

Identification of the extent and cause of parsnip canker

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The extent and cause of parsnip canker

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Purpose of project:

This report details the outcomes of a 24-month project investigating parsnip canker. This project carried out surveys to investigate both the cause and extent of parsnip canker in Victoria, Western Australia and Tasmania.

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Media Summary

Ground breaking research into parsnip canker in Australia

Scientists have taken the first step in determining the cause and extent of parsnip canker in Australian parsnip production. Parsnip canker can cause crop losses up to 80 %. Parsnip is a long-term crop, harvested after 5–7 months. It is a difficult crop to grow and there are few major growers in Australia. Victoria produces over 80 % of the total parsnip production in Australia.

Surveys of parsnip canker at harvest in 2006 and 2007 showed that canker levels peaked between September and November, on crops sown in February to March. The peak average losses due to canker for this period were 45 % for 2006 and 23 % for 2007. This represents a potential loss of A\$3 million for the parsnip industry over the two years.

Several fungi were isolated from cankers and there was strong evidence that the debilitating disease is caused by more than one organism. The fungi isolated included *Itersonilia perplexans*, the main cause of this disease overseas, and fungi such as *Cylindrocarpon* spp., *Fusarium* spp., *Mycocentrospora acerina*, *Pythium* spp. and *Rhizoctonia* spp.

Observations in the field, as well as laboratory studies, indicated that superficial damage to the roots predisposed parsnips to fungal attack and canker.

Symptoms of parsnip canker are large black lesions on mature parsnip roots, mostly on the shoulder or crown, that can spread to other sections of the root and in extreme cases, cover the entire root, making the product unusable.

Information resulting from this research was presented in conference posters, at Steering Committee meetings and field days.

This research was led by scientists at the Department of Primary Industries Victoria Knoxfield Centre. The project was facilitated by Horticulture Australia Limited (HAL) in partnership with Federation of Potato and Vegetable Growers Australia Limited (AUSVEG) and was funded by the National Vegetable Levy. The Australian Government provides matched funding for all of Horticultural Australia's Research and Development activities. The researchers gratefully acknowledge the financial support of the Department of Primary Industries through Primary Industries Research Victoria.

Technical summary

Little or no research into parsnip canker has been undertaken in Australia despite the fact that growers have reported persistent crop losses in spring-harvested crops of up to 80 % for 40 years.

This 24 month scoping study:

- Surveyed parsnip crops in the major cropping regions of Victoria, Tasmania and Western Australia to identify the extent of parsnip canker affecting production;
- Identified the causes of parsnip canker in Victorian crops by conducting pathogenicity tests (Koch's postulates) on fungi isolated from cankers;
- Used field trials to confirm pathogen trials;
- Determined that root damage predisposed parsnip roots to canker.

Systematic surveys involving four growers in Victoria, as well as one in Tasmania and two in Western Australia showed that parsnip canker was a major problem in Australia. In Victoria, where 80 % of Australian parsnips are grown, parsnip canker accounted for losses of up to 45 % in spring-harvested crops, and in one case, losses were over 85 %. This represented a potential loss of A\$3 million for the parsnip industry over two years.

Pathogenicity tests were conducted on fungi that were consistently isolated from diseased parsnip roots in Victoria. The confirmed overseas pathogens *Itersonilia perplexans*, *Cylindrocarpon* spp., *Mycocentrospora acerina* and *Phoma* spp. all caused canker-like symptoms on fresh parsnip roots. Other fungi such as *Acremonium* spp., *Fusarium* spp., *Microdochium* spp., *Pythium* spp. and *Rhizoctonia* spp. caused similar lesions. Pathogen trials also established that root damage predisposed parsnip to pathogen attack.

Trials involving growers' own seed and a commercial variety, Tusk, showed that there was potential to alleviate canker levels using resistant cultivars. The use of Tusk reduced canker levels by 45 % compared to some of the growers' own seed stocks.

The effectiveness of metalaxyl-m in one out of three trials, in reducing canker incidence by 77 %, reinforced the notion of root damage contributing to parsnip canker. In a second trial, metalaxyl-m reduced canker incidence by 34 %, although, in this case, it was not significantly different from the control. These results, as well as the pathogen tests, indicate that oomycete fungi such as *Pythium* spp. may have a role in predisposing parsnip roots to canker.

Future directions

Our research has increased knowledge of parsnip canker but it has revealed how little we know about this disease complex.

Future investigations need to determine the:

- Predisposing nature of *Pythium* species and other pathogenic fungi;
- Biological succession of fungi on parsnip root to determine the time of initial infection, by microbiological sampling;
- Influence of predisposing abiotic factors such as soil moisture, temperature, pH, nutrients and fertilization on canker development;
- Initiation of field infections, using targeted fungicides;
- Cultural, biological and alternative soft chemical controls.

Chapter 1

A brief history of parsnip canker

1.1 Parsnip canker symptoms

Cankers primarily form on the crown and shoulder of roots, although these lesions can extend along the length of the root (Cerkauskas 2002). Four types of parsnip canker were described by Channon (1965). Overseas, the disease has been attributed to *Itersonilia perplexans*, *Phoma* spp., *M. acerina* and *S. scabies* in the UK (Channon 1965, Fox 2002, Jones 1953). In Canada it was attributed to *P. complanata* (Cerkauskas 1985), while in the USA *Itersonilia* was pathogenic (Wilkinson 1952) and in Scotland *C. destructans* was responsible for canker (Channon and Thomson 1981). It is not considered to be associated with bacteria (Green and Hewlett 1950), but has been associated with the carrot fly larvae *Psila rosae* in the UK (Stone 1954). Fortunately carrot flies are not present in Australia (see 1.3.5).

1.2 *Itersonilia*

I. perplexans is thought to be the main cause of parsnip canker, and as a consequence, most research into parsnip canker and its control has targeted this organism. It is a basidiomycete which can infect roots, leaves, inflorescences and seed (Channon 1969), with an optimal temperature for growth of 20 °C. According to the Commonwealth Mycological Institute, *I. perplexans* is endemic to Canada, North America, England, Italy, Australia and New Zealand (Anon 1978). Sowell and Korf (1960) also obtained isolates from the Netherlands.

1.2.1 Initial studies

The type species of *Itersonilia*, *I. perplexans* Derx was first isolated in 1948 (Derx 1948). Not long after, *Itersonilia* sp. was isolated from parsnip crops in the United States, and both cankers and ‘typical’ leaf spots were induced on parsnips from pure isolates (Wilkinson 1952). Sowell (1953) reported that germinating ballistospores of *Itersonilia* sp. were responsible for both the leaf spot and the canker. Channon (1956) observed similar symptoms in Great Britain and obtained pure cultures from cankers in 1954. Only one of these isolates produced cankers in both unwounded and wounded parsnip tissue. The isolate was subsequently identified as *I. perplexans*, the same species that had been isolated previously (Derx 1948). Similarly, this isolate also produced leaf lesions on young parsnip plants. The leaf lesions were fairly distinctive, with a necrotic centre surrounded by a light-green halo.

A study of 6 isolates, from parsnip around the world, as well as 43 other local isolates, determined that all isolates were a single species, *I. perplexans*, and all were proven to be pathogenic on parsnip (Sowell and Korf 1960).

1.2.2 Pathogenicity

Channon initially isolated *Itersonilia* from cankers in parsnip in Great Britain (Channon 1956) and wrote a series of papers on his studies on parsnip canker (Channon 1963abc, 1964, 1965). He described two kinds of canker in his initial paper, a black canker, caused by *Itersonilia*, *Phoma* or both, and an orange-brown canker, with an unknown cause (Channon 1963a). Over 60% of black cankers yielded pathogenic *Itersonilia*, but only 16% yielded pathogenic *Phoma*. There was some evidence of a ‘consortium’ of fungi causing black cankers, since isolates from brown cankers failed to induce similar symptoms, but *Itersonilia* isolates from the same brown cankers produced ‘typical’ black cankers. These ‘brown’ cankers appeared to be associated with growth splitting. *Itersonilia* isolates from diseased

parsnip roots and leaves were to be pathogenic on parsnips, but similar isolates from chrysanthemums were non-pathogenic on parsnip, and vice-versa. On the basis of profuse chlamyospore production, i.e. the resting stage of the fungus, it was decided that these pathogenic *Itersonilia* isolates were different from the original *I. perplexans* (Derx 1948), and a new species was named that was exclusively pathogenic on parsnip, *I. pastinacae*. Channon also noted that wounding of parsnip roots prior to inoculation with *I. pastinacae* resulted in more rapid and larger cankers (Channon 1963a).

1.2.3 Epidemiology

In his second paper, Channon reported the seasonal presence of ballistospores, which appeared on leaves of parsnip, and were the presumptive cause of canker in parsnip roots (Channon 1963b). Ballistospores were present in parsnip crops in late summer and their presence peaked in autumn. Numbers were higher in the morning, and appeared to be associated with dew periods. Drier conditions in subsequent years resulted in fewer *Itersonilia* spores and were associated with less canker incidence in the following season. There was a clear link between rainfalls, numbers of spores collected and canker incidence. It was speculated that abundant spore formation on leaves during the wet season leads to profuse numbers of spores washed down into the soil and subsequent canker. The optimal temperature for growth is 20 °C and abundant soil moisture and low temperatures promote the disease whilst hot and dry conditions retard it (Cerkauskas 2002).

1.2.3.4 Presence on seed

The presence of *Itersonilia* in seed was attributed to contamination from infected trash and to infected flowers. Infection of parsnip seedlings from previously pristine fields led to the discovery that *Itersonilia* could be seed-borne (Channon 1967, Smith 1966). A simple bioassay of unsorted seeds stuck to petri dish lids over media demonstrated that *Itersonilia* was present in 20% of seed lots, with an equal weight of contaminated seed in lots from grower and commercial sources. It was speculated that the presence of *Itersonilia* in seed lots was probably due to exposure to dried plant trash. The level of contamination of the seeds (1–4%) was enough to induce seedling infection. Channon (1969) found *Itersonilia* in flowers, which led to a reduction in seed production and could be a potential problem for emerging seedlings.

1.2.3.5 Persistence in soil

The survival of *Itersonilia* in soil was demonstrated by Smith (1967). When parsnip roots with canker were buried in the soil, *Itersonilia* was still viable after 12 months. When the tops of the roots were excised to simulate harvest damage and stimulate breakdown, viability was cut to 7 months. Soil saprophytes such as *Bacillus subtilis* and *Streptomyces* sp. were introduced to sterile soil inoculated with *Itersonilia* and rapidly lysed both ballistospores and hyphae, but left the more resistant and hardier resting spores (i.e. chlamyospores). This mirrored ‘natural soils’ and demonstrated the effectiveness of ‘hilling’ by covering the parsnip crowns progressively with soil and thus encouraging rapid breakdown of the fungus. However, the survivability of *Itersonilia* in soil showed that infected parsnip roots are an obvious source of carry-over in the soil with chlamyospores persisting in a cycle of infection.

1.2.6 Host specificity

I. perplexans has been found to be pathogenic on a wide variety of crops and flowers, including parsnip, dill, chrysanthemum, Chinese aster, sunflower, and edible burdock (Channon 1963a, Horita and Yasuoka 2002, Koike 2001, McGovern and Seijo 1999, Seijo *et al.* 2000). It is generally accepted that those isolated from flowers such as chrysanthemum are not pathogenic on parsnip and vice-versa, possibly indicating that *I. perplexans* is a weak pathogen at best (Koike 2001). Alternatively, these differences may be due to different

pathotypes. However, isolates from edible burdock were also capable of infecting chrysanthemums, causing petal blight (Horita and Yasuoka 2002).

1.2.7 *I. perplexans* or *I. pastinacae*?

In his initial studies of parsnip canker, Channon (1963a) isolated an *Itersonilia* strain which was thought to be sufficiently different from the type strain of *I. perplexans*, and was named *I. pastinacae*. However, studies of nutrition requirements, mating and DNA homology determined that *I. pastinacae*, *I. perplexans*, and another species *I. pyriformans* were *Itersonilia perplexans* (Boekhout 1991, Boekhout *et al.* 1991).

1.3 Other causes of parsnip canker

In initial studies of parsnip canker in the UK, other fungi were associated with cankers, lending weight to the theory of a ‘complex’ of fungi was responsible for parsnip canker (Channon 1963c, 1965).

1.3.1 *Phoma*

The initial studies by Channon (1963a) found *Phoma* as well as *Itersonilia*, and both were capable of producing ‘black cankers’. Researchers in Canada found that *P. complanata* caused wide spread losses (up to 80 % incidence in field crops) and confirmed that the pathogen was seed-borne (Cerkauskas 1985). There was a strong correlation between the severity and incidence of the foliage phase of *Phoma* and the severity and incidence of the canker phase (Cerkauskas 1987). Unlike *Itersonilia*, *P. complanata* had a narrow pathogenicity range, but like *Itersonilia*, it was capable of over-wintering and surviving in soils for up to 5 months (Cerkauskas 1987).

1.3.2 *Mycocentrospora*

Canker symptoms very similar to those caused by *Itersonilia* yielded another pathogen, *M. acerina*. Cankers associated with this pathogen were also black, but were usually surrounded by a pale brown/red band. Unlike *Itersonilia*, there was no difference in the severity or size of the cankers caused by *M. acerina* on wounded and unwounded parsnip roots. It was pathogenic on a wide variety of crops including parsnip, beetroot, peas, cabbage, cauliflower, tomato and carrot. *M. acerina* was found to be capable of growing and infecting at temperatures below 0°C, which would encourage the advent of canker symptoms under winter conditions (Channon 1965).

1.3.3 *Cylindrocarpon*

C. destructans, a fungus similar to *Fusarium*, was also isolated from black or dark brown cankers on parsnip. Isolates of this fungus were capable of re-infecting and causing canker symptoms on damaged parsnip roots (Channon and Thomson 1981). The fungus has a wide range of hosts and is considered to be a weak pathogen in most hosts, but a major pathogen of ginseng (Zeizold 1997).

