Journal of Plant Protection Research ISSN 1427-4345

ORIGINAL ARTICLE

Resistance risk assessment: realized heritability, cross resistance and resistance stability of acetamiprid in the cotton aphid, *Aphis gossypii* Glover (Homoptera: Aphididae)

El-Sayed Mohammad Soliman Mokbel*

Central Agricultural Pesticides Laboratory, Agriculture Research Center, 12618 Giza, Egypt

Vol. 58, No. 4: 328–334, 2018

DOI: 10.24425/jppr.2018.124641

Received: February 4, 2018
Accepted: October 17, 2018

*Corresponding address:
sayedmokbel@yahoo.com

Abstract

The cotton aphid, *Aphis gossypii* is an economically significant insect pest infesting various important crops and vegetables. The neonicotinoid, acetamiprid was recommended against aphids with excellent results. Resistance emergence and environmental pollution makes acetamiprid a favorable alternative to conventional insecticides. The aims of the present work were to predict acetamiprid resistance risk in *A. gossypii*, investigate cross resistance to other tested insecticides and explore acetamiprid stability in the absence of selection. A field-collected population from Sharqia governorate, Egypt was selected with acetamiprid. After 16 generations of selection, there was a 22.55-fold increase in LC_{50} and the realized heritability (h^2) of resistance was 0.17. Projected rates of resistance indicated that, if $h^2 = 0.17$ and 50% of the population was killed at each generation, then a tenfold increase in LC_{50} would be expected in 12.2 generations. If h^2 was 0.27 then 7.63 generations would be needed to achieve the same level. In contrast, with h^2 of 0.07 it necessitates about 30 generations of selection to reach the same level. Cross resistance studies exhibited that the selected strain showed obvious cross resistance to the other tested neonicotinoid members, moderate cross resistance to alpha-cypermethrin and no cross resistance to pymetrozine. Fortunately, resistance to acetamiprid in the cotton aphid was unstable and resistance reverses the nearby susceptible strain throughout five generations without exposure to acetamiprid. Our results exhibited cotton aphid potential to develop resistance to acetamiprid under continuous selection pressure. The instability of acetamiprid makes *A. gossypii* amenable to resistance management tactics such as rotation with pymetrozine.

Keywords: acetamiprid, *Aphis gossypii,* heritability, resistance

Introduction

The cotton aphid, *Aphis gossypii* Glover (Homoptera: Aphididae) is a key insect pest infesting a wide range of vegetables and field crops around the world. Aphid infestation causes major yield losses due to their deleterious effects through direct feeding, honeydew excretion and virus transmission (Xu *et al.* 2004). Cotton aphid control is mainly dependent on the application of various insecticide classes particularly neonicotinoids which are widely used as alternatives to conventional insecticides against sucking insects (Tomizawa and Casida 2003). Acetamiprid showed excellent activities against Homoptera, Thysanoptera and Lepidoptera. Therefore, it represents an ideal alternative to combat resistant pest populations. Despite the successful use of neonicotinoids to combat target pests, several species have now showed resistance levels that threaten the control success of these insecticides (Bass *et al.* 2015). Repeated use of neonicotinoids has led to a resurgence of resistant cotton aphid populations (Hirata *et al.* 2015). In Japan, a neonicotinoid-resistant cotton aphid population exhibited high resistance to seven neonicotinoids with an acetamiprid resistance ratio

value of 104-fold (Matsuura and Nakamura 2014). Similarly, in Australia, *A. gossypii* exhibited resistance to the neonicotinoids acetamiprid, clothianidin and thiamethoxam (Herron and Wilson 2011). Resistance to neonicotinoids in other insect species was reported including tobacco thrips (*Frankliniella fusca* Hinds) (Huseth *et al.* 2016) and the brown planthopper, *Nilaparvata lugens* L. (Yang *et al.* 2016).

Due to pest resistance potential, estimating the resistance risk to a particular pesticide before its actual occurrence in the field is of great importance to avoid or at least delay resistance problems (Keiding 1986). Resistance risk assessment can originate from several sources, including selection experiments (Jutsum *et al.* 1998). Selection experiment data can be subjected to quantitative genetic methods and the heritability of resistance estimated. Realized heritability (h^2) is an index to quantify pushing degree to a trait in a population by selection. It is defined as the ratio of genetic variance to total phenotypic variance. It provides an effective way to predict future resistance evolution in response to selection (Tabashnik 1992). Therefore, resistance risk assessment before resistance breaks out in the field can provide useful information supporting a proactive implementation of strategies to manage and maintain susceptibility in field populations and sustain the efficacy of acetamiprid.

