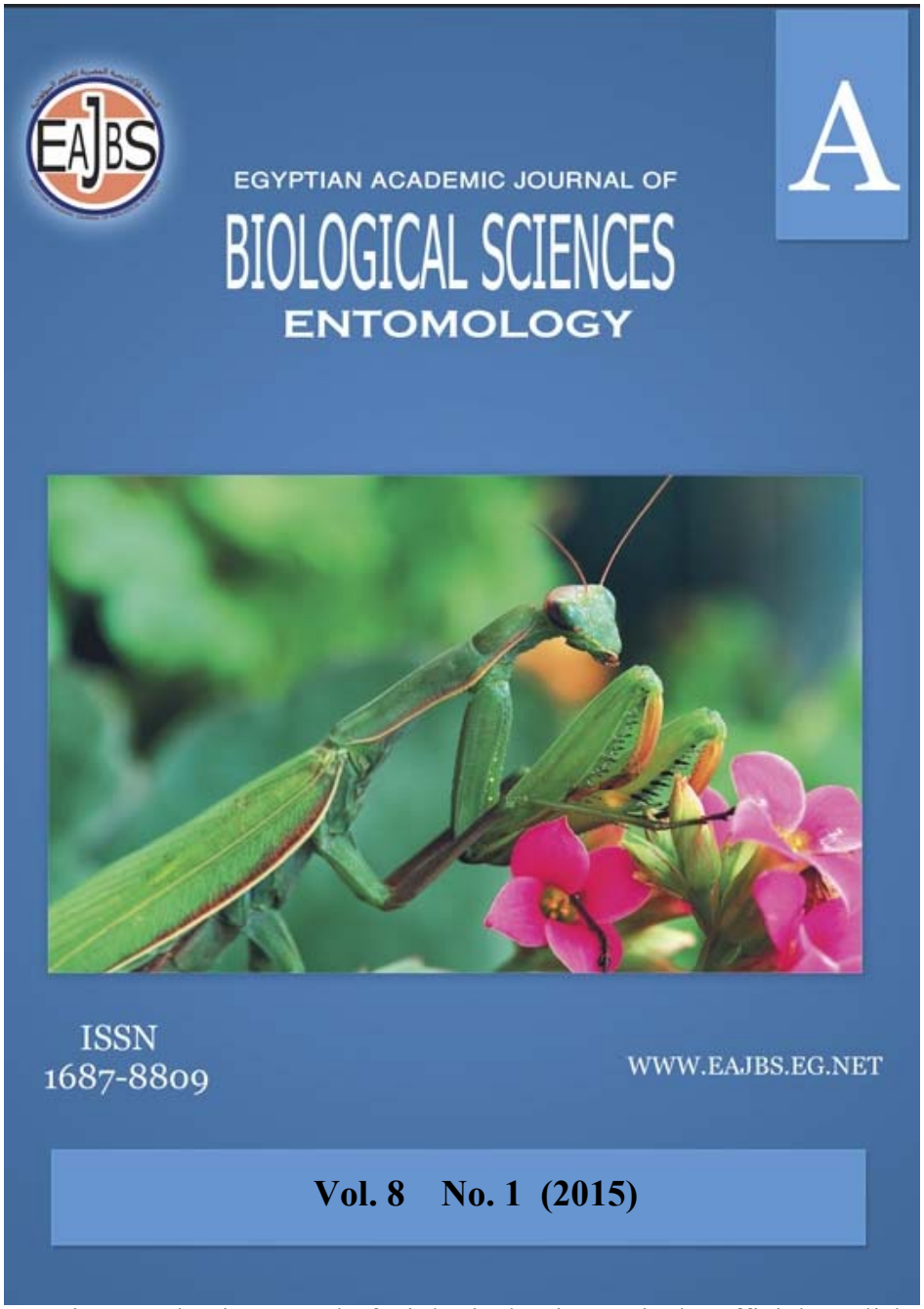


Egyptian Academic Journal of Biological Sciences is the official English language journal of the Egyptian Society for Biological Sciences, Department of Entomology, Faculty of Sciences Ain Shams University.

Entomology Journal publishes original research papers and reviews from any entomological discipline or from directly allied fields in ecology, behavioral biology, physiology, biochemistry, development, genetics, systematics, morphology, evolution, control of insects, arachnids, and general entomology.

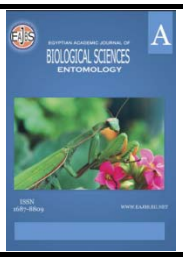
www.eajbs.eg.net

Citation: *Egypt. Acad. J. Biolog. Sci. (A. Entomology) Vol.8 (1)pp.89-98 (2015)*

Egypt. Acad. J. Biolog. Sci., 8(1): 89-98 (2015)

Egyptian Academic Journal of Biological Sciences
A. Entomology

ISSN 1687-8809
www.eajbs.eg.net



Dominant Inheritance of *Plutella xylostella* selected with Flufenoxuron

Hanan Salah-El-Din Taha Diab
Central Agricultural Pesticides Laboratory, Dokki, Giza, Egypt.

