Violet Root Rot of Carrots – Summary of fumigation trials, 2007-2010

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Plant & Food Research, Hawke's Bay

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Violet root rot of carrots – summary of fumigation trials, 2007-2010 Report to Horticulture NZ Incorporated, Project SFF HRNZF 07/048

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Violet root rot (VRR), caused by the fungal pathogen *Rhizoctonia crocorum*, causes substantial economic losses and threatens the long-term viability of the carrot industry in the Ohakune region. Previous attempts at control have been largely unsuccessful. This report summarises the third year of a three-year programme to evaluate the potential of commercial fumigants to control VRR.

In the 2007/08 season, soil fumigation trials in heavily infested Ohakune carrot fields failed to provide commercially useful VRR control. Deficiencies in fumigant placement and distribution, plus inadequate sealing of the soil were identified as likely reasons for the failure.

In 2008/09, trials using VRR-infested soil in plastic crates demonstrated that Telone®, chloropicrin, and Fumasol™ were each capable of killing the pathogen under suitable conditions. Replicated field trials in Ohakune and Karioi, established in November 2008, proved that joint applications of Fumasol with TriForm60 or chloropicrin, combined with improved soil sealing with a heavy tyre-packer, could give excellent control of VRR. However, there were significant problems with phytotoxicity causing suppression of carrot growth in some treatments, most notably those including Fumasol. The phytotoxicity was attributed to the very high fumigant rates used, sowing of seed just over 3 weeks from fumigation, and the minimal cultivation of soil between fumigation and sowing.

The 2009/10 trials built on previous results, and aimed to fine-tune fumigant rates, combinations, and application techniques, plus investigate factors contributing to phytotoxicity problems. Trials comparing various fumigant rates and combinations were established in the Sue (Karioi) site using plots from the previous year's trial, and a new site in an A.S. Wilcox block near Ohakune. Also included in the Sue site were treatments designed to test the carry-over effect of treatments into a second season following fumigation and carrot cropping the previous year. A further trial at the Sue site investigated planting time, soil cultivation to aid gas release, and subsequent growth suppression caused by gas residues.

Main fumigant rate and combination trial (Sue site)

The main trial in the Sue site was established in November 2009 in 'untreated control' areas from the previous season, and thus had high inoculum pressure. Combination treatments of Fumasol™ at high or low rates and chloropicrin or TriForm®60 at high or low rates were compared with TriForm60-only and untreated control plots. Fumasol at rate of 30 ml/m^2 (low) or 50 ml/m^2 (high) was applied to the surface and hoed in to a depth of 10 to 15 cm. TriForm60 or chloropicrin, at rates of 33 g/m^2 (low) or 55 g/m^2 (high), was shank-injected to 25 to 30 cm, then plots were rolled with a heavy tyre-packer to seal the surface. 'Paramount' carrot seed was sown 6½ weeks after fumigation.

Weed seeds and *Phytophthora* spores were buried during fumigation, retrieved 7 days later, and tested for viability. Survival rates were high in untreated control plots, but very low in all combinations of Fumasol and TriForm60 or chloropicrin. Survival was high near the soil surface in TriForm60-only plots, but low at deeper in the soil profile. Natural weed numbers, measured in plots 5 weeks after fumigation, were significantly lower in all fumigation treatments than in untreated controls. Early carrot plant growth was slightly slower in some fumigation treatments, probably a result of phytotoxic effects from fumigant residues. By mid-season this difference in growth was no longer apparent.

VRR disease incidence was assessed in April and June 2010. Compared with untreated controls, there was significantly lower disease incidence in all treatments combining Fumasol with either TriForm60 or chloropicrin. There was a trend toward lower disease incidence where rates of the respective fumigants were high. The TriForm60-only treatment did not control disease.

A.S.Wilcox trial

A trial on an A.S. Wilcox site in Ohakune was established in November 2009 on ground that had violet root rot problems in a potato crop grown the previous season. A replicated trial testing various combinations of Fumasol with TriForm60 at different rates was established. The ‘high’ and ‘low’ rates tested were similar to those at the Main Sue rate & combination trial, with added ‘very low’ rates (Fumasol at 20 ml/m^2, TriForm60 at 22 g/m^2). Survival of seeds and *Phytophthora* spores buried in plots during fumigation was low in all treatments, but high in untreated controls.

Violet root rot incidence, assessed in June 2010, was moderately high in untreated control plots (mean 16.6%). VRR incidence was significantly less in all but one of the Fumasol/ TriForm60 treatments. With the one exception, all fumigant treatments averaged less than 4% disease.

Re-treatment/carry-over effect trial (Sue site)

Plots that had been fumigated in the previous season’s trial were either re-fumigated or left untreated in November 2009. There was good evidence for a carry-over effect from the previous season’s fumigation treatment. In plots that had no fumigation in the second season, there was significantly lower disease incidence in plots that had been fumigated the previous year, compared with those untreated in the previous season. In plots fumigated in the previous season, a combined treatment of TriForm60 and Fumasol (both at the low rate) in the second year reduced disease incidence compared with that in similar plots left untreated in the second year. Re-treating plots with a low rate of TriForm60 alone was of no apparent benefit.

Plant-back trial

To test factors contributing to early season stunting of plants following fumigation, a trial with various hoeing and sowing regimes was established on the Sue site in November 2009. Treatments included high rates of Fumasol and TriForm60 applied as sole treatments, and combined treatments of the two products at both low and high rates. Carrot seeds were sown 2, 3, 4 or 5 weeks after fumigation, into plots that had been hoed at different times between fumigation and sowing. Fumigant gas concentrations in soil, measured periodically in hoed and un-hoed plots, indicated that gas release (Telone in particular) was more rapid in hoed than in un-hoed plots.

In comparisons made within the same hoeing/sowing regime, carrot seed germination and percentage plant establishment were similar across all fumigant treatments and untreated controls, i.e. there was no evidence for reduced germination with any of the fumigants. In contrast, early plant size was reduced, compared with untreated controls, in all fumigation treatments. The growth reduction was generally the greatest in the high Fumasol/high TriForm60 treatment, and least in the Triform60-only treatment. There was no obvious pattern in the magnitude of the growth reduction across the various hoeing/sowing regimes, with early growth stunted, compared with untreated controls, under all regimes. Despite the observed inhibition in early seedling growth, within a few weeks these differences were no longer apparent, i.e. the plants had outgrown any early inhibition. Early season growth inhibition was also noted in some fumigation treatments in the main Sue rate and combination trial, but once again these differences were outgrown later in the season.

Although plant-back remains a potential problem with fumigation, there are ways to minimise its impact, such as use of lower fumigant rates, long waiting periods before sowing, plus cultivation and aeration of soil between fumigation and sowing.

Overall, the various trials carried out over the past three years have identified fumigant treatment combinations that will effectively control violet root rot. Joint application of chloropicrin or TriForm60 (shank-injected), with Fumasol (surface-applied and incorporated) followed by soil sealing using a heavy roller, has provided good control of VRR in heavily infested fields. A range of fumigant rates were effective, but in general higher rates provided better disease control. Selection of what rates to use will depend on site history and disease pressure. There will have to be a balance between higher rates potentially providing better VRR control and perhaps giving multiple-year effectiveness, against factors such as cost, longer waiting periods, and more potential for phytotoxicity problems. Benefits other than VRR control, such as nematode, weed and general pathogen control should also be considered.

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Introduction

Violet Root Rot (VRR), caused by the fungal pathogen *Rhizoctonia crocorum*, is a significant disease of carrots in the Ohakune region. It causes substantial economic losses and threatens the long-term viability of the carrot industry in the region. The pathogen has a relatively wide host range, including other crops such as potatoes and parsnips. Symptoms on carrot start as a superficial, purplish rot, later progressing to rot the carrot completely, leaving only a blackened outer sheath (Figure 1).

Figure 1. Symptoms of violet root rot on carrot. Left: diseased patch in early May. Centre: early stage of infection, showing violet colour. Right: diseased carrots, with just the blackened outer sheath remaining.

Past attempts at control have been unsuccessful, and the magnitude of the VRR problem has increased with time. The primary aim of the current research programme is to determine whether or not soil fumigation with commercially available fumigants is capable of controlling VRR in Ohakune carrot fields. There is a need to find an effective short- to medium-term solution to the VRR problem, while finding out more about the pathogen, which could ultimately lead to integrated and more sustainable control.