1.3.4 *Streptomyces scabies*

S. scabies, which causes common scab in potato, was also found to be capable of producing canker in parsnip (Jones 1953).

1.3.5 *Psila rosa* (Carrot rust fly)

Psila rosa (Carrot rust fly) is a major contributor to the incidence of parsnip canker in the UK, because of the damage it causes to parsnip roots, pre-disposing them to infection (Stone 1954). Controlling carrot fly incidence was found to be consistent with a significant reduction in parsnip canker incidence (Collingwood and Croxall 1954). Control of carrot fly usually involves pre-drilling the soil before seeding and treating with insecticides such as phorate and

diazinon (Sivasubramaniam *et al.* 1997). Research is continuing into the location and the amount of insecticide needed to effectively control this pest (Sivasubramaniam *et al.* 1999).

1.3.5.1 Biosecurity issues with carrot fly

Although carrot rust fly is not in Australia, it is widely distributed around the world. It is in North America, the UK, Europe and Eurasia (Factsheet 17/2001). Its presence has been documented in New Zealand, and thus has the potential to be a major biosecurity issue for carrot and parsnip growers in Australia.

1.4 Control of parsnip canker

Management of parsnip canker associated with *Itersonilia* has included cultural practices, cultivar resistance and fungicide treatments.

1.4.1 Cultural practices

The cultural control of canker is limited in scope, but the following practices have been investigated and promoted.

- Gradual hilling and covering of parsnip shoulders encourages breakdown of *Itersonilia* ballistospores in soil (Channon 1963b, Smith 1967). In the UK, this practice led to a 45% reduction in canker incidence and a 70% reduction in the size of the lesions. However, growers in Australia are adamant that this only increases the incidence of *Phoma* canker.
- Sowing and spacing practices were reported to alleviate canker incidence (Channon 1964). There was at least a 3-fold reduction in canker incidence when the crop was late-sown and thinned to 3-cm intervals. However, this was offset by a reduction in root size, with small roots having less canker than larger roots, and there was a balance between a loss in total yield and a reduction in canker incidence and severity that increased marketable roots. While there was a 75% reduction in canker incidence and a 60% reduction in lesion size, there was a large drop in marketable yields (50%), so this method was deemed to be impractical.
- Crop hygiene consisting of removal of all roots and plant trash from beds was suggested by Smith (1967). No hard figures are available for this practice, but it is logical to assume there would be a reduced *Itersonilia* presence in the soil, leading to less canker.
- Crop rotation is imperative as *Itersonilia* can survive on parsnip roots after burial for 12 months. The air-borne stage is not viable after 2 days in soil (Smith 1967).

1.4.2 Fungicide treatments

There is very little literature concerning fungicide control of canker caused by *Itersonilia*. Some authors have recommended the application of copper (every 7–10 days) as a foliar spray to eliminate ballistospores on foliage and thus reduce the incidence of canker (Chupp and Sharp 1960). In New Zealand maneb sprayed at fortnightly intervals from February to June, had some efficacy on *Itersonilia* canker (Brandenburg 1965). *Bacillus subtilis* and *Streptomyces* spp. were antagonistic to *Itersonilia* *in vitro* in Australia (Smith 1967), but biological options do not appear to have been examined in the field. Up to 7 fungicide sprays per crop were required for the control of canker. This frequency of calendar spraying is considered uneconomic by the Australian industry.

Treatment of parsnip seed with hypochlorite or mercuric chloride was insufficient to eliminate the fungus. Hot water treatment eliminated *Itersonilia*, but significantly reduced germination. Thiram only inactivated *Itersonilia* located on seed surfaces (Channon 1969,

Smith 1966). The most successful treatment was by steam air at 45.5 °C for 30 min which removed it from seed trash without significantly affecting germination (Smith 1966).

Canker caused by *P. complanata* has been successfully controlled by fungicides such as chlorothalonil and mancozeb in Canada (Cerkauskas and McGarvey 1988). The effectiveness of the chemical treatments was dependent on the area in which they were grown. In the UK tebuconazole is registered for canker control (Assured Food Standards 2006).

1.4.3 Cultivar resistance

The control of parsnip canker using resistant cultivars is complicated by the fact that more than one organism is responsible for the disease. Parsnip lines have been bred for resistance with varying success against *I. perplexans* (Anon 1966, Channon *et al.* 1970, Davis *et al.* 1989), *P. complanata* (Cerkauskas 1986ab), *Streptomyces scabies* (Green and Hewlett 1954) and *M. acerina* (Channon 1965). Breeding for resistance against *I. perplexans* and *Phoma* also gave rise to resistance against canker caused by *M. acerina* (Channon 1965, Channon *et al.* 1970).

1.5 The parsnip industry in Australia

In Australia parsnip production is estimated at 10,360 t on 415 ha and valued at A\$20 million. Victoria produces 75 % of the crop which is estimated at 8,535 t on 313 ha and valued at A\$15 million (ABS 2001).

Parsnips are direct seeded and grown for 6–8 months. Most Victorian growers have selected their own seed over the years but commercial varieties are still grown both in Victoria and interstate. Locally bred seed produces a whiter rooted parsnip compared with the creamier coloured root of commercial parsnip varieties. The latter are considered less susceptible to canker, but are least preferred by supermarkets. Parsnips are a demanding crop to grow and harvest since their soft root is not amenable to mechanical harvesting. Ongoing issues with parsnip crops affecting marketability include (i) variability in size and shape, (ii) colour, (iii) forking, (iii) powdery mildew and (iv) canker.

1.6 Conclusions

There is tremendous scope for further research into the management of this debilitating disease. Despite the long history of parsnip canker, little research has been done to control this problem. There has been some progress in managing canker, which has been widely accepted by growers, but more research into the impacts of irrigation, nutrition and soil conditions, such as pH and nutrient levels, is required to further understand and meet this challenge.

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Chapter 2

Surveys of parsnip canker extent and cause for the years 2006 and 2007

Summary

Surveys of the incidence of canker were undertaken systematically in both 2006 and 2007 to determine the presence of canker in crops at harvest. Canker incidence was low throughout the year (<1 %), except in the spring period of August–October, when there was a sharp and dramatic increase in canker incidence, peaking in October (2006, 45 %; 2007, 22 %), before dropping by December (<1 %). Canker incidence in 2006 was worse than in 2007 (a mean of 25 % compared to 17 %). Economic losses due to parsnip canker were estimated at A\$3 million for 2006/2007.

2.1 Introduction

Canker of parsnip has been a serious and persistent problem for the past 40 years. The disease affects the quality of parsnip roots and anecdotally, growers have reported that in severe cases 50–80 % of the crop is unsaleable with entire fields of spring-maturing crops abandoned. Under the direction of growers, systematic surveys at harvest were undertaken to establish when canker incidence was rising in crops, as well as establishing exact levels of canker incidence.

2.2 Methods

2.2.1 Visual surveys

In order to determine the presence of canker, systematic, seasonal surveys (summer, autumn, winter, spring) were undertaken in 2006 at four properties in Victoria, as well as less frequently in both Tasmania and Western Australia. At the request of the growers, surveys were performed on the washline as shown (Fig. 2.1). In a timed interval (of 2 or 3 minutes), repeated 10 times, total numbers of parsnip roots and roots that displayed obvious signs of canker were counted (Fig. 2.2). In the case of one grower, surveys were undertaken in the field. In this instance, 20 parsnips were pulled out of the ground at harvest and visually assessed for canker. Another sample of 20 parsnips was pulled out after 5 metres, and this process was continued until at least 200 parsnips were assessed for canker incidence. In 2007, at the behest of growers, monthly surveys were performed at the Victorian properties.

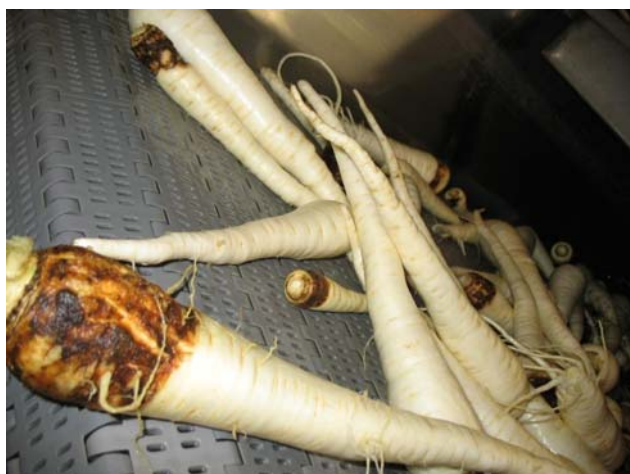


Fig. 2.1 Parsnips on a washline prior to sorting. Note the presence of parsnips with and without cankers



Fig. 2.2 Parsnip samples from a washline prior to microbial sampling of cankers. Note the presence of black cankers on parsnip shoulders

2.2.2 Microbial surveys

A representative sample of parsnips displaying signs of typical canker was removed from each washline/field for the isolation of fungi from the cankers. Parsnips were stored at 4 °C for no more than a week. Each parsnip root was sprayed with 70 % (v/v) ethanol for surface sterilisation. Photographs were taken and a sample number was assigned (Fig. 2.3). Parsnip lesions were cut to expose the interface between healthy and diseased tissue (Fig. 2.4). Sections of root with that interface were placed in a small ceramic sieve, treated as specified below, and placed on three types of agar, malt extract agar (MEA), water agar (WA) and potato dextrose agar with tetracycline (0.05 %, PDAA).

Samples were treated thus,

- MEA, sections of parsnip were attached to the lid of the agar plates with petroleum jelly without prior sterilisation. This method encourages ballistospore colonisation of the plate from *I. perplexans* (Smith 1966).
- WA, for isolation of oomycetes, sections were dipped in sterile deionised water alone for 30 seconds (s) before placement on WA, since it has been demonstrated that surface sterilisation of roots with sodium hypochlorite can suppress oomycetes (Davidson and McKay 2001).
- PDAA, sections were sterilised in sodium hypochlorite (2.5 %) for 30 s, then washed in sterile deionised water before placement on PDAA for isolation of all other fungi except oomycetes.

All plates were sealed with parafilm and incubated for at least 7 days at room temperature. Individual fungal isolates were then subcultured for decontamination before pathogen testing (see Chapter 3).



Fig. 2.3 Parsnip with sample number



Fig. 2.4 Parsnip lesion cross-section

2.3 Results

2.3.1 Washline surveys

The levels of canker are presented for both 2006 and 2007 (Fig. 2.5). In 2006, seasonal surveys were undertaken, but in 2007, at the behest of growers, more frequent surveys were performed.

Canker incidence was highest for September–October in 2006 for most growers with the incidence decreasing markedly in November. An exception was a WA grower, where canker incidence remained high throughout 2006, increasing dramatically in November. Similarly, canker incidence was high in Tasmania, in both summer and winter for 2006.

Canker levels in 2007 were in general, lower in 2006, with canker incidence not exceeding 45 %, but, similar to 2006, there was a noticeable increase in canker incidence for most growers in the September–October period.

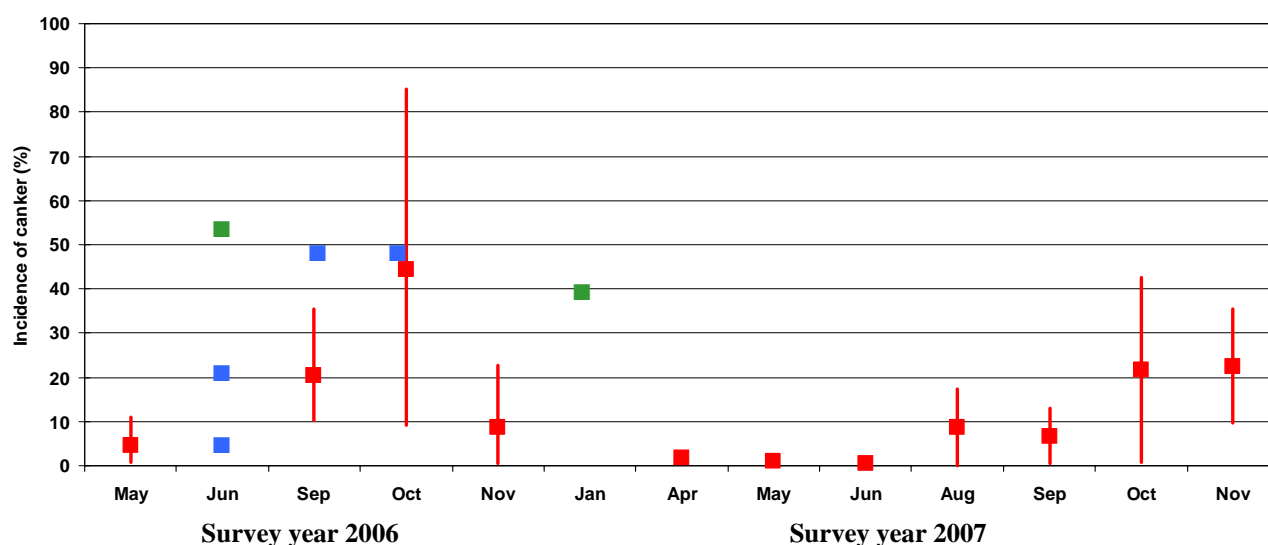


Fig. 2.5 Parsnip canker incidence in survey years 2006 and 2007. (Box=mean, line=range; red squares, Victoria surveys; blue squares, Western Australia surveys; green squares, Tasmania surveys)

2.3.2 Microbial surveys

Sampling of parsnips for the presence of fungi emphasised the superficial nature of the disease, with no cankers penetrating more than 5 mm below the surface of the root (Fig. 2.4). Microbial samples were derived from parsnips at harvest, so not surprisingly, a suite of fungi were isolated from these cankers. *Itersonilia perplexans* was routinely isolated, but other fungi such as *Acremonium* spp., *Cylindrocarpon* spp., *Fusarium* spp., *Microdochium* spp., *Mycocentrospora acerina*, *Phoma* spp., *Pythium* spp. and *Rhizoctonia* spp., were also isolated. Over 500 individual isolates were extracted from cankers and their pathogenic ability was determined (see Chapter 3).

