Side-effects of insecticides used in tomato fields on *Trichogramma pretiosum* (Hymenoptera, Trichogrammatidae)

Geraldo Andrade Carvalho[1], Paulo Rebelles Reis[2], Luiz Carlos D. Rocha[1], Jair Campos Moraes[1], Loriney Costa Fuini[1] and Carvalho Carlos Ecole[1]

*[1]Departamento de Entomologia, Universidade Federal de Lavras, C.P. 37, 37.200-000, Lavras, Minas Gerais, Brasil. [2]Epamig-CTSM/EcoCentro, C.P. 176, 37.200-000, Lavras, Minas Gerais, Brasil. *Author for correspondence. e-mail: gacarval@ufla.br*

ABSTRACT. The side-effects of lufenuron (0.4g a.i./L), triflumuron (0.15g a.i./L), imidacloprid (0.28g a.i./L), cyromazine (0.11g a.i./L), methoxifenozide (0.12g a.i./L), pirimicarb (0.25g a.i./L) and abamectin (0.18g a.i./L) on different developmental stages of the egg parasitoid *Trichogramma pretiosum* Riley, were investigated. Abamectin was the only insecticide to affect parasitoid emergence and sex ratio, regardless of the developmental stage and parasitoid generation exposed. Abamectin, lufenuron and pirimicarb also decreased the lifetime of F_1 females exposed during the egg-larva stage. The capacity of parasitism was significantly reduced by all the products when females were treated in pupal stage.

Key words: selectivity, egg parasitoid, biological control, integrated pest management.

RESUMO. Efeitos colaterais de inseticidas utilizados em campos de tomate sobre *Trichogramma pretiosum* (Hymenoptera, Trichogrammatidae). Avaliou-se a seletividade dos inseticidas lufenurom (0,4g i.a./L), triflumurom (0,15g i.a./L), imidaclopride (0,28g i.a./L), ciromazina (0,11g i.a./L), metoxifenozide (0,12g i.a./L), pirimicarbe (0,25g i.a./L) e abamectina (0,18g i.a./L) para *Trichogramma pretiosum* Riley. Abamectina foi o único inseticida prejudicial à emergência e a razão sexual, independentemente do estágio de desenvolvimento dos embriões expostos ou da geração do parasitóide pesquisado. Abamectina, lufenurom e pirimicarbe também prejudicaram o tempo de vida de fêmeas da F_1 que receberam esses inseticidas no período de ovo-larva. A capacidade de parasitismo foi reduzida significativamente por todos os produtos quando fêmeas foram tratadas em sua fase de pupa.

Palavras-chave: seletividade, parasitóide de ovos, controle biológico, manejo integrado.

Introduction

Tomato (*Lycopersicon esculentum* Mill.) is cultivated in about 40.000ha throughout Brazil and the production is up to two million tons (Lopes and Santos, 1994). There are about 200 arthropod species associated with the tomato crop (Lange and Bronson, 1981), and the intensive and widespread use of pesticides required for the pest control in this crop has led to the development of pesticide resistance in key tomato pests, such as the tomato leafminer *Tuta absoluta* (Meyrick) (Siqueira *et al*., 2000a, b; 2001). The social-economic importance of this crop and the environmental risks, due to the management practices employed, support an ever-increasing research interest in the phytosanitary problems of this crop (Michereff Filho and Vilela, 2001).

The genus *Trichogramma* shows different species responsible for natural control of several insects-pest, mainly lepidopterous. Faria *et al*. (2000) related that the species *Trichogramma pretiosum* Riley, has been studied in Brazil for the control of several pests in corn, cassava and cotton crops. Furthermore, in the tomato crop, the control of the moth *T. absoluta* has been studied in some countries, through inundative releases of *T. pretiosum* (Amaya-Navarro, 1988; Faria Jr., 1992; Haji, 1996, 1997). The use of parasitoids of the genus *Trichogramma* has also been evaluated for the control of other pests in this crop. Blackmer *et al*. (2001), accomplishing experiments in tomato crop, verified natural parasitism rates of *Neoleucinodes elegantalis* (Guenée) eggs for *T. pretiosum* about 28.7%, with the reduction of pesticide utilization.

