THE TOXICITY OF PICLORAM, DICAMBA, 2,4-D AND OTHER GROWTH REGULATING HERBICIDES TO RUBBER VINE

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Summary. The toxicity of 2,4-D, 2,4,5-T, dichlorprop, fenoprop, MCPA, dicamba, triclopyr and picloram to rubber vine, *Cryptostegia grandiflora* was studied using probit analysis. Herbicide toxicity decreased in the order picloram>triclopyr>dicamba>2,4-D>2,4,5-T>MCPA>fenoprop>dichlorprop.

INTRODUCTION

Picloram, dicamba and 2,4-D are effective in controlling rubber vine, *Cryptostegia grandiflora* (9). 2,4,5-T and triclopyr are also effective, but less so than might be expected given their effectiveness against other species.

Basal bark treatment with herbicides is a particularly effective way of controlling woody plants (8, 15), because the ester-in-diesel solution gives almost 100% uptake of the herbicide (Harvey, unpublished data) and there is little or not effect of translocation. The aim of this experiment was to determine the relative toxicity of a number of phenoxy herbicides, and picloram, triclopyr and dicamba, to rubber vine, using basal bark treatments to overcome any effects of differential uptake and translocation.

The results were analysed using probit analysis (7), an establihsed technique often used for insecticide studies.

METHODS

A preliminary trial was conducted to determine the useful range of herbicide doses. Subsequently a larger trial was commenced in late October 1982 and terminated in late February 1983.

Over 4,000 plants were grown in the glasshouse until 12 months of age, at which stage approximately 3,500 were used in this experiment. Plants were treated with 10 or 20 *u*L of the following herbicides in diesel distillate:

1. 2,4-D, ethyl ester
2. 2,4,5-T, butyl/isobutyl ester
3. fenoprop, butyl/isobutyl ester
4. triclopyr, butoxyethyl ester
5. dicamba, oil-soluble amine
6. MCPA, iso-octyl ester
7. dichlorprop, iso-octyl ester
8. picloram, iso-octyl ester

Dosage rate were: 1,000, 800, 600, 500, 400, 300, 200, 100, 50, 30 and 10 *u*g a.e./plant, except for picloram which differed only at the lower end of the range, i.e. 100, 90, 70, 50, 30, 10 *u*g/plant.

There was 16 plants/treatment, with two replicates. Control plants were treated with 10 or 20 *u*L diesel distillate without herbicides.

RESULTS AND DISCUSSION

The number of dead plants was recorded, and the data analysed using a probit computer programme held by the Entomology Branch of the Department of Primary Industries.

None of the control (diesel distillate only) plants died. Only two picloram-treated plants survived, so that the picloram data was unsuitable for analysis, and we can conclude only that the LD_{50} value for picloram is less that 10 *ug*.

Results of the analysis are reported below.

Table 1. Fiducial limits, LD_{95} values and slope of the probit line

Herbicide	LD_{95} (*ug*/plant)	5% fiducial limits		Slope ± s.e.
2,4-D	488	365	714	1.71 ± 0.15
2,4,5-T	544	416	771	1.88 ± 0.16
Dichlorprop	1989	1034	6972	0.95 ± 0.16
Fenoprop	690	507	1040	1.62 ± 0.14
Dicamba amine	288	226	393	2.27 ± 0.20
MCPA	667	509	972	1.94 ± 0.19
Triclopyr	59	44	97	2.98 ± 0.47
Picloram[a]	<10	-	-	

[a] Data not analysed

Since, in the field, we are looking to kill >95% of treated rubber vine, we may use the LD_{95} values to determine the order of toxicity of these herbicides. The order of toxicity is then:
picloram>triclopyr>dicamba>2,4-D>2,4,5-T>MCPA>fenoprop>dichlorprop.

The order of toxicity is as found in previous experiments in the glasshouse and the field (8, 9) which indicates that differences resulting from foliar application of these herbicides in the field are not due just to differential uptake and translocation. Rather, uptake and translocation are probably very similar, although it is possible that differential metabolism i.e. the rate at which the different herbicides are metabolised, is responsible for the differences found.

However, analogy with insecticide studies suggests that the differences probably and more properly reflect differences in toxic action at the biochemical level. Very little is known of the biochemical modes of action of these herbicides, but growth responses suggest that changes in nucleic acid and protein metabolism, and metabolic aspects of cell wall plasticity are primary mechanisms of action (6, 11). The phenoxyalkanoic acid herbicides also act as uncouplers and inhibitors of oxidative phosphorylation (12, 13).

2,4-D affects plant cell membranes (10, 14), and the work by Brian (2, 3, 4) and Brian and Rideal (5) on herbicide resistance by adsorption to non-specific sites indicates that adsorption to plant cell membranes is central to the expression of herbicide toxicity, either by reaction with the membrane itself or, perhaps more likely, by attachment to a specific protein embedded in the membrane.

This experiment does not prove, but it is likely, that the order of toxicity is determined by the strength of interaction with some unknown receptor.

Of the herbicides tested, picloram is the most effective arboricide (1), while the 2,4,5-trichlorophenoxy herbicides (2,4,5-T, fenoprop) are usually more effective than their 2,4-dichloro-analogues (2,4-D, dichlorprop) against woody plants, particularly so in the genus *Eucalyptus* (1, 16). There are, however, exceptions (woody plants that are more susceptible to 2,4-D that 2,4,5-T) of which rubber vine is confirmed as an example, as are guava, *Psidium guajava*, (15), groundsel bush, *Baccharis halimifolia*, and lantana.

With the notable exception of 2,4-D and 2,4,5-T, the order of herbicide toxicity is what would be expected against many woody plants. This indicates that the results obtained here are of much wider applicability than to rubber vine alone.

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