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RESEARCH ARTICLE

Susceptibility of eight potato cultivars to tuber infection by *Phytophthora erythroseptica* and *Pythium ultimum* and its relationship to mefenoxam-mediated control of pink rot and leak

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Abstract

In addition to cultural practices, the application of the fungicide mefenoxam is an important disease management tactic used to control both pink rot and leak on potato tubers grown in the USA. Mefenoxam resistance has been identified in many of the potato growing regions, and therefore resistance management strategies are very important for retaining this fungicide as a tool to manage these storage rot diseases. The relationship between mefenoxam efficacy and cultivar susceptibility to pink rot and leak was assessed in post-harvest inoculation studies. Mefenoxam was applied to potato (*Solanum tuberosum*) cultivars known to express varying levels of susceptibility to pink rot and leak caused by *Phytophthora erythroseptica* and *Pythium ultimum*, respectfully. Tubers harvested from plants treated with in-furrow and foliar applications of mefenoxam were inoculated with isolates sensitive to the fungicide. Incidence and severity of both diseases ranged widely among cultivars. Russet Norkotah was the most susceptible to infection by *P. erythroseptica*, while cvs Pike and Atlantic were the most resistant. Cultivars Dark Red Norland, Russet Norkotah, Goldrush and Russet Burbank were most susceptible to infection by *P. ultimum* whereas Snowden was most resistant. Control of pink rot differed significantly among cultivars following mefenoxam treatment, ranging from 28% (cv. Goldrush) to 67% (cv. Snowden) and generally provided the greatest level of disease control on susceptible and moderately susceptible cultivars such as Russet Norkotah and Snowden, respectively. In contrast, the impact of mefenoxam on leak development was minimal and disease control did not differ significantly among the cultivars. The fungicide failed to control leak in the susceptible cvs Atlantic and Pike and control ranged from 1.7% to 5.2% in cvs Goldrush, Russet Norkotah, Dark Red Norland, Russet Burbank and Kennebec. The greatest level of leak control was achieved with the moderately resistant cv., Snowden, at 12.7%. Cultivars most likely to benefit from mefenoxam treatments should be targeted as part of a pink rot management programme. Judicious use of the fungicide, when matched with the level of cultivar susceptibility, may prove to be an efficient and effective approach to reduce infection rates and possibly manage mefenoxam resistance thereby maintaining longevity of the compound.

Introduction

Potato (*Solanum tuberosum* L.) is a cool climate crop ranking only behind wheat, maize and rice in importance as a world food staple (Thurston, 2001). Like other root-bearing and tuber-bearing species, many of the most important potato diseases affect the below ground starch storage organ. Two of these, soil-borne diseases pink rot

and leak, can cause severe losses in the field prior to harvest and after tubers are placed in storage facilities (Blodgett, 1945; Powelson *et al.*, 1993; Secor & Gudmestad, 1999; Lambert & Salas, 2001; Salas & Secor, 2001). Both diseases may become particularly serious in low-lying areas of fields, locations with poorly drained soils as well as during periods of heavy rainfall. Pink rot and leak often are collectively referred to as 'water rot' diseases because of the watery breakdown of infected tuber tissue. The diseases are, however, differentiated by unique symptomatology.

The oomycete *Phytophthora erythroseptica* Pethyb. is the primary cause of pink rot. The pathogen infects potato tubers through stolons, eyes or lenticels via zoospores and through cuts, cracks or other wounds made during harvest and handling operations (Pethybridge, 1913; Lonsdale *et al.*, 1980; Secor & Gudmestad, 1999; Salas *et al.*, 2000; Lambert & Salas, 2001; Taylor *et al.*, 2004). In contrast, the oomycete *Pythium ultimum* Trow, the principal leak pathogen (Jones, 1935; Blodgett & Ray, 1945), cannot penetrate into the undamaged periderm tissue, therefore infection occurs exclusively through abrasions, cuts or other wounds (Hawkins & Harvey, 1919; Salas & Secor, 2001; Taylor *et al.*, 2004). *P. erythroseptica* and *P. ultimum* are widely distributed throughout potato growing regions of the world. They are endemic to most soils and are able to survive for many years as oospores in the soil and in the infected plant debris. Both pathogens are capable of infecting many other solanaceous as well as non-solanaceous species (Lambert & Salas, 2001; Salas & Secor, 2001). These factors make effective control of water rots difficult to achieve.