Consequently, preserving the efficacy of newly introduced pesticides is an important goal of resistance management. When resistance appears, certain tactics are practiced such as rotation with non-cross resistance pesticides cross generations. However, the success of these tactics depends on certain factors such as patterns of cross resistance with alternative pesticides. Positive cross resistance correlation reduces the effectiveness of resistance management programs. Cross resistance in neonicotinoid resistant pests to other neonicotinoid members and to other insecticides has been reported in the cotton aphid (Wang *et al.* 2002; Cui *et al.* 2016; Wei *et al.* 2017), the bird cherry-oat aphid, *Rhopalosiphum padi* L. (Wang *et al.* 2017) and the brown planthopper, *Nilaparvata lugens* (Stal) (Yang *et al.* 2016).

In addition, it has been demonstrated within several species that exploring resistance stability in the absence of selection is essential and may be required as a prerequisite for resistance management strategies (Bush *et al.* 1993). Resistance reversion could be attributed to fitness disadvantages associated with resistance. Resistance exerts negative effects on life history traits such as pupae weight which correlates with reduced fecundity as well as the inability of the resistant individuals to compete effectively with the susceptible ones in reproductive potential and other biotic factors (Ninsin and Tanaka 2005). Acetamiprid resistance reversion was investigated in the cotton mealy bug, *Phenacoccus solenopsis* (Tinsley), and found to be unstable in the absence of acetamiprid selection (Afzal *et al.* 2015).

The current study aimed to investigate the risk of resistance evolution in *A. gossypii* to acetamiprid as a result of laboratory selection, cross resistance to other insecticides and stability of acetamiprid resistance in the absence of acetamiprid selection. The obtained results will contribute in developing resistance management strategies.

Materials and Methods

Insecticides

Tested insecticides were: acetamiprid (Acetamiprid 20% SP, Barighat India), dinotefuran (Oshin 20% SG, Mitsui Chemicals, Inc., Japan), imidacloprid (Best 25% WP, El-Help Pesticides and Chemicals, Egypt), thiacloprid (Thiacloprid 48% Sc, Jiangsu Flagchemicals Industry Co., China), alpha-cypermethrin (Alpha-cyper 10% EC, the National Company for Agrochemicals & Investment, Egypt) and pymetrozine (Chess 25% WP, Syngenta Agro., Switzerland).

Insects

The cotton aphid, *A. gossypii* was collected from cotton fields in Sharqia Governorate, Egypt, in September 2015 and divided into two parts. The first part was maintained in the laboratory without exposure to any insecticides and served as the susceptible strain (S-strain). The other part was selected with acetamiprid to develop the resistant strain (R-strain). Aphids were reared on cotton seedlings under constant laboratory conditions [22 ± 2°C, 70 ± 5% relative humidity (RH) and 12 : 12 light-dark photoperiod]. Cotton seedlings were planted in plastic pots (15 cm diameter) and after cotton seeds germination were thinned to one seedling per pot and then used in four true leaf stages. Cotton seedlings were continuously supplied until the necessary aphids were obtained.

Bioassays

Leaf – dip bioassay was used to assess the toxicities of the aforementioned insecticides according to Moores *et al.* (1996). Cotton leaves were dipped in the desired insecticide solution for about 10 s, allowed to dry on a paper towel and then placed upside down on an agar bed in Petri dishes (60 mm diameter). Ten apterous adults were moved to the treated leaf for each replicate. Leaves dipped in water were considered to be the control. Five to seven insecticide concentrations and five replicates per each concentration were used. Aphids were kept under laboratory conditions until mortality

was recorded after 48 h. The resistance ratio (*RR*) was calculated according to the formula:

$RR = LC_{50}$ value of R-strain/LC_{50} value of S-strain,

where: LC_{50} – the median lethal concentration.

Selection for resistance

Based on initial bioassay results, selection for acetamiprid resistance was accomplished by the dipping technique of Guo *et al.* (1996). Apterous adults (more than 500 adults) were moved to four true leaf cotton seedlings (10–15 cm high) 24 h before treatment. The plants bearing the aphids were dipped in insecticide dilution for 10 s, allowed to air dry for about 1 h, and then set in a rearing room. The aphids which survived were moved to new plants and kept until they became apterous adults of the next generation and then used for bioassay.

