INFLUENCE OF PLANT GROWTH REGULATORS ON BUD DEVELOPMENT IN KIWIFRUIT

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SUMMARY

Axillary bud movement on upright kiwifruit (*Actinidia chinensis*) canes was inhibited with NAA or CPPP (each at 0.1% in lanolin) applied to the stump. Stem-nicking halfway along upright canes at bud swell increased bud break by 13%. CPPP (⩾ 0.05% in lanolin) applied to these nicks on horizontal canes gave 90% reduction in shoot growth distal to the nicks and promoted bud break proximal to them. For horizontal canes, chlormequat chloride (0.8% in lanolin) and paclobutrazol (⩾0.2% in lanolin) increased bud break and decreased shoot growth distal to the nicks by 30% respectively. NAA and CPPP residues moved considerable distances within canes.

INTRODUCTION

Previous plant growth regulator (pgr) studies on mature kiwifruit have involved broadcast treatments of daminozide (Alar), applied in autumn or spring (Davison 1971). Both treatments reduced summer vegetative growth and increased yields but fruit flattening occurred in some cases.

In the present study daminozide and five other pgr's, likely to either retard or promote axillary bud development, were tested in two different assays on young kiwifruit vines. This was an initial study in a programme to assess the potential of pgr's in kiwifruit management.

METHODS

All tests used single 1 y.o. canes on 2 y.o. Hayward plants in plastic bags (15 cm dia.) containing a balanced fertiliser mix. The plants were grown in a glass-topped screen house and watered continuously by placing them on tables covered with wet capillary cloth.

Assay 1. Stump treatment

Upright plants were in five blocks each containing six rows of eight plants. Chemicals allocated at random to one of the rows were tested in a two-fold dilution series over a 64-fold concentration range. A guard row of untreated plants was placed across the outer end of each block.

On 3 February 1983, the canes were pruned approximately 2 cm above the sixth or seventh axillary bud about 40 cm above soil level. This removed all current season's growth. Immediately after pruning, glass caps (15 x 6 mm ID) containing the regulator-lanolin mixtures (0.4 ml) were placed over the stumps.

All pgr's were of technical grade. Solutions (2-4%) of each chemical were prepared in 1-propanol and an aliquot stirred into warm (50°C) Isocreme (Croda Chemicals Ltd, UK). Compounds tested over the 0.0125-0.8% concentration range in this lanolin base were: NAA (1-naphthylacetic acid), CCC (chlormequat chloride), daminozide, and gibberellic acid (90% min. gibberellin A_3). Two CSIRO chemicals viz., 5-(2-carboxyphenyl)-3-phenylpyrazole (CPPP; Geissler *et al* 1975) and β(6-bromopyridin-3-yl) alanine (BPA; Phillips *et al* 1981) were tested at concentrations 0.0063-0.4% and 0.0016-0.1% in lanolin respectively. The latter compound was dissolved in HC1:1-propanol (1:2500).

Bud break and shoot development were assessed at 8 weeks after application.

Assay 2. Stem nicking

Canes varied between 60 and 90 cm long and contained 10-14 dormant buds. Plants were in four blocks each containing six groups of 10 plants. Within each group, half the plants were held upright (U) and half bent over with most of the cane held on the horizontal (H) about 30 cm above the table.

At bud swell on 3 August 1983, about 10 days prior to bud burst, two nicks (5 x 4 x 1 mm deep) were cut on opposite sides of the stem about halfway along each cane. Slit PVC tube (20x7.9mm ID) containing 0.5g of a regulator-lanolin (1:1 Isocreme:Cremba) mixture was then sleeved over the nicks. Each chemical was tested at four rates as in assay 1 and included NAA, CPPP, BPA (0.0125-0.1% in lanolin base) and CCC, gibberellic acid and paclobutrazol (0.1-0.8% in lanolin base). Paclobutrazol (PP 333) was a 250 g ai/litre SC formulation. Two nicked controls (U and H containing lanolin alone) were included with each chemical tested. Nine untreated U plants were also placed at random within the blocks.

