Table 2 *Effect of surface wax on germination of spores of Colletotrichum capsici and Helminthosporium rostratum*

Treatment	*C. capsici* (% germination)	*H. rostratum* (% germination)
5-Day old fruit wax	93.63 (80.02)	96.63 (79.73)
15-Day-old fruit wax	94.50 (76.73)	95.00 (77.77)
25-Day old fruit wax	96.25 (79.57)	95.75 (79.74)
Control (sterile water)	99.13 (87.27)	99.76 (88.57)

Figures in parentheses are transformed values.

	C.D. ($P=0.05$)
Fungus	NS
Treatment	4.55
Fungus × treatment	NS

In the present study, the amount of wax was higher in resistant green fruits than in susceptible ripe fruits, when expressed on dry weight basis. The chief components of cuticle are cutin and wax. Cutin forms the framework of the cuticular membrane which carries the wax on its surface and embedded within it[7]. A difference in quantity of wax between susceptible and resistant tissues has been reported in leaves of *Citrus aurantifolia* with reference to infection by *Gloeosporium limetticola*[1,2].

Dickinson[8] discussed the possibility that the waxy surface may present the first barrier by repelling the water film required by the pathogen for germination. Nutmen and Roberts[9] ascribed differences in susceptibility of coffee varieties to berry disease caused by *Colletotrichum coffeanum* to some physical and chemical differences in the cuticle, which made penetration of the resistant variety more difficult.

The possible mechanisms of interrupting infection are: (i) thickness of natural waxy layer in barley against *Erysiphe polygoni* f. sp. *hordei*[3], (ii) repelling of film of water on the leaf surface in sorghum against *Peronosclerospora sorghi*[10], and (iii) chemical substance in the cuticle of chrysanthemum against *Botrytis cinerca*[11]. After a critical examination of the role of wax in disease resistance, Royle[12] suggested that there was some evidence that cuticle provided protection against pathogens.

21 October 1988; Revised 14 February 1989

1. Roberts, M. F. and Martin, J. T., *Ann. Appl. Biol.*, 1963, **51**, 411.
2. Wheeler, B. E. J., *Trans. Br. Mycol. Soc.*, 1963, **46**, 193.
3. Edwards, H. H. and Allen, P. J., *Phytopathology*, 1970, **60**, 1504.
4. Martin, J. T. and Batt, J. F., *Ann. Appl. Biol.*, 1958, **46**, 375.
5. Blakeman, J. P. and Sztejnberg, A., *Physiol. Plant Pathol.*, 1973, **3**, 269.
6. Shipton, W. A. and Brown, J. F., *Phytopathology*, 1962, **52**, 1313.
7. Baker, F. A., Batt, R. F. and Martin, J. A., *Ann. Appl. Biol.*, 1964, **53**, 59.
8. Dickinson, S., In: *Plant Pathology—An Advanced Treatise*, (eds) J. G. Horsfall and A. E. Dimond, Academic Press, New York, 1960, vol. II, p. 203.
9. Nutman, F. J. and Roberts, F. M., *Trans. Br. Mycol. Soc.*, 1960, **43**, 489.
10. Frederikson, R. A., *Plant Dis.*, 1980, **64**, 903.
11. Blakeman, J. P. and Atkinson, P., In: *Microbiology of Aerial Plant Surface*, (eds) C. H. Dickinson and F. F. Prrece, Academic Press, New York, 1976, p. 441.
12. Royle, D. J., In: *Biochemical Aspects of Plant Parasitic Relationship*, (eds) J. Friend and D. R. Tredfall, Academic Press, New York, 1976, p. 161.

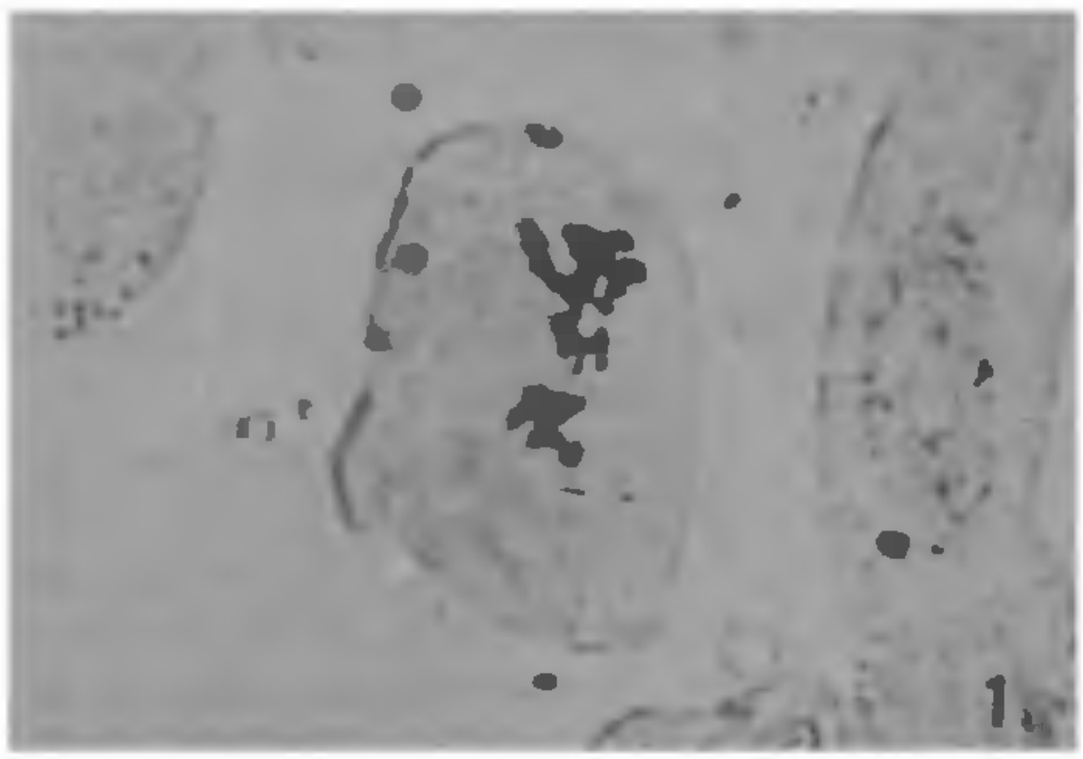
MANGIFERINE-INDUCED CHROMOSOME ABERRATION IN ROOT-TIP CELLS OF *SOLANUM INCANUM* L.

