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Targeted insecticide regimes perform as well as a calendar regime for control of aphids that vector viruses in seed potatoes in New Zealand

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ABSTRACT

Over two seasons (2002–03; 2003–04) at Pukekohe in the North Island and three seasons (2002–03; 2003–04; 2004–05) at Lincoln in the South Island of New Zealand, a common calendar-based insecticide regime was compared with a targeted insecticide regime for control of aphids on potatoes, and for any subsequent change in potato virus transmission by aphids. Treatments were an untreated control (1), calendar-applied fortnightly applications of methamidophos to foliage grown from untreated seed (2) and seed treated with imidacloprid (3), λ-cyhalothrin applied at an aphid threshold to foliage from untreated (4) or imidacloprid-treated seed (5) and pymetrozine at an aphid threshold to foliage from untreated seed (6). The action threshold for spraying was when apterous aphid populations exceeded 10 per 150 potato leaves. As indicated from suction traps at the trial sites, aphid flights peaked in autumn and spring. At Pukekohe in both years, aphids were not found on the potato foliage in any treatments until late January (midsummer), making 3–4 of the 5–7 methamidophos applications for the season unnecessary. Further applications of methamidophos kept apterous aphid populations at negligible levels until late summer, but populations in autumn tended to be higher than in untreated plots, due probably to the suppression of aphid predators (syrphids and lacewings) by the insecticide. No apterous aphids were found on potato foliage above the threshold in imidacloprid-treated or untreated plots until 3 weeks before desiccation of the foliage, necessitating only one application of λ-cyhalothrin or pymetrozine for each season. At Lincoln over all years, the imidacloprid seed treatment and the calendar-based 7–11 foliar applications of methamidophos kept plots free of aphids. However, the imidacloprid seed treatment alone, or one foliar application of λ-cyhalothrin alone or pymetrozine alone, were all that were required to keep aphid populations below the threshold during two of the three seasons, and were not required in the 2003–04 season. The incidence of tubers infected with potato leafroll virus (PLRV) and potato virus Y (PVY) at both sites was not significantly reduced by any insecticide treatment compared with the untreated control. The insecticide treatments also had no significant effect on total potato yields. In New Zealand, an imidacloprid seed treatment followed by a foliar application of λ-cyhalothrin or pymetrozine whenever apterous aphid populations exceed 10 per 150 potato leaves appears sufficient to maintain aphid populations below the action threshold, without compromising yields or increasing virus risk in tubers.

1. Introduction

Viruses that are transmitted by aphids can cause significant yield reductions in potatoes internationally (van de Zaag, 1987) and in New Zealand (Ovenden et al., 1985). Currently, virus disease is controlled through the widespread use of virus-tested seed tubers. Several viruses may still be transmitted to potato crops by aphids. These include potato viruses Y (PVY), M (PVM) A (PVA) and S (PVS), alfalfa mosaic virus (AMV), potato aucuba mosaic virus (PAMV), and potato leafroll virus (PLRV) (Fletcher, 1997). PVY (Fletcher, 1989) and PLRV are considered the most important, but PAMV has not been recorded in New Zealand for decades (J.D. Fletcher, pers. comm.). At least 28 aphid species in New Zealand are actual or potential potato virus vectors (D.A.J. Teulon, unpubl. data), and nine of these can transmit PLRV, of which only three species are reported to be common on potatoes in New Zealand. In Canterbury, the region in New Zealand where most of the potato crops are grown for seed and process markets, the green peach aphid (*Myzus persicae* (Sulzer)) is the most common aphid found on potato plants, followed by the foxglove aphid (*Aulacorthum solani* (Kaltenbach))

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and the potato aphid (*Macrosiphum euphorbiae* (Thomas)) (Stufkens and Teulon, 2001). *M. persicae* is the most important vector of PLRV, with *M. euphorbiae* and *Aulacorthum solani* not known to carry PLRV (Miln, 1978). The melon aphid (*Aphis gossypii* Glover), has been found in sizable numbers on sprayed potatoes in the Hawke's Bay, North Island (T. Herman, pers. comm.), but few have been found in Canterbury (Stufkens and Teulon, 2001). *A. gossypii* is known to transmit PLRV and PVY in potatoes (Sertkaya and Sertkaya, 2005).

Insecticides are applied to potatoes to control potato tuber moth (*Phthorimaea operculella*) (Foot, 1979) and to seed potatoes to control several species of virus-transmitting aphids (Stufkens and Teulon, 2001). The requirement to keep potatoes, especially seed potatoes, virus free, necessitates a thorough and exacting aphid control programme often resulting in multiple applications of insecticides from a range of chemical classes. In the 2001–02 and 2003–04 seasons, most potato seed crops in Canterbury received a single seed/tuber treatment as well as 1–10 foliar applications (van Toor and Teulon, 2006). The seed/tuber treatments were mostly imidacloprid, a neonicotinoid, but also phorate, an organophosphate. The foliar applications included mostly organophosphate (methamidophos), but also carbamate (pirimicarb), pyrethroid (λ-cyhalothrin) and pyridine–azomethine (pymetrozine) insecticide classes.

Insect control programmes that rely on the multiple uses of the same or related insecticides can lead to insecticide resistance in the absence of insecticide resistance management strategies (Martin et al., 2005). Insecticide resistance to aphids on potatoes has already been documented in New Zealand. Fellowes and Fergusson (1994) considered that insecticide resistance caused control failure of *M. persicae* in field trials in potatoes at Pukekohe. Insecticide resistance was confirmed in *M. persicae* to selected organophosphates, carbamates and pyrethroids through laboratory studies (Cameron and Walker, 1988). Insecticide-resistant melon aphid populations have been found on chrysanthemum in New Zealand (Martin and Workman, 1997).

