The medullary extract from the thiamine deficient chicken depressed the activity of AChE in all the three regions studied (table 1). The activity of AChE in the cerebrum was significantly reduced, while in medulla it was reduced considerably. Cerebellum did not show a significant response. The profound effect of thiamine deficiency in the CNS in a wide variety of mammalian species including man have been described. Biochemical lesions in the CNS were indicated in birds exhibiting polyneuritis[3]. In many animal species studied different patterns of regional involvement of the CNS as a consequence of thiamine deficiency were described[1]. It has been found consistently that the brain stem or the medulla suffers the brunt of injury. The deficiency state besides impairing the oxidative metabolism[4] and the efficient utilisation of glucose[8] and pyruvate[10] might produce some toxic substance as a consequence of several metabolic alterations. These substances could be inhibitory on several neuronal or neurobumoral mechanisms. Such an inhibitory factor was indicated in the blood of thiamine deficient chicken[6]. The present results also indicate the presence of the toxic factor/factors in the medulla capable of bringing about a depression in the activity of AChE (table 1). The results also point out a definite pattern of regional involvement in the CNS. Cerebral AChE seems to be more susceptible to the medullary inhibiting factor. The cerebellar AChE is the least susceptible (table 1). This could be due to the differences in the grey matter contents of the regions studied.

Table 1 *Effect of the medullary extract of the Thiamine Deficient chicken on the activity level of AChE in the different regions of the brain of normal chicken*

	Cerebrum	Cerebellum	Medulla oblongata
Controls	9.61	8.2	14.38
	+	+	+
	S D 0.35	0.42	1.62
Tests (Homogenates incubated with the medullary extract)	6.2	7.78	10.98
	(±)	(±)	(±)
	S D 0.78	0.38	0.61
Percent changes	−35.2	−5.2	−23.64
P	*P* > .01	NS	*P* > .05

Values as mean ± S D of 5 observations. Values are expressed as micromoles of ACh hydrolysed/mg tissue/minute. NS; Not Significant.

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1. Philip, R. D., Arthur, L. P. and Ralph, D. F., *Nutrition and the developing nervous system.* The C V Moshy Company, 1975, 422.
2. Victor, H., Adams, R. D. and Collins, G. H., *Contemporary Neurology series*, Ed. by Plum and Mec Dowell, F. H. Philadelphia, Davis Co., 1971, No. 7.
3. Peters, R. A., *Biochemical lesions and lethal synthesis*, Pergamon Press, Ed. International series of Monographs on pure and applied Biology, 1963.
4. Nalini, U. P. and Nayeemunnisa, *Experientia*, 1976, **32,** 198.
5. Nayeemunnisa, *Curr. Sci.*, 1976, **45,** 582.
6. Walshe, F. M. R., *Med. Sci. Abstr.*, 1920, **2,** 411.
7. Nayeemunnisa and Abdul Azeem, *Curr. Sci.*, 1976, **45,** 693.
8. Hestrin, S., *J. Biol. Chem.*, 1949, **180,** 249.
9. Gaitonde, M. K., Nixey, R. W. K. and Sharman, I. M., *J. Neurochem.*, 1974, **22,** 53.
10. Murdock, D. S. and Gubler, C. J., *J. Nutr. Sci. Vitaminol*, 1973, **19,** 237.
11. Dreyfus, P. M. and Victor, M., *Am. J. Clin. Nutr.*, 1961, 9, 14.

LABORATORY EVALUATION OF LEAF EXTRACT OF A NEW PLANT TO SUPPRESS THE POPULATION OF MALARIA VECTOR *ANOPHELES STEPHENSI* LISTON (DIPTERA: CULICIDAE).

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No report has yet appeared pertaining to the efficacy of leaf extract of *Ipomoea carnea* Jacq *fistulosa* Mart ex Choisy for pest population suppression. Since in Rajasthan this widely grown shrub is planted as a hedge to protect the plants from animals it was selected for toxicity to mosquitoes.

The mosquito *Anopheles stephensi* was reared in the laboratory according to the procedure suggested by Ansari *et al*[1]. The leaves of *I. carnea fistulosa* were collected from the fields around Jaipur and dried. Forty mesh powder (90 g) was extracted in a soxhlet

Table 1 *Effect of the leaf extract of Ipomoea carnea fistulosa on Anopheles stephensi.*

Concentration of extract (ppm)*	Average larval period ±.S E. (Days)	Percent larval mortality	Average pupal period ±.S.E.	Percent pupal mortality	Percent adult emergence (a)	Average developmental period ±.S.E. (Days) (b)	Growth index (a/b)
200	18.93 + 0 03 $P < 0.05$	68	6.36 ± 0.10 $P < 0.005$	8	24	25.29 ± 10.04 $P < 0.005$	0 94
300	19.24 ± 0.03 $P < 0.005$	75	8.08 ± 0.08 $P < 0.005$	15	10	27.31 ± 0.11 $P < 0.005$	0.54
Control	14.44 ± 0.01	0	3.58 ± 0.01	0	100	18 02 ± 0.007	5.54
Untreated	14 44 ± 0.01	0	3.58 ± 0.01	0	100	18.02 ± 0.007	5.54

* 100 second instar larvae were treated at each concentration.

apparatus using acetone as the solvent. After complete evaporation of the solvent, the residue of the extract represented by 6% of the total dry weight of the material was redissolved in acetone to prepare 10% stock solution. Different test concentrations ranging from 200 to 300 ppm were prepared by adding desired volumes of stock solution, to distilled water followed by vigorous stirring. Tween-40 was used as an emulsifier at the concentration of 0.02% in the final test solution. One hundred second instar larvae (20/beaker) were released in each 250 ml beaker containing 100 ml test formulation. Separate controls using acetone and emulsifier and untreated sets of plain water were also run simultaneously for comparison. The powdered yeast was supplied (125 mg yeast/beaker) as food throughout the larval period. The effects of the extract on development, moulting and metamorphosis of larvae and pupae of *A. stephensi* were observed.

The observations (table 1) demonstrate a significant difference ($P < 0.005$) in average larval-, pupal-, and developmental periods of treated and control mosquitoes. The growth index of the treated mosquitoes is also shorter than the control and the untreated sets. Further, mortality in larvae and pupae results in 76% and 90% fall in population.

The imbalance between growth stimulating and growth-inhibiting hormones caused by the leaf extract may prolong the larval, pupal and developmental periods[2]. Prolongation of larval and pupal periods may be due to the inhibition of moulting process caused by an increase titre in JH in the insect body[3]. Larval and pupal mortality during respective stages and moulting could either be due to the presence of toxic ingredients in the extract or the imbalance between growth stimulating and growth inhibiting hormones. It needs further probe to establish the cause. Death during moulting in larval stage, emergence of pupae and adults within the exuviae may be the consequence of insufficient availability of chitin during development, especially during metamorphosis. If the chitin is insufficient, the larvae, pupae and adults get entangled in the weak integument in an effort to increase the haemolymph pressure for casting off the exuviae and die.

The extract has the potentiality to cause mortality and disrupt the development and growth of *Anopheles stephensi*.

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1. Ansari, M. A., Sharma, V. P. and Razdan, R. K., *J. Com. Dis.*, 1978, **10**, 131.
2. Novak, V. J. A., *Insect hormones*, Methun and Company, 1966, p. 478.
3. Hargartner, W. and Masner, P., *Experientia*, 1973, **29**, 1358.