

Buprofezin Effects on Two Parasitoid Species of Whitefly (Homoptera: Aleyrodidae)

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ABSTRACT The effect of buprofezin on *Bemisia tabaci* (Gennadius) and its parasitoids *Eretmocerus* sp. and *Encarsia luteola* Howard was determined in the laboratory. The effect on immature whiteflies was tested, and LC_{50} and LC_{95} were estimated. Immature parasitoids were exposed to the material in both the egg and pupal stages. Adults were tested to determine the effect of buprofezin on oviposition. First- and second-instar sweet-potato whiteflies were most sensitive to the material, with the LC_{50} for the fourth instar being ≈ 80 -fold that of the first instar. Young *Eretmocerus* sp. were affected by buprofezin but young *E. luteola* were not. The reverse was true for pupae. No effect on oviposition occurred with females that were exposed to buprofezin either as immatures or when they were mature. We concluded that the following three factors should be considered when using buprofezin for pest management of *B. tabaci*: (1) parasitoids attack the whiteflies mainly at the third stadium and beyond; later instars are not as sensitive to buprofezin; (2) even under laboratory conditions, at least 20% of the parasitoids will probably survive; and (3) the rate of parasitoid oviposition is unaffected by buprofezin.

KEY WORDS *Bemisia tabaci*, parasitoids, *Encarsia*

THE SWEETPOTATO WHITEFLY, *Bemisia tabaci* (Gennadius), is a pest of economic importance that attacks numerous vegetable and field crops (Byrne et al. 1990). This species is controlled currently with insecticides. However, development of resistance and damage to the natural-enemy complex caused by the frequent use of insecticides increasingly have hampered control of *B. tabaci* (Eveleens 1982; Dittrich et al. 1986, 1990). Consequently, new means of control, including use of new types of insecticides, are needed.

One candidate group, insect growth regulators (IGRs), disrupts development of immatures by interference either with the hormonal balance of the developing insect, or with chitin synthesis. IGRs are specific to certain taxonomic groups and, in addition to their efficacy, are considered to be relatively nondisruptive to the agroecosystem (Staal 1975, Perng & Sun 1987).

One of the chitin synthesis-inhibiting IGRs, buprofezin, is effective against Homoptera, including whiteflies. It has been tested extensively against the greenhouse whitefly, *Trialeurodes vaporariorum* (Westwood), with good results. Buprofezin is particularly effective against young nymphs but not against the pupae. This IGR also has a detrimental effect on eggs, especially when the mother comes in contact with the material before oviposition (Yasui et al. 1985, 1987). Buprofezin also affects *B. tabaci*; the IGR killed

young nymphs that had been exposed either by direct contact (Ishaaya et al. 1988) or by vapor action (De Cock et al. 1990). In addition, eggs of females that come in contact with the material fail to hatch (Ishaaya et al. 1988). Because *B. tabaci* is a prominent cotton pest in Israel, it is important to determine the efficacy of buprofezin for its control and to determine how it can be used most effectively for control.

A chemical's ability to control a pest includes not only its capacity to kill but also its selective advantage (or disadvantage) to the natural enemies present. Therefore, we studied the relative susceptibility of different stages of *B. tabaci* in the laboratory, together with the chemical's effect on two parasitoid species of the sweetpotato whitefly. Here, we report the results of these tests.

Materials and Methods

Sweetpotato Whiteflies. The experiments were done in a temperature cabinet at $26 \pm 1^\circ\text{C}$ with 60% RH and a photoperiod of 14:10 (L:D) h^{-1} . Cotton seedlings ('Acala SJ2') with one true leaf each were used. About 100-200 sweetpotato whiteflies from our permanent laboratory cultures were placed on each plant for 24 h for oviposition. Eggs were allowed to develop, and nymphs were observed until they reached the desired stage, when they were counted under a

dissecting microscope. Thereafter, they were dipped either in water (for the control) or in water containing a known concentration of buprofezin. The buprofezin concentrations ranged from 0.5 to 1,000 mg(AI)/liter. The leaves were kept for 24 d to allow the sweetpotato whiteflies to develop to adulthood and to emerge. Thereafter, they were examined, and emergence was calculated. Treatments began with the lowest concentration; after treated sweetpotato whiteflies in the stadium being tested showed at least 95% mortality at a given concentration, the experiment with this instar was terminated. If mortality was <95%, a higher concentration was applied in the next experiment.

