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RESEARCH ARTICLE

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Bifenthrin toxicity, inheritance of resistance, cross-resistance to insecticides in *Helicoverpa armigera*

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Abstract

Aim of study: It is first report to sort out resistance development; its mode and inheritance in *Helicoverpa armigera* against bifenthrin till several generations using progeny reciprocal crosses and back crosses, combined with observing the cross resistance of bifenthrin against pyrethroid, organophosphate, pyrazole and new chemistry insecticides.

Area of study: This study was conducted at agriculture fields of University of Agriculture, Faisalabad, Pakistan.

Material and methods: Bifenthrin selected strain of *H. armigera* was reciprocally crossed to bifenthrin susceptible strains. Resulting F1 progeny was back-crossed to resistant strain. Cross resistance of bifenthrin to six insecticides (cypermethrin, triazophos, emamectin benzoate, fipronil, lambda-cyhalothrin, profenofos) was observed.

Main results: Resistance ratio was higher in bifenthrin selected strain. *h* value showed that resistance was autosomal with incomplete dominance. Polygenic mode of resistance; resistance controlled by more than one gene; was found against bifenthrin in *H. armigera.* Cross resistance of bifenthrin selected strain against different insecticides was found higher.

Research highlights: Reciprocal crosses of F1 progeny combined with LC50 exhibits that resistance can be controlled using multiple insecticides at different intervals against *H. armigera.* These results can be implicated to develop an integrated pest management strategy to control *H. armigera.*

Additional keywords: American bollworm; genetic resistance; insecticide resistance

Abbreviations used: FL (fiducial limits); IRM (integrated resistance management); MRR (resistant parent); MRS (F_1 offspring); RR (resistant allele); RS (offsprings); XF (log LC50 of reciprocal crosses); XRR (bifenthrin-sel population); XSS (susceptible population).

Authors' contributions: This manuscript, having one author, he designed and performed the experiment, analyzed the data and wrote the manuscript.

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Introduction

American bollworm, *Helicoverpa armigera,* being polyphagous pest feeds on wide range of host plants worldwide, causes significant economic losses (Xu *et al.,* 1958; King, 1994; Zalucki *et al.,* 1994). Continued use of broad-spectrum insecticides has resulted in selection pressure of pests and caused resistance development in insects. Adoption of insecticide use against *H. armigera* has steadily increased which resulted in selection pressure against insecticides. *H. armigera* resistance evolution against pyrethroids was firstly reported in Australia (Gunning *et al.,* 1984). In Turkey, *H. armigera* showed higher resistance ratio to pyrethroids (Karaağaç *et al.,* 2013). In China, Yang *et al.* (2013) observed resistance development in *H. armigera* against insecticides sprayed in Bt cotton. In Indonesia, (McCaffery *et al.* (1991) reported insecticide resistance development of *H. armigera*. *Helicoverpa* sp. was found to be resistant against pyrethroids (Pietrantonio *et al.,* 2007). *Helicoverpa* sp. tested in transgenic and conventional cotton sprayed with spinosad and thiodicarb showed least evolved resistance (Brickle *et al.,* 2001).

In Pakistan, Ahmad *et al.* (1995) reported *H. armigera* resistance to pyrethroids; Ahmad *et al.* (2006) and Khan *et al.* (2014) reported *H. armigera* resistance to deltamethrin, and alpha-cypermethrin, respectively. Resistance development in *H. armigera* against insecticides including profenophos, lambda cyhalothrin, emmamectin benzoate, chlorpyrifos, bifenthrin, deltamethrin, thiodicarb, methoxy fenozide, lufenuron under field conditions has also been

reported in Pakistan (Hussain *et al.,* 2014). In Pakistan, *H. armigera* showed resistance against carbamates (Ahmad *et al.,* 2001). Bt cotton expressing Cry1Ac was developed to control lepidopteran pests, but these pests have also developed resistance against Bt cotton in Pakistan (Alvi *et al.,* 2012). Similarly, *H. armigera* was found to show least developed resistance against new chemical insecticides, while moderate level of developed resistance against pyrethroids, and maximum resistance against organophosphate insecticides (Qayyum *et al.,* 2015). There are also reports of multiple resistances against different insecticides in Pakistan (Ahmad *et al.,* 2003).

