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Assessment of the impact of insecticides on *Anagrus nilaparvatae* (Pang et Wang) (Hymenoptera: Mymanidae), an egg parasitoid of the rice planthopper, *Nilaparvata lugens* (Hemiptera: Delphacidae)

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Abstract

The parasitoid, *Anagrus nilaparvatae* (Pang et Wang), is a major natural enemy of the rice planthopper *Nilaparvata lugens*. It plays an important role in the integrated pest management (IPM) of the rice planthopper, although chemical control is also effective. However, compatibility of biological and chemical control has never been investigated with this system. This study was designed to assess potential insecticide toxicities to the wasps, including acute and residual toxicity through contact and oral ingestion. Fourteen insecticides, including organophosphates, carbamates, pyrethroids, insect growth regulators (IGRs), neonicotine, phenylpyrazole, and antibiotics were selected to test their toxicities against the adult parasitoid. Median lethal concentration (LC_{50}) of each insecticide was first determined. Acute contact toxicity tests indicated that chlorpyrifos had the highest toxicity to the wasp, requiring the least chemical to achieve 50% mortality. Imidacloprid was the second most toxic insecticide, while IGRs had the lowest toxicity. Results of oral toxicity tests of 14 pesticides showed that dichlorvos was the most toxic, which generated 100% mortality only 2 h after treatment. Isoprocarb, imidacloprid, and thiamethoxam were the second most toxic insecticides and killed all wasps in a 4-h period. Residual toxicity results indicated that imidacloprid was the most persistent insecticide, and it retained residual toxicity (80.7% mortality) on rice leaves up to 7 d after treatment. Thiamethoxam, triazophos, and fipronil also had long residual toxicity to the wasps with 7-d mortalities as 66.8%, 54.6%, and 50.0%, respectively. IGRs showed very low contact and residual toxicity, but exhibited certain chronic effects of oral toxicity on longevity, fecundity, and offspring emergence.

Keywords: Insecticides; *Anagrus nilaparvatae*; *Nilaparvata lugens*; Integrated pest management; IPM; Risk; Sublethal effect; Lethal effect

1. Introduction

Brown rice planthopper (BPH), *Nilaparvata lugens* (Stål), is one of the most serious pests on rice in Asia and causes substantial yield loss in most rice-producing countries (Heong et al., 1992; Cheng et al., 2003). In China, outbreaks of this planthopper have happened frequently in recent years (Wang and Wang, 2007). Chemical control remains a major strategy in the integrated pest management (IPM) system for its advantages of being quick, efficient, easy to use, cost-effective, notably amendable, and reliable effectiveness against the insect (Zhao, 2000; Endo and Tsurumachi, 2001). However, lethal and sub-lethal effects of broad-spectrum and non-selective pesticides are usually considered a high risk to beneficial species (Croft, 1990; Ruberson et al., 1998). Misuse of chemical insecticides might be accountable for the outbreaks of the pest because extensive and intensive use of insecticides applied heavy selection pressure on target pests and accelerated resistance

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development (Kilin et al., 1981; Hirai, 1993). Another important factor for pest outbreaks was that insecticides indiscriminately or even differentially killed a wide range of more natural enemies than the target pest (Way and Heong, 1994; Tanaka et al., 2000).

Anagrus nilaparvatae (Pang et Wang) (Hymenoptera: Mymanidae), an egg parasitoid of the rice planthopper, has a remarkable ability to suppress population density of rice planthopper (Luo and Zhuo, 1980, 1981; Cheng and He, 1996). Most recent research focused mainly on biology and ecology of *A. nilaparvatae* (Xu and Cheng, 1989; Lou and Cheng, 1996, 2001; Ma et al., 2004; Lou et al., 2002, 2005a, b). Luo et al. (1981) found that some broad-spectrum pesticides, such as dimethoate and others used in the 1970–1980s, were toxic to adult and other different stages of *Anagrus* spp. Very little information and knowledge about the adverse effects of currently popular insecticides on *A. nilaparvatae* is available, except a report showing that buprofezin and imidacloprid diminished the parasitism of the wasp (Xu et al., 2006).

