the intensification of cross-equatorial flow and westerlies north of the equator over the Arabian sea thus providing favourable conditions for the formation of tropical disturbance. Intense frontal system as observed on 12 and 13 June over western south Indian ocean played an important role for strengthening of cross-equatorial flow and its spreading towards central and eastern Arabian sea. This provided a favourable environment for the intensification of ITCZ and formation of onset vortex over the Arabian sea which leads to an onset of the monsoon over India[10].

In the present study we have stressed the cause of intensification of the low-level jet, and its effect on the modulation of low-level air circulation over the Arabian sea during onset phase of summer monsoon 1979. It suggests that after the northward passage of cold front to the south of the Mozambique channel, strong southerlies are established in the Mozambique channel within a day or two. It increases the strength of low-level jet and westerlies over the Arabian sea. Thus, strengthening of monsoonal westerlies makes favourable environment for the formation of an onset vortex on the leading edge of the stream. Though an onset vortex is not an indispensible condition for the monsoon's onset, it does form during some of the years and gives onset over India. Thus, satellite-derived low-level winds over the western Indian ocean are useful for monitoring large scale fluctuation of the monsoon circulation over the Arabian sea.

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DEVELOPMENT OF KNOCKDOWN RESISTANCE [KDR] AGAINST FENVALERATE IN A DDT-RESISTANT STRAIN OF *ANOPHELES STEPHENSI*

K. V. S. VERMA and S. J. RAHMAN*
Desert Medicine Research Centre, Jaipur Unit, S.M.S. Medical College, Jaipur 302004, India.
* *National Institute of Communicable Diseases, 22-Sham Nath Marg, Delhi 110054, India.*

PYRETHROIDS play an important role in controlling outbreaks of mosquito-borne diseases. The pyrethroids, owing to their fumigant action, cause instant knockdown of the vectors. Synthetic pyrethroids, popularly known as 'emergency insecticides', have been reported to be more effective than natural pyrethroids[1] and can control OP- and OC-resistant strains[2, 3]. These compounds are now being recommended as alternative insecticides in vector control programmes. Keeping in view their high potentiality[2] and quick action[4] selection studies were initiated against an important urban malaria vector *Anopheles stephensi*, using fenvalerate as an experimental compound. While estimating the insecticide susceptibility status of different generations of *A. stephensi* against fenvalerate, it was observed that the test populations of subsequent generations exhibited longer knockdown time against the same concentration of the fenvalerate. This indicated the development of knockdown resistance. To confirm the above observation and generate systematic data on the development of knockdown resistance (KDR) in *A. stephensi* against fenvalerate, the following experimental study was undertaken.

Fourth instar larvae of DDT-resistant strain of *A. stephensi* were taken from NICD insectary and selected against fenvalerate (sumididin) up to 12 generations. The larvae were exposed to 0.5 ppm of fenvalerate. Beyond this concentration, the insecticide got precipitated. At this concentration, the highest mortality (85%) of parent generation (normal generation) was obtained. The insecticide pressure was applied during larval stage in each generation and the development of knockdown resistance was observed in the adults.

Three-day-old freshly fed females of each generation were exposed in 0.5% fenvalerate impregnated papers in the laboratory. Insecticide papers were impregnated using Bushvine method[5]. Knockdown was observed in the exposure tubes, supplied with the W.H.O. test-kit for the estimation of insecticide resistance. The observations were made regularly at 10 min interval till