Insects evolved resistance due to the wide-spread and prolonged use of pesticides, thus suppressing the target pests while resulting in selection of resistant population (Melander, 1914). Different strategies have been developed to counter or delay the resistance in insects (Sudo *et al.,* 2018), which include application of two insecticidal toxins in rotation to delay the resistance evolution against single toxin insecticides (Coyne, 1951). Reviewed by Ma *et al.* (2017), knowledge of genetic basis of insecticide resistance is important for observing, monitoring and managing resistance (Bouvier *et al.,* 2001; Abbas *et al.,* 2014a). In order to know the development of resistance, pattern of dominance and number of genes involved in resistance are important tools (Abbas *et al.,* 2014b). Higher insecticidal resistance either recessive or incomplete recessive was due to one or more autosomal genes (Sayyed *et al.,* 2003; 2004; Pereira *et al.,* 2008), while low resistance was because of dominant inheritance mechanism (Gould *et al.,* 1992; Tang *et al.,* 1997). Reviewed by Tabashnik (1991), single backcross technique is commonly conducted to detect the mode of inheritance of resistance which is either monogenic or polygenic in nature (Georghiou, 1969).

Material and methods

Insect collection and rearing conditions

Two strains of *H. armigera*, a bifenthrin susceptible strain and a bifenthrin resistant strain, were colonized in the laboratory. Approximately 3000 larvae were chosen for this experiment. Bifenthrin susceptible strain was collected in a field in Punjab province of Pakistan within the cotton region (Multan, Khanewal, and Vehari districts) in 2016, and was reared using standard rearing techniques for 11 generations without exposure to any insecticide before bioassays were conducted. Bifenthrin resistant strain was selected from a laboratory colony derived from field collection from Vehari district in 2016. Oral permission was taken from private landlords rather than special permit. In order to ensure resistant generations and to produce sufficient progeny for testing in bioassays, selection regime was exposing larvae to tender cotton young leaves sprayed with bifenthrin. Insects were kept in jars and were incubated at 16:8 L:D, 65% RH, 27±2°C conditions. Cotton tender leaves were refreshed each day. Insects used for experiment were exclusively reared on cotton.

Bifenthrin-unselected strain was collected from Khanewal cotton field and was kept on bifenthrin recommended dose sprayed cotton till 11 generations. The field resistant strain named field population was collected from Multan fields and was kept on cotton sprayed with recommended doses till one generation.

Insecticide formulations and recommended rate of application

Common insecticides were purchased from Pakistan including bifenthrin (Talstar, 10EC) recommended rate is 0.075 %/L; lambda-cyhalothrin (Karate, 2.5 EC) recommended rate is 50 mg/kg; profenofos (Curacron, 500EC) recommended rate is 0.197 mL/m^2; emmamectin benzoate (Proclaim, 1.9 EC) recommended rate is 0.049 mL/m^2; cypermethrin (Arrivo, 10 EC) recommended rate is 0.081 mL/m^2; triazophos (40EC) recommended rate is 0.247 L/m^2; fipronil (5SC) recommended rate is 0.123 mL/m^2.

Bifenthrin selection for H. armigera

H. armigera population was selected on bifenthrin till 11 generations (G1-G11) and was considered as bifenthrin-selected (bifenthrin-sel mentioned hereinafter) strain. For susceptible strain, concentrations ranging from 0.5 to 10 µg/m L a.i. were chosen. For bifenthrin-selected rearing, concentrations ranging from 10 to 400 µg/mL a.i. till 11 generations were prepared (Table 1). Different number of larvae from each generation were exposed to the insecticide (Table 1) depending upon their survival. For G1 to G11, 2000, 975, 1050, 950, 870, 900, 1015, 1050, 950, 1000 and 900 larvae, respectively, were used. Surviving larvae of each generation were taken for the next selection.

