

INSECTICIDE RESISTANCE IN FIELD-COLLECTED COTTON APHID.

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Abstract

There is speculation that current secondary pests of cotton may be more troublesome following the introduction of transgenic cotton. There is concern that increased sprays targeted at the secondary pests, such as cotton aphid, may exacerbate resistance. To quantify resistance in cotton aphid eleven strains were collected from Qld, NSW, NT and WA and tested. Carbamate and organophosphate resistance was detected in strains from Qld, NT and WA but not NSW. Profenofos resistance was low (max. 4.7x), omethoate resistance high (max. 42.1x) and pirimicarb resistance extreme (max. 1,736x). Low level endosulfan (max. 1.4x), and pyrethroid resistance (max. 4.1x esfenvalerate) was detected in one NSW strain. The implications of the findings of this continuing study are discussed.

Introduction

Cotton aphid is a worldwide pest of many plant species including cotton (Blackman and Eastop, 1984). It is the main aphid pest of cotton throughout the world causing significant problems in Thailand, The Sudan, USSR and the USA (Schepers, 1989). In Australia, cotton aphid has not as yet caused wide spread problems. However, reports of field control problems against cotton aphid, with chemicals usually used for its control, continue to be reported. Recent investigations into the resistance status of Australian cotton aphid confirmed the presence of pyrethroid and endosulfan resistant aphids. (Herron *et al.*, 1996)

Overseas studies have found cotton aphid resistant to organophosphate, pyrethroid and carbamate insecticides. In Hawaii, where cotton aphid is a major pest of cucurbits, resistance levels to the organophosphate insecticide oxydemeton-methyl were >2,000x (Hollingsworth *et al.*, 1994). Kerns and Gaylor (1992) found organophosphate (80x) and pyrethroid (50x) resistance in cotton aphid from cotton fields in Texas and Alabama. O'Brien *et al.*, (1992) found carbamate and organochlorine resistance in cotton aphid from Mississippi and Tang (1992) found 1000x resistance to deltamethrin in cotton from China. Recently, Chinese researchers documented 16492x deltamethrin, 1901x fenvalerate, 2068x monocrotophos, 297x parathion, 1310x omethoate, 29x methomyl, and 3x aldicarb resistance against cotton aphid (Guilin *et al.*, 1997). Such high level resistance invariably leads to complete control failure.

With the introduction of Ingard® cotton there is expected to be an overall reduction in insecticide use (Wilson, 1996). The reduction in insecticide use is likely to cause an increase in secondary pests such as aphids. Secondary pest problems are especially expected where growers favour the 'sprayed refuge' option, compared to the smaller but less profitable 'unsprayed refuge' option (Wilson, 1996).

Pyrethroid and endosulfan resistance have now been detected in Australian cotton aphid (Herron *et al.*, 1996), but the abundance of resistance and the resistance range within field collected populations is not known. Carbamate and organophosphate resistance still have not been detected (Herron *et al.*, 1996).

The aims of this still continuing project are:

1. To generate data for field-collected strains of cotton aphid against endosulfan bifenthrin, esfenvalerate, deltamethrin pirimicarb, dimethoate and profenofos using a discriminating-dose technique
2. To conduct full log-dose assays on 'suspect resistant' strains that do not show 100% mortality at the discriminating-dose.
3. Establish additional base-line data.

MATERIALS & METHODS

Strains tested

A total of eleven strains were collected from Queensland, New South Wales, the Northern Territory, and Western Australia. Strains BAN (Bancary Narrabri 17/4/97), ELL (Ellengerah Macquarie Valley 17/4/97), MOR (Moree 17/4/97), KU1 (Kununurra W.A. 18/7/97), KU2 (Kununurra W.A. 11/9/97), WAV (Waverly N.S.W. 23/4/97), BOW (Bowen Qld. 2/9/97) and KAT (Katherine D.P.I., N.T. 16/9/97) were from cotton. However, strain BER (Berrimah N.T. 12/8/97) was from cucumbers and the two 'susceptibles' ADE-sus (Adelaide 6/3/97) and JAB-sus (Jabiru N.T. 2/4/97) were collected from hibiscus in home gardens.