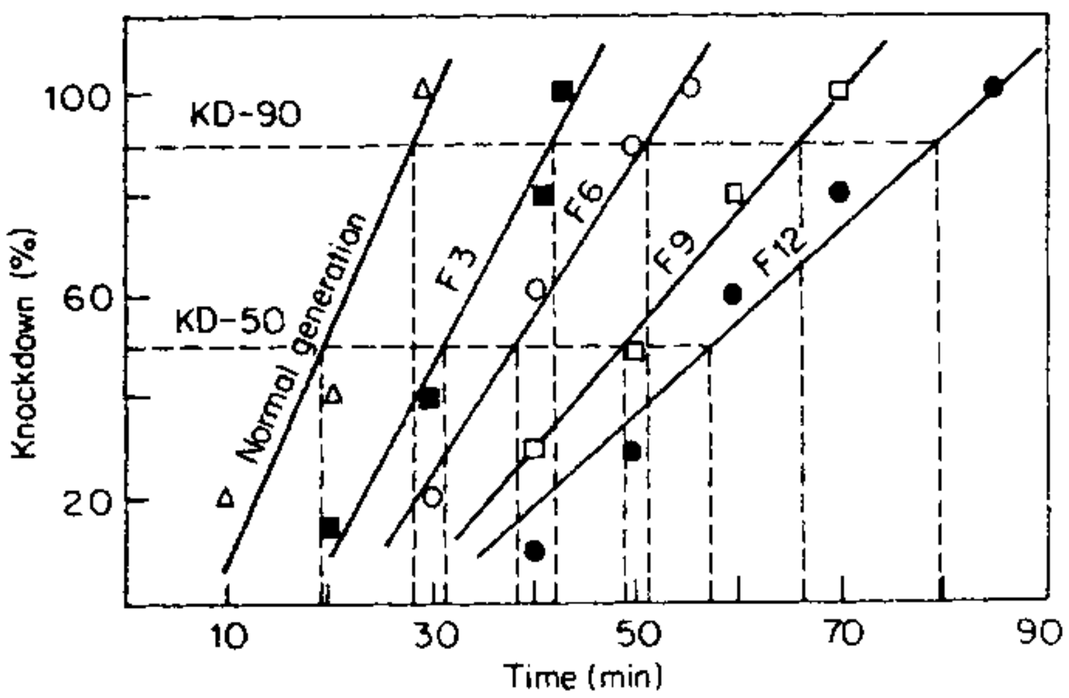

Figure 1. KD_{50} and KD_{90} values of different generations of *A. stephensi* against fenvalerate.

Table 1 *Data showing development of knockdown resistance (KDR) in Anopheles stephensi against fenvalerate*

Generations tested	No. of females exposed	Knockdown time in minutes	
		KD^{*}_{50}	KD^{**}_{90}
Normal/ parent	100	19.00	28.00
F-1	100	22.50	32.00
F-2	100	26.00	37.00
F-3	80	31.00	41.75
F-4	80	32.75	46.00
F-5	80	36.25	47.50
F-6	60	38.00	50.50
F-7	60	43.50	57.50
F-8	80	45.00	60.00
F-9	60	48.75	65.00
F-10	80	53.00	67.75
F-11	60	54.00	70.00
F-12	60	58.00	79.50

The fenvalerate concentration was 0.5%.
* KD_{50}-Time (in minutes) required to obtain 50% knockdown. ** KD_{90}-Time (in minutes) required to obtain 90% knockdown.

100% knockdown was achieved. The KD_{50} and KD_{90} values were calculated by graphical method (figure 1) and the degrees of KDR development were calculated according to Finney's method[6].

The data collected during the experiments given in table 1 show that the time taken by each generation to obtain total knockdown, in comparison to parent/normal generation, is higher, in the subsequent generations. In the parent generation, the total knockdown was observed within 30 min; however, in F-12 generation it was within 85 min using the same concentration (0.5%). Among other generations the knockdown time was recorded between the above values.

In F-1 generation, the KD_{50} and KD_{90} were 22.5 and 32 min respectively, whereas in F-12 generation the corresponding values were estimated as 58 and 79.5 min. This shows that F-12 generation took 39 and 51.5 min more time than parent/normal generation, to seek 50% and 90% knockdown respectively. This increase in knockdown time is obviously due to the development of KDR among test population.

The degree of KDR development (ratio of knockdown times of normal/parent and the subsequent generations at respective knockdown percentage) was calculated for each generation (table 2). In F-3 generation, the degrees of KDR, at KD_{50} and KD_{90} levels, were calculated as 1.63 and 1.49, respectively. The values in F-6 generation were determined as 2.0 and 1.8 respectively. The degrees of KDR in F-12 generation were 3.05 and 2.82 at KD_{50} and KD_{90} levels respectively.