Estimation of realized heritability

Realized heritability (h^2) of resistance to acetamiprid in *A. gossypii* was estimated according to Tabashnik (1992) as follows:

$$h^2 = R/S,$$

where: R – the response to selection, S – the selection differential.

Response to selection was estimated as:

$$R = [\log(\text{final } LC_{50}) - \log(\text{initial } LC_{50})]/n,$$

where: n – the number of selected generations.

The final LC_{50} is the LC_{50} of *A. gossypii* offspring after n generations of selection. The initial LC_{50} is the LC_{50} of the parent generation before selection. The selection differential (S) was estimated as: $S = i\sigma_p$, where i represents the intensity of selection according to Falconer and Mackay (Appendix A) (Falconer *et al.* 1996). The phenotypic standard deviation (σ_p) is the reciprocal of the mean slopes originated from the probit analysis of the selected strain (σ_p = 1/average slope).

Genetic parameters (such as R, S, and h^2) were calculated separately for both the first and the second round of selection experiments to investigate their changes during selection. Based on the R-value, the number of generations required for a tenfold increase in LC_{50} (G) is the reciprocal of R:

$$G = R^{-1} = (h^2S)^{-1}.$$

The decline in resistance (*DR*) in R-strain was estimated according to the following formula:

$$DR = [\log(\text{final } LC_{50}) - \log(\text{initial } LC_{50})]/n,$$

where: n – the number of generations reared without acetamiprid selection pressure.

Data analysis

Mortality was corrected using Abbott's formula (Abbott 1925) and data were analyzed by probit analysis (Finney 1971) using the software package EPA probit analysis version 1.5.

Results

Selection of resistance to acetamiprid

Selection with the LC_{50} of acetamiprid for 16 consecutive generations caused an increase in LC_{50} values from 0.55 to 12.18 ppm. The resistance ratio (*RR*) increased 36.98-fold as compared to the *S*-strain (Table 1).

Realized heritability (h^2)

The overall estimated h^2 mean of acetamiprid resistance in *A. gossypii* (F0–F15) was 0.17. The h^2 of acetamiprid resistance was 0.22 and 0.13 in the first half and in the second round of selection, respectively. Response to selection was higher in the first round than in the second round of selection causing an increased h^2 in the first round compared to the second round of selection (Table 2).

Projected rate of acetamiprid resistance development

The projected rate of resistance development is directly proportional to h^2 and selection intensity (Fig. 1). For example, if we assume that the slope = 1.64 [the value of mean slope for (R-strain) generations in this study] and h^2 = 0.17, then 12–6 generations would be required for a tenfold increase in the LC_{50} at a 50–90 percent selection intensity, respectively. However, at a similar slope, if h^2 = 0.07, then 30–14 generations would be required for a tenfold increase in the LC_{50} at a 50–90 percent selection intensity. Likewise, if h^2 = 0.27, then the same would occur in 8–3 generations at 50–90 percent selection intensity (Fig. 1).

The projected rate of resistance development is inversely proportional to the slope. For example, if we assume that h^2 = 0.17 (heritability of acetamiprid resistance estimated in this study) and slope = 1.64, then a tenfold increase in the LC_{50} would occur in 12–6 generations at a 50–90 percent selection intensity, respectively. However, at the same h^2, if the slope = 2.64, then 20–11 generations would be required for a tenfold increase in the LC_{50} at 50–90 percent selection intensity, respectively. Likewise, if the slope = 0.64, then the same would occur in 5–2 generations at 50–90 percent selection intensity, respectively (Fig. 2).

Table 1. Resistance levels of *Aphis gossypii* to acetamiprid during the laboratory selection process