Bioassay

To assess the toxicities of insecticides a bioassay using third instar of *H. armigera* with seven concentrations of bifenthrin was conducted. The experiment was repeated three times. A leaf dip bioassay was performed with different doses of bifenthrin ranging 0-10 µg/mL a.i. for susceptible strains. Similarly, bifenthrin-sel strain was tested with doses ranging 0-300 µg/mL a.i.

For the cross resistance experiment, dilutions were prepared ranging 0-350 µg/mL a.i. of the insecticides cypermethrin, triazophos, emmamectin, fipronil,

Table 1. History of generations selected and their percent mortalities

Generation	Concentration (µg/mL)	No. of larvae exposed (n)	No. of larvae dead	Mortality (%)
G1	50	2000	1100	55
G2	70	975	39	4
G3	100	1050	29	2.76
G4	120	950	25	2.63
G5	150	870	21	2.41
G6	170	900	19	2.11
G7	200	1015	20	1.97
G8	220	1050	9	0.85
G9	230	950	8	0.84
G10	250	1000	8	0.8
G11	250	900	4	0.44

lambda-cyha lothrin and profenofos. Seven concentrations of each insecticide were used and each experiment was repeated three times.

Range of concentration for toxicity bioassay over generations selected for bifenthrin was 0-150 µg/mL (G1-G6), 0-300 µg/mL (G7-G10), and 0-350 µg/mL (G11). Range of concentrations for toxicity bioassay for susceptible was 0-5 µg/mL, for field population 0-150 µg/mL, and for unselected population 0-150 µg/mL. Fresh leaves were cut and dipped for 15 sec into each dilution and were air-dried. Treated leaves were kept in petri dishes, each dish having one larva. In total, 48 dishes for one replication and 3 replications for each dilution were used. Mortality data were observed after 24 hours until 7 days in toxicity, as well as cross resistance experiments.

Genetic crosses

Reviewed by Gorman *et al.* (2010) reciprocal crosses and selection are the extensive way to determine the true cross resistance (conferred by single mechanism) as compared to multiple resistances (conferred by multiple mechanisms). In order to get bifenthrin-selected population, larvae were reared on bifenthrin-treated leaves till 11 generations and susceptible generations larvae were reared on non-sprayed leaves till 11 generations. Following Tabashnik (1991), these populations were considered as homogenous resistant and susceptible. To observe the genetic basis of American boll worm, F1 progeny was result of reciprocal cross conducted between bifenthrin-selected and susceptible (bifenthrin-sel♂ × S♀) and (S♂ × bifenthrin-sel♀). Four back crosses were conducted F1♀ (S♀ × bifenthrin-sel♂) × SS♂, F1♂ (S♂ × bifenthrin-sel♀) × SS♀, SS♀ × F1♂ (S♂ × bifenthrin-sel♀), SS♂ × F1♀ (S♀ × bifenthrin-sel♂). For each genetic cross, mating of pair of male and female was allowed for 2 days, then these adults were separated. For their egg laying paper sheets were kept inside the cage. These sheets were taken out each day and were kept separately for further hatching.

Statistical analysis

Data analysis for LC_{50}, LC_{90} and LC_{95} was done by Probit analysis (Finney, 1971), with LeOra software (2003), in order to determine LC50 values, confidence intervals and their standard errors; POLO Plus was used. Resistance ratio (RR) was calculated by dividing LC_{50} of resistant by LC_{50} of susceptible. RR was considered significantly different if 95% fiducial limits (FL) did not include the value of 1, which was RR value of susceptible (Robertson & Preisler, 1992).

Inheritance pattern

LC_{50} for toxicity and reciprocal crosses was done by following formula (Stone, 1968):

$$D = (2XF – XRR - XSS) / (XRR - XSS)$$

where XF is the log LC_{50} of reciprocal crosses; XRR is the bifenthrin-sel population (G11); XSS is the susceptible population. This value can range from -1 to 1, where -1 is completely recessive, and 1 is completely dominant.

