# Selectivity of growth regulators and neonicotinoids for adults of *Trichogramma pretiosum* (Hymenoptera: Trichogrammatidae)

La Selectividad de los reguladores de crecimiento y neonicotinoides para los adultos de *Trichogramma pretiosum* (Hymenoptera: Trichogrammatidae)

GERALDO ANDRADE CARVALHO[1*], MAURÍCIO SEKIGUCHI GODOY[1,2], DOUGLAS SILVA PARREIRA[1,3], OLINTO LASMAR[1,4], JANDER RODRIGUES SOUZA[1,5] and VALERIA FONSECA MOSCARDINI[1,6]

**Abstract:** The objective of this study was to evaluate the residual and sublethal effects of the insecticides acetamiprid (0.05 g a.i./L), lufenuron (0.04 g a.i./L), imidacloprid (0.14 g a.i./L), novaluron (0.02 g a.i./L), triflumuron (0.14 g a.i./L), and pyriproxifen (0.1 g a.i./L) on adults from the maternal generation of *Trichogramma pretiosum*, as well as their subsequent effects on the $F_1$ and $F_2$ generations. Eggs of *Anagasta kuehniella* were glued to paper cards, UV-killed, and dip-treated in insecticide solutions. Next, the eggs were exposed for parasitism 1, 24, and 48 h after treatment, maintained that way for 24 h under controlled conditions (24 ± 2°C, 70 ± 10% relative humidity, 12-h photophase), until the emergence of the parasitoids. The insecticide toxicity was calculated based on the longevity and parasitism capacity of the maternal generation, as well as on the emergence rate, sex ratio, longevity, and parasitism capacity of the $F_1$ and $F_2$ generations. The insecticides were toxicologically classified according to IOBC. Pyriproxifen was slightly harmful (class 2) for the parasitism capacity of *T. pretiosum* maternal and $F_1$ generation females. Novaluron was slightly harmful for the emergence rate of the $F_1$ generation. Acetamiprid, imidacloprid, lufenuron, and triflumuron were harmless (class 1) to *T. pretiosum*.

**Key words:** Solanaceae. Egg parasitoids. Pesticides. Side-effects.

**Resumen:** Se evaluaron los efectos residuales y subletales de insecticidas acetamiprid (0,05 g i.a./L), lufenurón (0,04 g i.a./L), imidacloprid (0,14 g i.a./L), novaluron (0,02 g i.a./L), triflumurón (0,14 g i.a./L) y piriproxifen (0,1 g i.a. L) en la generación parental de adultos de *Trichogramma pretiosum*, tanto como sus efectos posteriores sobre las generaciones $F_1$ y $F_2$. Huevos de *Anagasta kuehniella* adheridos con goma árabe a cuadros de cartón fueron sacrificados con luz UV y tratados por inmersión en las soluciones de insecticida. Luego, se expusieron al parasitismo 1, 24 y 48 horas después del tratamiento durante 24 horas bajo condiciones controladas (24 ± 2°C, humedad relativa 70 ± 10%, 12-h fotofase) hasta la emergencia de los parasitoides. Se calculó la toxicidad de los insecticidas basados en la longevidad y la capacidad de parasitismo de las hembras de la generación materna, así como en la tasa de emergencia, la proporción de sexos, la longevidad y la capacidad de parasitismo de las generaciones $F_1$ y $F_2$. Los compuestos se clasificaron según la IOBC. Piriproxifen fue levemente perjudicial (clase 2) para la capacidad de parasitismo de las hembras maternales y la generación $F_1$ de *T. pretiosum*. Novaluron fue levemente perjudicial para la emergencia de la generación $F_1$. Acetamiprid, imidacloprid, lufenurón y triflumurón fueron inocuos (clase 1).

**Palabras clave:** Solanaceae. Parasitoides de huevos. Pesticidas. Efectos colaterales.

## Introduction

Parasitoid insects are well known for their efficient control of pests in several cultures. Among these pest control agents, those from the *Trichogramma* genus have attracted attention worldwide (Scholz *et al.* 1998) for parasitizing eggs and killing hosts before pest emergence and plant attack (Lundgren *et al.* 2002).

In Brazil, 28 species of *Trichogramma* have been reported in almost all regions (Querino and Zucchi 2003) and associated with hosts such as *Tuta absoluta* (Meyrich, 1917) (Lepidoptera: Gelechiidae), *Neoleucinodes elegantalis* (Guenée, 1854) (Lepidoptera: Pyralidae), and *Helicoverpa zea* (Boddie, 1850) (Lepidoptera: Noctuidae), which are tomato crops pests (Zucchi and Monteiro 1997). Due to the importance of the *Trichogramma* species as a natural enemy of several tomato culture pests, studies on its use as a biological pest control agent together with other methods, particularly insecticides, as they are still used in large quantities in pest control in tomato crops, are fundamental. The information obtained will be instrumental in decision-making in integrated pest management programs aiming at the use of these natural enemies in agroecosystems, the reduction of pesticide use, and the minimization of the related human health hazards (Ruberson and Tillman 1999; Carvalho *et al*. 2001; Medina *et al*. 2003; Moura *et al*. 2005).

