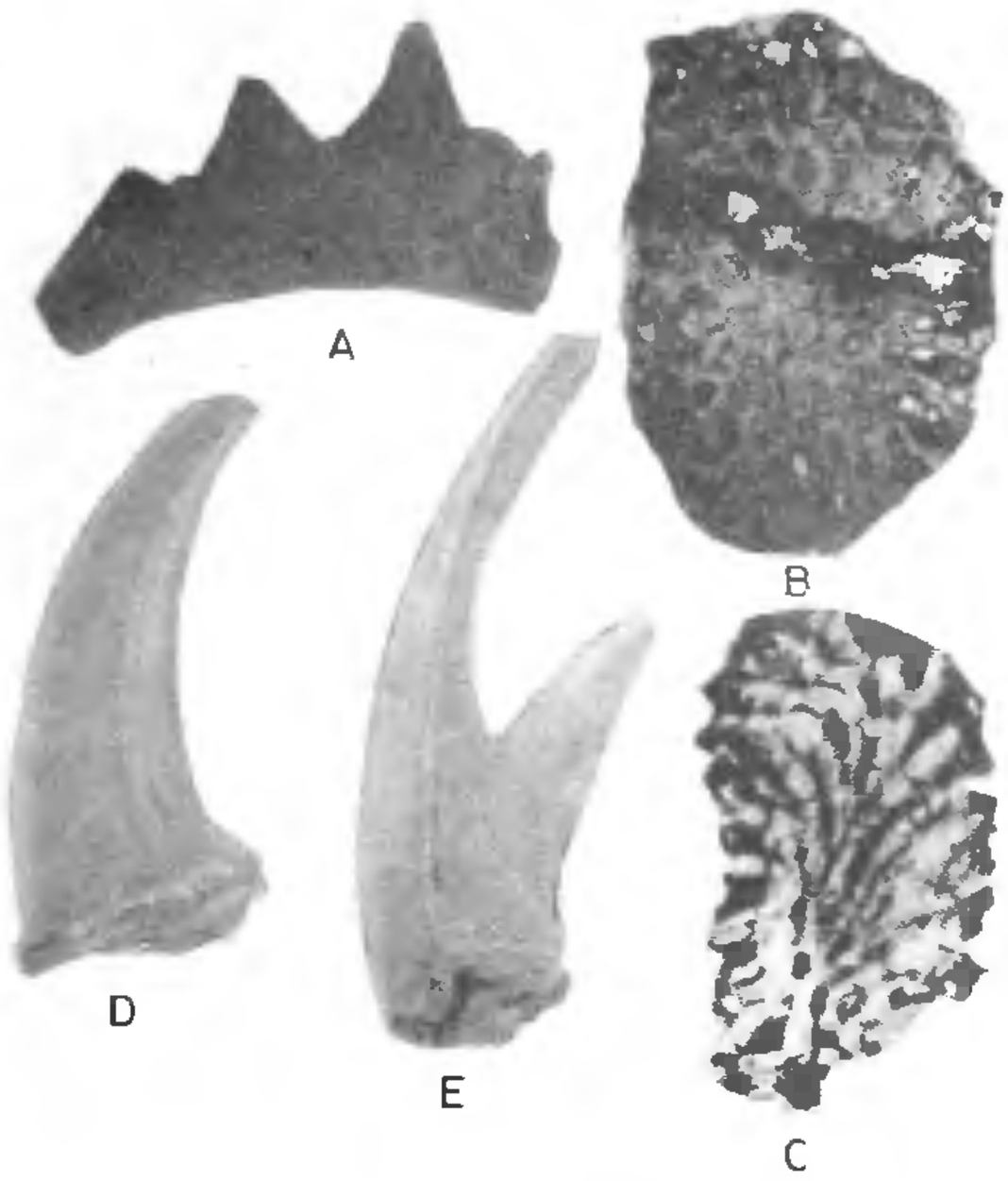

Figure 2. A—*Protacrodus* (×50). Locality-Gethia. B—*Strotopora* Tangential section (×40). C—*Strotopora* Longitudinal section (×40). Locality-Bhawali. D—*Paltodus* (×50). Locality-Gethia. E—*Strachanognathus* (×250). Locality-Bhawali.

curved posteriorly and has acute apex. The base is hollow and wide.

