The Pyrethroids - How they work and why they fail.

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The pyrethroids have provided spectacular control of the cotton bollworm *Heliothis armigera* and have enabled the production of a high yielding, quality cotton product in Australia. However, intense selection pressure has led to the development of insecticide resistance. The reasons for insecticide resistance are complex and the purpose of this article is to describe how the pyrethroids act on *Heliothis* and to indicate the mechanisms by which *H. armigera* have become resistant.

Insecticides can poison insects in many ways. Insecticides can interfere with metabolic processes, of energy production, cuticular growth and hormone production. Many insecticides, being potent nerve poisons, can stop nervous transmission through the insect nervous system. Resistance is now apparent to most, if not all, types of insecticides and research on the mechanisms of resistance has always been part of the multidisciplinary approach which should be adopted for all cases of insecticide resistance management. To deal with pyrethroid resistant *H. armigera* it is necessary to know exactly why the insects are resistant.

Resistance Mechanisms

Insecticide resistance is usually due to any of three basic resistance mechanisms, they are:

1. Reduced insecticide penetration - some insects can take advantage of peculiarities of cuticular chemistry which can delay or stop insecticide penetration into the insect body.
2. Detoxification - Detoxification is the most common resistance mechanism that protects insects against foreign compounds. Insects use enzymes to convert toxic substances into water soluble excretable metabolities. Resistants can simply produce more enzymes than normal, or synthesise enzymes of slightly differing chemical structures which are more effective.
3. Insensitivity of the nervous system - Most insecticides in use today interfere with the insect nervous system, either chemically or by electrical interference with nerve conductance. Insensitivity of the nervous system target toward insecticides is relatively common. They usually produce intractable resistance mechanisms that produce the highest levels of resistance.

Since 1983 Robin Gunning, Entomologist at the Tamworth Agricultural Research Centre, has been studying the mechanisms of pyrethroid resistance in Heliothis armigera. There have been three independent resistance mechanisms identified, one from each of the categories described above. They are reduced pyrethroid penetration through the cuticle into the Heliothis larva, metabolic breakdown of the pyrethroid compounds, and a change in the nervous system reducing sensitivity to pyrethroid poisoning.

Reduced Pyrethroid Penetration

Using 14C radiolabelled pyrethroids, Robin Gunning and Pesticide Chemist Ian Ferris studied the penetration and distribution of pyrethroids into large resistant and susceptible H.armigera larvae. Findings, (summarised in Figure 1), were that even 4 hours after pyrethroid treatment 55% of the C14 labelled pyrethroid remained outside the bodies of resistant lavae whilst an average of 25% of the dose remained outside the cuticle of pyrethroid susceptible H.armigera. The progeny of resistant susceptible crosses behaved similarly to the resistant parent indicating that the inheritance of this factor was dominant.

Pyrethroid Detoxification

By using known enzyme inhibitors pyrethroid metabolism in H.armigera has been studied. With piperonyl butoxide it was found that pyrethroid resistant H.armigera possessed microsomal oxidase enzymes capable of metabolising and detoxifying the pyrethroids. These resistants are widespread in New South Wales and Queensland.

Nervous system insensitivity

Pyrethroids are recognised as neurotoxicants that act directly on excitable membranes, they cause repititive nerve firing by interfering with axonal transmission in the inseact sensory and central nervous system. Using electrophysiological techniques, which studied the effects of insecticides on nerve action of Heliothis taste receptor hairs (the procedure is summarized in Fig 2), located on the larval mouthparts, it was shown that the nerves of resistant H.armigera were clearly less sensitive to pyrethroids than were susceptibles. Normal nerve firing of a Heliothis taste receptor is shown in Figure 3(a), almost continuous repetitive firing induced by pyrethroid treatment of a susceptible in Figure 3(b) and the lack of nerve response after similar treatment of a pyrethroid resistant H.armigera is shown in Figure 3(c).

As well, the time interval between insecticide treatment and any nerve response showed a clear difference between resistants and susceptibles and probably indicated heteozygous and homozygous resistant genotypes.

