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: offspring); RR (resistant allele); RS (offsprings); XF (log LC50 of reciprocal crosses); XRR (bifenthrin-selected 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 profenofos, lambda cyhalothrin, emmamectin benzoate, chlorpyrifos, bifenthrin, deltamethrin, thiodicarb, methoxyfenozide, 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 (Karat, 2.5 EC) recommended rate is 50 mg/kg; profenofos (Curacron, 500EC) recommended rate is 0.197 mL/m²; emamectin benzoate (Proclain, 1.9 EC) recommended rate is 0.049 mL/m²; cypermethrin (Arrivo, 10 EC) recommended rate is 0.081 mL/m²; triazophos (40EC) recommended rate is 0.247 L/m²; fipronil (5SC) recommended rate is 0.123 mL/m².

**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/mL 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, 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 was performed with 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, emamectin, fipronil,
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Lambda-cyhalothrin 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:

\[ h = (wRS - wSS) / (wRR - wSS) \]

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**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          |
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 (X²) values. The observed and expected mortalities of the backcross population at different bifenthrin concentrations were evaluated with X² 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 \( F1 \times RR \) will produce 50% RS and 50% RR offsprings. Mortality probabilities estimated at concentration \( x \) for assumed \( F1 \) offspring (MRS) and resistant parent (MRR) genotypes were used to estimate the expected mortality (Yx) in the backcross progeny as insecticide dose X as:

\[
Y_x = 0.5 \times (MRS + MRR)
\]

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:

\[
X^2 = \frac{(F-pn)^2}{pqn}
\]

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). \( \text{LC}_{50} \) 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 \( \text{LC}_{50} \), ultimately supporting the hypothesis of resistance development against bifenthrin in \( H. \) armigera.

#### Cross resistance

\( \text{LC}_{50} \) 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

| Selection          | LC50        | Slope       | X²     | 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|

### Table 2. Response of \( H. \) armigera to bifenthrin at different concentrations
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 F₁ progeny was tested for bifenthrin. Toxicity of bifenthrin (LC₅₀) for reciprocal cross from F₁ progeny was significantly higher than susceptible parent (Table 4) while significantly lower than resistant parent (Table 2) with LC₅₀ 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 consistent with mono-factorial model (Table 6). At lower concentration, there was higher X² value, while at higher concentration, X² 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, pea, 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

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

| Strain          | Insecticide  | LC₅₀         | RR      | Slope   | X²    | 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 |
| Bifenthrin-sel  | Lambda cyhalothrin | 35.23 (33.08-37.35) | 31.73   | 6.78±0.50 | 4.48  | 16 |
| (G11)           | Profenofos   | 36.72 (34.59-38.83) | 33.08   | 7.75±0.58 | 2.52  | 16 |

RR: resistance ratio

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

| Strain             | LC₅₀         | Slope   | X²    | df |
|--------------------|--------------|---------|-------|----|
| Susceptible        | 1.06 (0.95-1.18) | 3.24±0.18 | 21.07 | 16 |
| Bifenthrin-sel ②  | 39.87 (37.47-42.08) | 6.75±0.50 | 13.14 | 16 |
| S ③ × Bifenthrin-sel ② | 37.67 (35.57-39.73) | 6.21±0.38 | 10.48 | 16 |

Resistance will be considered as significantly different if LC₅₀ will not overlap on 95% fiducial limit (FL) and will be non-significantly different if LC₅₀ will overlap on 95% FL.
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

| Table 5. F1 progeny back-cross with resistant parents |
|--------------------------------------------------------|
| Strain | LC₅₀ (μg/mL) | Slope | X² | 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 (%) | X² |
|---------|---------------------|------------------------|-----|
| F₁♀ (S♀ × Bifenthrin-sel♂) × RR♂ | 20 | 2.08 | 0.5 | 1 |
| | 40 | 39.58 | 19.58 | 0.71 |
| | 80 | 72.91 | 19.58 | 0.65 |
| | 120 | 100 | 30.25 | 0.35 |
| F₁♂ (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 |
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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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