Generation	LC_{50} (95% CL) [mg · l^{-1}]	Slope ± SE	χ^2	RR^a	RR^b
S-strain	0.33 (0.03–0.65)	1.15 ± 0.44	0.51	1.00	
F0	0.55 (0.15–0.91)	1.62 ± 0.47	1.35	1.63	1.00
F1	1.75 (1.06–2.92)	1.38 ± 0.33	0.41	5.30	3.18
F2	2.58 (1.57–3.80)	1.78 ± 0.39	1.24	7.06	4.69
F3	3.10 (1.91–4.57)	1.89 ± 0.33	1.77	9.39	5.63
F4	3.34 (1.96–4.83)	1.95 ± 0.38	2.08	10.12	6.07
F5	4.16 (2.23–5.97)	2.07 ± 0.49	1.45	12.00	7.56
F6	4.53 (1.96–9.64)	1.81 ± 0.39	1.66	13.72	8.23
F7	4.66 (3.05–6.42)	1.76 ± 0.35	0.09	13.57	8.47
F8	5.33 (3.67–7.53)	1.36 ± 0.20	0.49	16.15	9.69
F9	6.69 (4.54–9.24)	1.58 ± 0.26	0.41	20.27	12.16
F10	8.20 (5.40–14.35)	1.26 ± 0.26	1.00	24.84	14.90
F11	9.19 (5.19–14.08)	1.63 ± 0.32	2.62	27.84	16.70
F12	10.02 (6.15–19.08)	1.58 ± 0.42	0.11	30.36	18.21
F13	10.37 (6.30–24.14)	1.06 ± 0.25	1.77	31.42	18.85
F14	10.57 (6.95–16.24)	1.50 ± 0.30	1.16	32.03	19.21
F15	12.18 (8.65–17.11)	1.71 ± 0.31	1.54	36.98	22.14

[a] Resistance Ratio = LC_{50} of tested generation/LC_{50} of susceptible strain
[b] Resistance Ratio = LC_{50} of tested generation/LC_{50} of parent generation (F0)
CL = Confidence Limit

Table 2. Estimated realized heritability (h^2) of resistance to acetamiprid in *Aphis gossypii*

Selected generations	No. of selected generations	Estimate of mean response per generation		R	Estimate of mean selection differential per generation				S	h^2
		Log initial LC_{50}	Log final LC_{50}		P	i	mean slope	σ_p		
(F0–F7)	8	–0.259	0.668	0.115	50.0	0.798	1. 75	0.57	0.456	0.25
(F8–F15)	8	0.726	1.085	0.044	50.0	0.798	1.48	0.68	0.539	0.08
(F0–F15)	16	–0.259	1.085	0.084	50.0	0.798	1.64	0.61	0.486	0.17

n = number of generations selected
P = mortality percent
R = response to selection R = [log(final LC_{50}) – log(initial LC_{50})]/n
i = intensity of selection (Falconer 1989)
σ_p = the phenotypic standard deviation σ_p = 1/2 (initial slope + final slope)$^{-1}$
S = selection differential $S = i\sigma_p$
h^2 = realized heritability $h^2 = R/S$

Cross resistance in the acetamiprid-selected strain

After 16 generations of selection, R-strain of *A. gossypii* had low levels of cross resistance to alpha-cypermethrin (5.33 fold) and dinotefuran (8.54 fold). In relation to other neonicotinoids members, cross resistance was 10.78 and 11.67-fold for thiacloprid and imidacloprid, respectively. In contrast, pymetrozine showed non-cross resistance (Table 3).

Resistance reversion

The stability of acetamiprid resistance was investigated. The R-strain strain was reared for an extra five generations without exposure to the insecticide with a declined resistance value of – 0.23 (Table 4).

Discussion

In our study, selection of a field population of cotton aphids, *A. gossypii* for 16 generations with the neonicotinoid, acetamiprid, resulted in the development of resistance. It seems that the intensive use of neonicotinoids is the key factor in resistance development in *A. gossypii*. In addition, several aphid species showed the ability to develop resistance to various insecticides as a result to selection pressure. For instance, *A. craccivora*

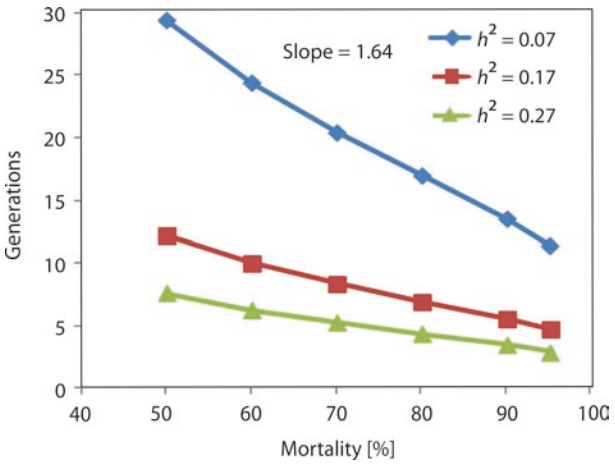

Fig. 1. Effect of realized heritability (h^2) on the number of generations of *Aphis gossypii* required for a tenfold increase in LC_{50} of acetamiprid (slope = 1.64) at different selection intensities

