COTTON RESEARCH COUNCIL

FINAL REPORT ON OVERSEAS TRAVEL 1987/88

1. Project : DAN 31L (Attendance at XVIII International Congress of Entomology, Vancouver, Canada)

2. Organisation : NSW Agriculture & Fisheries

Officer : Mr N.W. Forrester
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3. Itinerary :

July 2, 1988	Sydney-Vancouver
July 3-9, 1988	Vancouver (XVIII International Congress of Entomology)
July 10, 1988	Vancouver-Sydney

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4. Final Report:

The main aim of this trip was to report to the international scientific community on the progress of the Insecticide Resistance Management Strategy that has been implemented in Australia to contain pyrethroid and endosulfan resistance. With this aim in mind, a paper was delivered on the "Field Selection for Pyrethroid Resistance Genes in *Heliothis armigera* in Australia". This paper discussed the importance of the mixed function oxidase gene for field resistance to pyrethroids in Australia.

More detailed notes on the presentations of other researchers at the conference are given in an Appendix.

5. Financial Summary:

Allocation		$2,500
Expenses incurred		
Sustenance	380.84	
Airfares/Internal Transport	2,073.45	
Travel Insurance	49.00	
Others (Exchange Commissions, Departure Tax, Expenses en route, Incidentals etc)	106.75	
	2,610.04	
Balance of Allocation	$0.00	

APPENDIX

Relevant papers presented at the XVIII International Congress of Entomology, Vancouver, CANADA.

TOXICOLOGY, PURE AND APPLIED

Feyereisen (Oregon State University, USA) - Described the isolation and sequence of a cytochrome P-450 mixed function oxidase enzyme from an insecticide resistant housefly. Could be useful for direct detection of mfo genes in field populations.

Hsu and Wei (National University, Taiwan) - Reported the synergism of carbaryl with propynl ether (PE) and piperonyl butoxide (PBO) on resistant houseflies. They showed PE was even better than PBO at lower carbaryl/synergist ratios. Could be a useful synergist for our mfo gene especially since Dr T. Brown (Clemson University, South Carolina) has found synergism with pyrethroid resistant *Heliothis virescens*. Dr Brown has forwarded a sample of his propynl ether compound for testing on our *Heliothis armigera* .

Ioannidis and Grafius (Michigan State University, USA) - Colorado potato beetle has three mechanisms of resistance to pyrethroids:- mfo, esterase and nerve insensitivity, but no studies on the relative importance of these mechanisms in the field.

Mullin and Siegfried (Pennsylvania State University, USA) - Studied the mechanism of resistance of corn rootworm to cyclodienes. No evidence of reduced cuticular penetration or increased metabolism. Probably due to a modification of the GABA-chloride ionophore.

Dowd (USDA/ARS, Peoria, Illinois, USA) - Described the results of the very large USDA screening programme for the insecticidal activity of mycotoxins and other fungal metabolites. Suggested that the fungal metabolite kojic acid is a useful mfo inhibitor for pyrethroids.

Moss (Wellcome, UK) - Compared the temperature response (15-35^{o}C) of pyrethroids and lipid amides on housefly. By 4 days, both pyrethroids and lipid amides (eg. pellitorine) showed strong negative temperature dependence from 15-23^{o}C. Only deltamethrin continued to show such a temperature dependence when the temperature was raised to 35^{o}C.

NATURAL PRODUCTS AS MODELS FOR DEVELOPING INSECTICIDES

Narahashi (University Medical School, Chicago, USA) - Discussed the mode of action of pyrethroids. Synthetic pyrethroids act by keeping the Na gate open. This is done by affecting the opening mechanism and not by blocking the gate. There are approximately 100 Na channels/micron of nerve fibre and only 1% of these need to be modified to poison the animal. Detoxification (eg mfo's) will be higher at higher temperatures. Therefore, try testing resistant *Heliothis armigera* larvae at different temperatures.

Casida (University of California, USA) - Discussed three natural compounds and their synthetic analogues.

