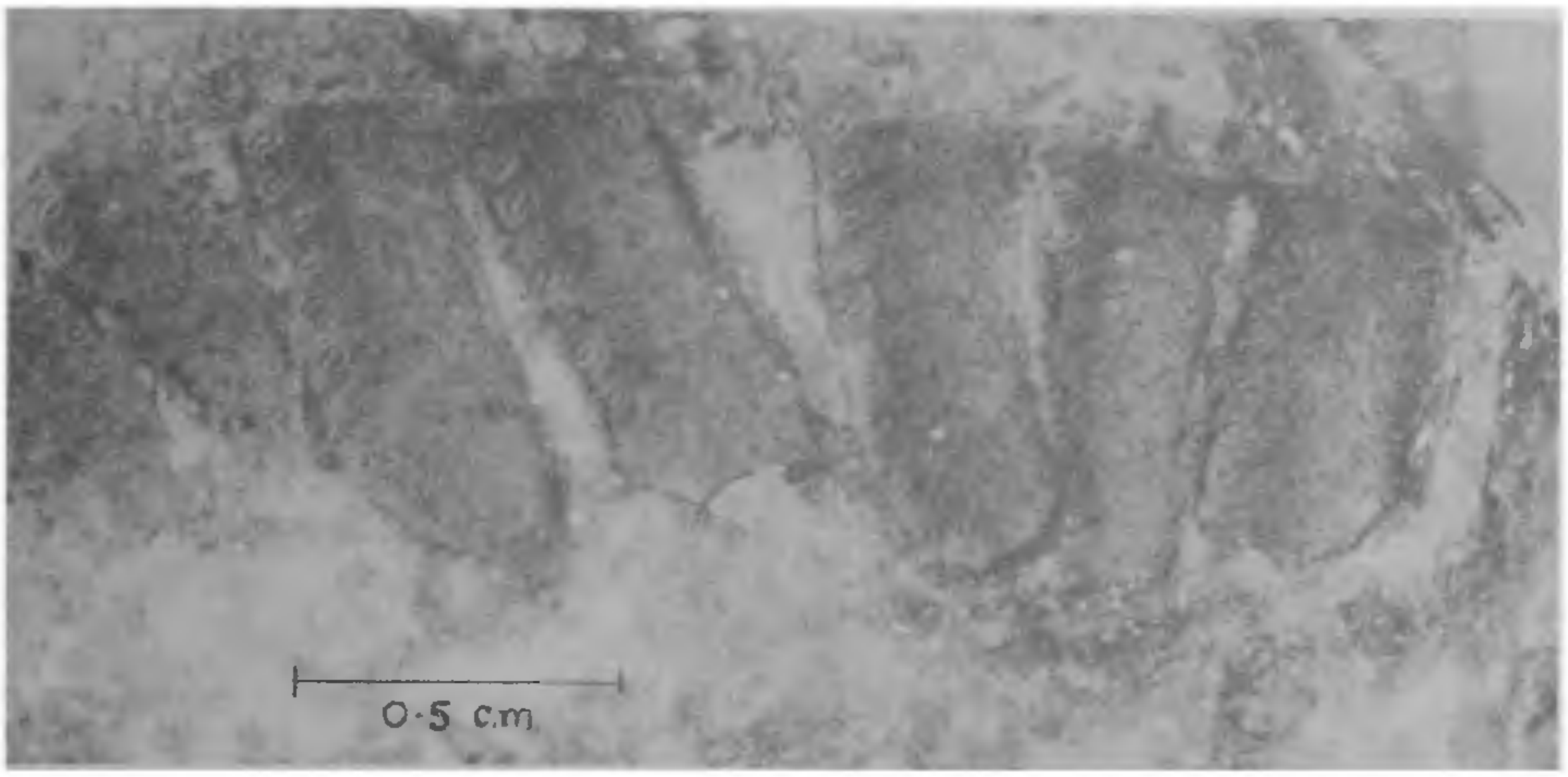

Figure 2. Specimen showing fertile pinnules on one side of the rachis (SGA/EKP/3)

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A NEW DISEASE OF COCONUT KERNEL

R. B. SHARMA*, A. N. ROY and M. P. SHARMA

Plant Disease Laboratory, Botany Department, Agra College, Agra 282 002, India.
**Department of Botany, S. D. College, Hathras 204 101, India*

FOLLOWING a survey of local fruit market during June-July, 1981 (temperature 26.5–38.6°C and RH 40–94.2%) in Agra, a soft rot disease was noticed on coconuts (*Cocos nucifera* Linn.). Fresh disease spots were irregular, water-soaked and reddish-brown or violet whereas the mature ones turned black. Profuse sporulation rendered the spots violet in colour. Following the usual mycological techniques, the causal organism was isolated, purified (single spore culture) and identified as *Drechslera hawaiiensis* (Bungicort) Subram and Jain.

The disease was reproduced on healthy fruits by inoculating the spore suspension (360 spores/ml) through cork borer[1] and pin pricks at natural depressions (eyes). Fifteen fruits were taken for each experiment and five for control. The treated and control fruits were incubated at 28 ±2°C for 15 days to record the symptoms.

The typical rot symptoms that included maceration, tissue discoloration and emission of the foul smell, appeared 12 days after inoculation (figure 1). Later the entire diseased portion was covered by mycelia bearing

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.

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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

U. P. NALINI and NAYEEMUNNISA
Department of Zoology, Bangalore University, Bangalore 560 056, India.

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].