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Full Length Research Paper

Susceptibilities of two populations of *Aphis gossiper* Glover to selected insecticides

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Two populations of *Aphis gossypii* were collected from cotton and melon crops treated with insecticides to control this aphid species. The susceptibility of both aphid populations to pymetrozine, Pirimicarb, Oxydemeton-methyl and Imidacloprid was evaluated using leaf deep bioassays in Laboratory which were commonly used to control this aphid on both crops. Results showed that LC_{50} values of these insecticides against clones of cotton aphid were 452, 1427, 1810 and 209 ppm, respectively. LC_{50} values of the above mentioned pesticides against clones of melon aphid were 625, 688, 523 and 125 ppm, respectively. Lc_{50} data showed that aphids reared on melon was 2.07, 1.6 and 3.4 times more susceptible than cotton aphids to Pirimicarb, Imidaclopride and Oxydemeton-methyl and 1.4 times more resistant to Pymetrozine, respectively. In conclusion, it has been shown that clones of cotton aphid is on average 3.4-fold less susceptible to Oxydemeton- methyl, 2-fold less susceptible to pirimicarb, 1.6-fold less susceptible to Imidacloprid and nearly 0.7-fold more susceptible to pymetrozine than clones of melon aphid. There was little difference in susceptibility to pymetrozine between the two populations. It is also suggested that continuous resistance monitoring should be conducted on a regional scale to identify the efficiency of compounds which are applied against this insect species.

Key words: *Aphis gossypii*, cotton, melon, insecticide resistance.

INTRODUCTION

The melon aphid, *Aphis gossypii* Glover (Hemiptera: Aphididae), is found throughout most of the temperate, subtropic and tropic regions of the world. This insect has a wide host range feeding on about 700 host plants, including watermelon, cucumbers, cantaloupes, squash pumpkin, cotton, citrus, eggplant, pepper, asparagus, bean, beet, potato and okra (Leclant and Deguine, 1994).

Melon aphids suck nutrients from the plant causing foliage to become chlorotic and die. Also, their feeding causes distortion and leaf curling, interfering with photo-synthetic capacity of the plant. Moreover, this insect species secretes honeydew which provides a growing media for saprophytic fungi (*Capnodium* spp *Cladosporium* spp and *Fumago* spp) on plant tissues (Hillocks and Bretell, 1992). Melon aphid transmits potyviruses and probably some other viruses such as cucumber mosaic virus, watermelon mosaic virus and zucchini yellow mosaic virus (Capinera, 2007). Cotton aphid had the potential to develop resistance to insecticides due to high reproductive potential (Mallet and Luttrell, 1991).

Insecticides such as pirimicarb, oxydemton- methyl, imidacloprid and pymetrozine are often used to manage *A. gossypii* in the cotton and the other crops. Imidacloprid acts on the nicotinic acetylcholine receptor, causing the insect to reduce or stop feeding and reduces mobility (Boiteau and Osborn, 1997). Pymetrozine impacts feeding behavior (Harrewijn and Kayser, 1999). Pirimi-carb, a selective aphicides and Oxydemeton- methyl both act as an acetylycholinesterase inhibitor (Mooress et al., 1996; Menozzi et al., 2004).

Intensive use of insecticides often leads to resistance development by sprayed aphids, forcing farmer to in-crease dosage of application frequency. Resistances of *A. gossypii* to some insecticide have been reported (Andrew et al., 2006; Wang et al., 2002). Mechanisms in

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Table 1. Concentrations (ppm) used in the experiments for all four insecticides against two populations (melon aphid and cotton aphid) of *A. gossypii*.