Aphids on potato crops may contain mechanisms that confer resistance to a range of chemical classes. Parthenogenetic lineages of *M. persicae* individuals collected on potatoes during February–March from main potato growing regions in New Zealand showed 60% contained one or more mechanisms (elevated carboxylesterase, modified acetylcholinesterase, knockdown, super-knockdown and imidacloprid resistance) that confer resistance to between one and three of any of four insecticide classes: organophosphates, dimethyl carbamates, pyrethroids and neonicotinoids (van Toor et al., 2008).

In response to these issues, in 2002 the New Zealand potato industry, supported by the New Zealand Vegetable and Potato Growers Federation (now incorporated into Horticulture NZ), the Ministry for Agriculture and Forestry Sustainable Farming Fund and the agrochemical industry, initiated a project to develop and implement a resistance management programme for aphids in potatoes. It aimed at reducing insecticide pressure on aphid populations to limit the development of resistance. Since this strategy may increase the risk of virus transmission to potatoes by virus-carrying aphids, we studied the effect of reduced insecticide use in seed potatoes on aphid numbers and on virus incidence in field trials. They compared the effects of the common calendar-based methamidophos spray regime with targeted regimes in which other insecticides were applied only when aphids were present.

2. Materials and methods

2.1. Trial design

2.1.1. Preparation

Trials were established in 2002 and 2003 at Lincoln and Pukekohe, and at Lincoln in 2004. Pre-basic or first generation potatoes (*Solanum tuberosum*) cvar. Russet Burbank seed potatoes, certified as virus-tested, were sown between 22 October and 5 November in each year. The potatoes were grown under standard management practice for each region. At Pukekohe, the fertilisers, 6–5–5% N–P–K at 2 t/ha and nitrogen at 92 kg/ha, were applied at sowing; herbicides metribuzin and linuron were applied prior to emergence; the fungicide mancozeb was applied weekly after emergence of the foliage to control early blight (*Alternaria solani*) and late blight (*Phytophthora infestans*). At Lincoln, 15% potassic superphosphate at 400 kg/ha or 13–14–15% N–P–K at 200 kg/ha, was applied at sowing and nitrogen at 50 kg/ha was applied in midsummer; the herbicides glyphosate and tribenuron methyl were applied pre-sowing; the fungicide copper oxychloride was applied once in early summer to control early and late blight. In all trials, the potato rows were moulded 7–8 weeks after sowing.

2.1.2. Treatments

The same treatments were applied in all trials (Tables 1–5). They comprised an untreated control (1), calendar-applied methamidophos to foliage grown from untreated seed (2) and seed treated with imidacloprid (3), or when aphid numbers reached a threshold, target applications of λ-cyhalothrin to foliage from untreated (4) or imidacloprid-treated seed (5) and pymetrozine to foliage from untreated seed (6). The aphid threshold for targeted spraying was set at 10 apterous aphids per 50 plants (Raman, 1984) as recommended to potato growers in New Zealand through the Crop & Food Research Broad sheet service (Fletcher and Herman, 2000).

The treatments were replicated six times and arranged in a randomised block design. Plots were 6 m long. In trials established in the first 2 years, the plots contained six rows each: row spacings were 0.76 m and plants were 0.30 m apart within the rows. The outside rows of each plot were buffer rows. Since there was a risk of spreading viruses between plants when sampling them for insect populations, two of the middle rows were sampled weekly for aphids and their invertebrate predators/parasitoids, and the other two middle rows were only sampled for potato yields and virus incidence. At Lincoln in 2004–05, the plots comprised only two sampling rows; one row for monitoring aphids/predators/parasitoids and the other for measuring yields and virus incidence, with one buffer row shared between plots and two buffer rows surrounding the trial.

In treatments 3 and 5, Gaucho® (600 g/kg imidacloprid) was applied at 90 mL/T seed, equivalent to 134 mL imidacloprid/ha. In treatments 2 and 3, Tamaron® (600 g/L methamidophos) was applied at 800 mL/ha (480 mL methamidophos/ha) every 10–14 days commencing 42 days after potato emergence. At the appropriate times, Karate® Zeon Technology (250 g/L λ-cyhalothrin) was applied at 40 mL/ha (10 mL λ-cyhalothrin/ha) (treatments 4 and 5), and Chess® WG (500 g/kg pymetrozine) was applied at 200 g/ha (100 g pymetrozine/ha) (treatment 6). All insecticides were applied at 500 L water/ha.

The insecticides were applied at Pukekohe using a tractor-mounted experimental sprayer with a 4 m wide boom containing 18 TXVK% Conjet nozzles, and at Lincoln using an experimental knapsack sprayer with an offset 1.5 m wide boom containing three, fan-jet 110°/08/3 spray nozzles. Water pressure was 207 kPa throughout all foliar spray applications.

Potato foliage was desiccated in mid-February at Pukekohe and in mid-March at Lincoln with one application of Reglone® (200 g/L diquat) at 3 L/ha in 600 L/ha water. There was no regrowth of foliage in any trial, which reduced the risk of virus transmission by aphids during this time. Potatoes were harvested as per commercial practice (in July at Pukekohe and in May at Lincoln).