FIELD EVALUATION OF AN INSECTICIDE MANAGEMENT STRATEGY FOR THE CONTROL OF PYRETHROID RESISTANT HELIOTHIS ARMIGERA

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Introduction

In response to the development of resistance to pyrethroids by Heliothis armigera in 1983, an insecticide management strategy was devised in an attempt to prolong the field effectiveness of this and indeed, the other groups of insecticides used against H. armigera on summer crops in Australia. Basically, the strategy allows the use of pyrethroids on only one generation of the four or five generations which can occur during the season. Control at other times relies on the rotation of unrelated chemical groups (e.g. organophosphates, carbamates, endosulfan etc.). The strategy was first introduced in the 1983/84 season and was closely monitored in the intensive cotton growing areas of the Namoi and lower Gwydir valleys. This paper deals with the acceptance of the strategy in that area and the monitoring results for the first season.

Methods

Eggs were collected off cotton in the study area (see Figure 1.) and treated as depicted in Figure 2. Extensive testing on susceptible H. armigera (20 strains) has indicated that the fenvalerate doses chosen for screening resistance (0.2 micrograms/30-40 mg larva and 0.5/40-60 mg larva) give 98.8% and 99.8% mortality of susceptible larvae, respectively and thus can be considered as satisfactory discriminating doses. Insecticide use patterns for the study area were obtained from information supplied by both consultants and growers.

Results and Discussion

Acceptance of the strategy was excellent with no pyrethroids being used outside of Stage II (see Table 1.). The main products used in Stage I were endosulfan and profenophos; in Stage II (pyrethroids, profenophos and endosulfan) and in Stage III (profenophos and sulprophos). Ovicide use was quite substantial with all products but particularly so with endosulfan and profenophos (see Table 2). Approximately 90% of the pyrethroids used in the study area were of the structurally closely related cypermethrin/

deltamethrin group (see Table 3).

The proportion of resistant individuals remained constant at about 9% between Stages I and II but increased to approximately 15% in Stage III (see Table 4). This latter increase would be no doubt due to pyrethroid selection in the previous generation in Stage II. The fact that resistance levels did not drop between Stages I and II is disappointing as some reduction was anticipated due to the lack of pyrethroid selection pressure in Stage I (under laboratory conditions, resistance levels nearly always drop back to almost susceptible levels in one to two generations, if left unselected).

The degree of resistance of the survivors of the discriminating dose (SELECTED) showed an interesting trend (see Table 4) with very low resistance in Stage I, moderate resistance in Stage II and higher resistance in Stage III (deltamethrin only). Although resistance to fenvalerate did seem to increase in stage III as well, it was just short of statistical significance. The increase in the degree of resistance to deltamethrin in Stage III would be no doubt due to pyrethroid selection in the previous generation in Stage II. As mentioned earlier, most of the pyrethroids used in Stage II were of the cypermethrin/deltamethrin group and as indicated by our laboratory screening studies selecting with this group may be expected to preferentially increase cypermethrin/deltamethrin resistance levels compared to those of fenvalerate. This may help to explain the differential response of the deltamethrin and fenvalerate resistance factors to pyrethroid selection in Stage II.

The increase in the degree of resistance in Stage II (approximately 6 fold to both fenvalerate and deltamethrin) was unexpected, as there had been no pyrethroid selection pressure in the previous generation in Stage I. This fact, along with the previously mentioned failure for resistance levels to drop between Stage I and II, indicate that some selection pressure has occurred between the two generations.

This may have taken place in a number of ways:-

(1) Pyrethroid selection of adults in Stage II

Pyrethroid sprays applied to larvae in Stage II may have also selected for resistant moths. If this is the case, then egg collections sampled just after a pyrethroid spray in Stage II, should have higher resistance levels than those where no pyrethroids were used. This is not so, even when samples

from smaller properties (where immigration after spraying would be an important source of dilution) have been deleted (see Table 5).

