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Resistant Risk Assessment in the Insecticide Resistant Strains of Predatory Mite, *Neoseiulus (=Amblyseius) longispinosus* (Evans)

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A B S T R A C T

In vivo experiment was conducted by utilizing *Neoseiulus longispinosus*, a promising and potential predator of the spider mite, *Tetranychus urticae*. *N. longispinosus* is subjected to the insecticides (monocrotophos and dicofol) selection pressure to ingrain resistance development. It was seen that the realized heritability (h^2) for the monocrotophos resistant population was 0.09 and 0.16 for generations F_0 to F_5 and F_{10} to F_{20} generations, respectively and realized heritability (h^2) for dicofol resistant population was 0.01 and 0.04 for the F_0 to F_5 and F_{10} to F_{20} generations, respectively. It was analysed that the h^2 value estimated at the end of six generations for monocrotophos selected population (F_0 to F_5) was 1.8 times less than that of F_{10} to F_{20} generations and it was four times less for dicofol resistant population at the end of six generations when compared to F_{10} to F_{20} generations. This indicates that a high level of resistance to monocrotophos and dicofol can only be realized after long periods of selections (10 generations) in the laboratory reared populations of phytoseiid mite, *Neoseiulus longispinosus*. The degree of dominance was estimated and it was found that the monocrotophos resistant strain of *N. longispinosus* had -0.52, -0.58, -0.96, -0.98, and -0.97 for 5th, 10th, 20th, 30th and 40th generations and the degree of dominance for dicofol resistant strain was -1 for all the generations. This indicated that the degree of dominance of insecticide resistance is completely recessive.

Introduction

The Spider mites belonging to the family Tetranychidae are the serious acarine pests on agricultural and horticultural crops. This mite has been reported infesting over 200 species of crop plants (Perry *et al*., 1998). Among horticultural crops, the spider mites are known to cause serious infestation on vegetable crops, plantation crops, flower crops and ornamental crops both in the field as well as in the protected environment. The loss caused by spider mites is related to the population level and stages of infestation. The yield reduction of 13.64 and 31.09 per cent at Bangalore and Varanasi, respectively was estimated due to the red spider mite (Anonymous. 1991). On flowers and ornamentals, mites are primarily an aesthetic concern, but they can kill plants if populations become very high on annual plants (Godfrey, 2011). In order to contain these spider mites, organochlorines,

organophosphates and carbamates were widely used. Due to heavy selection pressure of acaro-insecticides, the spider mites gained insecticide resistance and these insecticides became ineffective in containing the pest. Acaro- insecticide resistance in the spider mites is a remarkable intrinsic potential for the rapid evolution of the resistance (van Leeuwen *et al.,* 2010). Besides insecticide resistance, the application of insecticides eliminated the potential predators of the spider mites. Among the several predatory mite species, *N. longispinosus* was found widely distributed in almost all horticultural crops and they have a intimate association with the spider mite population. By considering integrated pest management (IPM) as key approach to modern plant protection (Hoy, 2011), developing an insecticide resistant predatory mites will be effective against the target pests and also compatible with the acaro-insecticides will reduce the number insecticidal application creating a safer environment to humans and other beneficial non target organisms in the field and protected environmental conditions.

The phytoseiid**,** *N. longispinosus* is a promising and potential predator of the spider mite, *T. urticae*. Since, the spider mites have developed high resistance to several insecticides biological control with *N. longispinosus* would be effective if it is tolerant / resistant to the chemicals used against the pest mites. In order to predict the rate of development of resistance in *N. longispinosus* in response to pesticide application, one has to study and understand the resistance risk assessment or realized heritability.

Materials and Methods

Two insecticides namely dicofol, an organochlorine and monocrotophos, an organo phosphorus compound were used for the insecticide resistant studies. Potters spray tower method was followed. The resistance to these two chemicals was induced in two different sets of the laboratory susceptible strain of phytoseiid mites, *N. longispinosus*. Initially, the susceptible strain was exposed to a concentration that produced 70% mortality. After 24 hour of treatment, the survivors were shifted to a fresh leaf bit containing spider mites. The concentration of insecticides was increased in an arithmetic progression after every generation and the mortality was recorded 24 hour after each treatment. Likewise, the selection of susceptible laboratory population to monocrotophos and dicofol respectively were maintained separately taking care to avoid mixing of the two populations.

Bioassay was conducted to determine the medium lethal concentrations of insecticides after five, ten and twenty generations of selections. The number killed was converted to % mortality and subjected to probit analysis to determine LC_{50} values.