Thus, the present work aimed to evaluate the residual and sublethal effects of the new insecticides recommended for tomato crops on adult specimens of the maternal generation of *Trichogramma pretiosum* Riley, 1879 and $F_1$ and $F_2$ generation parasitoid specimens.

## Material and Methods

Bioassays were carried out with *T. pretiosum* adult insects collected from *Spodoptera frugiperda* (J. E. Smith, 1797) (Lepidoptera: Noctuidae) eggs in a maize crops in Piraçicaba city, São Paulo, Brazil. The parasitoid was reared on eggs

[1] Selectivity of pesticides for natural enemies. Universidade Federal de Lavras (UFLA), Departamento de Entomologia, Lavras, Minas Gerais, Brazil, C.P. 3037, CEP: 37200-000. * Prof. Dr. in Entomology, *gacarval@den.ufla.br.* Corresponding Author. [2] Pós Dr. in Entomology. [3] M. Sc. in Entomology. [4,5,6] M. Sc. students in Agronomy / Entomology.

of factitious host *Anagasta kuehniella* (Zeller, 1879) (Lepidoptera: Pyralidae) in laboratory at 2 4 ± 2°C , 70 ± 10% relative humidity, and 12-h photophase.

The assayed insecticides in dosages higher recommended by the manufacturer for the tomato crop were acetamiprid (0.05 g a.i./L), lufenuron (0.04 g a.i./L), imidacloprid (0.14 g a.i./L), novaluron (0.02 g a.i./L), triflumuron (0.14 g a.i./L), and pyriproxifen (0.1 g a.i./L). Distilled water was used as a control treatment. Newly-emerged females (20) were submitted to individual treatment in 8-cm x 2.5-cm glass tubes and fed with honey droplets laid on the inside wall of the tubes. The tubes were closed with polyvinyl chloride (PVC) film.

About 125 eggs of *A. kuehniella* aged no more than 24 h were glued to 5cm x 0.5cm paper strips with 50% Arabic gum diluted in distilled water. The eggs were sterilized with a germicidal lamp (Parra 1997) and dip-treated in insecticide solutions or distilled water (control) for 5 s. The strips with the treated eggs were presented to *T. pretiosum* females one, 24, and 48 hours after treatment for 24 h. Next, the females were kept in the tubes and the paper strips with supposedly parasitized eggs were transferred to new recipients and kept in acclimatized chamber in the conditions previously described until the emergence of the $F_1$ generation.

The $F_1$ generation females newly-emerged from treated *A. kuehniella* eggs were placed in individual glass tubes with untreated host eggs glued to new paper strips. The same procedures described before for the maternal generation females were adopted in this step (number of females, paper strip size, number of host eggs). The longevity and parasitism capacity of the maternal generation females, the emergence ratio, the sex ratio, and the longevity and parasitism capacity of $F_1$ and $F_2$ generation specimens, were evaluated. Each treatment involved five repetitions. The control treatment involved four paper strips with parasitized host eggs. A completely randomized three x seven (three periods of parasitoid development vs. seven substances, totaling 21 treatments) factorial experimental design was used. The data obtained were submitted to variance analysis and the means were compared by the Scott-Knott grouping test at 5% significance (Scott and Knott 1974).

The evaluated insecticides were toxicologically classified in relation to their reduction of the parasitism capacity of maternal, $F_1$, and $F_2$ generation females, as well as the emergence of $F_1$ and $F_2$ generation specimens in relation to the control treatment as follows: 1 = harmless (< 30% reduction), 2 = slightly harmful (30-79% reduction), 3 = moderately harmful (80-99% reduction), and 4 = harmful (> 99% reduction), as recommended by the "International Organization for Biological and Integrated Control of Noxious Animals and Plants" (IOBC) (Sterk *et al*. 1999). The mean percent reduction of survival of the parasitoid was calculated with the following equation: % reduction = 100 - [(% general mean of the treatment with the insecticide/% general mean of the control treatment) x 100].

## Results and Discussion

**Longevity of Maternal Generation.** Insecticides acetamiprid, imidacloprid, lufenuron, triflumuron, and novaluron reduced the longevity of maternal generation females exposed to their residues 1 h after the treatment of the host eggs. Pyriproxifen was the only insecticide that did not affect this biological characteristic. No significant differences were observed in longevity of females exposed to treated host eggs 24 h after the insecticide treatment (Table 1). In turn, acetamiprid and pyriproxifen reduced the longevity of females exposed to treated host eggs 48 h after treatment by 6.1 and 6.0 days on average, respectively. Similar results were obtained for the toxicity of imidacloprid by Moura *et al*. (2004), who reported a reduction in longevity for females of *T. pretiosum* exposed to *A. kuehniella* eggs treated with 1.16 a.i./L of this insecticide 1 h after treatment.