A total of 10 plants (=replicates) was used for each treatment. Percentage mortality figures were transformed by the arcsine transformation before statistical analysis. Means of mortality among instars were compared in a one-way analysis of variance and were considered significant at $P < 0.05$. A Tukey's *B* test was used to compare differences among means after analysis of variance (Sokal & Rohlf 1981). Mortality regression lines were estimated by probit regressions (SAS Institute 1985). Regression line slopes were compared for significant differences as described by Sokal & Rohlf (1981).

Parasitoids. *Effect of Buprofezin on Immature Parasitoids Shortly After Oviposition.* Adult *Encarsia luteola* Howard and *Eretmocerus* sp. from California (USA) were taken from the culture and kept for 24 h in a vial with honey. Thereafter, 5–10 female wasps were placed on a cotton leaf (we used a rooted seedling as described above) bearing either second- and third-instar sweetpotato whiteflies (for *Eretmocerus* sp.) or third- and fourth-instar sweetpotato whiteflies (for *E. luteola*). The leaf was placed under a dissecting microscope, and the wasps were observed. Presumed oviposition on whiteflies was recorded by making a small mark near each appropriate sweetpotato whitefly nymph. Thereafter, the leaf was dipped in a buprofezin solution (20 mg/liter for *E. luteola*, which attacks the third and fourth instars; 5 mg/liter for *Eretmocerus* sp., which attacks the second and third instars). Controls were dipped in water. We used 9–13 replicates for each experiment.

Effect of Buprofezin on Parasitoid Pupae. Whiteflies on leaves were parasitized by releasing several females on them for 24 h. Six to eight d later (for *E. luteola*) or 9 to 11 d later (for *Eretmocerus* sp.), they were examined, and the parasitized pupae were counted. This procedure was facilitated by the fact that *B. tabaci* nymphs and pupae are transparent, allowing a clear view of the parasitoid pupae through them. Thereafter, the leaves were dipped in 1,000 mg/liter buprofezin solution for the test; leaves were dipped in water for the controls. We used 11–18 replicates of each experiment. The 1,000 mg/liter

concentration was chosen because we assumed that insect pupae are more resistant to buprofezin than are younger instars.

Effect of Buprofezin on Parasitoid Adults. We tested the effect of buprofezin on adults that were exposed as well as testing the residual effect on adults that had been exposed to the treatment as larvae or pupae. The effect of contact with buprofezin was tested by exposing female parasitoids to cotton leaves bearing sweetpotato whitefly nymphs (second to fourth instars). The leaves had been dipped in a solution of 100 mg/liter of buprofezin and allowed to dry. Control samples were dipped in water. The wasps were retained in small cages on these leaves for 48 h, during which they were seen feeding and ovipositing on the sweetpotato whitefly nymphs. Thereafter, the wasps were removed and were given fresh, undipped leaves bearing ≈ 100 –200 hosts for oviposition. Five females were placed on each leaf for 24 h. The experiment was replicated five to six times, and the number of parasitized sweetpotato whiteflies on each leaf was counted about 1 mo later. The differences between treatments were tested for significance by a one-way analysis of variance after a square root transformation of the data (Sokal & Rohlf 1981).

