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# TOXICITY OF IMIDACLOPRID AND DIAFENTHIURON TO *CHRYSOPERLA CARNEA* (STEPHENS) (*NEUROPTERA: CHRYSOPIDAE*) IN THE LABORATORY CONDITIONS

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**Abstract:** Insecticides are unavoidable in pest management programs especially when the pest crosses Econimic Threshold Level (ETL). Nevertheless, often the plant protection products kill the natural enemy population making the pest to resurge and thus demanding more sprays. Therefore, insecticides used in IPM programs should be selective enough to spare the beneficials. Laboratory studies were conducted to find out the toxicity of imidacloprid and diafenthiuron to the eggs, larvae and adults of *Chrysoperla carnea*. Imidacloprid at the recommended dose of 0.28 ml/l caused 15.38% egg mortality, 26.67 and 33.33% larval mortality by ingestion and contact, respectively and 50.00% adult mortality. The egg mortality was about 15.38% and larval mortality of 23.33% and adult mortality of 26.67% was caused by diafenthiuron. Based on the classification given by IOBC/WPRS working group on Pesticides and non-target invertebrates, both the insecticides were classified as harmless to *C. carnea*, since the recommended dose caused less than 50% mortality in the laboratory conditions.

**Key words:** imidacloprid, diafenthiuron, *Chrysoperla carnea*, toxicity

## INTRODUCTION

Presence of predators and parasitoids in field crops, orchards and vegetables has been a subject for many studies of reducing the insecticide usage and thereby environmental pollution (WhiteComb and Bell 1964; Dean and Sterling 1992). Sometimes the role played by the predators itself reduces the need of pesticide application. *Chrysoperla carnea* (Stephens) is one such predator highly useful in pest management especially of aphids. The larva of *C. carnea* (aphid lion) has relatively a broad range of prey acceptance (Hydron and WhiteComb 1979), which includes aphids whiteflies, eggs of moths and other soft-bodied insects. Due to the polyphagous and voracious nature and vast geographical distribution (New 1975), ease of mass multiplication (Araujo and Bichao 1990) and tolerance to some pesticides (Hassan *et al*. 1985) it has received much attention of farmers as well as researchers as a potential biological pest control agent. Effectiveness of *C. carnea* as biological control agent has been demonstrated in field crops, orchards and in green houses (Hagley and Miles 1987) and reported to give about 100% Lepidoptran pest control when used along with *Trichogramma* spp. (Rincon-Vitova 1999). Inspite of all these benefits, *C. carnea* with many other beneficials has almost been eliminated from fields due to frequent use of some non-selective agrochemicals (Nasreen *et al*. 2005). Now, the importance of biointensive pest management has been recognized as a holistic approach for integrated pest management. This can be achieved only by using an insecticide which is selective enough to kill the pest and spare the beneficials.

Neonicotinoids are the new group of crop protection products found highly effective against the sucking pests. Neonicotinoids act on receptor protein of insect nervous system and thus possess a new mode of action (Leicht 1996). They are acute, contact and stomach poisons with translaminar and systemic properties. At lower concentrations they act as antifeedants, a property they share with nicotine. Their selectivity, lower use rate and safety to beneficial insects especially when used as seed dressings make neonicotinoids an ideal component in any IPM programme.

Similarly, a thiourea compound, diafenthiuron has a novel mode of action on acting on the biochemical sites such as respiratory sites (Ishaaya *et al*. 2001) inhibiting mitocondrial action, energy metabolism (ATP synthesis) (Ruder and Kayser 1992) and moult inhibition and hence it is seen as a viable tool for managing insects and mites. It is very effective against *Plutella xylostella* (L.) (Ishaaya *et al.* 1993), *Bemisia tabaci* Genn. (Chinnabbai *et al.* 2000) and *Conogethes punctiferalis* and *Scirtothrips cardamomi* (Stanley 2007). Diafenthiuron is a selective chemical and the selectivity is associated to the metabolism by microsomal oxidation (Kayser and Ellinger 2001). Diafenthiuron is reported to be safe to parasitoids and predators (Ismail 1997; Zuhua and Shusheng 1998; Toress *et al*. 2002) and it can fit in integrated

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pest management (Delbeke *et al*. 1997; Sun and Soo 2000). Diafenthiuron also acts as insect growth regulator, selective and less toxic to beneficial insects (Kranthi *et al*. 2004).

