Pest Manag Sci 61:577–582 (2005)
DOI: 10.1002/ps.1029

Insecticide resistance in *Bemisia tabaci* (Homoptera: Aleyrodidae) populations from Crete

Emmanouil Roditakis,* Nikos E Roditakis and Anastasia Tsagkarakou

Plant Protection Institute of Heraklio, National Agricultural Research Foundation, 71003 Heraklio, Greece

Abstract: The resistance levels to α-cypermethrin, bifenthrin, pirimiphos-methyl, endosulfan and imidacloprid were determined in *Bemisia tabaci* (Gennadius) from Crete. Five *B tabaci* populations collected from greenhouse and outdoor crops were bioassayed and compared with a reference susceptible strain. *Bemisia tabaci* collected in a floriculture greenhouse exhibited the highest resistance against all insecticides: at LC_{50}, resistance factors were 23-fold for bifenthrin, 80-fold for α-cypermethrin, 18-fold for pirimiphos-methyl, 58-fold for endosulfan and 730-fold for imidacloprid. A population collected on outdoor melons was more susceptible than the reference strain against all insecticides tested, suggesting the occurrence of local highly susceptible *B tabaci* populations in 'refugia'. In pairwise comparisons of resistance levels, correlation was observed between the LC_{50} values of the pyrethroid insecticides bifenthrin and α-cypermethrin.

Keywords: insecticide resistance; *Bemisia tabaci*; imidacloprid; endosulfan; bifenthrin; α-cypermethrin; pirimiphos-methyl; Greece

1 INTRODUCTION

The sweet potato whitefly *Bemisia tabaci* (Gennadius) is one of the most damaging pests of numerous crops world-wide. It is considered to be a highly cryptic species complex, with 24 described biotypes.[1] Differences in host range, host plant adaptability, induction of phytotoxic reactions, insecticide resistance and virus-transmission capabilities between biotypes have been recognized.[2–7] It has been shown to transmit 111 virus species, some of which are of high economic importance.[7]

In the island of Crete (southern Greece; Lat: 35N; Long: 25E; Fig 1) more than 2000 ha of vegetable crops are cultivated in greenhouses. *Bemisia tabaci* (non-B biotype) was recorded for the first time in Crete in 1992[8] although earlier records exist for the mainland Greece.[9] From a secondary pest in the early 1990s, *B tabaci* gradually became a very harmful pest for greenhouse tomato, cucumber and eggplant crops as well as field crops (beans).

In 2000 a sudden outbreak of Tomato yellow leaf curl virus (TYLCV, Israeli species) transmitted by *B tabaci* was first reported on greenhouse tomatoes in the region of Ierapetra (south Crete).[10] Soon, TYLCV infections were reported from greenhouses all over Crete and continental Greece, causing substantial crop losses. Growers relied on intensive use of insecticides to control *B tabaci* and minimize viral spread. However failures to control the pest have been reported, probably due to the development of insecticide resistance. Resistance to approximately 35 active ingredients has been reported for *B tabaci* in at least 20 countries world-wide.[11–23]

The aim of this study was to determine whether populations of *B tabaci* from Crete were resistant to five insecticides commonly used to control whiteflies and other pests in Crete. Here we present results of toxicological bioassays for the major insecticide groups currently used in whitefly control, ie pyrethroids, organophosphates, cyclodienes and neonicotinoids.

2 MATERIALS AND METHODS

2.1 Whiteflies

The susceptible reference strain SUD-S initially collected on cotton (*Gossypium hirsutum* L) in Sudan in 1978 was obtained from IACR Rothamsted, UK, where it has been maintained in the absence of insecticides for the past 25 years. In our laboratory SUD-S was maintained on cotton plants at 25 (±1) °C, 50–60% RH and a photoperiod 16:8 h light:dark.