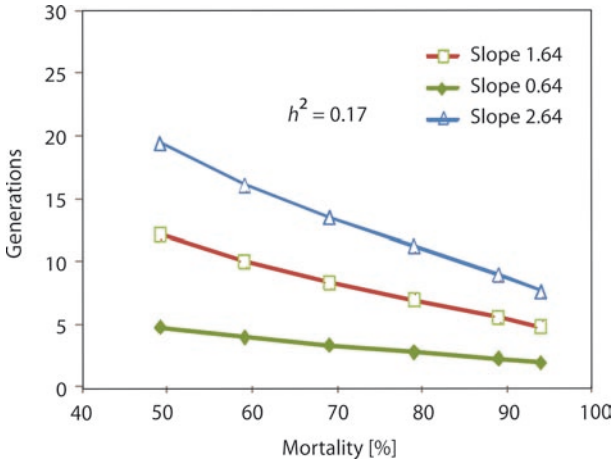

Fig. 2. Effect of slope on the number of generations of *Aphis gossypii* required for a tenfold increase in LC_{50} of acetamiprid (h^2 = 0.17) at different selection intensities

Table 3. Cross-resistance to some tested insecticides in R-strain of *Aphis gossypii*

Strain	Insecticide	LC_{50} (95% CL) [mg a.i. · ml^{-1}]	Slope ± SE	χ^2	*RR*
S -strain					
	acetamiprid	0.33 (0.20–0.44)	1.53 ± 0.44	0.161	–
	thiacloprid	0.95 (0.49–1.43)	1.45 ± 0.30	0.67	–
	dinotefuran	2.20 (1.05–3.49)	1.56 ± 0.33	0.81	–
	imidacloprid	1.07 (0.24–2.31)	0.87 ± 0.21	1.21	–
	alpha-cypermethrin	0.15 (0.059–0.34)	0.57 ± 0.14	0.12	–
	pymetrozine	35.01 (22.29–64.57)	1.08 ± 0.25	0.74	–
Aceta-SEL (F15)					
	acetamiprid	12.18 (8.65–17.11)	1.71 ± 0.31	1.54	36.98
	thiacloprid	10.25 (6.55–14.03)	2.29 ± 0.45	1.67	10.78
	dinotefuran	18.78 (9.21–30.39)	1.64 ± 0.40	0.63	8.54
	imidacloprid	12.49 (6.91–19.59)	2.14 ± 0.65	0.41	11.67
	alpha-cypermethrin	0.80 (0.48–1.31)	1.09 ± 0.25	0.21	5.33
	pymetrozine	35.73 (24.61–58.28)	1.32 ± 0.26	2.86	1.02

RR = Resistance Ratio

Table 4. Acetamiprid resistance reversion in R-strain of *Aphis gossypii*

Generation	LC_{50} (95% CL) [mg · l^{-1}]	Slope ± SE	χ^2	*RF*
S-strain	0.33 (0.20–0.44)	1.53 ± 0.44	0.161	–
Reversed strain				
F16	6.69 (4.76–10.04)	1.66 ± 0.37	1.9242	20.27
F17	5.16 (3.81–7.88)	1.61 ± 0.33	0.6247	15.6
F18	2.55 (1.80–3.83)	1.43 ± 0.27	0.4264	7.72
F19	2.23 (1.65–3.45)	1.49 ± 0.31	0.2212	6.75
F20	0.89 (0.32–1.43)	1.12 ± 0.36	0.5766	2.70

RF (Resistance Factor) = LC_{50} of R-strain/LC_{50} of susceptible strain

(Koch.) developed a high level of resistance to chlorpyrifos-methyl as a result of repeated selection pressure (Mokbel 2015). Selection pressure of pesticides resulted in increasing frequencies of resistant individuals in pest populations, and consequently, resistance evolution. Selection experiments can be used to estimate h^2 which represents an effective tool to estimate resistance development rate. The realized heritability value can be used to predict future resistance status to any pesticide in certain pests (Abbas and Shad 2015). Realized heritability is defined as the contribution of additive genetic variation in phenotypic variation. Lower h^2 reflects higher phenotypic variation and lower additive genetic variation and vice versa (Tabashnik 1992).