Chemical control and biological control are the two important strategies used in an IPM program (Zhao, 2000). Integration of chemical and biological control systems is a key for the success of any IPM program (Wright and Verkert, 1995). Chemical control should be used only when it is necessary and is least disruptive to biological control. Knowledge of compatibility and impact of pesticides (lethal and sub-lethal) on beneficial species is essential for effective integration of chemical and biological control (Greathead, 1995).

Lethal or adverse effects of insecticides on beneficial arthropods are often expressed as acute or chronic mortality resulting from contact with or ingestion of insecticides (Haseeb et al., 2004). Desneux et al. (2007) recently pointed out that the determination of the acute toxicity of pesticides to beneficial arthropods had traditionally and largely relied on the measurement of an acute median lethal dose or concentration and the estimated lethal dose or concentration might only be a partial measure of the deleterious effects. In addition to direct mortality induced by pesticides, their sub-lethal effects on arthropods must be considered for a complete analysis of their impact (Desneux et al., 2007). The sub-lethal effects on natural enemies are typically chronic and not obvious. These may be expressed as changes in life-history traits of insects, such as parasitism rate, longevity, egg viability, consumption rate, or behavior (Ruberson et al., 1998). Therefore, chemical insecticides need to be correctly and selectively used to ensure sustainable crop protection and environmental stability (Jepson, 1989; Greathead, 1995; Haseeb et al., 2000; Haseeb, 2001).

Currently, many low-toxicity organophosphates, pyrethroids, and other novel insecticides are being investigated as potential alternatives to replace highly toxic organophosphate insecticides. In addition to evaluating their toxicological effect against target insects, these insecticides must be assessed for their adverse impact on natural enemies. In this study, 14 insecticides with different modes of action were selected to investigate sublethal and lethal effects on adult *A. nilaparvatae*, the parasitoid of the BPH, to examine their acute and chronic toxicities for potential compatibility of biological and chemical control for improved IPM of rice pests.

2. Materials and methods

2.1. Insects

The hybrid rice Shanyou63 (30–50 days old after transplanting into plastic pots [15 cm high × 19 cm diameter]) was used as the host plant in this study. Brown planthopper (BPH) and *A. nilaparvatae* were collected from an insecticide-free rice field at Zhengjiang Plant Protection Station in Jiangsu, China. Both BPH and the parasitoid were subjected to verification for correct species and were reared on rice plants, which were kept in cages (55 × 55 × 90 cm) covered with a 0.1 mm mesh nylon cloth. All plants and insects were maintained at 20–30 °C, 60–80% RH, and 14:10 h (L:D) photo-cycle in the greenhouse. Adults of the parasitoids at a uniform age of 2–4 h after emergence were used in the experiment.

2.2. Insecticides

Fourteen insecticides from seven classes (Table 1) were selected for this study. For oral and residual toxicity bioassays, an emulsible concentrate (EC) was prepared by dissolving each insecticide (technical grade) into 90% (v/v) acetone supplemented and mixed uniformly with 10% (v/v) emulsifier Triton X-100 (Shanghai Ling Feng Chemical Reagent Co., Ltd., China). Insecticide of technical grade was dissolved in acetone and was used directly for contact bioassays. The test concentration for each insecticide was the recommended field rate (Ministry of Agriculture of China, 2001, 2006).

2.3. Experiment protocols

2.3.1. Contact toxicity (LC_{50})

We determined the LC_{50} under laboratory conditions. A preliminary experiment was conducted to determine the range of insecticide concentrations (Desneux et al., 2006). Starting with the recommended field application rate, a deceasing set of serial dilutions (10-fold) was made. Adult parasitoids were exposed to these dilutions to determine a concentration yielding approximately 50% mortality. This experimentally derived concentration was then used as a starting point to prepare a set of 2-fold increasing and a set of 2-fold decreasing serial dilutions (5–6 concentrations per insecticide). Adult parasitoids were exposed to the serial concentrations to establish a dose–mortality relationship. The insecticide solutions (in acetone) were applied to scintillation vials (5 cm high, 2 cm diameter). Acetone only was used as the control. To obtain uniform coverage, 500 μl

Table 1
Detailed information of insecticides used in this study: names, manufacturers, stock, and final test concentrations