ARTICLE INFO

Article History
Received: 5/4/2015
Accepted:16/5/2015

Keywords:
Plutella
Flufenoxuron
Resistance
Dominance

ABSTRACT

Dominance study was a petition needs to measure insecticide resistance problems to a notorious pest as *Plutella xylostella* and for IRM strategy planning success. Flufenoxuron insecticide was chosen for doing a laboratory selected strain for some resistance assessments. Studies on cross resistance of flufenoxuron resistant strains revealed a slight tolerance to lambdacyhalothrin, alphacypermethrin, thiocyclam and chlorfenapyr but no cross resistance to dimethoate, imidacloprid. Resistance stablility examined in both the flufenoxuron selected strain and the backcross selected x susceptible in the absence of exposure to insecticides, results revealed that the mortality were declined slowly at F3, F5 and F7. Results of reciprocal crossing experiments suggested that flufenoxuron resistance was inherited as an incomplete recessive trait. Values of the degree of dominance were estimated -0.63 and -0.66 for F1 larvae with resistant and susceptible female parents, respectively. Results of bioassays on F1 progeny of the backcrossed with the resistant parent strain and the F2 generations suggested that resistance autosomaly inherited and might be controlled by some loci. Analysis of probit lines from F1 reciprocal crosses indicated that resistance to flufenoxuron was inherited autosomaly as an incompletely recessive and probit lines showed no plateau at 50% mortality (probit = 4.0) and 3.4 for F2 at 25-75% mortality. The x^2 analysis of response ratio statistics of a monogenic model from F1 susceptible back crosses suggested that more than one locus is responsible for resistance to flufenoxuron.

INTRODUCTION

All conventional growers observed that diamondback moth (*P. xylostella*), cabbage moth, is one of the most important pests of cruciferous crops because the validation of highly fecund and capable of migrating long distances in the Mediterranean region and worldwide. The moth has a short life cycle (14 days at 25°C) and larvae damage leaves, buds, flowers, and seed-buds foundation causing complete removal of foliar tissue except for the leaf veins. This is damaging young seedlings and may disrupt head formation in cabbage, broccoli and cauliflower. Chemical and microbial control was used to be effective but since1980s resistance developed to recently introduce synthetic pyrethroids and the other compounds in many countries (Abdel-Razek *et al.*, 2006 & Liu *et al.*, 1981).

Evolution of insecticide resistance among various populations of this species worldwide has been documented, for monitoring insecticide resistance many techniques include biochemical (Sayyed *et al.*, 2000), immunological tests (Bourguet *et al.*, 2000) and DNA probes (Keiding, 1986), those are of limited use at present. The sustainable use of chemical control depends on a good understanding of resistance development as an evolutionary process, not just on characterization of resistant individuals and their populations. The traditional approach the multiple concentration test technique, that would produce 5-95% mortality, resistance is then expressed as resistance ratio of LC_{50} or LC_{90} of the test strain in comparison with a susceptible strain (Bliss, 1935), but further investigation developed the supplementary hypothesis about how response to selection is affected by various factors, including dominance, gene flow, fitness tradeoffs, genetic constraints, major and minor genes, haplodiploidy, founder events, life-history traits and multitrophic interactions (Tabashnik *et al.,* 2004). The success of insecticide resistance strategies depends on a variety of factors including the mode on inheritance of resistance and the genetic mechanisms that enhance the ability to design and apply. The effectiveness of the selection of any genetic factor depends on the gene frequency, its dominance, and the selection pressure intensity. Thus a change in the allele frequency requires the survival of the heterozygotes until the gene frequency has risen to a high level (Rawlings *et al.*, 1981). Thus selection for dominance is sensitive to the frequency of heterozygotes. Dominance cannot progress unless special conditions lead to the presence of a high frequency of mutant alleles in the population. In this study Inheritance patterns were examined to determine the degree of dominance, and the monogenic or polygenic nature of the resistance.

MATERIALS AND METHODS

Rearing of insects:

Collection of cabbage plant infested with larvae was brought out to the laboratory from Kalubia governorate. Transported larvae kept in glass container (20x15 cm) in lab condition (20-23^{o}C with 75% RH and a 16 light: 8 dark, photoperiod). Rearing procedure described by Liu and Sun (1984) were followed till pupation. After adult emergence, around 200 adults were placed in mating chamber containing cabbage seedlings for egg laying and absorbent cotton packed with a 10% honey solution were added for moth feeding. Cabbage seedling with deposited eggs was transferred to a clean glass container. Hatched larvae were allowed to feed on fresh cabbage plants which changed when consumed. Susceptible strain (S) was developed by rearing *P. xylostella* in the laboratory for 10 generations under insecticide free conditions before the beginning of the selection experiment.

