
Potato Yield Gap 2013–14

Sarah Sinton, Richard Falloon, Farhat Shah, Esther Meenken, Alex Michel, Steve Dellow, Sarah Pethybridge

June 2014



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Report approved by:

Sarah Sinton
Scientist, Sustainable Production
Date: June 2014

Paul Johnstone
Science Group Leader, Sustainable Production
Date: June 2014

This report has been prepared by The New Zealand Institute for Plant & Food Research Limited (Plant & Food Research).
Head Office: 120 Mt Albert Road, Sandringham, Auckland 1025, New Zealand, Tel: +64 9 925 7000, Fax: +64 9 925 7001.
www.plantandfood.co.nz

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Executive summary

Potato Yield Gap 2013–14

Sarah Sinton, Richard Falloon, Farhat Shah, Esther Meenken, Alex Michel, Steve Dellow, Sarah Pethybridge
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A yield gap survey of 11 commercial potato crops in Canterbury in the 2012-13 growing season confirmed grower concerns that a “yield plateau” of approximately 60 t/ha was common, a level at which potato growing is becoming uneconomic. The most important factors found to be reducing yield were soil compaction, the soil-borne diseases *Rhizoctonia* stem canker and *Spongospora* root galls.

This report outlines research in the 2013-14 growing season, which attempted to quantify the effects of these soil-borne diseases on tuber yield and quality by treating the soil with a range of pesticides to control all or some of the diseases, and to assess effects of seed tuber generation on potato yields.

Pesticide trial

Using farm-scale equipment, a replicated trial was planted on 4 November 2013, in a field known to have high levels of pathogen inoculum. Six pesticide treatments were applied, including: a non-treated control, three levels of soil fumigation (chloropicrin at 90, 112 or 146 kg/ha) for complete pathogen control, one rate of in-furrow azoxystrobin (1.5 l/ha Amistar®) to manage *Rhizoctonia* stem canker, and one rate of in-furrow flusulphamide (400 ml/ha Nebijin™) for control of *Spongospora subterranea*.

Soil samples from the trial site were tested for DNA of a range of soil-borne pathogens before and after treatment application. Above- and below-ground plant health and canopy cover were monitored every 10 days, underground stems and roots were scored for diseases and final yields were measured at commercial maturity.

According to the soil DNA tests, the levels of pathogen inoculum before and after the pesticide treatments were largely unchanged and were similar to or greater than those found in the crops surveyed in the previous growing season. *Rhizoctonia* stem canker developed as the plants emerged and by early January, all of the sampled plants in all of the treatments had stem canker symptoms (100% infection). However, throughout the season, severity of this disease was consistently less from the azoxystrobin treatment. Also in early January, *Spongospora* root galls appeared on the roots and stems in plants from all treatments, and the severity of root galling increased as the season progressed.

Throughout growth, the crop showed little above-ground effect of disease, and tuber dry matter accumulation and canopy cover followed that predicted by the potato growth model, until a severe hail storm hit on 23 February 2014. This reduced canopy cover by about 20%, hastening the onset of crop senescence and ending the chance of finding any treatment differences at crop maturity.

Final harvest yields did not differ between pesticide treatments and averaged 58 t/ha. All of the treatments gave similar amounts of powdery scab and black scurf on the harvested tubers.

Possible causes of lack of disease control were:

- the fumigation treatments were ineffective
- pathogen resistance to azoxystrobin (*Rhizoctonia*) and flusulphamide (*Spongospora*)
- the amounts of pathogen inoculum in the soil were too great for soil-applied pesticides to be effective
- planting operations transmitted pathogen inoculum from untreated (control) plots to pesticide-treated plots
- the scale of sampling failed to detect differences between the treatments due to the localised nature of the diseases
- the seed tubers were infected by the pathogens, thus distributing a range of pathogens evenly across the field. This is deemed the most likely, with the source of the inoculum coming from either the field that the seed tuber crop was grown in or from the seed cutting equipment.

Seed tuber generation trial

For a given cultivar, tuber seed is multiplied up from its original germplasm to give enough material for commercial potato crop production. It can take several years and “generations” to achieve this. Each time a generation is planted it can become infected with soil-borne diseases and this problem can become magnified during the seed tuber multiplication phase.

To monitor current seed quality, five generations (G 1-5) of the cultivars ‘Russet Burbank’ and ‘Innovator’ were planted (4 November) adjacent to the pesticide trial, in soil that had been treated with a high rate of fumigation. G1 is the first generation resulting from mini tubers planted in the field.

A hail storm on 23 February 2014 damaged the crop canopy, hastening crop senescence and confounding final analysis of the relative performance of the cultivars and generations.

Tuber fresh weight ranged from 54 t/ha (‘Russet Burbank’, G5) to 68 t/ha (‘Innovator’ G1). Apart from two exceptions, higher yields were associated with younger generations of both cultivars. ‘Russet Burbank’ had a lower total fresh yield but higher tuber dry matter content than ‘Innovator’, giving a similar overall tuber dry matter yield. ‘Russet Burbank’ had a lower stem number per plant, a higher tuber number per stem and a lower mean tuber weight than ‘Innovator’.

Early generations of ‘Innovator’ (1–3) and ‘Russet Burbank’ (2–3) yielded between 5 and 10 t/ha more than the ‘Russet Burbank’ generation (4) that was used in the pesticide trial.

Yield anomalies were ‘Russet Burbank’ G2, which had a high mean tuber weight and a very high level of powdery scab infection compared to all other treatments and ‘Innovator’ G2, which had a lower total yield than G3, G4 and G5.

‘Russet Burbank’ was more susceptible than ‘Innovator’ to both *Rhizoctonia* stem canker and the two forms of *Spongospora* (root galls and powdery scab).

A range of viruses were found in all generations.

1 Introduction

In the 2012–13 growing season, 11 surveyed crops had yield gaps of between 20–42 t/ha. For that season, potential yield for the crops was calculated at 87 t/ha, but average field yield was 60 t/ha. Fertiliser trials showed that nutrients were not limiting, but soil-borne diseases, soil compaction, and to lesser extent, foliar disease, weed competition, inadequate irrigation management and variable seed quality, contributed to yield losses. *Rhizoctonia* stem canker and *Spongospora* root galls were found in most of the surveyed crops and these diseases are known to reduce resource use efficiency in plants which in turn slows dry matter accumulation in the tubers, reducing yields.

So far, funding from Plant & Food Research, Potatoes New Zealand and Ravensdown Ltd, plus a substantial input of in-kind assistance from Canterbury potato growers, McCain Foods Limited and agronomists, has assisted with prioritising the importance of a range of yield limiting factors. The work reported here attempts to build on this new knowledge, and quantify the impact of particular soil-borne diseases on potato production.

2 Materials and methods

2.1 Pesticide trial

For this study, a field was selected that had a cropping history which included potatoes within the last 10 years and that was known to have high levels of a range of soil-borne pathogen inoculum. The aim was to significantly reduce all pathogens in some areas of the crop and retain individual diseases in other areas in order to estimate their individual and combined impacts on tuber yield and quality.

The trial design adopted was restricted by the commercial method of applying the different pesticides. These pesticides are normally sprayed in-furrow at planting. A large rig with spray tank, cultivator and a 6 row planter could only operate in long strips across the field. The trial was set up as a randomised block design with 3 replicates and 6 treatments randomised within each replicate (Appendix I), giving 18 plots. Each plot was 130 m long by 6 rows (5.3 m) wide. Five sampling points were established along each plot to attempt to determine spatial effects of disease development.

Treatments were:

- Control – no pesticides
- Azoxystrobin, 1.5 l/ha (as Amistar®), applied at planting
- Flusulphamide, 400 ml/ha (as Nebijin™), applied at planting
- 'Low' chloropicrin rate, 90 kg/ha, applied 3 weeks before planting
- 'Medium' chloropicrin rate, 112 kg/ha, applied 3 weeks before planting
- 'High' chloropicrin rate, 146 kg/ha, applied 3 weeks before planting

The effectiveness of azoxystrobin and flusulphamide in controlling plant damage that can result in yield loss depends upon the timing of disease infection relative to tuber set.

For the effect of flusulphamide on infection by the powdery scab pathogen, *Spongospora subterranea*, the main action in New Zealand seems to be significantly reducing the incidence and severity of early infection of the roots (e.g. galls). Early infection can reduce the ability of affected plants to extract water and nutrients from the soil. This could reduce the numbers of tubers set by each plant. Root infection is also likely to reduce growth of tubers later in crop development. Infection of tubers manifests as powdery scab symptoms on tuber surfaces, and these reduce tuber quality. Therefore, flusulphamide could be effective for improving tuber set, yields and quality.