The lower realized heritability value (0.17) after 16 generations of selection with acetamiprid suggests that the *A. gossypii* field strain has a lower potential to develop resistance to acetamiprid. Other findings with several insect pests exhibited the same trend. Shah *et al*. (2015) found that the h^2 value in the housefly, *Musca domestica* to methoxyfenozide was 0.17. Realized heritability (h^2) of resistance to lambda-cyhalothrin, bifenthrin, methomyl, imidacloprid, and fipronil was 0.07, 0.05, 0.01, 0.08, and 0.08, respectively (Abbas and Shad 2015). Realized heritability values indicate lower additive genetic variations for acetamiprid resistance in *A. gossypii*. Response to selection was declined as the selection pressure of acetamiprid was continued, causing lower h^2 in the second round than in the first round of selection. Although laboratory trials are not similar to field circumstances, the h^2 value can theoretically predict the future status resistance (Tabashnik 1992). In the present study, the estimated realized heritability was affected mainly by the response to the selection value. The h^2 was higher in the first round than in the second round of selection. This indicates the reasonable presence of a resistance allele in the collected field population and that the continuous selection h^2 was declined.

The realized heritability of resistance to certain insecticide varies between populations and within the same population with time as a result of changes in allele frequencies and/or environmental circumstances. So, it is difficult to extrapolate laboratory selection results to the field. However, the projected rate of resistance evolution can be obtained with the equation $G = R^{-1}$. This equation enables us to predict the occurrence of resistance before its appearance in the field. Resistance evolution is directly proportional to h^2 and selection intensity while it is inversely proportional to the slope of the probit line (Tabashnik 1992).

In the current study, acetamiprid induced cross resistance to other neonicotinoids, imidacloprid (11.67 fold), thiacloprid (10.78 fold) and dinotefuran (8.54 fold). Alpha-cypermethrin showed moderate cross resistance (5.33 fold). In contrast, the R-strain showed no cross resistance to pymetrozine (1.02 fold). Cross resistance generally arises within the same group of pesticide classes. But, it is difficult to forecast cross resistance between chemicals with different modes of action (Gorman *et al.* 2010). Cross resistance in neonicotinoids resistant pests have been investigated by several authors and in several insect species. In cotton aphids, the imidacloprid resistant strain exhibited resistance to fenvalerate on cotton and cucumber. In contrast, in other investigations the imidacloprid resistant strain showed no cross resistance to the novel neonicotinoid, cycloxaprid (Wang *et al.* 2002; Cui *et al.* 2016). Similarly, the thiamethoxam-resistant strain showed increased levels of cross resistance to clothianidin and the tested pyrethroids but did not show cross resistance to imidacloprid, organophosphate, carbamate and sulfoxaflor (Wei *et al.* 2017). In the bird cherry-oat aphid, *Rhopalosiphum padi*, an imidacloprid resistant strain showed high cross resistance to acetamiprid and thiamethoxam, low cross resistance to chlorpyrifos and no cross resistance to cycloxaprid (Wang *et al.* 2017). In the brown planthopper, *Nilaparvata lugens*, an imidacloprid resistant strain showed no cross resistance to pymetrozine (Yang *et al.* 2016). The obtained results explain the opportunity to rotate the insecticides without cross resistance to reduce selection pressure. Rotating insecticides without cross resistance can delay resistance development and sustain the efficacy of new chemicals by optimizing their use (Pu *et al.* 2010).

Resistance reversion in the absence of selection pressure is of great importance in resistance management. In the present study, acetamiprid resistance was not stable and the LC_{50} value was decreased under laboratory conditions. Without selection pressure the R-strain almost reached the S-strain after about five generations. Resistance instability was noticed in several insects with various insecticides. In the cowpea aphid, *A. craccivora* (Koch), chloropyrifos-methyl resistance was unstable and almost reached the S-strain within 10 generations (Mokbel 2015). In agromized leafminers, *Liriomyza sativa*, resistance to chloropyrifos was unstable in the absence of selection (Askari-Saryazdi *et al.* 2015) and in the cotton mealybug, *Phenacoccus solenopsis*, acetamiprid resistance was unstable in the absence of selection (Afzal *et al.* 2015).

In conclusion, the present work reveals that *A. gossypii* has the potential to develop resistance to acetamiprid. Acetamiprid resistance instability provides the opportunity to restore its efficacy by stopping its use for a certain time. A cross resistance study showed that pymetrozine has no cross resistance with acetamiprid. So, it can be used in acetamiprid resistance risk management programs either through rotation to reduce the possibility of resistance development in the field or as an alternative in the case of a resistance crisis. Finally, further studies are needed to monitor resistance, investigate resistance mechanisms and explore the role of insecticide rotation in risk management.