Though both the insecticides are reported to be effective against the pest species and selective enough to spare the beneficials, detailed study is needed to find the toxicity against the important predator, *C. carnea*, which forms the aim of the present study.

## MATERIALS AND METHODS

Laboratory experiments were carried out to find the toxicity of imidacloprid and diafenthiuron on *C. carnea* during 2006–2007. The insects for the bioassay were obtained form the Biological Control Laboratory, Tamil Nadu Agricultural University, Coimbatore and the commercial formulation made by Mahamaya Agrisciences is used for the study.

**Toxicity to eggs**

The method described by Krishnamoorthy (1985) was used to assess the effect of imidacloprid and diafenthiuron on the eggs of *C. carnea*. The brown paper strips containing stalked eggs were uniformly sprayed with insecticides using an atomizer. Each treatment was replicated thrice and contained approx. 20 eggs in each replication. The eggs sprayed with distilled water alone served as control. The number of larva hatched from each treatment was recorded and per cent hatchability was estimated.

**Toxicity to the larvae**

*Diet contamination method*

Eggs of *Corcyra cephalonica* (Stainton) were exposed to UV radiation of 15 W for 15 min to kill the embryo. The UV killed *Corcyra* eggs were taken in kada cloth and dipped in insecticide solutions of prescribed concentrations as given in the tables. The treated eggs were shade dried for 15 min and then transferred to test tubes at the rate of 1 $cm^3$ per test tube. The untreated check was maintained by dipping the eggs in distilled water. Second instar larvae of *C. carnea* (10 nos) were transferred into the test tubes containing *Corcyra* eggs. The larvae were allowed to feed the treated eggs and once they complete feeding, untreated *Corcyra* eggs were provided until pupation. The treatments were replicated thrice. Observations were made on the per cent larval mortality (12, 24 and 48 h after treatment), pupation and adult emergence.

*Dry film method*

The bioassay method developed by McCutchen and Plapp (1988) and modified by (Chelladurai 1999) was adopted for the study. Different concentrations of insecticide solutions were prepared using acetone and water at the ratio of 8:2. Glass scintillation vials of 20 ml capacity were coated evenly with 0.5 ml of different doses of insecticides and dried thoroughly by rotating the vials placed in between the palms. For untreated check, acetone:water (8:2) was used. Second instar larva were released at the rate of 10 per vial and covered with muslin cloth secured with a rubber band. The experiment was conducted with three replications for each treatment. After exposure of the larva for 1 h, they were transferred to test tubes and fed with 1 cc of *Corcyra* eggs. Observations were recorded and the per cent mortality of the larva (12, 24 and 48 h after treatment), pupation (%) and adult emergence (%) was estimated.

**Toxicity to adults**

Ten freshly emerged adults of *C. carnea* were released in separate containers and were fed with food solution containing different doses of insecticides. The food consists of one part of honey and one part of Protinex® with water. The adults were fed with uncontaminated food in untreated check. Likewise, three replications were made with 30 insects per treatment. Mortality of the adults was recorded 12, 24 and 48 h after treatment and the per cent mortality was calculated.

All the test units were kept in a controlled room maintained at 27±3°C and 70±5% relative humidity.

