Biotic and abiotic factors affecting potato yields in Canterbury, New Zealand

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Abstract
Potato yields in Canterbury have remained static at c. 60 t/ha for the last decade. In contrast, potato growth models predict potential yields of up to 90 t/ha, which have previously been achieved by some commercial growers. A two-year project conducted by industry and research partners has examined factors constraining crop yields. In year 1, 11 processing crops were intensively monitored (final yield, plant health and soil quality assessments) throughout the growing season. Soil-borne diseases (*Rhizoctonia* stem canker and *Spongospora* root infection) were identified as consistent factors in reduced yields, along with subsurface soil compaction and inadequate irrigation management. Cropping histories that included potatoes within the last 10 years resulted in faster onset of symptoms of *Rhizoctonia* stem canker (by emergence), compared with fields with periods of grass growth and no previous potato crops (8 weeks after emergence). In year 2, a controlled field experiment in a commercial crop (known to have high levels of soil-borne pathogens) attempted to isolate and quantify the impacts of soil-borne diseases on yield. Treatments included soil fumigant (90, 112 and 146 kg/ha chloropicrin), in-furrow application of azoxystrobin (1.5 l/ha) or flusulphamide (400 ml/ha), and a nil pesticide control. Soil-borne pathogen DNA tests before and after treatment showed a slight reduction in DNA levels of *Rhizoctonia solani* and *Spongospora subterranea* in the soil (plots treated with fumigant), but results were very variable. Final total fresh yield averaged 58 t/ha and did not differ between treatments. Throughout the season, the severity of *R. solani* on underground stems was consistently less for the azoxystrobin treatment compared to all other treatments.

Key words
Solanum tuberosum L., Rhizoctonia solani, Spongospora subterranea, irrigation, fumigation, crop survey

Introduction

Yields from processing potato crops in Canterbury are currently static, at c. 60 t/ha paid yield (Pyke 2014). Computer-based potato yield modelling shows that yields of 80 to 90 t/ha are achievable if optimum growth conditions (climate, fertiliser, pest and disease control, irrigation) prevail, yields which have been previously achieved by some growers (Jamieson et al. 2003; Jamieson et al. 2006). This highlights a yield "gap" of c. 30 t/ha in current production, which could be caused by abiotic and biotic stresses. Jamieson *et al*. (2006) showed that nitrogen supply can limit potato yields, but previous research has shown that inadequate nutrient supply was not a major yield-limiting factor in Canterbury (Sinton et al. 2014). *Rhizoctonia solani* and *Spongospora subterranea* are soil-borne pathogens that have been shown to reduce commercial potato yields (Otrysko and Banville 1992; Hide and Horrocks 1994; Nitzan *et al*. 2010). *Rhizoctonia solani* causes stem canker and black scurf on tubers, while *S. subterranea* causes galls on roots and powdery scab on tubers. Both of these diseases have been commonly observed in Canterbury (Sinton *et al*. 2014). Inadequate irrigation management can also cause yield reductions (Jamieson et al. 2006). Soil compaction can also reduce potato yields through delayed emergence, restricted light interception and root development (Stalham *et al*. 2007). We summarise results from a 2-year project, including crop surveys and a targeted field experiment, investigating the factors responsible for the potato yield gap.

Materials and methods

Year 1 (2012-2013 growing season)
Processing potato crops (n = 11) ranging in area from 7 to 80 ha were intensively monitored throughout the growing season. The sites were located in Mid- and South Canterbury, New Zealand. Seven sites were planted with 'Russet Burbank' and four with 'Innovator'. For each cultivar, some sites had grown potatoes within the last 7 years, while other sites had not included potatoes in the rotation for at least 10 years. The sites were monitored for disease at 10- to 14-day intervals, from crop emergence to harvest. In each crop,

