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The effect of pretreatment with S,S,S-tributyl phosphorotrithioate on deltamethrin resistance and carboxylesterase activity in *Aphis gossypii* (Glover) (Homoptera: Aphididae)

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ABSTRACT

The synergism of S,S,S-tributyl phosphorotrithioate (DEF) and its effect on carboxylesterase activity were investigated in deltamethrin-selected resistant (DRR) and susceptible (DSS) strains of cotton aphids, *Aphis gossypii* (Glover). Compared to the DSS strain, the DRR strain showed 23,900-fold resistance to deltamethrin, and 7560- and 99-fold cross-resistance to bifenthrin and ethofenprox, respectively. The synergist, DEF, increased the toxicity of both deltamethrin and bifenthrin, but not of ethofenprox when DEF was pretreated of 15 h. DEF exhibited significant inhibition on the carboxylesterase activity in the DRR strain, but no significant effect on that of the DSS strain *in vitro*. After the cotton aphids exposing to DEF, the carboxylesterase activity decreased gradually until 15 h and then gradually recovered until 24 h in the DRR strain, which fluctuated according to the effect of DEF on the deltamethrin toxicity detected using DEF pretreatment in the DRR strain. Therefore, our studies suggested that the effect of DEF on carboxylesterase was associated with deltamethrin resistance in the DRR strain.

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1. Introduction

Aphis gossypii (Glover) (Hemiptera: Aphididae), the cotton aphid, is a major pest of agriculture throughout the world because of damage through feeding and virus transmission [1]. In China, the cotton aphids have been subjected to widespread intensive chemical control such as organophosphates, carbamates and synthetic pyrethroids. Synthetic pyrethroids possess a unique combination of desirable properties including exceptional insecticidal activity, low mammalian toxicity, rapid biodegradation and little or no persisting residues [2], and pyrethroid insecticides have become a major class of insecticides for pest insect control. With heavy application of these chemical insecticides, cotton aphids have developed different levels of resistance to pyrethriods, such as cypermethrin, alpha-cypermethrin, cyfluthrin, bifenthrin and deltamethrin [1,3–6]. Some researches have confirmed the pyrethroid-resistance mechanisms in *A. gossypii* included the increased activity of detoxifying enzymes including P450s, GSTs esterases [4,5,7,8] and target site insensitivity (*Kdr*) [7,8]. The esterase-mediated resistance has been found as a prevalent resistant mechanism in *Boophilus microplus* [9], *Culex* mosquito [10] and *A. gossypii* [4–6]. Cao et al. have reported the increased expression of the carboxylesterase due to the high transcription levels of carboxylesterase mRNA was related to deltamethrin resistance in cotton aphids [5].

Pyrethroid resistance is still a major problem in the resistance management program of cotton aphid. Synergists with specific inhibition to some detoxification enzymes have long been exploited as tools for the laboratory diagnosis of particular insecticide resistance mechanisms. Piperonyl butoxide (PBO) has been used as a tank mix to improve the efficacy of pyrethroids, carbamates and neonicotinoids against resistant pests resulting in effective control highly resistant *Helicoverpa armigera*, B-type *Bemisia tabaci*, *Myzus persicae* and *Musca domestica* [11–13]. Esterase inhibition by PBO did not occur immediately after dosing, but reached a maximal enzyme inhibition (64%) after 11 h in pyrethroid-resistant B-type *B. tabaci* [11]. PBO exhibited maximum esterase inhibition in 3–4 h after dosing in *H. armigera* [12]. S,S,S-tributyl phosphorotrithioate (DEF) is a widely recognized insecticide synergist that functions by inhibiting hydrolytic metabolism [14]. The research result of Ahmad and Hollingworth [14] exhibited that DEF synergized indoxacarb, cypermethrin, chlorpyrifos, azinphosmethyl and tebufenozide in the resistant strain of *Choristoneura rosaceana*.

In the present study, we established a deltamethrin-resistant strain of *A. gossypii* by continuous selection with deltamethrin. The synergism and esterase-inhibition effects of DEF against this strain were investigated. These results are useful to clarify the pyrethroid-resistance mechanism and synergism of DEF in *A. gossypii*.

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2. Materials and methods

2.1. A. gossypii strain

The parental colony of *A. gossypii* was supplied by Donghai Zhang (Shihezi University, Xinjiang Uygur Autonomous Region, China). Two strains of cotton aphid *A. gossypii* were obtained as follows. The progeny of the parental colony was divided into two groups. One group was used for deltamethrin susceptible (DSS) selection by continuous opposite selection for four generations. The other group was used for deltamethrin resistant (DRR) selection with deltamethrin for 30 generations according to Moores et al. [15]. All the aphid clones were reared in the insectary at 20–23 °C on a photoperiod of 16:8 (L:D) h and fed with fresh cotton leaves.

2.2. Chemicals

Deltamethrin (98%) was supplied by Bayer and bifenthrin (97%) and ethofenprox (95%) were provided by Jiangsu Yangnong Chemical Group Co. Ltd. S,S,S-tributylphosphorotrithioate (DEF, 98%) was purchased from Chem Service (West Chester, PA). α-Naphthyl acetate (α-NA), Fast Blue RR salt, α-naphthol, sodium dodecyl sulfate (SDS) and bovine serum albumin (BSA) were products of Sigma Chemical Co. (St. Louis, MO) at the highest purity available. All other chemicals and solvents were purchased from commercial suppliers.

