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RESEARCH ARTICLE

Determination of optimal timing of 2,4-dichlorophenoxyacetic acid foliar applications for common scab control in potato

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Abstract

Application of 2,4-dichlorophenoxyacetic acid (2,4-D) has been shown to be an effective control method for common scab of potato. Prior research targeted applications at or shortly after tuber initiation during the period of known susceptibility, with later treatment providing little or no control. This study examined the effect of a range of application dates and the influence of multiple applications at low and high rates on common scab control in both Russet Burbank and Desiree cultivars. We show that applications as early as 5 days after average plant emergence gave the greater disease control for both cultivars. These early treatments provide sufficient material to the tuber to induce resistance that lasts throughout the tuber susceptibility period with no requirement for, or benefit of repeated applications. Agronomic assessments suggested minor effects on total tuber mass produced with 2,4-D treatment but these were not consistent. Effective treatments result in levels of 2,4-D at harvest below the Australian maximum residue limit.

Introduction

Common scab is a globally important soil-borne disease of potato characterised by distinctive lesions on the tuber surface (Loria *et al*., 2006). The disease is induced following infection of expanding tubers with pathogenic *Streptomyces* spp., the most common being *Streptomyces scabies* (Loria *et al*., 2006). All pathogenic *Streptomyces* spp. produce the phytotoxin thaxtomin A (King *et al*., 1989), a modified dipeptide that inhibits cellulose biosynthesis (Bischoff *et al*., 2009), whereas non-pathogenic strains do not (King *et al*., 1991), and its essential role in disease induction appears implicit (Goyer *et al*., 1998; Kers *et al*., 2005).

There is no single effective management option for this disease, rather an integrated approach is required to minimise disease. Control strategies used include management of planting dates (Waterer, 2002*a*), use of resistant cultivars (Pasco *et al*., 2005; Wilson *et al*., 2010), manipulation of soil pH (Lacey & Wilson, 2001; Waterer, 2002*b*) and strategic irrigation during early tuber development (Lapwood *et al*., 1973; Wilson *et al*., 2001). Treatment of potato seed tubers with fungicides or other biocides can reduce disease from seed-borne inoculum (Wilson *et al*., 1999). Pentachloronitrobenzene was a widely used soil-applied fungicide that gave good disease suppression from soil-borne inoculum (Davis *et al*., 1974; Hooker 1981); however, the material is a carcinogen and its use has been withdrawn from most potato production regions around the world without an effective replacement. Subsequently, trials were conducted in the UK to test a range of alternate chemicals for their ability to control common scab. These initially focussed on materials incorporated into the soil; however, effective compounds often displayed phytotoxicity (McIntosh, 1973, 1976). Later trials evaluated treatments applied to potato foliage at or just after tuber initiation, known to be a critical period for tuber infection (Lapwood *et al*., 1973; Khatri *et al*., 2011). These trials discovered 2,4-dichlorophenoxyacetic acid (2,4-D) and 3,5-dichlorophenoxyacetic acid (3,5-D) to be highly effective in reducing common scab in glasshouse trials. However, both materials produced phytotoxic effects including reduced yield, an increased numbers of small tubers and tuber deformation. McIntosh *et al*. (1981) found applications of 3,5-D prior to tuber initiation increased the efficacy of disease control, but also increased

phytotoxic effects and that the levels of disease control following 3,5-D treatment observed in glasshouse trials were not replicated in field trials. As such, these treatments were not considered commercially viable (McIntosh *et al.*, 1981, 1982). Subsequent studies with 2,4-D foliar treatments applied at or shortly after tuber initiation have repeated effective disease control in glasshouse trials, but have also shown highly effective control in the field (Tegg *et al.*, 2008) and also provided evidence towards a putative mechanism of activity. At concentrations applied 2,4-D does not affect pathogen growth (McIntosh *et al.*, 1981; Tegg *et al.*, 2008) nor its ability to produce thaxtomin A (Tegg *et al.*, 2008). However, there is a strong relationship between disease control and the level of tolerance of tuber tissues to thaxtomin A-induced necrosis following foliar application of 2,4-D and other substituted phenoxy acids, benzoic acids or picolinic acids (Tegg *et al.*, 2008, 2012). This suggested that 2,4-D and the other active materials may operate by reducing the sensitivity of potato tubers to thaxtomin A, and through this reducing the invasive capacity of the pathogen.

Timing of treatment to coincide with tuber initiation can be problematic in the field. Plants must be periodically dug and examined for hook development on underground stolons, a precursor to tuber initiation. However, tubers are produced asynchronously and initiation between plants will vary with time of emergence and early plant development. Failure to apply suppressive materials prior to the tubers entering the susceptible phase will negate any protective effect. Also, delays in material reaching tubers following foliar application must be accounted for. Burrell (1982) found that after 1 day only 0.3% of 2,4-D applied to foliage had been translocated to tubers, and after 4 days it increased to only 2–3%.

In an attempt to widen the window of treatment, multiple sprays have been applied commencing at tuber initiation that did provide greater common scab suppression than single applications (Tegg *et al.*, 2008). This article describes studies to determine the optimal timing for application of 2,4-D foliar treatments to provide the greatest common scab control; to provide indicative assessment of treatment effects on tuber yield and quality; to evaluate levels of 2,4-D in tubers at harvest following treatments and to further examine the effect of single and multiple treatments on disease control when applied at various stages of plant development.

Materials and methods

Inoculum preparation for pot trials

Pathogenic *S. scabies* strain G#20, initially isolated from a common scab-infected tuber harvested from north-west Tasmania in 1990, was used in these experiments. The isolate was grown on 10-mL ISP2 (Shirling & Gottlieb, 1966) agar slopes (10 g L^{-1} malt extract, 4 g L^{-1} yeast extract, 4 g L^{-1} glucose and 12 g L^{-1} agar, pH 7.3) until sporulation. Colonised agar slopes were aseptically transferred to a sterilised mixture of 120-g vermiculite and 500-mL SAY solution (20 g L^{-1} sucrose, 1.2 g L^{-1} L-asparagine, 0.6 g L^{-1} K_2HPO_4 and 10 g L^{-1} yeast extract, pH 7.2; Labruyère, 1971). The inoculum was incubated in the dark at 24°C until profuse sporulation. Approximately 1 L of vermiculite inoculum was added to 25 L of potting mix containing sand, peat and composted pine bark at a ratio of 10:10:80, at pH 6.0, premixed with Osmocote 16:3.5:10 N : P : K resin-coated fertiliser (Scotts Australia Pty Ltd, Sydney, Australia) at the rate of 6 kg m^{-3} and thoroughly mixed using a cement mixer. The inoculum containing potting mix was then used to fill plastic planter bags of 5-L capacity (200 mm × 200 mm, Botany Horticultural Pty Ltd, Gold Coast, Australia).

Planting material

Potato tubers of the varieties Russet Burbank (moderate resistance to common scab) and Desiree (moderate susceptibility) were used in all trials. For the glasshouse trials 15-g seed pieces from disease-free mini-tubers harvested from glasshouse grown tissue-culture plants were used, whereas for the field trials 50-g seed pieces cut from visibly clean, certified commercial seed tubers were used.

Preparation of 2,4-dichlorophenoxyacetic acid treatments

For pot trials treatment solutions were prepared from crystalline 2,4-D (Sigma Aldrich, St. Louis, MO, USA) to provide 200 mg L^{-1} and 100 mg L^{-1} solutions. Tween-80 (0.5 g L^{-1}) was added as a surfactant to both the 2,4-D treatments and water controls. For field trials the less volatile amine form of 2,4-D was used to minimise the chance of damage to nearby horticultural crops. Amicide 625 (Nufarm Pty Ltd, Melbourne, Australia) with 625 g L^{-1} 2,4-D present as dimethylamine and diethanolamine salts was used. Treatment solutions prepared had an equivalent of 25 mg L^{-1} and 100 mg L^{-1} 2,4-D with 0.5 g L^{-1} Tween-80 added.

Pot trials

Two pot trials designed to determine the effect of application date on treatment efficacy were planted in 2009 and 2010. Both trials had 15 2,4-D spray treatments, grouped in four application strategies: (a) 'single' – single

treatment of 200 mg L^{-1} 2,4-D applied at 10, 20, 30, 40 or 50 days after emergence (DAE); (2) 'double' – two treatments, each of 200 mg L^{-1} 2,4-D, the first applied at 10, 20, 30, 40 or 50 DAE, the second applied 10 days after the first application (i.e. at 20, 30, 40, 50 and 60 DAE, respectively), with a total of 400 mg L^{-1} 2,4-D applied; (3) 'double (half rate)' – two treatments, each of 100 mg L^{-1} 2,4-D, the first applied at 10, 20, 30, 40 or 50 DAE, the second applied 10 days after the first application (i.e. at 20, 30, 40, 50 and 60 DAE, respectively), with a total of 200 mg L^{-1} 2,4-D applied and (4) 'control' – three water only treatments, one applied as a single spray at 10 DAE and two applied as double sprays at 10 and 20 DAE. Each treatment was applied to five pots of each variety (180 pots in total) with pots arranged in a randomised block design. Prior to application, plants receiving treatment were separated from other trial plants to prevent possible spray drift contamination. Sprays were applied to foliage as a fine mist until run off. Plants were allowed to dry before being returned to the trial.