The importance of resistance mechanisms in the field

Using the information about pyrethroid resistance mechanisms outlined above, NSW Entomologists Robin Gunning and Neil Forrester have, in the 1987/88 season, assessed resistance mechanism frequency in resistant sprayed and non-sprayed populations. We wanted to see how, as the season progressed, the resistance management strategy affected resistance mechanism frequency and distribution.

The two resistance mechanisms chosen for study were pyrethroid metabolism and nervous insensitivity toward the pyrethroids (known as the "Kdr Factor"). The presence of microsom al pyrethroid detoxifying enzymes was monitored using the enzyme inhibitor piperonyl butoxide, whilst H.armigera having the Kdr factor were detected via electrophysiological techniques.

In this paper, data on the distribution of the nerve insensitivity mechanism in various pyrethroid resistant populations are presented (Figure 4). There were two unsprayed areas, Inverell and the NSW North Coast/Hunter Valley; and two pyrethroid sprayed populations, Emerald QLD and the Namoi/Gwydir in NSW. Figure 4 shows in these four populations, the relative frequencies of pyrethroid resistant H.armigera both heterozygous and homozygous for Kdr. Data are presented for stages 1,2 and 3 of the season. Results indicate that there were no differences between stages of the season or between any of the 4 resistant H.armigera populations studied. Approximately 50% of resistants carried the Kdr resistance factor, but only 10-20% were homozygous for this mechanism.

The finding that Kdr, at the moment, is probably not the major cause of pyrethroid resistance in <u>H.armigera</u> is significant because insensitivity of the pyrethroid target site, the nervous system, usually produces an intractable resistance mechanism.

Fig. 1 Penetration of ^{14}c-cypermethrin through the integument of pyrethroid resistant, susceptible and RxS matings of *H. armigera*