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1. Isobutylamides (Lipid amides) -
   - based on products from blackpepper from India (eg, pipericide)
   - synthetic analogues since 1955
   - act on the Na channel in the nerve?
   - photolabile
   - no cross resistance from nerve insensitivity pyrethroid/DDT resistance (in fact, some evidence of negative cross resistance)
   - strong synergism by mfo inhibitors

2. Picrotoxinin
   - isolated from seeds of *Anamirta* (poison berry)
   - act on the Gaba chloride channel (antagonist) as do cyclodienes (eg endrin/toxaphene)
   - same potency as most synthetic pyrethroids
   - highly synergised by PBO
   - positive temperature coefficient
   - selective toxicity to insects over-mammals

3. Ryanodine
   - a synthetic analogue of the natural occurring rotenone (ex *Derris* spp.)
   - muscle poison (modifies Calcium ion transport in muscles)
   - selective toxicity to insects over mammals
   - ester hydrolysis destroys potency
   - has a complex structure
   - synergised by PBO
   - contact action

Matsumura (University of California, USA) - Discussed the mode of action of picrotoxinin. Picrotoxinin resistance gives cross resistance to dieldrin and toxaphene but not to parathion, deltamethrin, chlordimeform or Mirex. Picrotoxinin antagonizes the inhibitory action of the GABA inhibitory transmitter in the nervous system.

Dybas (Merck, Sharp and Dome, USA) - Discussed the development of first and second generation avermectins. Avermectins are produced by fermentation of the soil microorganism *Streptomyces avermitilis*. They were discovered in the mid 70's. Avermectin B1 (abamectin) is a mix of 2 isomers and is used on agricultural pests. It has a wide range of variation in toxicity to insects. Ivermectin has a single bond instead of a double bond as in abamectin. It is used on veterinary pests. Avermectins have a unique action and appear to stimulate Cl ion flow at the GABA receptor. This is in contrast to the antagonistic action of picrotoxinin and cyclodienes (It would be interesting to check for cross resistance to both picrotoxinin and abamectin in our cyclodiene resistant *Heliothis armigera* colony). Abamectin is very active on mites and is more active on *Heliothis virescens* than on *Heliothis zea*.

The addition of an amine group to abamectin yielded the second generation avermectins (epimethyl amino abamectin). These are much more active on lepidoptera but unfortunately are about 15 x less toxic to mites.

Avermectins are translaminar and have long residual within the plant. However, surface residues degrade quickly. Said to be less toxic to predatory mites. Cost $US 30/acre ≡ $A 90/ha. No cross resistance from any current resistances (including pyrethroids and cyclodiene resistance).

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Usherwood etc. - Several papers discussed the possibility of using wasp and spider venoms as models for future insecticides.

Zoebelein (Bayer, West Germany) - Bayer have screened over the last 4 years, 530 plant species from 115 families for activity against lepidoptera, coleoptera, homoptera, diptera and a *Tetranychus* species. The most promising leads so far:-

1. harringtonln - derived from *Cephalotaxus* sp., just as active as pyrethrum on beetles and caterpillars.

2. annonin - derived from *Annona* sp., broad spectrum insecticidal activity.

WHITEFLY SYMPOSIUM

This one day symposium discussed the bionomics, pest status and management of *Bemisia tabaci* and *Trialeurodes vaporariorum*. Both these whitefly species have been recorded from Australia on cotton but neither has reached pest status yet. It is important to attempt to understand why, as *Bemisia tabaci* has the potential to become one of our major pest species in cotton. The proceedings of this symposium will be reproduced in a book.

Mound (British Museum, UK) - When whitefly invades a country, only a portion of the gene pool is introduced. This leads to the possibility of the introduction of specific biotypes which may not be adapted to local host varieties. This may explain the often long periods between the first records of *Bemisia tabaci* in a country and its emergence as a key pest of cotton.

de Ponti (Wageningen, Netherlands) - Host plant resistance to whiteflies has been identified in tomato and in cotton in the Sudan.

Dittrich (Ciba-Geigy, Switzerland) - Gave a good review of insecticide resistance problems with *B. tabaci*. To be published later in the proceedings.

van Lenteren (Wageningen, Netherlands) - Discussed host plant selection and host plant preference in whiteflies.