No differences were observed in the mean longevity of *T. pretiosum* females exposed to host eggs treated with acetamiprid, lufenuron, and triflumuron between the exposure periods. Imidacloprid was the only insecticide that did not affect the longevity of females exposed to treated eggs after 48 h, while pyriproxifen reduced the longevity of females 24 and 48 h after exposure in relation to females exposed to host eggs 1 h after treatment. The longevity of females exposed to imidacloprid and novaluron increased with time. When the eggs were presented 1 h after treatment, the female longevity was 5.9 and 5.5 days, respectively. However, the exposure to host eggs 48 h after the treatment resulted in mean parasitoid longevity values of 7.8 and 8.6 days, respectively (Table 1).

**Eggs Parasitized by Maternal Generation.** The parasitism capacity of *T. pretiosum* females exposed to host eggs one, 24, and 48 h after treatment with pyriproxifen was reduced, presenting means of 21.8, 18.9, and 18.1 parasitized eggs per female, respectively, without significant differences

**Table 1.** Longevity (days) (±EP) of *Trichogramma pretiosum* females (maternal generation) exposed to *Anagasta kuehniella* treated eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h |
|---|---|---|---|
| Control | 7.4 ± 0.64aA[1] | 7.3 ± 0.30aA[1] | 7.9 ± 0.84aA[1] |
| Acetamiprid | 5.7 ± 1.06aB | 7.1 ± 0.24aA | 6.1 ± 0.23aB |
| Imidacloprid | 5.9 ± 0.81bB | 6.3 ± 0.50bA | 7.8 ± 0.35aA |
| Lufenuron | 6.3 ± 0.65aB | 7.0 ± 0.23aA | 7.8 ± 0.41aA |
| Triflumuron | 6.3 ± 0.62aB | 6.3 ± 0.52aA | 7.3 ± 0.63aA |
| Novaluron | 5.5 ± 0.53bB | 7.5 ± 0.27aA | 8.6 ± 0.49aA |
| Pyriproxifen | 8.2 ± 0.69aA | 5.9 ± 0.44bA | 6.0 ± 0.55bB |
| CV (%) = 18.4% | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$).

**Table 2.** Number (±EP) of eggs parasitized by the maternal generation of *Trichogramma pretiosum* exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h | Reduction (%)[2] | Class[3] |
|---|---|---|---|---|---|
| Control | 29.8 ± 2.07aA[1] | 30.1 ± 3.63aB[1] | 27.7 ± 2.17aA[1] | – | |
| Acetamiprid | 24.4 ± 3.28aA | 28.6 ± 2.70aB | 29.1 ± 1.25aA | 6.2 | 1 |
| Imidacloprid | 21.2 ± 2.98bB | 27.8 ± 2.05aB | 18.1 ± 2.06bB | 23.3 | 1 |
| Lufenuron | 28.0 ± 1.00aA | 33.6 ± 1.91aA | 27.6 ± 1.02aA | 0.0 | 1 |
| Triflumuron | 19.6 ± 2.67cB | 37.6 ± 1.32aA | 27.2 ± 1.15bA | 3.8 | 1 |
| Novaluron | 25.5 ± 2.16aA | 26.9 ± 4.63aB | 31.6 ± 1.73aA | 4.1 | 1 |
| Pyriproxifen | 21.8 ± 2.00aB | 18.9 ± 1.97aC | 18.1 ± 1.29aB | 32.9 | 2 |
| CV (%) = 19.8 | | | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test (P < 0.05), [2]Percent parasitism reduction, [3]Toxicity class recommended by Sterk *et al.* (1999): class 1 = harmless (< 30% reduction of parasitism) and class 2 = slightly harmful (30-79% reduction of parasitism).

between the exposure periods. The exposure to eggs treated with imidacloprid and triflumuron also reduced the parasitism capacity of insects exposed to the eggs 1 h after the treatment (Table 2). Similar results obtained in studies by Rocha and Carvalho (2004) also evidenced a reduction in the parasitism capacity of *T. pretiosum* females that had been exposed to triflumuron residues present on treated surfaces. Castelo Branco *et al*. (2003) also reported that triflumuron reduced the percentage of *Helicoverpa zea* (Boddie, 1850) eggs parasitized by *T. pretiosum*, which led the authors to recommend not employing it in areas where the use of the insecticide-parasitoid association was planned.