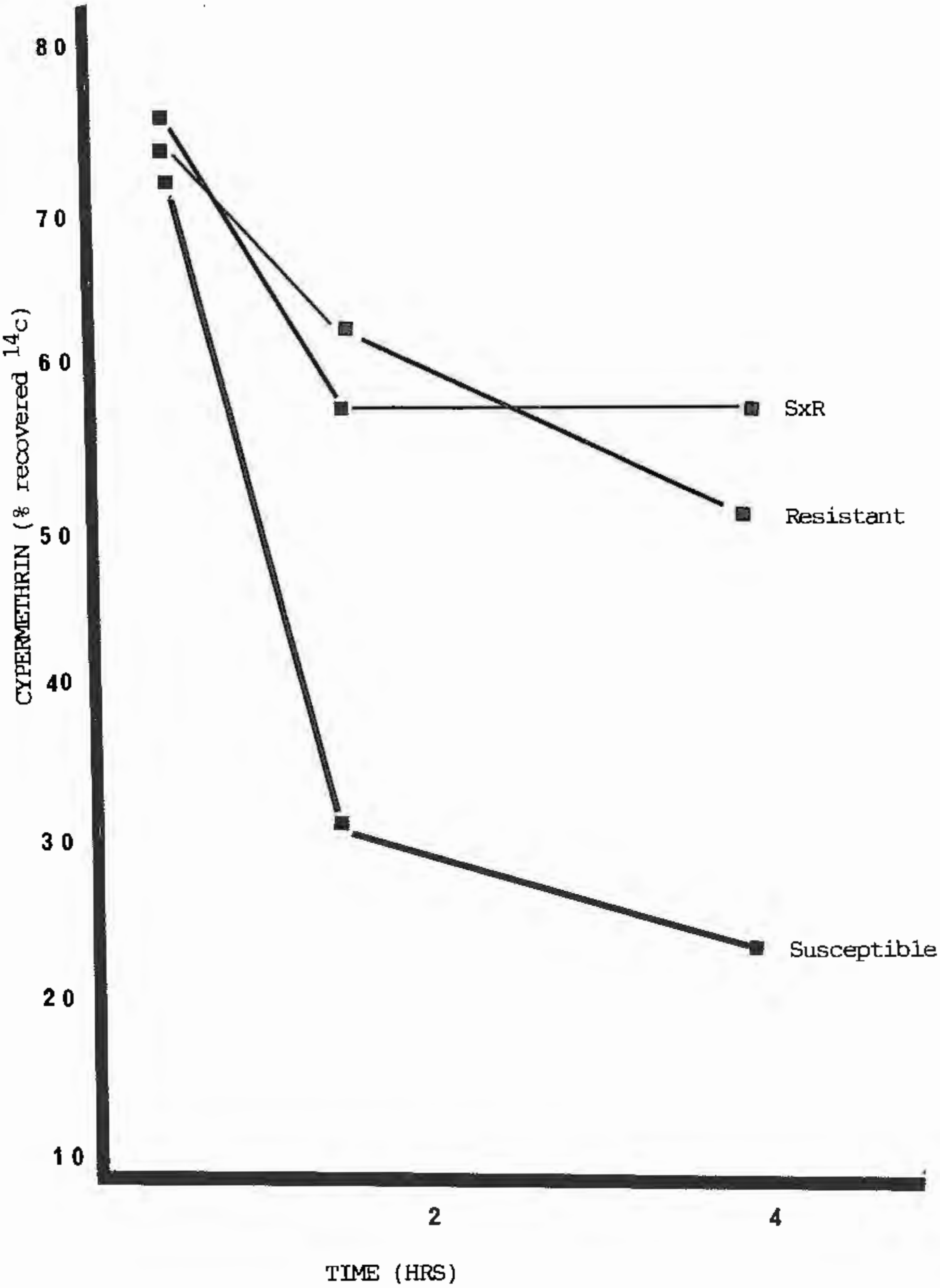
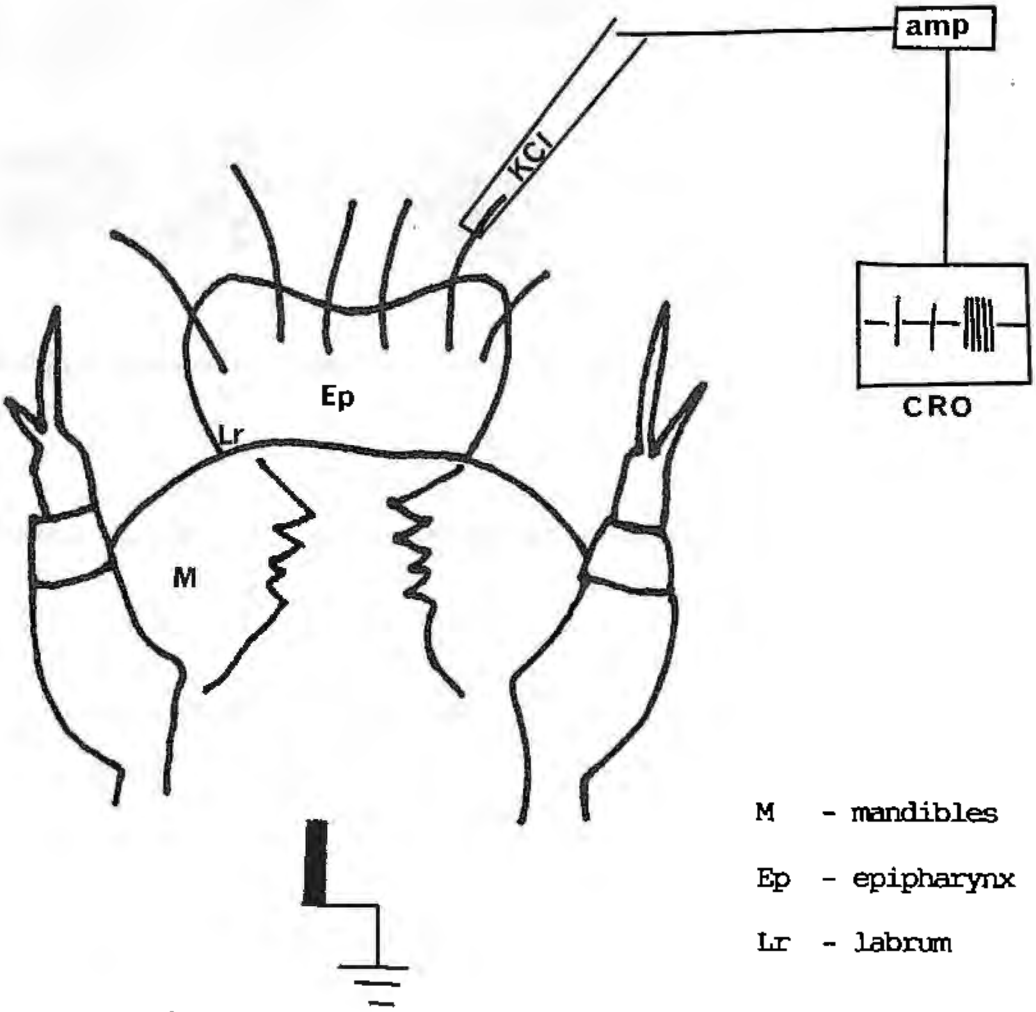