Insecticides	Technical grade (a.i. %)	Manufacturers	ECs[a] (a.i. %)	Concentration (mg a.i./l)
Buprofezin	98.2	Jiangsu Changlong Chemical Co., Ltd.	5	200
Chlorfluazuron	85.7	Japan Ishihara Industry Co., Ltd.	5	50
Hexaflumuron	97	Jiangsu Yangnong Chemical Co., Ltd.	5	50
JS118[b]	97	Jiangsu Pesticide Research Institute Co., Ltd.	10	120
Silafluofen	93.5	Jiangsu Yangnong Chemical Co., Ltd.	4	40
Isoprocarb	99.7	Jiangsu Changlong Chemical Co., Ltd.	5	200
Abamectin	97.2	Hebei Weiyuan Biochemical Co., Ltd.	1	16
Fipronil	87	Hangzhou Bayer Crop Science Ltd.	4	40
Imidacloprid	95.3	Jiangsu Changlong Chemical Co., Ltd.	5	50
Thiamethoxam	95.87	Swiss Syngenta Crop Protection Inc.	1	10
Chlorpyrifos	96	Nanjing Redsun Co., Ltd	40	400
Dichlorvos	97	Jiangsu Jiangshan Chemical Co., Ltd.	20	200
Methamidophos	70	Shandong Huayang Technology Co., Ltd.	50	500
Triazophos	85	Hubei Xianlong Chemical Co., Ltd.	20	200

[a]Emulsible concentrate.
[b]Insect growth regulator.

of solution was used to ensure complete coverage of the internal surface of the vial. The vial was then manually rotated until no more droplets were seen on the glass wall. Vials were kept at room temperature for 1 h to allow complete evaporation of acetone before introducing the parasitoids. By dividing the applied amount of the insecticide by the internal surface area, the concentration of the insecticide residue on the internal surface of the vial was determined as quantity per unit area. Ten wasps were placed in each vial supplied with a small plastic strip containing 4 μl of 10% honey. The vials were covered with fine nylon mesh to allow air circulation, and were maintained at $27\pm1\,^{\circ}C$, 60–80% RH, and 14:10 h (L:D) photo-cycle. Three replicates were conducted for each dose of each insecticide. After 1 h of exposure, the wasps were transferred into insecticide-free vials supplied with honey solution. After 8 h, dead parasitoids were counted, and the dose response (LD_{50}) was calculated for each insecticide.

2.3.2. Oral toxicity

The testing arena for the oral toxicity test was a transparent plastic Petri dish (3 cm height × 4.8 cm diameter). It had three air vents (1 cm diameter), one on the base and two on the side, which were covered with nylon mesh (0.1 mm). Each insecticide EC was diluted to the experimental concentration (in Table 1) with 10% honey solution. The honey-only solution was used as a control. One microscopic glass coverslip (1.8 × 1.8 cm, 0.12–0.17 mm thick) was placed in each Petri dish. Four droplets (1 μl each) of insecticide–honey mixture or honey-only solution were dispensed on the glass coverslip. Ten female wasps were introduced to each Petri dish. Each treatment had three replicates. Mortality was recorded after 1, 2, 4, and 8 h.

The surviving wasps of each treatment (all replicates) of JS118, hexaflumuron, and chlorfluazuron were removed and transferred to untreated tubes (8.0 × 1.5 cm). Honey solution (10%) was provided as food. Equal numbers of male wasps were also introduced into the tube for mating for an hour. After mating, five females were randomly picked for subsequent assessment of the longevity, fecundity, and emergence of the new generation. An individual female wasp was introduced into a tube (8.0 × 1.5 cm) including a rice stem containing approximately 40–50 BPH eggs, which were well beyond the maximal daily egg production (26 per female wasp) to prevent hyper-parasitism. The longevity of the wasp was recorded every 12 h, and food and BPH eggs were replaced daily until the death of the wasp. Parasitized BPH eggs were recovered from plant tissue and maintained on moist filter paper. The total number of parasitized eggs was counted as the total number of eggs laid per female wasp. The emergence rate was computed as number of new wasps divided by the total number of eggs laid per female wasp.