Azoxystrobin) is applied primarily for the control of stem canker and black scurf diseases (caused by *Rhizoctonia solani*), and black dot (caused by *Colletotrichum coccoides*). *Rhizoctonia* stem canker disease will also reduce the ability of affected plants to gain water and nutrients, and therefore the numbers of tubers set. Black scurf and black dot are primarily diseases occurring on tubers later in potato crop development, and these diseases reduce tuber quality.

Chloropicrin is a gas fumigant that is held as a liquid in pressurised containers. The fumigant is most effective when soil temperatures are between 7 and 25°C, the soil is moist and well cultivated, and there is little undecayed organic matter present. Fine textured soils (with high clay content) have fewer air spaces and more binding sites to hold the fumigant, and so rates

need to be greater in fine soils. The converse is true for coarser soils. The soil should be sealed in some way immediately following fumigant application. Chloropicrin can take up to one month to act on soil microorganisms, and completely dissipate.

Soon after the liquid fumigant is injected into the soil, it forms a gas which immediately begins to disperse from the initial area of high concentration. Eventually the soil concentration becomes negligible as the gas decomposes in the soil. Fumigants have no residual activity and will not control pests that are introduced after the fumigant disperses.

Due to chloropicrin supply problems, the earliest the fumigation could be applied was 17 October 2013. After bed forming, the plots (6 rows (5.3 m) wide by 130m long) were marked out. The fumigation treatments were applied by Brian Leicester Ltd (Hawke's Bay), and were recommended and supplied by his contact in the USA (TriEst Ag Group, Inc.). Given there was no previous experience using fumigation in potato crops in Canterbury soils, low, medium and high rates were recommended to see which (if any) was the most effective. The gas was injected at a depth of approximately 30cm, directly into the zone where the seed tuber pieces were to be planted. A custom-made press-pan attached to the injection rig was used to help slow the dissipation of the gas and therefore increase fumigant efficacy (Figure 1). The experienced operator applying the gas was satisfied that the soil conditions were ideal at the time of injection.



Figure 1. Applying the fumigation treatments, 17 October 2013.

Immediately before fumigation, 10 soil samples were taken at 10 m intervals along the length of each plot and bulked. This procedure was repeated on 12 November 2013, after all pesticide treatments were applied. A soil sample (300 - 500g field weight) was taken from the 5 sampling points in each plot, air dried at 40 °C for 24 h and submitted for pathogen testing to the Root

Disease Testing Service, SARDI, Australia. The tests routinely identify DNA of the following pathogens (respective diseases): *Spongospora subterranea* (powdery scab), *Rhizoctonia solani* AG2.1, and AG3 (*Rhizoctonia* stem canker (RSC) and black scurf), *Verticillium dahliae* (one pathogen of the early dying complex), *Meloidogyne fallax* and *M. Hapla* (root nematodes), *Streptomyces scabies* (common scab), and *Colletotrichum coccodes* (black dot).

Before the crop could be planted, it was necessary to check that all traces of chloropicrin gas had gone from the soil. On 30 October 2013, 1-2 tomato seedlings (cultivar 'Russian Red') were planted into the centre rows of each plot, watered, and sealed by placing a bucket over them for 24 h. The next day all tomato plants had survived, indicating that all chloropicrin had dissipated. The potato crop was planted and the azoxystrobin and flusulphamide treatments applied in furrow on 4 November, which was towards the end of the normal planting window in this region.

The cultivar planted in the trial was 'Russet Burbank', sourced from Canterbury, cut 2 days earlier and treated with fir bark, and the fungicides pencycuron (Monceren®) to control *Rhizoctonia* stem canker and black scurf, and mancozeb (Manzate®) to control tuber rots. Planting depth was 20 cm, plant spacing was 32 cm and row spacing was 88 cm.

Every 10 days (total of 12 occasions), one pre-determined plant from each of 5 sampling points in each plot (a total of 90 plants) was harvested. For each plant, the shoots were cut off at the soil surface, and these were inspected for foliar diseases, then discarded. Underground stems, roots and tubers were excavated, and taken to a field laboratory, where they were washed and assessed for incidence and severity of diseases.

The incidence and severity of *Rhizoctonia* stem canker was scored using two scales, one for percent disease coverage on each stem of each plant (3 December 2013 to 13 March 2014), and the other by scoring the form of the stem canker symptoms (30 January to 13 March). Percent coverage was recorded as 1 = no stem canker, 2 = 0–20% of stem area affected; 3 = 20–50%; 4 = 50–80% and 5 = >80% stem area affected. Although not part of the standard scale, dead stems were given a score of 6. The form of the stem canker varied between brown "speckling" on the stem (score of 1), a combination of speckling and solid brown lesions (score of 2) and solid brown lesions only (score of 3, i.e. a more severe form than speckling). The product of these two scales gave a final severity × symptom score. For example, a stem with more than 80% area affected by solid brown lesions received a score of 15 (5 (>80%) × 3 (brown lesions) = 15), and a dead stem scored the maximum of 18 (6 (dead) × 3 (brown lesions) = 18).

Spongospora galls on stems and roots were rated as; 1 = <5 galls/plant; 2 = 5–20 galls, 3 = >20 galls/plant.

On each sampling occasion tubers were counted, weighed fresh and scored for the surface diseases, black scurf (*Rhizoctonia solani*) and powdery scab (*Spongospora subterranea*). Tubers from each plant were inspected and given a combined score of 1 = 0-2% tuber surface area affected, 2 = 2–5%, 3 = 5–10%, 4 = 10–25% and 5 = >25% tuber surface area affected. Numbers of deformed tubers per plant (probably resulting from *Rhizoctonia*) were also recorded.

During the course of crop growth, plants with early above-ground disease symptoms were marked and later assessed for yield at crop maturity. Ten plants were selected that showed top wilting and leaf curling, typical symptoms of *Rhizoctonia* stem canker. Another ten plants that had emerged late due to a blackleg infection were also marked.

At each visit, crop cover was assessed at a sampling point in each plot (approx. 2 rows by 2 m), using a Greenseeker® radiometer 505 Handheld sensor (NTech Industries, Trimble Navigation Ltd. Trimble Agriculture, Westminster, CO, USA). When held at 0.8 – 1.2 m above the crop canopy, this instrument records reflectance from a strip approximately 60 cm wide (similar to row width) every 100 milliseconds. The sensor measures reflectance in the near infrared (NIR, 774 nm) and visible red (656 nm) light wavelengths. The sensor calculates a normalized difference vegetative index (NDVI, maximum value = 1). Crop canopies with more green foliage absorb more red light and reflect larger amounts of NIR than those with less green foliage. High values for NDVI are therefore indicative of large amounts of living plant tissue.

The Greenseeker® was used every 10 days until near the end of crop senescence, when weeds began to emerge and confounded the readings. Senescence was hastened by damage from a hail storm on 23 February, after which few green leaves were left but many stems remained green.

Measured canopy growth and crop yields were compared to a potato crop growth model (Jamieson et al, 2008), which was used to predict potential yield for the crop using weather data collected from the Ashburton weather station (20 km from the trial site). The potential yield is that which can be attained where biotic (e.g. disease) and abiotic (e.g. irrigation and fertiliser management) factors are non-limiting.

For a final tuber yield assessment, a 6 plant by one row final harvest plot was marked out in each of the five sampling points in each plot, the row length was measured in order to calculate plot area, and total stem number counted. On 13 May, tubers from each of these final harvest areas in each plot were dug by hand, combined and graded into length categories; <67 mm (reject), 67-90 mm and >90 mm. Tubers in each grade were counted and weighed. For each plot, 16 tubers were then randomly selected from each grade (total of 48 tubers). These were removed to the field laboratory where they were washed and assessed for tuber diseases. Powdery scab was scored using a standard disease severity scale and black scurf scored as presence/absence. From this sample, tuber dry matter was also determined by oven drying at 90°C for 3 days.

