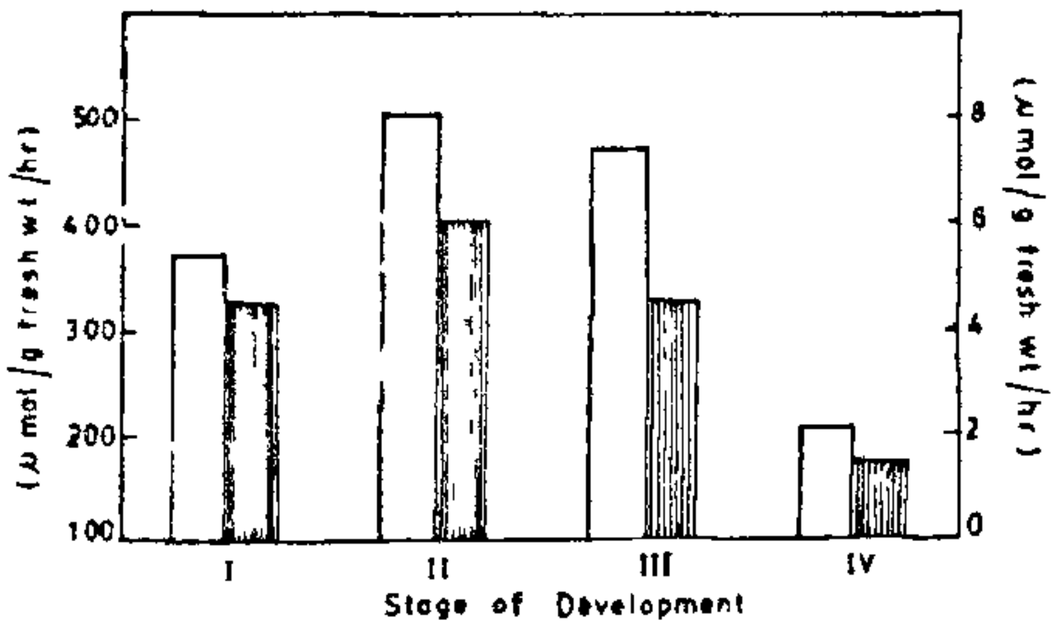

Figure 2. Activities of NAD-malate dehydrogenase and NADP-malic enzyme in the maize silks at four phases of development. [□. NAD-malate dehydrogenase; ▨. NADP-malic enzyme.]

synthetic carbon metabolism is hardly expected and a C_4 non-photosynthetic function seems to be required for their development.

In comparison to stage I, the activity of PEP carboxylase increased markedly through stages II and III and declined steeply at stage IV (figure 1). Thus, the increase in enzyme activity corresponded to the period of active elongation of developing silks. The developmental profiles of NAD-malate dehydrogenase matched with those of PEP carboxylase except that the activities were manifold higher and the decline at stage IV was much greater (figure 2). Glutamate-oxaloacetate transaminase activity showed a rising trend of activity during the silk development (figure 1). The above observations may be interpreted to mean that oxaloacetate, the product of PEP carboxylase activity, is very rapidly reduced to malate by quite high activities of NAD-malate dehydrogenase and a small amount may be used by glutamate-oxaloacetate transaminase to form aspartate. Thus, the predominant C_4 acid formed in elongating maize silks seems to be malate rather than aspartate.

The malate can subsequently be decarboxylated by NAD- and NADP-malic enzymes. Reasonable levels of NADP-malic enzyme were found in the maize silks but the values at each developmental stage investigated were lower compared with the other three enzymes (figures 1 and 2). Therefore, the decarboxylation of malate by NADP-malic enzyme in the cytosol to provide pyruvate and NADPH can only be of minor quantitative importance. It is quite likely that the malate formed may get accumulated in the vacuoles to achieve necessary turgor changes and as well as be a source of carbon for the anaplerotic operation of TCA cycle upon oxidation in the mitochondria.