(2) Pyrethroid selection of larvae in Stage II giving rise to resistant moths within Stage II

This seems highly unlikely, particularly as the period chosen for Stage II (42 days) is the shortest possible generation time (eggs to peak egg lay) for H. armigera. However, larvae may have been present at the beginning of Stage II and selected with early Stage II pyrethroid sprays, thus possibly reducing the generation time below 42 days. However, under the temperatures experienced during January and February 1984, it can be clearly seen that moths developed from larvae present on the first day of Stage II, even up to late third instar, would still be only just starting to lay their eggs during the last few days of Stage II (see Table 6). Larvae larger than late third instars would be uncommon in most commercial cotton crops and because of their position in the crop would not probably be effectively dosed anyway.

(3) Cross resistance from endosulfan selection on larvae in Stage I

There is mounting laboratory evidence that endosulfan selection can at least maintain pyrethroid resistance levels and vice versa. However, the practical significance of this to the field situation has been unclear. Endosulfan and the organophosphates (profenophos, monocrotophos, sulprophos) were the only insecticides of any consequence used in Stage I. There has been no evidence of any cross resistance between pyrethroids and organophosphates and so the very heavy use of endosulfan in Stage I must be strongly implicated as the cause of the selection pressure experienced between the generations in Stages I and II.

(4) Immigration of resistant moths from other crops

Non cotton hosts of H. armigera were very limited during the period of Stage I which would have generated Stage II moths (i.e. late Nov - early Jan). These would have been mainly late safflower, early sunflowers and early sorghum and all these crops would have received no pyrethroids and very little endosulfan (see Figure 1.). In fact, immigration from these basically unsprayed crops would be expected to dilute Stage II resistance levels, rather than maintain them.

(5) Immigration of resistant moths from diapause pupae

Diapausing pupae at the end of the 1982/83 season would have been exposed as larvae to pyrethroids and as such could be reasonably expected to have a high proportion of resistant individuals. However many studies have shown that these pupae emerge as moths from early spring to early summer (i.e. September to December). It is highly unlikely that any significant emergence from diapause, of resistant moths would have occurred in Stage II (i.e. January 10 - February 20).

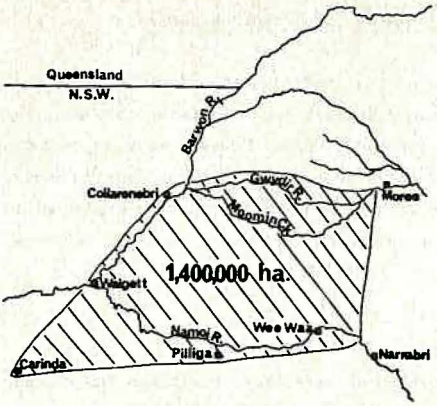
Conclusions

Endosulfan selection in Stage I

- maintained the proportion of pyrethroid resistant individuals
- increased the degree of pyrethroid resistance
- marginally increased endosulfan resistance

Pyrethroid selection in Stage II

- increased the proportion of pyrethroid resistant individuals
- increased the degree of pyrethroid resistance
- maintained endosulfan resistance at a low level.



NAMOI/LOWER GWYDIR
STUDY AREA

CROPPING AREAS 1983/84			
CROP	AREA (ha.)	% AREA TREATED WITH	
		PYR	ENDO
WINTER			
Safflower	1 600	nil	17
Wheat	280 000	nil	
SUMMER			
Cowpeas beans	350	nil	nil
Soybeans	2 700	6	9
Sunflowers	6 000	nil	7
Sorghum	8 000	5	4
Cotton	50 000	235	386
BALANCE			
Native-pasture Fallow			