2.2 Seed tuber generation trial

Five seed tuber generations (G) of 'Russet Burbank' and 'Innovator' were respectively sourced from two separate seed producers. A generation takes one season to produce and G1 is the first crop resulting from mini tubers being planted in the field. While the different generations had been grown on the same properties, they were not grown in the same fields. The seed tubers were uncut, apart from a sixth treatment, which was each cultivar from G5 cut in half (same final weight as G5 uncut). Seed tuber weight ranged from 89–124 g for 'Innovator' and 87–121 g for 'Russet Burbank' (Table 1). The soil at the trial site was treated with chloropicrin (at 146 kg/ha), and the soil was sampled before and after treatment for soil-borne pathogen DNA analyses (as described above).

Table 1. Average fresh weights of seed tubers of 'Russet Burbank' and 'Innovator', potatoes planted in the seed tuber generation trial

Seed tuber generation	Uncut/Cut	'Russet Burbank'	'Innovator'
		Average fresh weight/tuber (g)	
1	Uncut	87	89
2	Uncut	103	100
3	Uncut	100	102
4	Uncut	102	101
5	Uncut	113	124
5	Cut	121	122

The trial was a randomised block design with three replicates. The 12 treatments were seed tubers of the different cultivars and generations. Treatments were applied to plots that were each two rows by 20 m long. No buffer rows were planted between the plots (Appendix II). The trial was planted using a 6 row planter, with two people on the planter guiding the tubers from the different treatments onto the planter cups.

On 6 December 2013, a few days after plant emergence, leaf samples were taken for assays of viruses. Fifteen leaves per row (30 leaves from each plot) were bulked into one bag. The samples were taken to a plant pathology laboratory and tested for presence of a range of viruses commonly found in New Zealand potato crops.

Crop cover was measured every 10 days in a 2 m by 2 row section of each plot using a Greenseeker® radiometer (see pesticide trial section for further description).

When the trial had reached the full canopy stage, two plants were taken from each plot and returned to the laboratory for disease assessments. Tuber and underground stem disease assessments were carried out as described above.

At the end of crop growth, and before final harvest, a 10 plant (approximately 3 m length) by 2 row harvest area was marked out in each plot, the row lengths were measured and stems counted. Tubers from four plants (one from each corner of the plot) were dug, washed and assessed for skin diseases. Samples were then sent McCain Foods where they were tested for specific gravity, average length and fry quality. The final harvest areas were dug by hand, the tubers were counted and weighed and the dirt fraction was estimated.

3 Results and discussion

3.1 Pesticide trial

Cropping history of the field

Continuous cropping can result in the build-up of a range of soil-borne pathogens that potentially contribute to potato yield losses. Without breaks from annual crops, pathogens are more likely to survive from one season to the next on hosts, such as volunteer potato plants and weeds (e.g. nightshades), as well as on the remaining organic matter from previous crops. Growers understand this, but can find it difficult to find suitable land that has not grown potatoes in recent years. The purpose of this pesticide study was to monitor development of major diseases and to quantify their effects on tuber yield and quality. The field selected was known to have high levels of diseases, probably resulting from long-term cropping (Table 2).

A crop of potatoes had been grown in the field 8 years previously, and this also increased the chances of pathogen carry-over.

Table 2. Cropping history for the field where two potato trials were carried out in the 2013/14 growing season

Year	Crop
2004	Wheat
2005	Barley
2006	Potatoes
2007	Wheat
2008	Beetroot seed
2009	Wheat
2010	Perennial ryegrass seed
2011	Clover seed
2012	Wheat
2013	Ryegrass seed
2014	Potatoes

Soil from the field, sampled before and after applications of different pesticide treatments to designated plots, was tested for soil-borne pathogen DNA. Results showed *Spongospora subterranea*, *Rhizoctonia solani* AGs 2.1, 3, *Colletotrichum coccodes* (black dot), *Streptomyces scabies* (common scab) and *Verticillium dahliae* were present in the field. No DNA of root nematodes (*Meloidogyne fallax* and *M. Hapla*) was detected. See sections below where results for each disease are presented and discussed.

A separate test initiated by Seed and Field Services (SI) showed that root lesion nematode (*Pratylenchus* spp., including *P. crenatus*) and the mycophagous nematode (*Aphelenchus avenae*) were also in the soil.

The relationship between the presence of pathogen DNA in pre-planting soil samples and disease incidence and severity in the crop or on harvested potato tubers for any particular season depends on the environmental conditions encountered, the cultivar susceptibility and soil physical and chemical conditions. Large amounts of pathogen DNA do not always translate into severe disease occurring in the subsequent crop. Additionally, these tests do not discern

between “living” and “dead” DNA. However, the levels of *R. solani* and *S. subterranea* DNA measured in the soil were consistent with the widespread severity of symptoms of these diseases later found in the crop.

Volunteer potato plants were noted across the trial areas, surviving from the potato crop in 2006. Nightshade weeds (*Solanum* spp.) were also observed (Table 2) and *Rhizoctonia* stem cankers and *Spongospora* root galls were observed on some of these plants.

Pesticide treatment application

Applying chloropicrin to fumigate soil prior to planting potato crops had probably not been previously attempted in Canterbury, so three rates of the fumigant were used to allow for the unknown effects of soil texture and other soil characteristics. Conditions were considered to be ideal at the time of application; the soil was well cultivated in pre-plant beds, it was moist and within the optimum temperature range. It is uncertain why fumigation did not reduce pathogen levels in the soil, or disease severity in the subsequent crop. Possible reasons could be that the press-pan system did not hold the gas for long enough and/or pathogens were re-introduced at planting (either on the seed or planting equipment) after the gas had dissipated.

Both azoxystrobin and flusulphamide are commonly used in Canterbury. The pesticides were applied at planting by a commercial operator at standard rates.

Spongospora subterranea

Before pesticide treatment application, the amount *S. subterranea* DNA averaged 1,400 pg/g of soil, and there was evidence of slight reductions in DNA of this pathogen after the three different chloropicrin fumigation treatments ($P = 0.06$, mean of 1,100 pg/g) (Figure 2). This was not of practical significance and may have been due to the different time of sampling rather than the treatments themselves. Overall, the amount of *S. subterranea* DNA found in the soil of this crop was greater than was found in 6 of the 11 surveyed crops the previous season (range 0–960 pg/g soil).

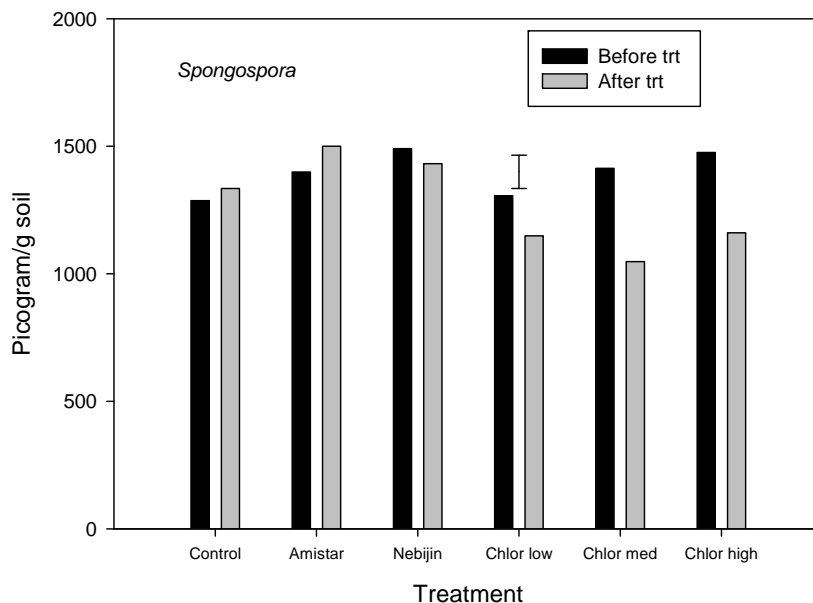


Figure 2. Mean amounts of *Spongospora subterranea* DNA detected in soil sampled from trial plots receiving different pesticide treatments; Control, Amistar, Nebijin and 3 rates of Chloropicrin (Chlor). Bar at LSD ($\rho = 0.05$), 22 df.

In the pesticide trial, *Spongospora* root galls were first noted on plants in early January. Soon after this, galls also began appearing on the underground stems, a phenomenon which was not seen in any of the surveyed crops the previous season (Sinton et al, 2013).

Powdery scab was equally present on the sampled tubers throughout crop growth across all of the pesticide treatments. Infection levels steadily increased from January to March (Table 3).

Table 3. Powdery scab score on tubers in the pesticide trial during crop growth.
 1 = 0–2%, 2 = 2–5%, 3 = 5–10%, 4 = 10–25% and 5 = >25% of the tuber surface area affected.