In pot trial No. 1 plants were grown in a glasshouse with all pots hand-watered ensuring the potting soil dried between waterings to maintain a suitable environment for disease. No other pesticides were applied. Glasshouse temperatures were maintained between 25°C and 30°C. At approximately 50 DAE an additional 1 L of a combination of inoculated vermiculite and potting mix at a ratio of 1:2 by volume was added to each pot to prevent exposure of developing tubers to sunlight. In pot trial No. 2 plants were kept outdoors and subject to natural weather events and temperatures with supplemental irrigation applied as required ensuring soil dried between waterings. The trials were planted on 30 January 2009 (pot trial No. 1) and 19 January 2010 (pot trial No. 2) with full emergence on 8 February 2009 (pot trial No. 1) and 1 February 2010 (pot trial No. 2). Tubers were harvested, following plant senescence, on 25 May 2009 (pot trial No. 1) and 1 May 2010 (pot trial No. 2) and stored at 4°C for up to 4 weeks before assessment. Soil was brushed from tubers and tuber number and mass (fresh weight, FW) were measured with any tuber disfigurements noted. Tubers were assessed for common scab and sensitivity to thaxtomin A.

Field trials

Field trial No. 1 was planted on 21 October 2008 at Bishopsbourne, north-west Tasmania, on a brown clay soil that had been sown with potatoes in the previous season. The trial was arranged in a randomised split plot design, each plot consisting of two subplots of five plants, one of each variety, with four replicates. The average emergence date was 30 November. There were 15 2,4-D spray treatments, grouped as three application strategies: 'single' – a single treatment of 100 mg L^{-1} 2,4-D applied at 10, 20, 30, 40, 50 or 60 DAE; 'double' – two treatments, each of 100 mg L^{-1} 2,4-D were applied, the first at 10, 20, 30, 40 or 50 DAE and the second 10 days after the first treatment (i.e. at 20, 30, 40, 50 and 60 DAE, respectively); and 'triple' – three treatments, each of 100 mg L^{-1} 2,4-D were applied, the first at 10, 20, 30 or 40 DAE, the second 10 days after the first treatment (i.e. at 20, 30, 40 and 50 DAE, respectively) and the third 20 days after the first treatment (i.e. at 30, 40, 50 and 60 DAE, respectively). Water only controls were applied once, twice or thrice (first application at 10 DAE). Sprays were applied with a backpack rig to the foliage until run off. Minimal irrigation was applied using a centre pivot to encourage disease. Plots were hand weeded when required. As plants began to senesce the plots were sprayed with a desiccant (Reglone®, Syngenta Crop Protection, Cambridge, UK) as per industry standards, with harvest on 2 April. All the tubers of each variety were collected and combined from the five plants within each plot. Tubers were stored for 1–2 weeks at 4°C prior to being washed and weighed to determine yield for each plot (the combined tubers from five plants) and assessed for common scab.

Field trial No. 2 (2009/2010) was planted on 17 November 2009 at Waterhouse, north-east Tasmania, on a predominately sandy soil that had been sown with potatoes in the previous season. The area around the trial site was sown with an oil-seed crop. The trial layout used in field trial No. 1 was repeated. The average emergence date was the 10 December. There were 22 2,4-D spray treatments, grouped in two application strategies: 'single' – a single treatment of either 25 or 100 mg L^{-1} 2,4-D, each applied at 5, 10, 20, 30, 40 or 50 DAE, and 'double' – two treatments of either 25 or 100 mg L^{-1} 2,4-D, each applied at 5, 10, 20, 30 or 40 DAE, with a second application at 10, 20, 30, 40 and 50 DAE, respectively. Water only controls were applied at 10 DAE for the single treatment, and at 10 and 20 DAE for the double treatment. Trial maintenance was as provided for field trial No. 1, with harvest on 8 April. All tubers were assessed for yield and disease. In addition, two tubers per replicate were assessed for toxin sensitivity and the levels of 2,4-D within selected tubers at harvest were quantified.

Disease assessment

Only tubers >2 g were assessed for common scab. Disease severity was assessed by tuber surface cover score (0: no visible disease on tuber surface, 0.5: <1%, 1: 1–<5%, 2: 5–<10%, 3: 10–<30%, 4: 30–<50%, 5: 50–<70% and 6: 70–100% tuber surface affected) with a percentage

tuber coverage estimated from the mid values of these score ranges, and by lesion depth score (LDS) with the depth of the deepest lesion present on a tuber recorded (0: no lesions present, 1: superficial lesions <1 mm deep, 2: lesions 1–<2 mm deep, 3: lesions 2–<3 mm deep and 4: lesions ≥3 mm deep). Disease incidence was assessed as the proportion of tubers with visible lesions present (Wilson *et al*., 2009).

Thaxtomin sensitivity of harvested tubers

Thaxtomin sensitivity of harvested tubers was measured using the following method of Tegg *et al*. (2008). In brief, harvested tubers stored for approximately 2 weeks at 4°C were surface sterilised and aseptically cut into 0.5-cm slices. Tuber slices were placed on filter paper moistened with sterile distilled water in a Petri dish. Thaxtomin A-amended (14 µM for Desiree and 7 µM for Russet Burbank) 6-mm-diameter Whatman No. 1 paper discs (2–4 per tuber slice) were placed on the cut surface of each tuber slice. The discs were moistened with 10 µL sterile distilled water to ensure attachment to the tuber slice and incubated in the dark at 24°C for 7 days after which visible necrosis beneath discs was scored (Fig. 1). Each treatment was replicated thrice with each replicate comprising 8–10 discs.

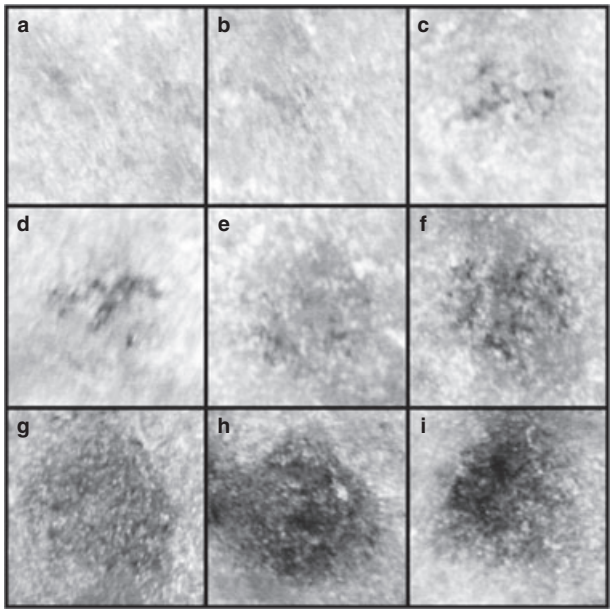

Figure 1 Thaxtomin A-induced necrosis in tuber slice assay (a: 0 = no necrosis; b: 0.5 = very sparse flecks; c: 1 = few light brown specks; d: 1.5 = few dark brown specks; e: 2 = light brown flecks in a circle; f: 2.5 = dark brown flecks in a circle; g: 3 = light brown necrosis; h: 3.5 = dark brown necrosis and i: 4 = black necrosis).

2,4-Dichlorophenoxyacetic acid quantification in harvested tubers

2,4-Dichlorophenoxyacetic acid was quantified within two to three disease-free tubers of a representative size from each treatment within a single replicate. Tuber tissue (10 g) was cut into 1-cm^2 pieces and stored in 80% methanol with butylated hydroxytoluene at −20°C. Samples were homogenised, left overnight at 4°C and vacuum filtered. Ninety nanograms of 2,4-D internal standard ([$^{13}C_6$] 2,4-D (Catalogue no. XA11940 200AC), Dr. Ehrenstorfer Laboratories, Augsburg, Germany) was added to 10% aliquots of each sample. Methanol was removed in a sample concentrator. Samples were purified by loading onto a preconditioned Sep Pak (Waters VacRC 500 mg) C18 cartridge in 0.4% acetic acid. The 2,4-D was eluted from the cartridge with 3 mL of acetonitrile, which was removed in a sample concentrator. One hundred microlitres of 1% acetic acid was then used to transfer the sample to a microcentrifuge tube. Samples were centrifuged at 13 000 g for 3 min, and 70 µL was analysed by UPLC-MS. The system comprised a Waters Acquity H-series UPLC coupled to a Waters Xevo triple quadrupole mass spectrometer. The column was a Waters Acquity UPLC BEH C18 (2.1 mm × 100 mm × 1.7 µm particles), with mobile phases A = 1% acetic acid in water and B = acetonitrile. The flow rate was 0.3 mL min^{-1}, using a gradient starting from a 70:30 ratio of solvents A : B, then a linear ramp to 30:70 A : B at 4.5 min and then held for 30 s, before immediate re-equilibration to initial conditions for 3 min. The column was held at 45°C. The mass spectrometer was operated in negative ion electrospray mode using multiple reaction monitoring (MRM) mode. The ion source was at 130°C, the desolvation gas was nitrogen at 950 L h^{-1}, the cone gas was nitrogen at 50 L h^{-1}, the desolvation temperature was 450°C, the capillary voltage was 2.7 KV and the cone voltage was 20 V. MRM transitions for quantitation using 18 V collision energy were 2,4-D – *m/z* 219–161 and $^{13}C_6$ 2,4-D – *m/z* 225–167. Confirmation channels monitored were 2,4-D – *m/z* 219–125, collision energy 28 V; *m/z* 221–163, collision energy 18 V; $^{13}C_6$ 2,4-D – *m/z* 227–169, collision energy 18 V. Dwell time for all channels was 95 ms.