The stratigraphic age of the genus *Protacrodus* is late Devonian[11]. *Strotopora* ranges from Devonian to Mississipian[12] and *Paltodus* from Ordovician to Silurian[13,14]. Although the bryozoans constitute the most abundant fossils in the Gethia member, the state of their preservation is far from good due to diagenetic changes suffered by the limestone. On the basis of general size and apparent habit, they seem to belong mostly to the Palaeozoic Fistuliporoidae. Thus the microfaunal assemblage, on the whole, indicates a Middle Palaeozoic age—somewhere between Ordovician age to Devonian.

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VECTOR-BORNE MLOs OF BRINJAL LITTLE LEAF

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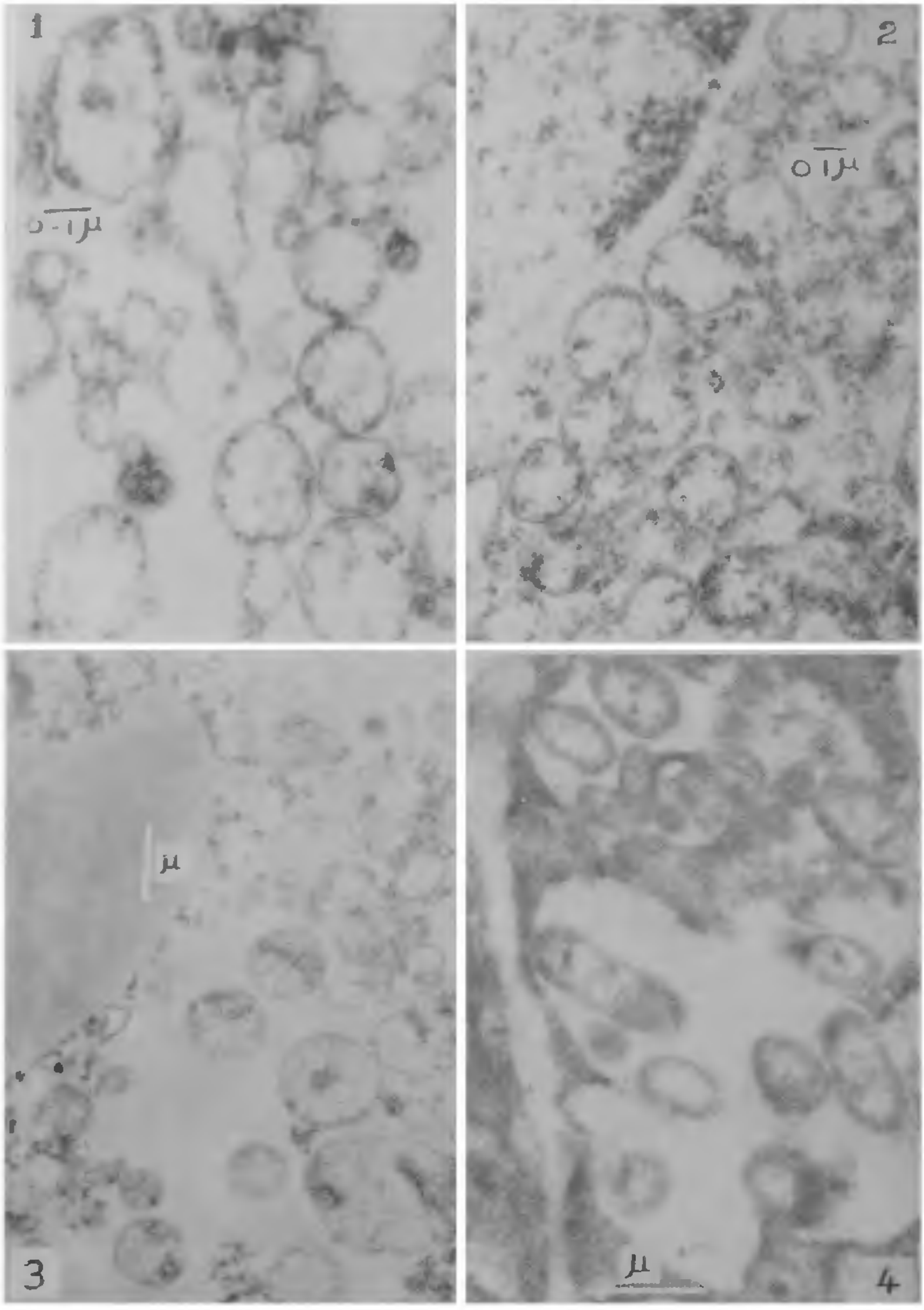
LITTLE leaf disease in brinjal comes under the Yellows group. This disease was first reported from Coimbatore by Thomas and Krishnaswami[1]. It has been proved to be MLO disease by electron micrographic evidence[2] and chemo[3] and thermo[4] therapic tests. Characteristic symptoms of this disease are reduction in leaf size, stunting of plant growth, phyllody and viruscence of floral parts. The growth of the fruits after