Overall, it is deduced that there may be a necessity in the formation of C_4 acids for anaplerotic metabolism (biosynthesis) and for making available the osmoticum for the development of maize silks.

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2. Basra, A. S. and Malik, C. P., *Biol. Rev.*, 1985, **60**, 357.
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A NEW STEM GALL ON *GNETUM SCANDENS* ROXB

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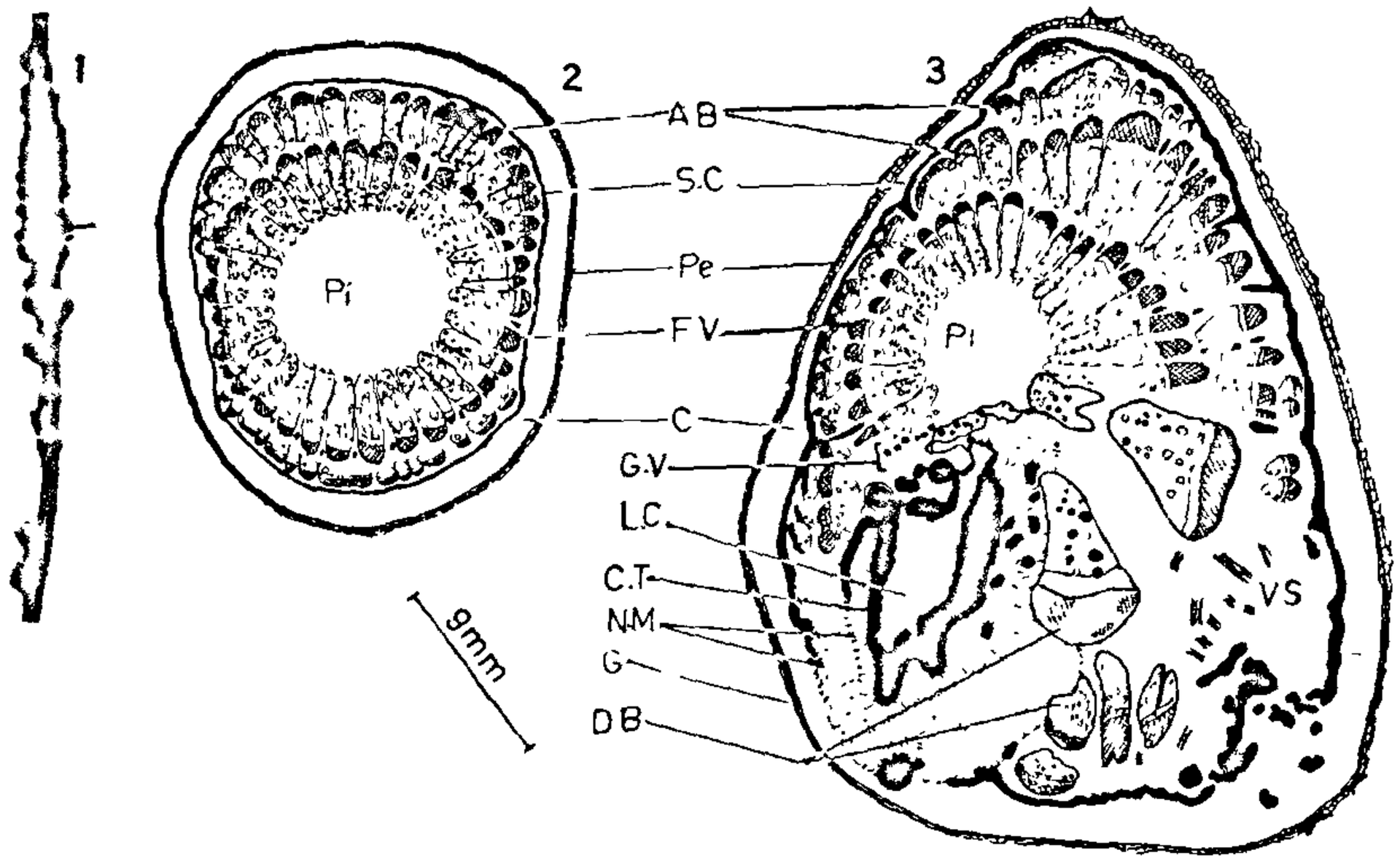
The insect-induced foliar gall on *Gnetum ula* seems to be of common occurrence. Swamy and Krishnamurthy[1] described 4 morphologically different leaf galls on this host plant which were presumably caused by different insects. As far as the author is aware, the present report of stem gall on *Gnetum*, induced by a midge (Cecidomyiidae) is the first record for this taxon.