FIGURE 1

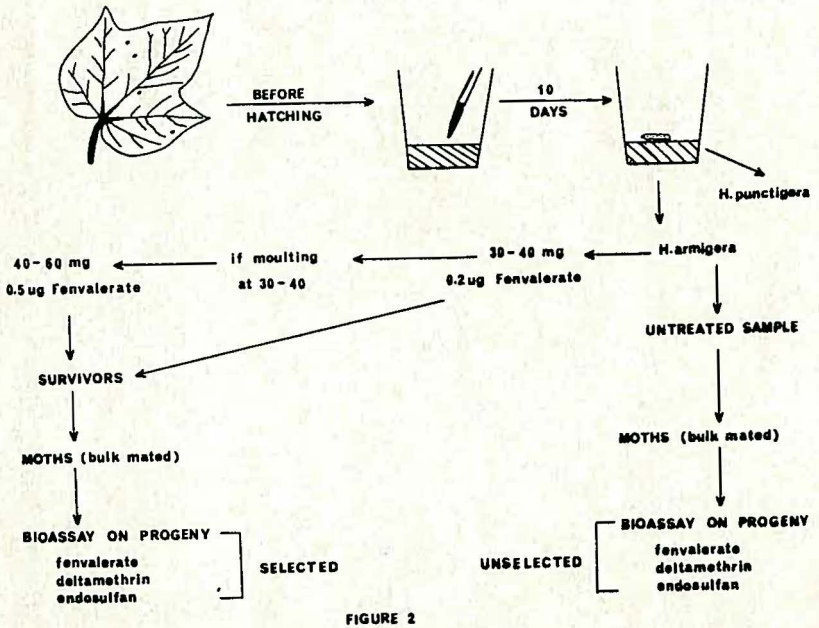


FIGURE 2

TABLE 1: Insecticide use in the Namoi/Lower Gwydir 1983/84 (No. sprays/total cotton area).

	Stage I	Stage II	Stage III	Total
Endosulfan	3.5	0.36	0	3.86
Pyrethroids	0	2.25	0	2.25
Chlordimeform	0.13	0.03	0	0.16
Profenophos	0.51	0.62	1.02	2.15
Monocrotophos	0.25	0.19	0.29	0.73
Sulprophos	0.1	0.03	0.44	0.57
Parathion	0	0.06	0.11	0.17
Chlorpyriphos	0.01	0	0	0.01
Acephate	<0.01	0	0	<0.01
Methomyl	0.03	<0.01	0.02	0.05
Thiodicarb	0	0	0.04	0.04
Dipel, CDM	<0.01	<0.01	<0.01	0.01
TOTAL	4.54	3.55	1.92	10.0

TABLE 2: Ovicide use/ in the Namoi/Lower Gwydir 1983/84 (for each insecticide, % of area treated).

	Alone	+ Chlordimeform	+ Methomyl
Endosulfan	23	56	21
Profenophos	24	76	0.001
Sulprophos	56	41	3
Pyrethroids	56	44	0.001

TABLE 3: Pyrethroid use in the Namoi/Lower Gwydir 1983/84 (% of Area Treated).

Fenvalerate	11
Cypermethrin	33
Deltamethrin	56

TABLE 4: Proportion of Resistant Individuals and their degree of resistance (Namoi/Lower Gwydir 1983/84)

	No.Collections	No.Larvae Tested	% Resistant	Resistance Factors of Survivors' Progeny		
				Fenvalerate	Deltamethrin	Endosulfan
Stage I	50	1207	9.4[a]	0 [a]	0 [a]	0 [a]
Stage II	73	842	8.5[a]	5.3[b]	6.8[b]	2.2[b]
Stage III	29	567	14.9[b]	11.5[b]	18.7[c]	2.1[b]
			Means followed by the same letter, are not significantly different at the 5% level.	Resistance Factors in the same column, followed by the same letter, are not significantly different at the the 5% level.		

TABLE 5: Average % Resistance in Stage II Egg Collections with or without a previous pyrethroid spray.

Farm Size	Pyrethroid Use in the 2-8 nights before egg collection.	
	0	1
Any	9.0[a]	9.5[a]
Over 250 ha	9.0[a]	12.2[a]
Over 500 ha	8.0[a]	10.7[a]

Means in the same row, followed by the same letter are not significantly different at the 5% level.

TABLE 6: Predicted date of first and peak egg lays of moths developed from various larval stages at the beginning of Stage II 1984. (allows 531 and 573 Day Degrees from hatching to first and peak egg lay respectively).

Stage present at Jan 10	Predicted First Egg Lay	Predicted Peak Egg Lay
newly hatched larva	Feb 21/22	Feb 25
late first instar	Feb 20	Feb 23
late second instar	Feb 18/19	Feb 21/22
late third instar	Feb 15/16	Feb 18/19

(Stage II Pyrethroid Sprays from Jan 10-Feb 20.)