

suppressed rhizogenesis and promoted embryogenesis in carrot (*Daucus carota* L.) cells *in vitro*. The concentration of reduced nitrogen (30 mM) used by Halperin<sup>5</sup> was higher than those used in the present study (4.38 g l<sup>-1</sup> as compared to up to 4 g l<sup>-1</sup> glutamine in the present study) but this difference appears to be rather small to account for the differences in the findings of the two studies. Another possible explanation may be that different species may respond differently reduce to nitrogen in the culture medium.

7 March 1983; Revised 9 August 1983

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## LONGEVITY OF GUARD CELL CHLOROPLASTS UNDER TOXIC ACTION

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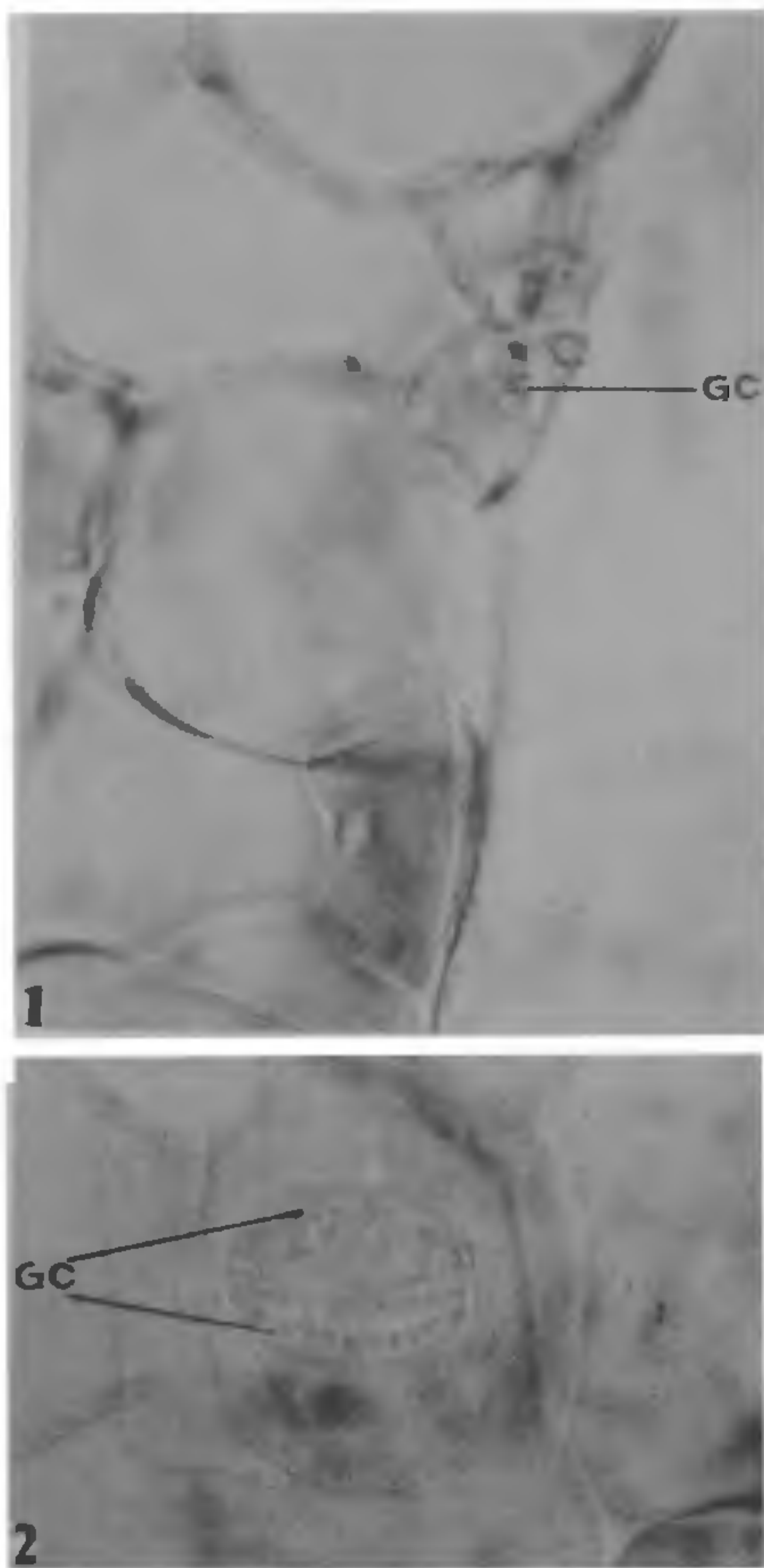
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THE unique property of the herbicidal chemical, 3-amino 1,2,4 triazole (AT) selectively degrading chloroplasts in green plant cells is reported<sup>1-4</sup>. At higher (lethal) concentrations, both mesophyll and guard cell chloroplasts of the treated leaves are destroyed. When applied in sublethal concentrations to growing buds it