The utilization of selective insecticides is a reasonable strategy in pest management, because it favors the conservation of natural enemies in the agroecosystem (Carvalho *et al*., 1999).

Works aiming to study the physiologic selectivity of different chemical groups of pesticides to *Trichogramma* spp. were accomplished. Cônsoli *et al*. (1998), evaluating the effects of several insecticides on the immature stages of *T. pretiosum*, verified that phenthoate and cartap were harmful, lambda-cyalothrin and abamectin were intermediate, tebufenozid and teflubenzuron were harmless to slightly harmful. Brunner *et al*. (2001) using Potter's tower, sprayed insecticides on adults of *T. platneri* Nagarkatti up to two days old and observed that the compounds oxamyl, imidacloprid and *Bacillus thuringiensis kurstaki* caused 100% mortality 48 hours after spraying, being observed selectivity of the products diflubenzuron, fenoxycarb and tebufenozide for that species.

This paper intends to study the impact of some pesticides, at dosage rates currently used in Brazilian tomato fields, to immature stages of the egg parasitoid *T. pretiosum*.

Material and methods

Fifteen newly-emerged females of *T. pretiosum* were individualized in glass tubes (8.5cm x 2.5cm) and, subsequently blue paper cards (3.0cm x 0.5cm) with eggs of *Anagasta kuehniella* (Zeller) previously UV-killed (±125 eggs) were offered. The parasitism period was 24h. Afterwards, the females were removed and the paper cards were maintained under controlled conditions at 25±2ºC, 70±10% RH and 14h of photophase for *T. pretiosum* development.

Pesticides used in the selectivity tests against *Trichogramma pretiosum* were: lufenuron (0.4g a.i./L), triflumuron (0.15g a.i./L), imidacloprid (0.28g a.i./L), cyromazine (0.11g a.i./L), methoxifenozide (0.12g a.i./L), pirimicarb (0.25g a.i./L) and abamectin (0.18g a.i./L). Eggs of *A. kuehniella* containing *T. pretiosum* at different developmental stages (egg-larval, prepupal and pupal with 24h, 96h and 192h after parasitism, respectively) were treated by dipping them in the insecticide solution for five seconds. After elimination of water excess from the egg surface, paper cards were individualized in glass tubes (8.5cm x 2.5cm) and maintained at the same conditions as previously described.

To evaluate the pesticide effects, 15 newly-emerged females from treated eggs (F_1 generation) were randomly taken from each treatment, individualized in glass tubes (8.5cm x 2.5cm) and provided with non-treated and inviabilized eggs of *A. kuehniella* for 48 hours, allowing the assessment of testing the pesticide side-effects in individuals of the next generation.

The biological parameters evaluated were: 1) the number of eggs parasitized by F_1 generation insects; 2) the parasitoid emergency of F_1 and F_2 generations; 3) the parasitoid of sex ratio in the egg-larval, prepupal and pupal stages and also 4) the F_1 female longevity.

The experimental design used was completely randomized with eight treatments (seven pesticides and a control treatment in which only water was applied) and 15 replicates, analyzing the pesticide effects on three parasitoid developmental stages and in two generations. A paper card containing about 125 parasitized host eggs was the experimental unit used.

The number of parasitized eggs and the longevity were transformed to $\sqrt{x+0.5}$, and the parasitoid emergency was transformed to $\text{arcsen}\sqrt{x/100}$ before subjected to Anova. The comparisons between treatments were carried out using Scott and Knott's groupment analysis test at 5% probability (Scott and Knott, 1974).

Results and discussion

Impact of pesticides on the F_1 and F_2 emergence of *T. pretiosum*

Abamectin was the pesticide which most affected parasitoid emergence when used at the egg-larval stage of *T. pretiosum*, independently of the generation. Cyromazine, imidacloprid and methoxifenozide were toxic for individuals of the generations F_1 and F_2. Lufenuron showed no effects at this stage (Table 1).