Toxicity bioassay:

The leaf dipping method reported by Tabashnik and Cushing (1987) was applied for all bioassays performed in this research. Commercial formulations products of dimethoate (cygon 40% EC), thiocyclam (evisect 50 % WP), imidacloprid (admire 20% SC), alphacypermethrin (fastac 10% EC), lambdacyhalothrin (karat 2.5 % EC), chlorfenapyr (challenger 36% SC) and flufenoxuron (cascade 10% DC), all were used for susceptibility, cross resistance bioassay and selection experiments were done by flufenoxuron only. All formulations were diluted with water to obtain a range of test concentrations from 6 to 7 for each insecticide by 0.5 fold. A piece of fresh cabbage leaves were dipped into these solutions for 15–20 s and dried by air at room temperature. Leaves treated with water alone were used as control. For each

concentration, 30 of one-day-old third-instars larvae in 3 replicate were treated and kept in three glass Petri dishes (15 cm). The mortality after treatment was assessed in 24, for fast acting insecticides, 48h for slow acting insecticides. The larvae that did not respond to stimulation with needle or whose bodies were deformed and less than half the size of those of controls were considered to be dead.

Selection Experiment:

Flufenoxuron resistant strain (R) was reared on cabbage leaves for six or seven consecutive generations. Selection regime, achieved by dipping cabbage seedlings in insecticide concentration equivalent to the LC_{70} resulted from the previous generation, allowed to dry and placed in the cages and introduced to the third instars larvae. After 2days of exposure, surviving larvae were transferred to untreated leaves and reared in the laboratory under the same previously described conditions until adult emergence. The number of larvae selected per generation ranged between 200 and 300 according to the standard method reported by Georghiou (1969). The general mean survival of larvae (after 48 h of treatment) over six generations of selection was 40%. Patches of flufenoxuron- selected strain surviving each selection was exposed to a range of concentrations using dip bioassay to determine the effect of selection on the susceptibility of the selected populations. Cross resistance bioassay between some insecticide and flufenoxuron-selected strain were performed by third instars larvae of F6.

Genetics of Resistance to Flufenoxuron:

The R and S strains were reciprocally crossed in pair-by-pair matings to produce the F1 offspring. There were four combinations in the cross pairs: (1) both female and male were the resistant (RR), (2) resistant female and susceptible (RS), (3) susceptible female and resistant male (SR), (4) both female and male were susceptible (SS). Newly emerged females and males of each strain were kept individually until use. Thirty pair's adults for each cross were set into separate glass container (20 cm x 25 cm) with food, for building a mass crosses to provide enough offspring for multiple concentration testing and calculation of LC_{50} values, (Kim *et al.*, 1991). After mating and laid eggs, hatched larvae were reared and the third-instar (1- day-old) larvae were used for the bioassay experiment as a Progeny (F1) from each cross. Ten pairs of back crosses for five to seven concentrations in the pooled F2 generation were done to determine whether resistance was inherited as a monogenic or polygenic trait.

Stability of Resistance in the absence of selection:

Around 300 larvae from each resistant strain, F6 of flufenoxuron-selected and backcrosse between resistant and susceptible were allowed to develop and established as generation 1 plus seven subsequent generations reared separately without exposure to insecticide. Susceptibility to flufenoxuron was evaluated with leaf dip bioassay at generations 3, 5 and 7. The proportion were survived from the concentrations 0.2, 2 and 20 ppm was used to calculate the change in frequency of resistant genotypes in the populations in the absence of insecticide exposure, as described by (Tabashnik *et al.*, 1994). The rate of change in the absence of insecticide exposure (R) can be calculated from the following:

$$R = (\log [\text{final proportion surviving treatment}] - \log [\text{initial proportion surviving}]) / n,$$

Where n is the number of generations not exposed to insecticide. A negative R value indicates a decline in the proportion of larvae surviving exposure to the insecticide and R values close to 0 indicate no change in the proportion of individuals surviving insecticide exposure according to (Wirth *et al.*, 2010).

Statistical analysis:

Observed mortalities were corrected using Abbott's formula (Abbott, 1925) for susceptible, flufenoxuron-resistance and reciprocal cross of hybrids, data were analyzed by probit analysis (Finney, 1971) and LeOra software (1989), Polo Pc programm. Resistance ratios were calculated by dividing the LC_{50} of test strain by LC_{50} of the susceptible strain.

Estimation of the degree of dominance of resistance (D): in F1 offspring was calculated by Falconer's formula (1964) according to Stone (1968): as follows D = (2RS- RR - SS) / (RR - SS). Where the RR, RS and SS were represent the resistant, heterozygote and susceptible populations, respectively. This will result in a value of -1 if the resistance is fully recessive; a value of 0 if there is no dominance and a value of + 1 if the resistance is fully dominant.

Test of monogenic inheritance: Chi-square test were calculated for the F1 back crossed progeny to determine whether the observed response of the third instar larvae at a full range of flufenoxuron testing concentration fit the predicted response based on the monogenic inheritance model (Georghiou and Garber (1965), Tabashnik 1991, Zhao *et al.,* 2000, and Preisler *et al.*, 1990).

