods after rainfall and irrigation. In Trial 1, carrots with tiger stripe were found in replicate 1 plots, which were situated in a low-lying area of the paddock (Table 4.5.1). In Trial 2, tiger stripe affected carrots were found only in one replicate plot in Treatment 4 (Table 4.5.2).

4. Crown rot disease (Cont.)

Table 4.5.1: Effects of fungicide treatments on carrot diseases in an area inoculated with *S. sclerotiorum* in Trial 1

No.	Fungicide	Fungicide Rates	% Healthy*	% Crown rot*	% Tiger stripe*	% Forking*
1	Amistar	0.4 kg/ha	80	2	0	12
2	Aliette	2.0 kg/ha	77	0	3	19
3	Bavistin	1.5 L/ha	80	0	1	14
4	Benlate	2.0 kg/ha	76	4	2	17
5	Folicur	0.5 L/ha	80	3	2	11
6	Fortress	1.5 L/ha	81	2	0	16
7	Fungaflor	0.5 kg/ha	80	3	0	13
8	Untreated control		79	3	1	14

* All mean values not significantly different at the 5% level according to analysis of variance.

Table 4.5.2: Effects of fungicide treatments on carrot diseases in an area inoculated with *R. solani* in Trial 2

No.	Fungicide	Fungicide Rates	% Healthy*	% Crown rot*	% Tiger stripe*	% Forking*
1	Amistar	0.4 kg/ha	73	2	0	18
2	Aliette	2.0 kg/ha	56	4	0	29
3	Bavistin	1.5 L/ha	63	6	0	21
4	Benlate	2.0 kg/ha	71	3	1	13
5	Folicur	0.5 L/ha	63	0	0	15
6	Fortress	1.5 L/ha	76	2	0	9
7	Fungaflor	0.5 kg/ha	54	3	0	22
8	Untreated control		68	6	0	8

* All mean values not significantly different at the 5% level according to analysis of variance.

Conclusions

There are two distinct types of crown rot symptoms on carrots, corky crown rot and smooth crown rot.

Three types of organisms found in association with smooth crown rot were *Fusarium* spp., *R. solani* and *S. sclerotiorum*. *Fusarium* spp. was always isolated from smooth crown rot, whereas in some crops, *R. solani* and *S. sclerotiorum* were isolated from affected crowns, as well as being observed on carrot foliage.

However, no single organism, including *R. solani* and *S. sclerotiorum*, was always related to high smooth or corky crown rot incidence. In field survey studies, the lack of correlation between any particular field condition and crown rot incidence may be due to the different type or isolate of pathogen involved.

Smooth crown rot did not develop in inoculated pot and field trials conducted in this project. Foliage infection by *R. solani* or *S. sclerotiorum* was also absent in the trials. In the field survey studies, in crops where crown rot was caused by *R. solani and S. sclerotiorum*, the affected carrots also had foliage infections. This indicates that, in the trials conducted, these organisms were not tested under an environment that is conducive to crown rot development. There were difficulties in maintaining high soil moisture due to various factors in the trials (an unsuitable field site, low plant density, the use of free draining soil and pot, and constant dry and windy conditions that prevailed during the trials).

Two types of organisms appear to be associated with corky crown rot, *Fusarium* spp. and *Streptomyces* spp. *S. scabies* (isolated from potato) was shown in a pot trial to cause corky lesions on carrots under relatively dry soil conditions. Corky lesions that occurred on the carrot crowns resembled corky crown rot observed in commercial crops. The role of *Fusarium* spp. in corky rot remains unclear. It is possible that the soil conditions in the study were not conducive to *Fusarium* infection.

In this project, even though no crown rot development occurred in pot and field trials inoculated with *Fusarium* spp., *R. solani* and *S. sclerotiorum*, their role in crown rot development could not be discounted. Field observations and fungal isolations conducted in this project indicate that in some crops, *R. solani* and S. *sclerotiorum* infections of lower stems spread directly onto the carrot crown, causing smooth crown lesions. In a pathogenicity test conducted in the laboratory, three species of *Fusarium* frequently isolated from crown rot of carrots, *F. culmorum, F. compactum and F. semitectum,* caused crown rot lesions on inoculated carrot crowns. Studies conducted elsewhere indicate that pathogenic *Fusarium* species (Howard et *al* 1994) and *R. solani* (Mildenhall & Williams 1973, Grisham & Anderson 1983) cause crown rot.

4. Crown rot disease (Cont.)

Different anastomosis groups of *R. solani* have also been found to show differences in pathogenicity on carrots (Grisham & Anderson 1983). They also show that isolates belonging to one anastomosis group (AG2-2 isolated from carrots) produce different symptoms on carrots of different growth stage. Pre-emergence and post-emergence damping-off occurred in a greenhouse study for 30 days after planting, whereas plants inoculated at 30-60 days after sowing developed crown rot symptoms. Under field conditions, *Rhizoctonia* infected plants of a similar age also developed crown rot. Damping-off and crown rot due to *R. solani* are most severe at high soil temperatures of 20 to 28°C, with little infection at 16°C (Mildenhall & Williams 1973).

Technology Transfer

- A carrot disease guide - "Description of Typical Carrot Diseases and Disorders in Tasmania". Compiled by Hoong Pung and Pam Cox, and printed in 1998.

- Pung, H. and Cox, P., 1999. The development of crown rot disease on carrots. Proceedings of the Australian Plant Pathology Societies 12th Biennial Conference, September 27-30, p 283.

- Pung, H., 1999. Correct identification of carrot diseases essential for disease control. Good Fruit and Vegetables, October 1999.

- A flier on "Violet root rot of carrots". Compiled by Pung, H. and Cheah, L. H, and printed in July 2000.

- Project findings were presented at Tasmanian vegetable extension days held at Burnie on 28th May 1998, and at Ulverstone on 27th July 1999 and 10th August 2000. These were well attended by Tasmanian growers, industry representatives and researchers.

- A carrot growers' day was held in October 1998 at Bellfield, Tasmania, where copies of the carrot disease booklet were distributed to growers, field officers, and processing company representatives.

- Copies of the carrot disease booklet have also been provided to carrot growers in Victoria, New South Wales, Queensland, South Australia and Western Australia.

- For the duration of this project, Tasmanian carrot producers and packers have been visited by research staff for training in identification of various diseases and their causes.

- Meetings were held with growers, agronomists and carrot processor representatives in Tasmania on June 3 and 9, 1999, to notify them of the new carrot disease, violet root rot.

- Project findings were presented in a poster at the "Carrot conference" on 24-27 October 2000, Perth, Western Australia.

- Copies of a carrot disease poster that was presented at the "Carrot conference", were supplied to the major fresh market carrot producers in Tasmania.

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Appendices