Strategies used to manage water rots generally target conditions that favour infection and disease development. This approach commonly involves implementing agronomic practices, such as crop rotation, planting in well-drained soils and avoiding excessive late season irrigation. Any action that promotes periderm development, such as allowing sufficient time between vine killing and harvest, or limits wounding, also is beneficial (Powelson *et al.*, 1993; Secor & Gudmestad, 1999; Lambert & Salas, 2001; Salas & Secor, 2001). Although growers can generally manage pink rot and leak successfully by implementing such techniques, field applications of mefenoxam (Ridomil Gold EC and Ultra Flourish EC) continue to be the most widely used means of controlling these diseases under high disease pressure conditions (Wicks *et al.*, 2000; Peters *et al.*, 2001, 2003*a*; Platt *et al.*, 2003*a*,*b*, 2004; Taylor *et al.*, 2004). Resistance to mefenoxam has been detected in the *P. erythroseptica* and *P. ultimum* populations across North America (Sanders, 1984; Lambert & Salas, 1994; Goodwin & McGrath, 1995; Taylor *et al.*, 2002*a*,*b*; Hamm *et al.*, 2004; Moorman & Kim, 2004); however, mefenoxam remains the most reliable method of controlling pink rot and, to a certain extent, leak in areas where the pathogen populations predominantly remain sensitive to the fungicide.

The efficacy of mefenoxam against the water rots is probably one reason why traditional breeding programmes have not directed efforts towards developing resistance to these diseases. As a result, genetic resistance has not played a significant role in any integrated approach to manage pink rot and leak. Limited information is available from earlier studies that attempted to quantify resistance to pink rot (Cairns & Muskett, 1939; Goss, 1949; Jones, 1945; Fernandez-Northcote *et al.*, 1972; Lennard, 1980; Stack *et al.*, 1994) and leak (Hawkins, 1916; Jones, 1945; Priou *et al.*, 1997) in potato genotypes. These studies demonstrated that cultivars vary greatly in susceptibility to *P. erythroseptica* and *P. ultimum* and, although some are less severely affected, most cultivars commonly grown in North America are susceptible to one or both of these pathogens. Although limited studies assessing pink rot resistance of currently grown genotypes have been conducted (Peters & Sturz, 2001; Peters *et al.*, 2004), only recently has a more concerted effort been made to undertake large scale evaluations of potato cultivars for resistance to the two pathogens (Salas *et al.*, 2003).

Disease aetiology (Taylor *et al.*, 2004), and the level of mefenoxam resistance present in the pathogen population (Taylor *et al.*, 2006), impact efficacy of the fungicide. Cultivar resistance may also have an impact upon these diseases, but it is not known whether the extent of mefenoxam efficacy is influenced by the level of susceptibility of a particular genotype. Observations made in earlier work with cvs Russet Burbank, Shepody and Yukon Gold suggested that control of pink rot or leak by mefenoxam may be cultivar specific and related to the local growing conditions; however, the results of these single-year studies were inconsistent (Platt *et al.*, 2003*a*,*b*, 2004). A similar 3-year study found that mefenoxam applications provided greater suppression of pink rot in Kennebec than in Russet Burbank (Peters *et al.*, 2003*a*), but only these two cultivars were evaluated.

Mefenoxam is largely used indiscriminately by potato producers without regards to cultivar susceptibility. Because of the development of resistance to mefenoxam in *P. erythroseptica* and *P. ultimum*, it would be beneficial to limit its use to those cultivars in which treatment is necessary to adequately manage pink rot and leak to reduce exposure of the pathogen to unnecessary applications of the fungicide. Our hypothesis was that mefenoxam would provide little or no added benefit in the control of pink rot and leak in resistant or moderately resistant cultivars. The current research was undertaken

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to further examine the relationship between cultivar resistance and mefenoxam efficacy related to disease development and ultimately, disease management. Experiments were conducted to determine the effectiveness of mefenoxam against mefenoxam-sensitive isolates of *P. erythroseptica* and *P. ultimum* by applying the fungicide to eight potato cultivars exhibiting varying degrees of susceptibility to these pathogens.

Materials and methods

Source of isolates

Isolates used in this study were obtained from tubers with symptoms of pink rot or leak collected as part of a survey of commercial potato fields as previously described (Taylor *et al.*, 2002*a*). *P. erythroseptica* isolate 266-2 and *P. ultimum* isolate 153-7, have been previously determined to be sensitive to mefenoxam and have been used in a number of studies (Salas *et al.*, 2003; Taylor *et al.*, 2004; Thompson *et al.*, 2007). These isolates were inoculated onto potato tubers (cv. Russet Burbank) to confirm pathogenicity prior to post-harvest challenge inoculations. Isolate aggressiveness was maintained each year by similarly inoculating tubers followed by re-isolation.