Powdery scab on tubers	
Sampling date	Score
30 Jan 14	0.01
11 Feb 14	0.52
20 Feb 14	1.12
3 Mar 14	1.47
13 Mar 14	1.60

At final harvest (13 May), powdery scab severity on the tubers ranged from 0 to 46% of tuber surface affected, with only a few tubers having severity greater than 46%. Tubers from the Control, azoxystrobin or flusulphamide treatments (from a subsample of approximately 50 tubers) had greater numbers ($P = 0.03$) of tubers free of powdery scab than did the fumigation treatments (Figure 3). Flusulphamide, specifically formulated to control powdery scab, had no additional control of powdery scab on these mature tubers and gave no yield increase compared with any of the other treatments, including Control (no soil-applied pesticides).

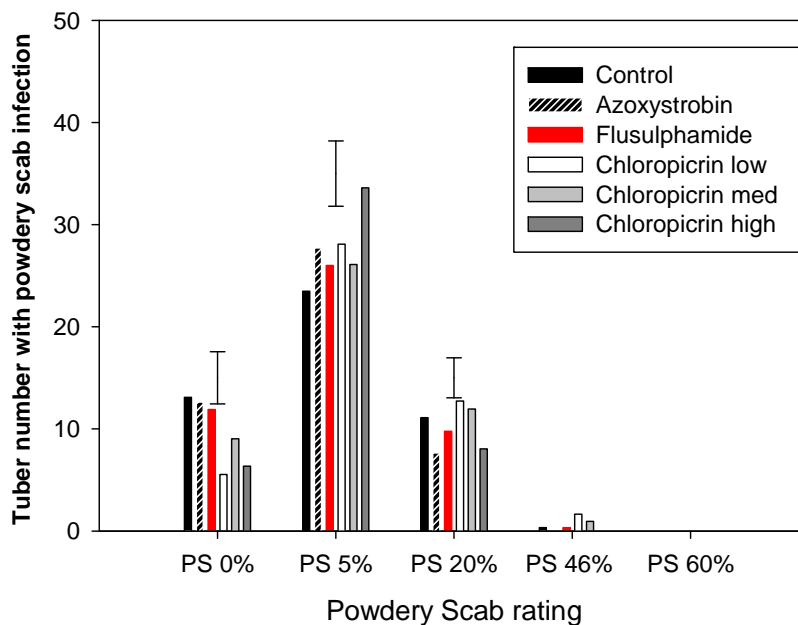


Figure 3. Mean numbers of potato tubers expressing powdery scab (0, 5, 20, 46 and 60% ratings) from plots treated with different pesticides prior to planting. Bars at LSDs ($p = 0.05$) 10 df.

Rhizoctonia solani

DNA of *R. solani* AG2.1 had an average pre-treatment quantity of 400 pg/g soil with a wide range of 250 to 700 pg/g. The amount of DNA was reduced by the low and medium rates of chloropicrin to less than 150 pg/g soil ($P = 0.002$) (Figure 4). However, the pre-treatment variability of the quantities of DNA of this pathogen makes these results more difficult to interpret clearly.

The range of amounts of DNA of *R. solani* AG2.1 found this season was about the same as measured for crops surveyed in the 2012–13 season, where this pathogen was detected in soils from most of the 11 surveyed crops.

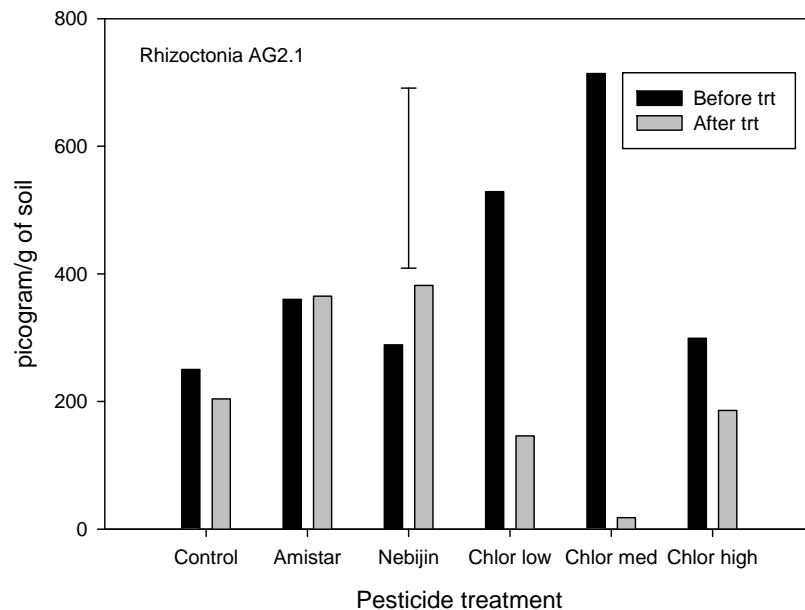


Figure 4. Mean amounts of DNA of *Rhizoctonia solani* AG 2.1 detected in soil sampled from trial plots receiving different pesticide treatments ; Control, Amistar, Nebijin and 3 rates of Chloropicrin (Chlor). Bar at LSD ($p = 0.05$), 10 df.

Rhizoctonia stem canker (RSC), generally caused by AG2.1, was present on the underground stems right from emergence on 3 December 2013 (Figure 5). By early January there was 100% of plants affected by RSC on underground stems from all treatments. However, by the end of crop growth, disease severity (measured as a product of percent stem cover, symptom appearance and whether or not the stems were dead) was less from the azoxystrobin treatment compared with all of the other treatments (Figure 6). This reduced disease did not translate into a yield benefit from azoxystrobin, however, probably because of the shortened canopy duration caused by the hail storm.



Figure 5. Recently emerged potato plant showing *Rhizoctonia* stem canker infection (middle stem).

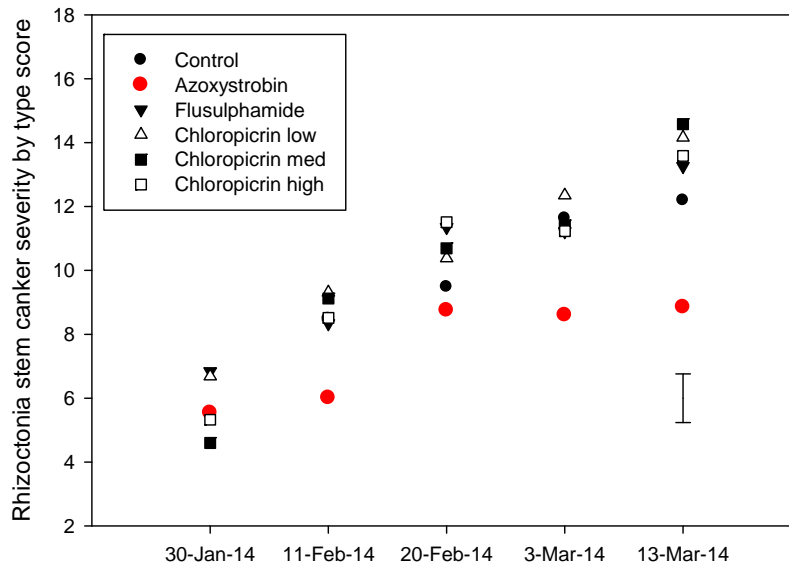


Figure 6. Mean *Rhizoctonia* stem canker severity scores (at different times during late crop growth) for potato plants grown in plots treated with different pesticides. Bar at LSD ($p = 0.05$), 10 df.

Tubers deformed by *Rhizoctonia* averaged 4% of the total tubers assessed and this proportion did not differ between pesticide treatments.

Throughout the growth of the crop, there were very few plants (less than 1%) showing above-ground effects of any soil-borne disease. Apart from lesions on underground stems, RSC can cause early wilting, yellow and curling of leaves and these plants usually die prematurely. Individual plants throughout the crop can be affected and also larger patches of plants where soil conditions are poor (e.g. waterlogged). During the crop survey in 2012–13, up to 20% of plants in some crops were showing above-ground symptoms of RSC by the end of February (Sinton et al, 2013).

Ten plants showing early above-ground wilting from RSC were marked in December and January, then harvested near the end of crop senescence. Plant yields ranged from 295 to 2,322 g/plant (equivalent to 10 to 77 t/ha tuber yield). This showed that the more vigorous plants had some resistance to the effects of RSC than weak plants. However, the mean yield of 34 t/ha was about 40% less than the overall crop yield of 58 t/ha.

