Figure 1. Coconut halves showing rot symptom due to infection by *D. hawaiiensis*.

spores which imparted violet-black colouration to infected regions. None of the controls showed any infection.

Eleven media (Ashour's medium, Richard's medium, pectin asparagine, peptone ammonium nitrate, glucose asparagine, PDA, Czapek's medium, oat meal, pectin peptone, glucose potassium nitrate and host extract) were used for *in vitro* growth of the fungus. Coconut kernel (200 g) was crushed, boiled in water for 1 hr and filtered through a muslin cloth. The final volume was made up to 1 litre. After adding the requisite amount of agar this liquid was used as host extract. Of these only host extract, PDA, oat meal and pectin peptone sustained good growth and the fungus covered the entire plate within three days. Of the seven fungicides (carbendazim, zineb, mancozeb, captafol, carboxin, ziram and sulfex tested) carbendazim completely checked the growth of the fungus *in vitro* at and above 1000 ppm concentrations at 28 ±2°C. Ziram also suppressed about 80% fungal growth at 2000 ppm. But the effective doses are very high to create residual problems and render the treatment a costly affair.

The disease incidence was low (about 3%). Since the pathogen was not present in the aerospora of this locality, it appears to have entered the fruit while still on plants either through eyes when soft or through injuries inflicted by insects, birds etc. Cracks developed during transportation might be the other avenue for infection.

The authors are indebted to Dr K. G. Mukerji, University of Delhi, for confirming the identification of the isolate and to CSIR, New Delhi, for financial assistance.

28 June 1984; Revised 21 September 1984

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EFFECT OF MEDULLARY EXTRACT OF THE THIAMINE DEFICIENT CHICKEN ON THE ACTIVITY LEVEL OF ACETYLCHOLINE ESTERASE IN THE BROAD COMPARTMENTS OF THE BRAIN OF NORMAL CHICKEN

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IT is known that birds develop poly-neuropathy in the absence of dietary thiamine[1–3]. In the CNS, the brain stem suffers the brunt of the injury[4]. Earlier studies have indicated several changes in the different compartments of the thiamine deficient brain[5,6]. These have been relatively significant in the medulla. The medullary nervous symptoms during thiamine deficiency have been reported to be due to toxic product accumulations[3,7].

The blood of thiamine deficient chicken has been demonstrated to depress the heart beat of normal chicken suggesting the presence of an inhibiting factor produced as a consequence of metabolic alterations[7]. The present study was therefore undertaken hoping that the medulla oblongata of athiaminotic bird produces some toxic factor which is responsible for producing biochemical lesions that accompany thiamine deficiency state.

Two-day old, white leg horn chicken, *Gallus domesticus* were reared in the laboratory in electrically heated cages. The controls were fed on standard chicken feed (Mysore Feeds, Bangalore, India). The experimental birds were fed on double-polished rice for 25–30 days as described by Peters[3] to induce thiamine deficiency. Water was made available *ad libitum*. The experimental birds developed polyneuritis by the 3rd week. The birds were decapitated at the acute phase of polyneuritis, along with the controls. Ten percent homogenates of the cerebrum, cerebellum and medulla oblongata of controls were prepared with phosphate buffer, pH 7. One ml of these homogenates was incubated with 1 mg of medullary tissue (in 0.1 ml of phosphate buffer) from thiamine deficient chicken for 30 min at room temperature (20°–25°). After the incubation period, the samples were analysed for AChE activity by Hestrin's method[8].

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.

The authors thank the CSIR, for financial assistance.

31 March 1984 Revised 22 October 1984

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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