Production of test tubers

Potato cvs Atlantic, Dark Red Norland, Goldrush, Kennebec, Pike, Russet Burbank, Russet Norkotah and Snowden were selected on the basis of levels of susceptibility to infection by *P. erythroseptica* and *P. ultimum* (Salas *et al.*, 2003). To maximize uniformity of fungicide application and more closely simulate actual production conditions, non-replicated production plots were used to generate tubers for post-harvest challenge inoculations as previously described (Taylor *et al.*, 2006). These production plots were established in designated areas of irrigated commercial fields near Park Rapids, MN in 2004 and 2005. Each cultivar was planted in three strips with whole or cut certified seed tubers obtained from North Dakota and Minnesota. Fungicide treatments included mefenoxam (Ridomil Gold 4EC) as an in-furrow application of 200 g a.i. ha^{-1} at planting followed by an additional sidedress application of 100 g a.i. ha^{-1} 21 days later. Split application of mefenoxam at these rates has previously been demonstrated to provide the highest level of pink rot and leak control (Taylor *et al.*, 2004, 2006). A second fungicide treatment included two foliar applications of 100 g a.i. ha^{-1} when tubers were 7–8 mm in diameter and again 14 days later. A non-mefenoxam-treated control was included for each cultivar to make comparisons and calculate percentage disease control for each disease. Fungicide treatments and non-treated plots were comprised of four rows, 15 m long and spaced 1 m apart. All were separated by buffers four rows wide planted to potatoes (cv. Russet Burbank). Seed was planted at 30 cm spacing in 2004 and at 36 cm spacing in 2005. The crop was managed each year using agronomic practices typical of those recommended for irrigated potato production in the region.

Post-harvest inoculation

To ensure an adequate quantity of tubers of the desired size and periderm development, plants were killed by mechanical flailing approximately 2–3 weeks prior to maturity. Following harvest, disease-free tubers (140–190 g) were held at 90% relative humidity (15°C) for approximately 2 weeks to optimise wound healing and were acclimated at room temperature (20–25°C) for 1–2 days prior to inoculation with *P. erythroseptica* or *P. ultimum* isolates. Previous research reports residual bioactivity of mefenoxam after up to 6 months in storage (Bruin *et al.*, 1982; Barak *et al.*, 1984; Platt, 1994). Therefore, to minimise the effect of declining levels of mefenoxam, tubers were stored at 10°C for no longer than 3 months prior to inoculation (Taylor *et al.*, 2004, 2006). Tubers harvested from the non-mefenoxam-treated control, in-furrow mefenoxam treatment and foliar mefenoxam treatment were inoculated with *P. erythroseptica* or *P. ultimum*. Inoculation experiments were arranged as randomised complete blocks consisting of 24 treatments (three field treatments × eight cultivars). Each experiment included four replications of 20 tubers each per treatment and the experiments were repeated as separate trials each year at approximately 2-week intervals. Challenge inoculations were conducted on a total of 240 tubers per treatment (4 replications × 20 tubers × 3 trials) in 2004 and 160 tubers per treatment (4 replications × 20 tubers × 2 trials) in 2005. All treatments in each trial were inoculated at the same time using a single preparation of inoculum.

Inoculum was prepared according to protocols used in previous studies (Salas *et al.*, 2003; Taylor *et al.*, 2004, 2006). Freshly prepared zoospore suspensions, adjusted to a concentration of 2×10^4 zoospores ml^{-1}, served as the inoculum for *P. erythroseptica*. Tubers of each cultivar were selected at random and placed in plastic moist chamber boxes (33 cm × 24 cm × 12 cm) lined at the bottom with no. 3 plastic mesh. The tubers were inoculated with 10 µl of the zoospore suspension (approximately 200 zoospores) on each of the three apical eyes and then were covered with four layers of paper towels moistened to saturation with deionised water. To promote infection, the chamber boxes were covered to

establish a high humidity environment and incubated in the dark at ambient temperature at 20–22°C for 10 days.

Inoculations with *P. ultimum* were carried out using mycelial cultures of the pathogen, as previously described (Salas *et al.*, 2003; Taylor *et al.*, 2004). The isolate was grown on modified V8 juice agar (100 mL V8 juice, 1.25 g $CaCO_3$, 15 g of agar, 900 mL deionised H_2O) for 36 h at 20–22°C. The periderm of tubers to be inoculated was manually wounded by abrasion using a commercially available no. 96 Scotch-Brite General Purpose Scouring Pad (3M; St Paul, MN 55144-1000, USA). *Pythium*-colonised 5-mm diameter agar plugs were cut from the margin of actively growing cultures and placed in the centre of the 1 cm^2 abraded area (one plug per tuber, mycelium side down). Tubers inoculated in this manner were placed in plastic moist chamber boxes, covered with moist paper towels and incubated as described above for *P. erythroseptica*.

Disease assessment

Disease incidence and severity were assessed using techniques similar to those described previously (Salas *et al.*, 2003; Taylor *et al.*, 2004, 2006; Thompson *et al.*, 2007). Inoculated tubers were cut and internal tissue was examined for the development of the pink colour characteristic of pink rot infection or watery, black discolouration diagnostic of leak. For pink rot, inoculated tubers were removed from the moist chambers after 10 days and infection was determined by cutting each tuber in half through the axis from the sites of inoculation on the apical bud end to the basal stem end. Leak evaluations were conducted after a 5-day incubation period. Tubers inoculated with *P. ultimum* were bisected through the point of inoculation, perpendicular to the longitudinal axis. In both cases, split tubers were covered with paper towels saturated with tap water and incubated at ambient temperatures of 20–22°C for approximately 30 min to enhance development of the colour characteristic of the specific disease. Infected tubers were counted and disease incidence (I) was expressed as I = (number of infected tubers/number of inoculated tubers) × 100. Disease severity was quantified and defined as a function of the rate of penetration (P) by the pathogen. The maximum depth (D) of rotted tissue was measured from the point of inoculation and P was estimated as $P = D/T$, where T is time in days following inoculation.