Five *B tabaci* populations from Crete were bioassayed: GR-IERE, GR-IERB, GR-IERC, GR-EPI and

* Correspondence to: Emmanouil Roditakis, Plant Protection Institute of Heraklio, National Agricultural Research Foundation, 71003 Heraklio, Greece
E-mail: e-roditakis@her.forthnet.gr
Contract/grant sponsor: Prefecture of Lassithi, Crete Greece
(*Received 9 June 2004; revised version received 6 October 2004; accepted 9 November 2004*)
Published online 15 February 2005

 Pest Manag Sci 1526–498X/2005 /$30.00

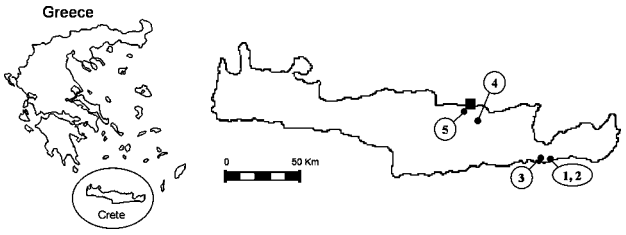

Figure 1. Whitefly collection sites, dates and host plants: (1) GR-IERE; Ierapetra, experimental greenhouse, June 2002, eggplants, (2) GR-IERB; Ierapetra, biological control greenhouse, November 2002, eggplants, (3) GR- IERC; Ierapetra, conventional control greenhouse, July 2002 eggplant, (4) GR-EPI; Episkopi, outdoor crops, August 2003, melons, (5) GR-MAL; Malades, greenhouse ornamentals, August 2002, *Hibiscus* sp and *Lantana camara.* Heraklio city as dark square. Map on the left: the island of Crete in relation to mainland Greece.

GR-MAL (Fig 1). Populations GR-IERE, GR-IERB and GR-IERC were collected from greenhouses in Ierapetra, the main greenhouse area located in south-eastern Crete. GR-IERE was collected on eggplants (*Solanum melongena* L) from an experimental greenhouse of the National Agricultural Research Foundation on 14 June 2002, the end of the greenhouse crop season. In this greenhouse a total of seven insecticide applications had been made (1× pirimiphos-methyl, 1× imidacloprid, 2× bifenthrin, 2 × α-cypermethrin and 1× acrinathrin) during the eight-month cropping season. A second *B tabaci* sample (GR-IERB) from Ierapetra was collected on greenhouse eggplants on 26 November 2002, the beginning of the greenhouse crop season. In this greenhouse, biological control based on predation and parasitism has been practiced for the past 3 years. The botanical insecticide rotenone was applied twice prior to collection. GR-IERC was collected on 2 July 2002, the end of the greenhouse crop season, on eggplants from a greenhouse in which pest control was heavily based on chemical insecticides: approximately 30 insecticide applications were conducted during the eight-month cropping season. GR-EPI was collected in the locality of Episkopi, from outdoor melon crops (*Cucumis melo* L). The field had not been treated with insecticides prior to collection on 20 August 2003, the middle of the field-crop season. Episkopi is located in central Crete, where the main crops are vine and olive trees. A few outdoor vegetables are also cultivated. GR-MAL was collected in the locality of Malades from greenhouse ornamentals (*Hibiscus* sp and *Lantana camara* L) on 9 August 2002. Plants were maintained in the greenhouse round the year. A three-month period of intensive insecticide use (approximately 20 applications with pyrethroids, neonicotinoids, organophosphates and carbamates) that failed to control the pest had been reported prior to collection. Malades is located in the north-central Crete where the main crops are vine and olives.

Once a collection site had been located, samples of adults and leaves infested with whitefly puparia were collected for species identification using the key by Martin *et al*.[2] For the toxicological tests, adult whiteflies were collected in the early morning hours using a mouth aspirator into large pots with ventilated lids modified to fit the aspirator. Insects were collected from at least ten different sampling spots at each site. Whiteflies were transported to the laboratory in a cool box and used within a few hours after collection for the toxicological tests.

2.2 Insecticides

The insecticides used in the bioassays were: the pyrethroids α-cypermethrin 100 g litre^{-1} EC (Glexor; Hellafarm, Athens, Greece) and bifenthrin 100 g litre^{-1} EC (Talstar; FMC, Philadelphia, PA); the organophosphate pirimiphos-methyl 500 g litre^{-1} EC (Actellic, Zeneca, Surrey, UK); the cyclodiene endosulfan 350 g litre^{-1} EC (Thionex, Alpha, Athens, Greece); the neonicotinoid imidacloprid 200 g litre^{-1} SL (Confidor, Bayer AG, Leverkusen, Germany). The insecticides endosulfan and imidacloprid were not tested against the populations GR-EPI and GR-IERB, respectively.