The galls were collected near Nagercoil (Tamilnadu) where *Gnetum scandens* Roxb occurs as a shrubby liane in shady habitats. The midge larva infests young shoot axis and the gall develops in such severe form that the liane becomes defoliated totally and eventually dies. Galling was noticed from the ground level of the shoot up to several feet above. The galls are subglobose or hemispherical, solid, hard unilocular, indehiscent dark brown cortical swellings of the tender branches. They are either solitary or crowded in large numbers close to each other and often agglomerated into complex masses or multilocular and irregularly knotted structures (figure 1). The surface of the gall has remnants of distorted epidermal layer. Fairly wide circular holes seen on the galls represent the exit holes of the insects.

The gall-free normal stem (figure 2) has a thin zone of periderm followed by fairly broad parenchy-



Figures 1–3. 1. A twig of *Gnetum scandens* Roxb bearing crowded galls (exit hole is indicated by an arrow); **2 and 3. 2.** *Gnetum scandens* Roxb; **2.** T.s. normal stem; **3.** T.s. gall. (A.B: accessory vascular bundles; C: cortex; C.T: crushed nutritive tissue; D.B: dilated vascular bundles; F.V: first ring of vascular bundles; G: gall portion; G.V: gum-filled vessels; L.C: larval chamber; N.M: nutritive meristem; Pe: periderm; Pi: pith; S.C: sclerotic cylinder; V.S: scattered vascular strands).

matous cortex wherein isolated and scattered brachysclereids occur. The cortex is bounded internally by a thin band of brachysclereids which corresponds to the contour of the coaxial cylinder of collateral vascular bundles, each bundle with bundle-cap fibers.

In a gall, nearly two-thirds of the stem exhibits normal organization whereas the remaining portion forms the gall (compare figures 2 and 3). The larval chamber is located initially between the sclerotic cylinder and the outermost ring of vascular bundles. The larva feed on the parenchymatous ground tissue by biting and chewing. The mechanical injury, combined with diffusing salivary secretion of the larva, stimulates the parenchyma tissue to proliferate profusely. Subsequently, the larval chamber is surrounded by dark crushed dead tissues resulting from the feeding activity of the larva. The disintegrated tissue is constantly renewed by a broad zone of meristem which is established on the outer boundary of the crushed tissue (figure 3). This meristem consists of radially aligned files of cambiform cells with dense cytoplasm and prominent nuclei. The meristem produces new cells bilaterally and the inner derivatives form the nutritive tissue and the outer derivatives remain in radial files of larger vacuolated cells. Thus, the nutritive meristem is the potential source of the gall tissues. The interfascicular parenchyma in the vicinity of the larval chamber undergoes diffuse proliferation, so that the vascular bundles become widely separated. The intrafascicular parenchyma also proliferates causing dilation and distortion of the vascular bundles (figure 3). The cortex of the gall does not exhibit significant proliferation, but the cells undergo limited enlargement. The sclerotic cylinder breaks into fragments in the gall, consequent to the hyperplastic activity of the gall tissues. Another notable feature is the deposition of a gummy substance in the lumen of the vessels occurring in the vicinity of the larval chamber. When the galls are agglomerated all round the stem, most of the vessels are plugged with gum deposition, eventually causing defoliation and death of the plants. The adult insect escapes through a circular hole which the larva prepares during later period of its growth.