Fumigation trials carried out in Ohakune in the 2007/08 and 2008/09 season have been fully reported previously (Horner et al. 2008, 2009), but are briefly summarised here. The trials identified problems with fumigant application and efficacy for VRR control, but also potential solutions. The 2009/10 field trials, reported in detail below, aimed to validate and ‘fine tune’ successful application systems identified in the 2008/09 trials, to address problems with phytotoxicity observed in previous trials, and to determine whether there were beneficial carry-over effects from treatment in the previous season.

Overview of 2007/08 trial

Two trials sites with a history of severe VRR infection (the Whale site near Ohakune, and the Sue site at Karioi) were selected for testing of a range of soil fumigants. Fumigants were applied in November 2007, to plots laid out in replicated block designs. Treatments were: untreated control, TriForm®60 (500 kg/ha), TriForm®60 (300 kg/ha), Fumasol™ (800 L/ha). TriFrom60 treatments were shank injected and sealed by rolling, and Fumasol was applied to the soil surface then rotary hoed in. On the Whale site there was also a TriForm60 300 kg/ha treatment covered with polyethylene film at fumigation.

Fumigant gases in the soil were monitored for up to 3 weeks using Gastec® tubes. Data indicated that with fumigants shank injected at 25 cm, the chloropicrin component of the Telone C60 in particular failed to reach high concentrations in the top few centimetres of soil. In contrast, the surface-applied and incorporated Fumasol reached high concentrations in surface layers, but not at greater depths. Bioassays using *Phytophthora* inoculum and ryegrass seed buried at various depths were used to determine the biological effectiveness of the various fumigants, and results reflected the gas concentration data. The shank-injected Telone treatments failed to kill Phytophthora or ryegrass seeds, particularly in the top few centimetres of soil. Fumasol was more effective, with almost 100% kill of ryegrass seed and *Phytophthora* near the surface. Natural germination of weed seeds and sprouting of volunteer potatoes were assessed following fumigation, and reflected the inoculum bioassays and gas readings; Fumasol was the most effective product at controlling weeds.

A carrot crop was sown four weeks after fumigation, and disease progress was monitored throughout the season. Assessments of carrot root disease were made in mid-March and early July 2008. The first above-ground symptoms of VRR were evident in March. By this time there was substantial infection below ground, with 80% of carrots in untreated control plots infected on the Whale site, and 22% infected on the Sue site. Fumasol appeared the most effective fumigant at reducing disease incidence, but because of wide variability between treatments, none of the observed differences were statistically significant. By July, disease incidence had increased further, with 90% and 49% infection of carrots in untreated plots at the Whale and Sue sites, respectively. On average, disease was lowest in the Fumasol-treated plots, but none of the differences were statistically significant. None of the treatments gave control effective enough to allow commercially viable harvest of the crop.

The disappointing result was attributed to extremely high disease inoculum pressure and a failure to get critical fumigant gas concentrations throughout the soil profile. The TriForm60 treatment, while possibly effective at the depth of application, failed to reach concentrations adequate to kill disease inoculum near the surface. The very open Ohakune soils probably contributed to the rapid loss of gas from the surface layers. In contrast, Fumasol effectively killed weed seeds and fungal inoculum near the surface, but failed to penetrate beyond the depth of incorporation.

The 2007/08 trials are reported fully in Horner et al. (2008).

Overview of 2008/09 trial

The 2008/09 season's trials aimed to determine whether commercial fumigants are capable of killing the VRR pathogen, and to test improved application techniques and fumigant combinations in infested fields.

To determine the potential of commercial fumigants (Telone®, chloropicrin, Fumasol™) to kill soil-borne inoculum of violet root rot, soil from heavily VRR-infested sites was fumigated in 45-L plastic crates, and then sown with carrot seed. Subsequent VRR disease incidence in carrots was greater than 90% in unfumigated control crates. No VRR was detected in any of the fumigated crates, demonstrating that each of the three fumigants is capable of killing the pathogen under suitable conditions.

Replicated trials were established in November 2008, on the same two Ohakune sites as in the previous season. On the Sue site, Tri-Form®60 (40% Telone, 60% chloropicrin) was shank-injected to 30 cm, combined with either a plastic (30 µm polyethylene) cover, or a surface application of Fumasol™ hoed in to 15 cm. Untreated control plots were included for comparison. On the second site (Whale), chloropicrin was shank-injected, with or without a surface application of Fumasol, and covered with plastic. Untreated controls were also included. Fumigant application rates were high (60 g/m^2 for TriForm60 and chloropicrin, 70 ml/m^2 for Fumasol), as these trials were for 'proof of concept' to establish whether fumigation had any potential for VRR control. A heavy tyre-packer towed behind the fumigation rig was used to help seal the soil surface in all plots.

Small mesh bags containing weed seeds and pathogen (*Phytophthora*) inoculum were buried at the time of fumigation, then retrieved a week later and assayed for survival of propagules. Following fumigation, gas concentrations in the soil were measured at depths of 5 and 25 cm for up to 3 weeks. Natural weed germination in plots was assessed 3 weeks after fumigation. Carrot seed was sown in all plots in late December, 3.5 weeks after fumigation. Disease incidence in the carrot crop was assessed in March, May and July by randomly harvesting carrots from every plot and carefully inspecting roots for signs of VRR.

Assessments of buried weed seed and pathogen kill, natural weed establishment and ultimate VRR control showed consistent trends within treatments:

- VRR disease incidence was high in untreated control plots on both sites, with 41 and 99% infection on the Sue and Whale trial sites respectively.

- Shank-injected chloropicrin alone sealed with plastic gave partial but not commercially adequate control of VRR, with 67% VRR incidence on the Whale site by July. Weed and pathogen control was also only partial, with survival of many seeds, especially near the soil surface. Gas readings indicated high chloropicrin concentrations at 25 cm, but much lower concentrations at 5 cm depth.

- Surface-applied Fumasol hoed in to 15 cm in the shank-injected chloropicrin plots, and then sealed with plastic provided excellent VRR control, with less than 1% disease incidence, immediately next to untreated plots with 99% incidence. Buried weed and pathogen control was also good.

- Shank-injected Tri-Form60 sealed with plastic was effective in controlling VRR on the Sue site, with less than 3% of carrots infected by July. Buried weed seed and fungal inoculum were also killed at both 5 and 25 cm depth, and there was minimal natural weed growth in plots.

- When the surface-applied Fumasol treatment was used in combination with the Tri-Form60 treatment on the Sue site, very good control of VRR was achieved (less than 4% incidence), even without a plastic cover. Gas analyses showed that Telone and chloropicrin concentrations were high at depth, and Fumasol concentrations were high near the surface, presumably accounting for the good pathogen control throughout the profile.

There were significant problems with phytotoxicity in some treatments, most notably those including Fumasol. Early plant growth was suppressed, presumably because fumigant residues were still present in soil at the time of planting. Carrots eventually grew out of the problem, and by the end of the season a healthy crop was produced. The phytotoxicity in these trials can be attributed to three factors: firstly, very high fumigant rates were used, in part to determine whether or not the fumigants were capable of controlling the disease; secondly, sowing of seed was just over 3 weeks after fumigation, probably a week or so too soon; and thirdly, soil was cultivated just once, to a relatively shallow depth, between fumigation and sowing. Because of the trial layout and the need to maintain plot integrity and avoid cross-contamination, more frequent cultivation and deeper working of the soil using a tractor-mounted hoe was not possible.

Overall, results from the 2008/09 trials were very encouraging with respect to the potential for using soil fumigation to control of VRR. Optimisation of fumigant rates, combinations and application techniques were identified as key goals for future work, together with further investigation of potential phytotoxicity problems.

The 2008/09 trials are reported fully in Horner et al. (2009 & 2010).

2009/10 Field Fumigation Trials

Following the success of the 2008/09 field trials with combined treatment with TriForm60 and Fumasol, or chloropicrin and Fumasol, it was deemed necessary to refine these treatments. In particular, trials were required to determine whether reduced chemical rates would still control disease, as the relatively high rate used in the 2008/09 trial would probably be uneconomical for carrot production. It addition, it was necessary to assess the phytotoxicity problems that were noted in earlier trials, to minimise problems with poor plant establishment following fumigation. It was also considered worthwhile to assess 'carry-over' effects from treatment in the previous season, to determine whether annual fumigation was required once fields were infected.

Trials comparing various fumigant rates and combinations were established in the Sue (Karioi) site using plots from the previous trial, and a new site in an A.S. Wilcox block near Ohakune. Also included in the Sue site were treatments designed to test the carry-over effect of treatments into a second season, following fumigation and carrot cropping the previous year. A further trial at the Sue site investigated planting time and soil cultivation to release gases, and subsequent growth suppression caused by gas residues.