a representative observation plot, 10 m × 8 rows, was set up. At each visit, plants showing poor vigour or disease symptoms were also identified in other parts of the crop, the symptoms recorded, and the plants marked for final yield assessments. These were compared to healthy plants within the same crops (Wilcoxon paired test): 43 pairs in eight crops for comparing early diseased plants with healthy plants; 12 pairs in two crops for comparing wilting plants, missed by irrigators, with irrigated plants; and a 2.5 m by four rows weedy and weed-free area in one crop. Agronomic data recorded in the observation plot included: soil tests for quantification of soil-borne pathogen DNA (Root Disease Testing Service, SARDI, Australia); emergence counts; stem and plant counts; tuber yield assessment at key crop development stages; canopy, underground stem and root system (including tubers) disease assessments; virus incidence (DAS-ELISA); soil physical properties (e.g. texture, aggregate size distribution and stability, penetration resistance); and root mapping (depth and horizontal distribution) and vigour assessments. At crop senescence, final tuber yield and tuber size distribution (frequency of tubers in these size ranges: <67 mm, 67-90 mm, >90 mm length) was measured in each observation plot by hand digging 2.5 m by four rows, and this yield was then related to the in-season observations of crop health at that site. Potential yield was estimated using a potato growth model (daily time step) which accesses local weather and crop management information to take up water and nitrogen from a simulated soil, grow a canopy, produce biomass and distribute it among its organs. The model is sensitive to shortages of water and nitrogen (either slows leaf expansion or accelerates senescence).

Year 2 (2013-2014 growing season)

A replicated trial aimed to quantify the impact of soil-borne diseases on 'Russet Burbank' tuber yield and quality. The trial site had a cropping history that included potatoes within the last 10 years and was known to have large amounts of soil-borne pathogens. The soil treatments included a control (nil pesticides), three different rates (90, 112 and 146 kg/ha) of chloropicrin applied 3 weeks before planting, azoxystrobin (1.5 l/ha Amistar®) and flusulphamide (400 ml/ha Nebijin™) applied in-furrow at planting. The aim was to reduce all pathogens in some areas of the crop (chloropicrin treatments) and retain individual pathogen populations in others (azoxystrobin against *R. solani* and flusulphamide against *S. subterranea*), to estimate their individual and combined impacts on yield and tuber quality. Soil samples were taken before and immediately after the treatments were applied and tested for the presence of soil-borne pathogen DNA. The trial was a randomised block design with six treatments within each of three replicates (18 plots). Each plot was 130 m × 6 rows. Within each plot, five sub-plots assessed the effects of the treatments on plant growth and development. Agronomic information was collected every 10 days, including: incidence and severity of diseases affecting the crop canopy, root systems and tubers; yield and number of tubers; and crop cover using a Greenseeker® radiometer 505 Handheld sensor (NTech Industries, Trimble Navigation Ltd, Westminster, CO, USA). Incidence and severity of *R. solani* stem canker (RSC) were scored using the proportion of disease coverage on each plant stem (1 = no stem canker, 2 = 0-20% of stem area affected, 3 = 20-50%, 4 = 50-80%, 5 = >80%, or 6 = dead stem); and the form of the symptoms (1 = brown stem "speckling", 2 = speckling and solid brown lesions, or 3 = solid brown lesions). A severity × symptom score was calculated as the product of the severity and symptom type scores; e.g., a stem with >80% area affected by solid brown lesions scored 15 (5 × 3), and a dead stem scored the max, 18 (6 × 3). *Spongospora subterranea* galls on stems and roots were rated as: 1 = <5 galls/plant; 2 = 5-20, or 3 = >20 galls/plant. Final tuber yield and size distribution assessments were carried out after canopy senescence by hand digging a10 m^2 area in each plot. Data were analysed by Analysis of Variance (ANOVA) using GenStat (14th Ed, VSN International Ltd).

