

DAN106C Executive Summary

SUMMARY

a) BACKGROUND OF THE PROJECT

The cotton whitefly *Bemisia tabaci* is a serious pest of fibre, horticultural and ornamental crops world wide. When present in sufficient numbers, it can cause extensive damage through direct feeding, the production of large quantities of honeydew and as a vector of many viruses. Australia has a benign native strain of *Bemisia tabaci*. but recently, a new biotype was identified by Robin Gunning, known as the B-type or poinsettia strain. Overseas, B-type *B. tabaci* is a primary pest on cotton, other vegetable crops (curcubits, tomatoes, rock melons) and ornamentals. This strain is extremely virulent, highly insecticide resistant, adapts to temperate climates and has a host range of over 500 plants. A nation-wide survey has now shown that this whitefly is widely distributed over eastern Queensland and NSW and the Darwin area of the NT (Fig 1).

The spread of this whitefly is expected to result in it becoming a major pest in Australia and a primary pest of cotton. B-biotype *B. tabaci* will effect field crops (such as cotton, maize, lucerne and sunflowers); field grown vegetables (curcubits, cole crops, melons, tomatoes); glasshouse vegetables; fruit crops (grapes) and glasshouse ornamentals (poinsettias, hibiscus, geberas and gloxinia).

Insecticides will be the primary weapon against this insect in Australia. B -type *B. tabaci* are resistant to many insecticides overseas and this insect may be very difficult to control. However, there are new insecticides to which this whitefly may still be susceptible. It is essential that the use of all chemicals is carefully managed to minimise or avoid resistance problems. This can only be achieved by establishing effective resistance detection and monitoring techniques and understanding the underlying mechanisms of resistance.

b) PROJECT OBJECTIVES:

- To use standard bioassay techniques to establish the particular resistance / susceptibility profiles of Australian native and B-biotype *B. tabaci*. Insecticide tested, will be organophosphates, carbamates, pyrethroids, newer pyrethroids, cyclodienes, novel control agents.
- Evaluation the mechanisms of resistance to organophosphates, carbamates and pyrethroids in B-biotype *B. tabaci*.
- Assess the applicability of rapid biochemical resistance monitoring techniques to *B. tabaci*.
- To investigate the relationship between B-biotype characteristics (silverleaf induction and B esterase bands) and insecticide resistance in B-biotype *B. tabaci*, will be investigated.
- Assessment of the potential of B- type *B. tabaci* to interbreed with and thus spread insecticide resistance into native *B. tabaci*.
- To investigate the relationship between "resistance status" and field control of resistant *B. tabaci*.
- Resistance management strategies will be devised and tested in the field and the laboratory.

The objectives of this project have largely been achieved or will shortly be accomplished. This is in spite of difficulties experienced because of a greatly increased workload associated with the *Helicoverpa* resistance monitoring programme.

c) RESULTS

(i) Distribution of B-biotype *B. tabaci* on cotton

Whiteflies were found on cotton in all principal cotton growing areas most NSW and Queensland and it is obvious that numbers of B-biotype *B. tabaci* on cotton in Australia is increasing, season by season. By 1998/99, approximately the number of whiteflies recovered, had increased approximately 15 times. Emerald was identified as an area of particular concern.

(ii) Insecticide bioassays

Bioassays confirm that B-type *B. tabaci* entered Australia with resistance to most pyrethroids (permethrin, cypermethrin, deltamethrin and es-fenvalerate), organophosphates (profenofos, methyl parathion, methamidphos, fenthion, sulprofos, and dimethoate) and carbamates (methomyl, methiocarb). B-type *B. tabaci* were, however, initially susceptible, or virtually susceptible, to endosulfan, bifenthrin, imidacloprid and amitraz but field use of these products on horticultural crops in Queensland has resulted in resistance selection. In particular, high levels of resistance to endosulfan, imidacloprid, and bifenthrin have been recently recorded in Queensland field populations. Native, non-B type *B. tabaci* are susceptible to all insecticides tested.

(iii) Field selection of insecticide resistance

Results show a very rapid build up of resistance, in response to selection at Ayr and Bowen. Resistance levels in unsprayed populations were low, however, application of bifenthrin and imidacloprid dramatically increased the resistance factor to 714 and 205 fold respectively (and 6 commercial applications of imidacloprid on Okra by a farmer) increased the resistance factor to over 500 fold). Amitraz selection increased the resistance factor from 2 to 22 fold.

iv) Organophosphate and carbamate resistance mechanisms

Studies of organophosphate resistance in B-type *B. tabaci* has shown that resistance is effectively monogenic, conferred by a single insensitive target site acetylcholinesterase (AChE), with differing levels of insensitivity to organophosphates tested. This resistant AChE is diagnosed by insensitivity to paraoxon or methyl paraoxon.

(v) Pyrethroid resistance mechanisms

Pyrethroid resistance in B-biotype *B. tabaci*, is largely to be as a result of metabolism by the B band esterase isoenzyme which may sequester and hydrolyse pyrethroids. B-type *B. tabaci* are resistant to most pyrethroids in Australia.

(vi) Bifenthrin resistance mechanism

In Australia, B-biotype *B. tabaci* were initially susceptible to bifenthrin, however, field use of bifenthrin has resulted in the selection of resistance. Bifenthrin resistant, B-biotype *B. tabaci* have developed a novel form of B band esterase, which binds more readily to bifenthrin than the susceptible form of the enzyme.

(vii) Interbreeding of native non-B type *B. tabaci* and B-biotype *B. tabaci*

While these experiments are as yet, incomplete, interesting data have been obtained. It is obvious that B-biotype *B. tabaci* readily interbreed with native non-B type *B. tabaci*. We are investigating whether insecticide resistance can be spread into the populations of native *B. tabaci*.

(ix) Insect growth regulators and juvenile hormone analogues

Insect growth regulators and juvenile hormone analogues, have been used successfully overseas against B-biotype *B. tabaci*. These insecticides act by disrupting the insect moulting process and were initially considered to be almost resistance proof. While B-biotype *B. tabaci* were initially susceptible to buprofezin and fenoxycarb in Australia, there has been no difficulty in the laboratory selection of resistant strains, within 2 generations. Buprofezin resistant B-biotype *B. tabaci* have extra esterase isoenzymes, (not occurring in susceptible insects), which appear to bind to and metabolise buprofezin