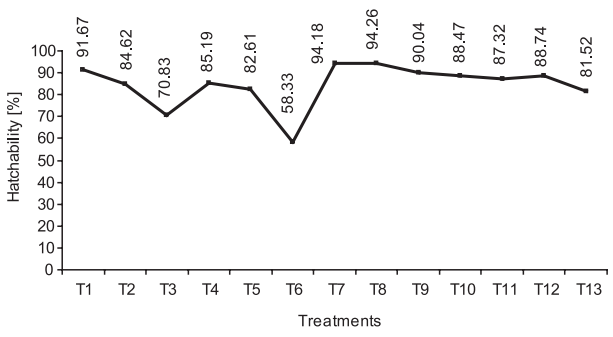
**Statistical analysis**

The per cent mortality in laboratory studies was corrected using Abbot's formula (Abbot 1925). The corrected per cent mortalities were transformed to arcsine percentage and subjected to statistical analysis adopting completely randomized design. The mean values of treatments were then separated by Duncan's Multiple Range Test (DMRT) (Gomez and Gomez 1984).

## RESULTS

**Toxicity to eggs**

The effect of imidacloprid on the egg hatchability of *C. carnea* was studied under laboratory conditions. The least egg mortality of 8.33% was observed in the lowest dose of imidacloprid tested i.e., 0.17 ml/l. About 82.61 and 58.33% of larva hatched out from the eggs treated with thiamethoxam at 0.28 ml/l and methyl demeton at 1 ml/l, respectively. In the case of diafenthiuron, the hatchability of the eggs varied from 87.32 to 94.26% for different doses tested. Diafenthiuron at all used doses were less toxic to *Chrysoperla* eggs. The hatchability in monocrotophos treatment was also not too low (81.52 %) (Fig. 1).




T1 – Imidacloprid 0.17 ml/l
T2 – Imidacloprid 0.28 ml/l
T3 – Imidacloprid 0.56 ml/l
T4 – Tatamida 0.28 ml/l
T5 – Thiamethoxam 0.2 g/l
T6 – Methyl demeton 1 ml/l
T7 – Diafenthiuron 0.8g/l
T8 – Diafenthiuron 1.2g/l
T9 – Diafenthiuron 1.6g/l
T10 – Diafenthiuron 2.4g/l
T11 – Diafenthiuron 3.2g/l
T12 – Pegasus 2g/l
T13 – Monocrotophos 2ml/l

Fig 1. Toxicity of insecticides to the eggs of *C. carnea*

Table 1. Toxicity of imidacloprid to larva of *C. carnea* – diet contamination method

(Mean from three observations)

| Treatments | Larval mortality [%] | | | Pupation [%] | Adult emergence [%] |
|---|---|---|---|---|---|
| | 12 HAT | 24 HAT | 48 HAT | | |
| Imidacloprid 17.8 SL 0.17 ml/l | 0.00 | 6.67 b (14.95) | 13.33 b (21.41) | 92.31 b (73.91) | 91.67 b (73.24) |
| Imidacloprid 17.8 SL 0.28 ml/l | 0.00 | 13.33 c (21.40) | 26.67 c (31.09) | 90.91 b (72.73) | 90.00 b (71.80) |
| Imidacloprid 17.8 SL 0.56 ml/l | 0.00 | 53.33 f (46.91) | 60.00 f (50.77) | 83.33 c (65.94) | 40.00 e (39.23) |
| Imidacloprid 17.8 SL 0.28 ml/l (Tatamida®) | 0.00 | 20.00 d (26.56) | 33.33 d (35.26) | 90.00 b (71.72) | 72.73 d (58.55) |
| Thiamethoxam 25 WG 0.2 g/l | 0.00 | 6.67 b (14.95) | 13.33 b (21.41) | 84.62 c (66.94) | 90.91 b (72.52) |
| Methyl demeton 25 EC 1 ml/l | 0.00 | 33.33 e (35.26) | 53.33 e (46.91) | 85.71 c (67.81) | 83.33 c (65.92) |
| Untreated check | 0.00 | 0.00 a (0.19) | 0.00 a (0.19) | 100.00 a (90.00) | 100.00 a (90.00) |