2.3.3 Economic impact of parsnip canker

Taking into consideration the losses due to canker, assuming that portion of the crop is unmarketable, and calculating the potential yields of parsnip from acreage data supplied by growers, a preliminary estimate of monetary losses due to parsnip canker was calculated. Monthly parsnip prices were obtained from Melbourne Market (see Appendix 2.5), and monetary losses in Victoria alone due to parsnip canker were calculated (Table 2.1). Losses for 2006 and 2007 combined were calculated at over A\$3 million, demonstrating the devastating effect parsnip canker has on grower earnings.

Table 2.1 Estimated monetary losses due to parsnip canker for Victorian growers

	Sep-06	Oct-06	Nov-06	Sep-07	Oct-07	Nov-07
Average Price /10kg box (\$) (Melbourne Market) – Class 1	36.5	52.75	49.52	49.5	43.2	44.5
Average canker loss - Victoria (%)	20.3	44.4	8.6	6.75	21.58	22.52
Average monthly production (10kg boxes)	53,960	53,960	53,960	53,960	53,960	53,960
Theoretical turnover (100%) [A\$]	1,969,540	2,846,390	2,672,099	2,671,020	2,331,072	2,401,220
Actual turnover [A\$]	1,569,723	1,582,593	2,442,299	2,490,726	1,828,027	1,860,465
Loss/month [A\$]	399,817	1,263,797	229,801	180,294	503,045	540,755
Total Loss [A\$]			1,893,414			1,224,094

2.4 Discussion

Parsnip surveys on the washline and in the field, supplemented with observations from field trials (see Chapters 4 and 5) confirmed that, in Victoria, the spring harvests of September–November exhibit the highest incidence of parsnip canker. Intermittent surveys in Western Australia and Tasmania showed a wide disparity, but overall, canker incidence in these states did not seem to be season-dependent. Canker incidence in Victoria for 2007 was comparable, but nominally lower than 2006.

In Victoria, canker incidence is highest on the February–March planted crops, but extremely low on crops planted at other times of the year. IPM strategies to control canker only need to be targeted at the February–March plantings. Also, the economics of any regular chemical applications may not be feasible due to the long-term nature of the crop.

The isolation of several different fungi from cankers is not surprising, since all microbial surveys were undertaken from harvest samples, and parsnips are in the ground for 5–7 months. Not only *I. perplexans*, the primary cause of canker, but several other fungi (as indicated above) were extracted, some of which have been previously implicated as a cause of parsnip cankers (e.g. Cerkauskas 1985, Channon 1963, 1965, Channon and Thomson 1981). Thus, any approach in combating parsnip canker must embrace plans to target all of these fungi, on the proviso that they also cause canker. The pathogenicity of these isolates will be discussed in the next chapter.

There is an obvious need to monitor a crop from seeding to harvest for fungi that are pathogenic, since growers report that there is little indication of canker problems up to the 4 month stage. Sampling at harvest probably misses the ‘window’ when parsnip roots are predisposed to infection and the primary cause of parsnip canker can be determined.

2.5 References

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2.5 Appendix: Melbourne Market parsnip prices for the period March 2006 to December 2007

Melbourne Wholesale Market Prices (A\$ per 10 kg carton) 2006

	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Low price	34	34	34	35	35	35	35	45	40	40
High price	36	36	38	38	38	38	60	60	50	55
Average price	35	35.1	35.8	35.7	35.3	35.3	36.5	52.8	49.5	49.2

Melbourne Wholesale Market prices (A\$ per 10 kg carton) 2007

	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Low price	45.5	45	45	45	47.5	50	50	40	42.7	44.5	44.5
High price	50	45	45	47.5	49	50	50	50	44.5	44.5	55
Average price	47.8	45	45	46.3	47.9	50	50	47	44.1	44.5	51.5

Information above on the wholesale price of parsnips was derived from the Melbourne Market Price Reporting Service and was used in calculations for Section 2.2.3.

Box 1 Melbourne Markets, 542 Footscray Road, West Melbourne,
Victoria 3003

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Chapter 3

Pathogen tests

Summary

Several fungi, including the presumptive cause of parsnip canker, *Itersonilia perplexans*, were isolated from parsnip lesions (Chapter 2). Fungal isolations from the parsnip cankers revealed a suite of fungi including *Acremonium* spp., *Cylindrocarpon* spp., *Fusarium* spp., *Mycocentrospora acerina*, *Phoma* spp. and *Pythium* spp. Over 500 individual isolates were tested for pathogenicity, with about 22 % producing lesions or canker-like lesions on fresh parsnip roots in dew chambers.

3.1 Introduction

Itersonilia perplexans has long been established as the primary cause of canker in parsnip overseas (Channon 1956, Smith 1966, Wilkinson 1963). In the literature *Phoma* spp., *Mycocentrospora acerina* and *Cylindrocarpon destructans* are also associated with parsnip canker (Channon 1965, Channon and Thomson 1981, Collingwood and Croxall 1954). This study attempted to ascertain the primary cause of parsnip canker in Australia by fungal isolation and by proving Koch's Postulates.

3.2 Methods

3.2.1 Fungal isolations

The procedures for fungal isolations detailed in Chapter 2 were followed, and the fungi isolated (Fig. 3.1) were tested for pathogenicity.

3.2.2 Pathogenicity tests

After growing fungi on suitable media (MEA, PDA, PDAA, V8) for 5–7 days at room temperature, 5-mm agar plugs were aseptically excised from the leading edge of fungal growth (Fig. 3.2). These plugs were then transferred to fresh parsnip roots, either damaged or undamaged. Parsnip roots were damaged by sterile needle at three locations on the root: collar, middle and bottom. One plug was aseptically transferred to each location, making a total of six plugs (Fig. 3.3). To promote lesion development parsnip roots were then placed in 1 L plastic take-away containers lined with tissue paper, sprayed with sterile deionised water, sealed and stored at 9 °C to simulate winter soil temperatures (Figs. 3.4, 3.5). Fresh plates, as well as slide preparations of each isolate, were also prepared. Plates were stored at 4 °C after incubation for 5–7 days at room temperature, and slide preparations were preserved by sealing the edges of cover-slips with nail polish. Parsnip roots were visually assessed every month for lesion development.

After lesion development, pieces of diseased tissue were placed on fresh agar plates and compared to the stored isolate, in relation to colony appearance and microscopic features to confirm Koch's Postulates. Isolates that caused lesions or canker-like symptoms were then identified to at least genus level. If no lesions developed after three months, the isolate was scored as non-pathogenic.

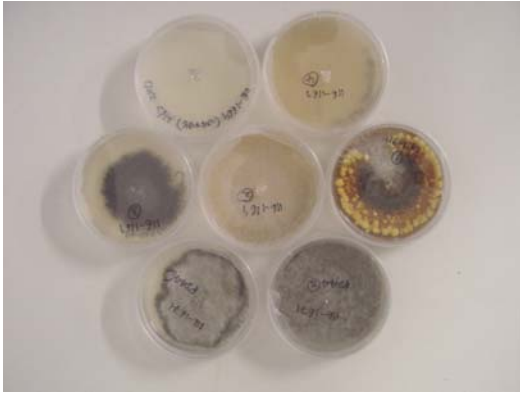


Fig. 3.1 Fungal isolates from one parsnip canker

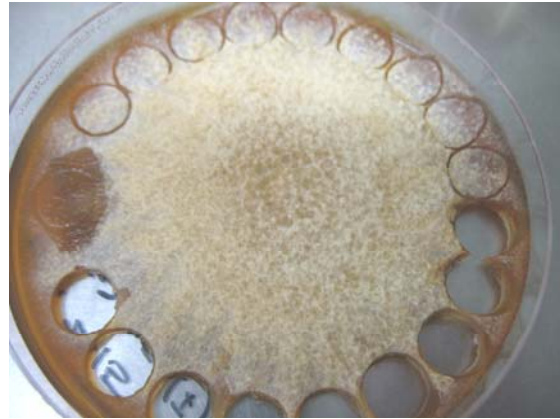


Fig. 3.2 Agar plugs excised from the leading (growing) edge of fungal growth on an agar plate



Fig. 3.3 Position of agar plugs on fresh parsnip roots

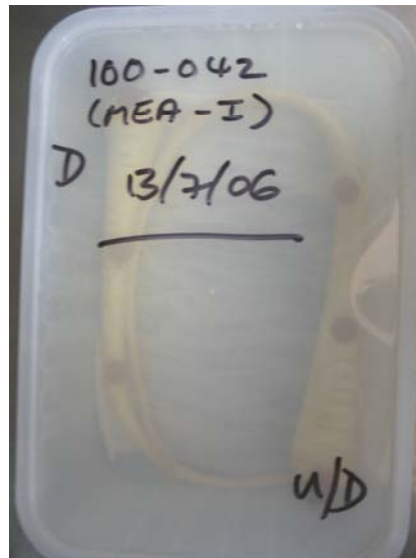


Fig. 3.4 A sealed chamber before storage at 9 °C



Fig. 3.5 Parsnip samples stored at 9 °C in a cold room

3.3 Results

Over 500 individual isolates were tested over the two-year period (Fig. 3.6). As well as the presumptive cause of parsnip canker, *Itersonilia perplexans*, fungi such as *Acremonium* spp., *Cylindrocarpon* spp., *Fusarium* spp., *Mycocentrospora acerina*, *Phoma* spp. and *Pythium* spp. were also isolated from cankers. Table 3.1 summarises the isolates that caused canker-like symptoms on fresh parsnip roots.



Fig. 3.6 Isolates from parsnip cankers for pathogen testing

Table 3.1 Isolates that caused canker or canker-like symptoms in parsnip

Fungus	Number of positive isolates	% of positive isolates
Unknown	26	23.4
<i>I. perplexans</i>	24	21.6
<i>Fusarium</i> spp.	21	18.9
<i>Cylindrocarpon</i> spp.	12	10.8
<i>Microdochium</i> spp.	7	6.3
<i>Rhizoctonia</i> spp.	5	4.5
<i>Mycocentrospora acerina</i>	5	4.5
<i>Phoma</i> spp.	4	3.6
<i>Acremonium</i> spp.	3	2.7
<i>Pythium</i> spp.	3	2.7
<i>Pithomyces</i> spp.	1	0.9
TOTAL	111	100

Of those tested, 111 were pathogenic (22.2 %), in that they produced lesions or canker-like symptoms on fresh parsnip roots. A total of 26 isolates (23.4 %) were unable to be identified, because they were sterile in culture and DNA typing was unsuccessful. Of those organisms known previously to cause parsnip cankers (see Chapter 1), *I. perplexans* represented 21.6 % of known pathogens; *Cylindrocarpon* spp., 10.8 %; *Mycocentrospora acerina*, 4.5 %; and *Phoma* spp., 3.6 %.

Lesion development could be assigned into three broad categories in roughly equal proportions. Isolates produced lesions on all areas of parsnip roots, both damaged and undamaged (Type I, Fig. 3.7); lesions on upper areas only (Type II, Fig. 3.8) or lesions on damaged root only, either all along the length, or at the shoulder only (Type III, Fig. 3.9). In a variation of Type III, lesions formed also at the shoulder of the undamaged root (Type IV, Fig. 3.10).



Fig. 3.7 Type I: Lesions on both damaged (left) and undamaged (right) parsnip roots



Fig. 3.8 Type II: Lesions on both damaged (left) and undamaged (right) parsnip roots



Fig. 3.9 Type III: Lesions on the damaged root only (left)



Fig. 3.10 Type IV: Lesions on the damaged root (left) and only on shoulder of the undamaged root (right)

There was little relationship between pathogen ‘type’ and fungal type/genus. Table 3.2 below lists all identified isolates along with their pathogen ‘type’ as described above.

Table 3.2: Pathogen ‘type’ of known fungal isolates

Fungi	Pathogen ‘type’
<i>Acremonium</i> spp.	II, IV
<i>Cylindrocarpon</i> spp. ²	I, II
<i>Fusarium solani</i>	II
<i>Fusarium oxysporum</i>	I
<i>Fusarium</i> spp.	I, II, IV
<i>Itersonilia perplexans</i> ²	I, II, III, IV
<i>Microdochium (tabachinum?)</i>	I
<i>Mycocentrospora acerina</i> ²	I
<i>Phoma</i> spp.; <i>Phoma exigua</i> ²	I
<i>Pythium</i> spp., <i>Pythium sulcatum</i> ¹	II
<i>Rhizoctonia</i> spp.	III

¹ From laboratory pathogen collection, originally isolated and pathogenic on parsley (see VG04064)

² Previously recorded as causes of parsnip canker (see discussion)

3.4 Discussion

Overseas, parsnip canker has been attributed to *I. perplexans*, as well as *Phoma complana*, *Mycocentrospora* spp. and *Cylindrocarpon destructans* (Cerkaukas 1985, Channon 1963ab, Channon and Thomson 1981). In Victoria, Australia, in addition to those isolates, other fungi are implicated, possibly forming a complex of organisms capable of causing canker. Pathogenic isolates also included several fungi that have not previously been implicated in parsnip cankers, such as *Acremonium* spp. and *Microdochium* spp., as well as *Fusarium* spp., *Pythium* spp. and *Rhizoctonia* spp., which are known to cause root and crown rot in parsnip (CAES webpage 2008).