When *T. pretiosum* was exposed to host eggs 48 h after treatment with imidacloprid, its parasitism capacity was reduced to 18.1 eggs/female on average, in relation to the other post-treatment periods. In contrast, acetamiprid, lufenuron, and novaluron did not reduce this biological characteristic, regardless of the post-treatment time of exposure of the females to the treated eggs (Table 2).These results confirm those reported by Moura *et al*. (2004), possibly because the presence of acetamiprid residues on the host eggs did not repel the females and consequently did not affect the *T. pretiosum* reproduction capacity.

Moura *et al*. (2006) reported divergent results from the current ones for adult *T. pretiosum*. They observed that the parasitism capacity of females exposed to a glass surface containing acetamiprid residues was reduced by 98.3%. The toxic effect of acetamiprid on *T. pretiosum* females is thought to be related to their larger exposure to residues on glass plates in relation to host egg cards. Due to the reduction of parasitism by pyriproxifen, it was placed in class 2, slightly harmful, while acetamiprid, imidacloprid, lufenuron, triflumuron, and novaluron were classified as harmless (class 1) (Table 2).

**Emergence of $F_1$ Generation.** The emergence of $F_1$ generation parasitoids was affected by novaluron when the females were exposed to host eggs one, 24, and 48 h after treatment (Table 3). Its toxicity increased with time, suggesting a larger concentration of these inside the host eggs, which increased the mortality of the parasitoid in the embryonic period, as also reported by Carvalho *et al*. (2001), Cônsoli *et al*. (2001), and Moura *et al*. (2005). Imidacloprid and triflumuron also affected the percent of emergence of $F_1$ generation parasitoids when the maternal generation females were exposed to the insecticides 1 h after egg treatment, resulting in mean emergence values of 79.1 and 72.8%, respectively (Table 3). Carvalho *et al*. (2003) obtained similar results for imidacloprid and triflumuron. They observed a reduction in the emergence of *T. pretiosum* $F_1$ generation specimens from factitious host eggs exposed for parasitism 1 h after treatment. They also confirmed the results of Moura *et al*. (2004), who observed that imidacloprid was harmful for the emergence of the *T. pretiosum* $F_1$ generation, regardless of the period of exposure of the maternal generation females to the treated host eggs.

**Table 3.** Emergence (%) (±EP) of $F_1$ generation specimens from *Trichogramma pretiosum* females exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h | Reduction (%)[2] | Class[3] |
|---|---|---|---|---|---|
| Control | 88.3 ± 1.91aA[1] | 84.6 ± 4.70aA[1] | 74.3 ± 8.68aB[1] | – | – |
| Acetamiprid | 83.1 ± 11.03aA | 83.0 ± 4.63aA | 87.6 ± 1.10aA | 0.0 | 1 |
| Imidacloprid | 79.1 ± 4.90aB | 73.0 ± 6.38aA | 59.9 ± 6.84aC | 14.2 | 1 |
| Lufenuron | 90.0 ± 2.88aA | 84.6 ± 2.35aA | 94.5 ± 1.38aA | 0.0 | 1 |
| Triflumuron | 72.8 ± 7.69bB | 77.2 ± 4.45bA | 90.2 ± 3.05aA | 2.8 | 1 |
| Novaluron | 69.9 ± 5.06aB | 51.2 ± 8.10bB | 45.0 ± 2.25bC | 32.8 | 2 |
| Pyriproxifen | 88.3 ± 4.67aA | 79.4 ± 6.76aA | 75.8 ± 2.63aB | 1.5 | 1 |
| CV (%) = 13.2 | | | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test (P < 0.05), [2]Mean percent reduction of emergence, [3]Toxicity class recommended by Sterk *et al.* (1999), class 1 = harmless (< 30% reduction of emergence), class 2 = slightly harmful (30-79% reduction of emergence).

**Table 4.** Longevity (%) (±EP) of *Trichogramma pretiosum* $F_1$ generation females exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h |
|---|---|---|---|
| Control | 5.6 ± 0.27aA[1] | 6.1 ± 0.48aB[1] | 6.2 ± 3.27aA[1] |
| Acetamiprid | 4.2 ± 0.26bA | 5.4 ± 0.76aB | 5.7 ± 2.03aA |
| Imidacloprid | 5.8 ± 0.33aA | 5.8 ± 0.35aB | 6.6 ± 2.90aA |
| Lufenuron | 5.7 ± 0.46aA | 5.7 ± 0.52aB | 6.5 ± 1.94aA |
| Triflumuron | 4.7 ± 0.15bA | 6.1 ± 0.25aB | 5.9 ± 1.59aA |
| Novaluron | 5.3 ± 0.41bA | 7.9 ± 0.50aA | 6.4 ± 3.53bA |
| Pyriproxifen | 4.9 ± 0.23aA | 4.5 ± 0.71aB | 4.6 ± 2.12aB |
| CV (%) = 17.2 | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$).