Calculated chi-square values = $\sum$ (Observed values - Expected values)^2/ Expected values

Males and females of F1 progeny were backcrossed to resistant (RR) females, and to susceptible (SS) males, respectively. F1 progeny were also allowed to intercross with F2 progeny. Where expected mortality was the average of the hybrid and parental LC_{50} values. The expected concentration mortality-response curve of the backcross progeny, assuming monofactorial inheritance, was calculated as followed (Georghiou, 1969):

(1) for backcross progeny to SS or RR parents: $Xy = W_{(SR)}\ 0.50 + W_{(SS\ or\ RR)}\ 0.50$

(2) for F2 progeny: $Xy = W_{(SS)}\ 0.25 + W_{(SR)}\ 0.50 + W_{(RR)}\ 0.25$

Where X = the expected response at a given concentration y; and W = the observed response of SS, SR and RR genotypes at concentration y, obtained directly from the respective regression lines. If a single gene is responsible for resistance then plateaus will occur in the F2 regression line at about 25 or 75 % mortality, (Georghiou, 1969).

RESULTS AND DISCUSSION

The LC_{50} value for flufenoxuron generations selected was significantly greater than the LC_{50} of the susceptible strain (Table 1) reflecting that resistance increased continually. The LC_{50} of the F6 flufenoxuron strain (Table 1) was not significantly different from the LC_{50} values for all F1 reciprocal crosses (Table 2). This result agrees with (Tabashnik *et al.*, 2004) who indicated that resistance development in laboratory selections follows a similar pattern to that observed in the field. Studies on cross resistance of flufenoxuron resistant strains revealed a slight tolerance to lambdacyhalothrin, alphacypermethrin, thiocyclam and chlorfenapyr but no cross resistance to dimethoate, imidacloprid (Table 3). Liang *et al.,* 2003 found a little cross-resistance in *P. xylostella* population between abamectin and deltamethrin, beta-cypermethrin, fenvalerate and bifenthrin and no cross-resistance between abamectin and chlorfluazuron or flufenoxuron. Selection of *P. xylostella* with teflubenzuron showed no cross-resistance to chlorfluazuron of various Taiwan and Thailand populations (Perng *el al.*, 1988).

Table 1: Responses of six generation of *P. xylostella* susceptible and selected strains to flufenoxuron

Gen.	Susceptible strain			Flufenoxuron resistant strain				
	LC_{50}(95% FL)	Slope ± SE	χ^2(df)	LC_{50}(95% FL)	LC_{70}(95% FL)	Slope ± SE	χ2(df)	rr
F0	0.011 (0.011 - 0.019)	1.669±0.200	2.460(5)	0.014 (0.011 - 0.019)	0.28(0.021-0.39)	1.769±0.20	2.460(5)	--
F1	0.007 (0.005 - 0.009)	1.849±0.216	2.5(5)	0.073 (0.051 - 0.100)	0.162(0.12-0.247)	1.502±0.22	0.853(4)	10.429
F2	0.008 (0.006 - 0.010)	1.612±0.195	0.798(5)	0.310 (0.210 - 0.436)	0.753(0.53-1.22)	1.362±0.21	0.317(4)	38.750
F3	0.009 (0.007 - 0.013)	1.503±0.188	2.21(4)	0.318 (0.213 - 0.451)	0.79(0.552-1.32)	1.323±0.21	0.779(4)	35.333
F4	0.009 (0.007 - 0.012)	1.532±0.188	0.876(5)	0.487 (0.332 - 0.668)	1.08(0.789-1.62)	1.511±0.22	0.795(4)	54.111
F5	0.008 (0.006 - 0.011)	1.993±0.226	3.86(5)	0.624 (0.440 - 0.853)	1.39(1.01-2.135)	1.509±0.22	0.210(4)	78.000
F6	0.010 (0.008 - 0.013)	1.985±0.226	2.01(5)	0.716 (0.482 - 1.027)	1.83(1.255-3.19)	1.287±0.21	0.853(4)	71.600

rr = Resistance ratio = LC_{50} of the resistance strain / LC_{50} of the susceptible starain

Table 2: Responses of *P. xylostella* hybrid F1 progeny of flufenoxuron resistant strain.

Generation	LC_{50}(95% FL)	Slope ± SE	χ2(df)	rr	D
Flufenoxuron R female x Lab S male	0.063 (0.045 -0.085)	1.668±0.236	0.740(4)	9.000	-0.6331
Lab S female x Flufenoxuron R male	0.047 (0.032 -0.063)	1.713±0.291	0.436(3)	5.875	-0.6651
F1 pooled	0.034 (0.024 -0.046)	1.595±0.230	0.558(4)	3.778	-0.6911
F1 female x Lab male	0.025 (0.017 -0.033)	1.748±0.293	0.558(3)	2.778	-0.7091
Lab female x F1 male	0.013 (0.009 - 0.017)	1.732±0.251	2.136(4)	1.625	-0.7331

rr = Resistance ratio = LC_{50} of the resistance strain / LC_{50} of the susceptible starain
D = Dominance (*D* varies from 0 to 1; where 0 is completely recessive and 1 is complete dominance).