2.3 Leaf dip bioassay

Aqueous dispersions of commercial insecticide formulations were used in a leaf dip bioassay previously described by Cahill *et al*.[13] Cotton leaf discs, cut to fit 39-mm diameter Petri dishes, were immersed for 5 s in serial insecticide concentrations containing 0.2 g litre^{-1} Triton X-100 as non-ionic wetting agent (Merck, Germany). Treated leaf discs were allowed to dry (*ca* 2 h) and placed with the abaxial surface uppermost on Petri dishes embedded with thin sterile water agar (15 g litre^{-1}). Leaf discs dipped in distilled water containing 0.2 g litre^{-1} Triton X-100 were used for control treatments. Four ventilation holes covered with thin mesh had been opened on the side wall of the dish to allow adequate ventilation.

Bemisia tabaci adults were immobilized using carbon dioxide and 20 females were placed on each leaf dish. Handling mortality was estimated within 1 h. The dishes were inverted for the insects to orientate normally and placed in a large controlled environment room at 25 (±1) °C, 50–60% RH and 16:8 h light:dark photoperiod. Five concentrations with five replications each (100 insects per concentration) that gave between 0 and 100% mortality were tested. Rangefinder assays for each insecticide were conducted on the respective population 2–3 days prior to the toxicological test. Final mortality was assessed after 48 h for bifenthrin, α-cypermethrin, pirimiphos-methyl and endosulfan and after 72 h only for imidacloprid. An insect was considered alive if any sign of movement was observed.

2.4 Data analysis

Mortality data were analyzed by probit analysis based on Finney[24] using the Probit software 3.3 (Praxeme).[25] This software tests the linearity of dose-mortality responses and provides the slope, lethal concentrations (LC) and 95% confidence limits (CL) of the LC for each mortality line. A population is considered to be

significantly ($P < 0.05$) more (or less) resistant that another population when there is no overlap of the 95% confidence limits of the LC_{50}. The resistance factors (RF) relative to the susceptible population SUD-S and among the Greek populations were also calculated.

Pairwise comparisons of the log LC_{50} values of all whitefly populations were used to investigate any associations between population responses against different insecticides.

3 RESULTS

The results of the probit analysis are shown in Table 1. The reference strain SUD-S was the most susceptible strain to endosulfan and pirimiphos-methyl, but not to α-cypermethrin, bifenthrin or imidacloprid. For SUD-S, linearity of the dose-mortality response was rejected ($P < 0.05$) for pirimiphos-methyl and endosulfan.

3.1 Bifenthrin resistance

Linearity of the dose mortality response was rejected for populations GR-IERE, GR-IERB, GR-IERC ($P < 0.001$) and GR-MAL ($P < 0.05$). The highest resistance was observed in the GR-MAL population, which exhibited a 23-fold resistance. The three Ierapetra populations displayed low (GR-IERE, RF = 1.8; GR-IERB, RF = 3.1) or no resistance to bifenthrin (GR-IERC). GR-EPI was significantly more susceptible than SUD-S against bifenthrin (RF = 0.22).

3.2 α-Cypermethrin resistance

Linearity of the dose-mortality responses was rejected ($P < 0.001$) for population GR-MAL because of a plateau at 10% mortality. GR-MAL was the most resistant population (RF = 80). GR-IERB and GR-IERC exhibited 11- and 5.8-fold resistance respectively. GR-IERE and GR-EPI were both

Table 1. Log-dose probit mortality data for Greek populations tested with pyrethroid, organophosphate, cyclodiene and neonicotinoid insecticides[a]