Maternal sex linkage

From reciprocal cross of bifenthrin-selected and susceptible strains, if there is significant difference between their LC_{50}, then resistance is considered as sex linked, while if LC_{50} is not significantly different then it is autosomal.

Effective dominance

Effectiveness of dominance (*h*) of resistance as well as cross resistance was calculated:

$$h = (wRS - wSS) / (wRR - wSS)$$

Table 2. Response of *H. armigera* to bifenthrin at different concentrations

Selection	LC50	Slope	X^2	df	RR
Susceptible	1.39 (1.22-1.56)	3.61±0.21	32.0	16	1
Field population	49.12 (46.39-51.85)	5.45±0.32	15.44	16	45.33
Unselected	52.54 (46.68-55.39)	5.05±0.29	18.33	16	37.79
Bifenthrin-sel (G1)	50.67 (47.92-53.39)	6.17±0.41	8.32	16	36.45
Bifenthrin-sel (G2)	57.11 (53.34-60.89)	5.48±0.31	20.85	16	41.08
Bifenthrin-sel (G3)	66.15 (61.30-71.09)	4.26±0.24	21.57	16	47.58
Bifenthrin-sel (G4)	78.69 (74.26-83.28)	4.24±0.25	11.70	16	56.61
Bifenthrin-sel (G5)	148.58 (132.79-171.72)	2.56±0.23	3.49	16	106.89
Bifenthrin-sel (G6)	165.28 (142.57-191.15)	3.80±0.23	77.66	16	118.90
Bifenthrin-sel (G7)	167.59 (144.34-144.34)	3.93±0.24	83.37	16	120.56
Bifenthrin-sel (G8)	184.29 (164.28-205.75)	5.60±0.35	82.29	16	132.58
Bifenthrin-sel (G9)	232.31 (218.71-248.72)	4.04±0.37	6.72	16	167.12
Bifenthrin-sel (G10)	269.98 (250.11- 297.50)	3.59±0.36	4.37	16	194.23
Bifenthrin-sel (G11)	326.10 (292.51-375.47)	2.69±0.24	3.52	16	234.60

where wRS is fitness of F1 progeny; wSS is fitness of susceptible parents; wRR is fitness of resistant parents; *h* can vary from 0 to 1 (completely recessive to completely dominant resistance).

Loci influencing inheritance/ monogenic or polygenic resistance test using chi square

Test for fitting the monogenic model of resistance was evaluated through assessing the corresponding chi-square (X^2) values. The observed and expected mortalities of the backcross population at different bifenthrin concentrations were evaluated with X^2 test for fitting the Mendelian single gene model of resistance (Tabsahnik, 1991; Zhao *et al.,* 2000). If the resistance is controlled by one locus with two alleles, the backcross of F_1 × RR will produce 50% RS and 50% RR offsprings. Mortality probabilities estimated at concentration x for assumed F_1 offspring (MRS) and resistant parent (MRR) genotypes were used to estimate the expected mortality (Y_x) in the backcross progeny as insecticide dose X as:

$$Y_x = 0.5\ (MRS + MRR)$$

In order to determine the number of factors involved in bifenthrin resistance, following Sokal & Rohlf (1981), chi-square fitness of good test was done for monogenic resistance using following the formula:

$$X^2 = (F\text{-}pn)^2 / pqn$$

where F is the observed mortality in F_1 population at a particular dose; n is the number exposed at a particular dose; p is the expected mortality at a given dose; qis is calculated as 1-p (Georghiou, 1969).

Results

Evolution and selection of resistance to bifenthrin in American boll worm

Bifenthrin resistant strain of American boll worm was selected for 11 generations with increased bifenthrin concentration in each generation (50-250 µg/mL), mortality ranged from 55% to 0.4% from 1st to 11th generation (Table 1). For evaluation of susceptibility, a bioassay for bifenthrin susceptible and bifenthrin-sel (G11) strains was conducted using bifenthrin. There was relationship between bifenthrin dose and mortality for the susceptible strain (as shown by the slope value). LC_{50} of bifenthrin-sel (G11) strain was 1.39 (1.22-1.56) µg/g, which was significantly higher than bifenthrin susceptible strain 326.10 (292.51-375.47) µg/g (Table 2). Compared to susceptible strain, bifenthrin-sel strain (G11) was 234.7 times more resistant at LC_{50}, ultimately supporting the hypothesis of resistance development against bifenthrin in *H. armigera*. A lower slope value of 2.67 for the bifenthrin-sel (G11) strain compared to 3.61 for the susceptible strain showed the heterogeneity of the response to bifenthrin in the population. The results showed that several selections with bifenthrin considerably increased the resistance ration (RR) 234.60 folds at LC_{50} (Table 2).