Statistical analysis of post-harvest challenge inoculation trials

A one-way ANOVA determined that disease incidence was significantly different in non-mefenoxam-treated control tubers among cultivars for both pink rot ($P < 0.0001$) and leak ($P < 0.0001$) as has been reported in previous research (Salas *et al.*, 2003). Therefore, to accommodate differences in cultivar susceptibility to both diseases, incidence data were used to calculate percentage disease control using the formula [(disease incidence of non-mefenoxam-treated control − disease incidence of treatment)/disease incidence of non-treated control] × 100. Variance homogeneity of percentage disease control was tested using Levene's method (Millikin & Johnson, 1992) among data from trials conducted for both pink rot and leak in both years of the study. Following confirmation of variance homogeneity, percentage disease control data from all trials among years were combined for each disease and a two-way (eight cultivars by two mefenoxam application methods) ANOVA was performed. Mean percentage disease incidence and severity as well as percentage disease control were differentiated using Fisher's protected least significant difference test ($P = 0.05$). All analyses were performed using the general linear model of SAS (PROC GLM; SAS Institute, Inc., Cary, NC, USA).

Results

Susceptibility of potato cultivars to pink rot and leak

Disease incidence and the rate of penetration of each pathogen were significantly different among cultivars ($P < 0.0001$) (Table 1). Incidences of pink rot infection were approximately 25% lower than those reported earlier (Salas *et al.*, 2003) (Table 1), however, relative rankings of cultivar susceptibility to pink rot and leak were similar. Cultivar susceptibility also can be characterised by quantifying disease severity as depth of penetration of the pathogen once infection is established (Salas *et al.*, 2003; Thompson *et al.*, 2007). The current study combined the rate of tuber tissue colonisation and disease incidence as indices of the host : pathogen interaction (Fig. 1). Based on combined incidence and penetration parameters reported here, the inoculation of susceptible cvs Russet Norkotah with *P. erythroseptica* resulted in the greatest amount of pink rot, significantly higher than any other cultivar (Table 1). Correspondingly, while susceptible cv. Goldrush had slightly lower incidence of pink rot infection than moderately susceptible cv. Snowden, the pathogen displayed a significantly higher rate of penetration into tuber tissue of cv. Goldrush, causing a greater amount of infected tissue overall. As expected, significantly less pink rot developed in the moderately resistant and resistant cvs, Russet Burbank, Atlantic and Pike. Fewer tubers were infected and in those that were,

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Table 1 Relative susceptibilities of eight potato cultivars to infection in non-treated tubers by *Phytophthora erythroseptica* (pink rot) and *Pythium ultimum* (leak)

	1998, 1999, 2000[a]		2004, 2005[b]		
Cultivar	Susceptibility rank	Incidence (%)	Susceptibility rank	Incidence (%)	Penetration (mm day)
Pink rot					
Russet Norkotah	1	88.1	1	77.9	5.5
Snowden	2	85.3	2	58.1	4.3
Goldrush	4	64.5	3	54.2	5.2
Kennebec	3	76.6	4	49.8	4.9
Dark Red Norland	5	61.9	5	47.7	5.0
Russet Burbank	6	50.1	6	36.3	4.8
Atlantic	8	31.6	7	29.7	4.0
Pike	7	47.8	8	24.1	4.2
LSD[c] (P = 0.05)				10.1	0.5
Leak					
Dark Red Norland	1	95.6	1	93.5	6.9
Russet Norkotah	5	76.7	2	92.5	5.3
Goldrush	3	85	3	91.8	8.4
Russet Burbank	6	73	4	91.6	6.4
Pike	2	95.6	5	85.4	8.0
Atlantic	4	77.3	6	78.1	7.8
Kennebec	7	67.4	7	73.4	6.8
Snowden	8	33.5	8	45.9	6.5
LSD[c] (P = 0.05)				10.8	1.4

[a]Based upon previous results of Salas *et al.* (2003).
[b]Results obtained in current study.
[c]LSD, Fisher's protected least significant difference.

the disease developed more slowly (Fig. 1A). In contrast, there were fewer differences in susceptibility to leak among the cultivars used in this study (Table 1; Fig. 1B). Cultivar Snowden was found to be significantly less susceptible to leak than all other cultivars with a mean infection incidence of 49.5% (Table 1). Cultivars Atlantic and Kennebec were significantly less susceptible than Dark Red Norland, Russet Norkotah, Goldrush, Russet Burbank and Pike; however, infection frequencies for all these cultivars were very high, ranging from 73.4% to 93.5% (Table 1). Although penetration of *P. ultimum* in potato tubers differed very little among cultivars, rotting