Products tested

Products tested included: the organochlorine endosulfan, the pyrethroid bifenthrin, the pyrethroid esfenvalerate, the pyrethroid deltamethrin, the carbamate pirimicarb, the organophosphate dimethoate and the organophosphate profenofos. All chemicals were commercially available formulations of the product.

Bioassay method

Aphids were tested using methods similar to that described in Herron *et al.*, (1996a) for western flower thrips. Briefly that required excised cotton plant leaf disks to be placed onto cooling liquid agar in a Petri dish. When the disks had fully cooled and set, batches of aphids were transferred to the Petri dishes and then sprayed with aqueous insecticide emulsions by Potter spray tower. Petri dishes were then covered with clear cling wrap and mortality was assessed after 24 h. The discriminating-dose sprayed was the calculated LC99.9 value of cotton aphid susceptible 'A' detailed in Herron *et al.* 1996. Full log-dose tests were subsequently done on each strain that had survivors at the discriminating-dose and LC50 and LC99.9 values calculated using Probit 5 for Windows (Gillespie, 1995). The two 'susceptible' strains, collected from garden yards, were subjected to full log-dose tests only. Resistance factors were calculated as the ratio of the LC50 / 99.9 of the field-collected strains (including the two from garden yards) by the LC50 / 99.9 of susceptible 'A'.

RESULTS

Profenofos, omethoate and pirimicarb resistance were not detected in any NSW samples (Table 1). In contrast, endosulfan, deltamethrin, bifenthrin and esfenvalerate resistance was restricted only to NSW and strain BAN (Table 1). Profenofos, endosulfan, deltamethrin, bifenthrin and esfenvalerate resistance was low order with little difference in response between strains (Table 2). High level omethoate resistance was evident in strains KU1 (LC99.9 level only), KU2 and KAT (Table 2). Extreme pirimicarb resistance was evident in strains KU2 and KAT at the LC50 level but was seen in all strains tested at the LC99.9 level (Table 2).

DISCUSSION

There is a clear dichotomy of response between strains collected from NSW and other than NSW (Table 1) and we think it likely these differences are related to overall insecticide use. We consider supporting evidence is apparent in the response of strains KU1 and KU2 to omethoate. Strain KU2 received at least two additional field applications of dimethoate compared to KU1. Resistance at the LC50 level consequently increased from 0.8x to 19.4x after the additional pesticide applications (Table 2). Additionally, the extra dimethoate selections have made strain KU2 more homogeneous for resistance (evidence by higher slope). However, paradoxically it reduced the LC99.9 estimate (Table 2).

There is high level organophosphate and pirimicarb resistance evident (Table 2), a finding

Table 1. Percent control corrected mortality at the discriminating-dose for nine field collected strains of cotton aphid collected from three Australian states (refer to 'strains tested') and tested against seven pesticides.

Strain:	BAN	ELL	MOR	KU1	KU2	WAV	BER	BOW	KAT
Origin:	NSW	NSW	NSW	WA	WA	NSW	NT	QLD	NT
profenofos	100	100	100	91.9	ca.90	100	71.9	99.1	66.7
omethoate	100	100	100	89.1	ca.40	100	70.8	72.7	0.9
pirimicarb	100	100	100	80.9	ca.0	100	55.7	81.7	0.8
endosulfan	97.8	100	100	100	100	100	100	100	100
deltamethrin	98.1	100	100	100	100	100	100	100	100
bifenthrin	83.8	100	100	100	100	100	100	100	100
esfenvalerate	95.1	100	100	100	100	100	100	100	100

Table 2. Dose responses for profenofos, omethoate, pirimicarb, endosulfan, deltamethrin, bifenthrin and esfenvalerate against field collected resistant strains and 'susceptibles' ADE-sus and JAB-sus.