Table 3: Responses of a susceptible strain of *P. xylostella* to some insecticides and Cross-resistance between flufenoxuron resistant strain (F6) and other insecticides.

Insecticide	Susceptible strain			Flufenoxuron resistant strain			
	LC_{50}(95% FL)	Slope ± SE	χ^2(df)	LC_{50}(95% FL)	Slope ± SE	χ^2(df)	RR
Dimethoate	13.096 (9.924 -	1.843±0.241	0.199(4)	21.310(15.702 -	1.730±0.236	0.797(4)	1.627
Thiocyclam	5.457(4.023 -	1.837±0.293	0.795(3)	17.267(12.738 -	1.631±0.227	0.536(4)	3.164
imidacloprid	1.678(1.258-	1.747±0.236	1.699(4)	3.032(2.269 -	1.776±0.237	0.254(4)	1.807
Alphacypermethrin	0.542(0.403-	1.887±0.296	0.994(3)	1.761(1.335 -	1.822±0.239	0.526(4)	3.249
Lambdacyhalothrin	0.354(0.254 -	1.535±0.224	1.30(4)	1.261(0.947 -	1.871±0.250	2.857(4)	3.562
Chlorfenapyr	0.043(0.032 -	1.764±0.235	0.534(4)	0.097(0.061 -	1.263±0.229	0.583(3)	2.256

RR= Resistance ratio = LC_{50} of flufenoxuron - resistant strain / LC_{50} of Lab strain.

There was also little or no cross-resistance between teflubenzuron or chlorfluazuron and two other acylureas, hexaflumuron and flufenoxuron (Fahmy and Miyata, 1991). The cross-resistance patterns and mechanisms of resistance to acylureas may vary between populations of *P. xylostella*, some strains remaining susceptible to one group of acylureas while becoming highly resistant to another (Cheng, 1988 and Sun, 1990). Goud *et al.*, 2009, find that the effectiveness of chitin synthesis inhibitors on *P. xylostella* was a progressive increase in larval mortality with increase in dose and at fifth day after treatment and flufenoxuron and novaluron were more potent than lufenuron. This potency is strongly related to the molecular structure of IGRs which affect the activity and degradation inside the pest, also, Kao and Cheng (1999) mentioned that there was a lack of cross- resistance between IGRs, carbamates and synthetic pyrethroids.

After 6 generations flufenoxuron selection, the LC_{50} value was increased from 0.014 to 0.716 ppm, and the slope of the log dose–probit line increased from 1.769 to 1.287 (Table 1). The LC_{50}'s of the reciprocal F1 crosses between the susceptible and resistant populations did not significantly different. Thus, maternal effects on resistance were not evident. But almost the genetic basis of resistance was autosomal rather than sex-linked.

Mortality responses to flufenoxuron in susceptible and resistant strains and in F1 progenies of reciprocal crosses F1 (R × S) and (S × R) were (0.010 - 0.716 - 0.063

and 0.047) respectively (Table 2), suggesting that the S and R strains were likely to be homogeneous for susceptibility and resistance to the insecticide. Virtually there were no significant differences in the LC_{50} values between F1 (R × S) and (S × R) (Table 2).

In Table 2, the results showed negative degree of dominance, indicating that the resistance to flufenoxuron was inherited as an incompletely recessive autosomal gene. The estimated dominance (D) was -0.633 and -0.665 for F1 (R × S) and (S × R) respectively.

When the slope of the probit line were resistance development interpretation include, the observed mortality (Table 4) (log dose–probit lines) of the backcross progenies showed no plateau at 50% mortality (probit = 4.0), and the observed mortality of the F2 progenies from inbreeding F2 of (R × S) and (S × R) showed no plateaus at 25% and 75% mortality levels (probit = 4.26 and 3.87 respectively). Practically the resistance in *P. xylostella* typically matched the polygenic inheritance characteristic.

Table 4: Deviation between observed and expected mortality for a monogenic test Flufenuxuron (backcross F1 x Lab).

Conc. ppm	Larvae No.	Obs.no.	Exp.no.	χ^2 (Df=1)	P> χ^2
0.1562	40	28	27	0.04	1.00
0.0781	40	23	22	0.05	1.00
0.03905	40	20	18	0.22	0.99
0.01952	40	13	12.5	0.02	1.00
0.0097625	40	7	6	0.17	1.00
Total χ^2	-	91	85.5	0.49	0.97

Obs.= observed mortality obtained from the backcoss regression lines

Exp.= Expected mortality at concentration X = 0.5 x (% mortality of F1 at X + % mortality of Lab population at X)

P= Probability values were considered significantly different at $P < 0.05$. Significant if less than 0.05.