2.3.3. Residual toxicity

Eleven insecticides were used for their residual toxicity assays. Three pots of rice plants were grouped and sprayed with insecticide until the plants were completely saturated with the solution. Treated plants were placed outside the greenhouse to allow extra insecticide solution on rice leaves to evaporate. Leaves from each treated group were removed and a leaf blade (ca. 7.0 × 1.0 cm) from each leaf was cut out using shears. Two leaf blades were inserted into 1% agar medium (ca. 1 ml) dispensed into the bottom of a tube (8.0 × 1.5 cm) to maintain moisture. Ten adult parasitoids were introduced into each tube. Each treatment was replicated three times. Mortality was recorded after 8 h. Rice leaves were periodically collected (0, 1, 3, and 7 days post insecticide treatment) and were used to investigate residual toxicity of the 11 insecticides to the adult parasitoids.

2.4. Statistical analysis

For contact toxicity bioassays, the EPA probit analysis program version 1.5 (EPA, USA) was used to determine dose–morality relationship and LD_{50} values. Mortality and emergence rates were subjected to arcsine transformation and subsequently analyzed by one-way ANOVA. Means were separated by using Tukey's Student range test (HSD) at $P = 0.05$ (SPSS11.5 (SPSS Inc., 2002)).

3. Results

3.1. Median lethal concentration (LC_{50})

Data of contact toxicity of 14 insecticides to adult *A. nilaparvatae* are summarized in Table 2. Mortality in all control groups was consistently below 5%. The slope of regression line indicates how fast the mortality occurs as concentration increases. Toxicities of the 14 insecticides were ranked as follows: chlorpyrifos > imidacloprid > fipronil and methamidophos > thiamethoxam, isoprocarb, and triazophos > abamectin, silafluofen, and dichlorvos > buprofezin, JS118, hexaflumuron, and chlorfluazuron (LC_{50}'s with overlapping confidence intervals were classified as the same rank). Among all 14 insecticides, the contact toxicity of chlorpyrifos was the highest, and its LC_{50} was only 0.002 mg a.i./l. Four insect growth regulators, buprofezin, JS118, hexaflumuron, and chlorfluazuron, showed the lowest toxicity to the parasitoid. The LC_{50}'s for the IGRs were more than 1000 mg a.i./l.

3.2. Acute and chronic effects of oral toxicity

For acute effects of the oral toxicity assay with female wasps, Tukey HSD tests indicated that the insecticide treatments were significantly different after 1 h ($P < 0.001$, $F = 80.35$, d.f. = 14), 2 h ($P < 0.001$, $F = 61.07$, d.f. = 14), 4 h ($P < 0.001$, $F = 73.34$, d.f. = 14), and 8 h ($P < 0.001$, $F = 108.09$, d.f. = 14) (Fig. 1). All four organophosphate insecticides were highly toxic to female adults, and wasp mortality reached 100% 8 h after exposure. Dichlorvos had the highest oral toxicity among all 14 insecticides, and 100% mortality was obtained only 2 h after treatment. Isoprocarb, imidacloprid, and thiamethoxam were the second most toxic insecticides and killed all wasps in a 4-h period. Eight hours after treatment, fipronil and avermectin generated 100% and 98.9% mortalities, respectively. Silafluofen had relatively low oral toxicity to wasps with 37.6% morality after 8 h. The IGRs had the lowest oral toxicity to the parasitoid. Buprofezin caused 29.4% mortality to the wasps. Chlorfluazuron, hexaflumuron, and JS118 were virtually non-toxic, and caused 4.0%, 1.3%, and 1.1% mortality, respectively, to the parasitoid.

Results of chronic effects of chlorfluazuron, hexaflumuron, and JS118 on female adults are illustrated in Fig. 2. After ingestion of the insecticides, the longevity of the parasitoid was significantly and adversely influenced ($P < 0.05$, $F = 4.190$, d.f. = 3) and oviposition ($P < 0.05$, $F = 7.492$, d.f. = 3). Longevity and fecundity of treated females was approximately 29% and 47% lower than control for JS118, 29% and 49% lower than control for chlorfluazuron, and 24% and 28% lower than control for hexaflumuronand, respectively. Longevity and fecundity of the female wasp treated with JS118 and chlorfluazuron were both significantly different from control. The emergence rates of the offspring adult wasps treated with the three IGRs were slightly lower than that of control, but were not significantly different ($P > 0.05$, $F = 1.208$, d.f. = 3) from that of the control.