prevents chloroplast development and the leaves produced after this chemical treatment turn white. The whitened leaves when examined showed all the mesophyll cells devoid of chloroplasts. Among the mesophyll cell chloroplasts, those in the cells adjoining to the stomatal openings were the last to be degraded. Nevertheless, the chloroplasts of the stomatal guard cells were fully green, intact, functional and appeared to be least affected by the chemical treatment. The stomata in the whitened area were found to open during day and close at night just like those of normal untreated leaves. Their stomatal pore in the open condition was in no case smaller indicating that their guard cell chloroplasts were neither defunct nor sluggish. Further, examination of AT treated whitened areas of leaves from several plants, both dicot and monocot showed similar results.

The striking conservation of the guard cell chloroplasts of the AT treated leaves in contrast with the complete degradation of their mesophyll counterparts is interesting and a consideration of how the guard cell chloroplasts survive longer even under the toxic action of the chemical would be profitable because it has implications for the understanding of cellular aging and stomatal function. Longevity of guard cell chloroplasts in senescing leaves where the mesophyll chloroplasts get degraded and defunct<sup>5</sup> is a case comparable to the present observations after AT treatment. Accumulation of toxic waste materials in cells either by the absorption of an externally applied chemical as in the present case or by their formation inside the tissues on account of degenerative processes during senescence could be one of the important ultimate reasons for chloroplast degradation. In the guard cells of the stomatal opening such accumulation of toxic materials is prevented by their immediate removal through oxidation or detoxification due to sufficient aeration since the guard cells are most exposed to or are in immediate contact with fresh air. On the other hand, in the case of mesophyll chloroplasts as they are situated away from the stomatal opening and thus lacking in sufficient ventilation, immediate removal of toxic materials through oxidation naturally is not possible. This should account for the survival and longevity of guard cell chloroplasts in both AT treated (figures 1 and 2) and senescing leaves.

The fact that at lethal concentrations AT degrades both mesophyll and guard cell chloroplasts alike shows that both mesophyll and guard cells are permeable to AT. This aspect of differential permeability of mesophyll and guard cells to AT though not extensively verified with higher plant cells, experi-



**Figures 1 & 2.** T. S. and L. S. of guard cells from AT treated (*Rhoeo discolor*) leaf showing intact green chloroplasts (G. C)  $\times 800$ .

ments with unicellular algal chloroplasts (unpublished work) have shown that these chloroplasts in well-aerated situations exhibit resistance to AT treatment. In sublethal concentrations AT affects not only differentiation of chloroplasts but it has been observed<sup>1-3</sup> that all the plastid elements are destroyed except the nucleic acid components of the proplastids. This is in contrast with the naturally white areas of

white or mosaic leaves which did contain colourless plastids (undifferentiated). It may be argued that the intracellular concentration of  $O_2$  is not so low as to stop oxidation of the AT absorbed. In that case there should not have been a gradation in the degradation and depletion of chloroplasts in mesophyll cells near and away from the stomatal opening. All these findings further point to a generalised conclusion; that is, 'breathe more fresh air to live longer'.

Thanks are due to Dr C. A. Ninan and the University of Kerala for facilities.

25 May 1983

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#### A NEW HYPHOMYCETES— *PHAEOSARIOPSIS CHONEMORPHAE* SP. NOV. FROM INDIA.

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THE authors describe here a new species, *Phaeoisariopsis chonemorphae* causing leaf spot disease of *Chonemorpha macrophylla* growing in the campus of Suman bungalow, Pachmarhi.

*P. chonemorphae* sp. nov. (figure 1)

Leaf spots 3–8 mm, quadrangular to irregular, reddish brown, becoming ash-coloured at maturity, surrounded by yellowish halo.

Stroma partly immersed, compact, bulbous, dark brownish black, 40–50  $\mu\text{m}$  in diam.; conidiophores macronematous, caespitose or forming loose to compact synnemata, up to 230  $\mu\text{m}$  long, 3.8  $\mu\text{m}$  thick at the base, septate, olivaceous brown to brown, simple, geniculate, lighter at tips; conidia solitary, dry, acropleurogenous, mostly obclavate, olivaceous to olivaceous brown, end cells subhyaline, conicotruncate at the base, broader in the middle, smooth, 3–8 septate, septa sometimes thick and dark, 30–50  $\times$  3.8–7  $\mu\text{m}$ .

On the living leaves of *C. macrophylla* G. Don. (*Apocynaceae*), Pachmarhi (Madhya Pradesh), India,