Rhizoctonia solani AG3 is known to cause black scurf on tubers. Soil DNA tests showed that there was about 16 pg/g soil of DNA of this pathogen (much less than for AG2.1), and this quantity was not affected by the pesticide treatments. At the final harvest, all the pesticide treatments (including the untreated controls) had approximately 50% of the tubers affected by black scurf.

Bacterial soft rot (blackleg)

During plant emergence, blackleg (not assessed in the soil DNA assays) was identified as a cause of slow emergence in some plants (Figure 7). This disease is usually transmitted on seed tubers. Ten plants affected by this disease were marked and harvested at crop senescence. Yields from these plants ranged from 300 to 2,300 g/plant (equivalent to 7 to 70 t/ha tuber yield, mean 30 t/ha). This showed that some plants outgrew the disease but others were weakened and/or were out-competed by more vigorous neighbours. Of the 990 plants sampled across the trial between 3 December 2013 and 13 March 2014, 41 (or 4%) had blackleg lesions on their underground stems.



Figure 7. Marked plant showing symptoms of delayed emergence as caused by bacterial soft rot (blackleg).

Black dot

There were similar amounts of *Colletotrichum* DNA in the soil before and after application of the pesticide treatments (approximately 500 pg/g soil), and this was generally greater than was found in soil from the crops surveyed in 2012–13.

Streptomyces scabies

Small amounts of *S. scabies* DNA were detected in the soil before and after application of the pesticide treatments, averaging from 0–9 pg/g soil. Only one crop in the 2012–13 survey had comparatively large amounts of DNA of this pathogen (>1,000 pg/g). All of the other 2012–13 crops either had no *S. scabies* DNA or very low amounts.

Verticillium dahlia

All treatments had less 1 pg/g soil of *V. dahlia* DNA in the soil, and this amount was the same before and after treatment with pesticides. Symptoms were not seen during crop growth. This disease was present at low levels in 5 of the 11 crops monitored in the 2011–12 season.

3.2 Seed tuber generation trial

Plants emerged on about 3 December 2013. Soon after emergence, leaf samples were tested to estimate levels of tuber-borne virus infection (Table 4). *Potato virus S* was present in young plants from all of the seed tuber generations, at high estimated incidence (10–100%). *Potato virus M* was also present in all generations but generally at lower incidence (1–100%). *Potato virus X* and *Potato virus Y* were similarly present in most ‘Russet Burbank’ generations but at low incidence (1–10%). PVX was present in most ‘Innovator’ generations (1–4%) and PVY in one generation (‘Innovator’ G2, 1%). *Potato leafroll virus* was detected in only one ‘Russet Burbank’ generation (‘Russet Burbank’ G1, 1%).

Table 4. Presence of virus (percent of leaves sampled) in plants from five generations of ‘Russet Burbank’ and ‘Innovator’ seed tubers. The samples were taken a few days after the plants emerged.

Estimated virus incidence (percent of leaves sampled)							
19 December 2013							
	Seed type	Virus X	Virus Y	Virus S	Virus M	Virus A	Virus Lr
RB							
Gen	cut/uncut						
G1	whole	0	10	10	6	0	1
G2	whole	0	1	100	1	0	0
G3	whole	1	1	100	100	0	0
G4	whole	1	1	100	20	0	0
G5	whole	1	1	100	100	0	0
G5	cut	1	0	100	100	0	0
Innovator							
Gen	cut/uncut						
G1	whole	1	0	100	100	0	0
G2	whole	4	1	100	100	0	0
G3	whole	4	0	100	100	0	0
G4	whole	1	0	100	100	0	0
G5	whole	0	0	100	100	0	0
G5	cut	1	0	20	100	0	0

Modelled NDVI is based on the cultivar 'Russet Burbank'. When compared to this, canopy expansion was faster in all generations of both 'Innovator' and 'Russet Burbank', including 'Russet Burbank' G4 that was used in the pesticide trial (Figures 8 and 9). The canopy of 'Innovator' G5 cut seed was smaller than from the other 'Innovator' lines and complete cover was not achieved. Other generations of 'Innovator' had similar canopy sizes, as did all generations of 'Russet Burbank'. Compared to 'Innovator', 'Russet Burbank' grew a larger canopy which was sustained for longer.

Maximum NDVI was maintained until a hail storm hit on 23 February 2014. The storm severely damaged the leaves and crop cover was reduced by about 20%. Following from this, crop cover declined rapidly, and plants from most of the seed tuber generations were dead by the end of March. The opportunity for the different generations to develop differences in canopy duration was therefore lost, due to the damaging hail storm.

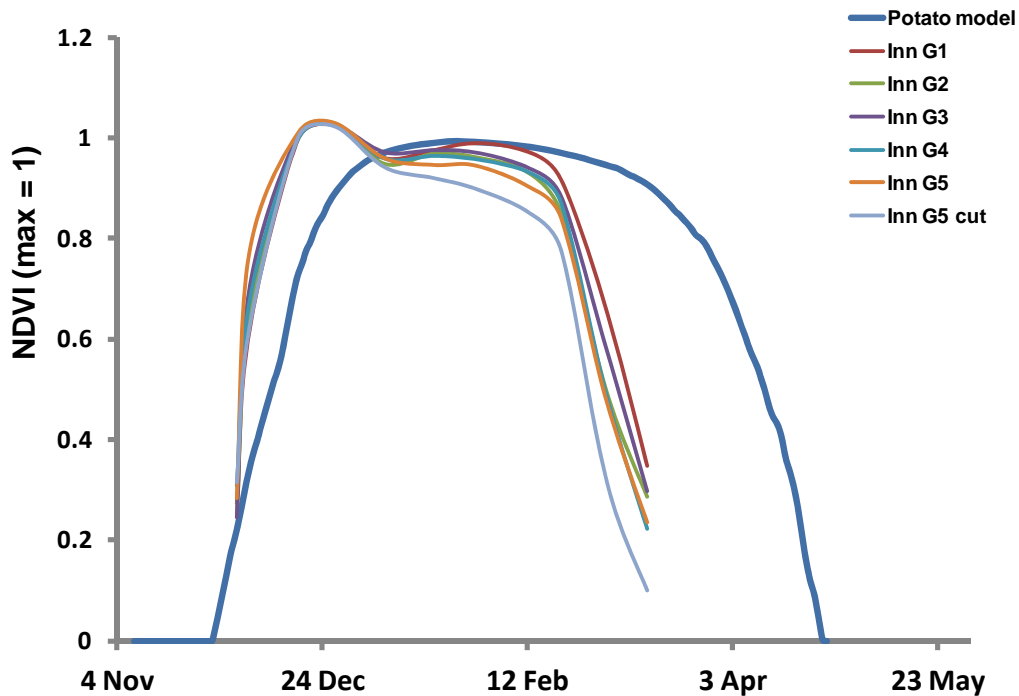


Figure 8. Modelled and measured canopy cover for 'Innovator' plants grown from five different generations of seed tubers.

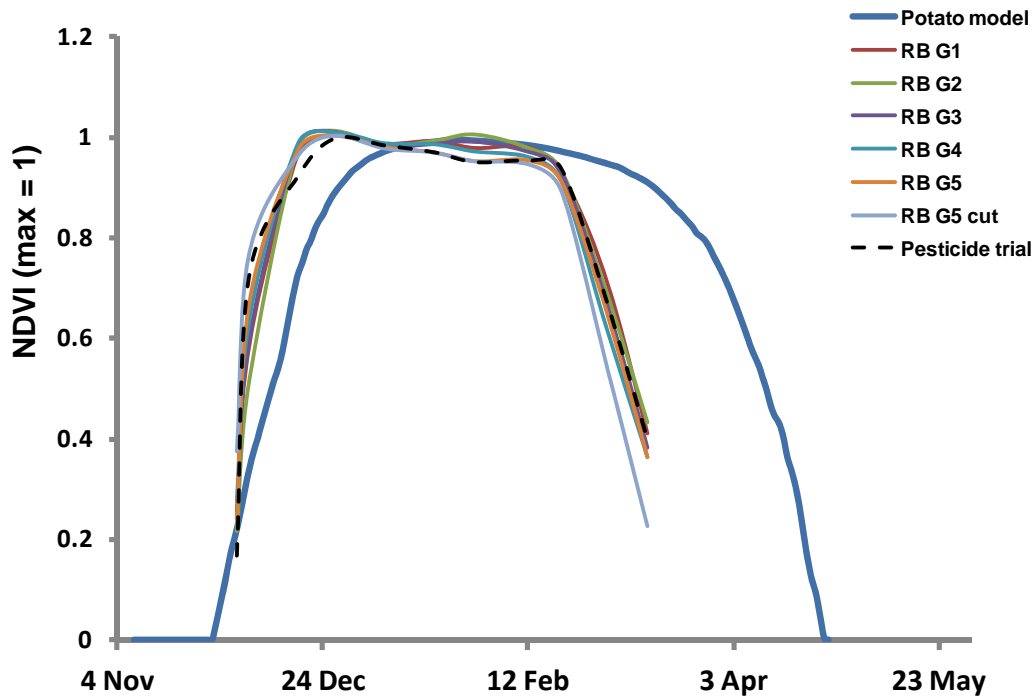


Figure 9. Modelled and measured canopy cover for 'Russet Burbank' plants grown from five different generations of seed tubers and also 'Russet Burbank' G4 that was used in the pesticide trial.