For the F_1 generation, when prepupae of *T. pretiosum* where exposed to pesticides, parasitoid emergence rates were smaller for abamectin, imidacloprid and lufenuron (Table 1). Despite the relatively high emergence rate observed with abamectin application, allowing 70% emergence of *T. pretiosum*, all the surviving insects died soon afterwards and subsequently there parasitism ability could not assessed (Table 2). This mortality may be due to the contact of the parasitoids with abamectin residue through the host egg chorion. The insects treated with cyromazine, methoxifenozide, pirimicarb and triflumuron showed emergence rates similar to those of the control treatment, suggesting their safety to parasitoids of the F_1 generation. Cyromazine, imidacloprid, lufenuron, methoxifenozide, triflumuron and pirimicarb allowed more than 80% emergence for parasitoids of the F_2 generation. Abamectin was highly toxic to *T. pretiosum* (Table 1).

Table 1. Emergence (%) (± SEM) of *Trichogramma pretiosum,* in the generations F_1 and F_2 treated with insecticides during the egg-larval, prepupal and pupal stages when developing in *Anagasta kuehniella* eggs

Treatments	Egg-larval stage	
	F_1	F_2
Abamectin	49.1 ± 3.6 aC	49.1 ± 3.3 aC
Cyromazine	74.1 ± 4.4 aB	69.9 ± 3.4 aB
Imidacloprid	68.4 ± 3.8 aB	68.4 ± 4.9 aB
Lufenuron	91.0 ± 1.4 aA	80.9 ± 2.9 bA
Methoxifenozide	76.6 ± 3.4 aB	65.3 ± 3.2 bB
Pirimicarb	79.7 ± 3.3 aB	84.0 ± 3.0 aA
Triflumuron	88.3 ± 1.6 aA	64.0 ± 4.8 bB
Control	89.6 ± 1.6 aA	88.1 ± 2.1 aA
Treatments	**Prepupal stage**	
	F_1	F_2
Abamectin	70.0 ± 2.4 aB	17.5 ± 9.4 bB
Cyromazine	80.7 ± 4.5 bA	93.4 ± 1.4 aA
Imidacloprid	71.5 ± 4.5 bB	92.1 ± 2.1 aA
Lufenuron	69.7 ± 2.1 bB	93.1 ±1.4 aA
Methoxifenozide	84.7 ± 2.4 aA	90.7 ± 3.5 aA
Pirimicarb	90.4 ± 2.8 aA	82.0 ± 3.3 bA
Triflumuron	81.4 ± 3.0 bA	88.8 ± 2.5 aA
Control	89.3 ± 2.5 aA	86.0 ± 3.1 aA
Treatments	**Pupal stage**	
	F_1	F_2
Abamectin	41.1 ± 3.3 bC	49.7 ± 3.3 aC
Cyromazine	70.4 ± 3.8 bB	81.2 ± 3.1 aA
Imidacloprid	70.3 ± 4.3 bB	79.6 ± 3.1 aA
Lufenuron	72.8 ± 3.2 aB	71.4 ± 3.8 aB
Methoxifenozide	74.1 ± 2.3 bB	79.0 ± 4.0 aA
Pirimicarb	76.7 ± 3.3 bA	82.3 ± 2.7 aA
Triflumuron	63.3 ± 4.2 bB	67.3 ± 3.1 aB
Control	82.2 ± 2.4 bA	84.3 ± 2.4 aA

* Means followed by the same capital letter in the column and lower case in the line are not significantly different by the Scott and Knott test at 5%

There were significant effects of the pesticides on parasitoid emergence when they were exposed during their pupal stage (Table 1). In F_1, the highest emergence rate was observed for pirimicarb, differing from those obtained with cyromazine, imidacloprid, lufenuron, methoxifenozide and triflumuron, which showed intermediate toxicity. The drastic reduction in the emergence rate of *T. pretiosum* was observed with abamectin. This last pesticide also affected insect emergence at the following generation, allowing only 49.7% emergence. Lufenuron and triflumuron also reduced the emergence of *T. pretiosum*. No significant difference was observed between the treatments cyromazine, imidacloprid, methoxifenozide, pirimicarb and control, for the individuals of F_2 generation (Table 1).

The results reported here resemble those of Carvalho *et al*. (2001) and Cônsoli *et al*. (1998), who applied abamectin on eggs of *A. kuehniella* containing the parasitoid *T. pretiosum* in the egg-larval, prepupal and pupal stages, and verified the reduction of the emergence rate from 10 to 40%.