Population	*n*	LC_{50}[b] (mg litre^{-1})		95% CL	Slope	SE	χ^{2}[c]	RF
Bifenthrin								
SUD-S	599	0.92	a	0.76–1.12	2.06	0.22	2.4	—
GR-IERE	492	1.63	b	1.28–2.05	1.56	0.14	16.5***	1.8
GR-IERB	565	2.86	b	1.72–4.72	2.29	0.57	24.5***	3.1
GR-IERC	644	1.08	ab	0.37–3.06	1.32	0.43	14.6***	1.6
GR-EPI	594	0.20	c	0.14–0.27	1.96	0.26	0.25	0.22
GR-MAL	959	21.5	d	13.5–34.7	1.67	0.52	4.8*	23
α-Cypermethrin								
SUD-S	598	1.61	a	1.05–2.47	0.79	0.067	7.5	—
GR-IERE	489	0.26	b	0.14–0.43	0.81	0.098	1.3	0.16
GR-IERB	733	17.1	c	10.5–25.4	1.37	0.18	1.9	11
GR-IERC	407	9.38	c	3.66–20.5	0.58	0.087	1	5.8
GR-EPI	558	0.08	d	0.05–0.13	1.24	0.15	5.6	0.05
GR-MAL	799	130	c	0.75–22 500	0.96	0.68	542***	80
Pirimiphos-methyl								
SUD-S	590	16.3	a	8.05–33.1	4.14	2.60	66***	—
GR-IERE	416	73.8	b	41.4–132	2.34	0.82	8.4	4.6
GR-IERB	606	31.4	a	22.8–43.3	3.14	0.71	5.1	1.9
GR-IERC	431	95.1	b	60.3–141	2.44	0.51	8.6	5.8
GR-EPI	545	22.5	a	16.8–28.2	2.67	0.40	4.5	1.4
GR-MAL	734	297	c	206–427	2.43	0.51	15.2**	18
Endosulfan								
SUD-S	598	1.65	a	1.21–2.24	2.77	0.47	12.2**	—
GR-IERE	526	31.7	b	29.4–34.0	7.32	0.70	0.94	19
GR-IERB	729	7.40	c	6.57–8.50	4.45	0.70	0.39	4.5
GR-IERC	541	39.7	b	33.8–43.6	7.68	1.49	0.34	24
GR-EPI	—	—		—	—	—	—	
GR-MAL	512	97	d	55.8–166.7	4.90	2.23	20.3***	58
Imidacloprid								
SUD-S	578	0.36	a	0.24–0.53	1.04	0.10	5.4	—
GR-IERE	539	9.55	b	4.14–21.8	1.30	0.33	10**	26
GR-IERB	—	—		—	—	—	—	
GR-IERC	508	76.4	c	43.3–119	1.46	0.22	0.14	210
GR-EPI	477	0.012	d	0.005–0.018	1.90	0.38	1.7	0.033
GR-MAL	581	266	c	23.5–3012	0.77	0.47	62***	730

[a] *n*, number of whiteflies tested. CL, confidence limits. RF, resistance factor.

[b] Different letters indicate non-overlap of confidence limits ($P < 0.05$).

[c] Chi-square testing linearity of dose-mortality response: * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

significantly more susceptible than SUD-S (RF = 0.16 and 0.05 respectively).

3.3 Pirimiphos-methyl resistance

Linearity of the dose-mortality responses was rejected for the population GR-MAL ($P < 0.01$). The highest resistance was observed in population GR-MAL (18-fold) followed by GR-IERC (RF = 5.80) and GR-IERE (RF = 4.6). Populations GR-IERB and GR-EPI did not exhibit resistance against pirimiphos methyl.

3.4 Endosulfan resistance

Linearity of the dose-mortality responses was rejected for the population GR-MAL ($P < 0.001$). All populations displayed significant resistance ranging from 4.5-fold in GR-IERB to 58-fold in GR-MAL (GR-EPI was not tested). High slope values (>4) were observed for all Greek whitefly populations, particularly for GR-IERE and GR-IERC, suggesting a high homogeneity of the populations against the cyclodiene insecticide.

3.5 Imidacloprid resistance

Linearity of the dose-mortality responses was rejected for population GR-IERE ($P < 0.01$) and GR- MAL ($P < 0.001$). The highest resistance was exhibited by population GR-MAL (RF = 730). Significant resistance, 26- and 210-fold, was observed for GR-IERE and GR-IERC, respectively (GR-IERB was not tested). GR-EPI was significantly more susceptible than SUD-S against imidacloprid (RF = 0.033).