Aggressive pathogens were those that caused lesions on both damaged and undamaged roots (i.e. types I, II, IV, Table 3.2). Weaker, opportunistic pathogens only caused lesions on the damaged roots. These included *Rhizoctonia* spp. *I. perplexans* isolates varied in their pathogenicity, displaying all ‘pathogen types’, possibly revealing variation within the species. Isolates were only tested for pathogenicity individually. Inoculating combinations (e.g. *Pythium* spp.), followed by *I. perplexans*, *Phoma*, *Rhizoctonia*, etc., could confirm the notion of a complex of fungi being responsible for parsnip canker, or certain fungi predisposing parsnip to canker formation.

A wide variety of fungi were derived from parsnip cankers. Apart from the fungal isolates that were shown to be pathogenic, 77.8 % did not form lesions or canker-like symptoms on

fresh parsnip roots. These probably represented saprophytic organisms that occurred naturally in the soil. Also, if there is a succession of fungi on parsnip roots, which is highly likely considering the long-term nature of the crop, the initial cause of the canker may have been missed, since microbial samples were only taken at harvest from samples off the wash-line when canker surveys were performed (see Chapter 2).

The variety of fungi proven to be pathogenic on parsnip causing canker-like symptoms emphasises that a holistic approach must be taken to combat this particular disease. The next two chapters outline potential strategies to alleviate the incidence and severity of canker in parsnips.

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Chapter 4

Field trials to complement pathogenicity tests

Summary

To confirm the pathogenicity tests of Chapter 3 and as part of a two pronged approach to identify the cause of parsnip canker, three field trials were undertaken with fungicides targeted to control specific fungi. The only fungicide with efficacy was Ridomil, which reduced canker in one trial by 78 % suggesting oomycetes, such as *Pythium*, may be associated with canker on one site, possibly on a second, but not on the third site. This observation is confounded by the positive site being fumigated on a regular basis. Canker may be associated with a complex of organisms or with abiotic factors. Biocontrol agents had no efficacy against parsnip canker. The 7–8 month duration of the crop suggests chemical controls will not be an option and re-enforces the idea of focussing on soil health to combat the disease.

4.1 Introduction

The results in Chapter 3 showed that a complex of microbes may be responsible for black canker in parsnips, with one, or several, fungi causing or pre-disposing parsnips to canker. Overseas parsnip canker has been associated with *Itersonilia perplexans*, *Phoma* spp., *Phoma complanata*, *Mycocentrospora acerina*, *Cylindrocarpon destructans* and *Streptomyces scabies* (Cerkauskas 1986, Channon 1965, Channon and Thomson 1981, Fox 2002, Green and Hewlett 1954, Wilkinson 1952). It is not considered to be caused by bacteria (Green and Hewlett 1950), but has been associated with the carrot fly larvae *Psila rosae* in the UK (Stone 1954). Fortunately carrot flies are not present in Australia. There is no evidence that nematodes are associated with the canker in Victorian parsnip crops (Crop Health Services DPI Vic., pers. comm.).

Management of parsnip canker associated with *Itersonilia* has included: (i) use of breeding resistant cultivars, (ii) seed treatment, (iii) cultural practices, (iv) hygiene, and (iv) fungicide strategies. Fungicide seed treatments are considered effective for 4 to 6 weeks. Fungicide treatments with efficacy to control the foliage phase of *Itersonilia* canker were copper and to a lesser extent maneb (Brandenburg 1965, Chupp and Sharp 1960). The foliage phase of *Phoma* canker was controlled with chlorothalonil or mancozeb in Canada (Cerkauskas and McGarvey 1988) or tebuconazole, which is registered in the UK (Assured Food Standards 2006).

Growers have a number of theories concerning predisposing factors for parsnip canker. These include (i) excessive soil moisture, (ii) nutrition, (iii) rapid growth in spring, (iv) top-dressing after winter, (v) changes in parsnip chemistry as the spring crop goes to seed, and (vii) *Pythium* damage to lateral roots predisposing parsnips to canker. Some parsnip seed is treated by the industry and crops are rotated. Parsnip crowns are covered but hilling up of crowns is avoided as it is believed to predispose parsnips to *Phoma* canker. Interviews with retired parsnip growers indicated that parsnip canker can be reduced with (i) soil pH 9 with GBA (ground burnt agricultural lime), (ii) additional potash to stimulate root growth, (iii) additions of organic matter (straw, lime and fowl manure), (iv) resting ground before planting and (v) a long rotation out of parsnips.

This chapter reports trials designed to complement the pathogenicity tests of Chapter 3, as part of a two pronged approach to identify the cause of parsnip canker. This set of trials used fungicides that were specific for oomycetes, assorted Ascomycetes and Fungi Imperfecti e.g.

Fusarium and Basidiomycetes (eg. *Rhizoctonia*) organisms, as epidemiological tools to ascertain which groups of fungi were responsible for parsnip canker under field conditions at several sites with different soil types. Soil types included sand and sandy loam at Rosebud and Cranbourne West, respectively. Biocontrol organisms were also evaluated in a preliminary investigation as potential options for an IPM program for the disease.

4.2 Materials and methods

There were 3 trials over the summer of 2006 and 4 trials over the winter of 2007, numbered 1–7, respectively (Table 4.1). All seed used in the trials was the growers' own seed. Seed planted at Cranbourne West was treated with Thiram[®] and seed planted at Rosebud was treated with Maxim[®] and Apron XL[®]. The Rosebud site had been fumigated with Metham Sodium[®] in the past.

Table 4.1 List of trial sites and activity dates for 2006 and 2007

Year	Trial No. No.	Site	Date		
			Planted	Emergence	Harvest
2006	1	Cranbourne (west)	21/07/2006	na	na
	2	Rosebud	26/07/2006	14/10/2006	8/01/2007
	3	Devon Meadows	14/09/2006	13/10/2006	14/02/2007
2007	4	Devon Meadows	30/03/2007	na	8/10/2007
	5	Rosebud	29/03/2007	7/05/2007	5/10/2007
	6	Cranbourne (west)	2/04/2007	7/05/2007	17/10/2007
	7	Rosebud (biocontrol)	23/04/2007	7/05/2007	5/10/2007

na, not assessed.

4.2.1 Trial designs

All trials contained an untreated Control and were laid out in randomised blocks. Each bed contained four rows of parsnips so an experimental unit comprised 4 rows of parsnips but varied in length. Plot dimensions for Trial 1 were 3m by 1.62m (an area of 4.86 m²) and for Trials 2–7, the dimensions were 5m by 1.62m (an area of 8.1 m²). The layouts for the trials varied. Trials 1, 5 and 6 had the same layout occupying two beds of 12 plots each. Each of the six replicates consisted of a 2 by 2 block of four plots to which the four treatments were allocated. Trials 2 and 3 had the same layout and consisted of 12 replicates laid out across 6 beds with each bed of six plots containing two replicate blocks of three plots each with a plot for each of the three treatments. Trial 4 contained six replicates across four beds. Each replicate consisted of a block of 2 by 2 plots to which the four treatments were allocated and the replicates were arranged in two stacks of three replicates each. Trial 7 occupied two beds with 9 plots in each bed. Each bed contained 3 replicates of three plots each.

4.2.2 Chemical applications

Treatments are listed in Table 4.2, and spray schedules are listed in Table 4.3. Granular applications were spread by hand. Drench applications of Polyversum, MicroPlus, Bavistin and Rizolex were applied by a Teejet 8003 VP (red and blue) nozzle and spray applications were applied by a Teejet SPX No. 12 nozzle (brown), at 30 psi using a Silvan Selectra 12 v knapsack (Silvan Pumps and Sprayers, Aus., Pty. Ltd.) with a 3 nozzle boom configuration. In the later stages of growth, from the 5th month, spray applications of all fungicides were applied using one nozzle, at equivalent rates.

Table 4.2 Treatments and rates of applications for the 2006 and 2007 trials

Trade name	Active	Target	Company	Label rate	Application rate	Plot rate	Trial
Control	na	na	na	na	na	na	1-7
Bavistin FL	carbendazim	<i>Fusarium, Septoria, Alternaria, Cercospora, Cladosporium</i> etc.	BASF	60 mL/100 m ² ^A	1000 L/ha.	486 mL	1
Rizolex 500WP	tolclofos-methyl	<i>Rhizoctonia</i>	Sumitomo	50 mL/100 m ²	500 L/ha	405 mL	4, 5, 6
Ridomil Gold RG25	metalaxyl	<i>Pythium, Phytophthora</i>	Syngenta	120 g/100 m ^c	1000 L/ha. 500 L/ha	486 mL 405 mL	1, 2, 3 4, 5, 6
Ridomil Gold RG25	metalaxyl	<i>Pythium, Phytophthora</i>	Syngenta	120 g/100 m ^c	1.2 g/1m row	3.6 g	1, 2, 3
Polyversum™	<i>Pythium oligandrum</i>	<i>Fusarium, Pythium, Phytophthora, Verticillium, Sclerotinia</i>	Biopreparaty	200 g/ha	400 L/ha	6.0 g 2 g/4 L	4, 5, 6 7
MicroPlus™	<i>Streptomyces lydicus</i>		Organic Farming Systems	500 g/ha	500 L/ha	2 g/4L	7

^A, turf rate, soil-borne; ^B, soil application rate; ^C, of row (carrot rate)

Table 4.3 Spray schedules for the 2006 and 2007 trials

Year	Trial No.	Site	Treatment	Application date	Week applied		
2006	1	Cranbourne (west) ^B	Bavistin FL	27/09/2006	10		
				25/10/2006	14		
				29/11/2006	19		
			Ridomil Gold RG25	27/09/2006	10		
				29/11/2006	19		
				Rizolex 500 WP	27/09/2006	10	
	2	Rosebud ^A	Ridomil Gold RG25	6/10/2006	8		
				8/12/2006	10		
				8/12/2006	19		
	3	Devon Meadows	Rizolex 500 WP	6/10/2006	10		
				Ridomil Gold RG25	27/09/2006	2	
					29/11/2006	11	
2007	4	Devon Meadows	Bavistin FL	27/09/2006	2		
				29/11/2006	11		
				Rizolex 500 WP	27/09/2006	2	
			Ridomil Gold RG25	30/03/2007	1		
				1/05/2007	6		
				4/06/2007	10		
	5	Rosebud ^A	Ridomil Gold RG25	2/07/2007	15		
				30/03/2007	1		
				1/05/2007	6		
			Rizolex 500 WP	30/03/2007	1		
				Bavistin FL	29/03/2007	1	
					30/04/2007	6	
	1/06/2007	10					
	6	Cranbourne (west) ^B	Ridomil Gold RG25	29/06/2007	14		
				29/03/2007	1		
				30/04/2007	6		
				2/07/2007	14		
				2/04/2007	1		
1/05/2007				5			
Rizolex 500 WP			2/04/2007	1			
			2/04/2007	1			
			7	Rosebud ^A	MicroPlus	28/05/2007	1
						28/06/2007	5
						28/07/2007	9
					Polyversum	28/08/2007	14
28/09/2007	18						
28/05/2007	1						
28/06/2007	5						
28/07/2007	9						
28/08/2007	14						
28/09/2007	18						

^A, seed treated with Maxim[®] and Apron XL[®]; ^B, Seed treated with Thiram[®]

4.2.3 Source of biological controls

Polyversum™ was sourced from Biopreparaty (Prague, Czech Republic) after approval for importation was granted by AQIS (Permit no. 200613869) and approval for use was granted by the AVPMA (PER 7250). Polyversum™ consists of *P. oligandrum* oospores dispersed in anhydrous silicon dioxide at a concentration of no less than 10^6 oospores g^{-1} . The formulation is readily dispersed in water prior to use.

Micro-Plus was sourced from Organic Farming Systems (PO Box 419, Cottesloe WA 6911; ACN 076 001 100). It contains 10^7 cfu mL^{-1} of *Streptomyces lydicus* WYEC108. This formulation was imported from the USA and repackaged for the Australian market. It was applied to the trial immediately after dispersing the preparation in water.

4.2.4 Assessment and analysis

Emergence or germination was analysed for Trials 3, 5 and 6 (Table 4.1), and measured as the number of plants in a 40-cm length of row across 4 rows of parsnips on a bed at the Rosebud site and a 30-cm length of row across 4 rows of parsnips on a bed at the Cranbourne (west) site.

At harvest, parsnips in all trials were ‘lifted’ by the growers then assessed visually for the presence or absence of canker by the research team. For each plot, the disease data consisted of the number of diseased plants in each plot and the total number of plants assessed.

Trial 1 had no disease. The data from Trials 2 to 7 were analysed using Analysis of Variance using Genstat®. The canker data from Trials 2 and 3 needed a square root transformation prior to analysis. The data from Bed 6 (containing replicates 11 and 12) were omitted from the analysis of Trial 2 as 5 of the 6 plots in that bed contained no disease at all. This may have been due to uneven distribution of the pathogen.

4.3 Results

4.3.1 Germination

The only treatment which affected emergence was Rizolex 500 WP, which reduced it significantly, compared with the control, but only in Trial 6 (Table 4.4). There were no evidence of damping off which Ridomil gold RG25 would be expected to control.

Table 4.4 Effect of treatments on the emergence parsnip seedlings at 3 sites during 2006 to 2007

Treatment	Emergence (%)		
	Trial No. 3	Trial No. 5	Trial No. 6
	Summer 2006 Devon Meadows	Winter 2007 Rosebud	Winter 2007 Cranbourne (west)
Control	30.5	18.6	25.8 b
Bavistin FL	na	17.8	23.0 ab
Ridomil Gold RG25	30.0	18.8	24.5 ab
Rizolex 500 WP	30.0	17.0	21.1 a
<i>lsd</i> (5%)	<i>ns</i>	<i>ns</i>	3.592

ns, not significant; na, not applicable.