Table 2. Number of parasited eggs/female (±SEM) and longevity (±SEM) of *Trichogramma pretiosum* in the F_1 generation, treated with insecticides during the egg-larval, prepupal and pupal stages when developing in *Anagasta kuehniella* eggs

Treatments	Number of parasited eggs/female*		
	Egg-larval stage	Prepupal stage	Pupal stage
Abamectin	17.3 ± 1.4 aD	-	17.7 ± 1.4 aC
Cyromazine	35.5 ± 2.7 aC	28.1 ± 2.1 bA	34.6 ± 1.8 aB
Imidacloprid	39.4 ± 2.4 aB	27.8 ± 2.9 bA	39.9 ± 2.9 aB
Lufenuron	41.1 ± 2.0 aA	31.6 ± 1.5 bA	37.8 ± 1.9 aB
Methoxifenozide	45.4 ± 2.3 aA	27.6 ± 1.8 bA	39.8 ± 2.7 bB
Pirimicarb	35.5 ± 2.7 aC	27.8 ± 2.2 bA	38.0 ± 2.5 aB
Triflumuron	44.4 ± 1.8 aA	30.6 ± 2.3 bA	41.4 ± 2.9 aB
Control	51.7 ± 3.2 aA	32.1 ± 2.5 bA	54.3 ± 2.6 aA
Treatments	**Longevity (days)***		
	Egg-larval stage	Prepupal stage	Pupal stage
Abamectin	9.3 ± 0.5 aD	-	10.2 ± 0.6 aB
Cyromazine	14.0 ± 0.7 aB	13.1 ± 0.7 aB	13.7 ± 0.6 aA
Imidacloprid	12.7 ± 0.7 aB	13.5 ± 0.9 aB	12.9 ± 0.5 aA
Lufenuron	10.7 ± 0.4 aC	11.6 ± 1.2 aB	11.1 ± 0.4 aB
Methoxifenozide	12.8 ± 0.6 bB	15.6 ± 0.9 aA	13.2 ± 0.5 bA
Pirimicarb	11.3 ± 0.6 bC	14.7 ± 0.9 aA	11.9 ± 0.8 bB
Triflumuron	11.6 ± 0.6 bC	13.6 ± 0.7 aB	13.4 ± 0.6 aA
Control	16.1 ± 1.2 aA	16.3 ± 0.9 aA	12.4 ± 0.8 bA

* Means followed by the same capital letter in the column and lower case in the line are not significantly different by the Scott and Knott test at 5%

Carvalho *et al*. (2001) did not observe significant reduction in emergence of parasitoids when *T. pretiosum* were treated with cyromazine, triflumuron and pirimicarb in the egg-larval stage. This different results may have occurred due to the different origin of the parasitoid populations used in the present study, which may influence the quality of the parasitoid population and the intensity of the insect exposition to pesticide residues (Prezotti *et al*., 1994; Pratissoli and Parra, 2001).

Regarding the prepupal and pupal stages, Cônsoli *et al*. (1998) also observed similar results to those reported in the present study with lufenuron and abamectin. Carvalho *et al*. (2001) also noted similar effect for the abamectin, cyromazine, pirimicarb and triflumuron on the same parasitoid species. Regarding the pupal stage, the results with triflumuron resemble those reported by Narayana and Babu (1992) for *T. chilonis* Ishii.

Pesticide impact on sex ratio of *T. pretiosum*

There was a significant effect of pesticides on sex ratio (Table 3). When the pesticides were applied during the egg-larval, prepupal and pupal stages, the smallest number of females was observed with abamectin (0.38, 0.37 and 0.41, respectively). For the egg-larval and prepupal stages, the sex ratio observed with cyromazine was not different from the control indicating its selectivity to the parasitoid based on this parameter (Table 3). Selective effect of cyromazine on the developmental stages (egg-larval, prepupal and pupal) of *T. pretiosum* has also been previously reported (Carvalho *et al.*, 2001).