Data analysis

Data were analysed in Genstat 12.1 (VSN International Ltd, Hemel Hempstead, UK). Multivariate analysis of variance was used to determine significant effects from and interactions between treatment factors. Probabilities less than 0.05 were considered to be significant and Fisher's least significant difference (LSD) test was used for comparison of treatment means.

Table 1 Pot trial No. 1: The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single or double treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants on common scab incidence and severity. There was a significant effect found from variety on DCS ($P < 0.001$, standard error difference; SED = 0.11, LSD = 0.21), LDS ($P < 0.001$, SED = 0.12, LSD = 0.23) and proportion of diseased tubers ($P = 0.003$, SED = 2.2, LSD = 4.4). Treatments with the same letter in the same variable are not significantly different at $P = 0.05$ using Fisher's least significant difference (LSD) test

		Disease Cover Score [Percentage Cover (%)][b]			Lesion Depth Score[c]			Diseased Tubers/Pot (%)[d]		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
200 mg L^{-1} (single)	10	0.00	0.40 (1.2)	0.20	0.00	0.60	0.30	0.0	10.7	5.3
	20	0.20 (0.6)	0.00	0.10	0.40	0.00	0.20	5.0	0.0	2.5
	30	0.40 (1.5)	0.00	0.20	0.40	0.00	0.20	10.0	0.0	5.0
	40	0.00	0.50 (2.8)	0.25	0.00	0.50	0.30	0.0	4.0	2.0
	50	0.00	0.40 (1.5)	0.20	0.00	0.20	0.10	0.0	3.3	1.7
Control (0 mg L^{-1})		0.00	1.00 (5.5)	0.50	0.00	1.00	0.50	0.0	25.0	12.5
200 mg L^{-1} (double)	10, 20	0.00	1.10 (4.9)	0.55	0.20	1.20	0.70	0.0	21.2	10.6
	20, 30	0.00	0.80 (3.0)	0.40	0.00	0.60	0.30	0.0	10.0	5.0
	30, 40	0.20 (0.6)	1.00 (5.5)	0.60	0.40	1.00	0.70	6.7	10.0	8.3
	40, 50	0.40 (1.5)	0.40 (1.5)	0.40	0.40	0.20	0.30	4.0	4.0	4.0
	50, 60	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.0	0.0
Control (0 mg L^{-1})		0.30 (0.7)	0.87 (3.8)	0.58	0.20	0.60	0.40	10.0	16.0	13.0
100 mg L^{-1} (double)	10, 20	0.00	0.50 (1.6)	0.25	0.00	0.60	0.30	0.0	5.1	2.5
	20, 30	0.00	0.00	0.00	0.00	0.20	0.10	0.0	0.0	0.0
	30, 40	0.30 (0.7)	0.80 (3.0)	0.55	0.40	1.00	0.70	9.0	15.0	12.0
	40, 50	0.40 (1.5)	1.10 (3.7)	0.75	0.40	1.30	0.90	3.3	21.4	12.4
	50, 60	0.20 (0.6)	1.10 (4.1)	0.65	0.20	1.10	0.70	4.0	25.0	14.5
Control (0 mg L^{-1})		0.20 (0.6)	0.60 (2.1)	0.40	0.20	1.20	0.70	4.0	5.3	4.7
Mean (first DAE)	10	0.00	0.67	0.33	0.07	0.80	0.43	0.0	12.3	6.1
	20	0.07	0.27	0.17	0.13	0.27	0.20	1.7	3.3	2.5
	30	0.30	0.60	0.45	0.40	0.67	0.53	8.6	8.3	8.4
	40	0.27	0.67	0.47	0.27	0.67	0.47	2.4	9.8	6.1
	50	0.07	0.50	0.28	0.07	0.43	0.25	1.3	9.4	5.4
Mean (controls)		0.17	0.82	0.49	0.13	0.93	0.53	4.7	15.4	10.1
	Mean (Var.)	0.14 a	0.59 b		0.18 a	0.63 b		3.1 a	9.8 b	

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

[b]Disease cover score (DCS; 0–6 scale; Wilson *et al.*, 2009) assesses surface area of tubers covered with lesions. Percentage cover is the estimated percentage of tuber surface coverage derived from the DCS.

[c]Lesion depth score (LDS; 0–4 scale; Wilson *et al.*, 2009) assesses mean of the lesion depth of deepest lesion per tuber.

[d]Proportion of diseased tubers measures the number of tubers with visible lesions as a proportion of total tubers per pot.

Results

Common scab control

In pot trial No. 1 the observed disease levels were very low. There was no significant effect of timing, rate or frequency of 2,4-D treatments on disease although there was a general trend towards greater disease in treatments applied later and in the control. There was a significant effect of variety with Desiree having greater disease cover scores (DCS), LDS and proportion of diseased tubers than Russet Burbank (Table 1). Pot trial No. 2 also had low disease. There were significant effects of treatment timing on DCS, LDS and the proportion of diseased tubers to healthy tubers. The control and 50 DAE had significantly greater disease than other treatments except for 20 DAE for LDS and proportion of diseased tubers. Once again there was a general trend for earlier application dates to show less disease across all parameters. There was no significant effect of treatment frequency, rate or variety except that Russet Burbank had greater proportion of diseased tubers than Desiree (Table 2).

Field trial No. 1 had greater disease than the pot trials. All harvested tubers had at least one lesion (100% incidence; Table 3). There was a significant interaction between the date of the first 2,4-D application, the frequency of application and variety on both measures of disease severity (Table 3). In both varieties DCS and LDS progressively increased with the increasing delay of the first spray date regardless of whether treatments were single, double or triple sprays. Treatments with the first application at the earliest time point (10 DAE) invariably gave the least disease. There were no obvious data trends for treatment frequency, rate or variety (Table 3). In field trial No. 2 disease incidence was again very high

Table 2 Pot trial No. 2: The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single or double treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants on common scab incidence and severity. There was a significant effect found from date of first spray on DCS ($P < 0.001$, standard error difference; SED = 0.11, LSD = 0.23), LDS ($P < 0.001$, SED = 0.14, LSD = 0.29) and from date of first spray ($P = 0.003$, SED = 4.41, LSD = 8.73) and variety ($P = 0.003$, SED = 2.25, LSD = 5.04) on proportion of diseased tubers. Treatments with the same letter in the same variable are not significantly different at $P = 0.05$ using Fisher's least significant difference (LSD) test

		Disease Cover Score [Percentage Cover (%)][b]			Lesion Depth Score[c]			Diseased Tubers/Pot (%)[d]		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
200 mg L^{-1} (single)	10	0.15 (0.4)	0.10 (0.1)	0.13	0.20	0.20	0.20	13.3	5.0	9.2
	20	0.20 (0.6)	0.00	0.10	0.60	0.20	0.40	10.0	0.0	5.0
	30	0.20 (0.2)	0.35 (1.0)	0.28	0.40	0.60	0.50	15.3	8.2	11.8
	40	0.10 (0.1)	0.20 (0.6)	0.15	0.20	0.40	0.30	2.9	5.0	3.9
	50	0.55 (1.6)	0.80 (2.3)	0.68	1.00	1.20	1.10	23.3	15.2	19.3
Control (0 mg L^{-1})		0.70 (1.5)	0.75 (2.5)	0.73	1.10	0.80	0.95	38.0	11.2	24.6
200 mg L^{-1} (double)	10, 20	0.10 (0.1)	0.00	0.05	0.40	0.00	0.20	4.0	0.0	2.0
	20, 30	0.10 (0.1)	0.50 (1.3)	0.30	0.40	0.70	0.55	8.0	10.3	9.2
	30, 40	0.10 (0.1)	0.50 (1.6)	0.30	0.20	0.40	0.30	6.7	5.3	6.0
	40, 50	0.10 (0.1)	0.70 (2.2)	0.40	0.40	0.80	0.60	5.0	8.1	6.5
	50, 60	0.40 (1.2)	0.30 (0.3)	0.35	0.40	0.60	0.50	16.7	8.5	12.6
Control (0 mg L^{-1})		0.30 (0.7)	0.55 (1.6)	0.43	0.40	0.80	0.60	23.3	12.9	18.1
100 mg L^{-1} (double)	10, 20	0.20 (0.2)	0.30 (0.7)	0.25	0.40	0.80	0.60	14.0	6.9	10.4
	20, 30	0.30 (0.7)	0.30 (0.3)	0.30	0.40	1.00	0.70	26.7	10.6	18.7
	30, 40	0.10 (0.1)	0.10 (0.1)	0.10	0.40	0.20	0.30	10.0	1.8	5.9
	40, 50	0.00	0.25 (0.8)	0.13	0.20	0.40	0.30	0.0	6.7	3.3
	50, 60	0.60 (1.0)	0.30 (0.7)	0.45	1.00	0.60	0.80	32.5	6.2	19.3
Control (0 mg L^{-1})		0.60 (1.8)	0.60 (1.8)	0.60	0.60	0.70	0.65	23.0	11.9	17.5
Mean (first application date)	10	0.15	0.13	0.14 a	0.33	0.33	0.33 a	10.4	4.0	7.2 a
	20	0.20	0.27	0.23 a	0.47	0.63	0.55 ab	14.9	7.0	10.9 ab
	30	0.13	0.32	0.23 a	0.33	0.40	0.37 a	10.7	5.1	7.9 a
	40	0.07	0.38	0.23 a	0.27	0.53	0.40 a	2.6	6.6	4.6 a
	50	0.52	0.47	0.49 b	0.80	0.80	0.80 b	24.2	10.0	17.1 bc
Mean (controls)		0.53	0.63	0.58 b	0.70	0.77	0.73 b	28.1	12.0	20.0 c
	Mean (Var.)	0.27	0.37		0.48	0.58		15.1 a	7.4 b	