In a column means followed by a common letter are not significantly different at p = 0.05 by DMRT
Figures in parentheses are arcsine $\sqrt{P}$ transformed values

Table 2. Toxicity of diafenthiuron to larva of *C. carnea* – diet contamination method

(Mean from three observations)

| Treatments | Larval mortality [%] | | | Pupation [%] | Adult emergence [%] |
|---|---|---|---|---|---|
| | 12 HAT | 24 HAT | 48 HAT | | |
| Diafenthiuron 50WP 0.8 g/l | 0.00 a (0.19) | 0.00 a (0.19) | 3.33 b (10.51) | 96.55 b (79.36) | 100.00 a (90.00) |
| Diafenthiuron 50WP 1.2 g/l | 3.33 b (10.50) | 13.33 b (21.41) | 16.67 c (24.08) | 96.30 b (78.99) | 92.59 b (74.23) |
| Diafenthiuron 50WP 1.6 g/l | 13.33 c (21.39) | 23.33 c (28.87) | 23.33 d (28.87) | 95.65 b (78.01) | 86.96 c (68.86) |
| Diafenthiuron 50WP 2.4 g/l | 16.67 d (24.09) | 20.00 c (26.55) | 26.67 e (31.08) | 90.91 bc (72.46) | 86.36 c (68.35) |
| Diafenthiuron 50WP 3.2 g/l | 23.33 e (28.87) | 40.00 d (39.23) | 43.33 f (41.16) | 88.24 c (69.97) | 82.35 d (65.17) |
| Diafenthiuron 50WP (Pegasus®) 2.0 g/l | 13.33 c (21.39) | 16.67 b (24.10) | 20.00 cd (26.56) | 95.83 b (78.34) | 91.67 b (73.25) |
| Monocrotophos 36 WSC 2 ml/l | 26.67 f (31.09) | 46.67 e (43.09) | 60.00 g (50.77) | 83.33 d (65.92) | 83.33 d (65.93) |
| Untreated check (water) | 0.00 a (0.19) | 0.00 a (0.19) | 0.00 a (0.19) | 100.00 a (90.00) | 100.00 a (90.00) |

In a column means followed by a common letter are not significantly different at p = 0.05 by DMRT
Figures in parentheses are arcsine $\sqrt{P}$ transformed values

**Toxicity to larvae**

*Diet contamination method*

All insecticidal treatments significantly affect the larva mortality, per cent pupation and adult emergence (Table 1). The maximum larvae mortality was recorded in the higher dose of imidacloprid (0.56 ml/l) at 24 and 48 HAT (53.33 and 60.00%), respectively which was significantly different from all other treatments. The recommended dose of imidacloprid at 0.28 ml/l recorded the mortality of 13.33 and 26.67% at 24 and 48 HAT, respectively. The lower dose of imidacloprid at 0.17 ml/l and thiamethoxam at 0.2 g/l were on par with each other recorded 6.67 and 13.33% at 24 and 48 HAT, respectively. Imidacloprid at 0.56 ml/l recorded 83.33% pupation followed by thiamethoxam at 0.28 ml/l (84.62%) and methyl demeton at 1ml/l ha (85.71%). Imidacloprid at 0.17 and 0.28 ml/l recorded 92.31 and 90.91% pupation, respectively while untreated check registered cent per cent pupation. With regard to adult emergence, the least emergence was achieved in imidacloprid at 0.56 ml/l (40.00%) followed by imidacloprid (Tatamida®) at 0.28 ml/l (72.73%) and methyl demeton at 1 ml/l (83.33%). The remaining treatments showed more than 90.00% adult emergence. However, 100% adult emergence was observed in untreated control.