- whitefly adults feed and lay eggs simultaneously, therefore on hairy leaves, eggs are scattered.
- whitefly react to blue UV and yellow light, no reaction to odour, shape etc (results in polyphagy).
- have a migratory phase (react to low UV light).
- egg plant is a good host for both *Bemisia* and *Trialeurodes*.
- prefer younger leaves (determined by adult probing).
- host plant rejection occurs before phloem is reached (it takes 30 mins to reach the phloem, 1 & half mins in parenchyma tissue).
- stylet amputation (drop on end proves it is in phloem).
- a 10% reduction in development time is equivalent to a 50% reduction in fecundity.

LYGUS BUG

Leigh (University of California, USA) - Cotton growers spray safflower to control Lygus bug in cotton.

RESISTANCE MANAGEMENT SYMPOSIUM

Scott (Cornell University, USA) - There are 3 components of the mixed function oxidase cycle:-

- cytochrome p-450
- cytochrome p-450 reductase
- cytochrome b5

The pyrethroid resistant strain of housefly (Learn) uses all 3 components to resist pyrethroid poisoning. Tefluthrin (a radically different pyrethroid designed for soil insect control blocks mfo resistance in this housefly strain. This concept may be used to produce resistance breaking pyrethroids to manage our mfo dominated field resistance to pyrethroids.

Plapp (Texas A & M, USA) - Put forward the theory that metabolic resistance in houseflies is due to the production of a protein from a gene on chromosome II. This protein binds insecticides and juvenile hormone. This then induces specific detoxifying enzymes. This means that resistant insects can't use juvenile hormone as well as susceptibles. This could affect fecundity of resistant moths which require high levels of juvenile hormone for egg production.

Pasteur (University of Montpellier, France) - Discussed the evolution of organophosphate resistance genes in *Culex pipiens* in France.

- OP use started in 1968.
- First resistance in 1972 (due to a single gene, esterase A1).
- Another gene in 1978 (insensitive cholinesterase).
- 1986/87 a survey showed that the above 2 genes were widespread (80-100% resistance frequency).
- this latter survey also showed 2 other esterases in low frequencies.

Thus, prolonged selection with OP's has successively selected several resistance genes. Unexpectedly, the more recently selected genes provide less efficient protection than the insensitive cholinesterase.

ffrench-Constant (Cornell University, USA) - Described an immunological resistance monotoring scheme for esterases in *Myzus persicae*. Interestingly he showed resistance in this aphid can revert to susceptibility but reselection of these susceptible strains can reselect for resistance. Obviously, the resistance gene is not being expressed at all times.

Sun (National University, Taiwan) - Discussed the management of insecticide resistance in diamondback moth.

- mfos give resistance to both pyrethroids and chitin synthesis inhibitors (however, a lack of cross resistance between these two groups suggests that different mfos are involved).
- suggested 3 mfo synergists (PBO, MGK 264, sulfoxide).

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- PBO synergised almost fully teflubenzuron (no cross resistance to chlorfluazuron yet, although this is expected in time).
- PBO synergises fenvalerate more than deltamethrin.
- there is no cross resistance between mfo synergists.

INSECT PATHOLOGY -

Van Mellaret (Swiss Federal Institute of Technology) - Discussed the mode of action of *Bacillus thuringiensis* delta endotoxins binding to lepidopteran midgut membranes.

- there is a heterogeneity of binding sites in the insect midgut for different strains of *B. thuringiensis* toxins.
- natural *Bacillus thuringiensis* crystals are invariably mixtures of different toxins.

While at Vancouver, I met Dr D.J. Wright from Imperial College. He has been involved in a joint research project on the toxicology of resistance to insecticides in diamondback moth in Thailand. On his return to the UK, he sent me a copy of a report on the meeting of his Joint Research Project held in Bangkok in March 1988. This report on field and laboratory testing of various insecticide groups against diamondback moth is quite disturbing. It indicates field and laboratory resistance to pyrethroids, organophosphates, chitin synthesis inhibitors and now *Bacillus thuringiensis*. Only the recently introduced abamectin gave any measure of control. The results with this product also indicate the potential for future resistance problems. The development of resistance to *Bacillus thuringiensis* in diamondback moth is an important warning for molecular geneticists to heed.