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

[b]Disease cover score (DCS; 0–6 scale; Wilson *et al*., 2009) assesses surface area of tubers covered with lesions. Percentage cover is the estimated percentage of tuber surface coverage derived from the DCS.

[c]Lesion depth score (LDS; 0–4 scale; Wilson *et al*., 2009) assesses mean of the lesion depth of deepest lesion per tuber.

[d]Proportion of diseased tubers measures the number of tubers with visible lesions as a proportion of total tubers per pot.

(87–100%), whereas severity was moderate (Table 4). There was a significant interaction found between the date of the first 2,4-D application, the frequency of application and between varieties on DCS and LDS, and between the date of the first 2,4-D application, the frequency of application and variety on proportion of diseased tubers (Table 4). Data trends again suggested that the earlier treatments result in lower disease. Early treatments reduced incidence of common scab in Russet Burbank only, and Desiree had significantly greater DCS and LDS than Russet Burbank (Table 4).

Agronomic effects

In pot trial No. 1 there was a significant effect found from the date of the first treatment on the total tuber mass per pot. Treatments at 20, 30 and 50 DAE gave significantly less tuber mass than the control. There were no other significant effects of treatment date, frequency, rate or variety, although all 2,4-D-treated plants showed a trend for lower yield (reduced total tuber mass and mean tuber mass). There were no trend effects on tuber number (Table 5). Pot trial No. 2 showed significant interaction between the first spray date and rate and frequency of application on the total tuber mass (Table 5). In the single application treatments, the control had a significantly higher total tuber mass than all treatments, except 50 DAE. In multiple application treatments, the control was only greater than the earliest treatment (10 DAE) at the highest rate (200 mg L^{-1}). There was no significant effect on mean tuber mass or on number of tubers per pot although tubers appeared slightly elongated from

Table 3 Field trial No. 1: The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single or double treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants on common scab incidence and severity. There was a significant interaction between date of first spray, number of sprays and variety on DCS ($P = 0.027$, standard error difference; SED = 0.23, LSD = 0.45) and LDS ($P = 0.024$, SED = 0.22, LSD = 0.44). Treatments with the same letter in the same column are not significantly different at $P = 0.05$ using Fisher's LSD test

		Disease Cover Score[b] (Percentage Cover %)			Lesion Depth Score[c]			Diseased Tubers/ Sample (%)[d]		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
100 mg L^{-1} (single)	10	1.57 (5.8) a	2.28 (12.2) ab	1.92	1.80 abc	2.49 abc	2.15	100	100	100
	20	1.66 (6.5) ab	2.44 (14.8) abc	2.05	2.12 bc	2.65 abcd	2.39	100	100	100
	30	2.08 (10.8) b	2.79 (19.3) cde	2.43	2.16 cd	3.03 def	2.60	100	100	100
	40	2.78 (18.4) cd	2.91 (20.8) de	2.84	2.95 efg	2.89 ef	2.92	100	100	100
	50	2.79 (19.5) cd	3.00 (23.1) e	2.89	2.79 ef	2.95 f	2.87	100	100	100
	60	2.95 (21.3) cd	3.03 (22.9) e	2.99	3.04 fg	3.00 f	3.02	100	100	100
100 mg L^{-1} (double)	10, 20	1.48 (5.4) a	2.14 (10.9) a	1.81	1.70 ab	2.52 a	2.11	100	100	100
	20, 30	1.68 (7.2) ab	2.51 (14.8) abcd	2.09	1.89 abc	2.78 abcde	2.33	100	100	100
	30, 40	2.55 (15.4) c	2.34 (13.5) ab	2.45	2.64 ef	2.82 abc	2.73	100	100	100
	40, 50	2.79 (19.0) cd	2.50 (16.3) abcd	2.64	2.60 de	2.68 abcde	2.64	100	100	100
	50, 60	2.77 (18.4) cd	2.66 (18.0) bcde	2.71	2.71 ef	2.92 cdef	2.83	100	100	100
100 mg L^{-1} (triple)	10, 20, 30	1.35 (4.6) a	2.25 (12.2) ab	1.80	1.58 a	2.73 abc	2.15	100	100	100
	20, 30, 40	1.78 (8.0) ab	2.21 (12.4) ab	1.99	2.01 bc	2.71 ab	2.36	100	100	100
	30, 40, 50	2.61 (16.7) c	2.61 (16.3) bcde	2.61	2.76 ef	2.68 bcdef	2.72	100	100	100
	40, 50, 60	3.21 (21.0) d	2.15 (12.3) a	2.68	2.94 efg	2.46 a	2.70	100	100	100
Control (0 mg L^{-1})		3.20 (25.2) d	3.58 (32.0) f	3.39	3.27 g	3.32 g	3.30	100	100	100
Mean (first application date)	10	1.47	2.22	1.84	1.69	2.58	2.14	100	100	100
	20	1.71	2.39	2.05	2.01	2.71	2.36	100	100	100
	30	2.41	2.58	2.50	2.52	2.84	2.68	100	100	100
	40	2.93	2.52	2.72	2.83	2.68	2.75	100	100	100
	50	2.78	2.83	2.80	2.75	2.93	2.84	100	100	100
	60	2.95	3.03	2.99	3.04	3.00	3.02	100	100	100
Control		3.20	3.58	3.39	3.27	3.32	3.30	100	100	100
	Mean (Var.)	2.33	2.59			2.43	2.79	100	100	100

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

[b]Disease cover score (DCS; 0–6 scale; Wilson *et al*. 2009) assesses surface area of tubers covered with lesions. Percentage cover is the estimated percentage of tuber surface coverage derived from the DCS.

[c]Lesion depth score (LDS; 0–4 scale; Wilson *et al*. 2009) assesses mean of the lesion depth of deepest lesion per tuber.

[d]Proportion of diseased tubers measures the number of tubers with visible lesions as a proportion of total tubers per pot.

plants receiving early applications of 2,4-D. In this trial, Desiree produced more and heavier tubers than Russet Burbank. Once again trends suggest that tuber mass increases with increasing delay to the first application date (Table 5).

In field trial No. 1 there was no significant effect of 2,4-D treatment on tuber mass or number. There were no obvious trends in the data set, suggesting that treatment had little effect on yields although tuber elongation was noted. Russet Burbank had significantly greater number of tubers than Desiree, but Desiree had a significantly higher mean mass per tuber than Russet Burbank (Table 6). In field trial No. 2 yields from the water-stressed plants were very poor. There was a significant interactive effect from spray rate, number of sprays and date of first spray on mean total mass; however, there is no trend apparent with controls not significantly different to any treatment. Russet Burbank had a significantly greater total tuber mass and number of tubers than Desiree, but Desiree had significantly higher mean mass per tuber than Russet Burbank (Table 7).

Toxin tolerance

Both pot trials No. 1 and No. 2 showed significant interactive effects of variety, timing of sprays and number of sprays on tuber toxin tolerance after harvest. Data trends suggest reduced necrotic response with the earliest applied 2,4-D treatment (Table 8). In field trial No. 2 there was a significant interactive effect found on toxin tolerance between date of the first application, application rate and variety. All 2,4-D treatments resulted in reduced thaxtomin A sensitivity. There were no obvious data trends for date, application rate or variety (Table 8).