The *B tabaci* population GR-EPI collected from open-field crops was significantly more susceptible against all insecticides tested than SUD-S. The resistance factors of the Greek populations collected from greenhouse crops against GR-EPI are shown in Table 2. The highest resistance ratios were observed for the insecticides imidacloprid and α-cypermethrin followed by bifenthrin and pirimiphos methyl. Resistance factors calculated using the local susceptible strain GR-EPI as reference were notably higher than RFs calculated using SUD-S for imidacloprid and α-cypermethrin. For example, the population GR-IERC, was 11- and 210-fold more resistant to α-cypermethrin and imidacloprid, respectively, when compared with SUD-S, but 112- and 6408 -fold more resistant to the same insecticides when compared with GR-EPI.

Table 2. Resistance ratios of the Greek populations, relative to the local susceptible population GR-EPI[a]

Population	BIF	A-CYP	PIR	IMI
GR-IERE	8.2	3.1	3.3	801
GR-IERB	14	205	1.4	
GR-IERC	7.2	112	4.2	6 408
GR-MAL	108	1 549	13	22 324

[a] A-CYP = α-cypermethrin; BIF = bifenthrin; PIR = pirimiphos-methyl; IMI = imidacloprid.

In pairwise comparisons of LC_{50} values, a significant correlation between the two pyrethroid insecticides was observed ($r = 0,856$, $P < 0.05$). No significant correlation between the LC_{50} values for the organophosphate insecticide pirimiphos-methyl and the pyrethroid insecticides bifenthrin or α-cypermethrin was found ($r = 0.767$ and 0.624 respectively). Imidacloprid and endosulfan were excluded from the correlation tests because of the low number of pairwise comparisons.

4 DISCUSSION

This study revealed variation in the level of insecticide resistance in *B tabaci* collected in Crete. The population GR-EPI collected on outdoor melon crops free of insecticide treatments was the most susceptible against all insecticides among the Greek populations. In addition, GR-EPI was more susceptible against bifenthrin, α-cypermethrin and imidacloprid than the reference susceptible strain SUD-S, and exhibited the same susceptibility to pirimiphos-methyl as SUD-S. No evidence of physical stress (ie increased control mortality compared with the other populations) of GR-EPI was observed that could be associated with the high susceptibility levels recorded. The highest resistance factors recorded at LC_{50} were exhibited by the population collected from Malades (GR-MAL) in a floriculture greenhouse after reports of repeated failures to control the pest with chemical insecticides. The high levels of resistance in the particular greenhouse could be explained by the fact that large numbers of potted plants (*Hibiscus* sp and *L camara*) were maintained in stock throughout the year and the *B tabaci* population had been continuously selected. The populations from the Ierapetra locality were collected from greenhouses where the vegetable crops were maintained only for the eight-month cropping season and displayed intermediate levels of resistance compared with GR-EPI and GR-MAL. The level of exposure to insecticides of the Greek whitefly populations ranged from no exposure (GR-EPI) to intensive insecticide application (GR-MAL). The succession of the populations ranked according to the exposure to insecticides was the same as that observed in the level of resistance with the insecticides pirimiphos-methyl, endosulfan and imidacloprid. This association between insecticide use and the resistance level was observed only in part with the pyrethroid insecticides. Although GR-EPI and GR-MAL exhibited the lowest and the highest levels of resistance respectively, GR-IERC and GR-IERE were less resistant to bifenthrin and α-cypermethrin than to GR-IERB (collected in greenhouse practicing biological control), despite the fact that pyrethroids are extensively used against *B tabaci* in conventional pest management. *Bemisia tabaci* colonizes a wide range of plant species including many wild host plants[26,27] and gene flow between treated and untreated populations may be the origin

of discrepancies between insecticide use and level of resistance.

Insecticide resistance is mainly based on mutations of the target protein decreasing the affinity to the respective insecticide and on increased detoxification by esterases, cytochrome P450-dependant monoxygenases or gluthatione *S*-tranferases (GST). In *B tabaci*, resistance to organophosphates involves, in addition to the insensitivity of the synapse acetylcholinesterase,[28,29] the enhanced detoxification of the insecticides by non-specific esterases and cytochrome P450-dependant monoxygenases.[22,30,31] Overexpression of cytochrome P450-dependant monoxygenases was also found to be responsible for the resistance of *B tabaci* to neonicotinoids.[14] Resistance of *B tabaci* to the cyclodiene endosulfan is associated with a replacement of a single amino acid (Ala_{302} to Ser or Gly) within the resistance to dieldrin gene (*Rdl*) encoding the γ-aminobutyric acid (GABA) receptor subunit, the primary target of cyclodiene insecticides.[32] Nevertheless, cyclodiene resistance not associated with replacement of Ala_{302} has been found in *Helicoverpa armigera* (Hübner)[33] and *Nasonovia ribisnigri* (Mosley), where GST detoxification was mainly involved.[34] Metabolism of endosulfan by oxidative and hydrolytic pathways has also been suspected in *B tabaci* populations by Pérez *et al*.[35] Resistance to pyrethroids has been associated with mutations in the *para* sodium channel gene, the target site of the insecticides in this class.[36] Detoxification by esterases and cytochrome P450-dependant monoxygenases has also been implicated in pyrethroid resistance in *B tabaci*.[22,30,37,38]