Cross resistance

LC_{50} values of cypermethrin, triazophos, emamectin, fipronil, lambda cyhalothrin, and profenofos were significantly higher in field-population of bifenthrin and in bifenthrin-sel (G11) strain as compared to susceptible

Table 3. Cross resistance of insecticides in field population and bifenthrin-sel populations of *H. armigera*

Strain	Insecticide	LC_{50}	RR	Slope	X^2	df
Susceptible	Bifenthrin	1.11 (0.99-1.23)	1	3.48±0.19	23.0	16
Field population	Cypermethrin	62.39 (58.80-66.07)	56.20	7.62±0.63	1.31	16
	Triazophos	49.14 (32.16-55.58)	44.27	7.69±0.99	103.5	16
	Emamectin	40.99 (35.88- 46.11)	36.92	4.16±0.23	40.21	16
	Fipronil	55.98 (49.90- 61.19)	50.43	6.12±0.50	38.31	16
	Lambda cyhalothrin	35.23 (33.08-37.35)	31.73	6.78±0.50	4.48	16
	Profenofos	36.72 (34.59-38.83)	33.08	7.75±0.58	2.52	16
Bifenthrin-sel (G11)	Cypermethrin	102.78 (83.44-122.35)	92.59	4.43±0.26	115.0	16
	Triazophos	86.04 (73.89-98.82)	77.51	4.59±0.29	92.17	16
	Emamectin	70.93 (59.94-81.74)	63.90	5.41±0.37	108.8	16
	Fipronil	59.67 (57.12-62.08)	53.75	9.35±0.79	11.23	16
	Lambda cyhalothrin	45.81 (42.87-48.61)	41.27	5.78±0.40	7.33	16
	Profenofos	53.49 (48.0-58.98)	48.18	5.73±16.23	47.76	16

RR: resistance ratio

strain. Selection with bifenthrin resulted in resistance ratio of 56.20, 44.27, 36.92, 50.43, 31.73, 33.08, respectively, in case of field population of bifenthrin resistant strain, while 92.59, 77.51, 63.90, 53.75, 41.27, 48.18 folds for laboratory selected bifenthrin resistant strain (Table 3), which shows that bifenthrin-sel (G11) was cross resistant to other 6 insecticides.

Maternal sex linkage

In order to determine the mode of inheritance at lethal concentrations, the susceptibility of F_1 progeny was tested for bifenthrin. Toxicity of bifenthrin (LC_{50}) for reciprocal cross from F_1 progeny was significantly higher than susceptible parent (Table 4) while significantly lower than resistant parent (Table 2) with LC_{50} values of 39.87 µg/g and 37.67 µg/g (Table 4) having overlap in FL of each other showing no significant difference. Further analysis of equality tests with equal slopes, equal intercepts and parallelism tests were not rejected. These analyses confirmed that the bioassay of reciprocal cross did not have significant difference. Backcross produced levels of resistance intermediate between those of the susceptible and resistant parents. LC_{50} value in F_1 progeny was intermediate the LC_{50} of susceptible and resistant parents (Table4) confirming that inheritance was autosomal with no maternal effects.

Table 4. Maternal sex linkage to determine either resistance evolved is related to heredity or not in *H. armigera*

Strain	LC_{50}	Slope	X^2	df
Susceptible	1.06 (0.95-1.18)	3.24±0.18	21.07	16
Bifenthrin-sel♂ × S♀	39.87 (37.47-42.08)	6.75±0.50	13.14	16
S♂ × Bifenthrin-sel♀	37.67 (35.57-39.73)	6.21±0.38	10.48	16

Resistance will be considered as significantly different if LC_{50} will not overlap on 95% fiducial limit (FL) and will be non-significantly different if LC_{50} will overlap on 95% FL.