Table 4 Field trial No. 2 The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single or double treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants, on common scab incidence and severity. There was a significant interaction between date of first spray, spray rate and number of sprays on DCS ($P = 0.007$, standard error difference; SED = 0.26, LSD = 0.51) and LDS ($P < 0.001$, SED = 0.20, LSD = 0.39), a significant interaction between date of first spray, spray rate, number of sprays and variety on proportion of diseased tubers ($P = 0.002$, SED = 0.04, LSD = 0.08) and a significant effect from variety on DCS ($P < 0.001$, SED = 0.07, LSD = 0.14) and LDS ($P < 0.001$, SED = 0.06, LSD = 0.11). Treatments with the same letter in the same column are not significantly different at $P = 0.05$ using Fisher's LSD test

		Disease Cover Score (Percentage Cover)[b]			Lesion Depth Score[c]			Diseased Tubers/ Sample (%)[d]		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
25 mg L^{-1} (single)	5	1.58 (7.5)	1.15 (3.6)	1.36 ab	1.02	1.06	1.04 a	87 a	94 ab	90
	10	1.00 (3.2)	1.70 (7.0)	1.35 a	1.09	1.06	1.07 a	96 ab	100 b	98
	20	1.19 (4.5)	2.00 (10.2)	1.60 abcde	1.04	1.29	1.17 abc	92 ab	100 b	96
	30	1.59 (7.5)	3.00 (21.4)	2.30 gh	1.29	2.25	1.77 ef	99 b	100 b	99
	40	1.82 (9.3)	2.45 (15.2)	2.14 fgh	1.38	1.64	1.51 cde	100 b	100 b	100
	50	1.20 (4.3)	1.86 (9.5)	1.48 abc	1.08	1.50	1.26 abcd	100 b	100 b	100
25 mg L^{-1} (double)	5, 10	1.56 (7.1)	2.70 (18.3)	2.13 fgh	1.36	1.80	1.58 de	100 b	100 b	100
	10, 20	1.47 (6.4)	2.58 (19.2)	2.02 defgh	1.13	1.80	1.47 cde	98 b	100 b	99
	20, 30	1.45 (6.2)	2.23 (11.6)	1.84 abcdefg	1.18	1.44	1.31 abcd	97 b	100 b	98
	30, 40	1.52 (6.1)	2.65 (16.7)	2.08 efgh	1.21	1.69	1.45 bcde	100 b	100 b	100
	40, 50	1.67 (7.7)	2.62 (16.0)	2.08 efgh	1.20	1.67	1.40 abcde	100 b	100 b	100
Control (0 mg L^{-1})		1.17 (4.2)	2.58 (16.1)	1.87 bcdefg	1.11	1.58	1.34 abcd	100 b	100 b	100
100 mg L^{-1} (single)	5	1.34 (5.4)	1.77 (8.6)	1.56 abcd	1.21	1.54	1.37 abcd	88 a	100 b	94
	10	0.82 (2.1)	2.67 (16.8)	1.74 abcdef	1.06	1.63	1.34 abcd	99 b	100 b	88
	20	1.50 (8.3)	2.15 (12.0)	1.82 abcdefg	1.15	1.29	1.22 abcd	100 b	100 b	100
	30	1.40 (5.6)	2.46 (16.7)	1.93 cdefgh	1.04	1.91	1.48 cde	100 b	100 b	98
	40	1.54 (6.4)	2.44 (13.1)	1.93 cdefgh	1.15	1.67	1.37 abcd	100 b	100 b	100
	50	1.22 (4.6)	2.67 (17.1)	1.94 cdefgh	1.10	1.67	1.38 abcd	100 b	100 b	100
100 mg L^{-1} (double)	5, 10	1.42 (7.0)	2.30 (12.6)	1.71 abcdef	1.27	2.05	1.53 cde	99 b	100 b	99
	10, 20	1.05 (4.1)	2.25 (12.4)	1.65 abcdef	1.17	1.68	1.43 abcde	100 b	100 b	99
	20, 30	1.05 (3.4)	1.94 (8.1)	1.50 abc	1.11	1.44	1.28 abcd	100 b	100 b	100
	30, 40	1.67 (8.7)	3.00 (23.0)	2.34 gh	1.23	1.93	1.58 de	100 b	100 b	100
	40, 50	1.70 (7.4)	3.33 (26.7)	2.40 h	1.32	2.83	1.97 f	100 b	100 b	100
Control (0 mg L^{-1})		1.67 (7.4)	2.62 (17.4)	2.15 fgh	1.22	1.42	1.32 abcd	100 b	100 b	100
Mean (first application date)	5	1.47	1.93	1.69	1.21	1.55	1.37	96	100	98
	10	1.08	2.30	1.69	1.11	1.54	1.33	98	100	99
	20	1.31	2.08	1.69	1.12	1.36	1.24	97	100	99
	30	1.56	2.80	2.18	1.20	1.95	1.57	100	100	100
	40	1.68	2.69	2.14	1.26	1.93	1.56	100	100	100
	50	1.21	2.32	1.73	1.09	1.60	1.33	100	100	100
Controls		1.42	2.60	2.01	1.16	1.50	1.33	100	100	100
	Mean (var)	1.40 a	2.38 b		1.17 a	1.64 b		97	100	

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

[b]Disease cover score (DCS; 0–6 scale; Wilson *et al*., 2009) assesses surface area of tubers covered with lesions. Percentage cover is the estimated percentage of tuber surface coverage derived from the DCS.

[c]Lesion depth score (LDS; 0–4 scale; Wilson *et al*., 2009) assesses mean of the lesion depth of deepest lesion per tuber.

[d]Proportion of diseased tubers measures the number of tubers with visible lesions as a proportion of total tubers per pot.

2,4-Dichlorophenoxyacetic acid quantification

In tubers from field trial No. 2 Russet Burbank had more 2,4-D in tubers at harvest than Desiree. In both varieties there was a strong trend towards increasing levels of 2,4-D in tubers at harvest with later application (Fig. 2a). For most application dates the 100 mg L^{-1} treatment had higher levels of 2,4-D in the tuber at harvest than the 25 mg L^{-1} treatment. Only one sample (Russet Burbank treated at 50 DAE with 100 mg L^{-1}) exceeded the Australian maximum residue level (MRL) of 100 ng g^{-1} of tuber FW (Commonwealth of Australia, 2011). With treatments applied at 20 DAE Desiree tubers had similar levels of 2,4-D at harvest for both application rates and both single and double applications. In contrast, Russet Burbank had similar levels to the Desiree samples for the 25 mg L^{-1} single treatment, but much greater levels for all other treatments, although no treatment exceeded the MRL. These data lack replication and as such have

Table 5 The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single or double treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants, on total tuber mass per pot, mean mass per tuber and number of tubers per pot in pot trials No. 1 and No. 2. In pot trial No. 1, there was a significant effect of the date of the first spray on total tuber mass ($P < 0.001$, standard error difference; SED = 2.36, LSD = 4.66). In pot trial No. 2, there was a significant effect of variety on total tuber mass per pot ($P < 0.033$, SED = 1.55, LSD = 3.06), mean mass per tuber ($P < 0.001$, SED = 0.72, LSD = 1.43) and number of tubers ($P < 0.001$, SED = 0.30, LSD = 0.59), and a significant interaction between date of first spray and number of sprays on total tuber mass per pot ($P = 0.013$, SED = 4.65, LSD = 9.19). Treatments with the same letter in the same column are not significantly different at $P = 0.05$ using Fisher's LSD test