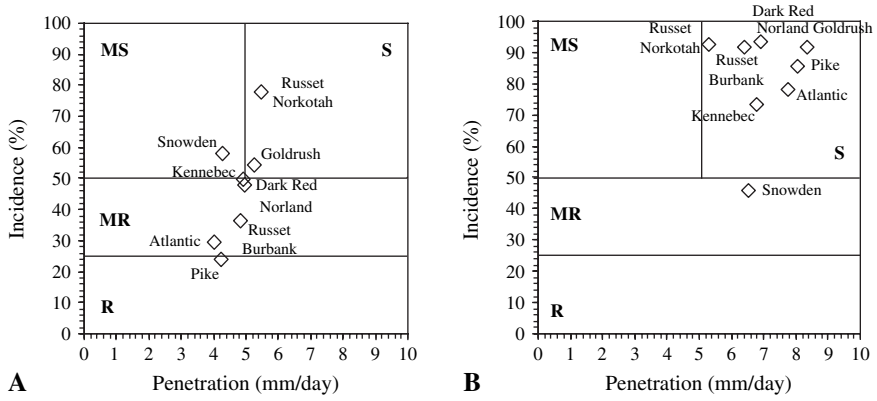

Figure 1 Relationship between percentage disease incidence and rate of penetration (mm day^{-1}) in non-treated tubers of potato cultivars inoculated with the pink rot pathogen, *Phytophthora erythroseptica* (A) and the leak pathogen, *Pythium ultimum* (B). Symbols of cultivars closer to the origin of the X–Y axes denote less disease. Graphs are divided to denote postulated host : pathogen interactions of susceptible (S), moderately susceptible (MS), moderately resistant (MR) and resistant (R). Separation of R and MR is based on infection incidence only. Separation of MS and S is based upon the rate of pathogen penetration.

progressed nearly twice as rapidly in Goldrush as in Russet Norkotah (Fig. 1B). For these reasons, we consider Dark Red Norland and Goldrush to be the most susceptible cultivars to leak and Snowden to be the least susceptible.

Mefenoxam and disease control

The control of pink rot ($P = 0.4402$) and leak ($P = 0.1883$) incidence obtained following in-furrow and foliar applications of mefenoxam did not differ significantly (data not shown), therefore, results obtained for these two application methods were combined for further analysis. In general, mefenoxam had a greater impact in reducing disease incidence on cultivars most susceptible to pink rot such as Russet Norkotah, Kennebec and Snowden and was less effective on moderately resistant and resistant cvs Atlantic and Pike (Fig. 2A). Notable exceptions were substantial reductions in disease incidence in moderately resistant cvs Russet Burbank and Dark Red Norland and less reduction of pink rot in susceptible cv. Goldrush. Contrary to results observed with pink rot, mefenoxam only reduced the incidence of leak when applied to Snowden, which is the only cultivar with any resistance to leak (Fig. 2B); however, levels of disease control were not significantly different among cultivars ($P = 0.0916$).

Mefenoxam applications provided substantially better control of pink rot compared with leak within cultivars (Fig. 3). Control of pink rot by mefenoxam varied significantly among the cultivars evaluated ($P = 0.0153$) from 28% (cv. Goldrush) to 67% (cv. Snowden) (Figs 2 and 3). Depth of penetration of *P. erythroseptica* was not affected by the presence of mefenoxam in any cultivar (Fig. 3),