Concentration tested (ppm)		Recommended dosage (ppm)	Insecticide type
Cotton aphid	Melon aphid		
500,1000,2000,4000, 8000	250,500,1000,2000, 4000	500 - 700	Pirimicarb
500,1000,2000,4000,8000	125,250,500,1000,2000	1000	Oxydemton-metyl
44,87,175,350,700	42,85,175,350,700	250	Imidacloprid
125,500,1000,2000,4000	125,500,1000,2000,4000	1000	Pymetrozine

which Insects develop resistance to insecticides include decrease in insecticide penetration through cuticle which reduce target site sensitivity and enhance metabolism (Plapp, 1976; Oppenoorth, 1984); Enhancement of chemical metabolism, decreasing the effective amount of insecticides that can kill insects. Cytochrome P450 monooxygenases, glutathione *S*-transferases and esterases are the major detoxifying enzymes that are involved in insecticide resistance (Oppenoorth, 1984).

In recent years there are complains about insufficient control results of available chemicals on both melon and cotton aphids. So, the aim of the current study was to evaluate susceptibilities of two populations of *A. gossypii* to insecticides used for control of this aphid species in Iran. These insecticides are from four different insecticide groups including pirimicarb, oxydemeton- methyl, imidacloprid and pymetrozine. So, the efficacy of these insecticides on two populations of *A. gossypii*, reared on cotton and melon were investigated to determine these insecticides effectiveness on the *A. gossypii* control. Bioassays using treated leaf disks were used to determine dose response curves for both populations.

MATERIALS AND METHODS

Insect rearing

A. gossypii used in these experiments were collected from melon and cotton farms of Torbat Jam, Iran. Two aphid colonies were established separately on *Cucumis melo* var khatooni and on *Gossypium hirsutum* var varamin in greenhouse at 20 ± 2°C, a 16 h light: 8 h dark cycle and relative humidity of 55 ± 5% as described by (Lashkari et al., 2008). These colonies were kept on each rearing plants for several generations. Every week (5 - 7 days) plants were replaced with new ones in order to keep colonies alive. Apterous adults from these colonies were used in this study. Seedlings used for aphids culturing as well as producing leaf disks for insecticide bioassays were grown in plastic pots in above mentioned conditions.

Toxicity bioassay

Four insecticides used in this experiment were Pirimicarb 50% WP (China's Jecom Company), Pymetrozine 25% WP (Iran's Moshkfaamfars Company), Imidacloprid (Confidor®) 35% SC (German's Bayer Company) and Oxydemton-methyl (Metasystox-R®) 25% EC (German's Bayer Company).

Leaf dip assays were performed according to the procedures described by Bandani and Butt (1999).. Initially, for each insecticide on each population, bracketing test was done to determine doses that produce satisfactory range (10 - 90% mortality). The used concen-trations were given in Table 1.

All four insecticides were diluted with distilled water and each assay consisted of 25 apterous adult per treatment (each dose) and each treatment replicated 5 times. Plant leaf was cut (three weeks old seedlings leaf) and dipped into insecticide solution for 10 s and allowed to dry for 30 min before exposing the insects to it. For controls, plant leaves were treated with distilled water alone. Mortality was assessed after 48 h. Mortality data were corrected with Abbott's formula (Abbot, 1925).

Data analysis

In these experiments, concentration-mortality regression for the adult from each bioassay was evaluated statistically using probit analysis (Polo-PC Probit and Logit analysis; LeOra Software 1997) to determine the lethal concentrations (LC_{50}s). Differences in toxicity were considered significant when 95% Fiducial Limit (FL) did not overlap (Adams et al., 1990). Unavailability of known susceptible strain of *A. gossypii* has led comparison of LC_{50} between two populations that had collected from cotton and melon fields and reared on them.

RESULTS

Susceptibilities to imidacloprid (Confidor®)

Toxicity of imidacloprid against *A. gossypii* obtained originally from melon and cotton is illustrated in Table 2. Lethal toxicity (LC_{50}) of imidacloprid to aphid was 125 ppm and to cotton aphid was 209 ppm. There was a 1.67 fold increase in response values (Table 2) that it shows that average LC_{50} for cotton aphid was 1.67 fold more than melon aphid. However, there was considerable overlap between individual values that it means there were not significant differences in tolerance to the insecticide. Both species are susceptible to imidacloprid but comparing the responses of the two clones showed that clones of cotton aphid were moderately more tolerant to the insecticide compared with those clones of melon aphid.