The fumigants used in the trials were:

- Chloropicrin, applied as a 100% product
- TriForm®60, a blend of 40% Telone® (1,3-dichloropropene) and 60% chloropicrin
- Fumasol™ (metam sodium).

Sue Site, Main trial – fumigant rates & combinations

Only 'untreated control' plots from the 2008/09 trial were used for the main fumigant rates and combinations trial, to ensure relatively high and even inoculum pressure among plots. Each untreated control plot from 2008/09 was split into four sub-plots, each measuring 8 x 2.2 m, and new treatments were assigned. Treatments are listed in Table 1. Triform60 rates were 55 and 33 g/m^2 for high and low rates, respectively. Fumasol rates were 50 and 30 ml/m^2 for high and low rates, respectively.

Fumigation was carried out in early November 2009, when soil temperatures ranged from 10 to 16°C and 11 to 14°C at 10 and 20 cm depth respectively. Soil was in good physical condition for fumigation.

Fumasol was applied to the soil surface using a watering can, then immediately rotary-hoed in to a depth of 10 to 15 cm. Immediately following Fumasol incorporation, the Tri-Form60 or chloropicrin was shank-injected to a depth of 25-30 cm in appropriate plots using a standard tractor-mounted fumigation rig. All plots were then rolled with a heavy tyre-packer to seal the surface (Figure 2).

Areas within plots to be planted were cultivated to a depth of approximately 15 cm, using a small motorised rotary hoe 2 weeks and 5 weeks after fumigation, to release remnant gases and prepare the seedbed. The need to minimise cross-contamination of treated/untreated soil between plots precluded the use of a tractor-mounted hoe and more frequent aeration. All plots were re-hoed in a similar manner 5 weeks after fumigation. 'Paramount' carrot seed was sown 6.5 weeks after fumigation using a standard tractor-mounted seed drill.

Table 1. Treatments used in the main carrot fumigant rate and combination trial at the Sue site, Karioi, 2009/10.

Fumigants	Code	Replicates
Chloropicrin (high rate) + Fumasol™ (high rate)	PH-FH	3
Chloropicrin (high rate) + Fumasol (low rate)	PH-FL	3
Chloropicrin (low rate) + Fumasol (high rate)	PL-FH	3
Chloropicrin (low rate) + Fumasol (low rate)	PL-FL	3
TriForm®60 (high rate) + Fumasol (high rate)	TH-FH	5
TriForm60 (high rate) + Fumasol (low rate)	TH-FL	5
TriForm60 (low rate) + Fumasol (high rate)	TL-FH	5
TriForm60 (low rate) + Fumasol (low rate)	TL-FL	5
TriForm60 (high rate)	TH	4
TriForm60 (low rate)	TL	3
Untreated control	Unt	7

Figure 2. Left: Shank injection of fumigants and pressing and sealing the soil surface using a tyre-packer. Right: Sue trial site, 2 weeks after fumigation, November 2009.

Assessments

Buried weed seed and fungal inoculum

Within one hour of fumigation, 5 x 6 cm mesh bags containing *Phytophthora*-infested soil and weed seeds (ryegrass and clover) were inserted into the soil at depths of 5-7 cm (top) and 16-18 cm (bottom) in four replicates of each treatment. Where there were only three replicate plots of a given treatment, two series were buried in one plot. This buried bag assay has been used successfully in a number of similar trials, as an indicator of fumigant activity and biocidal effectiveness in the soil. Ideally, *R. crocorum* inoculum would be used, but this fungus is particularly difficult to retrieve into culture and assay for viability, thus the *Phytophthora* and weed assay was chosen. The bags were retrieved 7 days after fumigation and assayed in the laboratory for seed viability (germination) and *Phytophthora* survival (cotyledon bait infection).

Results of seed and *Phytophthora* survival are presented in Table 2. Seed germination and Phytophthora survival rates were high in all untreated control bags. In contrast, in all combinations of either chloropicrin or Triform60 with Fumasol, ryegrass and clover germination, plus *Phytophthora* survival was substantially reduced or completely prevented. TriForm60 alone (i.e. without Fumasol) worked well at the deeper burial depth, preventing seed and *Phytophthora* survival, but nearer the surface it gave minimal control.

Table 2. Survival of weed seeds and Phytophthora inoculum in mesh bags buried at depths of 5-7 cm (top) or 16-18 cm (bot) in plots treated with various rates and combinations of soil fumigants at the Sue site, Karioi.

Fumigation treatment		Seed Germination (%) Ryegrass		Seed Germination (%) Clover		*Phytophthora* survival (out of 4)	
		top	bot	top	bot	top	bot
Chloropicrin (high rate) + Fumasol™ (high rate)	PHFH	0	0	0	0	0	0
Chloropicrin (high rate) + Fumasol (low rate)	PHFL	0	0	0	0	1	0
Chloropicrin (low rate) + Fumasol (high rate)	PLFH	0	0	0	0	0	0
Chloropicrin (low rate) + Fumasol (low rate)	PLFL	0.8	0.8	1.7	0.8	0	0
TriForm®60 (high rate) + Fumasol (high rate)	THFH	0	0	0.8	0	0	0
TriForm60 (high rate) + Fumasol (low rate)	THFL	0	0	0.8	0	0	0
TriForm60 (low rate) + Fumasol (high rate)	TLFH	0	0	0	0	0	0
TriForm60 (low rate) + Fumasol (low rate)	TLFL	0	0	0	0.8	0	0
TriForm60 (high rate)	TH	58.3	0	10.0	0	3	0
TriForm60 (low rate)	TL	79.2	0	44.2	0	4	0
Untreated control	U	83.3	75.8	45.0	46.7	4	4

Natural weed establishment

Weed assessments were made 5 weeks after fumigation, just before the second rotary hoeing the seedbeds, by counting germinated weeds in five quadrats placed randomly within each plot.

Weed growth was significantly less in all fumigant treatments than in untreated controls (Table 3, Figure 3). Among the various fumigant combinations, the TriForm60 alone (without Fumasol) gave the highest weed counts, reflecting results from the buried bag assay above. However, none of the differences in weed numbers among the various fumigant combinations was statistically significant.

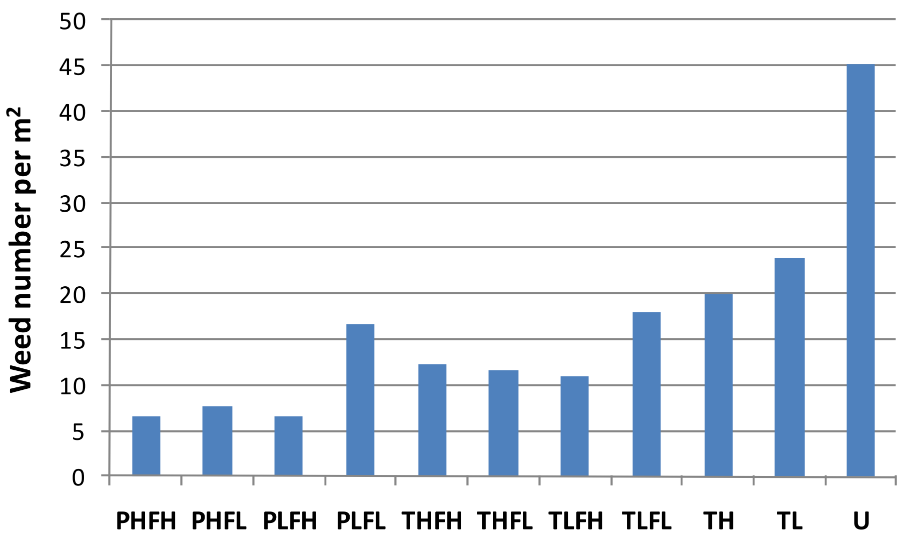

Figure 3. Weed counts in plots, 5 weeks after treatment with a range of fumigants at various rates and combinations. $P<0.001$, LSD=2.17. Treatment key: P=chloropicrin, T=TriForm®60, F=Fumasol™. H=high rate and L=low rate.

Early carrot plant growth

Carrot plant growth was first assessed in early February, six weeks after sowing. This was primarily to assess potential suppression of growth caused by fumigant residues. Twelve plants along transects in each plot were pulled, oven dried and weighed.

A summary of carrot plant weights is given in Table 3 and Figure 4. There were significant differences in early plant growth between treatments. Where TriForm60 alone was used, early season plant size was greater than in plots where Fumasol was included in the treatments, perhaps indicating that phytotoxic Fumasol residues were suppressing early plant growth. In all cases, early plant growth was less in when high rates rather than low rates of Fumasol were used (i.e. where the other fumigant in the mix remained the same).