No negative effects were observed on the emergence of $F_1$ generation specimens when maternal generation females were exposed to treated eggs 24 h after treatment with acetamiprid, imidacloprid, lufenuron, triflumuron, and pyriproxifen. Imidacloprid reduced the emergence of descendents of females exposed to *A. kuehniella* eggs 48 h after egg treatment (Table 3). Only acetamiprid and lufenuron did not affect the emergence of *T. pretiosum* $F_1$ generation specimens after exposure to treated host eggs at any of the periods.

When eggs of factitious host were treated with triflumuron and offered to the maternal generation females, one and 48 hours after contamination, the number of insects that emerged did not decrease, presenting averages of 72.8% and 90.2%, respectively. As novaluron reduced the emergence of $F_1$ generation parasitoids, it was classified as class 2 = slightly harmful (30-79% emergence reduction) and the other insecticides fell into class 1 = harmless (<30% emergence reduction) (Table 3).

**Longevity of $F_1$ Generation.** The longevity of $F_1$ generation females was not negatively affected by any of the insecticides when the maternal generation was exposed to treated host eggs one and 24 h after treatment (Table 4). However, Carvalho *et al*. (2003) observed negative effects on the longevity of *T. pretiosum* $F_1$ generation females exposed to host eggs dip-treated with lufenuron and triflumuron 1 h after treatment. The differences found for lufenuron are thought to result from the larger dose of this insecticide (0.4 g a.i.\L) used in this study.

In contrast, pyriproxifen was the only product to reduce the longevity of exposed $F_1$ generation females 48 h after host egg treatment, with a mean of 4.6 days (Table 4). No significant difference was observed in mean longevity of $F_1$ generation females of *T. pretiosum* exposed to host eggs treated with imidacloprid, lufenuron, and pyriproxifen at the post-treatment exposure times investigated. Acetamiprid and triflumuron affected longevity only when the females were exposed to treated eggs 1 h after treatment. Novaluron reduced female longevity at 1-h and 48-h post-treatment exposure times in relation to a 24-h post-treatment exposure time (Table 4).

**Eggs Parasitized by $F_1$ Generation Females.** The parasitism capacity of $F_1$ generation *T. pretiosum* was not affected by any of the insecticides when the maternal generation females were exposed to treated eggs 1 h after treatment (Table 5). Exposure to acetamiprid and imidacloprid reduced the parasitism capacity of $F_1$ generation females 24 h after egg treatment, giving means of 24.1 and 18.9 parasitized eggs/female, respectively. Means of 14.3 and 8.8 parasitized eggs/female were obtained at 24 and 48 h after host egg treatment with pyriproxifen, demonstrating a reduction in parasitism capacity. It was also observed that this product caused a decrease in the number of eggs per female in relation to the evaluated times (Table 5).

Considering the effects of the compounds for the different post-host egg treatment times of exposure of *T. pretiosum* females, no significant difference was observed in the mean

**Table 5.** Number (±EP) of parasitized eggs per *Trichogramma pretiosum* $F_1$ generation females exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h | Reduction (%)[2] | Class[3] |
|---|---|---|---|---|---|
| Control | 39.0 ± 2.77aA[1] | 33.6 ± 1.91aA[1] | 25.6 ± 3.27bA[1] | | |
| Acetamiprid | 30.0 ± 2.32aA | 24.1 ± 3.80aB | 26.0 ± 2.03aA | 18.3 | 1 |
| Imidacloprid | 29.7 ± 5.79aA | 18.9 ± 3.44bB | 28.0 ± 2.90aA | 22.0 | 1 |
| Lufenuron | 33.3 ± 3.95aA | 32.2 ± 2.70aA | 25.6 ± 1.94aA | 7.0 | 1 |
| Triflumuron | 31.0 ± 4.18aA | 30.3 ± 3.33aA | 25.3 ± 1.59aA | 11.6 | 1 |
| Novaluron | 28.4 ± 3.37aA | 34.4 ± 1.95aA | 19.4 ± 3.53bA | 16.2 | 1 |
| Pyriproxifen | 25.9 ± 3.05aA | 14.3 ± 0.74bB | 8.8 ± 2.12cB | 50.2 | 2 |
| CV (%) = 19.8 | | | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$), [2]Mean percent reduction of emergence, [3]Toxicity class recommended by Sterk *et al.* (1999), class 1 = harmless (< 30% reduction of emergence), class 2 = slightly harmful (30-79% reduction of emergence).