Table 2
Acute contact toxicity (LC_{50}) of 14 insecticides to the adult wasps of *Anagrus nilaparvatae*

Insecticides	Slope ± SE[a]	LC_{50} (mg a.i./l)[b]	95% confidence limits
Chlorpyrifos	0.708 ± 0.131	0.002	0.001, 0.004
Imidacloprid	1.416 ± 0.228	0.021	0.013, 0.034
Fipronil	3.044 ± 0.417	0.180	0.147, 0.222
Methamidophos	3.123 ± 0.429	0.191	0.155, 0.234
Thiamethoxam	1.695 ± 0.407	0.520	0.306, 0.908
Isoprocarb	1.921 ± 0.308	1.071	0.781, 1.427
Triazophos	3.525 ± 0.462	1.253	1.045, 1.503
Abamectin	2.657 ± 1.237	8.499	3.309, 33.124
Silafluofen	2.608 ± 0.478	14.220	10.541, 18.530
Dichlorvos	1.960 ± 0.387	15.946	9.440, 24.197
Fuxian (JS118)	–	>1000[c]	–
Hexaflumuron	–	>1000[c]	–
Chlorfluazuron	–	>1000[c]	–
Buprofezin	–	>1000[c]	–

[a]Slope represents increasing speed, SE is standard error.
[b]Median lethal concentration.
[c]When the concentration was 1000 mg a.i./l, the wasp mortality was less than 10%.

3.3. Residual toxicity

Treatments with 11 different insecticides exhibited significant differences ($P < 0.001$) in residual toxicity to the wasps at day 0 ($P < 0.001$, $F = 50.36$, d.f. = 11), day 1 ($P < 0.001$, $F = 46.82$, d.f. = 11), day 3 ($P < 0.001$, $F = 36.02$, d.f. = 11), and day 7 ($P < 0.001$, $F = 18.32$, d.f. = 11) (Fig. 3). Imidacloprid had the longest residual toxicity among 11 tested insecticides, and it still caused 80.7% mortality 7 d after treatment, although 97.6% of wasps were killed on day 0. Thiamethoxam, triazophos, and fipronil also had long residual toxicity to the wasps with 7-d mortalities of 66.8%, 54.6%, and 50.0%, respectively. Methamidophos, chlorpyrifos, and avermectin had relatively short residual toxicity to the wasps, and the 7-d mortality rates were reduced to less than 10%. Silafluofen showed relatively low residual toxicity (1.1% mortality) after 7 d. Dichlorvosand and isoprocarb had short residual toxicity, and no wasp died at day 3. Buprofezin, an IGR, was nearly harmless to the wasps with as little as 1.1% wasps dead on day 0.

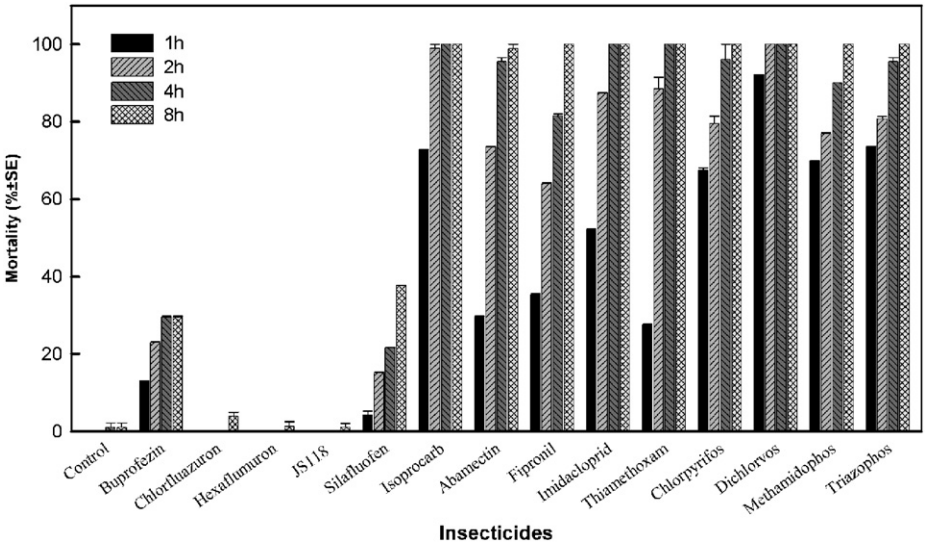

Fig. 1. Acute oral toxicities expressed as mean percentage of mortality of female *A. nilaparvatae* after ingestion of 14 different insecticides. Mortalities were recorded 1, 2, 4, and 8 h after treatment.