Table 3. Toxicity of imidacloprid to larva of *C. carnea* – dry film method

(Mean from three observations)

| Treatments | Larval mortality [%] | | | Pupation [%] | Adult emergence [%] |
|---|---|---|---|---|---|
| | 12 HAT | 24 HAT | 48 HAT | | |
| Imidacloprid 17.8 SL 0.17 ml/l | 0.00 a (0.19) | 13.33 b (21.41) | 20.00 b (26.56) | 91.67 a (73.35) | 90.91b (72.56) |
| Imidacloprid 17.8 SL 0.28 ml/l | 13.33 b (21.41 | 26.67 c (31.09) | 33.33 c (35.26) | 90.00 a (71.70) | 88.89 b (70.64) |
| Imidacloprid 17.8 SL 0.56 ml/l | 33.33 c (35.26) | 53.33 e (46.91) | 66.67 d (54.74) | 60.00 c (50.77) | 33.33 e (35.26) |
| Imidacloprid 17.8 SL 0.28 ml/l (Tatamida®) | 13.33 b (21.41) | 33.33 d (35.26) | 33.33 c (35.26) | 90.00 a (71.68) | 66.67 c (54.75) |
| Thiamethoxam 25 WG 0.2 g/l | 66.67 d (54.74) | 80.00 g (63.45) | 80.00 e (63.45) | 66.67 b (54.74) | 50.00 d (45.00) |
| Methyl demeton 25 EC 1 ml/l | 33.33 c (35.26) | 73.33 f (58.91) | 80.00 e (63.45) | 66.67 b (54.74) | 50.00 d (45.00) |
| Untreated check | 0.00 a (0.19) | 6.67 a (14.97) | 6.67 a (14.97) | 100.00 a (90.00) | 100.00 a (90.00) |

In a column means followed by a common letter are not significantly different at p = 0.05 by DMRT
Figures in parentheses are arcsine $\sqrt{P}$ transformed values

Table 4. Toxicity of diafenthiuron to larva of green lacewing bug, *C. carnea* – dry film method

(Mean from three observations)

| Treatments | 12 HAT | | 24 HAT | | 48 HAT | | Pupation [%] | Adult emergence [%] |
|---|---|---|---|---|---|---|---|---|
| | mortality [%] | corrected mortality [%] | mortality [%] | corrected mortality [%] | mortality [%] | corrected mortality [%] | | |
| Diafenthiuron 50WP 0.8 g/l | 3.33 b (10.51) | 3.33 | 10.00 b (18.39) | 3.57 | 10.00 b (18.39) | 3.57 | 96.29 b (78.94) | 96.15 b (78.89) |
| Diafenthiuron 50WP 1.2 g/l | 10.00 c (18.43) | 10.00 | 13.33 c (21.33) | 7.14 | 16.67 c (24.04) | 10.71 | 96.00 b (78.59) | 100.00 a (90.00) |
| Diafenthiuron 50WP 1.6 g/l | 13.33 d (21.39) | 13.33 | 20.00 d (26.55) | 14.28 | 23.33 d (28.87) | 17.85 | 100.00 a (90.00) | 95.65 b (78.13) |
| Diafenthiuron 50WP 2.4 g/l | 13.33 d (21.40) | 13.33 | 30.00 e (33.21) | 25.00 | 33.33 e (35.26) | 28.57 | 85.00 c (67.39) | 88.24 c (69.97) |
| Diafenthiuron 50WP 3.2 g/l | 20.00 e (26.55) | 20.00 | 33.33 e (35.26) | 28.57 | 43.33 f (41.17) | 39.28 | 82.35 c (65.23) | 71.43 e (57.69) |
| Diafenthiuron 50WP (Pegasus®) 2.0 g/l | 13.33 d (21.39) | 13.33 | 23.33 d (28.87) | 17.85 | 23.33 d (28.87) | 17.85 | 86.96 c (68.95) | 95.00 c (77.17) |
| Monocrotophos 36 WSC 2 ml/l | 53.33 f (46.91) | 53.33 | 73.33 f (58.94) | 71.42 | 76.67 g (61.13) | 75.00 | 71.43 d (57.72) | 80.00 d (63.49) |
| Untreated check (water) | 0.00 a (0.19) | – | 6.67 a (14.90) | – | 6.67 a (14.90) | – | 96.43 b (76.14) | 100.00 a (90.00) |