4.3.2 Chemical trials

Of the two summer trials that showed disease (Trials 2 and 3, Table 4.5), the trial at Devon Meadows showed some evidence of a difference between the two chemical treatments because the chemical Rizolex 500 WP appeared to increase levels of disease. Overall,

however, there was no significant difference between the Control and either of the two chemical treatments, Rizolex 500 WP and Ridomil Gold RG25. Of the four winter trials, only Trial 5 showed a significant difference between treatments. Ridomil Gold RG25 reduced disease significantly, by 76%, when compared to the control (Table 4.5). Larger plot sizes may have produced significant reductions in canker, especially in Trials 2 and 4. The biocontrols had no efficacy on parsnip canker (Table 4.5).

Table 4.5 Effects of fungal specific chemical treatments on incidence of parsnip canker

Year	Trial No.	Site	Treatment	No. sprays	Incidence of canker (%)
2006	1	Cranbourne (west) ^A	Control	0	0
			Bavistin FL	3	0
			Ridomil Gold RG25	2	0
			Rizolex 500 WP	1	0
			<i>lsd (5%)^C</i>		<i>ns</i>
	2	Rosebud ^B	Control	0	1.05
			Ridomil Gold RG25	3	0.31
			Rizolex 500 WP	1	0.72
			<i>lsd (5%)^C</i>		<i>ns</i>
	3	Devon Meadows	Control	0	0.90
			Ridomil Gold RG25	2	0.49
			Rizolex 500 WP	1	1.94
<i>lsd (5%)^C</i>				<i>ns</i>	
2007	4	Devon Meadows	Control	0	12.8
			Bavistin FL	4	13.9
			Ridomil Gold RG25	2	8.4
			Rizolex 500 WP	1	16.5
			<i>lsd (5%)^C</i>		<i>ns</i>
	5	Rosebud ^B	Control	0	16.0 a
			Bavistin FL	4	12.0 a
			Rizolex 500 WP	1	11.7 a
			Ridomil Gold RG25	2	3.80 b
			<i>lsd (5%)^C</i>		6.6
	6	Cranbourne (west) ^A	Control	0	35.0
			Bavistin FL	4	30.6
			Ridomil Gold RG25	2	30.3
			Rizolex 500 WP	1	30.7
			<i>lsd (5%)^C</i>		<i>ns</i>
	7	Rosebud ^B	Control	0	10.1
			MicroPlus™	5	9.1
Polyversum™			5	9.6	
<i>lsd (5%)^C</i>				<i>ns</i>	

^A, seed treated with Thiram®; ^B, seed treated with Maxim® and Apron XL®; ^C, data required transformation for analysis therefore there is no *lsd*; *ns*, not significant.

4.4 Discussion

The only fungicide with efficacy was Ridomil Gold RG25, which suggests that oomycetes may be associated with canker on one site, possibly on a second but not on the third site. Ridomil Gold RG25 had efficacy in sandy soil, but not on the sandy loam which would have a higher organic content. However, the sandy site (Rosebud) was fumigated, which may have confounded the observations. There are several possibilities:

- A complex of organisms may have been associated with canker at the Cranbourne site, as none of the chemicals had efficacy, but these were removed with fumigation at the

Rosebud site. If several organisms can cause canker, and if one is removed with a specific chemical, the others can still cause canker;

- The chemicals may not have targeted the appropriate fungus or were not applied at the appropriate time;
- Other fungi, organisms or abiotic factors such as nutrients or irrigation, may be associated with canker;
- *Pythium* spp. may be causing damage in the early phases of crop growth with other pathogens succeeding, them in the later stages, hence the low levels of *Pythium* recovered from surveyed parsnips;
- Fungicides did not target *Itersonilia* and an *Itersonilia*-specific fungicide is required;

Parsnips are a 5–7 month crop and growers report that canker symptoms first appear at the three-quarter stage in June–July. The causal agent of the disease is probably infecting parsnip roots much earlier in the field. It is very interesting that retired growers all pointed to a high soil pH and lime application as a good method to reduce canker. A similar situation has also been reported for clubroot (Caroline Donald, pers. comm.). Few chemicals have been trialled for the control of *Itersonilia* canker, except maneb and copper (Brandenburg 1965, Chupp and Sharp 1960). Both chemicals required 7–14 day applications, exhibited limited efficacy and the high frequency of applications would limit their economic benefit. The long cropping period for parsnips would render regular spraying uneconomical and would not align with an IPM strategy. The long term control of the disease will probably rest with breeding resistant varieties.

Ridomil

Metalaxyl was the only chemical with efficacy against canker. The efficacy of metalaxyl at the Rosebud site (Trial 5) suggests that oomycetes were associated with canker. *Pythium* spp. have a low incidence of isolation from parsnip cankers, but are pathogenic on parsnip roots (Chapter 2 and 3). *Phytophthora* spp. have also been isolated from parsnip cankers (Crop Health Services, pers. comm.). Metalaxyl was applied early at weeks 1 and 6. It has efficacy against oomycetes for about 6 weeks on parsley in the region (Minchinton *et al.* 2006). It is unlikely to be a sustainable option for management of *Pythium* because of its biological degradation in sandy soil (Davidson and McKay 1999, 2001) and the potential for fungi to develop resistance to it (Bailey and Coffey 1985). The Rosebud site was fumigated on a regular basis. While it is possible that the fumigation removed pathogens, oomycetes may have re-enter the system via the irrigation water. The efficacy of metalaxyl on this site suggests that oomycetes could be associated with disease in the early stages of parsnip crops. The mean incidence for plots treated with metalaxyl at Devon Meadows was 34 % lower than the Control, but this difference may be due to chance as the statistical analysis showed no significant difference. Metalaxyl had no efficacy at the Cranbourne west site, implying that other organisms are associated with canker or the timing of the applications was inappropriate.

Rizolex

Rizolex has efficacy against *Rhizoctonia* spp. (Tomlin 2003). The one application had no efficacy against canker in any of the trials and it often increased canker incidence, although not significantly. It is also possible the timing of the application was inappropriate. *Rhizoctonia* had a low frequency of isolation (Chapter 3). Some isolates were identified as binucleate and could therefore be saprophytic (Joanna Petkowski pers. comm.). They may be competing with other pathogens and consequently their removal could increase canker levels. It appears that *Rhizoctonia* is probably not a major cause of canker. It was also thought that Rizolex may have some efficacy for *Itersonilia*, as it is also a basidiomycete, but the one

application to parsnips had no efficacy for canker. In hindsight a plate trial to evaluate fungicides for specificity to *Itersonilia* would have been useful.

Bavistin

This treatment was designed to target *Fusarium* spp., which were pathogenic on parsnips and had a high frequency of isolation from parsnip cankers (Chapter 3). Up to 4 applications of Bavistin during the crops' life had absolutely no efficacy on canker. Assuming it did target *Fusarium* spp., it appears that these were also not the primary cause of canker.

Biocontrols

None of the biocontrols had efficacy for canker. The lack of efficacy could be associated with low soil temperatures. It has recently been discovered that Polyversum™ has an optimum temperature of 15 °C for efficacy. This could account for its lack of efficacy during winter when soil temperatures drop below 10 °C in the region (Oscar Villalta, pers. comm.). The lack of efficacy of MicroPlus™ could be associated with timing of the first application, which was applied after commencement of the trial due to its late arrival. This biocontrol, along with several others on the market, may be worth another trial in the interests of IPM, however, the number of applications may render these products uneconomical.

Winter and summer trials

The low incidence of canker in the summer mirrored the survey data of Chapter 2. Assuming the fungicides selected targeted these fungi, the low levels of canker on parsnips in summer do not appear to be associated with the fungi targeted. Although none of the treatments were effective in the summer trial, the little disease that was present may not warrant control economically. Any strategies to control canker need to be targeted at the February planted crop which grows over winter and is harvested in October. The 6–8 month growing season may not make chemical controls an option.

4.5 References

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Chapter 5

Cultivar evaluation

Summary

Field trials were conducted to evaluate the commercial variety Tusk and growers' own seed lines for resistance to canker. Tusk performed well in terms of both emergence and canker incidence, but several of the growers' seed lines were comparable in performance. The use of Tusk reduced canker incidence by 40 % at one site, and 55 % at the other, compared to the worst-performing seed line. Thus, cultivar resistance could be an effective tool as part of an overall IPM strategy to combat parsnip canker.

5.1 Introduction

Selection of resistant cultivars has been investigated as a way to combat fungal disease in a variety of crops including broccoli (*Albugo candida*, Minchinton *et al.* 2007) alfalfa (*Verticillium*, Papadopolous *et al.* 1989), chickpeas (*Phytophthora*, Dale and Irwin 1991) and carrots (*Pythium*, Cooper *et al.* 2006, Davidson and McKay 2001, Hiltunen and White 2002) to name a few. These studies have shown that an effective breeding program for disease resistance can have a profound influence on alleviating the incidence and severity of disease in vegetables.

5.2 Methods

5.2.1 Seed selection

Seed stock was collected from 4 Victorian growers and sown at two sites, Devon Meadows (Site 1) and Cranbourne East (Site 2). All seed stock was from the current year. The seed stock was untreated, except for the standard fungicide treatment with Thiram (Table 5.1). One exception was G3, which was coated with Apron XL (metalaxyl-m) and Maxim (fludioxinol) or Maxim XL to manufacturer specifications by Seed Solutions (4 Concorde Cres., Carrum Downs, Victoria). The new parsnip cultivar Tusk was supplied *gratis* by Terranova Seeds (Smithfield, NSW, Australia) for use in these trials.

Table 5.1 Seed lines used in the cultivar trials at Devon Meadows and Cranbourne East

Seed	Source
Tusk	Terranova seeds
G1	Cranbourne West
G2	Cranbourne East
G3	Rosebud
G4	Devon Meadows

5.2.2 Trial layout and location.

Trials were conducted at Devon Meadows and Cranbourne East. For the trial at Devon Meadows, 4 beds of two bays were utilised. Each bed was 35 m long and replicate plots were 8 m x 1.62 m, a total of 13 m² per plot. Seeds were sown in 4 rows per bed on 30 April 2007. There were 6 replicates for each of the grower seeds, and 8 replicate plots of Tusk. At Cranbourne East, 3 beds of one bay were utilised. Each bed was 80 m long and replicate plots were 9 m x 1.62 m, a total of 14.6 m² per plot. Seeds were sown in 4 rows per bed on 2 May 2007. There were 5 replicates of grower seeds and 4 replicates of Tusk.

5.2.3 Assessment

Both trials were assessed for seed emergence at 23 days after sowing. In both cases, a 40 cm strip was measured in the middle of each replicate plot and the total number of seedlings across the four rows was counted in that area. Canker incidence was determined at harvest after 214 days at Devon Meadows and 216 days at Cranbourne East). At both Devon Meadows and Cranbourne East, 6 m of each replicate plot was visually assessed for canker.

5.2.4 Statistical analysis

For each plot, the disease data consisted of the number of diseased roots in each plot and the total number of roots assessed. In addition, emergence in each plot was assessed for both trials. All data were analysed in Genstat® using linear models and maximum likelihood estimation.

5.3 Results

Table 5.2 Emergence (%) of cultivars at Devon Meadows and Cranbourne East

Seed Source	Devon Meadows	Cranbourne East
G1	41.6a	39.4a
G2	52.0b	35.2a
G3	78.4c	46.8a
G4	74.6c	51.4b
TUSK	105.3d	59.4b
Isd (approximate)	14.2	13.2

A similar pattern in emergence rate was seen at both sites, with significant differences apparent (Table 5.2). There were significant differences in seed lines in terms of emergence, with Tusk having the highest level of emergence. From best to worse at Devon Meadows, the sequence was: Tusk, G3/G4, G2, G1 and for Cranbourne East: Tusk and G4, followed by G3/G2/G1.

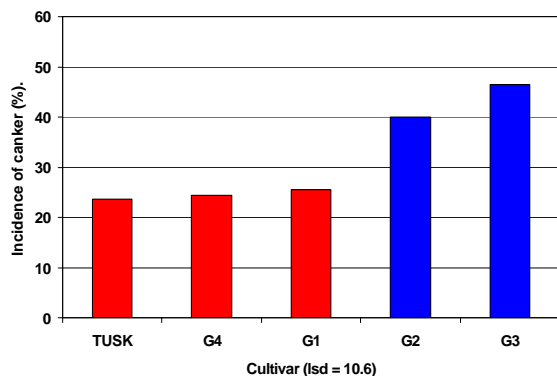


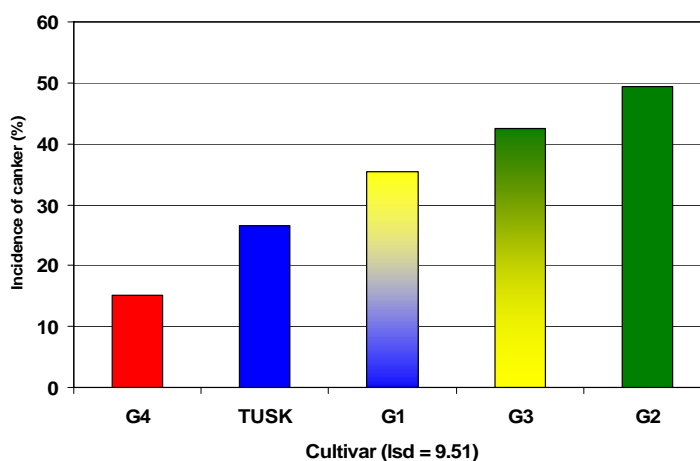
Fig. 5.1 Incidence of canker at Devon Meadows
Bars of different colours differ significantly at the 5% level.

Canker incidence at Devon Meadows ranged from an average of 23–46% (Fig. 5.1), with Tusk, G4 and G1 performing better than G2 and G3.

Canker incidence at Cranbourne East ranged from an average of 15–48%, with G4 exhibiting the least canker, followed by Tusk, G1, G3 and then G2 (Fig. 5.2).