		Pot trial No. 1									Pot trial No. 2								
		Total Tuber Mass/ Pot (g)			Mean Tuber Mass (g)			Number of Tubers/Pot			Total Tuber Mass/Pot (g)			Mean Tuber Mass (g)			Number of Tubers/Pot		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
200 mg L^{-1} (single)	10	25.6	28.2	36.0	9.0	7.2	8.1	5.2	5.2	5.2	23.5	30.0	26.7 a	6.9	7.6	7.2	3.8	5.8	4.8
	20	27.7	23.1	23.4	4.7	5.6	5.2	6.8	4.0	5.4	34.1	40.7	37.4 bcd	9.6	7.3	8.5	4.2	6.6	5.4
	30	22.9	21.2	27.7	7.0	5.7	6.4	5.0	4.8	4.9	29.5	30.7	30.1 ab	5.6	5.6	5.6	5.6	6.0	5.8
	40	33.7	30.2	28.1	4.8	6.2	5.5	4.6	7.2	5.9	36.8	29.4	33.1 abc	9.1	5.9	7.5	4.2	5.4	4.8
	50	21.2	28.1	25.2	6.5	5.4	6.0	4.2	5.8	5.0	41.3	47.7	44.5 de	13.2	7.3	10.3	4.0	6.8	5.4
Control (0 mg L^{-1})		32.0	36.7	33.2	7.0	9.2	8.1	4.6	5.4	5.0	44.8	55.1	50.0 e	12.7	8.4	10.5	3.8	7.2	5.5
200 mg L^{-1} (double)	10, 20	28.4	19.2	23.8	6.1	3.9	5.0	6.2	5.6	5.9	25.2	28.8	27.0 a	7.5	3.7	5.6	3.6	7.2	5.4
	20, 30	20.6	23.5	22.1	5.6	5.3	5.5	4.4	5.2	4.8	29.2	29.0	29.1 ab	12.4	6.6	9.5	3.2	6.0	4.6
	30, 40	18.7	19.2	19.0	5.0	4.1	4.6	4.0	5.0	4.5	41.6	39.4	40.5 cd	14.9	6.5	10.7	3.2	7.6	5.4
	40, 50	30.0	29.3	29.6	5.8	8.3	7.0	6.0	4.0	5.0	28.3	35.8	32.1 abc	11.0	5.6	8.3	3.4	6.6	5.0
	50, 60	22.4	24.5	23.4	5.4	4.9	5.2	4.8	5.4	5.1	33.5	41.5	37.5 bcd	12.4	6.4	9.4	3.0	7.8	5.4
Control (0 mg L^{-1})		27.4	37.7	32.6	3.5	7.3	5.4	8.2	5.4	6.8	38.7	33.6	36.2 bcd	15.9	8.1	12.0	2.8	6.0	4.4
100 mg L^{-1} (double)	10, 20	38.5	33.6	26.9	4.2	5.0	4.6	6.4	6.2	6.3	29.3	39.1	34.2 abc	10.0	6.6	8.3	3.4	5.8	4.6
	20, 30	25.3	21.5	25.4	5.7	3.7	4.7	5.6	6.6	6.1	32.2	30.9	31.6 abc	10.8	5.6	8.2	3.8	6.4	5.1
	30, 40	27.5	27.9	22.0	5.0	8.7	6.8	5.0	4.8	4.9	36.9	38.7	37.8 bcd	14.2	6.8	10.5	3.0	6.8	4.9
	40, 50	20.8	35.4	32.0	8.9	6.4	7.6	4.4	4.8	4.6	33.0	39.9	36.5 bcd	12.4	6.9	9.6	3.6	5.8	4.7
	50, 60	27.2	23.2	24.6	5.8	5.4	5.6	4.6	6.0	5.3	39.1	36.4	37.7 bcd	10.2	5.3	7.7	4.4	7.2	5.8
Control (0 mg L^{-1})		29.0	37.3	34.3	7.2	8.9	8.0	4.8	5.4	5.1	32.5	42.8	37.6 bcd	9.6	6.4	8.0	3.8	7.4	5.6
Mean (first DAE)	10	30.8	27.0	28.9 bc	6.4	5.4	5.9	5.9	5.7	5.8	26.0	32.6	29.3	8.1	6.0	7.0	3.6	6.3	4.9
	20	24.5	22.7	23.6 a	5.4	4.9	5.1	5.6	5.3	5.4	31.8	33.5	32.7	10.9	6.5	8.7	3.7	6.3	5.0
	30	23.0	22.8	22.9 a	5.7	6.2	5.9	4.7	4.9	4.8	36.0	36.3	36.1	11.6	6.3	8.9	3.9	6.8	5.4
	40	28.2	31.6	29.9 c	6.5	7.0	6.7	5.0	5.3	5.2	32.7	35.1	33.9	10.9	6.1	8.5	3.7	5.9	4.8
	50	23.6	25.3	24.4 ab	5.9	5.2	5.6	4.5	5.7	5.1	38.0	41.8	39.9	11.9	6.3	9.1	3.8	7.3	5.5
Mean (controls)		29.5	37.2	33.4 c	5.9	8.5	7.2	5.9	5.4	5.6	38.7	43.9	41.3	12.7	7.6	10.2	3.5	6.9	5.2
	Mean (Var.)	26.6	27.8		6.0	6.2		5.3	5.4		33.9 a	6.5 b		3.7a	6.6b				

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

Table 6 The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single, double or triple treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants, on total tuber mass per plot, mean mass per tuber and number of tubers per plot in field trial No. 1. There was a significant effect found from variety on mean tuber mass ($P < 0.001$, standard error difference; SED = 4.92, LSD = 9.76) and number of tubers ($P < 0.001$, SED = 1.68, LSD = 3.35). Treatments with the same letter in the same row and factor are not significantly different at $P = 0.05$ using Fisher's LSD test

		Mean Total Mass (g)			Mean Mass/Tuber (g)			Number of Tubers		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
100 mg L^{-1} (single)	10	3134	4152	3643	93.7	145.0	119.3	33.5	28.5	31.0
	20	3419	3148	3283	107.5	172.7	140.1	33.3	18.8	26.0
	30	3854	3131	3492	111.7	166.0	138.8	35.3	19.0	27.1
	40	2956	2694	2825	91.0	135.9	113.4	32.3	18.5	25.3
	50	3131	3506	3319	125.3	175.2	150.3	25.5	20.3	22.9
	60	3192	3718	3455	102.7	176.5	139.6	30.0	21.3	25.6
100 mg L^{-1} (double)	10	3161	3250	3206	70.4	153.8	112.1	44.8	20.8	32.8
	20	3285	3656	3471	97.0	148.0	122.5	34.0	25.3	29.6
	30	3675	2508	3091	112.7	159.1	135.9	34.0	15.5	24.8
	40	4081	3734	3908	115.1	178.0	146.5	35.3	21.5	28.8
	50	3312	2990	3151	111.4	156.6	134.0	33.3	20.0	26.6
100 mg L^{-1} (triple)	10	3029	3902	3466	70.7	144.2	107.4	42.8	27.3	35.0
	20	3775	3841	3808	104.9	163.1	134.0	35.8	23.5	29.6
	30	2610	2952	2781	107.1	158.6	132.8	24.0	19.0	21.5
	40	2891	3188	3039	80.6	170.0	125.3	35.8	18.8	27.3
Control (0 mg L^{-1})		2332	3761	3047	111.1	198.5	154.8	22.8	19.5	21.1
Mean (first DAE)	10	3108	3768	3438	78.2	147.6	112.9	40.3	25.5	32.9
	20	3493	3548	3521	103.1	161.3	132.2	34.3	22.5	28.4
	30	3380	2864	3122	110.5	161.2	135.8	31.1	17.8	24.5
	40	3310	3205	3257	95.6	161.3	128.4	34.4	19.6	27.0
	50	3222	3248	3235	118.3	165.9	142.1	29.4	20.1	24.8
	60	3192	3718	3455	102.7	176.5	139.6	30.0	21.3	25.6
Mean (controls)		2332	3761	3047	111.1	198.5	154.8	22.8	19.5	21.1
	Mean (Var.)	3240	3383		100.8 a	162.6 b		33.3 a	21.1 b	

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

only been analysed statistically for varietal differences (Fig. 2).

Discussion

A discrete window of susceptibility of potato tubers to common scab occurs during the rapid growth phase of tuber development. Tuber internodes are susceptible to infection by *S. scabies* after lenticels have been formed from stomata but have not yet completely suberised (Adams, 1975). Lapwood & Adams (1973) determined that lenticels in the third and fourth internode from the apical bud enter this susceptible phase that lasts approximately 10 days under normal tuber growth rate (Adams & Lapwood, 1978). More recent research has found that earlier formed lenticels in the first and second internode are more susceptible to infection than those formed later, and 68% of tubers inoculated 14 days after initiation become infected, whereas only 4% of tubers succumbed if inoculated 8 weeks after initiation (Khatri *et al.*, 2011). This suggested that tubers will be most susceptible at the beginning of the infection window.

In this study, the greatest disease suppression was generally associated with the most early application date at 5 or 10 DAE, much earlier than any tested previously (McIntosh *et al.*, 1981; Tegg *et al.*, 2008). We suggest that treatments timed to the estimated commencement of the infection window may inadvertently miss the start of this period for all tubers. Potatoes produce tubers asynchronously (Vreugdenhil & Struik, 1989); thus, the time of both tuber initiation and commencement of the disease infection window varies for each tuber. Moreover, first tuber initiation is influenced by emergence date more so than planting date (Struik *et al.*, 1999), as well as cultivar genetics (Celis-Gamboa *et al.*, 2003) and environmental factors such as day length. As such, the infection window of a crop may extend for a significant period of time, and the onset for the first tubers formed may be difficult to predict. This is exacerbated by the difficulty in monitoring tuber initiation under the soil particularly as tuber initiation does not correspond to plant development above ground (Celis-Gamboa *et al.*, 2003). McIntosh *et al.* (1982) recognised that the greatly diminished disease

Table 7 The effect of 2,4-dichlorophenoxyacetic acid (2,4-D) applied in single, double or triple treatments at various times to the foliage of Russet Burbank (RB) and Desiree (DE) plants, on total tuber mass per plot, mean mass per tuber and number of tubers per plot in field trial No. 2. There was a significant effect found from variety on total tuber mass ($P < 0.001$, standard error difference; SED = 48.7, LSD = 96.4) and mean tuber mass ($P < 0.001$, SED = 2.68, LSD = 5.31). There was a significant effect found from variety $P < 0.001$, SED = 0.81, LSD = 1.60) and date of first spray ($P < 0.001$, SED = 1.79, LSD = 3.53) on tuber number. There was a significant interaction between spray rate, number of sprays and date of first spray on total tuber mass ($P = 0.027$, SED = 175.6, LSD = 347.4). Treatments with the same letter in the same row and factor are not significantly different at $P = 0.05$ using Fisher's LSD test