In a column means followed by a common letter are not significantly different at p = 0.05 by DMRT
*Figures in parentheses are arcsine $\sqrt{P}$ transformed values

Among the diafenthiuron doses, diafenthiuron at high dose (3.2 g/l) recorded a high per cent mortality of 40.00 followed by diafenthiuron 2.4 g/l (20.00 %) a day after exposure. At 48 h of exposure diafenthiuron at the lowest dosed 0.8 g/l recorded 3.33% mortality whereas in the highest dose 3.2 g/l it was 43.33% (Table 2). Almost all the larvae pupated ranging from 88.24 to 96.55% in diafenthiuron treatments. None of the treatments affected adult emergence which was evident from 82.35 to 100.00% adult emergence in diafenthiuron treatments.

*Dry film method*

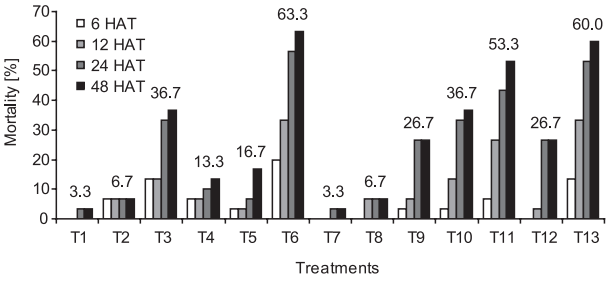
Similar to diet contamination method, all the treatments caused larva mortality, pupation and adult emergence significantly in the dry film method also. Among the imidacloprid treatments, the treatment with imidacloprid at 0.17 ml/l emerged as the least toxic by registering 0.00, 13.33 and 20.00% mortality of larva at 12, 24 and 48 HAT, respectively. The recommended dose of imidacloprid at 0.28 ml/l and Tatamida® at 0.28 ml/l recorded the per cent mortality of 26.67 and 33.33 (24 HAT) and

33.33 (48 HAT), respectively. Thiamethoxam at 0.2 g/l was found to be highly toxic to the larva of *C. carnea* which recorded 80.00% mortality at 24 and 48 HAT. The per cent pupation and adult emergence were found to be 100% in untreated check followed by imidacloprid at 0.17 ml/l (91.67 and 90.91%), respectively. Imidacloprid at 0.28 ml/l and Tatamida® at 0.28 ml/l recorded 90.00% pupation and 88.89 and 66.67% adult emergence, respectively whereas the respective values for higher dose of imidacloprid at 0.56 ml/l was 60.00 and 33.33% (Table 3).

The results on the influence of diafenthiuron to *C. carnea* larva determined by dry film method revealed that diafenthiuron was not very toxic to the predator. In diafenthiuron treatments (0.8 to 3.2g/l) the mortality at 24 HAT ranged between 10.00 and 33.33% and it was 23.33 in another formulation Pegasus® at 2.0 g/l. However, monocrotophos registered 73.33% mortality, which implies that monocrotophos is highly toxic to the larva (Table 4). Almost all the live larva pupated invariable of the treatments and the per cent pupation ranged from 82.35 to 100.00 in diafenthiuron treatments, while it was 71.43 in monocrotophos at 2 ml/l. Diafenthiuron at the highest dose of 3.2 g/l registered 71.43% adult emergence but it went up to 100% in other treatments of diafenthiuron.

**Toxicity to adults**

The mortality of the adults of *C. carnea* when fed with the higher dose of imidacloprid (0.56 ml/l) contaminated food was 13.33% at 12 HAT, whereas it was 33.33% in methyl demeton. At 24 and 48 HAT, 33.33 and 36.67% adults died at the higher dose of imidacloprid (0.56 ml/l), respectively whereas it was only 3.33% in the lower dose of imidacloprid (0.17 ml/l). When fed with Tatamida®, thiamethoxam and methyl demeton contaminated food the mortality per cent was 13.33, 16.67 and 63.33, respectively at 48 HAT (Fig. 2).