Resistance risk assessment was made by calculating realized heritability (h^2) values as described by Tabashnik (1992).

h^2=R/S where R is the response and S is the selection differential (Hartl, 1988; Falconer, 1996)

$$R = \frac{\log (\text{Final } LC_{50}) - \log (\text{initial } LC_{50})}{n}$$

Where final LC_{50} is the LC_{50} of the offspring after 'n' generations of selection and initial LC50is the LC_{50} of the parental generation before 'n' generations of selection.

S= i$\boldsymbol{\sigma}$p where 'i' is the intensity of selection and '$\boldsymbol{\sigma}$p' is the phenotypic standard deviation. Intensity of selection (i) was estimated from p, which is the % of the population with values above the selection threshold using appendix of Falconer (1996), based on the properties of normal population.

The phenotypic standard deviation (σp) was estimated as the reciprocal of the mean of the estimated slopes of profit regression lines (Finney, 1971) from the parental selection before insecticidal selection (initial slope) and the offspring after 'n' generations of selection (final slope).

$\sigma p = ½ (\text{initial slope} + \text{final slope})^{-1}$

Results and Discussion

Selection of resistance to monocrtophos

The median lethal concentrations of monocrotophos after selection pressure for several generations are shown in the Table 1. The LC50 value gradually increased from 3.639 to 202.89 ppm, it was highest in the 30th generation. When the resistant ratios were worked, it was observed that the resistant strain showed 55.89 folds resistance to monocrotophos.

Selection of resistance to dicofol

The median lethal concentrations of dicofol after selection pressure for several generations are shown in the Table 2. The LC50 value gradually increased from 0.867 to 20.533 ppm. The highest LC50 value of 20.533 was recorded in the 40th generation which was equivalent to 23.59 folds resistance. From the above data it was evident that *N. longispinosus* developed resistance to monocrotophos very rapid compared to dicofol.

Realized heritability(h^2)

The realized heritability of the susceptible and the resistant strains is provided in the Table 3. The estimated h^2 for the monocrotophos resistant population was 0.09 and 0.16 for generations F_0 to F_5 and F_{10} to F_{20} generations, respectively and estimated h^2 for dicofol resistant population was 0.01 and 0.04 for the F_0 to F_5 and F_{10} to F_{20} generations, respectively. The R and S were higher in the first five generations compared with the second F_{10} to F_{20} generations in both monocrtophos and dicofol resistant strains. Therefore, the estimated h2 in both the strains was higher in the second *i.*e from F_{10} to F_{20} than in the first five generations *i.e.* From the Table 3, it was analysed that the h^2 value estimated at the end of six generations for monocrotophos selected population (F_0 to F_5) was 1.8 times less than that of $F_{10}$to F_{20} generations and it was four times less for dicofol resistant population at the end of six generations when compared to F_{10} to F_{20} generations.

Table.1 Median lethal concentrations of monocrotophos to *N. longispinosus* during different generations of selection

Generations	Chi square (DF)	Slope	LC50 (ppm)	Resistant ratios
0	0.146 (4)	$\hat{Y}$ = -0.6621+1.1803X	3.639	-
5th	1.846 (4)	$\hat{Y}$ = -0.8424+0.7956X	11.449	3.15
10th	2.656 (4)	$\hat{Y}$ = -1.2599+1.1451X	12.598	3.47
20th	5.738 (4)	$\hat{Y}$ = -1.9731+0.9817X	102.325	28.19
30th	4.406 (4)	$\hat{Y}$ = -1.8372+0.7963X	202.888	55.89
40th	6.738 (4)	$\hat{Y}$ = -1.5835+0.6949X	189.916	52.32

Table.2 Median lethal concentrations of dicofol to *N. longispinosus* during different generations of selection

Generations	Chi square (DF)	Slope	LC50 (ppm)	Resistant ratios
0	0.669 (4)	$\hat{Y}$ = 0.6671+1.0782X	0.867	-
5th	1.073 (4)	$\hat{Y}$ = -0.0314+1.3031X	1.057	1.22
10th	0.694 (4)	$\hat{Y}$ = -0.3876+1.0079X	2.424	2.796
20th	1.546 (4)	$\hat{Y}$ = -0.8114+0.7723X	11.237	12.92
30th	0.198 (4)	$\hat{Y}$ = -1.3797+1.0835X	18.769	21.57
40th	4.602 (4)	$\hat{Y}$ = -1.1889+0.9059X	20.533	23.59

Table.3 Realized heritability (h^2) of insecticide resistance in the phytoseiid mite *N. longispinosus*