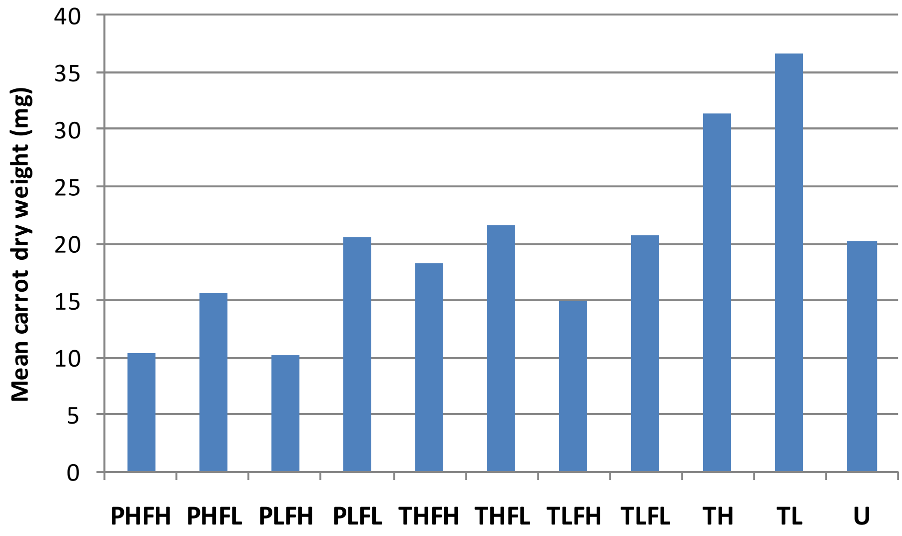

Figure 4. Average carrot seedling dry weight in February, six weeks after sowing. P<0.001, LSD = 7.4 mg. Treatment key: P=chloropicrin, T=TriForm®60, F=Fumasol™. H=high rate and L=low rate.

Disease incidence and carrot productivity

Assessments of carrot plant health and growth were made in early April and late June 2010. To minimise edge effects, only plants in the central 4 m of plots were assessed. At the two sampling times, 16 (April) or 30 (June) plants were assessed in each plot by lifting plants at uniform intervals, brushing and washing carefully to remove soil, and scoring VRR disease symptoms on the roots. Disease severity ratings were: 'healthy' = no symptoms, 'trace' = minor/superficial infection, 'moderate' = up to 50% of the carrot infected, 'major' = 50-75% of carrot infected, 'severe' = more than 75% of the carrot infected. Disease incidence data (proportion of plants with VRR symptoms) were analysed in Genstat® using an angular transformation. Healthy carrots from each plot were weighed, and average weight was calculated.

Disease data are presented in Table 3 and Figure 5. There was a positive response to fumigation in all treatments where either chloropicrin or TriForm60 was applied with Fumasol, with significantly lower VRR disease incidence than in untreated control plots. TriForm60 alone, at either the high or the low rate, did not reduce disease incidence from that in the untreated control.

There were no statistically significant differences in VRR incidence among the various combinations and rates of Chloropicrin, TriForm60 and Fumasol. However, the trend among treatments does suggest a rate effect. In all cases where chloropicrin or TriForm60 were combined with Fumasol, the high rate of a given fumigant resulted in less disease than the low rate of the same fumigant (within the same combination of fumigants). The 'high-high' fumigant combination had the lowest VRR disease incidence, and the 'low-low' combination had the highest disease incidence, for both chloropicrin/Fumasol and TriForm60/Fumasol combinations. There was also evidence that both the chloropicrin or Trifrom60 component, and the Fumasol component, contributed to disease control.

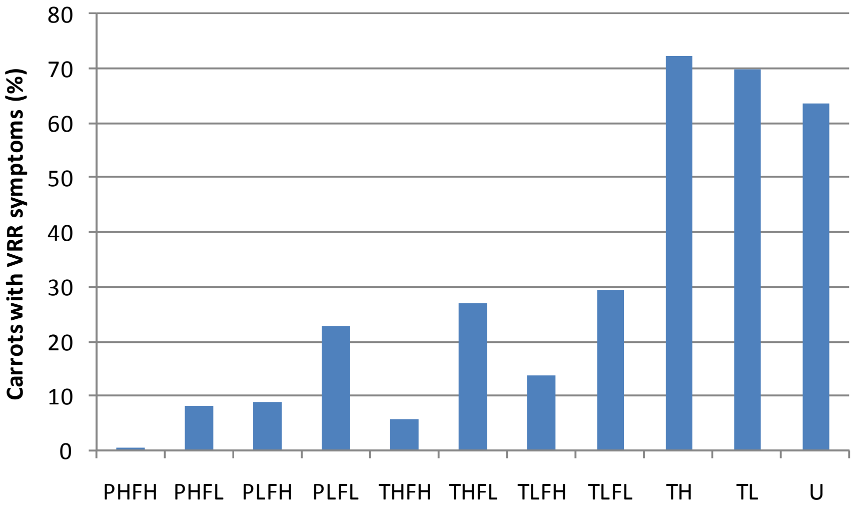

Figure 5. Percentage of carrots with violet root rot symptoms in June 2010, following pre-plant treatment with a range of fumigants at various rates and combinations. P<0.001 Treatment key: P=chloropicrin, T=TriForm®60, F=Fumasol™. H=high rate and L=low rate.

Analyses of carrot root weights (Table 3) showed that for the April and June assessments there were no statistically significant differences in average carrot size among the various treatments. This indicates that any potential delays in early establishment and growth, as were shown at the six-week assessment of plant size (Figure 4), were outgrown by the time of final harvest.

Table 3. Carrot plant weight and violet root rot (VRR) incidence assessed at various times after pre-plant fumigation (Nov. 2009) with various combinations and rates of Fumasol™, chloropicrin or Triform®60, Sue trial site, Karioi. * Fresh weight data are mean weights per carrot for all carrots, or for healthy carrots only, for April and June assessments, respectively.

Means in the same column, followed by the same letter, are not significantly different (α= 0.05)

Treatment		Weeds/m^2		Mean carrot plant dry weight, February (mg)		Mean carrot fresh weight* in April (g)		Mean carrot fresh weight* in June (g)		VRR incidence (%) April		VRR incidence (%) June	
Chloropicrin (high rate) + Fumasol (high rate)	PHFH	6.7	a	10.4	a	48.9	a	101.4	a	3.3	a	0.4	a
Chloropicrin (high rate) + Fumasol (low rate)	PHFL	7.7	a	15.7	ab	50.7	a	102.4	a	1.4	a	8.3	ab
Chloropicrin (low rate) + Fumasol (high rate)	PLFH	6.7	a	10.1	a	50.0	a	85.3	a	7.6	ab	8.8	ab
Chloropicrin (low rate) + Fumasol (low rate)	PLFL	16.7	a	20.5	b	62.2	a	114.5	a	17.3	ab	22.8	ab
TriForm60 (high rate) + Fumasol (high rate)	THFH	12.3	a	18.3	b	57.2	a	112.5	a	4.2	a	5.8	ab
TriForm60 (high rate) + Fumasol (low rate)	THFL	11.7	a	21.5	b	56.5	a	107.2	a	12.4	ab	26.9	b
TriForm60 (low rate) + Fumasol (high rate)	TLFH	11	a	15.0	ab	59.3	a	96.6	a	2.1	a	13.6	ab
TriForm60 (low rate) + Fumasol (low rate)	TLFL	18	a	20.7	b	54.2	a	98.2	a	39.3	ab	29.4	b
TriForm60 (high rate)	TH	20.0	a	31.4	c	53.6	a	126.4	a	47.2	b	72.2	c
TriForm60 (low rate)	TL	24.0	a	36.6	c	57.1	a	105.7	a	50.0	b	69.7	c
Untreated control	U	45.1	b	20.1	b	53.1	a	92	a	49.0	b	63.6	c
	P-value	*<0.001*		*<0.001*		*0.859*		*0.13*		*0.015*		*<0.001*	

Wilcox Site

The A.S. Wilcox site near Ohakune had grown potatoes in the 2008/09 season, and there were reportedly severe violet root rot problems throughout the block. An area approximately 210 × 16 m was selected for the carrot trial, and divided into plots measuring 16 m × 2.2 m. Fumigation treatments, outlined in Table 4, were applied in mid November 2009, using the same application techniques as outline for the Sue site, above. Triform60 rates were 55, 33, and 22 g/m2 for high, low, and very low rates, respectively. Fumasol rates were 50, 30, and 20 ml/m2 for high, low, and very low rates, respectively. Except for the 'very low rates', the fumigant application rates were the same as those in the main rate and combination trial on the Sue site. There were nine replicate plots of each treatment, arranged in a randomised complete block design (Figure 6). Plots were rotary hoed twice between fumigation and planting. A carrot crop was sown in early January, 7 – 8 weeks after fumigation.