T1 – Imidacloprid 0.17 ml/l
T2 – Imidacloprid 0.28 ml/l
T3 – Imidacloprid 0.56 ml/l
T4 – Tatamida 0.28 ml/l
T5 – Thiamethoxam 2 g/l
T6 – Methyl demeton 1 ml/l
T7 – Diafenthiuron 0.8g/l
T8 – Diafenthiuron 1.2g/l
T9 – Diafenthiuron 1.6g/l
T10 – Diafenthiuron 2.4g/l
T11 – Diafenthiuron 3.2g/l
T12 – Pegasus 2g/l
T13 – Monocrotophos 2ml/l

Fig. 2. Toxicity of insecticides to the adults of *C. carnea*

Nearly 27% of the adults of *Chrysoperla* when fed with diafenthiuron 3.2 g/l contaminated food, died at 12 HAT, whereas it was 33.33% in monocrotophos. At 24 and 48 HAT, 43.33 and 53.33% adults died at the highest concentration of diafenthiuron (3.2 g/l) whereas it was only 3.33% in the lower dose of diafenthiuron (0.8 g/l). Sixty per cent of adults died when fed with 2 ml/l of monocrotophos contaminated food and 26.67% in Pegasus® 2.0 g/l contaminated food (Fig. 2).

## DISCUSSION

Extensive use of synthetic chemicals results in the destruction of non-target organisms directly or where they are transported and entered into the niche of the organisms. So, safety studies on predators should be done to know the selectivity of the chemical. The insecticidal effect on non-target organisms are categorized as per the recommendations of the International Organisation for Biological Control, West Palaearctic Regional Section (IOBC/WPRS) working group (Hassan 1989; Nasreen *et al.* 2000) as harmless (< 50% mortality), slightly harmful (50–79% mortality), moderately harmful (80–89% mortality) and harmful (> 90% mortality) when tested at the field recommended dose.

Recommended dose of imidacloprid 17.8 SL at 0.58 ml/l (25 g a.s./ha) had less impact on *C. carnea* registering 15.38% egg mortality, certifying the chemical as harmless. The above was in accordance with Kumar (1998), who stated that no significant adverse effect had been observed due to imidacloprid at 0.28 ml/l on the egg hatchability of *C. carnea.* The effect of the recommended dose of chemical on *C. carnea* larva by diet contamination and dry film methods indicated 26.67 and 33.33% mortality after 48 h of exposure, respectively. Higher larval mortality of more than 60% was reported in a dose above the recommended dose of imidacloprid on chrysopa larvae (Suganthy 2003). Toda and Kashio (1997) reported that imidacloprid and acetamiprid showed low toxicity to *C. carnea* larva in the dipping test and a high toxicity in the residual contact test. Patil and Lingappa (2001) reported that the higher dose of imidacloprid (40 g a.s./ha) as toxic to *C. carnea* larva. It is noteworthy to note that thiamethoxam registered the least mortality of larva in diet contamination method but in dry film method, 80% mortality of larvae was observed indicating it was moderately toxic and similar to the conventional insecticide methyl demeton which may be due to higher contact toxicity than stomach poison. In the present study, imidacloprid at the recommended dose caused less than 50% adult mortality rendering it as harmless. Nevertheless, imidacloprid was reported to affect the longevity of *C. carnea* adults (Mathirajan and Regupathy 2002).