Fig. 5.2 Incidence of canker at Cranbourne East

Bars of different colours differ significantly at the 5% level.



There was little difference in the severity of canker in the cultivars at harvest, although there was a significant difference in the incidence of canker as stated in the previous section (Figs. 5.3 A-E).

Fig. 5.3 Canker severities in all seed cultivars. LHS, healthy roots, RHS, diseased roots.



5.4 Discussion

All seed from growers' stocks has been bred for yield, resistance and appearance within their own soil systems over several decades. For example, the seed stock G3 has shown canker resistance in the soils that it was bred in (<13% at the height of canker incidence), but showed high levels of canker at both Devon Meadows and Cranbourne East (42.5 and 45 % respectively). Soil profiles showed that G3 seed was bred in a light, sandy soil with low organic carbon (0.5 %), whereas both Devon Meadows and Cranbourne east were medium soils with higher organic carbon content (1.25 and 1.3 % respectively, see Chapter 6).

Breeding for resistance to parsnip canker is complicated by the fact that this particular disease has more than one cause (see Chapter 1 for details). Screening and breeding for resistance to canker in parsnip, particularly in the UK has had some success (Cerkaukas 1986, Davis *et al.* 1989), with a 95% decrease in the incidence of canker in Phoma-induced canker. Breeding for resistance to canker caused by *Itersonilia* also imparted similar resistance to cankers caused by both *Phoma* and *Mycocentrospora* (Channon *et al.* 1970). However, in a separate study under a variety of different soil conditions, this resistance was not sustainable (Day 1978). This emphasises the importance of testing cultivars in all soil types and conditions.

5.5 Conclusions

- The new commercial cultivar Tusk performed well in terms of both emergence rate and canker incidence with significant reduction in canker levels (40 and 55 %) and higher emergence rates (300 and 210 % increase) compared to the worst seed stocks,
- No growers' seed lines, apart from G4, had consistently lower canker at the Devon Meadows or the Cranbourne East site. Variations in the performance of the growers' seed were surprising, since all seed stocks of Victorian growers are derived from the same source, B4 'Hollow Crown',
- Breeding resistant cultivars will alleviate canker incidence, not eliminate it, but could be useful as part of an overall IPM strategy.

5.6 References

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Chapter 6

Epidemiology

Summary

Preliminary monitoring of abiotic factors, including meteorological data, leaf sap and soil nutrient levels was undertaken to ascertain if there was any association with incidence of parsnip canker. In Australia, parsnip canker occurs after winter and is more common on wet sites with heavier soils. No individual nutritional factor appeared to be associated with parsnip canker. A survey of retired growers consistently pointed to a high pH in the soil for the reduction of canker.

6.1 Introduction

There is little information on environmental factors associated with parsnip canker, irrespective of its various causes. Most research on epidemiology of parsnip canker has concentrated on *Itersonilia*.

Itersonilia has an optimum temperature for growth of 20 °C (Cerkauskas 2002) and does not cause symptoms above 25 °C (Smith 1966). Parsnips were more prone to *Itersonilia* canker on heavy soils compared with light soils (Green and Hewlett 1950). Abundant soil moisture and low temperatures promote the disease whilst hot and dry conditions retard it (Cerkauskas 2002, Green and Hewlett 1950). Brown *et al.* (1964) reported canker was directly correlated with summer rainfall, but there was no correlation with nutrient deficiencies. Although the Australian crop which is highly susceptible to canker does not grow through summer, any high rainfalls in the Australian winter could conceivably promote canker. *Itersonilia* can infect roots, leaves, inflorescences and seed (Cerkauskas 2002). It can over-winter on infected parsnip roots, or as chlamydospores in soil for at least 12 months in Australia (Smith 1966).

There is little epidemiological information on the other fungi capable of causing parsnip canker, *Phoma complanata*, *Mycocentrospora acerina* and *Cylindrocarpon destructans*. *P. complanata* has a foliage phase and can survive in soils for 5 months (Cerkauskas 1987). *M. acerina* can grow and infect parsnips at temperature below 0 °C suggesting it is pathogenic under winter conditions (Channon 1965). These fungi represented approximately 20 % of confirmed pathogens isolated from parsnip cankers in Australia (Chapter 3).

Parsnip canker is widespread in southern Australia, with severe losses reported from Tasmania, Western Australia and Victoria. Losses are most common on crops planted in February and harvested in October, although significant losses from summer harvested parsnips have been reported in WA (F. Natoli, pers. comm., Chapter 2). In the worst cases, entire fields are abandoned. Interestingly Victorian growers report that canker can be sporadic, affecting one planting but not the next, even though both crops were planted with the same seed and received the same treatments.

This chapter reports on preliminary environmental, soil and nutrient data in parsnip crops, including phone surveys of observation by retired parsnip growers.

6.2 Material and methods

Environmental conditions within a parsnip crop were monitored; soil was analysed, sap tests of foliage were undertaken and area rainfall data were obtained from the Bureau of Meteorology (BOM) for preliminary correlation with incidence of parsnip canker in crops.

6.2.1 Weather data

A ModelT weather station (Western Electronic Design, Loxton, South Australia) was used to record average leaf wetness, air and soil temperature, relative humidity and total rainfall at 30 min. intervals. The leaf wetness sensor was placed in the parsnip crop and its height was adjusted as the crop grew. The monitored crop was located at Devon Meadows, and planted on 30/3/2007. The weather station was set up on 30/08/2007 and the crop was harvested on 30/10/2007. Area rainfall was obtained from the BOM for Moorabbin Airport about 30 km NW of the Cranbourne market garden area and 32 km from the weather station located at the Devon Meadows site.

6.2.2 Nutrient monitoring

Parsnip foliage was sampled once per month for 4 months, starting when plants were aged three months in a crop planted on 30/3/2007 at Devon Meadows, Vic. The weather station was also located on this site. Soils in parsnip crops at Devon Meadows, Cranbourne East and West and Rosebud were sampled at harvest when crops were being assessed for canker incidence (see Chapter 4). The foliage sap and soil were analysed by Serve-Ag Pty Ltd. (P.O. Box 690 Devonport 7310).

6.2.3 Grower interviews

A phone interview of 4 retired parsnip growers was undertaken during October 2007. Growers were asked about crop rotations, pH of soil and their practices for controlling canker.

6.3 Results

A snapshot of environmental conditions within the parsnip crop at Devon Meadows are shown in Appendix 1. There were issues with coordinating equipment to monitor conditions in crops. A thorough investigation of environmental effects on canker requires continual and systematic monitoring and sampling of crops with a high and low potential of canker for meaningful correlations.

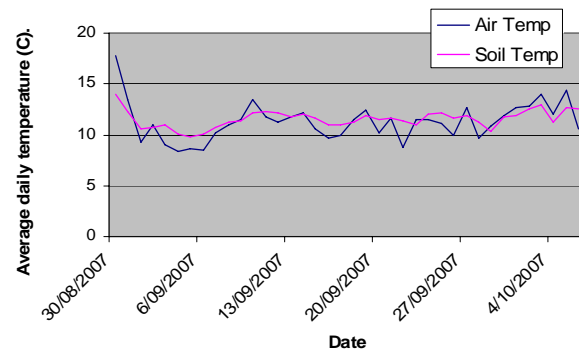


Fig 6.1 Average air and soil temperatures in the parsnip crop at Devon Meadows in 2007

6.3.1 Temperature

During winter, soil temperatures in the Devon Meadows and Cranbourne region dropped below 10 °C (Figs. 6.1 and 6.2).

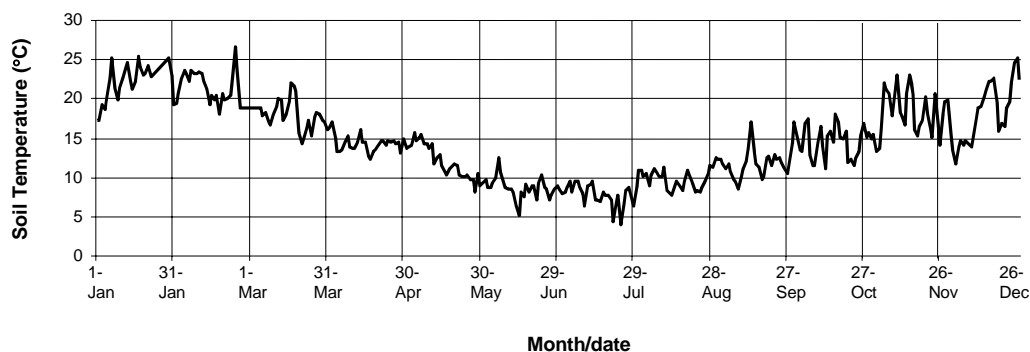


Fig 6.2 Average daily soil temperature for the Cranbourne area in 2002 (courtesy of Oscar Villalta)

6.3.2 Rainfall

Most of the water on the crop in Fig. 6.3 was irrigation, as well as rainfall. Rainfall at Moorabbin Airport which is 20 km NW of the Cranbourne west site, 25 km NW of the Cranbourne east site, 32 km NW of the Devon Meadows site and 48 km NNE of the Rosebud site is shown in Fig. 6.4. High rainfall events in June and July corresponded to a period when the growers suspected that canker appears in crops.

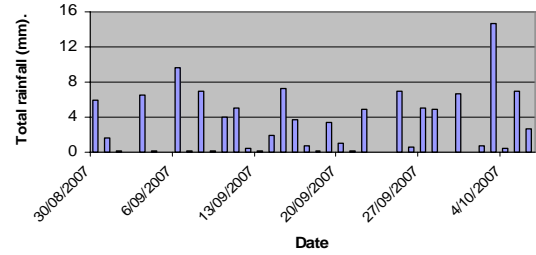


Fig 6.3 Daily rainfall for the parsnip canopy at Devon Meadows in 2007

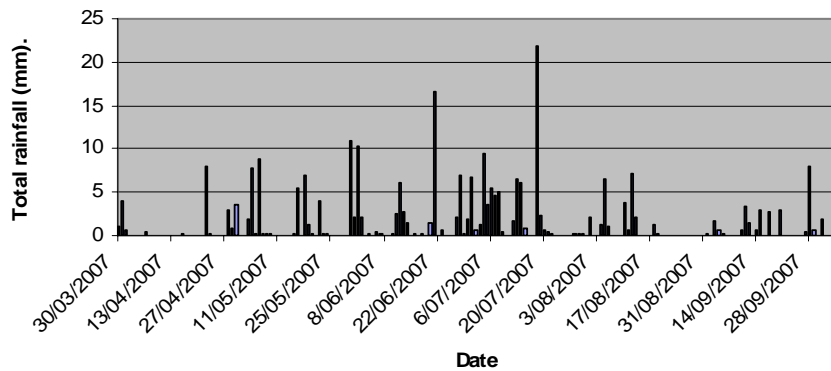


Fig 6.4 Daily rainfall data from Moorabbin Airport

6.3.3 Nutrition

The sap tests of parsnip foliage from the monthly surveys at the Devon Meadows ‘monitored site’ showed that nitrate (NO₃), sulphur (S), calcium (Ca) and molybdenum (Mo) levels decreased over time, whilst potassium (K), sodium (Na), phosphorous (P) and magnesium (Mg) levels either increased or remained relatively steady (Fig. 6.5). The growth stages are based on carrots because none exist for parsnips (Appendix 2). Table 6.1 details soil nutrient levels at harvest.

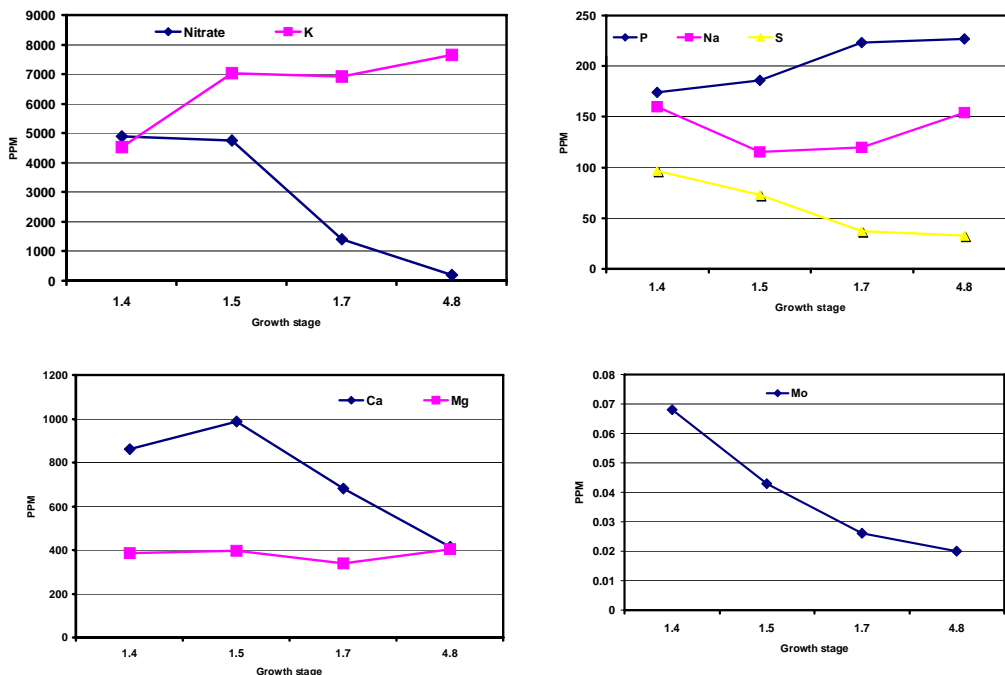


Fig. 6.5 Nutrient analysis of parsnip leaves from the Devon Meadows crop

Table 6.1 Analysis of soil nutrient levels and incidence of parsnip canker at 4 sites in 2007