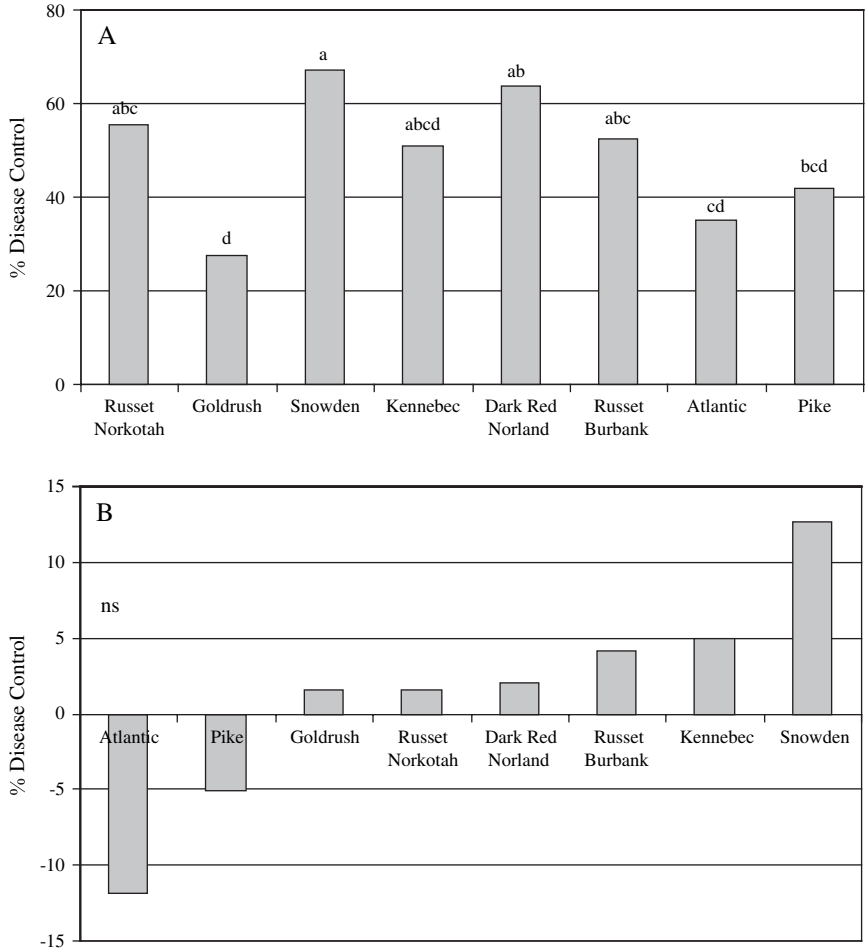

Figure 2 Control of pink rot (A) and leak (B) in tubers of potato cultivars treated with furrow and foliar applications of mefenoxam and challenge inoculated after harvest with *Phytophthora erythroseptica* and *Pythium ultimum*, respectively. Percentage disease control of pink rot (A) was significantly different based on Fisher's protected least significant difference test ($P = 0.05$). ns, not significant.

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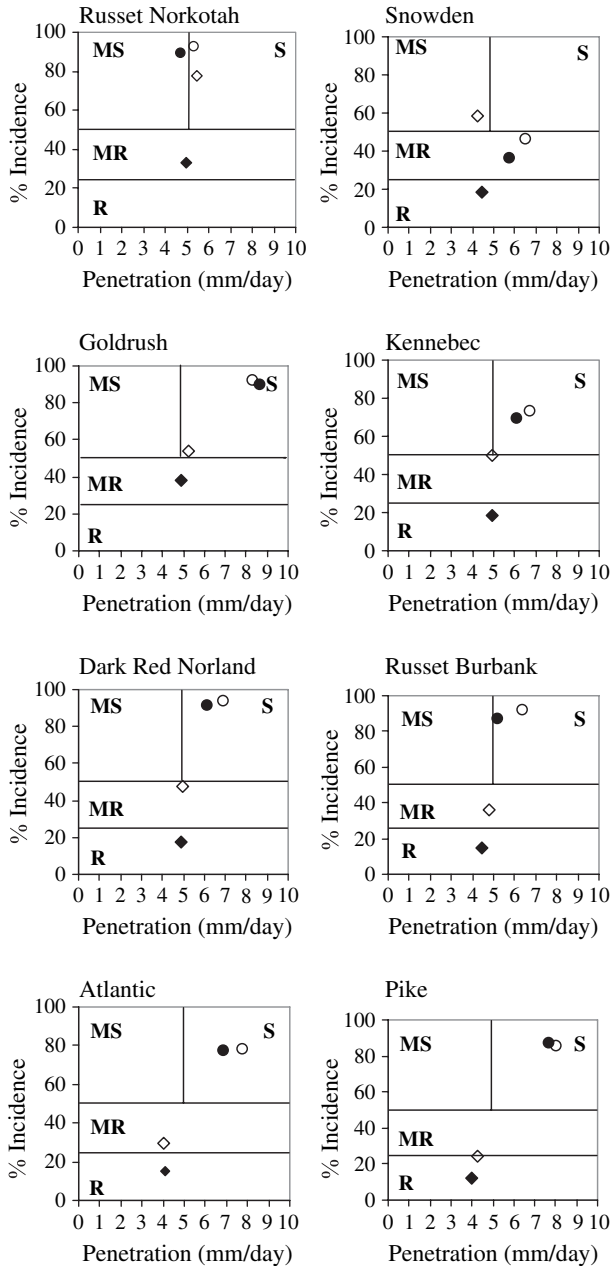

Figure 3 Percentage disease incidence and rate of penetration in non-treated tubers (open symbols) and tubers from plants treated with furrow and foliar applications of mefenoxam (closed symbols) following challenge inoculation with *Phytophthora erythroseptica* (◇,◆) and *Pythium ultimum* (○, ●). Symbols of treatments closer to the origin of the X–Y axes denote less disease relative to the non-treated control. Graphs are divided to denote postulated host : pathogen interactions of susceptible (S), moderately susceptible (MS), moderately resistant (MR) and resistant (R). Separation of MS and S is based upon the rate of pathogen penetration.

however, penetration of *P. ultimum* was significantly reduced from 7.0 mm day^{-1} in non-treated tubers to 6.4 mm day^{-1} in mefenoxam-treated tubers among cultivars.