No. of generations	Estimate of mean response per generation			Estimate of mean selection differential per generation						h^2
	Initial LC_{50} (log)	Final LC_{50} (log)	R	P	I	Initial slope	Final slope	σ^2	S	
Monocrotophos										
F_0 to F_5	0.56	1.06	0.08	44.87	0.90	1.18	0.8	1.01	0.91	0.09
F_{10} to F_{20}	1.10	2.00	0.08	68.5	0.52	1.14	0.98	0.94	0.49	0.16
Dicofol										
F_0 to F_5	-0.06	0.02	0.01	32.80	1.15	1.08	1.30	0.84	0.97	0.01
F_{10} to F_{20}	0.38	1.00	0.05	41.73	0.97	1.00	0.77	1.13	1.09	0.04

R = Response to selection
p = percentage survival
i = intensity of selection
σ^2 = phenotypic standard deviation
S = selection differential
h^2 = heritability

Table.4 Degree of dominance in strains of *N. longispinosus* resistant to monocrotophos and dicofol

Generations	Degree of dominance for monocrotophos resistance	Degree of dominance for dicofol resistance
5th	-0.52	-1.0
10th	-0.58	-1.0
20th	-0.96	-1.0
30th	-0.98	-1.0
40th	-0.97	-1.0

Degree of dominance

The degree of dominance of resistance is represented in the Table 4. The degree of dominance of the resistant trait in the strains of *N. longispinosus* selected for monocrotophos was -0.52, -0.58, -0.96, -0.98, and -0.97 for 5th, 10th, 20th, 30th and 40th generations and the degree of dominance for dicofol resistant strain was -1 for all the generations. These estimates of degree of dominance in this study indicated that insecticide resistance is nearly completely recessive.

In this study, the selection of *N. longispinosus* with monocrotophos and dicofol after 40 generations resulted in the development of high resistance (55.89 and 23.59 folds, respectively). Evolution of resistance occurs faster under high selection pressure when susceptible genes are replaced by the resistant genes, resulting in a number of high resistant individuals (Ijaz *et. al.*, 2016). The value of realized heritability obtained at the end of 6 generations (F_0-F_5) of monocrtophos and dicofol -selected strains is less (1.8 and 4 times) than those for F_{10}-F_{20} generations and could indicate a less level of risk in the development of resistance to monocrotophos and dicofol, initially. The high levels of resistance to monocrotophos and dicofol can only be realized after long periods of selections (10 generations) in the laboratory reared populations of phytoseiid mite, *N. longispinosus*. One of the important uses of heritability estimates is the prediction of future response (Hartl, 1988; Falconer, 1996). The purpose of resistance risk assessment is to predict the rate of development of resistance in response to insecticide application (Via, 1986; Firko and Hayes, 1991). Heritability estimate after one generation of selection is often a reliable approximation of the heritability of the trait in the parental population because laboratory environment has minimal effects (Tabashnik, 1992). As the mean response for the F_0 to F_5 generations is less in case of monocrotophos and dicofol, the risk for development of resistance is less during earlier generations. As the selection progresses, high risk in the development of resistance was achieved. Almost 40 generations were required to develop 53-fold increase in LC50 of monocrotophos and 23.59 fold increase was observed in LC_{50} of dicofol. The differences in the folds of increase might due to the fact that the dicofol being an absolute acaricide affects the growth and survival of phytoseiid mites.

The degree of dominance in this study indicated that insecticide resistance is nearly completely recessive (Table 4). This pattern results from the additive inheritance of multiple genes (Keena and Granett, 1990). The inheritance of insecticide resistance according to Raymond *et al.* (1987) was controlled by multiple genes in the early generations under continuous selection pressure while single gene in the later generations. According to Lande's (1981) formula the insecticide resistance against monocrotophos and dicofol was controlled by more than 1 gene. Roush and McKenzie (1987) noted that the insecticide resistance is caused by allelic variants at one or two loci. They reasoned that the laboratory selected populations for any insecticide is favoured by multiple gene resistance due to the usage of small size population for the experiment where as under field condition when large population of mites is constantly exposed to an insecticide, the resistance development is favoured by a single gene of rare alleles. Without suitable genetic markers, only with the use of bioassays it is highly difficult to discriminate between inheritance mediated by a single gene and multiple genes and is almost impossible with overlapping concentration-response lines (Tsukamoto, 1963).

It can be concluded that although high levels of resistance were achieved by these laboratory-selected populations, one cannot expect the same under field or polyhouse conditions. This is because the field populations are usually more heterogeneous and exhibit more complex and diverse response to insecticide pressures. Interactions among environment, population structure and selection intensity greatly affect the field response (Bloch and Wool, 1994). The

evolution of insecticide resistance under field conditions could be delayed due to immigration of susceptible populations from other crops and the alteration of insecticides. The degree of insecticide resistance has been shown to reduce down when the insecticide pressure is removed (Servín-Villegas *et al*., 2001).

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