In our study, a significant correlation of the LC_{50} values was observed between bifenthrin and α-cypermethrin, but no correlation was found between the two pyrethroids and pirimiphos-methyl. Absence of correlation between resistance to pyrethroids and organophosphates in *B tabaci* has been reported by Pérez *et al*[35] and Cahill *et al*.[13] Our finding suggests that the resistance mechanism to the two pyrethroids is different from the mechanism involved in the resistance to pirimiphos-methyl.

In Ierapetra, the largest greenhouse area in Crete, the highest resistance was exhibited against imidacloprid by the population GR-IERC (RF = 210) collected in the greenhouse practising conventional control. This resistance level was aproximately 10 times higher than that of the other Ierapetra population GR-IERE (RF = 26), collected in an experimental greenhouse, indicating a strong selection against imidacloprid in the commercial greenhouses. Since a common detoxifying resistance mechanism against insecticides of this class has been recently demonstrated,[14] resistance of the local populations to other neonicotinoids is expected. Preliminary results have indicated that cross-resistance occurs between the insecticides thiacloprid and imidacloprid (data not shown). GR-IERC and GR-MAL, both collected from greenhouses practising conventional control, were found 6408 and 22 324 times, respectively, more tolerant to imidacloprid than the local susceptible population (GR-EPI). Resistance of this level against the insecticide imidacloprid has been reported recently by Rauch and Nauen[14] for populations collected from Spain, Germany and Israel. The current status of *B tabaci* resistance to neonicotinoid insecticides requires urgent attention. Since the recent virus outbreaks, pest management in the greenhouse crops of Crete heavily relies on chemical control, and particularly on neonicotinoids. An average of 30 insecticide applications per crop season does not allow the design of a rational insecticide rotation scheme. The presence of highly susceptible *B tabaci* pool in wild host plants could possibly be a major advantage regarding the implementation of insecticide resistance management in the area.

Development of resistance depends in part on the occurrence of 'refugia' for susceptible populations[39] and on gene flow among populations colonizing different habitats in a given area. The biotype status as well as genetic exchanges between *B tabaci* populations collected on different plant species and on greenhouse or outdoor crops are currently been examined using microsatellite markers.[40] Studies on the genetic structure of the *B tabaci* population together with the identification of resistance genes will allow a better understanding of the evolution of resistance. The development of a resistance management scheme is being considerate through the area-wide implementation of integrated pest management.

ACKNOWLEDGEMENTS

We thank Ian Denholm, IACR Rothamsted, who kindly provided the susceptible strain SUD-S and Nikos Giannios for technical assistance. This project was funded by the Prefecture of Lassithi, Crete Greece.

REFERENCES

1 Perring TM, The *Bemisia tabaci* species complex. *Crop Prot* **20**:725–737 (2001).
2 Martin JH, Mifsud D and Rapisarda C, The whiteflies (Hemiptera: Aleyrodidae) of Europe and the Mediterranean Basin. *Bull Entomol Res* **90**:407–448 (2000).
3 Bedford ID, Briddon RW, Brown JK, Rosell RC and Markham PG, Geminivirus transmission and biological characterization of *Bemisia tabaci* (Gennadius) biotypes from different geographic regions. *Ann Appl Biol* **125**:311–325 (1994).
4 Bedford ID, Briddon RW and Markham PG, *Proc Brighton Crop Prot Conf—Pests Dis*, BCPC, Farnham, Surrey, UK, pp 1235–1240 (1992).
5 Sanchez-Campos S, Navas-Castillo J, Camero R, Soria C, Diaz JA and Moriones E, Displacement of tomato yellow leaf curl virus (TYLCV)-Sr by TYLCV-Is in tomato epidemics in Spain. *Phytopathology* **89**:1038–1043 (1999).
6 Oliveira MRV, Henneberry TJ and Anderson P, History, current status, and collaborative research projects for *Bemisia tabaci*. *Crop Prot* **20**:709–723 (2001).
7 Jones DR, Plant viruses transmitted by whiteflies. *Eur J Plant Pathol* **109**:195–219 (2003).