Discussion

Pink rot and leak are storage rot diseases that frequently occur together in the same potato production areas and traditionally, similar approaches have been used to manage them. Most of these practices are cultural in nature and although these management tactics may reduce disease incidence in many situations, application of mefenoxam remains a widely practiced control measure. However, the effectiveness of mefenoxam has become compromised in areas where resistance has developed in pathogen populations (Taylor *et al.*, 2002*a*, 2006; Peters *et al.*, 2003*a*). Fungicide resistance can be a mitigating factor in the management of many plant diseases but resistance to mefenoxam is of particular importance with water rots because mefenoxam currently is the only fungicide effective against both the diseases, providing very good control of pink rot and fair control of leak when applied in furrow (Taylor *et al.*, 2004).

As the frequency and scope of mefenoxam resistance increases, the need to redirect efforts and modify strategies employed to manage these diseases becomes more urgent. Management of pink rot and leak within the context of mefenoxam resistance is undoubtedly a daunting task. Alternative approaches, including unique crop rotations, conservation tillage and the development of suppressive soils (Peters *et al.*, 2003*a*,*b*, 2005), foliar and post-harvest applications of phosphorous acid (phosphonate, phosphite) (Johnson *et al.*, 2004; Miller *et al.*, 2006) and establishment of transgenic potato lines expressing antimicrobial peptides (Osusky *et al.*, 2004, 2005) have shown some potential, particularly against *P. erythroseptica*. None of these management strategies and tactics has been widely adopted. Cultivar resistance has yet to become a major feature of management programmes, but recent successes in the development of germplasm with resistance to both diseases demonstrates the potential of developing resistant cultivars through traditional breeding programmes (Thompson *et al.*, 2007).

Previous work with cvs Russet Norkotah and Russet Burbank demonstrated that mefenoxam can influence the host : pathogen interaction and subsequently, the extent of pink rot development (Taylor *et al.*, 2006). The results reported here demonstrate that field applications of mefenoxam will control pink rot and that potato genotypes respond differentially to *P. erythroseptica*, resulting in significant differences in disease control among cultivars. These differences were not related to cultivar maturity, but a trend towards greater mefenoxam efficacy in the least resistant cultivars was apparent. Similar

observations were made in experiments involving two potato cultivars used in the present work (Peters *et al.*, 2003*a*). Although disease control in these two cultivars did not differ significantly in the current study, the earlier study found that mefenoxam provided significantly greater suppression of pink rot in the moderately susceptible cv. Kennebec relative to the moderately resistant cv. Russet Burbank. This relationship might extend to control of other diseases as well because similar results were obtained in work involving black shank of tobacco caused by *Phytophthora nicotianae* (Reilly, 1980; Van Jaarsveld *et al.*, 2002). In those studies, metalaxyl treatments most effectively controlled black shank in susceptible and moderately resistant cultivars, but the fungicide did not significantly reduce the level of disease in resistant cultivars. In light of these observations, additional research assessing this association in other pathosystems may be warranted.

Mefenoxam was not as effective against *P. ultimum* and the low levels of control (0–12%) may have contributed to our inability to identify differential susceptibility or cultivar specific efficacy as most cultivars used in this study were highly susceptible to leak. Inconsistent disease control and conflicting results have been noted previously in field trials designed to assess the efficacy of mefenoxam for leak control (Mulrooney, 1982, 1998; James & Stevenson, 1999; Kirk *et al.*, 2001; Platt *et al.*, 2003*b*). Movement of mefenoxam to the developing tubers, and ultimately its efficacy, can be affected by environmental conditions, particularly soil moisture levels (Mulrooney & Gregory, 2002; Peters *et al.*, 2003*a*). Spatial and temporal variations in soil moisture content may partially explain inconsistencies and erratic efficacy of mefenoxam on leak occasionally observed by growers. Additionally, mefenoxam (metalaxyl) is not uniformly distributed within a potato tuber but preferentially concentrated near the periderm surface (Bruin *et al.*, 1982). Because leak is a wound pathogen, the wounds necessary for infection likely breech this barrier allowing entry and penetration (Taylor *et al.*, 2004). From the studies reported here, it is apparent that the level of mefenoxam distributed within the medullary areas of tuber tissue are insufficient to affect further growth and penetration of *P. erythroseptica* but is sufficient to affect the penetration of *P. ultimum* (Fig. 3). Bruin *et al.* (1982) reported metalaxyl concentrations in the periderm of 0.039 μg g^{-1} and 0.055 μg g^{-1} following foliar applications at 0.25 kg ha^{-1} and 0.50 kg ha^{-1}, whereas concentrations in the cortex (0.016 μg g^{-1}, 0.022 μg g^{-1}) and medullary tissues (0.017 μg g^{-1}, 0.034 μg g^{-1}) were lower. Results from this and previous research indicate that the level of fungicide present in the periderm is sufficient in reducing infection of *P. erythroseptica,* but clearly, the low mefenoxam concentration in the medullary tissue is not sufficient to reduce further penetration. Interestingly, the level of mefenoxam in the medullary tissue was adequate to reduce the rate of penetration of *P. ultimum*. This is difficult to understand since the sensitivity of *P. ultimum* to mefenoxam is substantially less than the sensitivity of *P. erythroseptica* to this fungicide (Taylor *et al.*, 2004).