**Table 6.** Emergence (%) (±EP) of $F_2$ generation *Trichogramma pretiosum* maternal generation females exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h | Reduction (%)[2] | Class[3] |
|---|---|---|---|---|---|
| Control | 68.7 ± 7.50aB[1] | 76.1 ± 5.95aB[1] | 66.6 ± 3.08aB[1] | – | – |
| Acetamiprid | 84.3 ± 5.44aA | 72.4 ± 7.77aB | 78.8 ± 7.54aA | 0.0 | 1 |
| Imidacloprid | 60.6 ± 10.14bB | 56.9 ± 4.74bB | 92.6 ± 1.80aA | 0.7 | 1 |
| Lufenuron | 73.3 ± 3.60aA | 66.9 ± 9.20aB | 62.4 ± 7.11aB | 4.3 | 1 |
| Triflumuron | 85.4 ± 5.63aA | 94.8 ± 1.90aA | 85.3 ± 6.56aA | 0.0 | 1 |
| Novaluron | 63.8 ± 9.99aB | 65.3 ± 9.19aB | 44.2 ± 6.02bC | 18.0 | 1 |
| Pyriproxifem | 54.6 ± 5.85aB | 67.3 ± 7.94aB | 68.5 ± 2.01aB | 9.9 | 1 |
| CV (%) = 21.0 | | | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$), [2]Mean percent reduction of emergence, [3]Toxicity class recommended by Sterk *et al.* (1999), class 1 = harmless (< 30% reduction of emergence), class 2 = slightly harmful (30-79% reduction of emergence).

parasitism capacity of $F_1$ generation females exposed to host eggs treated with acetamiprid, lufenuron, and pyriproxifen. Imidacloprid had an effect only 24 h after treatment, while novaluron was active only after 48 h in relation to parasitoids exposed to host eggs 1 h after treatment (Table 5). Pyriproxifen reduced the parasitism capacity of $F_1$ generation females, fitting into class 2 = slightly harmful (30-79% reduction), and the other insecticides fell into class 1 = harmless (< 30% reduction) (Table 5).

**Emergence of $F_2$ Generation.** The emergence of $F_2$ generation specimens was not affected by any of the insecticides when maternal females were exposed to treated eggs 1 h and 24 h after treatment. Novaluron negatively affected the emergence percentage of $F_2$ generation insects from maternal generation females exposed to treated host eggs 48 h post-treatment, affording 44.2% emergence. Triflumuron produced higher emergence percentages for $F_2$ generation specimens, with means ranging from 85.3 to 94.8% (Table 6).

When the effect of exposure to the insecticides after the various times was evaluated, significant differences were found for mean emergence values of $F_2$ generation specimens exposed to host eggs treated with acetamiprid, lufenuron, triflumuron, and pyriproxifen. Imidacloprid did not have an effect at 48 h after treatment, while novaluron reduced only the parasitism capacity of females at this time in relation to those exposed to treated host eggs 1 h and 24 h after treatment (Table 6). Despite the negative effects on $F_2$ specimen emergence observed for some of the tested insecticides, all fell into class 1 = harmless, according to the IOBC toxicity categories (Table 6).

**Longevity of $F_2$ Generation.** Acetamiprid, lufenuron, triflumuron, and novaluron produced a longevity reduction in *T. pretiosum* $F_2$ generation females when maternal generation females were exposed to treated host eggs 1 h after treatment. Yet only imidacloprid and triflumuron reduced the longevity of $F_2$ generation females exposed to their residues 24 h after treatment, giving means of 4.7 and 5.2 days, respectively. In contrast, none of the insecticides affected the longevity of $F_2$ generation females when maternal generation females were exposed to host eggs 48 h after treatment (Table 7).

No significant difference was observed in longevity of the $F_2$ generation females of *T. pretiosum* exposed to host eggs treated with triflumuron and pyriproxifen at the studied exposure times. Lufenuron and novaluron only affected the longevity of maternal generation females exposed to treated eggs 1 h after treatment, while imidacloprid reduced the longevity of females only at 24-h exposure after treatment in relation to specimens exposed to treated host eggs 1- and 48-h post treatment (Table 7).

**Eggs Parasitized by $F_2$ Generation Females.** Pyriproxifen reduced the parasitism capacity of $F_2$ generation females when the maternal generation was exposed to its residues one, 24, and 48 h after the treatment of host eggs. Acetamiprid, imidacloprid, and novaluron also reduced the parasitism of $F_2$ generation females, but only when the maternal generation was

**Table 7.** Longevity (days) (±EP) of *Trichogramma pretiosum* $F_2$ generation females from maternal generation females exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h |
|---|---|---|---|
| Control | 6.0 ± 0.31bA[1] | 7.4 ± 0.28aA[1] | 4.5 ± 0.61cB[1] |
| Acetamiprid | 5.3 ± 0.16cB | 7.6 ± 0.33aA | 6.4 ± 0.34bA |
| Imidacloprid | 7.0 ± 0.46aA | 4.7 ± 0.60bB | 6.5 ± 0.42aA |
| Lufenuron | 5.2 ± 0.46bB | 6.9 ± 0.32aA | 6.4 ± 0.70aA |
| Triflumuron | 5.3 ± 0.21aB | 5.2 ± 0.41aB | 4.9 ± 0.46aB |
| Novaluron | 4.8 ± 0.11bB | 6.5 ± 0.49aA | 6.5 ± 0.27aA |
| Pyriproxifen | 6.5 ± 0.41aA | 6.7 ± 0.19aA | 6.1 ± 0.36aA |
| CV (%) = 6.9 | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$).