Table 4. Treatments used in 2009/10 carrot fumigation trial at the Wilcox site, Karioi.

Fumigants	Code
TriForm®60 (high rate) + Fumasol™ (high rate)	TH-FH
TriForm60 (high rate) + Fumasol (low rate)	TH-FL
TriForm60 (high rate) + Fumasol (v. low rate)	TH-FvL
TriForm60 (low rate) + Fumasol (high rate)	TL-FH
TriForm60 (low rate) + Fumasol (low rate)	TL-FL
TriForm60 (low rate) + Fumasol (v.low rate)	TL-FvL
TriForm60 (v.low rate) + Fumasol (high rate)	TvL-FH
TriForm60 (v.low rate) + Fumasol (low rate)	TvL-FL
TriForm60 (v.low rate) + Fumasol (v.low rate)	TvL-FvL
Untreated control	Unt

Figure 6. Wilcox trial site in Ohakune. November 2009, a week after fumigation (left), and in April 2010 (right).

Assessments

Gas concentrations in soil

Gastec® tubes were used to measure concentrations of Telone, chloropicrin and Fumasol gases present in the soil following fumigation. Gas readings were taken in the TriForm60 (high)/Fumasol (high) treatment and TriForm60 (v.low)/Fumasol (v.low) treatments only, 4 and 5 weeks after fumigation. Chloropicrin and Telone were measured at 20 cm depth and Fumasol was measured at 10 cm depth, with four replicate plots sampled for each gas in each treatment at each sample time.

Results are summarised in Table 5. Chloropicrin residues could not be detected in soil 4 weeks after fumigation. Telone residues were high four weeks after fumigation, and although they had decreased in the following week (probably a result of cultivation in the block), residues were still detectable, and planting was delayed for another 2 weeks. Fumasol residues were not detected at week 5. However, other work (Horner, unpublished) has shown that the detection tubes used for Fumasol do not detect the various breakdown products of Fumasol; there could still be plant-inhibiting residues in the soil, even with zero readings for Fumasol.

Table 5. Fumigant gas readings (ppm) in soil 4 and 5 weeks after fumigation with Triform®60 and Fumasol™, Wilcox trial site, Ohakune.

Treatment	Week	Telone	Chloropicrin	Fumasol
TriForm60 (high rate) + Fumasol (high rate)	4	15.4	0	5
TriForm60 (high rate) + Fumasol (high rate)	5	2.4	0	0
TriForm60 (v.low rate) + Fumasol (v.low rate)	4	5.6	0	2.5
TriForm60 (v.low rate) + Fumasol (v.low rate)	5	1.5	0	0

Buried weed seed and fungal inoculum

Within one hour of fumigation, 5 x 6 cm mesh bags containing *Phytophthora*-infested soil and weed seeds (ryegrass and clover) were inserted into the soil at depths of 5-7 cm (top) and 16-18 cm (bot) in four replicates of each of four treatments (TH-FH, TL-FL, TvL-FvL, Unt). The bags were retrieved 7 days after fumigation and assayed in the laboratory for seed viability (germination) and *Phytophthora* survival (cotyledon bait infection).

Results of seed and *Phytophthora* survival are presented in Table 6. Seed germination and *Phytophthora* survival rates were high in all untreated control bags. Ryegrass and clover germination, plus *Phytophthora* survival was completely prevented in the high and low rates of Triform60 and Fumasol. There was a small amount of germination/survival at the very low rate of TriForm60 and Fumasol.

Table 6. Survival of weed seeds and Phytophthora inoculum in mesh bags buried at depths of 5-7 cm (top) or 16-18 cm (bot) in plots treated with various rates and combinations of soil fumigants at the Wilcox site, Ohakune.

Fumigation treatment	Seed Germination (%)				*Phytophthora* survival (out of 4)	
	Ryegrass		Clover			
	top	bot	top	bot	top	bot
TriForm®60 (high rate) + Fumasol™ (high rate) TH-FH	0	0	0	0	0	0
TriForm60 (low rate) + Fumasol (low rate) TL-FL	0	0	0	0	0	0
TriForm60 (v.low rate) + Fumasol (v.low rate) TvL-FvL	1.2	2.0	0.8	0.8	1	1
Untreated control Unt	85	82	56	50	4	4

Disease incidence and carrot productivity

Checks of carrot plant growth in March 2010 show even establishment in all plots, with no obvious signs of phytotoxicity or delayed growth in any of the fumigated plots.

The first plant health assessment was carried out in mid April 2010. Sixteen plants were lifted in all nine replicates of six of the treatments (TH-FH, THFvL, TL-FL, TvL-FH, TvL-FvL, Unt). All carrots were washed and assessed for VRR symptoms, using the same scoring system noted above for the Sue trial. Of 54 plots sampled, only four plots had any VRR among the sampled plants; three of these were untreated control plots and the fourth was a TvL-FvL plot. Disease incidence was not further analysed for these data.

Carrots sampled from each plot in April were weighed, and average weight was calculated. There were no significant differences (P =0.279) in mean carrot weight among the various fumigation treatments (Table 7), and thus no evidence for fumigant residues delaying plant growth, as had been seen in previous trials.

A second assessment of carrot growth and health was made in late June 2010. In every plot, 50 carrots (i.e. 450 carrots/treatment) were harvested, carefully washed, and assessed for violet root rot incidence and severity as outlined for the Sue site above.

The distribution of disease across the trial block was not even. At one end of the trial area, there appeared to be an infection focus, with very high disease incidence. This 'hot-spot' was just inside the gate at the top of the block, adjacent to and downhill from a loading dock and vehicle turning area. It is possible that waste carrots were dumped at this point some time in the past (a common practice), contributing to a high inoculum pool.

Averaged across the block, disease incidence was 16.6% in untreated control plots and less than 4% in all but one fumigation treatment (Table 7, Figure 7). The exception was the TvL-FL treatment, with 12.7% disease incidence. Apart from the one replicate plot of TvL-FL in the 'hot-spot', mean disease incidence for this treatment was 6.8%.

Except for the TvL-FL treatment, there were no statistically significant differences in disease incidence among the various fumigation treatments; all had significantly (P=0.002) lower disease incidence than the untreated control. There were no significant trends in disease incidence in the high, medium and low rates of fumigants used, although it is worth noting that the best disease control was in the TriForm60 high/Fumasol high (TH-FH) treatment (Figure 7).

On average, carrots grown in untreated control plots had the smallest weight in the June harvest, but none of the differences in carrot weight among the different treatments were statistically significant (Table 7).

Table 7. Mean weight and violet root rot (VRR) incidence in carrots harvested from plots treated with fumigated with various rates and combinations of TriForm®60 and Fumasol™.

Fumigant treatment		Mean carrot weight (g), April	Mean carrot weight (g), June	VRR incidence April (%)	VRR incidence June (%)
TriForm60 (high rate) + Fumasol (high rate)	TH-FH	33.15	77.1	0	0.8 a
TriForm60 (high rate) + Fumasol (low rate)	TH-FL	-	81.8	-	1.9 a
TriForm60 (high rate) + Fumasol (v. low rate)	TH-FvL	41.76	76	0	2.6 a
TriForm60 (low rate) + Fumasol (high rate)	TL-FH	-	71.6	-	3.1 a
TriForm60 (low rate) + Fumasol (low rate)	TL-FL	40.61	74.8	0	3.5 a
TriForm60 (low rate) + Fumasol (v.low rate)	TL-FvL	-	79.7	-	3.3 a
TriForm60 (v.low rate) + Fumasol (high rate)	TvL-FH	40.74	74.6	0	1.9 a
TriForm60 (v.low rate) + Fumasol (low rate)	TvL-FL	-	77.6	-	12.7 b
TriForm60 (v.low rate) + Fumasol (v.low rate)	TvL-FvL	41.23	76.1	0.7	2.7 a
Untreated control	Unt	36.43	70.1	5.6	16.6 b
	P-value	*0.279*	*0.507*	-	*0.002*

Means in the same column, followed by the same letter, are not significantly different (α= 0.05)