Bud break and shoot weight were assessed at 11 weeks after application.

Residue analysis

NAA and CPPP were extracted with 0.1N sodium hydroxide. Clean-up was by sequential acid — base/chloroform extraction. Analysis was by reversed-phase HPLC.

In assay 1, the two nodes and internode segments immediately below the stump were analysed. In assay 2, two plants from each of the 0.1% NAA and CPPP treatments were cut above each node and the segments plus any terminal shoots analysed.

RESULTS

Assay 1

Shoot growth from axillary buds occured in 43% of control plants, but on only three of the 35 decapitated plants (9%) treated with 0.0125% NAA and higher concentrations. Bud inhibition was complete with 0.1% NAA and higher concentrations and callus tissue formed on all stumps treated with 0.025% NAA and higher concentrations.

Similarly, CPPP ($\geqslant$0.025%) inhibited bud development on all but 8% of plants and any shoots that did develop were abnormal with epinastic leaves, shortened internodes, prominent nodes and a maximum length of 13 cm. Control plants produced shoots well in excess of 1 m.

NAA and CPPP moved downward in the stem. Residues below 0.1 ppm were common with the 0.1% NAA rate and compared with up to 9.8 ppm at the 0.8% rate. CPPP tended to be more concentrated near the point of contact. Within two nodes of the stump, levels ranged between 0.13 and 9.2 ppm depending on application rate. At and beyond the second node, levels were generally between 0.1 and 0.5 ppm for all rates.

Both gibberellin ($\geqslant$0.1%) and BPA ($\geqslant$0.0063%) promoted ($P < 0.05$) shoot growth from the apical bud. This effect was induced in 70% of the treated plants. In some plants treated at higher gibberellin levels ($\geqslant$0.2%) where the apical bud did not shoot, short shoots with virtually no stems developed from several lower axillary buds.

Neither chlormequat chloride nor daminozide had any influence on bud development in these tests.

Assay 2

A comparison between nicked and untreated upright plants indicated that stem-nicking increased the overall bud break along the canes by 13.2%. The influence of nicking was in an upward direction as the bud break for U and H controls below the nicks (Table 1) was significantly ($P < 0.001$) less than that above the nicks (Table 2). Table 1 also shows that NAA reduced bud break below the nicks and was more effective ($P < 0.05$) on U than H canes. In addition, NAA had no upward influence on bud break with either cane orientation. CPPP ($\geqslant$0.025%) had the reverse effect of NAA in that it promoted bud break below the nicks on both U and H canes.

Shoot growth below the nicks was significantly ($P < 0.05$) less on U than H control plants (Table 1). The downward influence of NAA on U canes was again apparent in the reduced shoot growth below the nicks. NAA residues were found in the stems both above and below the nicks. While only traces of NAA (< 0.1 ppm) were found above the nicks on one U cane, the other contained 0.56 and 0.34 ppm in the two upper internode segments

TABLE 1: Influence of NAA and CPPP on axillary bud development below the area of chemical entry into upright (U) and horizontal (H) kiwifruit canes.

Chemical	Rate‡ (%)	Bud break (%) U	Bud break (%) H	Shoot weight (%)* U	Shoot weight (%)* H
nicked control	0	42.2	45.2	39.6	52.5
NAA	0.1	28.2	33.1	27.2	48.9
,,	0.05	17.7	52.5	22.1	56.9
,,	0.025	25.5	40.5	29.4	36.7
CPPP	0.1	74.5	61.1	74.9	91.6
,,	0.05	55.2	62.3	71.7	91.4
,,	0.025	67.9	73.6	68.1	68.0

* wt of all shoots below nicks as a percentage of total shoot wt on canes.

‡ plants treated with 0.0125% rates were not significantly different from nicked controls.

TABLE 2: Influence of chlormequat chloride and paclobutrazol on axillary bud development above the area of chemical entry into upright (U) and horizontal (H) kiwifruit canes.