4. Discussion

Chemical insecticides that kill target insects may also kill parasitoids directly because of their common physiology. Complete evaluation of an insecticide on natural enemies should include not only its acute toxicity but also its sublethal and chronic effects, which are often ignored because they are not as distinct as acute toxicity (Desneux et al., 2007). A recent study indicated that the sublethal effects of insecticides on natural enemies may ultimately cause beneficial insects to become less effective as biological control agents in the field due to their low performance in parasitizing and preying on hosts (Poletti et al., 2007). Therefore, in addition to mortality, an assessment of the impact of an insecticide on beneficial insects should include sublethal effects, together with information on the residual activity of insecticides, as we and others have shown that certain insecticides can cause sublethal effects on beneficial insects for many days after their application (Tipping and Burbutis, 1983).

While egg and larva are double-shielded (i.e. host egg shell and plant tissue) from insecticide spray, the adult *A. nilaparvatae* is most likely to be exposed to insecticides through contacting host insects and plants, which have been newly sprayed with insecticides. In addition, natural enemies might be poisoned when their food sources are contaminated with insecticides. The secretion of honeydew from the BPH is an important food source for *A. nilaparvatae* (Zheng et al., 2003), and it is easily contaminated when a pesticide is applied. The toxic chemical will be ingested along with the food. Subsequently, parasitoids may die or their performance may be adversely influenced (Haseeb and Amano, 2002). This study, aiming to improve compatibility of chemical and biological control, was designed to simulate field conditions of insecticide sprays and to assess potential risks of acute contact and oral, residual, and chronic effects of 14 insecticides, all potential candidates for replacing highly toxic organophosphate insecticides. Our study demonstrated that different insecticides possessed significantly different risks to *A. nilaparvatae*, which in turn might provide more choices for integration of chemical control with biological control, such as using different insecticides at sensitive and less sensitive stages of the parasitoid.

Among the 14 insecticides evaluated, three IGRs, JS118, hexaflumuron, and chlorfluazuron, had no obvious contact or oral toxicity to adult wasps. Because JS118 mimics the action of the insect molting hormone ecdysone, and hexaflumuron and chlorfluazuron function as chitin synthase inhibitors, these chemicals should be safe to adults and are not acutely toxic to the wasps (Gerling and Sinai, 1994; Jones et al., 1998). However, we found that JS118 and chlorfluazuron substantially reduced longevity and fecundity of female wasps, but they did not affect filial adult wasp emergence. Haseeb and Amano (2002) also reported a reduction in parasitism when chlorfluazuron was ingested by *Cotesia plutellae* adults. It is likely that the IGRs adversely affect oogenesis of adult wasps and subsequently the development of the eggs within host larvae. Unlike the other two chitin synthase inhibitors, buprofezin had a certain degree of acute oral toxicity. However, several studies have shown no or minimal impact of buprofezin on hymenopteran parasitoids (Garrido et al., 1985; Smith and Papacek, 1990; Gerling and Sinai, 1994; Hoddle et al., 2001). IGRs were highly toxic to the target pest, but relatively safe to natural enemies (Zhou et al., 2003). These insecticides are potential candidates for further consideration in rice IPM. Further tests of these IGRs, under field conditions, need to be conducted.