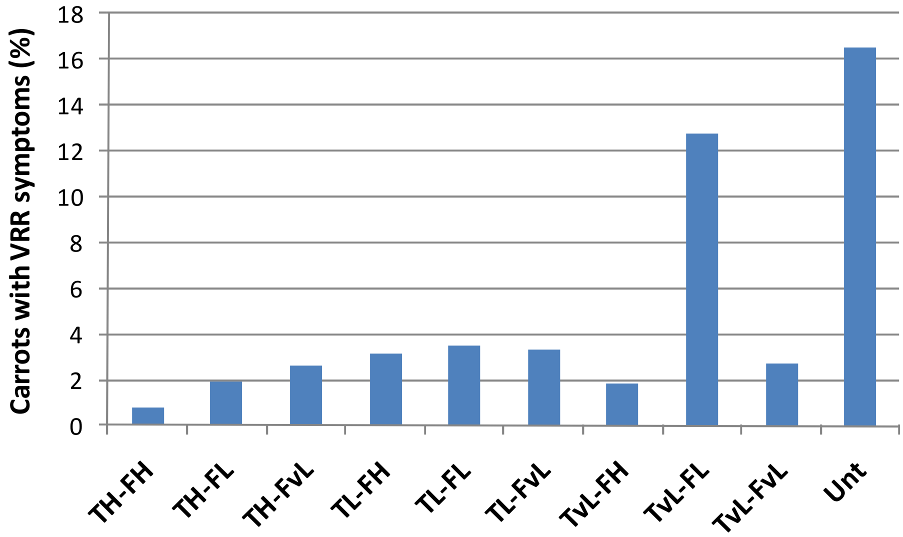

Figure 7. Violet root rot (VRR) incidence in carrots harvested from plots fumigated with various rates and combinations of TriForm®60 and Fumasol™, Wilcox trial site, Karioi. T=TriForm60, F=Fumasol, H=high rate, L=low rate, vL=very low rate, Unt=Untreated control.

Sue Site: Re-treatment/carry-over effect trial

To test the carry-over effect from previous season fumigation, and whether fumigation in successive seasons is of benefit, plots on the Sue site that had been fumigated the previous season were either re-fumigated or left untreated. Plots used had previously been treated with either TriForm60 + plastic, or TriForm60 + Fumasol, as indicated in Table 8. Plots in the re-treatment trial were fumigated in early November 2009, and the same day and using the same techniques as outlined in the 'main rate and combination' Sue trial above. Similarly, hoeing, planting and carrot plant disease and productivity assessments were the same as outlined for the 'main' trial above.

Table 8. Fumigants used in the re-treatment/carry-over effect fumigation trial at the Sue site 2009/10.

Fumigant treatment in current season	Treatment in previous season	Code	Reps
TriForm®60 (low rate, 33 g/m^2)	TriForm60 + plastic	TL/tp	4
TriForm60 (low rate, 33 g/m^2)	TriForm60 + Fumasol	TL/tf	4
TriForm60 (low rate 33 g/m^2) + Fumasol™ (low rate, 30 ml/m^2)	TriForm60 + plastic	TLFL/tp	4
TriForm60 (low rate 33 g/m^2) + Fumasol (low rate, 30 ml/m^2)	TriForm60 + Fumasol	TLFL/tf	4
Untreated	TriForm60 + plastic	Unt/tp	5
Untreated	TriForm60 + Fumasol	Unt/tf	6
Untreated	Untreated	Unt/U	7

Assessments

Carrot plant health and productivity data from April and June assessments are summarised in Table 9 and Figure 8.

There was good evidence for a carry-over effect from the previous season's fumigation treatment. In plots that had no fumigation in the second season, there was significantly lower disease incidence in plots that had been fumigated the previous year, compared with those untreated in the previous season. In plots fumigated in the previous season, there was no apparent benefit in re-treating plots with a low rate of TriForm60 alone, but a combined treatment of TriForm60 and Fumasol (both at the low rate) in the second year, reduced disease incidence compared with that in similar plots left untreated in the second year. There was no evidence for differences in the magnitude of carry-over effect from the two different treatments from the previous season.

Table 9. Average carrot root weight and violet root rot incidence in plots treated or not with various fumigant combinations following treatment or not in the previous season. * Fresh weight data are mean weights per carrot for all carrots, or for healthy carrots only, for April and June assessments, respectively.

Fumigant treatment in current season	Treatment in previous season	Code	Root weight April* (g)	Root weight June* (g)	% disease April	% disease June
TriForm®60 (low rate)	TriForm60 + plastic	TL/tp	62.0 a	119.3 bc	0.4 a	24.0 a
TriForm60 (low rate)	TriForm60 + Fumasol	TL/tf	60.6 a	110.5abc	8.0 a	16.2 a
TriForm60 (low rate) +Fumasol™ (low rate)	TriForm60 + plastic	TLFL/tp	45.1 a	96.3 ab	1.2 a	3.6 a
TriForm60 (low rate) + Fumasol (low rate)	TriForm60 + Fumasol	TLFL/tf	58.4 a	109.3abc	0.4 a	1.9 a
Untreated	TriForm60 + plastic	Unt/tp	53.1 a	98.1 ab	14.9 a	19.0 a
Untreated	TriForm60 + Fumasol	Unt/tf	53.4 a	122.8 c	9.0 a	13.7 a
Untreated	Untreated	Unt/U	58.6 a	92.0 a	49.0 b	63.6 b
		P-value	*0.344*	*0.033*	*0.003*	*0.007*

Means in the same column, followed by the same letter, are not significantly different (α= 0.05)

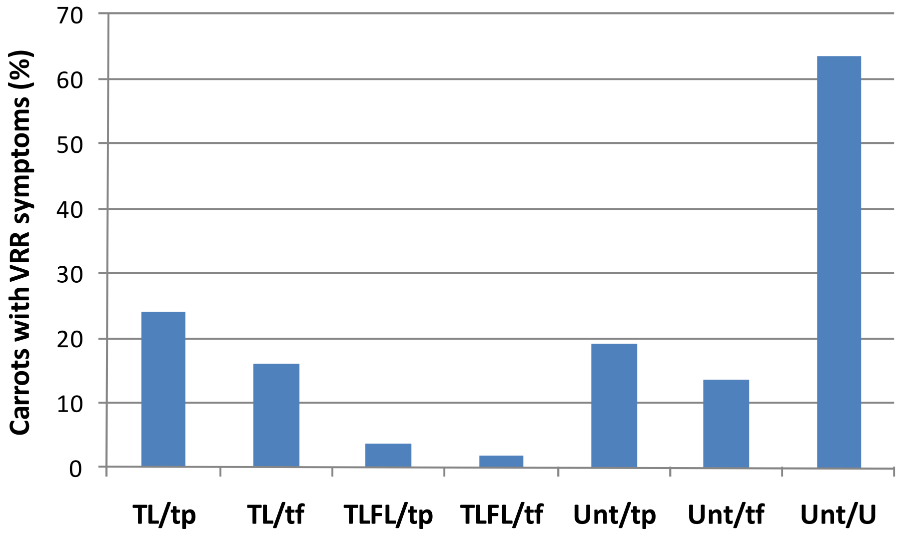

Figure 8. Violet root rot (VRR) incidence recorded in June 2010, in plots treated or not with various fumigant combinations following fumigation or not in the previous season. Key: Current year treatments TL=TriForm®60 low rate, FL=Fumasol™ low rate, Unt=untreated Previous year treatments tp=Trifrom60+plastic cover, tf=Trifrom60+Fumasol, U=untreated.

Plant-back trial

In the 2008/09 trials, early-season plant growth was stunted in some of the fumigation treatments, probably because of phytotoxic residues remaining in the soil at planting time. Possible contributing factors were the high fumigant rates used, the minimal cultivation to release fumigant gases, and the relatively short time between fumigation and planting. In the 2009/10 season, a trial was established at the Sue site to investigate further the effect of fumigant rate, degree of cultivation, and planting time on subsequent germination, establishment and growth of carrots.

The trial was established on ground that had been fumigated in the previous season, and consequently had relatively low VRR disease incidence that might otherwise interfere with the trial. The following fumigant treatments were applied in early November, on the same day and using the same techniques as the 'main rate and combination' Sue site trial described above:

- Fumasol (high rate, 50 ml/m^2) [FH]
- TriForm60 (high rate, 55 g/m^2) [TH]
- TriForm60 (high rate, 55 g/m^2) + Fumasol (high rate, 50 ml/m^2) [TH-FH]
- TriForm60 (low rate, 33 g/m^2) + Fumasol (low rate, 30 ml/m^2) [TL-FL]
- Untreated control [UNT].

There were three replicate plots of each fumigation treatment, each measuring 12 × 2.2 m, arranged in a randomised complete block design.

Following fumigation, five different rotary hoeing and seed planting regimes were imposed in subplots within each plot, as outlined in Table 10.