**Table 8.** Number (±EP) of parasitized eggs per *Trichogramma pretiosum* $F_2$ generation females when maternal females were exposed to *Anagasta kuehniella* eggs 1, 24, and 48 h after treatment.

| Treatment | 1 h | 24 h | 48 h | Reduction (%)[2] | Class[3] |
|---|---|---|---|---|---|
| Control | 45.8 ± 3.32aA[1] | 37.1 ± 1.70bA[1] | 32.3 ± 1.46bA[1] | | |
| Acetamiprid | 35.0 ± 1.57aB | 40.1 ± 1.21aA | 32.9 ± 0.70aA | 6.3 | 1 |
| Imidacloprid | 39.8 ± 1.21aB | 42.4 ± 1.21aA | 29.0 ± 3.10bA | 3.4 | 1 |
| Lufenuron | 49.0 ± 1.56aA | 37.5 ± 3.48bA | 28.8 ± 0.77cA | 0.0 | 1 |
| Triflumuron | 43.6 ± 1.88aA | 34.5 ± 4.11bA | 37.1 ± 2.16bA | 0.0 | 1 |
| Novaluron | 42.0 ± 2.72aB | 34.7 ± 2.69bA | 34.5 ± 2.43bA | 3.4 | 1 |
| Pyriproxifen | 41.0 ± 1.30aB | 22.5 ± 4.22bB | 19.5 ± 2.19bB | 27.9 | 1 |
| CV (%) = 14.7 | | | | | |

[1] Means followed by the same letter, rows with lower case letters and columns with upper case letters, do not differ by the Scott-Knott test ($P < 0.05$), [2]Mean percent reduction of emergence, [3]Toxicity class recommended by Sterk *et al.* (1999), class 1 = harmless (< 30% reduction of emergence), class 2 = slightly harmful (30-79% reduction of emergence).

exposed to treated host eggs 1 h after treatment. Lufenuron and triflumuron were harmless for parasitism capacity, resulting in mean oviposition values ranging from 29 to 49 eggs per female (Table 8).

No reduction in parasitism capacity was observed for $F_2$ generation females for acetamiprid, while lufenuron, triflumuron, and novaluron had no affect on the parasitism capacity of maternal generation females only in the circumstance where they were exposed to treated eggs 1 h after treatment. Imidacloprid reduced the parasitism capacity of females only for exposure 48 h after treatment in relation to specimens exposed to treated host eggs 1- and 24-h post treatment (Table 8). Considering the effects of the insecticides on the parasitism capacity of $F_2$ generation, they belong to class 1 = harmless, according to IOBC (Table 8).

In summary, pyriproxifen was slightly harmful (class 2) for the parasitism capacity of maternal and $F_1$ generation females of *T. pretiosum*. Novaluron was slightly harmful (class 2) for the emergence of $F_1$ generation specimens. Acetamiprid, imidacloprid, lufenuron, and triflumuron were harmless (class 1) to *T. pretiosum* and are recommendable for integrated pest management programs aiming at the preservation of this parasitoid species.

## Acknowledgements

For research grants from Fundação de Amparo à Pesquisa do Estado de Minas Gerais (FAPEMIG) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) for financial support.

## Literature cited

CARVALHO, G. A.; PARRA, J. R. P.; BAPTISTA, G. C. 2001. Impacto de produtos fitossanitários utilizados na cultura do tomateiro na fase adulta de duas linhagens de *Trichogramma pretiosum* Riley, 1879 (Hymenoptera: Trichogrammatidae). Ciência e Agrotecnologia (Brasil, Lavras) 25 (3): 560-568, Jul./Set.

CARVALHO, G. A.; FUINI, L. C.; ROCHA, L. C. D.; MORAES, J. C.; ECOLE, C. C. 2003. Avaliação da seletividade de inseticidas utilizados na tomaticultura a *Trichogramma pretiosum* Riley, 1879 (Hymenoptera: Trichogrammatidae). Ecossistema (Brasil, Espírito Santo do Pinhal) 28 (1/2): 23-30.

CASTELO BRANCO, M.; PONTES, L. A.; AMARAL, P. S. T.; MESQUITA FILHO, M. V. 2003. Inseticidas para o controle da traça-do-tomateiro e broca-grande e seu impacto sobre *Trichogramma pretiosum*. Horticultura Brasileira (Brasil, Brasília) 21 (4): 652-654.

CÔNSOLI, F. L.; BOTELHO, P. S. M.; PARRA, J. R. P. 2001. Selectivity of insecticides to the egg parasitoid *Trichogramma galloi* Zucchi, 1988 (Hym., Trichogrammatidae). Journal of Applied Entomology 125 (1/2): 37-43.