To test whether flufenoxuron resistance in *P. xylostella* was monogenically inherited, expected mortalities were calculated and compared with observed mortalities at proper concentrations using the chi-square test (table 4), results showed that the observed mortalities of all crosses were not significantly different from the expected mortality (χ^2 = 0.49, 5.17 and 5.72 for F1 SxR, F1 RxR and SxS respectively), where the hypothesis of monogenic mode of inheritance were not rejected. The conclusion is flufenoxuron resistance was conferred by some loci. More demonstration about the count of locus and responsibilities need further investigation. (Imai and Mori, 1999) reported that the data of two reciprocal crosses offsprings of Japanese *P. xylostella* population suggested that resistance to *B.t* was incompletely recessive and autosomally inherited and data of backcross offsprings suggested that resistance might be controlled by some loci. Liang *et al.*, (2003) stated that the testing of F1 progeny from reciprocal crosses between *P. xylostella* abamectin-resistant and susceptible strains indicated that resistance might be autosomal and incompletely recessive with a degree of dominance of -0.13. Chi-squared analyses from the response of a backcross of crossed F1 progeny and the resistant strain and F2 progeny were highly significant, suggesting that the resistance was probably controlled by more than one gene. kim *et al.*,(1991), found that there were no differences in the concentration-mortality relationship between f1 progenies of *P. xylostella* of the susceptible and fenvalerate-selected strains reciprocal crosses, indicating the absence of sex-linked inheritance. Noppun *et al.*, (1984) detected high levels of resistance to

phenthoate, prothiophos, and cyanophenphos, and moderate level to acephate, methomyl, and cartap when tested after 12 months and 5 months of laboratory rearing, respectively. Inheritance of resistance to phenthoate in *P. xylostella* was derived by more than one gene, incomplete dominant genes and no sex linkage. (Noppun *et al.,* 1987).

Resistance remained stable in both the flufenoxuron selected strain and the backcross selected x susceptible in the absence of exposure to insecticides, whereas the mortality were declined slowly from F1 to F3, 5 and 7 (Table 5). Overall, r values were slightly close to 0 and positive, indicating no decline in resistance toward them. Most searches about the stability of resistance were agree with that r values always positive and indicating no change in resistance in class Lepidoptera (Tabashnik 1994) and in strains Cq11A and Cq4AB *Culex quinquefasciatus* (Diptera) (Wirth *et al*., 2010). In most cases, instability of resistance was attributed to fitness costs linked to the resistance alleles (Tabashnik 1994) or attributed to fixation at the various loci in response to prolonged selection pressure (Strickberger 1976 & Furlong and Wright 1993).

Table 5: Stability of resistance to selected and backcrosse of resistant and susceptible strains in the absence of selection pressure

Generation	Conc. ppm	Strain Mortality		r values	
		Sel-strain	Backcross	Sel- strain	Backcross
3	0.2	7	5	0.1549	0.105
	2	18	11	0.0872	0.060
	20	28	20	0.0300	0.025
5	0.2	6	3	0.2218	0.136
	2	16	7	0.1383	0.088
	20	22	15	0.1347	0.043
7	0.2	5	2	0.3010	0.161
	2	12	6	0.2632	0.098
	20	20	13	0.1761	0.052

r value measures the rate of change in the absence of insecticide exposure.

Tang *et al.,* 1997 stated that *P. xylostella* strain resistant to *B.t.* kurstaki, the $_2$nd generation in the absence of selection, such high levels of resistance were unstable and declined significantly. If the resistance controlled by two or more genes it would develop more slowly than that determined by a single gene. The degree of dominance alleles may play a role in the expression and distribution of the resistance gene. If the resistance controlled by a dominant gene, it will make chemical control more difficult since heterozygotes are also resistant. The resistance with dominant alleles may develop faster than that inherited as a recessive trait, because the heterozygotes might have a higher chance of surviving when treated with insecticide, and then it tend to increase and spread faster in field populations (Mallet 1989), the inferior intrinsic rate of natural increase and inferior fitness of resistant population were exist, or whether the population is homogeneous or heterogeneous to resistance (Imai and Mori 1999).

REFERENCE

Abbott, W. S. (1925). A method of computing the effectiveness of an insecticide. J. Econ. Entomol. 18: 265–267.

Abdel-Razek, A.S.; Abbas, M.H.; El-Khouly, M. and Abdel-Rahman, A. (2006). Potential of Microbial Control of Diamondback Moth, *Plutella xylostella* (Linnaeus), (Lepidoptera: Plutellidae) on Two Cabbage Cultivars under Different Fertilization Treatments. J. Appl. Sci. Res., 2(11): 942-948.

Bliss, C. I. (1935). The calculation of dosage-mortality curve. Ann. Appl. Biol. 22:134–167

Bourguet, D., A. Genissel, and M. Raymond (2000). Insecticide resistance and dominance levels. J. Econ. Ent. 93: 1588-1595.

Cheng,E.Y. (1988). Problems of control of insecticide-resistant *Plutella xylostella*. Pestic. Sci. 23:177-188.