2.3. Bioassays

The toxicity of deltamethrin, bifenthrin and ethofenprox to the resistant and susceptible strains of *A. gossypii* was determined by the leaf-dipping method described by Moores et al. [15]. A stock of insecticide was prepared in acetone and diluted to five to seven concentrations with distilled water containing 0.05% (v/v) Triton X-100 and 1% acetone for bioassays. Cotton leaves were dipped into insecticide solution for 5 s and then placed in the shade to air dry. Bioassays were carried out by exposing 60 apterous adults to insecticide-treated leaves. Control cotton aphids were treated with distilled water containing 0.05% (v/v) Triton X-100 and 1% acetone. Each concentration was replicated at least three times, and the mortality was assessed at 25 °C, 24 h after the treatment. LC_{50} value was calculated by POLO software (LeOra Software Inc., Cary, NC).

2.4. Synergism bioassays

Toxicity of insecticide in the presence and absence of synergist DEF was evaluated by bioassay method as described previously. According to Sun et al. [16], the maximum sublethal dose and treatment time for synergist based on the susceptible strain were determined with the method of bioassay as in Section 2.3. At least five concentrations of DEF and a control only containing acetone were used. The maximum dose lead to zero mortality in the susceptible strain was adopted as the maximum sublethal concentrations in our study. Cotton aphids were exposed to cotton leaf discs (22 mm diameter), which were treated with synergist, DEF (which was applied at the maximum sublethal dose, final concentrations was 30 μg/ml), for varying periods of time up to 24 h, and then were transferred to treated leaf with pyrethroid insecticides. For estimated the effect of DEF + insecticides mix, cotton aphids were exposed to cotton leaves treated with DEF + insecticides mix. Mortality was recorded after 24 h. Synergistic ratio was calculated using the conventional approach of dividing the LC_{50} without DEF by the LC_{50} with DEF.

Esterase activity was determined after cotton aphids were exposed to the treated cotton leaf discs with DEF (final concentrations was 30 μg/ml) for varying periods of time up to 24 h.

2.5. Measurement of carboxylesterase activity

The carboxylesterase activity was measured using the method described by Moores et al. [15], with modification. Wingless adult females were homogenized in 1000 μL of ice-cold phosphate buffer (0.04 M, pH 7.0) containing 0.1% Triton X-100. The homogenates were centrifuged at 4 °C, 12,000*g* for 15 min, and resulting supernatants were diluted 25-fold for the susceptible strain and 100-fold for the resistance strain respectively with the same buffer. Twenty-five micro liter aliquots were removed and added to a new microplate where the final volume was adjusted to 50 μL with buffer and used for subsequent carboxylesterase activity assay. The reaction was started by adding 200 μL mixture solution containing 100 μM α-naphthyl acetate (containing eserine 0.3 mM) and 6 mg/mL Fast Blue RR salt (diazotized 4-(benzoylamino)-2,5-dimethoxyaniline/$ZnCl_2$) to each column of wells using eight-channel multipipettes. The assay was run for 15 min at 450 nm in a microplate reader (Spectra Rainbow; TECAN, Austria), utilizing SOFT 2000 to fit linear regressions to the kinetic plot, and the slopes were determined as the activity of the carboxylesterases.

2.6. Determination of carboxylesterase inhibition

The carboxylesterase inhibition was measured using the method described by Liang et al. [17], with modification. One hundred wingless adult females with similar size and color from each strain were homogenized in 1000 μL of ice-cold phosphate buffer (0.04 M, pH 7.0) containing 0.1% Triton X-100. The homogenates were centrifuged at 4 °C, 12,000*g* for 15 min. The supernatant was diluted 25-fold for the susceptible strain and 100-fold for resistance strain respectively with the same buffer. The diluted supernatants were used as the enzyme source for inhibition experiments.

A stock solution of DEF was prepared in acetone, and serial diluted of DEF from 0.26 to 100 mM with a solution of 100 μM α-NA (containing 0.3 mM eserine) + Fast Blue RR salt (6 mg/mL) as inhibitor solution. The inhibition of DEF on carboxylesterase was determined by adding 200μL inhibitor solution into each column of wells containing 50 μL of enzyme source, and the reactions were monitored for 15 min in the microplate reader. A row of wells without inhibitor served as a control for normal carboxylesterase activity determination. Inhibiting rate was summarized by expressing the carboxylesterase activity remaining as a percentage of the uninhibited activity. IC_{50} values were concentration of inhibitor resulting in a 50% reduction in enzyme activity. Three repetitions were set up for each concentration of inhibitor.

3. Results

3.1. Pyrethroid toxicity and synergism bioassay

Results of pyrethroid and DEF bioassays in *A. gossypii* are shown in Table 1. LC_{50} value of deltamethrin for the resistant strain (DRR) is 23,900-fold of the susceptible strain (DSS). Cross-resistance of the DRR strain to bifenthrin was 7560-fold and only 99.0-fold to ethofenprox compared with the DSS strain.

DEF increased deltamethrin efficacy by a DEF pretreatment of 15 h, and the LC50 value of deltamethrin for resistance strain decreased gramatically from 25,800 to 5390 mg/L. However, no synergism of DEF was observed in DSS strain with LC_{50} values 1.08 mg/L without DEF and 0.95 mg/L with DEF pretreatment respectively.

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