Fig 2 Electrophysiological techniques on *Heliothis* larvae



M - mandibles

Ep - epipharynx

Lr - labrum

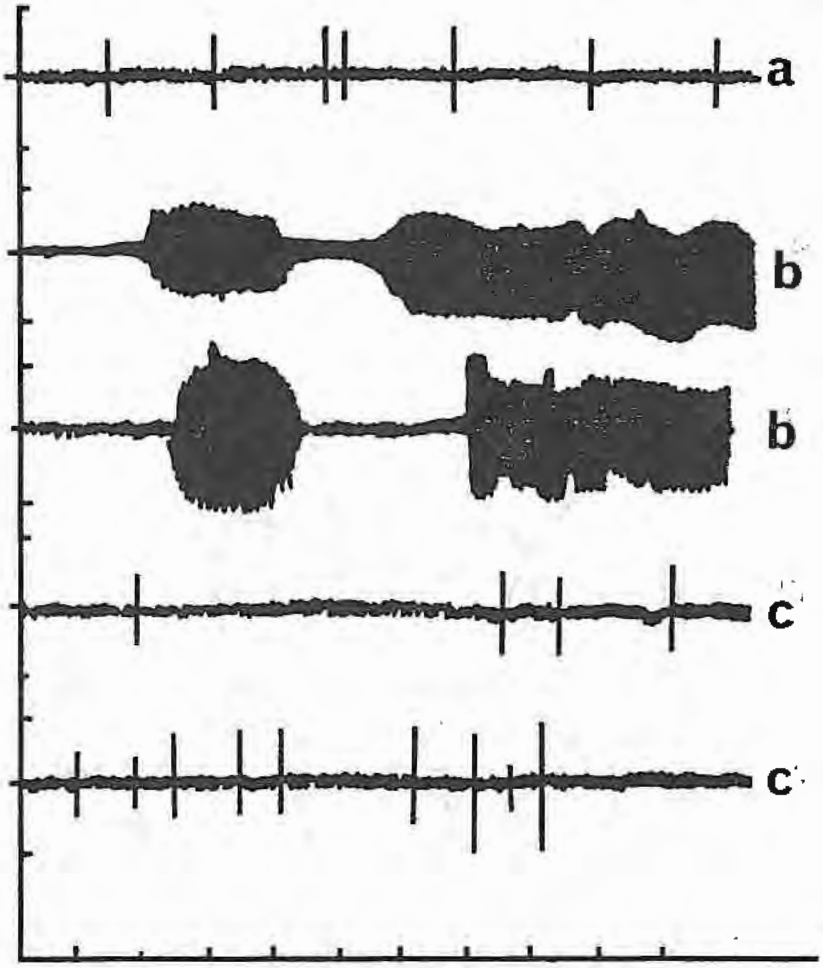


FIG. 3 Nervous activity recorded chemosensory receptors of pyrethroid susceptible and resistant 6th instar larvae of <u>Heliothis armigera</u>

Trace A untreated, susceptible
B pyrethroid poisoned, susceptible
C pyrethroid treated, resistant

Fig 4 Sprayed and non-sprayed pyrethroid resistant *H.armigera* populations tested for Kdr resistance