Test	Devon Meadows				Cranbourne east	Cranbourne west	Rosebud	Range	
	Cultivar trial	Chemical trial	Monitored-wet soil (G4 seed)	Monitored-dry soil	Cultivar trial (G2 seed)	Chemical trial (G1 seed)	Chemical trial (G3 seed)	from	to
Incidence of canker (%)	24.85	12.8	22.94	6.83	49.2	35	16		
Organic matter	2.48	3.1	1.58	1.62	2.8	2.36	1		
Organic Carbon (Walkely-Black) %	1.24	1.55	0.79	0.81	1.29	1.18	0.5	2.5	4.0+
pH water 1:5 H2O	7.08	8.18	7.62	6.72	7.16	7.67	7.77	6	7
pH CaCl2 1:5 CaCl2	6.44	7.41	6.79	6.14	6.58	6.85	6.86	5.3	6.5
Lime requirement (to neutralise Al+H)	0	0	0	0	0	0	0	na	na
Electrical conductivity (EC) 1:5 dS/m	0.29	0.36	0.21	0.29	0.17	0.13	0.1		<0.15
Electrochemical Stability Index	0.08	0.07	0.04	0.06	0.05	0.11	0.04		>0.05
Chloride mg/kg	139	177	148	113	61	26	38		<200
P saturatin raton	1.15	0.91	1.36	1.17	1.86	0.97	1	0.062	0.23
Phosphorous mg/kg	543.7	421.3	513.8	283.2	617	433.6	251.7	30	70
Potassium mg/kg	219.8	343	121.4	35.7	99.4	200.1	89.4	230	330
Sodium mg/kg	60.4	77.3	27.4	103.9	41.7	15.1	11.3	10	40
Calcium mg/kg	1470.9	1768.6	1514.7	937.2	1573.4	1607.4	911	1500	2500
Magnesium mg/kg	168.9	328.5	186.8	90.2	136.1	154.9	43.4	180	360
Sodium mg/kg	90.4	153.8	115.8	86.2	70.6	27.4	30.6	20	80
Cation exchange Capacity mg/kg	10.7	13.1	9.9	7.2	10	10	5.3		
Calcium %	68.8	67.5	76.2	65.2	78.5	80.8	86.3	60	70
Magnesium %	13.1	20.7	15.5	10.4	11.2	12.9	6.8	12	20
Potassium %	5.2	6.7	3.1	1.3	2.5	5.1	4.4	4	6
Sodium %	3.7	5.1	5	5.1	3.1	1.2	2.5	0	<4
Hydrogen & Other %	9.1	0	0	18.00	4.8	0	0	13	20
Base saturation %	90.8	100	99	82.0	95.2	100	100	80	87
Ca/Mg Ratio	5.3	3.3	4.9	6.3	7	6.3	12.6	3	5
K/Mg Ratio	0.4	0.3	0.2	0.1	0.2	0.4	0.6	0.3	0.5
Zinc mg/kg	15.66	9.81	16.83	14.24	23.2	10.89	9.35	2	10
Iron mg/kg	190.1	218.2	175.8	156.9	223.2	213.7	129.8	30	120
Manganese mg/kg	18.9	20	19.2	18.7	35.2	14.1	19.5	15	60
Copper mg/kg	5.39	4.08	5.51	4.66	8.66	4.9	4.37	2	8
Boron mg/kg	1.56	1.6	1.2	0.68	1.25	1.73	0.92	2	5

Yellow bars, data under the range; **orange bars**, data over the range.

No individual factor appears to be consistently associated with either high or low levels of parsnip canker in the industry. On one property the wetter site had more canker than the drier site (22.94 % compared to 6.83 %), even though the crop was the same in every other respect.

6.3.4 Grower interviews

Retired growers consistently reported high pH, long crop rotations and drier soils were important for the successful management of parsnip canker (Table 6.2).

Table 6.2 Comments on parsnip canker management from 4 retired parsnip growers

Grower	Comments
A	Long crop rotation. Rotated parsnips every 2 years with 3–4 crops of either broccoli, lettuce and onions. Limed parsnip crop at ½ t/acre at pH 6.5. Top dressed with fowl manure to prevent crusting after sowing and not again. Noticed that spotting was associated with high P.
B	Green manure, prepared ground with disc, added lime (GBA, 9 t/acre), kept the soil pH high, up to pH 9. Leaves were yellow but there was no canker. Hungry ground had less canker. Canker was a problem when the ground was too damp and there was plenty of top growth.
C	Preparation of ground very important. Found the addition of potash reduced canker. <ol style="list-style-type: none"> (i) Murate of potash applied at 3 bags+1 bag nitrate of soda to 2/3 acre. (ii) Harrowed lime 10 days after potash. (iii) Added straw to ground, disced to incorporate and watered for 7 days. (iv) Fowl manure on top of straw and disced in. Rotated crop with leeks and peas.
D	Applied twice the amount of lime and sulphate of potash as required. Rotated parsnips every 3 years and always planted after potatoes. Normally ground was pH 6 but grew parsnips in soil up to pH 10. <ol style="list-style-type: none"> (i) Lime applied to potato crop (ii) 1 week before planting parsnips, work in base lime and potash (iii) At sowing top dress with fowl manure. (iv) Fertilize a little and often.

6.4 Discussion

No individual nutritional factor appeared to be associated with parsnip canker. Canker was worse on the crop grown through winter (Chapter 2) and there is some evidence from the monitored site that canker incidence was higher on wetter ground. This observation is consistent with those of Green and Hewlett (1950) and Cerkauskas (2002). The anecdotal comments from growers concerning high pH reducing canker require further investigation, but this is unlikely to be a ‘silver bullet’ based on current observations. The drop in calcium and low boron levels are of concern because the former is associated with cell wall structure and storage performance, while the latter is a co-enzyme. This preliminary report indicates that more work is required on soil moisture, soil pH and some soil nutrients such as calcium and boron.

Weather data

Parsnip canker is first noticed in the February planting at the three-quarter growth stage, about June/July. During this period, soil temperatures are low and there is often heavy rainfall. At these temperatures many fungi can be inactive, whilst the oomycetes, such as *Pythium* spp., which are active at a wide range of temperatures, can still be pathogenic (Minchinton *et al.* 2006).

Nutrients – sap tests

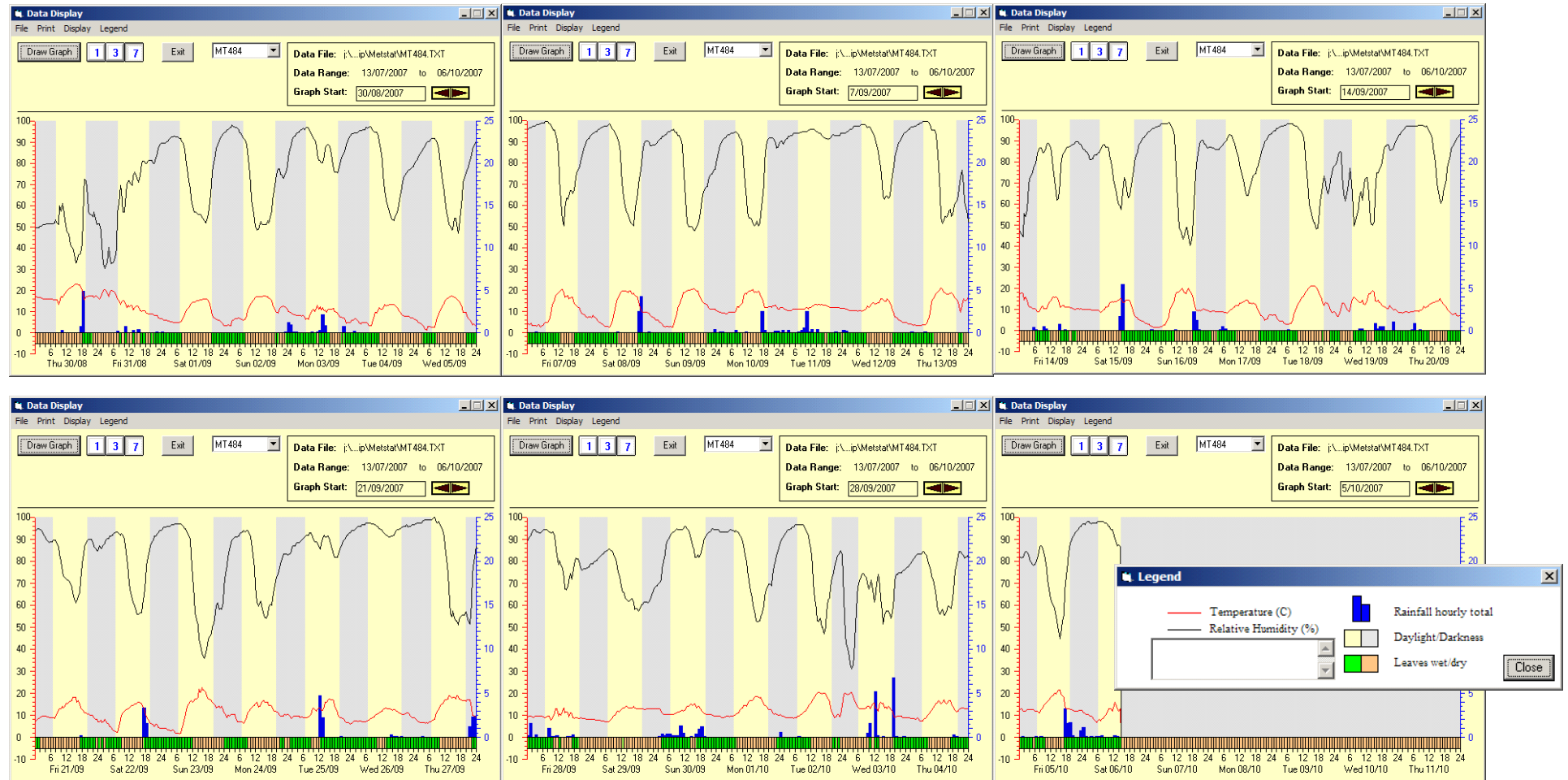
The drop in nitrate is expected, as the plant takes it up and converts it to ammonia for use. Certainly, excess nitrate levels are not desirable in a crop. According to Dr Doris Blaesing (Project Management, Serve-Ag Pty Ltd, PO Box 690, 6181 Frankford Rd, Devonport 7310), ‘Apart from calcium, sulphur and some trace elements, the crop was well supplied with nutrients. Calcium is important for storage performance. We are finding that sulphur correlates well with sap Brix in most crops’.

6.5 References

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- Cerkauskas RF (1987) Phoma canker severity and yield losses in parsnip. *Canadian Journal of Plant Pathology* **9**, 311–318.
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- Minchinton EJ, Auer DPF, Martin H, Tesoriero L, Thomson F, Trapnell LN, Foresberg L, Nadesan S, Vujovic S (2006) Scoping study to investigate management of root-rot diseases in parsley. Horticulture Australia VG04025. Primary Industries Research Victoria, Knoxfield Centre, 87pp.
- Smith PR (1966) Seed transmission of *Itersonilia pastinaceae* in parsnip and its elimination by a steam-air treatment. *Australian Journal of Experimental Agriculture and Animal Husbandry* **6**, 441–444.

6.6 Appendix 1

Meteorological data from the trial at Devon Meadows in 2007



Appendix 2

Growth stages of carrot (template for parsnip by Nu-test)



Carrot



Timing: Begin sampling at stage 1.5 (Vegetative growth), continue fortnightly until stage 4.6

Sample Volume: 30 plants for stage 1, later 15-20 plants. Return to the same representative sampling area for subsequent samples.

Plant part for analysis: Send entire tops of young plants. Later, the root and leaflets should be removed, retaining the center petioles and stalks for testing.

Primary Stage	Secondary Stage	Description (old stages)	Comments
1			Leaf Development (main shoot)
	1.5	Vegetative growth	(1) 5th true leaf unfolded.
	1.9		9 or more true leaves unfolded.
4			Development of harvestable vegetable plant part
	4.1	Bulking up of roots for harvesting	Roots begin to expand (diameter > 0.5 cm)
	4.2		(2) 20% of the expected root diameter reached.
	4.4		40% of the expected root diameter reached.
	4.5		(3) 50% of the expected root diameter reached.
	4.6		60% of the expected root diameter reached.
	4.8		(4) 80% of the expected root diameter reached.
	4.9		Expansions complete; typical form and size of roots reached.
5			Inflorescence emergence
	5.1	Stages for seed production	Main shoot begins to elongate
	5.5		First individual flowers of main shoot inflorescence visible (still closed).
6			Flowering
	6.5		Full flowering: 50% of flowers open.
7			Development of fruit
	7.5		50% of fruits have reached typical size
	7.9		Fruits have reached typical size.
8			Ripening of fruit and seed
	8.9		Fully ripe: seeds on the whole plant of typical colour and hard.
9			Senescence
	9.7		Plants or above ground parts dead.
	9.9		Harvested product (seeds).

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A Member of Leading in Agribusiness



Chapter 7

Technology transfer and recommendations

Summary

This chapter reports the benefits of a project advisory group established to oversee research projects. This group increased communication and cooperation between growers, researchers and allied support businesses and resulted in an accelerated impact of research and development within the parsnip industry. Recommendations for future research are presented.

7.1 Introduction

The research reported herein is the result of collaboration between parsnip growers, industry advisory groups and project steering committees. These groups consisted of vegetable growers, crop consultants and chemical resellers, with diverse experiences which they brought to the project. The groups provided an opportunity for researchers to describe their approach and current progress thus promoting the impact of research and development projects. The advisory groups also enabled growers and allied industries to ensure that their needs were being met by the research project. The advisory group approach worked very well and is DPI's preferred method of involvement with the vegetable industry. This interaction and collaboration with growers and vegetable industry development officers (IDOs), along with the subcontracting of sections of work to industry experts has been of enormous benefit to the project. The IDOs identified parsnip growers in other states. The advisory committee encouraged the researchers to promote results of the research to growers nationally in industry publications.