Figures 1–4. **1.** Sparcely arranged MLOs of different sizes and **2.** Densely packed MLOs of uniform sizes. **3.** MLOs of spherical and ovoid forms from hemolymph and **4.** MLOs of different shapes from fat body, containing RNA-like granules towards the periphery and DNA like strands in the centre.

infection is restricted or arrested ultimately failing to mature. The well-known little leaf vector is a jassid, *Hishimonus phycitis* Dist. Though transmission was proved through insect vector in the laboratory[5,6], MLOs presence in the insect host was not yet shown through electron micrographs. Externally, the infection causes noticeable damage to the plant hosts. It is interesting that the disease agents do not cause any external change in the insects. Rather the extent of damage caused to the insect vector is not visible. At the same time the vector lives long and multiplies fast on infected plants. This paper elucidates the development of etiologic agent in its insect vector.

Healthy and infected brinjal and the insects from healthy and infected colonies were fixed for electron microscopic study to understand the nature of the etiologic agent of the little leaf and its occurrence in different parts of plants and insect vectors. The materials were fixed in 3% glutaraldehyde, post fixed in 2% osmium tetraoxide and embedded in araldite. Ultra thin sections of 60–80 nm thick were cut with glassknives on a Sorvall ultra-microtome. The sections were mounted on grids with formvar supporting film and double-stained with 2% uranyl acetate and lead citrate. Electron micrographs were taken in a Hitachi (H-500) transmission electron microscope.

Spherical, ovoid, irregular and elongated pleomorphic MLOs were found intracellularly in the phloem sieve tube cells of the infected stems, phyllodes, petiole and roots. Various sizes may denote the active stages of the developing MLOs (figure 1). But in some places they were of uniform sizes (figure 2). In the viruliferous insects MLOs were found only in hemolymph (figure 3), and fat bodies (figure 4) found in the hemocoelic cavity nearer the filter chamber of the intestine. MLOs from both plant and insect hosts exhibited low electron opacity with less dense matrices, well-defined RNA like granules and DNA like strands (figure 4) inside the unit membranes. No evidence was obtained for the occurrence of MOLs in other parts of the infected insects although several sections were examined. In India this is the first report which has shown MOLs inside insect vector.

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REDISTRIBUTION OF POTASSIUM WITHIN EPIDERMIS OF *COMMELINA BENGHALENSIS* LINN. DURING FUSICOCCIN STIMULATED STOMATAL OPENING

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THE changes in guard cell turgor drive the stomatal movements—stomata open when they are turgid and close when they are flaccid. The increased turgor is believed to be the result of an uptake of potassium by guard cells[1–3]. The epidermal tissues use malate or chloride or nitrite ions to balance K^+[4,5]. FC, a fungal toxin, enhances the potassium influx into guard cells and opens stomata[6,7]. We have investigated in detail the FC stimulated stomatal opening in relation to the uptake of potassium by guard cells. In the presence of FC, potassium moved specifically into the guard cells not only from subsidiary cells but also from the epidermal hairs.

The plants of *Commelina benghalensis* were raised in 30 cm diameter seed pans outdoor with a natural approximately 12 hr photoperiod and average temperatures of 30°C day/20°C night. Second to fourth leaves from the top were used for experiments. Strips of 10 × 5 mm were prepared from the lower epidermis as described by Raghavendra[5,8]. Strips were placed in 5 cm diameter petridishes containing 20 ml of 25 mM MES-tris buffer (pH 7.55) with 0.05 mM $Ca(NO_3)_2$, 10^{-5} M FC, if present and KCl or NaCl at the required