15 December 1986

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REVERSIBLE AND IRREVERSIBLE CHANGES IN THE ATPASE DISTRIBUTION DURING HEXACHLOROCYCLOHEXANE-INDUCED LIVER LESIONS IN INBRED SWISS MICE

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The hexachlorocyclohexane (HCH) is a persistent type of organochlorine pesticide. It is widely used in India to control malaria vector and pests as an alternative to DDT. The levels of HCH residues have been reported to be quite high in the Omental fat samples[1]. Moreover, the distribution of several enzymes has been reported during HCH-induced liver tumour in inbred Swiss mice[2–4]. However, no attempt has yet been made to throw light on the reversibility of histopathological and histochemical changes in the HCH-induced liver lesions. The present study is therefore aimed at finding reversible and irreversible changes in ATPase distribution in the HCH-induced liver lesions.

Male, healthy 6-week-old, Swiss mice (15 in number) were exposed to technical grade HCH (containing 13.5% γ-isomer obtained from Hindustan Insecticides, New Delhi) in the diet at 500 ppm level for 4 months. Subsequently, the animals were kept on the normal diet for 10 months. The age and sex matched animals of control group were fed normal diet without HCH throughout the experiment. The animals of both the groups were killed by cervical dislocation and their livers were immediately dissected out and fixed in chilled 10% neutral formaldehyde solution for a brief period. The 10 μm thick sections were cut on freezing microtome. The sections were briefly washed in cold distilled water and processed for the localization of ATPase. Both, Padykula and Herman's, and Wachstein and Meisel's techniques were used in the present investigation. Prescribed controls were also simultaneously employed.

All the animals have shown the development of the tumours and neoplastic nodules of the liver suggesting multifocal alteration in the liver of experimental animals. Histoenzymological preparations of the liver after 4 months of HCH exposure and 10 months discontinuation of HCH in diet revealed decline in ATPase activity in the tumour (figures 5, 6 and 7) as compared to nontumour areas (figures 5 and 6). The identical ATPase activity has been observed in the sinusoids and blood capillaries of normal (figures 1 and 2) and nontumour part of the liver (figures 5 and 6). Irrespective of the central or periportal areas, the ATPase activity in both the groups is uniformly spread over the entire area in normal (figures 1 and 2) and the nontumour part of the liver (figures 5 and 6). At the same time, cellular morphology in the nontumour part (figures 5 and 6) is almost similar to the control livers (figures 1 and 2).

Cells in the neoplastic areas reveal altered cellular morphology while ATPase activity in them is similar to the non-nodular part of the liver (figures 3 and 4). However, the tumours are very much deficient in the ATPase activity as compared to nontumour areas (figures 5 and 7). In the tumour, ATPase activity is mainly present on the plasma membrane.

The distribution of ATPase and morphology of the cells in nontumour, non-nodular and control liver is identical in the present study whereas continuous exposure of HCH in the previous study has revealed marked changes in the distribution of ATPase and morphology of nontumour and non-nodular cells[3]. Based on these two studies the following facts have come to the light. i) Alteration in the cell morphology and ATPase distribution in nontumour and non-nodular part of the liver as reported previously is perhaps the direct action of HCH; therefore, these changes are reversible and within homeostatic level. ii) The changes in the tumour are probably genetic and therefore are of permanent type and irreversible even if the HCH is withdrawn from the diet for sufficiently long time. iii) Four months of maximum tolerable dose of HCH is more than sufficient to irreversibly transform certain cells in the liver to induce cancer after 10 months. iv) The pattern of ATPase distribution in neoplastic nodule suggests only morphological changes in these cells. v) Both, Wachstein and Meisel's, and Padykula and Herman's techniques are suitable for ATPase localization but the former method gave more precise localization of ATPase.

In conclusion, it may be suggested that the changes in the nontumour and non-nodular part of