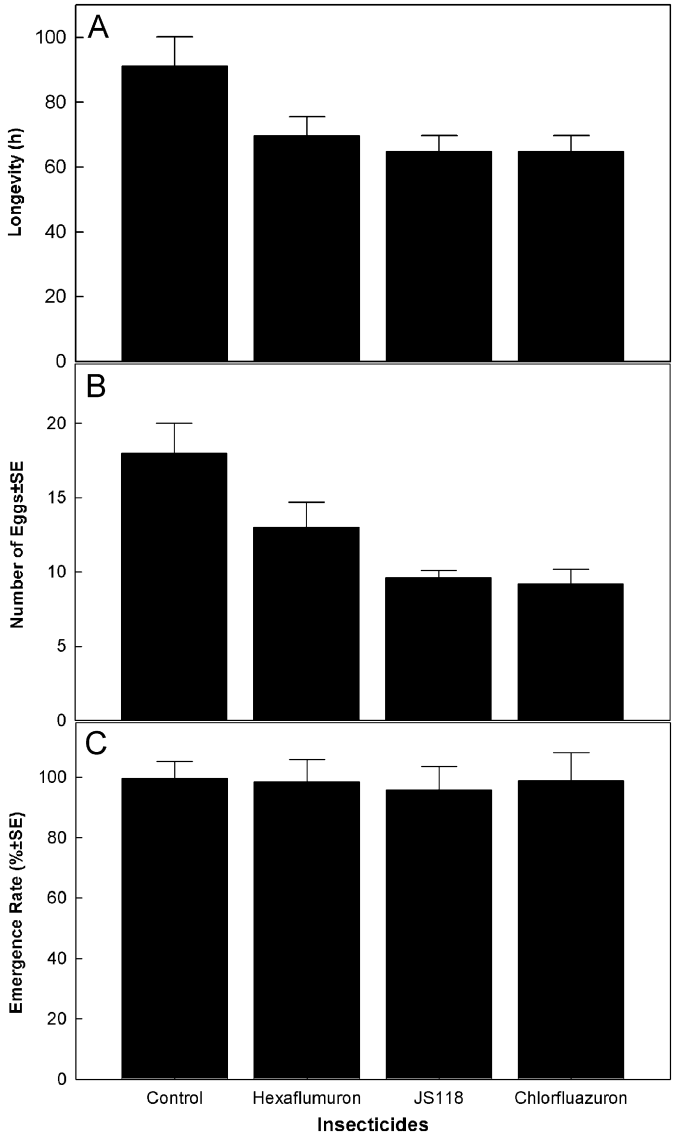

Fig. 2. Chronic effects of oral toxicity of three insect growth regulators on (A) longevity (hours); (B) fecundity (number of eggs); and (C) emergence rate (%) of offspring.

In addition, the influence of IGRs on parasitoid physiology and behavior needs to be investigated.

Silafluofen, a pyrethroid insecticide, has high insecticidal and miticidal efficacies and is safe to mammals and fish (Xue, 2004). However, limited research has been done to clarify its toxic effect on natural enemies. In this study, we found that silafluofen had low toxicity to adult *A. nilaparvatae*. Tao et al. (2006) found that silafluofen had high efficacy against *Chilo suppressalis* (walker), another important rice insect. Because of its higher efficacy against many rice pests and relatively low toxicity to the parasitoid, silafluofen is another potential insecticide for rice IPM.

Isoprocarb, a carbamate insecticide, had relatively low contact and residual toxicity, but high acute oral toxicity to adult *A. nilaparvatae*. Zhang et al. (1988) found that isoprocarb exhibited high efficacy against target pests and low toxicity to spiders and pupae of *Anagrus* spp. Limited available information about isoprocarb necessitates further physiological and behavioral studies of this insecticide on natural enemies in rice.

Kaspi and Parrella (2005) indicated that direct contact or ingestion of abamectin caused a significant negative effect on *Diglyphus isaea*. Our results also showed that abamectin displayed low contact and residual toxicity, but high oral toxicity to *A. nilaparvatae*. Its short residual toxicity was also observed in adult parasitoids of *Dacnusa sibirica* (Shipp et al., 2000). The adverse effect of abamectin on parasitoids might be associated primarily with oral toxicity. Compared with many traditional insecticides, however, the oral toxicity of abamectin was relatively low, and its use for rice IPM is still feasible, provided caution is properly taken (Prijono et al., 2004).