Effect on adults that emerged from treated immatures was tested by subjecting immature, parasitized hosts to buprofezin and examining the number of progeny produced by the emerging adults. For both *Eretmocerus* sp. and *E. luteola*, we used concentrations that were effective against some of the progeny but that did not cause total parasitoid mortality. For *E. luteola*, we used hosts that had been exposed to parasitoids for 2 d. The leaves were dipped in a 20 mg/liter solution of buprofezin, a concentration that allowed at least some parasitoid emergence. For *Eretmocerus* sp., we used pupated parasitoids rather than larvae because of the high larval mortality experienced in the previous experiments. Parasitoids were exposed to 1,000 mg/liter of buprofezin; water was used as a control. Parasitoids that emerged from the experiments were placed on leaves with fresh hosts, and their progeny production was examined as described previously. Six to eight replicates of each experiment were done.

Results

Effect of Treatment on Sweetpotato Whiteflies. Mortality of sweetpotato whiteflies usually occurred either during the molt to the next instar or immediately thereafter. Sensitivity to treatment was high in the first and second instars and decreased as nymphs grew (Table 1). In most cases, the effect on the fourth instar took the form of pupal mortality; occasionally we observed adult mortality either during emergence from the pupal skin or when adults were free.

Table 1. Influence of buprofezin concentration on *B. tabaci* nymphs

Concn of buprofezin mg/liter	Mean mortality of <i>B. tabaci</i> nymphs \pm SEM			
	L1	L2	L3	L4
Control	7.80a \pm 2.36	9.11a \pm 2.49	6.31a \pm 2.42	2.34a \pm 0.79
0.5	48.41b \pm 10.24	—	—	—
1	63.53bc \pm 7.67	31.06b \pm 7.28	32.10b \pm 8.21	1.52a \pm 0.66
5	83.35cd \pm 6.08	40.63b \pm 5.78	43.44b \pm 5.61	11.07ab \pm 3.10
10	90.76d \pm 1.78	79.73c \pm 2.67	66.13c \pm 4.84	21.39abc \pm 5.70
20	96.97d \pm 1.15	80.83c \pm 6.34	73.74c \pm 5.73	23.78bc \pm 7.35
40	—	96.41c \pm 1.88	77.89c \pm 3.03	39.51c \pm 5.89
100	—	—	74.94c \pm 2.49	59.60d \pm 8.50
250	—	—	98.43d \pm 0.83	81.16e \pm 4.20
1,000	—	—	—	99.81f \pm 0.14

Each value is an average of 10 replicates. Within a column, means followed by the same letter are not significantly different ($P > 0.05$; Tukey's *B* test [Sokal & Rohlf 1981]).

The difference in LC_{50} between the first- and fourth-instar nymphs was ≈ 80 -fold, and the difference in LC_{95} was ≈ 50 -fold. However, the slope of the regression lines for the fourth instar was not significantly different, indicating that the rates of nymphal mortality resulting from increases in buprofezin concentrations were similar for all instars (Table 2).

Effect of Treatment on Young Immature Parasitoids. Both treated *Eretmocerus* sp. and *E. luteola* had lower emergence rates than the controls (Table 3). However, we expected some reduction in percentage emergence of the parasitoids because buprofezin affects sweetpotato whiteflies directly, and young parasitoids die when their hosts die. Therefore, we determined whether mortality was greater than that expected for the sweetpotato whitefly nymphs alone.

For *Eretmocerus* sp., we observed 78% reduction in parasitoid emergence compared with the control (Table 3). This value is significantly higher than the 40% that could be expected by only considering nymphal mortality at 5 mg/liter buprofezin (Table 1). Therefore, treatment with buprofezin had a negative effect on the immatures of this parasitoid, reducing emergence success by $\approx 40\%$. We observed cases in which differential mortality of *Eretmocerus* sp. occurred (i.e., all of the affected parasitized sweetpotato whitefly nymphs died, even those in which the parasitoid was no longer alive). In contrast,

E. luteola treated with 20 mg/liter buprofezin showed 56% mortality (Table 3), a level that is not significantly different from the $\approx 50\%$ mortality observed when both third and fourth instars were treated with 20 mg/liter buprofezin (Table 1). Consequently, we observed no effect of buprofezin on young instars of this species.