8 Kirk AA, Lacey LA, Roditakis N and Brown JK, The status of *Bemisia tabaci* (Hom, Aleyrodidae), *Trialeurodes vaporariorum* (Hom, Aleyrodidae) and their natural enemies in Crete. *Entomophaga* **38**:405–410 (1993).
9 Mound LA and Hasley SH, *Whitefly of the world. A systematic catalogue of the Aleyrodidae (Homoptera) with host plant and natural enemy data*, John Wiley London (1978).
10 Avgelis AD, Roditakis N, Dovas CI, Katis NI, Vassilakos N and Bem F, First report of tomato yellow leaf curl virus on Tomato Crops in Greece. *Plant Dis* **85**:678 (2001).
11 Michigan State University, The database of arthropod resistance to pesticides, at http://www.cips.msu.edu/resistance/rmdb/index.html (2004).
12 Elbert A and Nauen R, Resistance of *Bemisia tabaci* (Homoptera: Aleyrodidae) to insecticides in southern Spain with special reference to neonicotinoids. *Pest Manag Sci* **56**:60–64 (2000).
13 Cahill M, Byrne FJ, German K, Denholm I and Devonshire AL, Pyrethroid and organophosphate resistance in the tobacco Whitefly *Bemisia tabaci* (Homoptera, Aleyrodidae). *Bull Entomol Res* **85**:181–187 (1995).
14 Rauch N and Nauen R, Identification of biochemical markers linked to neonicotinoid cross resistance in *Bemisia tabaci* (Hemiptera:Aleyrodidae). *Arch Insect Biochem Physiol* **54**:165–176 (2003).
15 El Kady H and Devine GJ, Insecticide resistance in Egyptian populations of the cotton whitefly, *Bemisia tabaci* (Hemiptera: Aleyrodidae). *Pest Manag Sci* **59**:865–871 (2003).
16 Ahmad M, Arif MI, Ahmad Z and Denholm I, Cotton whitefly (*Bemisia tabaci*) resistance to organophosphate and pyrethroid insecticides in Pakistan. *Pest Manag Sci* **58**:203–208 (2002).
17 Kranthi KR, Jadhav DR, Kranthi S, Wanjari RR, Ali SS and Russell DA, Insecticide resistance in five major insect pests of cotton in India. *Crop Prot* **21**:449–460 (2002).
18 Horowitz AR, Weintraub PG and Ishaaya I, Status of pesticide resistance in arthropod pests in Israel. *Phytoparasitica* **26**:231–240 (1998).
19 Prabhaker N, Toscano NC, Perring TM, Nuessley G, Kido K and Youngman RR, Resistance monitoring of the sweetpotato whitefly (Homoptera, Aleyrodidae) in the Imperial-Valley of California. *J Econ Entomol* **85**:1063–1068 (1992).
20 Cahill M, Denholm I, Ross G, Gorman K and Johnston D, Relationship between bioassay data and the simulated field performance of insecticides against susceptible and resistant adult *Bemisia tabaci* (Homoptera: Aleyrodidae). *Bull Entomol Res* **86**:109–116 (1996).
21 Bouharroud R, Hanafi A, Murphy B, Haouass F and Serghini MA, *3rd International Bemisia Workshop*, Barcelona (2003).
22 Dittrich V, Ernst GH, Ruesh O and Uk S, Resistance mechanisms in sweet-potato whitefly (Homoptera: Aleyrodidae) populations from Sudan, Turkey, Guatemala and Nicaragua. *J Econ Entomol* **83**:1665–1670 (1990).
23 Denholm I, Cahill M, Dennehy TJ and Horowitz AR, Challenges with managing insecticide resistance in agricultural pests, exemplified by the whitefly *Bemisia tabaci*. *Philos Trans R Soc Lond Ser B—Biol Sci* **353**:1757–1767 (1998).
24 Finney DJ, *Probit analysis*, Cambridge University Press, Cambridge (1964).
25 Raymond M, Prato G and Ratsira D, Probit analysis of mortality assays displaying quantal response, *Version 33 License 193019* (1993).