7.2 Industry advisory group

The Department of Primary Industries Victoria took the approach of inviting growers and private allied support business representatives to volunteer their time and join with researchers to plan and discuss parsnip disease issues first hand. Not all growers were in the position of being able to volunteer their time due to the demands of growing and marketing vegetables and consequently the researchers are extremely grateful to those who were able to contribute. The parsnip growers were very supportive of the project and provided many field sites for trials, which was enormously appreciated by the researchers.

The advisory group members who supported project VG06046 were:

Silvio and Glenn Favero – Market Gardeners, Hillcrest Farm, Cranbourne, Vic.

Peter, Darren and Paul Schreurs – Market Gardeners, Peter Schreurs and Sons, Devon Meadows, Vic.

Karl Riedel – Vegetable Crop Agronomist, EE Muir and Sons, Cranbourne, Vic.

Russell Lamattina – A and G Lamattina and Sons Market Gardeners, Boneo, Vic.

Mark Milligan – Farm Manager, A and G Lamattina and Sons Market Gardeners, Boneo, Vic

Ross Arnott – Market Gardener, R, C, G, and D Arnott, Boneo, Vic.

Joe Kelly – Market Gardener, Tullamore Gardens, Cranbourne, Vic.

Glenn Moore - Market Gardener, Scottsdale, Tas

Carlo Galati - Market Gardener, Galati Produce, Anketell, WA.

Figaro Natoli - Market Gardener, Natoli Produce Farms, Wanaroo, WA.

7.3 Dissemination of information to industry

The current project was enthusiastically received by all growers and new insights into this devastating disease were gleaned. Adults acquire information in different ways such as reading, talking and visual cues. Some forms of information distribution will be more useful or accessible than others. There are many methods for distributing information to growers, such as field days, industry publications,

workshop meetings and steering committees. During the course of this project we have endeavored to utilize a broad range of information delivery methods and take every opportunity to report to industry. Records of publications and extension activities are listed below.

Publication list

Extension:

Minchinton L (2006) Growers, researchers seek parsnip canker control. Good Fruit and Vegetables, Vegetableplatter, p. 17.

Minchinton L (2006–07) Parsnip growers check their R and D project. Victorian VegeLink Newsletter **28**, 5.

Minchinton L (2007) Parsnip growers check their R and D project. Good Fruit and Vegetables, January, Vegetableplatter, p. 18.

Minchinton E (2006) Parsnip canker project under way. Good Fruit and Vegetables, Vegetableplatter, p. 11.

Auer D, Minchinton E, Cunnington J, and Thomson F (2007) Identification of the extent and cause of parsnip canker. Poster, AUSVEG Conference Sydney 29–31 May 2007.

Auer D, Minchinton E. First steps in parsnip canker management. In press.

Workshops:

5th May 2006: Parsnip Canker Steering Committee Meeting No 1, Vic.

15th September 2006: Parsnip Canker Steering Committee Meeting No. 2, Vic.

20th April 2007: Parsnip Canker Steering Committee Meeting No. 3, Vic.

14th December 2007: Parsnip Canker Steering Committee Meeting No. 4, Vic.

Field Day:


8th October 2007: Field day discussing the evaluation of trials to combat parsnip canker

Scientific:

Auer D, Minchinton E, Cunnington J, and Thomson, F (2007) Identification of the extent and cause of parsnip canker. Abstract. Proceedings 16th Biennial Australian Plant Pathology Society Conference. Back to Basics: Managing Plant disease. Adelaide 24–27 September 2007, p. 198.

DEPARTMENT OF PRIMARY INDUSTRIES - VICTORIA

An invitation to learn more about:



Identification of the extent and cause of parsnip canker


Steering Committee Meeting for HAL Research project VG05045

Research supported by your Vegetable Industry Levy and Horticulture Australia

Speakers:
Liz Minchinton-DPI-Knoxfield
 A briefing on the scoping study to investigate management of canker in parsnip: HAL VG05045
Desmond Auer-DPI-Knoxfield
 Overview of organisms causing canker in parsnip.

When: 4.30 – 5.30 pm Friday 5th May
 Where: Annsid Golf Club – Cranbourne
 1000 Frankston-Cranbourne Road
 Melway Map 131 D5


Contact: Elizabeth Minchinton - 0427 01 2460
 Slobodan Vujovic – 03 9210 297



DEPARTMENT OF PRIMARY INDUSTRIES - VICTORIA

Second Steering Committee Meeting for HAL Research project VG05045

Research supported by your Vegetable Industry Levy and Horticulture Australia




Identification of the extent and cause of parsnip canker

Speakers:
Liz Minchinton-DPI-Knoxfield
 An update on the scoping study to investigate management of canker in parsnip: HAL VG05045
Desmond Auer-DPI-Knoxfield
 An update on organisms causing canker in parsnip.


When: 1.00 – 2.00 pm Friday 15th September
 Where: Blue Hill Restaurant Village
 125-135 Berwick-Cranbourne Road
 CRANBOURNE VIC 3997
 Melways Map 134 D6

Contact: Elizabeth Minchinton - 0427 01 2460
 Slobodan Vujovic – 03 9210 297



DEPARTMENT OF PRIMARY INDUSTRIES - VICTORIA

An invitation to attend a review of the following project:



Identification of the extent and cause of parsnip canker



Review Steering Committee Meeting for HAL Research project VG05045

Research supported by your Vegetable Industry Levy and Horticulture Australia

Speakers:
Liz Minchinton-DPI-Knoxfield
 A review on the scoping study to investigate management of canker in parsnip: HAL VG05045
Desmond Auer-DPI-Knoxfield
 An update on organisms causing canker in parsnip.


When: 2.00 – 3.00 pm Friday 20th April, 2007
 Where: The Boardroom
 A&G Lamattina & Sons Pty. Ltd.
 125 Bowen Road Bonoro VIC 3939
 Melways Map 170 D12

Contact: Elizabeth Minchinton - 0427 01 2460
 Slobodan Vujovic – 03 9210 297

Veg Cheque
 extension for the vegetable industries

An invitation to a Field Walk for Parsnip Canker



Research supported by your Vegetable Industry Levy, Horticulture Australia and DPI-Victoria.


Where: 1380 North Road, Devon Meadows (Peter Scheurer's property, starting at the sorting shed)

When: Monday 8th October 2007 any time between 10am and 12 pm (Morning Tea provided)

Speaker: Desmond Auer DPI Knoxfield

This is an opportunity to look at current trial(s) and discuss recent progress with the research scientists on the current study to investigate the extent and cause of parsnip canker (VG05045)

For more details contact: Desmond Auer on 9210-9352, Liz Minchinton on 9210-9224 or Slobodan Vujovic on 9210-9297




Veg Cheque
 extension for the vegetable industries

An Invitation to a Parsnip Canker Workshop



Research supported by your Vegetable Industry Levy, Horticulture Australia and DPI-Victoria.

Where: E. E. Muirs Cranbourne
 Cnr Sth Gippsland Hwy 7 Huon Park Rd, Cranbourne
 Melway Map 129 G7

When: Friday 14th December 2007
 1.30 – 3.00 pm (afternoon tea provided)

Speakers: Desmond Auer DPI Knoxfield
 De Liz Minchinton DPI Knoxfield
 Rob Velthuis Agriculture Consultant

This is an opportunity to look at current trial(s) and discuss recent progress with the research scientists on the current study to investigate the extent and cause of parsnip canker (VG05045)

For more details contact: Liz Minchinton on 9210-9224
 Des Auer 03 9210 9352
 or Slobodan Vujovic on 9210-9297



IDENTIFICATION OF THE EXTENT AND CAUSE OF PARSNIP CANCKER

Desmond P.F. Auer, Elizabeth Minchinton, James Cunnington and Fiona Thomson
DPI Victoria-Knoxfield Centre, Private Bag 15 Ferntree Gully DC Victoria 3156



INTRODUCTION

Growers report that canker causes up to 80% crop losses in parsnip in Australia. The presumptive causal agent for this disease is the fungus *Itersonilia perplexans*.

This investigation details an ongoing HAL-funded project to determine both the extent and the cause of parsnip canker in Australia.

Fungal isolations & pathogenicity testing

Parsnip roots with canker symptoms were sampled for the presence of fungi to establish the fungus responsible for canker.

Pathogenicity of purified fungal isolates from cankers was tested by Koch's postulates.

In over 400 individual isolates, only 24% (97) produced canker-like symptoms on fresh parsnip roots. Positive isolates included *Itersonilia perplexans*, *Acremonium*, *Cylindrocarpon*, *Fusarium*, *Microdochium*, *Mycocentrospora*, *Phoma*, *Pythium* and *Rhizoctonia*.

Canker surveys

Four properties in Victoria and properties in Western Australia and Tasmania were sampled seasonally (autumn, spring, summer, winter) in 2006.

Visual surveys for parsnip canker incidence were performed on washlines (Fig. 1).

Canker incidence was highest in spring, but this varied between properties (Fig. 2).



Figure 1

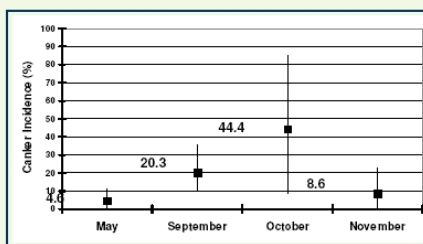


Figure 2 Average and range of incidence of canker during 2006.

Monetary losses due to canker

Potential monetary losses due solely to canker can be established using Melbourne Market data on parsnip prices (Figs. 3, 4). Losses at the height of canker incidence were estimated at \$A1.3M.

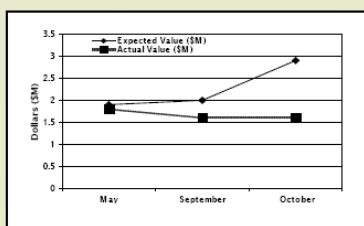


Figure 3 Expected and actual value based on Melbourne Market parsnip prices (2006)

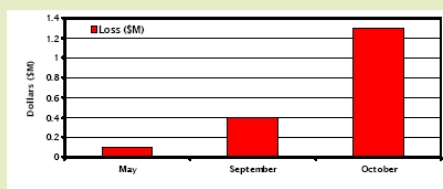


Figure 4 Losses (\$M) due to canker alone (2006)



Fig. 5

Figure 5 illustrates fungi which caused rot only on damaged roots eg. *Itersonilia*, *Fusarium*, *Rhizoctonia*, *Microdochium*. Parsnip inoculated with isolated fungi. LHS: Damaged root; RHS: Undamaged root (for Figs. 5-7).

Figure 6 illustrates fungi which caused rot only on the crown of infected parsnip roots. eg. *Fusarium*, *Cylindrocarpon*, *Acremonium*, *Pythium*. Parsnip inoculated with isolated fungi. LHS: Damaged root; RHS: Undamaged root.



Fig. 6



Fig. 7

Figure 7 illustrates fungi which caused rot on all areas of both damaged and undamaged roots eg. *Mycocentrospora*, *Phoma*, *Fusarium*. Parsnip inoculated with isolated fungi. LHS: Damaged root; RHS: Undamaged root.

CONCLUSIONS

Canker incidence is highest in spring months, which corresponds to autumn plantings (crop is 3-7 months old).

Apart from *Itersonilia perplexans*, a range of fungi including *Acremonium*, *Cylindrocarpon*, *Fusarium*, *Microdochium*, *Mycocentrospora*, *Phoma*, *Pythium* and *Rhizoctonia* cause canker-like symptoms on fresh parsnip roots.

FURTHER WORK

Management strategies to combat parsnip canker are being formulated.

7.4 Recommendations

The major recommendations to growers from this work are:

- Long-term management of the disease may rely on resistant cultivars.
- On specific sites, 2 applications of metalaxyl may be effective for controlling parsnip canker. The influence of metalaxyl on canker incidence suggests water moulds (oomycetes) are involved in the development of parsnip canker, but not consistently at all sites.
- Avoid planting parsnips in February. Parsnip canker levels were high in spring-harvested crops, representing crops sown in February–March. This reiterated previous anecdotal evidence from parsnip growers.
- *Itersonilia perplexans* is not the only fungus responsible for parsnip canker in Australia, with the possibility of a consortium of fungi being responsible. The complexity of the problem highlights the need for a more holistic approach to the management of this disease.

Areas of future research which would benefit the industry are:

Future research needs to have a national focus due to the extent of parsnip canker in southern Australia. We suspect that a complex of organisms is responsible for parsnip canker in Victoria, with the water moulds, such as *Pythium* spp., having a leading role in predisposing parsnips to canker. We also suspect soil moisture, soil temperature, pH and nutrients may have an influence on canker development. Future work on canker should be aimed at how and when the disease is occurring, conditions promoting it, control options including IPM, as well as extension.

Areas of future research of benefit to industry should:

- Determine if combinations of pathogens, such as *Pythium* spp., are predisposing parsnips to canker.
- Determine the influence of pH, nutrients and irrigation on parsnip canker, as they could be predisposing parsnips to canker.
- Establish the electrostatic behaviour of *Pythium* zoospores, as Ca^{++} interferes with root infection by zoospores.
- Establish when canker first attacks the crop and tailor control measures to this time.
- Establish optimal seed treatment regimes.
- Test a range of biocontrols for efficacy against canker.
- Test parsnip cultivars for canker tolerance under a wider variety of conditions, nationally.
- Identify cultural, biological and alternative soft chemical controls.
- Conduct systematic surveys for carrot rust fly as it is a biosecurity issue.
- Develop a national management strategy for parsnip canker and ensure it is economical.
- Benchmark control measures and establish the economics of IPM strategies
- Produce and publish a user-friendly protocol on best practice integrated management of parsnip canker.