Each subplot measured 1.3 m long × 2.2 m wide. When hoed, the central 1.0 m across the full width of the subplot was rotary hoed to a depth of approximately 12-15 cm, leaving a small un-hoed buffer between each subplot. Where hoeing and planting was in the same week after fumigation, there was a 6- to 24-hour interval after hoeing before the seed was sown, to allow soil aeration. Forty 'Paramount' carrot seeds were hand sown in a grid pattern in the central region of each sub-plot.

Table 10. Hoeing and carrot sowing regimes implemented in sub-plots within plots treated with a range of fumigants in the plant-back trial at the Sue property, Karioi, 2009/10.

Hoeing/ sowing regime	Weeks following fumigation					
	0	1	2	3	4	5
A	Fumigate	-	Hoe & sow			
B	Fumigate	-	Hoe	Hoe & sow		
C	Fumigate	-	Hoe	-	Hoe & sow	
D	Fumigate	-	Hoe	-	Hoe	Hoe & sow
E	Fumigate	-	-	-	-	Hoe & sow

Assessments

Gas concentrations in soil

Following fumigation, Gastec® tubes were used to measure concentrations of Telone, chloropicrin and Fumasol gases present in the soil. Surplus areas in the TriForm60 (high)/Fumasol (high) treatment plots were used, with four replicate readings of each gas taken 1, 14, 21, 28 and 42 days after fumigation. For the 21, 28 and 42-day samples, readings were taken in both hoed (2 weeks after fumigation) and un-hoed sub-plots. Chloropicrin and Telone were measured at 20 cm depth and Fumasol was measured at 10 cm depth.

Results are presented in Table 11. Chloropicrin dispersed rapidly from soil, and was at very low concentrations by week 3. In contrast, both Telone and Fumasol residues remained in soil for longer, with moderate concentrations still present after 4 weeks. Rotary hoeing of plots after 2 weeks appeared to enhance dispersal of Telone residues, with a more rapid decline in measured concentration in hoed than in un-hoed plots. This difference was not as obvious with Fumasol. However, measurement of Fumasol residues is often inaccurate after two or more weeks and results are sometimes misleading, because of the presence of breakdown products not detected by the Gastec tubes used in this assay.

Table 11. Fumigant gas readings in soil measured at various times after fumigation with Triform®60 and Fumasol™, Sue trial site, Karioi. * '-' was not assessed

Days after fumigation	Telone		Chloropicrin		Fumasol	
	Un-hoed	Hoed	Un-hoed	Hoed	Un-hoed	Hoed
1	57	-*	378	-	130	-
14	35	-	21	-	40	-
21	40	6.2	2.5	1.7	15	10
28	11.5	1.0	0	0	7.5	5
42	7	1.9	0	0	-	-

Seed germination/establishment and seedling growth

Seed germination (plant establishment) was assessed in week 6 (for regimes A, B and C) and in weeks 11 and 20 (all regimes). Plant weight was assessed in early February and mid April, 12 and 22 weeks, respectively, after fumigation. For each weight assessment, between five and ten plants per sub-plot were harvested. In February, the whole plant was oven dried and weighed. In April the root only was weighed, fresh.

For each assessment, germination or weight data for each fumigation treatment in each regime was compared with that of the untreated control in the same regime. It was assumed that if there were minimal differences for a given treatment and the control, then there was no evidence for phytotoxic effects. Conversely, if germination or plant weight was significantly lower in any treatment or hoeing/sowing regime than in the comparable untreated control, this was seen as evidence for phytotoxic effects.

In general, in comparisons made within the same hoeing/sowing regime, percentage germination/plant establishment was similar across all fumigation treatments and untreated controls (Table 12). The only exception was lower plant numbers under regimes D and E in the Fumasol (high)/TriForm60 (high) treatment, a surprising result given that plant establishment rates were similar to the untreated control in regimes A, B and C with earlier sowing dates. Apart from this one result, there was no evidence for any reduction in seed germination and early plant establishment in fumigated plots.

Table 12. Percentage germination and establishment of carrot seed sown after various hoeing and sowing regimes, following soil treatment with various fumigants. The assessment was made 20 weeks after fumigation. Data with an asterisk* indicate mean germination significantly (P<0.05) less than that in the untreated control in the same planting regime.

Treatment	Hoeing/sowing regime				
	A	B	C	D	E
Fumasol™ (high) FH	85.6	74.4	78.9	58.9	62.2
Triform®60 (high) TH	81.1	73.3	76.7	52.8	63.3
Fumasol (high) + Triform60 (high) THFH	80	77.8	72.2	43.3*	58.9*
Fumasol (low) + Triform60 (low) TLFL	74.4	80	71.1	50	73.3
Untreated control U	74.2	79.2	77.5	67.5	72.5

Although germination and establishment were not greatly affected by the fumigation treatments, there was a negative effect in terms of early plant growth (Table 13, Figure 9). In all but one of the fumigation treatments, under all hoeing/sowing regimes, there was a significant reduction in plant size compared with that in the untreated control, when measured 12 weeks after fumigation. The only exception was the TriForm-only treatment, which although it had lower plant weight than the untreated control under all regimes, these differences were not always statistically significant. Overall, the poorest early growth was in the THFH treatment, suggesting a possible rate effect.

Table 13. Mean dry weight (g) of carrot plants sown after various hoeing and sowing regimes, following soil treatment with various fumigants, measured in early February, 12 weeks after fumigation. Data with an asterisk* indicate mean plant weight significantly (P<0.05) less than that in the untreated control in the same planting regime.

Treatment	Hoeing/sowing regime				
	A	B	C	D	E
Fumasol™ (high) FH	0.854*	0.875*	0.351*	0.133*	0.126*
Triform®60 (high) TH	1.758	1.379*	0.546	0.444	0.265*
Fumasol (high) + Triform60 (high) THFH	0.516*	0.587*	0.288*	0.143*	0.115*
Fumasol (low) + Triform60 (low) TLFL	0.736*	0.945*	0.373*	0.243*	0.240*
Untreated control U	2.705	2.529	0.873	0.505	0.601

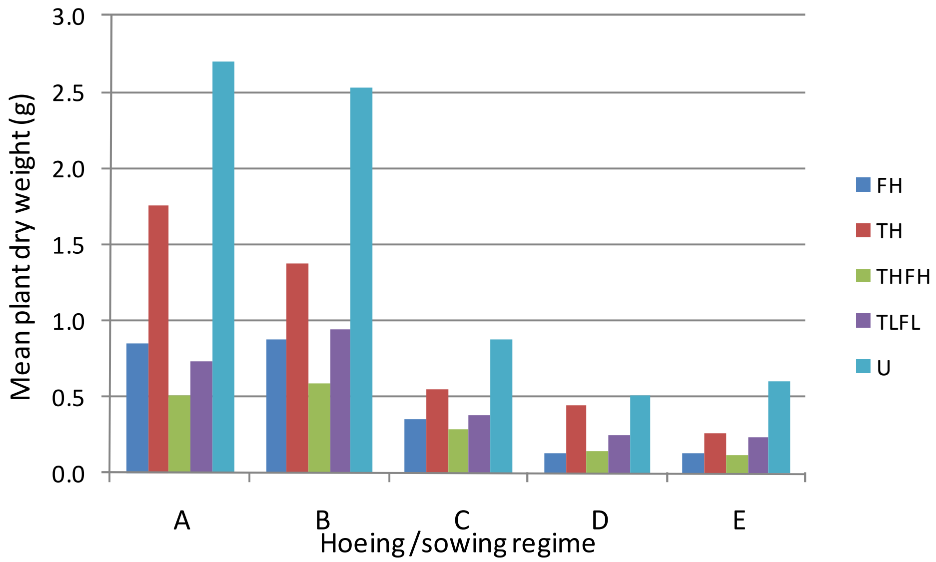

Figure 9. Mean dry weight of carrot plants, measured in early February, 11 weeks after soil treatment with various fumigants followed by various hoeing/sowing regimes. F=Fumasol™, T=TriForm®60, U=untreated, H=high rate, L=low rate.

Despite the early season suppression of carrot growth in most of the fumigation treatments and hoeing/sowing regimes, by mid April (22 weeks post-fumigation) plant growth had apparently caught up, with few differences in plant size compared with the untreated controls (Table 14).

Table 14. Mean fresh weight (g) of carrot roots sown after various hoeing and sowing regimes, following soil treatment with various fumigants, measured in mid April, 22 weeks after fumigation. Data with an asterisk* indicate mean plant weight significantly (P<0.05) less than that in the untreated control in the same planting regime.