Loci influencing inheritance/ monogenic or polygenic test using chi-square

Pooled F_1 progeny were backcrossed to resistant parents resulting in the progeny which showed more resistance than F_1 and less resistance than resistant parents to confirm that it was inherited (Table 5). Back-cross of resistant strain and F_1 progeny showed that LC_{50} value was 57.20 mg/L and RR was 40.85 (Table 5). Pattern of response was not consistent with mono-factorial model (Table 6). At lower concentration, there was higher X^2 value, while at higher concentration, X^2 value was lower, which indicates a polygenic resistance against bifenthrin (Table 6).

Effective dominance

Effective dominance was obtained to know the degree of dominance at three different concentrations of bifenthrin. *h* value varied with concentration, from dominant inheritance at higher concentration to recessive inheritance at lower concentrations (Table 7). Results showed partially recessive inheritance at 5 mg/L, *h* value was 0.83; and incomplete dominant inheritance at 50 mg/L, *h* value was 0.57; at concentration of 100 mg/L, h value was 0.27 (Table 7). It shows that higher concentration of single insecticide (bifenthrin) can cause dominant inheritance of resistance.

Discussion

H. armigera ranks among the most damaging lepidopteran pest of cotton, maize and vegetable crops (potato, tomato, peas, okra, and cabbage) in Pakistan (Talekar *et al.,* 2006; reviewed by Qayyum *et al.,* 2015). It is successful in its dispersal due to higher mobility, fecundity, and ability to develop resistance against insecticides (Wakil

Table 5. F1 progeny back-cross with resistant parents

Strain	LC_{50}	Slope	X^2	RR
Susceptible	1.40(1.28-1.51)	3.35±0.19	10.44	1
F1♀ (S♀ × Bifenthrin-sel♂) × RR♂	50.79 (37.07-64.30)	5.01±0.54	8.57	36.27
F1♂ (S♂ × Bifenthrin-sel♀) × RR♀	51.36 (39.18-64.12)	4.90±0.49	7.99	36.68
RR♀ × F1♂ (S♂ × Bifenthrin-sel♀)	57.20 (32.61-77.91)	4.91±0.56	14.85	40.85
RR♂ × F1♀ (S♀ × Bifenthrin-sel♂)	50.57 (45.14-56.13)	5.74± 0.63	3.79	36.12

RR: resistance ratio

Table 6. Test of monogenic model for inheritance of resistance to bifenthrin in bifenthrin-sel strain of *H. armigera*

Strain	Actual mortality (%)	Expected mortality (%)	χ^2
F_1♀ (S♀ × Bifenthrin-sel♂) × RR♂			
20	2.08	0.5	1
40	39.58	10.54	0.71
80	72.91	19.58	0.65
120	100	30.25	0.35
F_1♂ (S♂ × Bifenthrin-sel♀) × RR♀			
20	2.08	0.5	1
40	35.41	10.58	0.40
80	70.83	19.08	0.64
120	100	30.25	0.35
RR♀ × F1♂ (S♂ × Bifenthrin-sel♀)			
20	4.16	2.04	0.0008
40	31.25	10.62	0.18
80	58.33	17.12	0.42
120	100	29.20	0.42
RR♂ × F1♀ (S♀ × Bifenthrin-sel♂)			
20	0	1.04	26.04
40	33.33	10.08	0.38
80	81.25	22.62	0.54
120	100	29.20	0.42

Table 7. Effective dominance (*h*) of resistance to bifenthrin- sel *H. armigera*