References

Abbas N., Shad S.A. 2015. Assessment of resistance risk to lambda-cyhalothrin and cross-resistance to four other insecticides in the house fly, *Musca domestica* L. (Diptera: Muscidae). Parasitology Research 114 (7): 2629–2637. DOI: 10.1007/s00436-015-4467-2

Abbott W.S. 1925. A method of computing the effectiveness of an insecticide. Journal of Economic Entomology 18 (2): 265–267.

Afzal M.B., Shad S.A., Abbas N., Ayyaz M., Walker W.B. 2015. Cross-resistance, the stability of acetamiprid resistance and its effect on the biological parameters of cotton mealybug, *Phenacoccus solenopsis* (Homoptera: Pseudococcidae), in Pakistan. Pest Management Science 71 (1): 151–158. DOI: 10.1002/ps.3783

Askari-Saryazdi G., Hejazi M.J., Ferguson J.S., Rashidi M.R. 2015. Selection for chlorpyrifos resistance in *Liriomyza sativae* Blanchard: cross-resistance patterns, stability and biochemical mechanisms. Pesticide Biochemistry and Physiology 124: 86–92. DOI: 10.1016/j.pestbp.2015.05.002

Bass C., Denholm I., Williamson M.S., Nauen R. 2015. The global status of insect resistance to neonicotinoid insecticides. Pesticide Biochemistry and Physiology 121: 78–87. DOI: https://doi.org/10.1016/j.pestbp.2015.04.004

Bush M.R., Abdel-Aal Y.A., Saito K., Rock G.C. 1993. Azinphosmethyl resistance in the tufted apple bud moth (Lepidoptera: Tortricidae): reversion, diagnostic concentrations, associated esterases, and glutathione transferases. Journal of Economic Entomology 86 (2): 213–225. DOI: https://doi.org/10.1093/jee/86.2.213

Cui L., Qi H., Yang D., Yuan H., Rui C. 2016. Cycloxaprid: A novel cis-nitromethylene neonicotinoid insecticide to control imidacloprid-resistant cotton aphid (*Aphis gossypii*). Pesticide Biochemistry and Physiology 132: 96–101. DOI: 10.1016/j.pestbp.2016.02.005

Falconer D.S., Mackay T.F., Frankham R. 1996. Introduction to quantitative genetics (4th ed.). Trends in Genetics 12 (7): 280.

Finney D.J. 1971. Probit Analysis. 3rd ed. Cambridge University Press, New York, 333 pp. DOI: https://doi.org/10.1002/jps.2600600940

Gorman K., Slater R., Bland J.D., Clarke A., Wren J., McCaffery A., Denholm I. 2010. Cross-resistance relationships between neonicotinoids and pymetrozine in *Bemisia tabaci* (Hemiptera: Aleyrodidae). Pest Management Science 66 (11): 1186–1190. DOI: 10.1002/ps.1989.

Guo Y.C., Shanxia Z., Liuji Z., Guifen C. 1996. Studies on resistance trends of cotton aphid and cotton bollworm to fenvalerate in fields. Acta Agriculturae Universitatis Henanensis 30 (3): 284–287.

Herron G.A., Wilson L.J. 2011. Neonicotinoid resistance in *Aphis gossypii* Glover (Aphididae: Hemiptera) from Australian cotton. Australian Journal of Entomology 50 (1): 93–98. DOI: https://doi.org/10.1111/j.1440-6055.2010.00788.x

Hirata K., Kiyota R., Matsuura A., Toda S., Yamamoto A., Iwasa T. 2015. Association between the R81T mutation in the nicotinic acetylcholine receptor β1 subunit of *Aphis gossypii* and the differential resistance to acetamiprid and imidacloprid. Journal of Pesticide Science 40 (1): 25–31. DOI: https://doi.org/10.1584/jpestics.D14-092

Huseth A.S., Chappell T.M., Langdon K., Morsello S.C., Martin S., Greene J.K., Herbert A., Jacobson A.L., Reay-Jones F.P., Reed T., Reisig D.D. 2016. *Frankliniella fusca* resistance to neonicotinoid insecticides: an emerging challenge for cotton pest management in the eastern United States. Pest Management Science 72 (10): 1934–1945. DOI: https://doi.org/10.1002/ps.4232

Jutsum A.R., Heaney S.P., Perrin B.M., Wege P.J. 1998. Pesticide resistance: assessment of risk and the development and implementation of effective management strategies. Pesticide Science 54 (4): 435–446. DOI: https://doi.org/10.1002/(SICI)1096-9063(199812)54:4<435::AID-PS844>3.0.CO;2-K

Keiding J. 1986. Prediction or resistance risk assessment. p. 279–297. In: "Pesticide Resistance: Strategies and Tactics for Management". National Academy Press, Washington, D.C., USA, 452 pp.