		Mean Total Mass (g)			Mean Mass/Tuber (g)			Number of Tubers		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	RB	DE	Mean
25 mg L^{-1} (single)	5	151.2	75.0	113.1 a	13.7	23.8	18.7	8.3	3.0	5.6
	10	291.2	178.8	235.0 abc	26.8	23.4	25.1	10.8	7.8	9.3
	20	327.5	70.0	198.8 ab	26.4	32.5	29.5	10.5	2.0	6.3
	30	455.0	300.0	377.5 abcdefg	26.3	52.3	39.3	13.5	5.5	9.5
	40	632.5	292.5	462.5 bcdefg	29.4	27.2	28.3	16.8	7.5	12.1
	50	247.5	163.3	211.4 abc	24.0	42.8	32.1	8.0	3.0	5.5
25 mg L^{-1} (double)	5, 10	677.5	497.5	587.5 defg	33.7	44.2	39.0	14.5	11.5	13.0
	10, 20	737.5	252.5	495.0 bcdefg	44.3	37.5	40.9	13.8	6.0	9.9
	20, 30	320.0	460.0	390.0 abcdefg	24.7	26.4	25.6	8.5	7.8	8.1
	30, 40	822.5	442.5	632.5 fg	38.6	53.0	45.8	15.0	5.8	10.4
	40, 50	358.8	110.0	252.1 abcde	29.6	24.1	27.2	11.3	4.0	7.6
Control (0 mg L^{-1})		327.5	311.2	319.4 abcdef	31.6	30.8	31.2	8.3	6.5	7.4
100 mg L^{-1} (single)	5	656.2	536.2	596.3 efg	37.1	38.7	37.9	15.0	10.5	12.8
	10	308.8	178.8	243.8 abcd	20.8	35.6	28.2	13.5	4.8	9.1
	20	586.2	237.5	411.9 abcdefg	41.5	27.8	34.7	12.0	5.8	8.9
	30	716.7	713.3	715.0 g	34.7	38.2	36.5	12.5	12.5	12.5
	40	408.3	91.7	250.0 abcde	33.5	29.4	31.5	10.0	2.8	6.4
	50	280.0	311.2	295.6 abcdef	37.7	74.2	55.9	6.8	4.0	5.4
100 mg L^{-1} (double)	5, 10	672.5	317.5	554.2 cdefg	27.5	50.1	35.0	20.3	3.3	11.8
	10, 20	465.0	328.8	396.9 abcdefg	29.7	32.4	31.1	15.5	10.0	12.8
	20, 30	271.7	91.7	181.7 ab	26.2	21.4	23.8	7.0	2.5	4.8
	30, 40	625.0	761.2	693.1 g	29.2	41.0	35.1	16.0	12.5	14.3
	40, 50	626.2	140.0	417.9 abcdefg	40.3	72.5	54.1	12.3	1.3	6.8
Control (0 mg L^{-1})		432.5	206.2	319.4 abcdef	28.5	36.2	32.4	12.5	6.0	9.3
Mean (first DAE)	5	539.4	362.1	456.7	28.0	37.7	32.5	14.5	7.1	10.8 c
	10	450.6	234.7	342.7	30.4	32.2	31.3	13.4	7.1	10.3 bc
	20	387.9	206.1	297.0	30.3	27.5	28.9	9.5	4.5	7.0 ab
	30	650.7	543.7	597.2	32.0	46.7	39.4	14.3	9.1	11.7 c
	40	513.0	168.8	353.2	33.2	37.4	35.2	12.6	3.9	8.2 abc
	50	263.8	247.9	256.3	30.8	60.7	44.8	7.4	3.5	5.4 a
Mean (controls)		380.0	258.8	319.4	30.1	33.5	31.8	10.4	6.3	8.3 abc
	Mean (Var.)	476.9 a	297.3 b		30.7 a	38.0 b		12.2 a	6.1 b	

DAE, days after emergence.

[a]Date (DAE) is the number of days after the average emergence date.

suppression of 3,5-D applications targeted to the estimated tuber initiation period in the field compared with glasshouse trials might be the result of extended tuber initiation in field conditions. They also questioned the persistence of the material in the tubers; however, our studies with 2,4-D suggest that single early applications provide sufficient material for protection throughout the season.

While the mechanism by which 2,4-D increases resistance in potato to common scab is yet to be directly proven, a clear relationship between 2,4-D (and other suppressive compounds) concentration and diminished thaxtomin A toxicity in plant tissues has been demonstrated (Tegg *et al.*, 2005, 2008, 2012). This relationship has repeatedly been shown in a wide range of plant species and specific tissues. Further, Tegg *et al.* (2013) have shown relationships between thaxtomin A sensitivity and auxin cellular efflux transport processes. However, we cannot rule out that 2,4-D treatments may also activate additional defence responses in the plant. Systemic acquired resistance (SAR) responses providing protection against pathogen attack are well known following various chemical treatments of plants. For example, prohexadione-calcium treatment provides a SAR response in pears and apples against fire blight,

Table 8 Mean tuber slice necrosis response following application of thaxtomin A in tubers harvested from plants of Russet Burbank (RB) and Desiree (DE) treated with different 2,4-dichlorophenoxyacetic acid (2,4-D) foliar treatments. Necrosis was rated using the scale described in Fig. 1. There was a significant interaction between first spray date, number of sprays and variety in pot trial No. 1 ($P < 0.001$, standard error difference; SED = 0.127, LSD = 0.250) and pot trial No. 2 ($P < 0.001$, SED = 0.107, LSD = 0.210) and between spray rate, first spray date and variety in field trial No. 2 ($P = 0.045$, SED = 0.120, LSD = 0.235). Treatments with the same letter in the same column are not significantly different at $P = 0.05$ using Fisher's LSD test

		Pot Trial No. 1			Pot Trial No. 2					Field trial No. 2		
2,4-D Rate (Frequency)	Date[a] (DAE)	RB	DE	Mean	RB	DE	Mean	2,4-D rate (frequency)	Date (DAE)	RB	DE	Mean
200 mg L^{-1} (single)	10	2.04 cd	1.63 a	1.83	2.22 j	1.43 abc	1.82	25 mg L^{-1} (single)	5	1.90	1.65	1.78
	20	2.06 cd	1.88 bcde	1.97	1.67 ef	1.59 bcdef	1.63		10	1.45	1.71	1.58
	30	1.73 ab	1.85 abcd	1.79	1.19 a	1.69 defg	1.44		20	1.66	1.44	1.55
	40	2.65 g	2.08 def	2.37	2.00 hi	1.26 a	1.63		30	1.37	1.48	1.42
	50	2.42 fg	1.77 abc	2.09	1.79 fgh	1.52 bcd	1.66		40	1.63	1.48	1.56
Control (0 mg L^{-1})		1.98 cd	2.06 def	2.02	2.17 ij	1.69 defg	1.93		50	1.54	1.30	1.42
								100 mg L^{-1} (single)	5	1.24	1.44	1.34
200 mg L^{-1} (double)	10, 20	1.69 ab	1.71 abc	1.70	1.59 def	1.39 ab	1.49		10	1.56	1.63	1.60
	20, 30	2.35 ef	1.88 bcde	2.12	1.54 cde	1.87 g	1.70		20	1.53	1.81	1.67
	30, 40	2.06 cd	1.88 bcde	1.97	1.45 bcd	1.90 g	1.67		30	1.33	1.15	1.24
	40, 50	1.52 a	1.81 abc	1.67	1.25 ab	1.63 cdef	1.44		40	1.29	1.56	1.42
	50, 60	2.06 cd	2.06 def	2.06	1.48 gh	1.57 bcde	1.76		50	1.31	1.22	1.26
Control (0 mg L^{-1})		2.17 de	2.25 f	2.21	1.93 gh	1.85 g	1.89	Control (0 mg L^{-1})		2.01	1.85	1.93
								25 mg L^{-1} (double)	5, 10	1.48	1.340	1.41
100 mg L^{-1} (double)	10, 20	1.88 bc	1.67 ab	1.77	1.69 ef	1.76 efg	1.72		10, 20	1.51	1.715	1.62
	20, 30	2.17 de	1.73 abc	1.95	1.92 gh	1.71 defg	1.81		20, 30	1.79	1.583	1.69
	30, 40	2.52 fg	1.92 cde	2.22	1.35 abc	1.60 cdef	1.48		30, 40	1.40	1.347	1.37
	40, 50	1.98 cd	1.94 cde	1.96	1.73 efg	1.80 fg	1.77		40, 50	1.47	1.458	1.47
	50, 60	2.04 cd	1.77 abc	1.91	1.52 cde	1.64 cdef	1.58	100 mg L^{-1} (double)	5, 10	1.35	1.177	1.26
Control (0 mg L^{-1})		1.98 cd	2.10 ef	2.04	2.21 ij	1.71 defg	1.96		10, 20	1.52	1.625	1.57
									20, 30	1.493	1.469	1.48
Mean (first DAE)	10	1.87	1.67	1.77	1.83	1.52	1.68		30, 40	1.46	1.201	1.33
	20	2.19	1.83	2.01	1.71	1.72	1.72		40, 50	1.26	1.229	1.25
	30	2.10	1.88	1.99	1.33	1.73	1.53	Control (0 mg L^{-1})		1.85	1.71	1.78
	40	2.05	1.94	2.00	1.66	1.56	1.61	Mean (25 mg L^{-1})	5	1.69 b	1.46 cde	1.58
	50	2.17	1.87	2.02	1.75	1.58	1.66		10	1.48 bcde	1.71 ab	1.60
Mean (controls)		2.04	2.14	2.09	2.101	1.75	1.93		20	1.71 ab	1.51 bcde	1.61
									30	1.38 cde	1.41 def	1.40
									40	1.55 bc	1.47 cde	1.51
									50	1.54 bc	1.30 ef	1.42
								Mean (100 mg L^{-1})	5	1.29 de	1.34 ef	1.32
									10	1.54 bc	1.63 abcd	1.95
									20	1.51 bcd	1.67 abc	1.59
									30	1.40 cde	1.18 f	1.29
									40	1.27 e	1.36 ef	1.32
									50	1.31 de	1.22 f	1.26
								Mean (controls)		1.93 a	1.78 a	1.86