The present results reveal that the development of knockdown resistance in *A. stephensi* is very quick and that too of a very high degree. In only the 12th generation, the degree of KDR was 3.03 (KD_{50}) which warrants caution in the use of synthetic pyrethroids both as larvicides and adulticides.

The quick development of fenvalerate knockdown resistance in *A. stephensi* can be attributed to the KDR

Table 2 *Data showing the degree of knockdown resistance (KDR) at KD_{50} and KD_{90} levels in different generations*

Levels of knock down	Degree* of KDR											
	F-1	F-2	F-3	F-4	F-5	F-6	F-7	F-8	F-9	F-10	F-11	F-12
KD_{50}	1.18	1.36	1.63	1.72	1.90	2.00	2.28	2.36	2.56	2.78	2.84	3.05
KD_{90}	1.14	1.32	1.49	1.64	1.69	1.80	2.05	2.14	2.32	2.41	2.50	2.82

* Ratio of knockdown time of parent and each subsequent generations at respective knockdown percentages.

factor, which is one of the factors responsible for the precipitation of resistance in most of the insect vectors. Chadwick *et al*[7] mentioned a similar factor in establishing relationship between DDT-resistance and cross-resistance to permethrin and bioresmethrin in *Aedes aegypti*. Priester and Georghiou[8] also consider the possibility of development of extremely high permethrin resistance in *Culex pipins quinquefasciatus* due to DDT-resistance factor (KDR).

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EFFECT OF VICOLIDE B ON MALE SEX ORGANS OF ALBINO RATS

T. SUSAN, MUZAFFER ALAM and K. K. PURUSHOTHAMAN

Captain Srinivasa Murti Drug Research Institute for Ayurveda, Madras 600 106, India.

VICOLIDE B was isolated[1] from the plant *Vicoa indica* (Fam: Compositae) and its antifertility activity in female rats has already been reported[2]. In the present study the effect of Vicolide B alone on male sex organs and in combination with testosterone has been reported.

Inbred male albino rats were used for the study. The animals were fed on Hindustan Lever rat feed and water was provided *ad libitum*. The drug was suspended in 0.5% carboxy methyl cellulose and administered at a constant volume of 0.1 ml/100 g. body weight.

The assay was carried out according to Ercoli *et al*[3] with following modifications. Young male albino rats weighing 60–90 g were castrated and divided into 3 groups of 8 animals each and treated as follows for 7 days from the first day of castration. The first group served as control and was fed with vehicle (0.5% carboxy methyl cellulose) alone in an appropriate quantity. The second group was administered testosterone phenyl propionate at the dose of 1 mg/kg body weight subcutaneously. The third group received testosterone phenyl propionate at the above mentioned dose and Vicolide B suspended in 0.5% carboxy methyl cellulose at the dose of 100 mg/kg body weight. On the 8th day animals were sacrificed to dissect out seminal vesicle, ventral prostate and levator ani muscle. These organs were weighed.

Effect of long time oral feeding of Vicolide B on male sex organs

Mature adult male rats weighing 100–150 g were divided into 2 groups of 8 animals each. The first group served as control and was treated with 0.5% carboxy methyl cellulose (vehicle) in appropriate quantity. The second group was fed with Vicolide B suspended in 0.5% carboxy methyl cellulose at the dose of 100 mg/kg body weight for 30 days. The animals were sacrificed on 31st day and the sex organs, testes, epididymis, vas differens, seminal vesicle and ventral prostate were dissected out. These organs were weighed (wet weight) and only the testes were fixed in 10% formal saline and processed for histopathological studies staining with haematotoxylin and eosin.

Vicolide B did not show any change in the wet weight of seminal vesicle, ventral prostate and levator ani in the testosterone administered rats (table 1). In the direct assay Vicolide B did not cause an increase in the weight of these organs suggesting that it is neither androgenic nor antiandrogenic in nature.

The long-time feeding of the compound did not cause any significant change in the weight of the male sex organs (table 2). The histopathology of the testes did not reveal any change in the organ.

Vicolide B, which is proved to be antiestrogenic preventing fertility in female rats, is devoid of antiandrogenic activity.

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