Matsuura A., Nakamura M. 2014. Development of neonicotinoid resistance in the cotton aphid, *Aphis gossypii* (Hemiptera: Aphididae) in Japan. Applied Entomology and Zoology 49 (4): 535–540. DOI: https://doi.org/10.1007/s13355-014-0289-4

Mokbel E.M.S. 2015. Prediction of resistance and its stability of cowpea aphid, *Aphis craccivora* (Koch) to chlorpyrifos-methyl. Egyptian Scientific Journal of Pesticides 1 (4): 24–29.

Moores G.D., Gao X., Denholm I., Devonshire A.L. 1996. Characterisation of insensitive acetylcholinesterase in insecticide-resistant cotton aphids, *Aphis gossypii* glover (Homoptera: Aphididae). Pesticide Biochemistry and Physiology 56 (2): 102–110. DOI: https://doi.org/10.1006/pest.1996.0064

Ninsin K.D., Tanaka T. 2005. Synergism and stability of acetamiprid resistance in a laboratory colony of *Plutella xylostella*. Pest Management Science 61 (8): 723–727. DOI: 10.1002/ps.1043

Pu X., Yang Y., Wu S., Wu Y. 2010. Characterisation of abamectin resistance in a field-evolved multiresistant population of *Plutella xylostella*. Pest Management Science 66 (4): 371–378. DOI: 10.1002/ps.1885.

Shah R.M., Abbas N., Shad S.A. 2015. Assessment of resistance risk in *Musca domestica* L. (Diptera: Muscidae) to methoxyfenozide. Acta Tropica 149: 32–37. DOI: 10.1016/j.actatropica.2015.05.009

Tabashnik B.E. 1992. Resistance risk assessment: realized heritability of resistance to *Bacillus thuringiensis* in diamondback moth (Lepidoptera: Plutellidae), tobacco budworm (Lepidoptera: Noctuidae), and Colorado potato beetle (Coleoptera: Chrysomelidae). Journal of Economic Entomology 85 (5): 1551–1559. DOI: https://doi.org/10.1093/jee/85.5.1551

Tomizawa M., Casida J.E. 2003. Selective toxicity of neonicotinoids attributable to specificity of insect and mammalian nicotinic receptors. Annual Review of Entomology 48 (1): 339–364. DOI: 10.1146/annurev.ento.48.091801.112731

Wang K.Y., Liu T.X., Yu C.H., Jiang X.Y., Yi M.Q. 2002. Resistance of *Aphis gossypii* (Homoptera: Aphididae) to fenvalerate and imidacloprid and activities of detoxification enzymes on cotton and cucumber. Journal of Economic Entomology 95 (2): 407–413.

Wang R., Fang Y., Mu C., Qu C., Li F., Wang Z., Luo C. 2017. Baseline susceptibility and cross-resistance of cycloxaprid, a novel cis-nitromethylene neonicotinoid insecticide, in *Bemisia tabaci* MED from China. Crop Protection 110: 283–287. DOI: https://doi.org/10.1016/j.cropro.2017.02.012

Wei X., Pan Y., Xin X., Zheng C., Gao X., Xi J., Shang Q. 2017. Cross-resistance pattern and basis of resistance in a thiamethoxam-resistant strain of *Aphis gossypii* Glover. Pesticide Biochemistry and Physiology 138: 91–96. DOI: 10.1016/j.pestbp.2017.03.007.

Xu Y., Yang X., Rui C., Wang H., Chen X. 2004. Susceptilbility of *Helicoverpa armigera*, *Aphis gossypii*, and *T. turkestani* (Ugarovet Nikolski) to insecticides in Xinjiang. Acta Agriculturae Boreali-Occidentalis Sinica 13 (2): 74–78.

Yang Y., Huang L., Wang Y., Zhang Y., Fang S., Liu Z. 2016. No cross-resistance between imidacloprid and pymetrozine in the brown planthopper: status and mechanisms. Pesticide Biochemistry and Physiology 130: 79–83. DOI: 10.1016/j.pestbp.2015.11.007