26 Attique MR, Rafiq M, Ghaffar A, Ahmad Z and Mohyuddin AI, Hosts of *Bemisia tabaci* (Genn) (Homoptera: Aleyrodidae) in cotton areas of Punjab, Pakistan. *Crop Prot* **22**:715–720 (2003).
27 Calvitti M and Remotti PC, Host preference and performance of *Bemisia argentifolii* (Homoptera:Aleyrodidae) on weeds in central Italy. *J Econ Entomol* **27**:1350–1356 (1998).
28 Byrne FJ and Devonshire AL, Kinetics of insensitive acetylcholinesterases in organophosphate-resistant tobacco whitefly, *Bemisia tabaci* (Gennadius) (Homoptera:Aleyrodidae). *Pest Biochem Physiol* **58**:119–124 (1997).
29 Anthony NM, Brown JK, Feyereisen R and ffrench-Constant RH, Diagnosis and characterization of insecticide-insensitive acetylcholinesterase in three populations of the sweet-potato whitefly *Bemisia tabaci*. *Pestic Sci* **52**:39–46 (1998).
30 Prabhaker N, Coudriet DL and Toscano NC, Effect of synergists on organophosphate and permethrin resistance in sweetpotato whitefly (Homoptera: Aleyrodidae). *J Econ Entomol* **81**:34–39 (1988).
31 Bloch G and Wool D, Methidathion resistance in the sweetpotato whitefly (Aleyrodidae, Homoptera) in Israel: selection, heritability and correlated changes in esterase activity. *J Econ Entomol* **87**:1147–1156 (1994).
32 Anthony NM, Brown JK, Markham PG and ffrench-constant RH, Molecular analysis of cyclodiene resistance-Associated mutations among populations of the sweet-potato whitefly *Bemisia tabaci*. *Pest Biochem Physiol* **51**:220–228 (1995).
33 ffrench-Constant RH, Anthony NM and Andreev D, Single versus multiple origins of insecticide resistance: inferences from the cyclodiene resistance gene *Rdl*, in *Molecular genetics and evolution of pesticide resistance*, ACS Symposium Series No 645, ed by Brown TM, American Chemical Society, Washington, DC, pp 106–116 (1996).
34 Rufingier C, Pasteur N, Lagnel J, Martin C and Navajas M, Mechanisms of insecticide resistance in the aphid *Nasonovia ribisnigri* (Mosley) (Homoptera: Aphididae) from France. *Insect Biochem Mol Biol* **29**:385–391 (1999).
35 Perez CJ, Alvarado P, Narvaez C, Miranda F, Hernandez L, Vanegas H, Hruska A and Shelton AM, Assessment of insecticide resistance in five insect pests attacking field and vegetable crops in Nicaragua. *J Econ Entomol* **93**:1779–1787 (2000).
36 Morin S, Williamson MS, Goodson SJ, Brown JK, Tabashnik BE and Dennehy TJ, Mutations in the *Bemisia tabaci* para sodium channel gene associated with resistance to a pyrethroid plus organophosphate mixture. *Insect Biochem Mol Biol* **32**:1781–1791 (2002).
37 Byrne FJ, Gorman KL, Cahill M, Denholm I and Devonshire AL, The role of B-type esterases in conferring insecticide resistance in the tobacco whitefly, *Bemisia tabaci* (Genn). *Pest Manag Sci* **56**:867–874 (2000).
38 Shchukin A and Wool D, Pyrethroid resistance and esterase activity in selected laboratory populations of sweetpotato whitefly *Bemisia tabaci* (Homoptera:Aleyrodidae). *Eur J Entomol* **91**:285–295 (1994).
39 Georghiou GP, Principles of insecticide resistance management. *Phytoprotection* **75**:51–59 (1994).
40 Tsagkarakou A and Roditakis N, Isolation and characterization of microsatellite loci in *Bemisia tabaci* (Hemiptera:Aleyrodidae). *Mol Ecol Notes* **3**:196–198 (2003).