chemical	Strain	slope	LC50 g ai / L	RF	LC99.9 g ai / L	RF
profenofos	ADE-sus	2.7	0.0036	1.0x	0.050	2.3x
	JAB-sus	3.0	0.0040	1.1x	0.043	1.9x
	KU1	2.1	0.0033	0.9x	0.094	4.3x
	KU2	2.9	0.0095	2.6x	0.11	5.0x
	BER	2.8	0.0027	0.7x	0.034	1.5x
	BOW	4.0	0.0034	0.9x	0.020	0.9x
	KAT	5.5	0.017	4.7x	0.061	2.8x
omethoate	ADE-sus	2.5	0.0017	2.4x	0.028	2.5x
	JAB-sus	2.6	0.0011	1.6x	0.016	1.5x
	KU1	1.1	0.00091	0.8x	0.45	41.8x
	KU2	2.3	0.013	19.4x	0.31	27.8
	BER	1.1	0.00076	1.1x	0.41	37.3x
	BOW	1.3	0.0012	1.7x	0.25	22.7x
	KAT	2.6	0.029	42.1x	0.45	41.8x
pirimicarb	ADE-sus	3.7	0.0014	1.3x	0.0098	1.1x
	JAB-sus	3.4	0.0016	1.4x	0.013	1.4x
	KU1	0.5	0.00026	0.2x	135.07	14,846x
	KU2	2.1	0.71	645x	21.21	2,329x
	BER	0.7	0.00043	0.39x	17.45	1,868x
	BOW	0.7	0.0013	1.2x	36.95	4,066x
	KAT	2.3	1.91	1,736x	40.69	4,471x

endosulfan	ADE-sus	3.7	0.059	1.8x	0.39	1.1x
	JAB-sus	4.4	0.037	1.2x	0.19	0.5x
	BAN	2.3	0.048	1.4x	1.11	3.1x
deltamethrin	ADE-sus	2.3	0.00037	1.5x	0.0081	3.1x
	JAB-sus	2.7	0.00021	0.9x	0.0027	1.0x
	BAN	1.7	0.00034	1.4x	0.022	8.5x
bifenthrin	ADE-sus	2.5	0.00017	2.0x	0.0030	5.7x
	JAB-sus	3.1	0.00024	2.7x	0.0023	4.3x
	BAN	3.0	0.00020	2.2x	0.0022	4.2x
esfenvalerate	ADE-sus	3.1	0.00018	1.6x	0.0018	1.0x
	JAB-sus	3.1	0.00018	1.6x	0.0018	1.0x
	BAN	2.22	0.00049	4.1x	0.012	6.7x

which is consistent with overseas studies. Overseas studies have identified organophosphates generally, and pirimicarb specifically, as developing high level resistance in cotton aphid (Moore *et al.*, 1996; Delorme *et al.*, 1997). Those studies identified insensitive acetylcholinesterase (AChE) as the major mode of action for resistance with little detoxification evident. Interestingly, low level pyrethroid resistance in cotton aphid is caused by detoxification but high level resistance is also thought due to a target site insensitivity (Han *et al.*, 1995).

These findings have implications for Australian cotton aphid resistance management. If the major underlying mechanism is insensitive AChE then organophosphate and carbamate insecticides are at risk, a point supported by our data (Table 2). Consecutive sprays of organophosphates or pirimicarb for cotton aphid control should be avoided and the number of sprays per season restricted. Results to date suggest profenofos would be a good first organophosphate choice to use against cotton aphid because resistance is currently low in all areas (Table 2). Pirimicarb can still be used in NSW but resistance has the potential to develop to an extreme level. Pyrethroids are currently not recommended for cotton aphid control, however, high level pyrethroid resistance has already been detected in our earlier study (Herron *et al.*, 1996). Cotton aphid probably endures high endosulfan and pyrethroid selection as there is high usage of these groups against the major cotton pest, *Helicoverpa* spp. Consequently, pyrethroids may not be a viable option for cotton aphid control if high-level organophosphate and carbamate resistance becomes more abundant. Therefore, any new study on cotton aphid should investigate alternative chemistry for cotton aphid control.

The BER strain was collected from cucumbers and not from cotton. However, it possessed a number of profenofos, omethoate and pirimicarb resistant aphids (Table 1). If crops that support cotton aphid, such as cucumbers or melons, are grown in the vicinity of cotton

immigrant aphids may have a deleterious effect on the cotton. If resistance is selected in cotton aphid in the non-cotton crop, and the resistant aphids find their way into the cotton, the aphids could be further selected causing control failures.

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