Final fresh yields varied between the generations and cultivars (Figure 10). With the exception of 'Innovator' G2 and 'Russet Burbank' G1, fresh yield generally declined as generation number increased ($P = 0.02$). 'Russet Burbank' mostly yielded less (average of 59 t/ha, $P = 0.01$) than 'Innovator' (62 t/ha). However, for G2, the 'Russet Burbank' yield was greater (65 t/ha) than 'Innovator' (57 t/ha). For 'Innovator', G1 yielded the most with 68 t/ha and G2 the least with 57 t/ha. 'Innovator' G5 was also low yielding at 59 t/ha. For 'Russet Burbank', G2 yielded the most with 65 t/ha and G5 the least with 54 t/ha.

Tuber dry matter (solids) percent averaged 20.8% for 'Russet Burbank', which was greater ($P = < 0.001$) than 19.3% for 'Innovator'. When tuber dry matter content was taken into account, there was no overall yield difference between the cultivars, but there was still a decline in yield from 13 to 11.5 t/ha from the early to the later generations ($P = 0.06$) (Figure 11).

Final yield was affected by a hail storm on 23 February 2014, which damaged the canopy and triggered early senescence of the crop. Without this damage, yield differences may have been more marked.

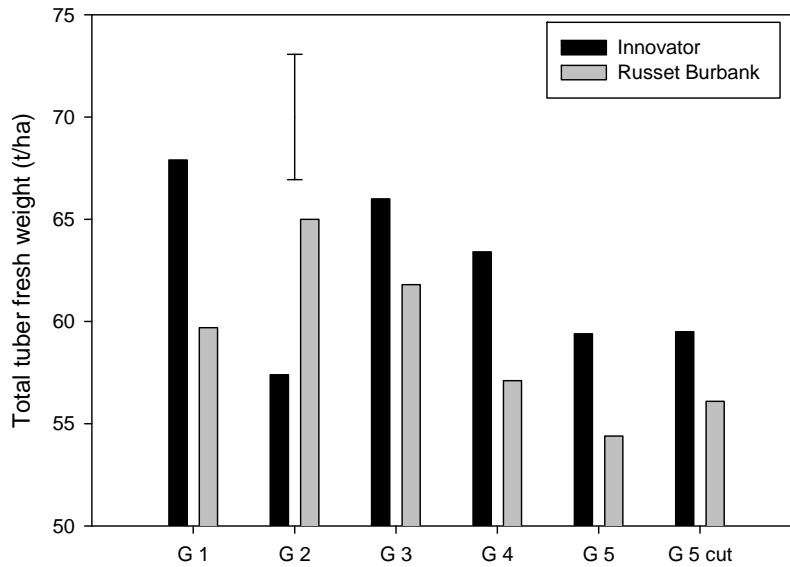


Figure 10. Mean total tuber fresh weight (t/ha) at final harvest for potatoes grown from five generations of 'Russet Burbank' or 'Innovator' seed tubers. Bar at LSD ($p = 0.05$), 22 df.

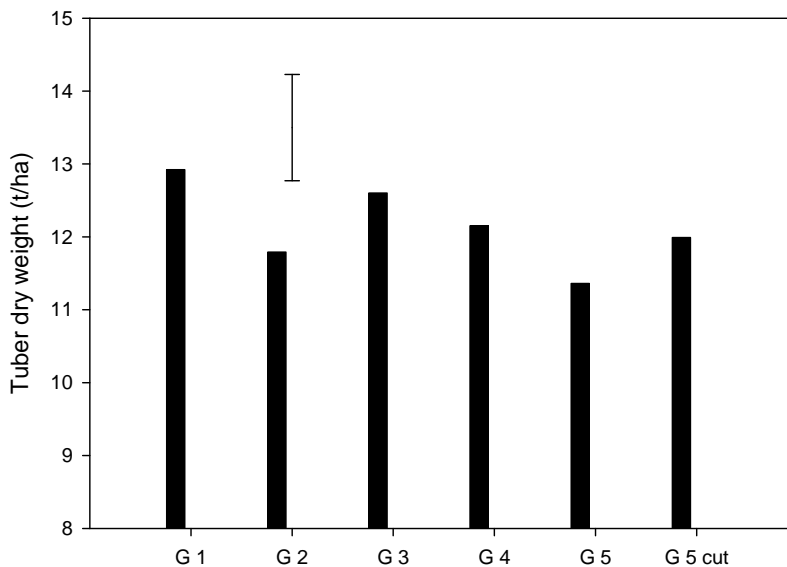


Figure 11. Mean total tuber dry weight (t/ha) at final harvest for potatoes grown from five generations averaged over 'Russet Burbank' and 'Innovator' seed tubers. Bar at LSD ($p = 0.05$), 22 df.

Mean stem number per plant was greater for plants from the older seed tuber generations ($P = <0.001$) and, compared to uncut seed, was also greater in G5 'Russet Burbank' plants where the seed tubers had been cut (Figure 12). G1 and G2 in both cultivars gave plants with fewer stem numbers (less than four) and for G3-5, 'Innovator' had greater stem numbers per plant (~5) than 'Russet Burbank' (~4).

For 'Innovator', mean number of tubers per stem decreased from 2.5 to 1.5 from G1 to G5 ($P = 0.004$), in response to increasing stem number (Figure 12). For this cultivar, mean tuber weights were similar for the different seed tuber generations, at between 200-250g (Figure 13). Mean tuber numbers per stem were similar for all generations of 'Russet Burbank' (2.8 tubers per stem), and were always greater than for 'Innovator' (1.7 tubers per stem, $P = 0.004$) (Figure 14). 'Russet Burbank' G2 gave a mean tuber fresh weight of 250g, whereas the other generations averaged about 150g.

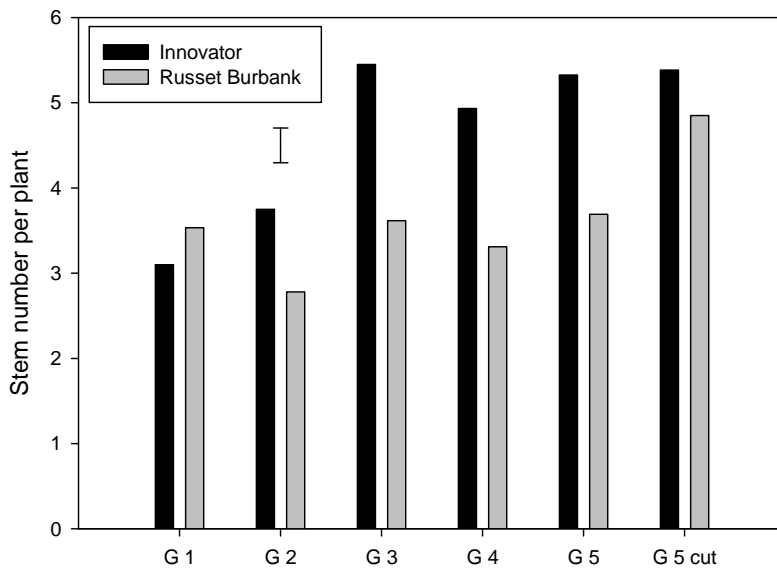


Figure 12. Mean numbers of stems number per plant for plants grown from five different seed tuber generations of 'Russet Burbank' and 'Innovator'. . Bar at LSD ($p = 0.05$), 22 df.