Table 3. Sex ratio (± SEM) of *Trichogramma pretiosum* treated with insecticides during the egg-larval, prepupal and pupal stages when developing in *Anagasta kuehniella* eggs

Treatments	Sex ratio/Stage of *T. pretiosum*		
	Egg-larval stage	Prepupal stage	Pupal stage
Abamectin	0.38 ± 0.02 C	0.37 ± 0.01 C	0.41 ± 0.15 C
Cyromazine	0.60 ± 0.03 A	0.61 ± 0.02 A	0.61 ± 0.06 B
Imidacloprid	0.55 ± 0.15 B	0.54 ± 0.05 B	0.55 ± 0.15 B
Lufenuron	0.51 ± 0.02 B	0.51 ± 0.02 B	0.56 ± 0.15 B
Methoxifenozide	0.57 ± 0.12 B	0.58 ± 0.23 B	0.59 ± 0.25 B
Pirimicarb	0.55 ± 0.02 B	0.54 ± 0.02 B	0.53 ± 0.16 B
Triflumuron	0.55 ± 0.13 B	0.57 ± 0.03 B	0.60 ± 0.19 B
Standard	0.68 ± 0.15 A	0.67 ± 0.32 A	0.63 ± 0.18 A

* Means followed by the same capital letter in the column are not significantly different by the Scott and Knott test at 5%

All the pesticides compromised the sex ratio of *T. pretiosum* when the host eggs containing the parasitoid in the pupal stage were treated; however, abamectin showed the highest distortions on sex ratio (Table 3).

Pesticide impact on the parasitism by *T. pretiosum*

There was a significant effect of the pesticides on egg parasitism by *T. pretiosum* of the F_1 generation. Abamectin reduced in 66.54% the number of parasitized eggs when applied at egg-larval stage. Cyromazine, pirimicarb and imidacloprid showed an intermediate effect on parasitism, while triflumuron, lufenuron and methoxifenozide results did not differ from control (Table 2).

When the insecticides were applied on the insects during the prepupal stage, there was no significant reduction of the number of eggs parasitized by *T. pretiosum*. On the other hand, insects exposed to abamectin died soon after emergence, not allowing the parasitism of the females. This was possibly due to the contact with residues of this pesticide through the host egg chorion, as reported before.

When pesticides were applied on *A. kuehniella* eggs in which parasitoids were at the pupal stage, abamectin remained the most harmful pesticide. The other pesticides were less toxic, but they significantly reduced the number of parasitized eggs per female of the F_1 generation (Table 2).

The results found here confirm those obtained by Carvalho *et al.* (2001) with the growth regulator triflumuron when applied on the egg-larval and prepupal stages of the *T. pretiosum*. Cônsoli *et al.* (1998) also reported a reduction in the parasitism of females emerged from eggs of *A. kuehniella* exposed to these two insecticides.

Pesticide impact on the longevity of F_1 of *T. pretiosum*

Females of the F_1 generation of *T. pretiosum* exposed to abamectin during the egg-larval stage had their longevity reduced (42.2%). The other pesticides were less toxic, but they significantly reduced the females longevity of the F_1 generation (Table 2).

The longevity of females treated at the prepupal stage with methoxifenozide and pirimicarb was not affected. However, abamectin has proved extremely toxic, killing the insects still inside the host egg, or after their emergence. The remaining insecticides showed intermediary effect (Table 2).

Cyromazine, imidacloprid, methoxifenozide and triflumuron did not affect the longevity of parasitoids female when they were exposed during the pupal stage. The pesticides abamectin, lufenuron and pirimicarb significantly reduced the longevity of the females treated at the pupal stage (Table 2).

The results here reported agree with those of Carvalho *et al.* (2001). However, Carvalho *et al.* (2001) did not observe some harmful effects of triflumuron on *T. pretiosum*, diverging from this study, since it was observed here that triflumuron was slightly toxic to females of *T. pretiosum.* These divergences are probably associated with the differences between populations of *T. pretiosum* (Carvalho *et al.*, 2001). Zaki and Gesraha (1987) also reported that diflubenzuron, another acylurea as lufenuron and triflumuron, reduced longevity of *T. evanescens* (Westwood) in about 44%.

The results showed that, regardless of the development stage of *T. pretiosum* and the generation, the pesticide abamectin is highly toxic to this species. Abamectin, lufenuron and pirimicarb reduced the longevity and the number of parasitized eggs when the parasitoids were exposed at the egg-larval stage. The prepupal stage was the most tolerant to the studied pesticides, except for abamectin.

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