Treatment	Hoeing/sowing regime				
	A	B	C	D	E
Fumasol (high) FH	194	181	143	135	131
Triform60 (high) TH	226	227	194	149	150
Fumasol (high) + Triform60 (high) THFH	209	178	162	119	98*
Fumasol (low) + Triform60 (low) TLFL	156*	165	175	155	161
Untreated control U	229	223	178	173	165

From this trial, there is evidence that both Fumasol and TriForm60 contribute to early suppression of plant growth. The 'Fumasol only' treatment in all cases suppressed growth more than the 'Triform60 only' treatment for the same planting regime, suggesting that Fumasol is the main contributor to the early growth suppression observed.

There was no clear evidence from this trial that the various hoeing and sowing regimes had a major impact on the degree of plant suppression, with a similar degree of stunting (compared with untreated controls) across the various regimes.

There was a clear difference in plant size relating to planting date, with significantly larger plants from the earliest sowing dates. This effect was still noticeable at the end of the growing season (data not recorded), suggesting that planting delays to accommodate fumigant residues might have to be balanced against growth benefits from earlier planting. It should be noted that the current trial was planted relatively late in the season (regime A was sown on November 20, regimes D & E were sown on December 11). The earlier regimes had good establishment and growth periods, whereas the later regimes were sown in relatively hot and dry conditions.

Discussion and Conclusions

Trials in Ohakune over the past three years have conclusively demonstrated that violet root rot of carrots can be controlled using soil fumigants. Joint application of chloropicrin or TriForm60 (shank-injected), with Fumasol (surface-applied and incorporated) followed by soil sealing using a heavy roller, has provided good control of VRR. Even in the heavily infested plots of the trial areas, providing a rigorous test for the trial fumigants, good disease control was achieved. Although questions remain about the most cost-effective rates and waiting times required before planting, the basic concept has been well proven.

Both fumigant components of the joint application are important, and neither works effectively in sole applications. In trials in all three season of the current programme, sole applications of TriForm60, chloropicrin or Fumasol have failed to provide effective control of VRR. In contrast, in almost all cases, the joint application of chloropicrin or TriForm60 with Fumasol provided good VRR control.

Studies of fumigant gas movement in soil, and pathogen and weed kill at various depths, have demonstrated that the Triform60 or chloropicrin component provides good control deep in the soil profile, and Fumasol provides control near the surface. Their zones of effectiveness reflect their respective application techniques and placement.

Theoretically, different application techniques to improve fumigant distribution in the soil could allow use of a single fumigant. But there are practical and economic issues preventing this. For either TriForm60 or chloropicrin to work effectively throughout the soil profile, improved soil sealing would be necessary. Attempts in previous years to seal in gases with polyethylene films were unsuccessful, except where heavy rolling was also used to seal the surface. Gases leaked rapidly through the plastic, and failed to build up to critical concentrations in the surface layers of soil. While virtually impermeable films (VIF) could be used, these films are significantly more expensive than standard polyethylene, and probably uneconomical for carrots. There is also the problem of plastic laying and retention in the windy and wet Ohakune environment, plus the monetary and environmental cost of plastic lifting and disposal, making plastic an impractical option. For Fumasol to fumigate effectively throughout the soil profile, higher rates and deeper incorporation of the product would be required. Although this is technically possible, by either shank injection or deep rotary hoeing to incorporate the product, Fumasol does not move very well through the soil. Deep incorporation of high rates of Fumasol is likely to lead to prolonged retention of residues, which could significantly delay planting or stunt early plant growth. However, such treatment has not yet been tested. Fumasol is a much cheaper product than either chloropicrin or TriForm60, and may be worthy of further investigation as a sole fumigant.

The trials on the Sue and Wilcox sites in the 2009/10 season demonstrated that a range of fumigant rates provided adequate control of VRR. There was a general trend for plots treated with highest fumigant rates to have the lowest VRR disease incidence, and for pathogen and weed survival in buried bags (as indicators of fumigant effectiveness) to be higher when low fumigant rates were used. Nevertheless, in few cases were the differences in disease control between high and low rates of the various fumigants statistically significant.

The requirement for a significant waiting period after fumigation, to avoid phytotoxicity and delayed growth in the young seedlings, remains a potential problem. Significant stunting of plants was observed in 2008/09 trials, but this followed high fumigant rates, minimal cultivation, and sowing just 3 weeks after fumigation.

There is some evidence that Fumasol residues are the major component of the plant-back problem (e.g. more growth suppression in Fumasol than in TriForm60 plots in the 2009/10 plant-back trial on the Sue site). However, evidence from the same trial, and experience with crops such as strawberries (Horner, unpublished) has shown that Telone/chloropicrin blends can also cause problems.

The plant-back trial in the 2009/10 season provided little evidence that delayed planting or increased hoeing reduced the phytotoxicity effect, although there was evidence that gas concentrations in soil dispersed more rapidly in hoed than in un-hoed plots. Early-season seedling growth suppression following fumigation was significant under a number of different cultivation and sowing regimes (seed sown up to 5 weeks after fumigation), yet by mid to late in the growing season, differences in carrot size between fumigated and un-fumigated plots were minimal. Similarly, in the main rate and combination trial on the Sue site in 2009/10, when seed was sown 6½ weeks after fumigation, early season plant suppression was noted in some fumigated treatments, but these effects were outgrown and nullified later in the season. No growth suppression was noted in the Wilcox trial, where sowing was 7-8 weeks after fumigation.

Even though plant-back remains a potential problem, there are ways to minimise its impact. Lower fumigant rates should result in fewer problems with plant-back. There was weak evidence for this in the 2009/10 plant-back trial, with slightly more suppression of early growth with high rates than with low rates of fumigants. A similar trend was noted in the main Sue rate and combination trial in 2009/10, with high rates in all comparisons resulting in more growth inhibition than low rates of the same fumigant. It is also worth noting that the phytotoxicity problem was much worse in the 2008/09 trial, when rates even higher than rates in the 'high-high' treatment on the 2009/10 trial were used (TriForm60 65 g/m^2 + Fumasol 70 ml/m^2 in 2008/09, TriForm60 55 g/m^2 + Fumasol 50 ml/m^2 in 2009/10).

Cultivation between fumigation and sowing will help to disperse lingering gas residues. Although no differences in plant suppression following fumigation were noted among the various hoeing regimes in the plant-back trial, there was a more rapid decline in Telone residues following hoeing of plots. Cultivation will be particularly important where a heavy roller has been used to seal in gases.

Later planting will reduces phytotoxicity problems, as fumigant residues will have time to disperse fully. But planting delays to minimise phytotoxicity must be balanced against poorer growth if planted late in season. This can be countered by fumigating as early as possible in the spring, then cultivating the soil within about 2 weeks to allow time for gas dissipation before planting.

Decisions on what fumigant rates to use will have to be a balance between potentially better VRR control with higher rates and perhaps multiple-year effectiveness, against factors such as cost, longer waiting periods with higher rates and more potential for phytotoxicity problems. The amounts of disease in the previous crop should also be taken into account when determining rates; when incidence was high in a previous crop there is likely to be a greater pool of inoculum in the soil and higher rates should be used. There is also potential to boost fumigant rates in disease 'hot spots' within treated fields, provided they are clearly marked at the time of fumigation. This would require mapping of problem areas (perhaps with GPS) during crop harvest. Where disease incidence is low or sporadic, lower fumigant rates could be used to keep the disease suppressed.

The choice of fumigants will depend on the situation. Telone, a major component of TriForm60, is an excellent nematicide, and will also broaden the spectrum of weed control. Chloropicrin alone is not as effective at killing weed seeds or nematodes, but is generally more effective than Telone at killing fungal inoculum, thus giving better disease control. Chloropicrin is a major component (60%) of TriForm60.

Although fumigation is expensive, and costs are likely to be marginal for carrot culture, the potential benefits are considerable. Trials have demonstrated that a single fumigation treatment of a heavily VRR-infested (and abandoned) field can once again allow carrot culture in that area. This has the potential to return large areas in the Ohakune district to production – areas that were previously abandoned because of VRR incidence. Once disease incidence and inoculum are lowered, occasional treatment, or fumigation with low chemical rates, may be adequate to maintain production with low VRR incidence. In the re-treatment trial, there was excellent disease control in the second year with such a regime.

Other potential benefits of fumigation should also be factored when evaluating the potential cost/benefit of treatment. Depending on the fumigants used, weed and nematode control should be achieved, reducing requirement for herbicides, and eliminating the need for nematicides. There should also be a lower incidence of carrot diseases other than VRR, and there is also the potential for increased carrot size in fumigated ground, provided that plant-back problems are minimised. If increased size is not desirable, plant spacing could be reduced slightly, thereby increasing overall productivity per hectare.

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