Two classes of insecticides showed relatively high acute contact and ingestion toxicity, as well as long residual toxicity to *A. nilaparvatae*. The phenylpyrazole insecticide, fipronil, disrupts normal nerve function by blocking the GABA-gated chloride channels (Cole et al., 1993). It kills insects by contact and ingestion. Our data showed that fipronil had not only high contact and acute oral toxicity but also a long residual effect on *A. nilaparvatae*. Hu and Zhao (2003) also found that fipronil was harmful to *Cotesia plutellae* and *Oomyzus sokolowskii*. Neonicotinoid insecticides were introduced into the market in the early 1990s and are currently one of the most important chemical groups used to control sucking insects (Nauen et al., 2001). However, the use of neonicotinoid insecticides should be evaluated carefully in IPM programs (Poletti et al., 2007). Results from this study showed that both imidacloprid and thiamethoxam had high acute contact and oral toxicity. A significantly high residual toxicity was also detected, indicating an association with their high stability. However, imidacloprid was relatively safe to *Trichogramma* nr. *brassicae* (Hewa-Kapuge et al., 2003). Different crop substrates (rice vs. tomato) and sensitivities of the two species might contribute to the discrepancy between our results and the findings of Hewa-Kapuge et al. (2003). Considering their high efficacy against rice insects and adverse effect on the parasitoid, all three of the insecticides representing those two classes must be used with precaution to avoid direct contact and minimize contamination of the food source for the parasitoid.

Another high-risk insecticide group is the organophosphate insecticides. Dichlorvos, chlorpyrifos, methamidophos, and triazophos had high acute oral toxicity to *A. nilaparvatae*. Li and Liu (2005) also found that both chlorpyrifos and dichlorvos were highly toxic to *Plutella xylostella*. But the contact and residual toxicities were different among the four organophosphates in our study. Chlorpyrifos had the highest contact toxicity to *A. nilaparvatae*. A similar result was also obtained with *Aphidius ervi* (Desneux et al., 2004). Dichlorvos had the shortest residual toxicity, which might be caused by its low stability in the rice environment due to its evaporative

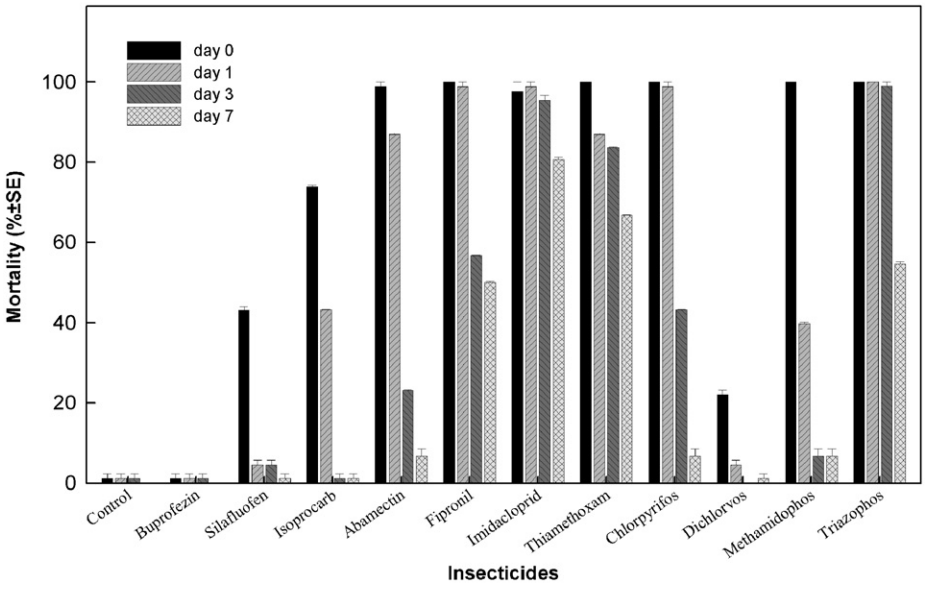

Fig. 3. Residual toxicity determined as mean mortality of *A. nilaparvatae* after the wasps were exposed to insecticide residues on rice leaves collected at day 0, day 1, day 3, and day 7 after spraying with 11 different insecticides. Mortality rates were assessed after 8 h of exposure to the insecticide residues.

nature. Unlike dichlorvos, triazophos is relatively stable in the environment (Rani et al., 2001) and exhibited the longest residual toxicity to *A. nilaparvatae*. Further studies on physiology and behavior may be necessary to fully understand the impact and use of these insecticides when the parasitoids are at less sensitive or less active stages.

In summary, 14 insecticides were evaluated for their lethal and sublethal effects on an important biological control agent in rice, *A. nilaparvatae*. This study provided important information for implementing compatible biological and chemical control for rice planthopper IPM. However, the impact of insecticides on parasitic wasps is a complex situation, which requires a systematic study to determine short-term and long-term influence on the biology, physiology, and behavior of both the host and parasitoid populations.

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