Results and discussion

Year 1

The average paid yield across the 11 crops was 55 t/ha, after an average deduction of approx. 10% by weight due to undersize tubers. The potential yield (paid) predicted by the model for that season was 87 t/ha, and the yield difference ("yield gap") ranged from 20 to 42 t/ha. At 90 t/ha, fresh gross yield (43 pairs of plants) was the greatest in sections of the crops where soil compaction, *S. subterranea* root galls or RSC were absent (Figure 1). Increasing severity of these diseases and the presence of soil compaction progressively reduced yield, and the smallest yield (30t/ha) where all three factors occurred. Soil- and/or seed tuber-borne diseases, along with the presence of soil compaction, were the most prevalent factors associated with yield reduction. Soil compaction zones were found in six of the 11 crops between 20-30cm below the ridge top and very close to the seed tuber position. Penetration resistance in these zones varied from 1 to 4.8 MPa and previous

has shown that potato root growth slows significantly beyond 1.5MPa (Stalham *et al*. 2007) . Few roots were found beneath these compaction zones, except where sub-soiling had taken place.

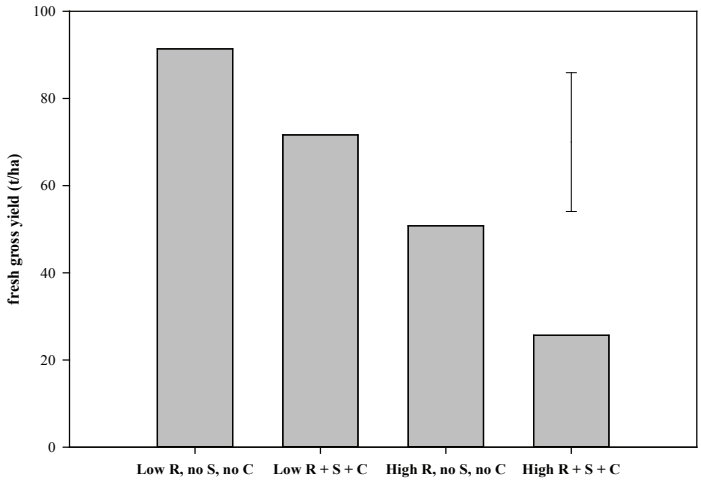

Figure 1. Averaged fresh "plant yield" from targeted areas in 11 potato crops. Categories are: "Low R, no S, no C" = low *Rhizoctonia* stem canker (RSC) incidence, no *Spongospora subterranea* root galls and no soil compaction; "Low R + S + C" = low RSC incidence, with *S. subterranea* root galls and soil compaction; "High R, no S, no C" = high RSC incidence, no *S. subterranea* root galls and no soil compaction; "High R + S + C" = high RSC incidence, with *S. subterranea* root galls and soil compaction. Bar represents LSD (5%) with approx. 76 df.

RSC occurred in all of the crops (but not in all plants), and *S. subterranea* root galls were also found in five of the crops. Inadequate irrigation management caused 13-28% yield loss (12 pair-wise comparisons). Poor weed control (a 15% yield loss was measured in a heavily infested area of one crop) also contributed to the yield gap in one crop. All five crops with *S. subterranea* root galls were in compacted soils, suggesting a link between poor water percolation and the presence of the disease. Whether or not potatoes were previously grown in a field did not directly affect yield in the 11 crops in the year of study, however levels of disease were related to history as well as the types of crops previously grown. Where grass had been grown the year before and potatoes were not part of the cropping history, the onset of RSC was delayed by up to 8 weeks. Conversely, annual crops grown the year before plus a recent cropping history of potato resulted in RSC symptoms showing at emergence of the current potato crop.Very low incidences of Potato virus X, Potato virus Y, Potato virus A, Potato virus M, Potato virus S and Potato leafroll virus were measured in most of the crops. This indicates that virus diseases were unlikely to have affected yields in the surveyed crops. Furthermore, these results were expected as the use of early generation (4^{th} and 5^{th}) seed tubers is common in Canterbury (S. Clelland pers. comm., 2012).