Although the cultivars did not differ significantly in their reaction to *P. ultimum* following application of mefenoxam, it is notable that the only level of leak control approaching that reported previously (Taylor *et al.*, 2004) was attained with the most resistant cv., Snowden. This suggests a possible relationship between mefenoxam efficacy and cultivar resistance, which should be re-examined in future experiments. Because mefenoxam often has only a limited effect upon leak development and its efficacy can be inconsistent, growers should seriously consider restricting the use of the compound, particularly if it is being used solely to target this disease.

Our results imply that a strategy of optimally combining cultivar resistance with judicious applications of mefenoxam applications could potentially be integrated into a programme to manage water rots, particularly pink rot. The susceptibility of many commonly grown cultivars is known (Peters & Sturz, 2001; Salas *et al.*, 2003; Peters *et al.*, 2004), therefore, those most likely to benefit from mefenoxam treatments might be targeted as part of a management scheme. In the case of pink rot, cultivars having the greatest susceptibility would be potential candidates for this approach. To reduce exposure of the *P. erythroseptica* population to mefenoxam and thereby limit development of resistance to the fungicide, it would be prudent to apply mefenoxam to those cultivars where disease control benefits can be maximised. This would be particularly important in the case of moderately resistant and resistant cultivars like Atlantic and Pike, where mefenoxan applications had the least effect upon pink rot development and presumably would derive the least economic benefit from its use. Therefore, in areas where pink rot is not a chronic problem, growers might consider cultivar resistance as part of their management strategy and apply mefenoxam either sparingly or not at all. This approach has been implemented successfully in some areas of the USA, where growers changed their cultivar to Russet Burbank after experiencing perennial problems with pink rot in Russet Norkotah.

Cultivar specific, targeted applications of mefenoxam could prove to be an efficient means of reducing infection rates and possibly managing mefenoxam resistance and maintaining longevity of the compound. However, the

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occurrence of mefenoxam-resistant strains would limit the effectiveness of such an approach. Previous work with *P. erythroseptica* suggested that insensitivity to mefenoxam endows the pathogen with a selective advantage. A mixed population of mefenoxam-sensitive and mefenoxam-insensitive genotypes can rapidly shift to a population solely comprised of resistant genotypes (Taylor *et al.*, 2002*b*). Moreover, some resistant isolates appear to be more aggressive than mefenoxam-sensitive isolates even in the absence of mefenoxam selection pressure (Taylor *et al.*, 2006). Other investigations have demonstrated that mefenoxam-resistant isolates grow faster and produce more oospores *in vitro* than insensitive phenotypes (Porter *et al.*, 2007) and that quantitative shifts towards higher levels of insensitivity can occur in populations derived from single oospore isolates obtained from phenotypes with intermediate levels of resistance to the fungicide (Abu-El Samen *et al.*, 2005). Although mefenoxam resistance in *P. ultimum* has been detected (Taylor *et al.*, 2002*a*), it has not spread rapidly. Therefore, even though mefenoxam does not control leak as effectively as it controls pink rot, resistance to the fungicide could still become an issue. The limited amount of protection afforded by mefenoxam undoubtedly would be lost if resistance continues to spread in that population.

In the future, cultivar resistance will provide the best means of addressing these factors and managing pink rot and leak. Based upon results reported in our previous work (Salas *et al.*, 2003), cultivar resistance generally appears to be stable and predictable. The nature of resistance to pink rot and leak has not been studied, but considering the differences in aetiology between these diseases, it is likely that the genetic basis for resistance to *P. erythroseptica* differs from resistance to infection by *P. ultimum* (Thompson *et al.*, 2007). Our results appear to support this view. The cv. Snowden is moderately susceptible to pink rot but moderately resistant to leak. The pattern is similar but reversed with cvs Pike and Atlantic. These observations also suggest that more than one gene may be involved in resistance. Interestingly, the cultivars showing the greatest resistance to pink rot (cvs Atlantic and Pike) and leak (cv. Snowden) share a common parent in their lineage B5141-6 (cv. Lenape) (Salas *et al.*, 2003). With sources of resistance such as this, it should be possible to develop germplasm with greater resistance to both diseases. As additional resistant germplasm is developed and new cultivar releases arise from them, mefenoxam also might be applied in a targeted manner to supplement the level of disease control afforded by such cultivars. The results reported here should be considered as new approaches to control the water rots of potato and strategies to manage mefenoxam resistance are developed and implemented.

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