Fahmy, A. R., and Miyata, T. (1991). Development and reversion of chlorfluazuron resistance in the diamondback moth. In Second international workshop on management of diamondback moth and other crucifer pests, Abstract volume, Asian Vegetable Research and Development Center, Shanhua, Taiwan.

Falconer, D. S. (1964). Introduction to quantitative genetics. New York: The Ronald Press Co., 365 p.

Finney, B. T. (1971). Probit Analysis, 3rd Ed. London: Cambridge University Press.

Furlong, M.J. and Wright, D.J. (1993). Examination of stability of resistance and cross-resistance patterns to acylurea insect growth regulators in field populations of the diamondback moth, Plutella xylostella, from Malaysia. Pesticide Science vol.39,(4): 305-312.

Goud, C.H.R.; Rao, S.R.K. and Chiranjeevi, C.H. (2009). Relative toxicity of chitin synthesis inhibitor against the diamondback moth *Plutella xylostella* (L.) larvae. Pest management and economic zoology, 17(1) 1-7.

Georghiou, G. P. (1969). Genetics of resistance to insecticides in house flies and mosquitoes. Exp. Parasitol. 26, 224-255.

Georghiou, G.P., and Garber, M.J. (1965). Studies on the inheritance of carbamate-resistance in the housefly (*Musca domestica* L.). Bull. World Health Organ. 32:181–196.

Imai, K. and Mori, Y. (1999). Levels, inheritance and stability of resistance to *Bacillus thuringiensis* formulation in a field population of the diamondback moth, *Plutella xylostella* (Lepidoptera: Plutellidae) from Thailand. Applied Entomology and Zoology 34 (1), 23-29 (1999).

Kao, C.H. and Cheng, E.Y. (1999). Study on Cross-resistance between BPU-type IGRs and other Insecticides Including *Bacillus thuringiensis* and abamectin in the diamondback Moth, *Plutella xylostella* L. J. Agric. Res, China 48(4) 84-95.

Keiding, J. (1986). Prediction of resistance risk assessment, In Pesticide resistance: strategies and tactics for management. National Academy of Science, Washington, DC. pp. 279-297.

Kim, G.H.; Lee, J.H. and cho, K.C. (1991). Inheritance of fenvalerate resistance in the diamondback moth, *Plutella xylostell*a (Linne.) (Lepidoptera: Yponomeutidae). Korean J. Appl. Entomolo. 30(2): 106-110.

LeOra Software (1989). POLO-PC: a user's guide to probit and logit analysis. LeOra Software, Berkeley, CA.

Liang, P.; Gao, X.W. and Zheng, B.Z. (2003). Genetic basis of resistance and studies on cross-resistance in a population of diamondback moth *Plutella xylostella* (Lepidoptera: Plutellidae). Pest management Science 59 (11); 1232-1236.

Liu, M.Y. and Sun, C.N. (1984). Rearing diamondback moth (Lepidoptera: Yponomeuidae) on rape seedlings by modification of the Koshihara and Yamada methods. J. Econ. Entomol.77(6): 1608-1609.

Liu, M. Y.; Tzeng, Y.J. and Sun, C.N. (1981). Diamondback moth resistance to several synthetic pyrethroids. J. Econ. Entomol. 74:393-396.

Mallet, j. (1989). The Evolution of Insecticide Resistanc: Have the Insect Won?. TREE vol. 4, no. 11.

Noppun, V., Miyata,T. and Saito.T. (1984). Decrease in insecticide resistance in the diamondback moth, *Plutella xylostella* L. (Lepidoptera: Yponomeutidae) on release from selection pressure. Appl. Entomol. Zool. 19:531-533.

Noppun, V., Miyata,T. and Saito.T. (1987). Insensitivity of acetylcholinesterase in phenthoate resistant diamondback moth, *Plutella xylostella* (L.) (Lepidoptera: Yponomeutidae). Appl. Entomol. Zool., 22, 116-1 18.

Perng, F. S., Yao, M. C., Hung, C. F., and Sun, C. N. (1988). Teflubenzuron resistance in diamondback moth (Lepidoptera: Plutellidae). J. Econ. Entomol., 81, 1277-1282.

Preisler, H. K., M. A. Hoy, and J. L. Robertson. (1990). Statistical analysis of modes of inheritance for pesticide resistance. J. Econ. Entomol. 83: 1649-1655.

Rawlings, P.; Davidson, G.; Sakai, R. K.; Rathor, H. R.; Aslamkhan, M. and Curtis, C. F. (1981). Field measurement of the effective dominance of an insecticide resistance in anopheline mosquitos. Bull.WHO, 59 (4): 631-640.

Sayyed, A.H.; Haward, R.; Herrero, S.; Ferre´, J.; and Wright, D.J., (2000). Genetic and biochemical approach for characterisation of resistance to *Bacillus thuringiensis* toxin Cry1Ac in a field population of the diamondback moth. Appl. Environ. Microbiol. 66, 1509–1516.