Concentration of bifenthrin	Strain	Survival (%)	Fitness	*h*
5.0	Susceptible	8.33	0.08	
	Bifenthrin-sel	100	1	0.83
	F1	100	1	
50	Susceptible	0	0	
	Bifenthrin-sel	72.91	1	0.57
	F1	41.66	0.57	
100	Susceptible	0	0	
	Bifenthrin-sel	37.5	1	0.27
	F1	10.41	0.27	

et al., 2009a,b; 2010). Resistance development against organophosphate (Ahmad *et al.,* 1999); potentiation of organophosphates and pyrethroids (Ahmad, 2004; 2008); cross resistance to different pesticides (Ahmad *et al.,* 2003) have already been reported in Pakistan. Till date, no work has been reported on resistance development, inheritance, maternal sex linkage of resistance of *H. armigera* against bifenthrin (pyrethroid), and bifenthrin cross resistance to other pesticides.

Our data suggest that a resistant colony of *H. armigera* reared in the laboratory under long-term selection pressure with bifenthrin has evolved moderate levels of resistance and cross resistance to several insecticides. Implication of these results exhibit that the frequency of bifenthrin resistance in field-collected population is higher than anticipated. These results are in agreement with Qayyum *et al.* (2015), who found that *H. armigera* developed resistance against organophosphates, pyrethroids and new

chemistry insecticides in Pakistan. In the present study the bifenthrin-sel strain was challenged with six structurally and functionally different insecticides: synthetic pyrethroids (cypermethrin, lambda-cyhalothrin); organophosphate (profenofos, triazophos); phenyl pyrazol (fipronil); and new chemistry insecticides (emmamectin benzoate). Bifenthrin-sel strain showed cross resistance against all tested insecticides. These results are consistent with other studies in which bifenthrin showed cross resistance to DDT against western corn rootworm. Similarly, our results are in agreement with Basit *et al.* (2013), who reported that cross resistance of bifenthrin to fenpropethrin, lambda-cyhalothrin, imidacloprid, acetamaprid, and diafenthuronin against whiteflies.

In the current study, when reciprocal crosses were performed between bifenthrin-sel and susceptible colonies, F1 offsprings showed no significant differences, what suggest that *H. armigera* have autosomal inheritance of resistance showing no sex linkage with maternal effects. Our results are in agreement with Alvi *et al.* (2012), who found that *H. armigera* showed autosomal resistance with no sex linkage and maternal effects. Similarly, our results are also in agreement with Narayanamma *et al.* (2013), who found *H. armigera* showing inheritance of resistance with no maternal effects. Insecticides belonging to different groups, and insect species from the same order, show different effects of susceptibilities in selected strains. So resistance in *Heliothis virescens* against different insecticides was sex linked (Heckel *et al.,* 1998), this difference with our studies may occur due to different species, suggesting that species genetic resistance model should be used in integrated resistance management (IRM) to better understand these phenomena.

In insects, resistance can be monogenic or polygenic in nature. In back-cross, null hypothesis is to test either resistance is controlled by one locus controlled by two alleles (reviewed by Tabashnik, 1991; Wang *et al.,* 2016). In this case resistance is controlled by one locus and two alleles, then RR allele was adjusted in multiple subsequent selection generations and no increase in resistance would occur (reviewed by Wang *et al.,* 2016). Evidence of genetic resistance was provided by the reciprocal back-crosses of the bifenthrin-sel strains with resistant strains. In order to determine number of loci for resistance based on expected mortality of offsprings resulting from back-cross of RSXRR using different doses of insecticides was performed in present study. Data did not support monogenic model thus resistance was polygenic in our laboratory selected strain of American bollworms. Our findings are in agreement with Abbas *et al.* (2014a)´s work, in which continuous selection pressure with insecticides results in polygenic resistance in insects. Our studies exhibit high resistance conferred by single recessive gene. Since recessive genes are linked with resistance, heterozygous individuals can be killed in field.

In the present study incomplete dominant resistance was found at higher dose while incomplete recessive resistance was found at lower dose in *H. armigera*. The level of dominance was dependent on the dose. It can be asserted that partial dominant resistance decreases with higher concentrations, so rotation of insecticides showing less cross resistance to bifenthrin can be used against *H. armigera*. These findings can be helpful further to sort out lepidopteran pest resistance at molecular level.

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