Chemical	Rate‡ (%)	Bud break (%) U	Bud break (%) H	Shoot weight (g) U	Shoot weight (g) H
nicked control	0	65.9	68.6	72.3*	63.9*
chlormequat	0.8	47.9	95.0	67.0	65.1
chloride	0.4	83.0	78.0	113.6	83.3
,,	0.2	59.6	81.0	93.3	78.7
paclobutrazol	0.8	60.0	81.3	45.2	46.4
,,	0.4	73.8	43.8	71.9	49.7
,,	0.2	82.5	53.3	61.3	37.2

‡ plants treated with 0.1% rates were not significantly different from nicked controls.

* ave shoot weights on U and H nicked controls were 123 and 133g respectively.

with traces elsewhere in the plant. For H canes, higher levels were found below than above the nicks. Traces were found in the terminal shoots on both U and H canes.

For CPPP ($\geqslant$0.025%) shoot growth increased below the nicks for U and H canes. Generally, toxic symptoms developed in shoots from buds above the nicks on plants treated with higher CPPP concentrations ($\geqslant$0.05%). Withered epinastic shoots developed within 40 cm of the nicks. Toxic symptoms developed below the nicks with the 0.1% CPPP concentration only and extended down to 15 cm. As was the case for NAA, CPPP also moved up and down the stem. In U and H canes without terminal shoots maximum levels of 0.8 and 0.2 ppm were found above and below the nicks respectively. For U and H canes with terminal shoots, residues above the nicks were only found in the shoots themselves at levels up to 0.14 ppm.

Concentrations of chlormequat chloride at and above 0.2% in lanolin increased ($P < 0.05$) bud break above the nicks on H canes (Table 2). Almost all the buds (95%) broke with the 0.8% concentration. The chemical had no retarding effect on shoot growth.

By contrast, paclobutrazol did not have a consistent influence on bud break but significantly reduced shoot growth on horizontal canes above the nicks by about one third at concentrations of 0.2% and above. Affected plants developed self terminating shoots above the nicks with increased shoot weight:shoot length ratios. There were significantly ($P < 0.05$) fewer vegetative shoots above the nicks on the paclobutrazol treatments.

DISCUSSION

Bud inhibition and callus formation induced by NAA in kiwifruit plants is consistent with similar tests with other species (Phillips 1975; Brown *et al* 1979). The influence of CPPP in also exerting apical dominance was unexpected. Generally auxin transport inhibitors promote (as in assay 2), rather than inhibit, bud activity in a downward direction (Phillips 1975). The timing of the decapitation assay may have favoured the activity of NAA and CPPP as the release of apical buds from dormancy for the control plants diminished from 100% in spring to 43% at the start of the trial in February. Nevertheless, both compounds are potentially useful chemical pruning agents for summer vegetative control in kiwifruit.

Apical dominance is diminished on H canes and as there was no difference between the bud break on H and U nicked controls, it is apparent that stem-nicking reduced the apical dominance of U canes. This would account for the 13% increased bud break on the nicked U canes compared with untreated U control plants.

The residue analysis showed that NAA and CPPP moved above (> 70 cm) and below the point of chemical entry. NAA concentrated more in the stem than the shoots while the reverse was the case for CPPP. This could account for the observed toxic effect of CPPP on shoots.

More pronounced effects on bud development would probably be expected on H than U canes and this was the case for paclobutrazol and chlormequat chloride. Paclobutrazol is an experimental growth regulator which needs to be in true solution for effective xylem transport within the plant. The lanolin formulation used may have led to the inconsistent effects. The results do suggest, however, that paclobutrazol is a promising chemical to introduce into kiwifruit replacement cane or fruiting wood to regulate growth. Likewise, chlormequat chloride is potentially useful for increasing kiwifruit yields by promoting bud break in fruiting canes which can be as low as 10% and seldom exceeds 50% on commercial orchards.

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