LUNDGREN, J. G.; HEIMPEL, G. E.; BOMGREN A. S. 2002. Comparison of *Trichogramma brassicae* (Hymenoptera: Trichogrammatidae) augmentation with organic and synthetic pesticides for control of cruciferous Lepidoptera. Environmental Entomology 31 (6): 1231-1239.

MEDINA, P.; SMAGGHE G.; BUDIA, F.; TIRRY, L.; VIÑUELA E. 2003. Toxicity and absorption of azadirachtin, diflubenzuron, pyriproxyfen, and tebufenozide after topical application in predatory larvae of *Chrysoperla carnea* (Neuroptera: Chrysopidae). Environmental Entomology 32 (1): 196-203.

MOURA, A. P.; CARVALHO, G. A.; RIGITANO, R. L. O. 2004. Efeito residual de novos inseticidas utilizados na cultura do tomateiro sobre *Trichogramma pretiosum* Riley, 1879 (Hymenoptera: Trichogrammatidae). Acta Scientiarum Agronomy (Brasil, Maringá) 26 (2): 231-237.

MOURA, A. P.; CARVALHO, G. A.; RIGITANO, R. L. O. 2005. Toxicidade de inseticidas utilizados na cultura do tomateiro a *Trichogramma pretiosum*. Pesquisa Agropecuária Brasileira (Brasil, Brasília) 40 (3): 203-210.

MOURA, A. P.; CARVALHO, G. A; PEREIRA, A. E.; ROCHA, L. C. D. 2006. Selectivity evaluation of insecticides used to *Trichogramma pretiosum*. Biocontrol 51 (6): 769-778.

PARRA, J. R. P. 1997. Técnicas de criação de *Anagasta kuehniella*, hospedeiro alternativo para produção de *Trichogramma*. cap.4, pp.121-150. In: PARRA, J. R. P.; ZUCCHI, R. A. (Eds.). *Trichogramma* e o controle biológico aplicado. FEALQ. Brasil. Piracicaba. 324 p.

QUERINO, R. B.; ZUCCHI, R. A. 2003. New species of *Trichogramma* Westwood (Hymenoptera: Trichogrammatidae) associated with lepidopterous eggs in Brazil. Zootaxa (Auckland) 163 (1): 1-10.

ROCHA, L. C. D.; CARVALHO, G. A. 2004. Adaptação da metodologia padrão da IOBC para estudos de seletividade com *Trichogramma pretiosum* Riley, 1879 (Hymenoptera: Trichogrammatidae) em condições de laboratório. Acta Scientiarum Agronomy (Brasil, Maringá) 26 (3): 315-320.

RUBERSON, J. R.; TILLMAN P. G. 1999. Effect of selected insecticides on natural enemies in cotton: laboratory studies. pp. 1210-1213. In: Proceedings, Beltwide Cotton Conferences. 3-7 January 1999, New Orleans, LA. National Cotton Council Orlando, Florida. USA.

SCHOLZ, B. C. G.; MONSOUR C. J.; ZALUCKI M. P. 1998. An evaluation of selective *Helicoverpa armigera* control options

in sweet corn. Australian Journal of Experimental Agriculture (Australia, Collingwood) 38 (6): 601-607.

SCOTT, A. J.; KNOTT, M. A. A. 1974. cluster analysis method for grouping means in the analysis of variance. Biometrics 30 (3): 507-512.

STERK, G.; HASSAN, S. A.; BAILLOD, M.; BAKKER, F.; BIGLER, F.; BLÜMEL, S.; BOGENSCHÜTZ, H.; BOLLER, E.; BROMAND, B.; BRUN, J.; CALIS, J. N. M.; COREMANS-PELSENEER, J.; DUSO, C.; GARRIDO, A.; GROVE, A.; HEIMBACH, U.; HOKKANEN, H.; JACAS, J.; LEWIS, G.; MORETH, L.; POLGAR, L.; ROVERSTI, L.; SAMSØE-PETERSEN, L.; SAUPHANOR, B.; SCHAUB, L.; STÄUBLI, A.; TUSET, J. J.; VAINIO, A.; VAN DE VEIRE, M.; VIGGIANI, G.; VIÑUELA, E.; VOGT, H. 1999. Results of the seventh joint pesticide testing programme carried out by the IOBC/WPRS-Working Group 'Pesticides and Beneficial Organisms'. BioControl 44 (1): 99-117.

ZUCCHI, R. A.; MONTEIRO, R. C. 1997. O gênero *Trichogramma* na América do Sul. cap.2, pp. 41-66. In: PARRA, J. R. P.; ZUCCHI, R. A. (Eds.). *Trichogramma* e o controle biológico aplicado. FEALQ. Brasil. Piracicaba. 324 p.

Recibido: 21-oct-2009 • Aceptado: 30-may-2010