DAE, days after emergence.
[a]Date (DAE) is the number of days after the average emergence date.

fosetyl-Al against late blight of potato and benzothiadiazole and acetylsalicylic acid against early blight, powdery mildew and Fusarium dry rot of potato, with treatments generally only effective when the treatments are applied earlier in the growing season or prior to symptom expression (Cooke & Little, 2001; Schupp *et al.*, 2002; Bokshi *et al.*, 2003; Andreu *et al.*, 2006). Another possible influence may be through the alleviation of abiotic stresses as has been reported for the osmoprotectant glycine betaine which following foliar treatments slightly reduced common scab severity (Tuomola *et al.*, 1996).

The earliest spray treatments were included in this study to determine if sprays applied prior to tuber initiation would be effective in controlling disease by ensuring that the entire infection window was completely covered. It was expected that treating plants even with low rates of a herbicide very early on in their development would be detrimental to the plant, and it was not known whether the 2,4-D would still be translocated to the tubers if it had been applied before they had developed. Our results show that the 2,4-D applied soon after plant emergence (between 5 and 10 DAE) is retained within

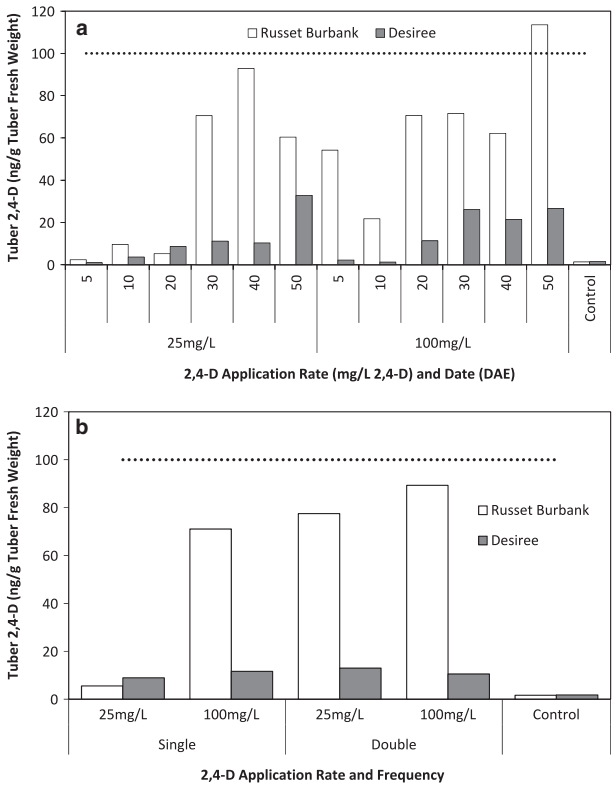

Figure 2 Concentration of 2,4-dichlorophenoxyacetic acid (2,4-D) in harvest tubers from plants treated with 2,4-D foliar sprays applied at various times and rates (Field trial No. 2). (a) Effect of date of application and rate. (b) Effect of frequency of application and rate. Dotted line indicates the maximum residue limit (MRL) for 2,4-D in potatoes at harvest.

the plant until tuber initiation, translocated to the tubers once they have initiated and gives good disease control in comparison to sprays applied later (after tuber initiation has already occurred). Quantification of 2,4-D within tubers at harvest shows that plants treated with 2,4-D prior to tuber initiation had small amounts of 2,4-D within the tubers at harvest. This confirms that the 2,4-D is translocated to the tubers after they have initiated, and that sufficient 2,4-D stays within the tuber to provide control against disease through the infection window.

As well as ensuring active material is present in the plant prior to tuber initiation and in doing so providing improved disease control, treatments prior to tuber initiation allow for a longer period in which the 2,4-D can be metabolised within the plant, resulting in less 2,4-D within tubers at harvest when it is not needed and unwanted. The current Australian MRL for 2,4-D in potatoes is 100 ng g^{-1} of FW tubers. Quantification of 2,4-D in tubers from field trial No. 2 at harvest suggests that when treated early with 2,4-D at the rates used, tubers would have a level of 2,4-D considered safe for human consumption, reaching levels of around 1/10 of the MRL. However, treatments applied later in the growing period, after tuber initiation, result in some tubers having 2,4-D levels near the MRL at harvest. This agrees with previous research that had shown that treatments of 2,4-D applied at higher rates than those used in this study targeted to the infection window resulted in levels of 2,4-D in tubers at harvest near or above the MRL (Tegg *et al*., 2008).

This study suggests that single applications are as equally effective in controlling disease as double or triple spray treatments. This is in contrast with previous work which found that multiple sprays resulted in lower disease severity than single sprays (Tegg *et al*., 2008). Chemically induced resistances can require multiple applications throughout the growing season to achieve disease control. The resistance of potato to late blight induced by foliar applied phosphorous acid was found to be three times more effective from double and triple applications applied at 14-day intervals than single applications (Taylor *et al*., 2011). However, induced control can be achieved with a single targeted application, as has been demonstrated with benzothiadiazole that induces resistance against post-harvest melon diseases and achieves similar levels of disease control as four applications made throughout the growing season (Bokshi *et al*., 2006). That single applications early in the growing season resulted in excellent disease control and low levels of 2,4-D in the tuber at harvest further suggests that one targeted application provided enough material to the developing tubers to effectively control common scab, and that multiple sprays spread across the growing season are not required. Additional sprays would likely be applied after tubers are susceptible, and this research has suggested that they would be less effective.

The 2,4-D treatments in this study were not shown to have a significant effect on the mean number of tubers or mean mass of tubers in any trials. There was an effect found from treatments on total tuber mass in one field trial and both pot trials; however, the effect was not consistent between trials, with treatments resulting in both higher and lower total tuber masses when compared with controls across the trials. In both pot trials there was a trend towards earlier sprays having a lower total tuber mass, and treatments applied around tuber initiation in pot trial No. 2 resulted in the lowest tuber masses. This result is similar to that of McIntosh *et al*. (1981), who observed that treatments applied at 21 DAE resulted in increased phytotoxic effects. Treatments prior to tuber initiation were not included in the trials of McIntosh *et al*. (1981). Tegg *et al*. (2008) also observed reduced total tuber weight in pot trials from treatments applied at a similar date. It may be that 2,4-D applications targeted towards the beginning of the infection window, when

tubers are initiating, has a greater effect on the total tuber mass of a plant than treatments applied prior to tuber initiation, or after tubers have finished rapidly growing, possibly as a result of changes in the hormone balance (Struik *et al*., 1999). In interpretation of these results we need to acknowledge that the main aim of this study was to determine the efficacy of 2,4-D as a disease control tool, and these trials were conducted to maximise disease levels with plants grown under continuous water stress. As a result, some agronomic outcomes, such as tuber size, tuber number and yield, are not indicative of what would occur in a commercial crop grown to maximise yield and productivity. Pot trials can also be poor indicators of agronomic performance in potato crops because of restrictions on root and tuber development, and care must be taken in the interpretation of these data. To determine more accurately the agronomic effects of these treatments, further trials would need to be undertaken using commercial growing practices, with standard irrigation and other management practices in place.

Both previous research (Tegg *et al*., 2008) and the results presented here suggest that the threshold of 2,4-D required for suppression of thaxtomin A is relatively low and that elevated levels do not necessarily give greater disease control. This is important as it allows application strategies that will ensure that residual levels should always remain below the recommended MRL. This recognises that 2,4-D may be naturally metabolised within the tuber (Burrell, 1982) and the level of 2,4-D in tubers at harvest will not necessarily reflect the levels present during the period of susceptibility. Therefore, the tuber slice assay and harvest 2,4-D levels need to be interpreted with rate and timing of the treatment, and date of harvest in mind.

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