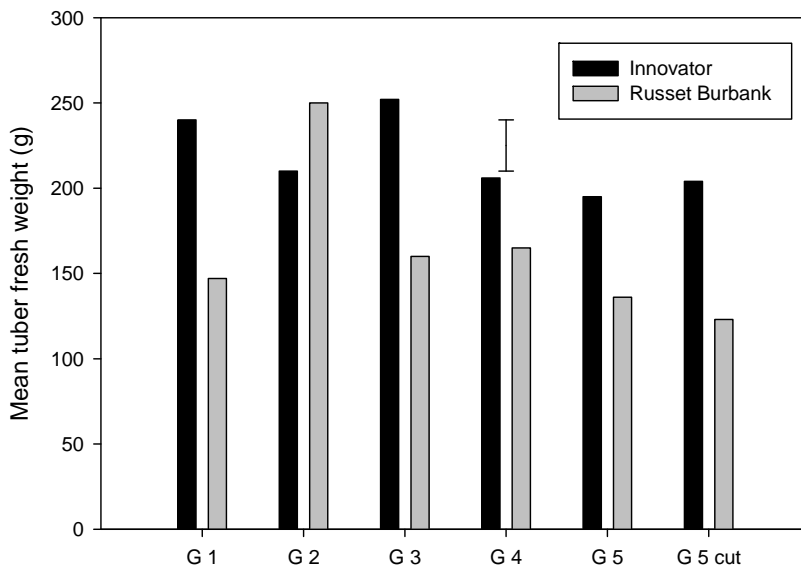


Figure 13. Mean tuber fresh weights for plants of 'Russet Burbank' and 'Innovator' grown from seed tubers of five different generations. . Bar at LSD ($p = 0.05$), 22 df.

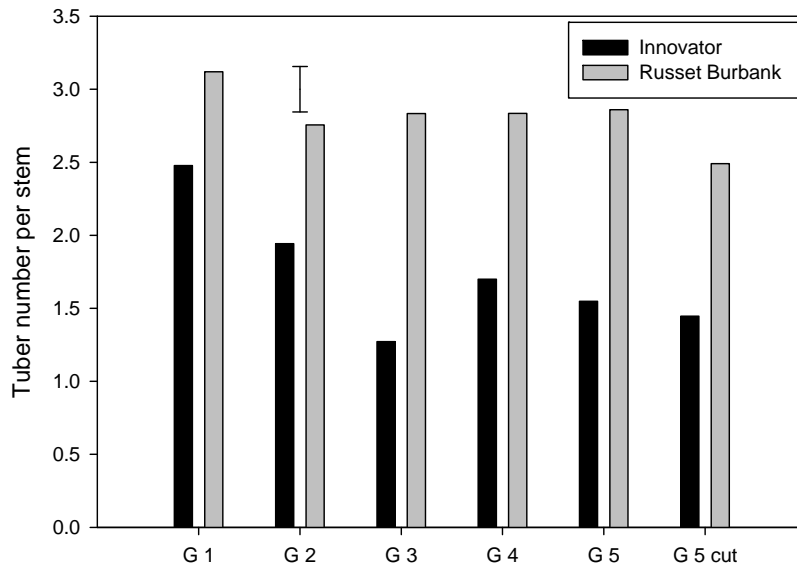


Figure 14. Mean number of tubers per stem for potatoes grown from five seed tuber generations of 'Russet Burbank' and 'Innovator'. . Bar at LSD ($p = 0.05$), 22 df.

The two main soil-borne diseases found in the crop were *Rhizoctonia* stem canker and *Spongospora*. Galls caused by *Spongospora* were frequently observed on the roots of the living plants, and powdery scab was common on the harvested tubers.

On 12 February 2014, at maximum canopy cover, stem canker was present on all the sampled stems from plants from all of the seed tuber generations (100% infection). Out of a maximum stem canker severity score of 18, the mean score for 'Russet Burbank' was 8, greater ($P = 0.008$) than that of 'Innovator' (6). There was some indication of variation in disease development across the seed tuber generation treatments, with less severe infections ($P = 0.10$) occurring in plants from the younger generations (Figure 15). This trend was similar for both cultivars.

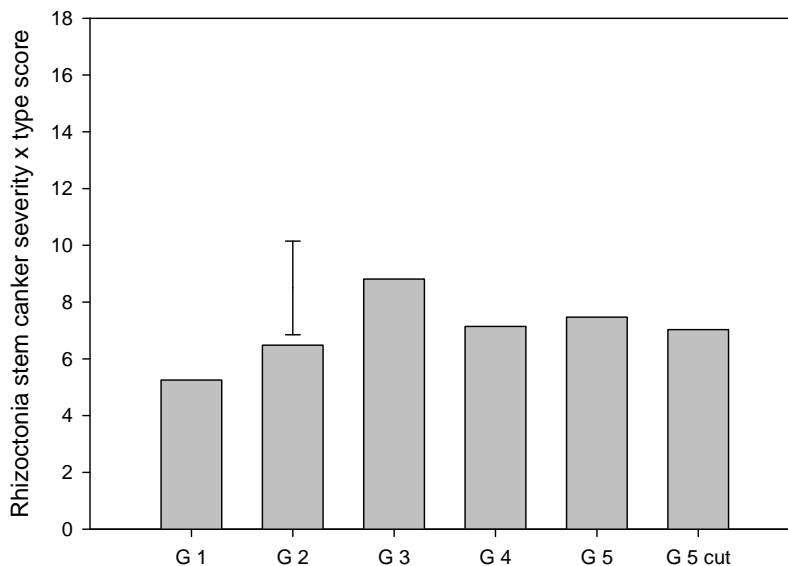


Figure 15. Mean *Rhizoctonia* stem canker severity scores on 12 February for potato plants grown from five seed tuber generations ('Russet Burbank' and 'Innovator' combined). Bar at LSD ($p = 0.05$), 58 df.

Spongospora root galls were found throughout all treatments on 12 February 2014. ‘Russet Burbank’ had a heavier infection (between 5 and 20 galls per plant, $P = <0.001$) than ‘Innovator’ (less than 5 galls per plant).

Powdery scab was found on tubers in both cultivars and from all the seed tuber generations at mid growth (12 February) and at final harvest (Figure 16). Amounts of tuber powdery scab were similar or greater by final harvest. ‘Russet Burbank’ G2 developed much greater powdery scab coverage on the tubers than all the other treatments.

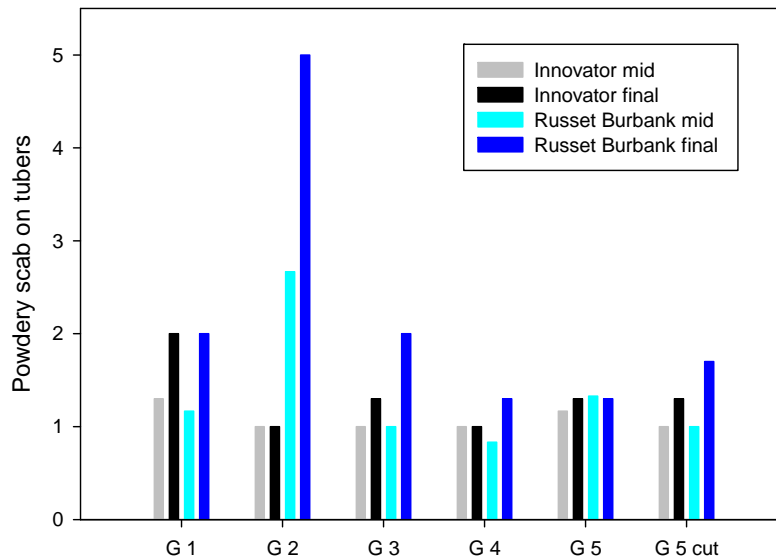


Figure 16. Mean powdery scab severities on the tubers from five seed tuber generations of ‘Russet Burbank’ or ‘Innovator’, for mid season (12 February) and final harvest (13 May).

3.3 Potential yield and yield gap

This project is part of an ongoing initiative to address the “yield gap” problem in Canterbury processing potato crops. During the 2013–14 season the focus was on quantifying the effects of soil-borne diseases on yield loss. The pesticides azoxystrobin and flusulphamide failed to selectively control any of the diseases observed in the trial, and all rates of chloropicrin failed to create disease-free environments. The treatment where no pesticides were applied had resulted in a similar yield (58 t/ha) to all of the different pesticide treatments.