Susceptibilities to oxydemeton-methyl (Metasystox-R®)

Average LC_{50} values ranged from 1810 ppm for cotton

Table 2. Susceptibility of two populations (melon and cotton aphids) of *A. gossypii* to imidacloprid based on probit analysis of mortality after 48 h.

Slop ± SE	X^2(df)	N.	Imidacloprid LC_{50} LC_{90}		Aphid population
1 ± 0.1	14.1(3)	750	673(333 - 7339)	125)43 - 231)	Melon aphid
1 ± 0.1	10.8(3)	750	1025(531.5 - 5680)	209(121 - 358)	Cotton aphid

LC values are based on ppm; values in parenthesis show Fiducial Limit (FL); N: number of insects treated. Slope: derived from regression equation of mortality values. SE: standard error.

Table 3. Susceptibility of two populations (melon and cotton aphids) *A. gossypii* to oxydemton-metyl based on probit analysis of mortality after 48 h.

Slop ± SE	X^2(df)	N.	Oxydemton-metyl LC_{50} LC_{90}		Aphid population
1 ± 0.2	1.7(3)	750	2521(1938 - 3622)	523(424 - 628)	Melon aphid
0.9 ± 0.2	6.7(3)	750	7296(4587 - 18489)	1810(1062 - 2672)	Cotton aphid

LC values are based on ppm; values in parenthesis show Fiducial Limit (FL); N: number of insects treated. Slope: derived from regression equation of mortality values. SE: standard error.

Table 4. Susceptibility of two populations (melon and cotton aphid) *A. gossypii* to pirimicarb based on probit analyses of mortality after 48 h.

Slop ± SE	X^2(df)	N.	Primicarb LC_{50} LC_{90}		Aphid population
1 ± 0.1	4.3 (3)	750	5186 (2928 - 16966)	688(380 -1038)	Melon aphid
1.1 ± 0.1	1.1(3)	750	13740(9309 - 24384)	1427 (1089 - 1792)	Cotton aphid

LC values are based on ppm; values in parenthesis show Fiducial Limit (FL); N: number of insects treated. Slope: derived from regression equation of mortality values. SE: standard error.

aphid to 523 ppm for melon aphid (Table 3). Fiducial limits do not overlap so differences in toxicity are considered significantly different.

There was a 3.46 fold increase in response values (Table 3) that it shows that average LC_{50} for cotton aphid was 3.46 fold more than melon aphid. Also, it shows that clones of cotton aphid were more tolerant (3.46 times) to the insecticide compared with those clones of melon aphid. Conversely, melon aphid was more susceptible to oxydemetom-methyl than cotton aphid.

Susceptibilities to pirimicarb (Pirimor®)

Toxicity of pirimicarb on *A. gossypii* was shown in Table 4. As indicated in the table, lethal toxicity (LC_{50}) of pirimicarb to melon aphid was 688 ppm and to cotton aphid was 1427 ppm. There was a 2.07 fold increase in response values (Table 4) that it shows that average LC_{50} for cotton aphid was 2.07 fold more than melon aphid.

Since there is no overlap between individual values of Fiduicial limit, it shows that there were significant differences in tolerance to the insecticide between two populations. These data showed that clones of cotton aphid were more tolerant to the insecticide compared to the clones of melon aphid.

Susceptibilities to pymetrozine (Chess®)

Susceptibility of two populations of *A. gossypii* to pymetrozine is shown in Table 5. Average toxicity (LC_{50}) of pymetrozine to cotton aphid was 453 ppm and to melon aphid was 625 ppm. These values show that cotton aphid is more susceptible (1.4 fold) to pymetrozine than melon aphid. However, the data show that there is overlap between individual values of Fiducial limit. Thus, there are not significant differences in susceptibility between the two populations.