Diafenthiuron was not found to have adverse effect on hatchability of *C. carnea* and did not very toxic to larvae both by contact and through stomach action. The recommended dose of diafenthiuron allowed 95.65% of the treated larvae to pupate and 86.96% of pupae to emerge as adults. Thus, if the larvae could tolerate the insecticide doses, then it can able to pupate and emerge as normal adults. The highest dose of diafenthiuron (3.2 g/l) killed 53% of *Chrysoperla* adults but it was only 3% for the lowest dose tried (0.8 g/l). No effect was observed on the adults of *C. carnea* treated with low dose of diafenthiuron (Nasreen *et al.* 2005). As per the safety norms discussed before, diafenthiuron can be declared as a harmless insecticide to *C. carnea*. It was also reported that low and

recommended dose of diafenthiuron was harmless to *C. carnea* and higher dose was slightly harmful (Nasreen *et al*. 2005). Diafenthiuron (Pegasus®) was reported to be less toxic to *Trichogramma* sp. with a residual toxicity for three days (www.bioresources.com/pretisoumchemicals) and no adverse effect on the parasitisation on *B. tabaci* was noticed in diafenthiuron sprayed fields (Otoidobiga 2003). Diafenthiuron was safer to Staphylinid beetle, *Oligota pygmaea* Kraatz, a red spider mite predator in the field (UPASI 2005). Fresh deposits of diafenthiuron on French bean caused a very minimal mortality to *Encarsia formosa* Gahan and *Eretmocerus eremicus* Rose and Zolnerowich. Adult parasitoid emergence from diafenthiuron treated pupae of *E. formosa* and *E. eremicus* was also found to be unaffected. Diafenthiuron was harmless to adults as well as pupae of both parasitoids irrespective of exposure periods (Javed and Matthews 2002). Diafenthiuron was safer to adults of Coccinellidae and Miridae and both adults and immature stages of predatory mites, *Amblyseius andersoni* (Chant) and *Typhodromus pyri* Scheuten. Spiders of the family Erigonidae and Lycosidae and *C. carnea* were not affected by diafenthiuron sprays in the field (www.kingtaichem.com).

Selection of a suitable insecticide in an IPM program not only depends on its efficacy against the target pest but also on its toxicity to beneficial insects and its withering and persistence. Both the chemicals tested are not highly toxic to the predator at the recommended doses, thus can be used in IPM programs.

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## POLISH SUMMARY

### TOKSYCZNOŚĆ IMIDAKLOPRIDU I DIAFENTIURONU W STOSUNKU DO *CHRYSOPERLA CARNEA* (STEPHENS) (*NEUROPTERA: CHRYSOPIDAE*) W WARUNKACH LABORATORYJNYCH

Insektycydy są nieodzowne w programach zwalczania szkodników, zwłaszcza, jeśli zostają przekroczone wartości progu szkodliwości. Jednak, często środki ochrony roślin zwalczają także naturalnych wrogów szkodników, co powoduje, że ich populacje odradzają się, a to wymaga większej liczby zabiegów. W takim przypadku insektycydy używane w programach IPM powinny być dostatecznie selektywne, ze względu na organizmy pożyteczne. W celu wyznaczenia toksyczności imidaklopridu i diafentiuronu w stosunku do jaj, larw i osobników dorosłych *Chrysoperla carnea* przeprowadzono badania laboratoryjne. Imidakloprid w zalecanej dawce 0,28 ml/l powodował śmiertelność jaj w 15,38%, w 26,67 i 33,33% śmiertelność larw, odpowiednio z powodu spożycia lub kontaktu i śmiertelność dorosłych owadów w 50,00%. Śmiertelność jaj wynosiła około 15,38%, larw 23,33, a dorosłych owadów 26,67% w przypadku użycia diafentiuronu. Opierając się na klasyfikacji wydanej przez grupę roboczą IOBC/WPRS, dotyczącej pestycydów i bezkręgowców nie będących celem zwalczania, oba insektycydy zakwalifikowano jako nieszkodliwe dla *C. carnea*, dopóki zalecana dawka powodowała mniejszą śmiertelność niż 50% w warunkach laboratoryjnych.