Effect of Treatment on Parasitoid Pupae. *Eretmocerus* sp. was unaffected, with emergence of adults from the treated pupae similar to that of the untreated ones (Table 3). *E. luteola* was affected by the treatment, with a reduction of $\approx 77\%$ in emergence after the treatment. Although most parasitoids died as pupae, a few reached adulthood and began to drill emergence holes before death.

Effect of Buprofezin on Progeny Production of Adult Parasitoids. Both parasitoid species were unaffected (Table 4). The number of progeny produced during 24 h by females that had been exposed to buprofezin either as adults or as immatures did not differ from that of controls treated with water.

Discussion

Our observations that buprofezin affects young instars more than older instars agree with the previous results with sweetpotato whitefly of Ishaaya et al. (1988). No explanation for this pattern is as yet available. However, the thicker wax cover of the more mature instars and their larger body mass may cause this phenomenon. Possibly, the capability of detoxifying and secreting is higher in later instars, as demonstrated for house flies, *Musca domestica* L., by Perry & Agosin (1974).

Two explanations for the high mortality rate caused in *Eretmocerus* sp. by buprofezin shortly after oviposition are possible (i.e., either parasitism affected the sweetpotato whitefly nymph so that it became more susceptible to buprofezin than unparasitized individuals or the immature parasitoid is affected directly by the material).

Table 2. Responses of *B. tabaci* nymphs exposed to buprofezin in the laboratory

Instar	n	(95% Fiducial limits)		Slope \pm SEM
		LC_{50} , mg/liter	LC_{95} , mg/liter	
1st	7,308	0.58 (0.27–0.95)	15.7 (8.70–42.5)	1.15 \pm 0.87
2nd	8,150	4.20 (0.35–9.88)	56.1 (18.3–174,000)	1.46 \pm 0.68
3rd	7,131	5.24 (1.61–10.1)	451 (131–10,400)	0.85 \pm 1.18
4th	10,488	47.3 (35.2–64.7)	767 (439–1,690)	1.36 \pm 0.74

Table 3. Emergence of adult *Eretmocerus* sp. and *Encarsia luteola* after dipping the parasitized hosts in buprofezin

Species	Treatment, mg/liter	n ^a	Stage ^b	No. parasitized	No. emerging	% emergence	% reduction ^c
<i>E. luteola</i>	0	13	E	288	230	79.86a	—
	20	9	E	232	82	35.34b	56%
<i>Eretmocerus</i> sp.	0	12	E	262	139	53.05a	—
	5	10	E	300	35	11.67b	78%
<i>E. luteola</i>	0	17	P	433	315	72.75a	—
	1,000	17	P	469	78	16.63b	77%
<i>Eretmocerus</i> sp.	0	11	P	768	669	87.11a	—
	1,000	18	P	644	550	85.40a	2%

Within a column for a species, means followed by the same letter are not significantly different ($P > 0.05$; one-way analysis of variance [Sokal & Rohlf 1981]).

^a n, number of replicates.

^b E, parasitoids treated in the egg stage; P, parasitoids treated in the pupal stage.

^c Reduction in percent emergence as compared with the control.

The latter cause is supported by our observation that no differential mortality of the host and its parasitoid occurred.

Eretmocerus sp. wasps do not injure their host during oviposition. Consequently, we anticipated no effect on host susceptibility before parasitoids hatch. However, buprofezin has a long-lasting effect and may affect the sweetpotato whitefly for several days, when *Eretmocerus* sp. larvae will have injured the host. Therefore, we have no sure way of determining the cause for the higher mortality that was observed. However, the fact that parasitization by *E. luteola*, which punctures its host during oviposition, does not cause higher mortality after treatment with buprofezin suggests that the effect is not on the host, but on *Eretmocerus* sp. As discussed below, this parasitoid may be susceptible to such treatment.