Year 2

Before fumigation or pesticide application, the amount of *S. subterranea* DNA averaged 1400 pg/g of soil across the treatments. There were slight reductions in *S. subterranea* DNA after the application of chloropicrin (all three rates) to an average of 1100 pg/g of soil, although this reduction was not significant (P = 0.06). Before application of the treatments, amounts of *R. solani* AG2.1 DNA (responsible for RSC) was highly variable and ranged between 250 to 700 pg/g of soil. Quantities of *R. solani* AG2.1 DNA were significantly reduced by the low (90 kg/ha) and medium (112 kg/ha) rates of chloropicrin to 150 pg/g soil (P = 0.002). The range of R. solani AG2.1 DNA quantities measured in the trial area was similar to that measured in most of the 11 crops surveyed in Year 1, but quantities were greater for *S. subterranea* compared with the five crops in Year 1 where the pathogen was present). Even though variable, the pre-treatment pathogen DNA results were consistent with anecdotal evidence that the field was likely to be highly diseased and therefore suitable for this type of experiment. Throughout the season, there was increasing incidence and severity of diseases caused by *R. solani* and *S. subterranea* in all treatment plots except those receiving azoxystrobin. Plants in the azoxystrobin-treated plots had less RSC (P = 0.013) than the other treatments (final mean severity × symptom score = 9 for azoxystrobin treatment cf. 13.5, mean of all other treatments (Figure 2). Final tuber yield did not differ between treatments (P = 0.782), which averaged 58 t/ha. This was attributed to the lack of control of soil-borne diseases, coupled with a severe hail storm in late February, which reduced canopy cover and hastened senescence.

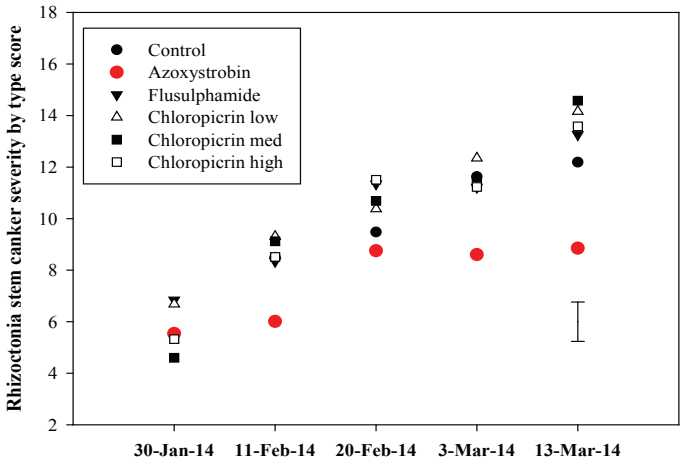

Figure 2. Mean *Rhizoctonia* stem canker severity scores (at different times during late crop growth) for potato plants grown in plots treated with different pesticides (cf. Materials and methods). Bar represents LSD (5%) with 10 df.

The lack of major disease reductions from the pesticides treatments may be explained by several factors. The treatments may have been ineffective because of poor soil penetration by fumigant, the local *R. solani* population was insensitive to azoxystrobin, and/or the diseases were too severe. Additionally, pathogen re-introduction may have occurred from surrounding untreated areas. Three rates of chloropicrin were tested in the experiment because this chemical had not been previously tested in commercial potato crops in New Zealand. Conditions were considered optimum at the time the chloropicrin treatments were applied.

Conclusion

This yield gap study of processing potato crops in Canterbury suggests that soil-borne diseases, in conjunction with soil compaction in the potato root zone, are likely to be the main factors reducing crop yields. Inadequate irrigation management and poor weed control were also identified as yield-limiting factors in some crops. These factors are also likely to be responsible for yield variability within individual crops. They can have cumulative impacts on potato yields and they can interact with one another. For example, soil compaction and excessive irrigation provide favourable conditions for development of soil-borne diseases. To reduce the yield gap, potato growers need to place more emphasis on a total package of best management practices. These would include: selecting clean cropping sites (no previous history of disease), optimising irrigation, fertiliser and foliar disease management to grow an unstressed crop. Future work should focus on improving soil health through crop rotation, improved irrigation management, and reducing soil compaction.

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