Stone, B.F., (1968). A formula for determining degree of dominance in cases of mono factorial inheritance of resistance to chemicals. Bull. WHO: 38, 325–326.

Strickberger, M. W. (1976). Genetics, 2nd ed. McMillan Publishing, New York.

Sun, C. (1990). Insecticide resistance in diamondback moth. In (N.S. Talekar ed.) Diamondback Moth Management Proceedings of the Second International Workshop, AVRDC. pp. 419-426

Tabashnik, B.E., (1991). Determining the mode of inheritance of pesticide resistance with backcross experiments. J. Econ. Entomol., 84, 703–712.

Tabashnik, B.E. (1994). Evolution of resistance to *Bacillus thuringiensis*. Annu. Rev. Entomol. 39: 47-79.

Tabashnik, B.E. and Cushing, N.L. (1987). Leaf residue vs. topical bioassays for assessing insecticide resistance in the diamondback moth, *Plutella xylostella* L. FAO Plant Protection Bulletin 35, 11–14.

Tabashnik, B.E.; Gould, F. and Carrie` Re, Y. (2004). Delaying evolution of insect resistance to transgenic crops by decreasing dominance and heritability. J .Evol. Biol. 17: 904–912.

Tang, J.D.; Gilboa, S.; Roush, R.T.; and Shelton, A.M. (1997). Inheritance, stability, and lack-of-fitness costs of field-selected resistance to *Bacillus thuringiensis* in diamondback moth (Lepidoptera: Plutellidae) from florida J. Econ. Entomol. 90(3): 732-741.

Wirth, M.C.; Walton, W. E. and Federici, B.A. (2010). Inheritance patterns, dominance, stability, and allelism of insecticide resistance and cross-resistance in two colonies of *Culex quinquefasciatus* (Diptera: Culicidae) selected with cry toxins from *bacillus thuringiensis* subsp. Israelensis. J. Med. Entomol. 47(5): 814-822.

Zhao, J.Z.; Bishop, B. A.; and Grafius, E. J. (2000). Inheritance and synergism of Resistance to Imidacloprid in the colorado potato beetle (Coleoptera: Chrysomelidae). J. Econ. Entomol. 93(5): 1508-1514.

ARABIC SUMMERY

الوراثه السائده للفراشه ذات الظهر الماسي المنتخبه بمبيد الفلوفينوكسيرون

حنان صلاح الدين طه دياب
المعمل المركزي للمبيدات ، دقي، جيزه، مصر.

لقياس وتقصي مقاومه افه هامه مثل الفراشه ذات الظهر الماسي للمبيدات ولانجاح اداره مقاومه الافات للمبيدات فانه من الضروره الملحه دراسه الحاله السائده للجينات المسؤله عن مقاومه هذه الافه للمبيدات. اختير مبيد الفلوفينوكسيرون لانه من المبيدات المنظمه للنمو والمعتاد استخدامه للمكافحه ، كاساس لعمل سلاله انتخابيه معمليه لاستخلاص المعلومات اللازمه لفهم طبيعه المقاومه لهذا المبيد. اوضحت دراسه المقاومه المشتركه للمبيد المذكورعن زياده طفيفه في قيم LC_{50} للمبيدات المختبره الاتيه: lambdacyhalothrin , alphacypermethrin , thiocyclam and chlorfenapyr ، وعدم وجود مقاومه مشتركه للمبيد dimethoate, imidacloprid . اختبرت قابليه مقاومه المبيد للثبات علي السلاله المقاومه وايضا السلاله الناتجه عن خلط اناث السلاله المقاومه مع الذكور للسلاله الحساسه في غياب التعرض للمبيد، وجد ان نسب الموت انخفضت في الاجيال المختبره ٣و٥و٧ . الاختبارات الحيويه علي السلالات الناتجه من خلط الاناث والذكور من السلاله المقاومه والسلاله الحساسه بالترتيب والعكس و حساب المعادلات الحسابيه الخاصه بالسياده اسفرت عن قيم سالبه تثبت ان الجينات المسؤله غير كامله التنحي (inherited as an incomplete recessive trait) وغير خاضعه لحسابات مندل الوراثيه، وكانت القيم كذلك (0.66- and 0.63-) لاناث المقاومه ثم لاناث الحساسه. ويقال ان هذه القيم الناتجه عن هذه الاختبارات تدل علي وجود اكثر من موقع علي الجين الواحد المسؤل عن المقاومه . وعند تحليل ميل خطوط السميه للجيل الاول الناتج من التهجين كان الميل =٤ عند نقطه ٥٠% من نسب الموت وللجيل الثاني الميل =٣,٤ عند نسب موت ٢٥,٧٥% . وعند حساب مربع كاي الاحصائي اللازم للتاكد من ان نسب الموت هذه ترجع الي وجود جين واحد او عده جينات للسلاله المقاومه والسلالات الناتجه عن التهجين وجد انه لا توجد فروق معنويه دلاله علي ان هذه المقاومه مسؤل عنها جين واحد فقط.