The detrimental effect of buprofezin on young *Eretmocerus* sp. probably is related to the ovipositional habits and immature development of that insect. *Eretmocerus* sp. lays its eggs under the host, where they hatch. Larvae then penetrate the host. After penetration, the larva develops within a capsule formed by the host that maintains contact with the outside (Gerling et al.

1991). The contact with the exterior environment exhibited by eggs and young *Eretmocerus* sp. larvae may expose them to buprofezin vapor. De Cock et al. (1990) demonstrated the toxic nature of such vapor.

As mentioned previously, young immature *E. luteola* are not affected by treatment with buprofezin. However, pupae of that species showed high mortality when treated. In liquid or vapor form, buprofezin may penetrate the thin remnants of the host's cuticle that envelop the pupa of the parasitoid, and such direct contact may cause parasitoid mortality. No such effect was found with *Eretmocerus* sp., which was less susceptible to buprofezin at the pupal stage.

On the basis of the results of our experiments, we speculate that buprofezin, because of its effects on sweetpotato whitefly immatures, can be used in a pest management strategy. In the field, especially in crops like cotton that have an extensive entomofauna, several types of organisms may be affected by insecticide treatments. These include the target pest itself, its parasitoids, its predators, other pests, and their natural enemies. In our experiments, we used *B. tabaci* and its parasitoids; consequently, our discussion emphasizes these organisms.

The natural enemy complex of *B. tabaci* is composed of the predators, parasitoids, and fungi. Thus far, fungi have not been reported to control *B. tabaci* in the field. The predators prefer to attack the early immature stages of sweetpotato whiteflies, whereas both *Eretmocerus* spp. and *Encarsia* spp. parasitize mainly the third and fourth instars of the host (Gerling 1990). In contrast, buprofezin is most effective against young sweetpotato whitefly immatures but also has some detrimental effect on both parasitoid species. Its effect on immature predators (e.g., on larvae of coccinellids and lacewings or on *Orius* spp.) has not been determined, but we anticipate some adverse effects.

Based on past experience, the importance of parasitoids in the control of sweetpotato whitefly populations is often of paramount importance

Table 4. Effect of buprofezin on adults of *Eretmocerus* sp. and *Encarsia luteola*

Species	Treatment ^a , mg/liter	n	Mean progeny ± SEM
<i>E. luteola</i>	0 (A)	6	24.67a ± 5.23
	100 (A)	5	34.80a ± 5.74
<i>Eretmocerus</i> sp.	0 (A)	5	74.40a ± 11.66
	100 (A)	5	63.20a ± 13.03
<i>E. luteola</i>	0 (B)	6	23.50a ± 5.77
	20 (B)	8	30.63a ± 4.78
<i>Eretmocerus</i> sp.	0 (C)	6	72.00a ± 13.50
	1,000 (C)	7	99.43a ± 17.78

Within a column for a species, means followed by the same letter are not significantly different ($P > 0.05$; one-way analysis of variance [Sokal & Rohlf 1981]).

^a A, parasitoids treated as adults; B, parasitoids treated as eggs within the hosts; C, parasitoids treated as pupae within host skins.

(whereas predators, whose role has not yet been studied adequately, are given less credit in that respect [Gerling 1990, Onillon 1990]). Consequently, the preservation of parasitoid activity is of great importance for sweetpotato whitefly management. The differential activity of buprofezin toward these whiteflies and parasitoids can be used by timing the treatments so that most of the pest population is in the young immature stage (nymphal first and second instars). Such timing will ensure a great reduction of the pest population but will have little effect on the parasitoids because immature parasitoids are affected only somewhat and adult parasitoids are not susceptible to the insecticide. Adult parasitoids that survive the treatment will encounter only those few hosts that escaped the insecticide treatment. These hosts probably can be kept under control by parasitoid activity, with little or no need for further human intervention.

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