The primary purpose of including the fumigation treatments was to have a “gold standard” to quantify the costs of soil-borne diseases to crop productivity and profitability by restarting the field at near zero pathogen presence. The assumption was that these “complete” treatments could have then been compared to the other treatments which targeted specific pathogens. There are a range of possible reasons the non-significant results obtained from these pesticide treatments. These include:

1. Fumigation was not effective. This could not be adequately assessed (using tomato indicator plants) because of health and safety issues associated with putting indicator plants in the field soon after chloropicrin applications.
2. The soil-borne pathogen population was not sensitive to azoxystrobin. This chemical is prone to the development of fungicide resistance in pathogens, and this could have been

the case as the fungicide is widely used for disease management in a range of arable crops.

3. The amounts of pathogen inoculum in the soil were too great for soil-applied pesticides to be effective.
4. Planting operations transmitted pathogen inoculum from untreated (control) plots to pesticide-treated plots.
5. The scale of the sampling was inadequate for detecting the differences between plots due to the localized nature of disease (unlikely but a possibility).
6. The seed tuber planting material was infected at high incidences with a range of pathogens which were then uniformly distributed across the field, i.e. the primary inoculum was seed tuber-borne. The predominant source of the inoculum could have been from the field where the seed crop was grown.

The potato crop model predicted a potential yield of 83 t/ha (Figure 17).

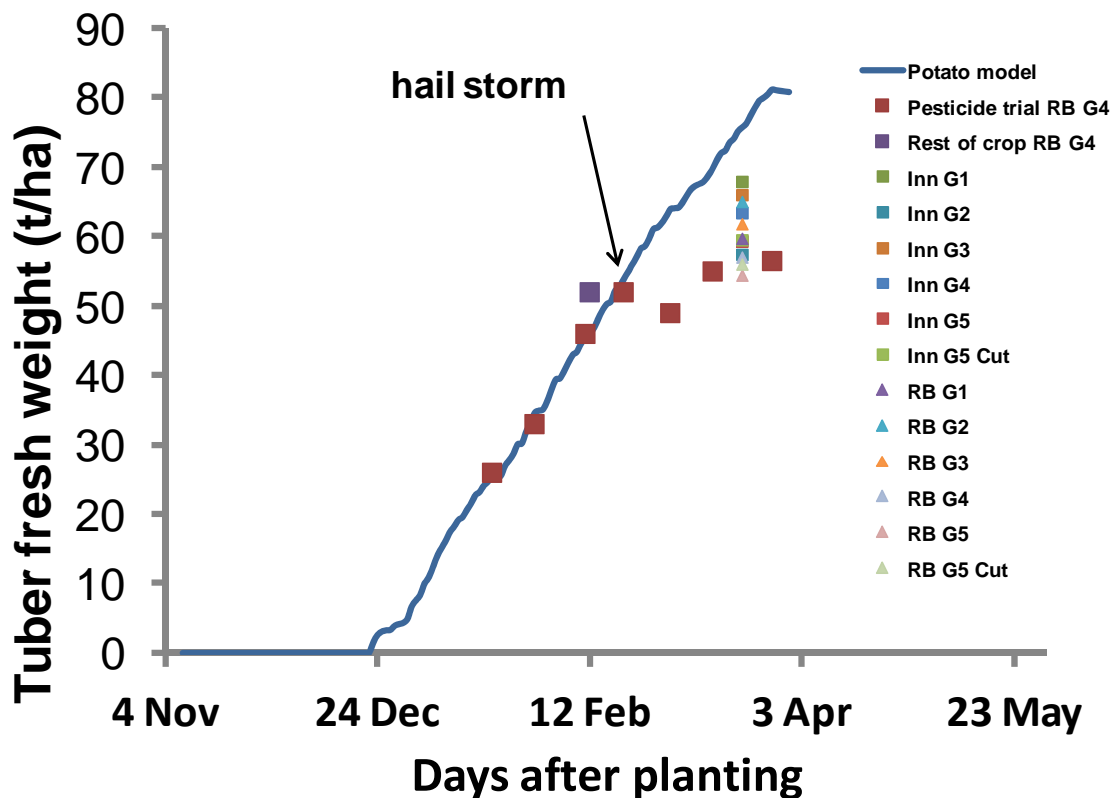


Figure 17. Accumulated potential yield (modelled) is compared to measured yield (t/ha) for the pesticide and generation trials, and also a yield estimate from the rest of the field.

Up until late February, both crop cover (Figure 18) and tuber yield accumulation (Figure 17) tracked closely to model predictions. This shows that, despite the presence of soil-borne diseases, adequate crop management allowed the plants to develop to potential for a period of time. However a hail storm on 23 February 2014 damaged the canopy and reduced crop cover by an estimated 20%. Crop senescence was hastened and the crop died about 3 weeks earlier than predicted.

Under these circumstances, the full yield potential of the crop could not be determined and yield-limiting effects of any particular factor(s) could not be apportioned. It is possible that the high levels of disease found in the crop could have eventually prevented yield accumulation from reaching potential.

It was noted that average yield of 58 t/ha from the 'Russet Burbank' G4 planted in the pesticide trial was similar that measured in G4 'Russet Burbank', grown in the generation trial (57 t/ha). These yields were both lower than that from the younger generations of 'Russet Burbank' planted in the generation trial. Generation trial treatment G2 yielded 65 t/ha and treatment G3 yielded 62 t/ha (Figure 17). Similar canopy development was recorded in all crops and soil-borne disease coverage of the stems was about the same, indicating there could be other reasons for difference in crop performance between generations.

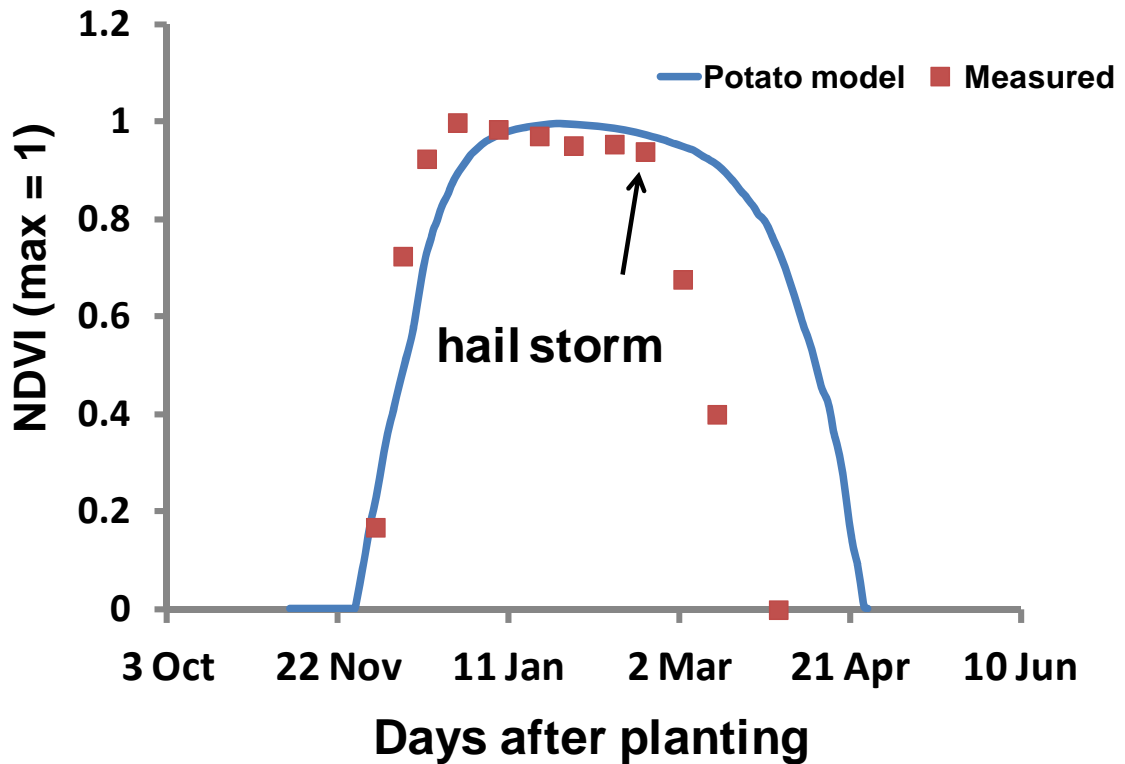


Figure 18. Modelled NDVI is compared to measured NDVI for the pesticide trial.

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