DISCUSSION

In this study, it was found that clones of aphid on melon was more susceptible to imidaclopride than clones of aphid on cotton explaining that application of more insecticide in cotton crop against pests including aphids. Thus, showing clones of cotton aphid are more tolerable to pesticides. This result was similar to finding of Wang et al. (2002) that found clones of cotton aphid was significantly more tolerable to fenvalerate and imidacloprid than cucumber (*Cucumis sativa* L.) clones. Hugh et al. (2003) found that *A. gossypii* had high susceptibility to imidacloprid even their finding showed that the

Table 5. Susceptibility of two populations (melon and cotton aphid) *A. gossypii* to pymetrozine based on probit analyses of mortality after 48 h.

Slop ± SE	X^2(df)	N.	Pymetrozine LC_{50}	LC_{90}	Aphid population
0.4 ± 0.1	7.5 (3)	750	5248 (2186 - 96129)	625 (287 - 1208)	Melon aphid
0.4 ± 0.1	0.2(3)	750	2774 (2054 - 4204)	453 (366 - 548)	Cotton aphid

LC values are based on ppm; values in parenthesis show Fiducial Limit (FL); N: number of insects treated. Slope: derived from regression equation of mortality values. SE: standard error.

insecticide has lower LC_{50} than recommended dosage. The largest difference in susceptibility to an insecticide for *A. gossypii* occurred after exposure to oxydemeton-methyl. The LC_{50} value for cotton aphid was 1810 ppm (AI) which was 3.4 fold of melon aphid. These LC_{50} values were more than recommended dosage, for two populations. After metasystox, the lowest suscep-tibility belonged to Pirimor®. The LC_{50} value for cotton aphid was 1427 ppm (AI) which was 2.07 fold of melon aphid. The LC_{50} values for cotton aphid were more than recommended dosage, but for melon aphid was almost equal to that.

O'Brien et al. (1992) found carbamate and organochlorine resistance in cotton aphid from Mississippi. By comparisons of LC_{50}s of several populations of *A. gossypii* Glover, Hollingsworth et al. (1994) showed up to > 2,000-fold resistance to oxydemeton-methyl that these values were positively correlated with the previous use of organophosphates. Sun et al. (1987) found that a combination of elevated carboxylesterase activity and reduced acetylcholinesterase sensitivity are responsible for the organophosphate resistance in some *A. gossypii* strains. Takada and Murakami (1998) using electro-phoresis detected esterase pattern of resistant *A. gossypii* to Malathion and pirimicarb. They showed that high esterase activity in this species plays an important role in resistance to malathion and pirimicarb. Devonshire (1989) suggested that *A. gossypii* had high tolerance to pirimicarb because of existence of a mutant form of acetylcholinesterase that is less sensitive to inhibition by pirimicarb.

Nauen and Elbert (2003) found that *Myzus persicae* and *A. gossypii* had no resistance to imidacloprid. In contrast they found that *M. persicae* and *A. gossypii* had a strong resistance to pirimicarb and oxydemeton-methyl and to a lesser extent to cyfluthrin.

Pymetrozine is a fast acting and selective inhibitor of aphid feeding and this compound is not a neurotoxin (Harrewijn and Kayser, 1999; Lowery et al., 2006). Thus, pymetrozine does not have a toxic effect on aphids but interferes with the nervous regulation of feeding behavior that result in death due to starvation within a few days (Harrewijn and Kayser, 1999). In the current study, mortality of aphids due to pymetrozine started after 48 - 72 h. Adult population of *A. gossypii* had high susceptibility to pymetrozine in both populations except for melon aphid that was more susceptible to the insecticide in low doses.

In conclusion, it has been shown that clones of cotton aphid is on average 3.4-fold less susceptible to oxydemeton-methyl, 2-fold less susceptible to pirimicarb, 1.6-fold less susceptible to imidacloprid and nearly 0.7-fold more susceptible to pymetrozine than clones of melon aphid. There was little difference in susceptibility to pymetrozine between the two populations. It is also suggested that continuous resistance monitoring should be conducted on a regional scale to identify the efficiency of compounds which are applied against this insect species.

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