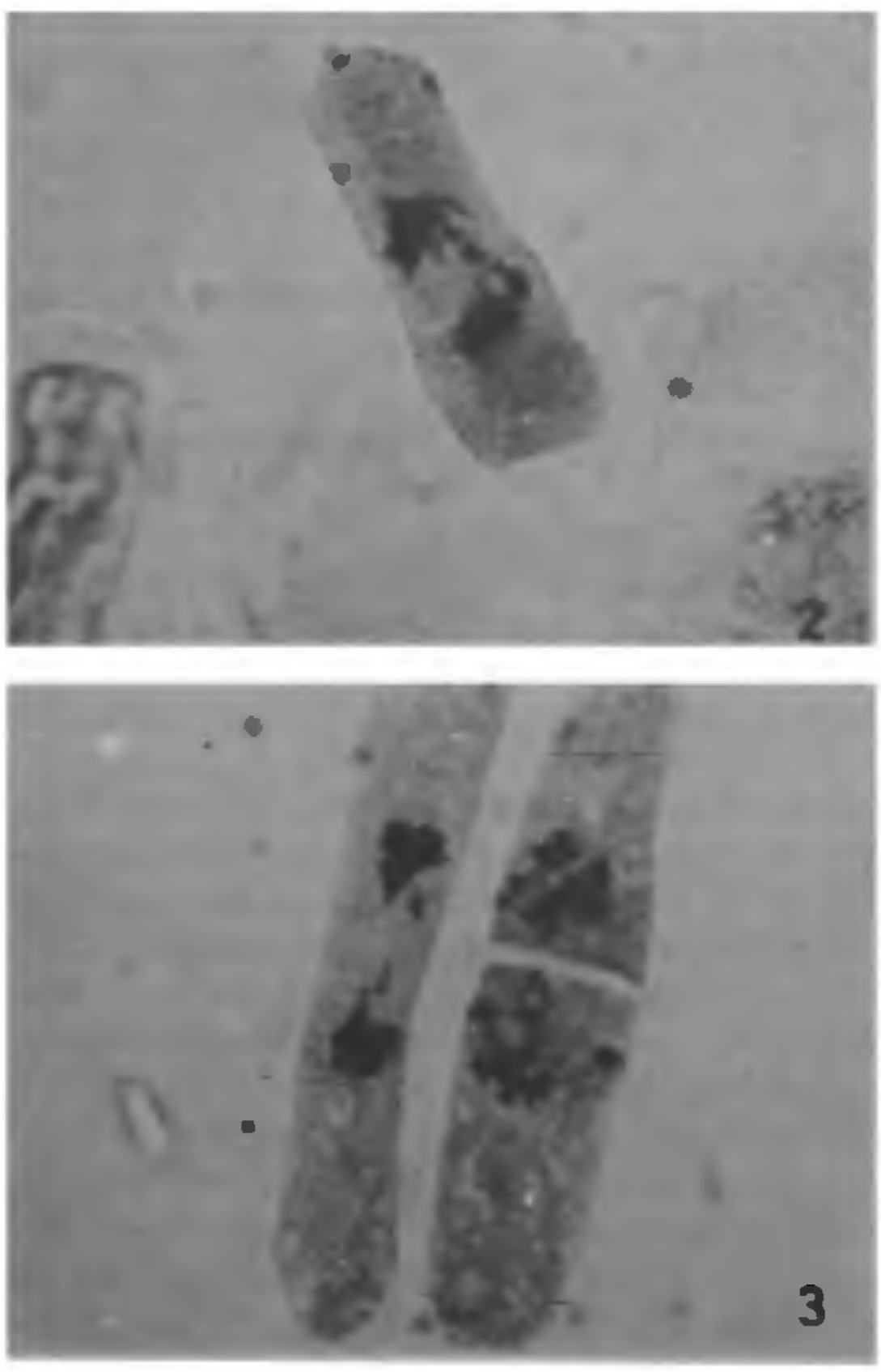
G. KUMAR
Department of Botany, Banaras Hindu University, Varanasi 221 005, India

ROOT-TIPS from mangiferine-treated seeds of *Solanum incanum* showed several kinds of chromosome aberrations. The frequency of aberration increased with concentration of mangiferine. These observations are suggestive of its mutagenic property. The correlation coefficient between the aberrations and concentration is significant at the 1% level.

Mangiferine, a naturally occurring glucosylxanthone, is widely distributed in higher plants[1] such as in the members of the families Gentianaceae[2] and Anacardiaceae[3]. It has been found to work as an antimitotic and antifertility agent in animals, particularly mammals[4,5]. These effects had not been known in plants. If this drug can cause antifertility in plants, it may be of use in plant breeding programmes, specially in those cases where fruits and seeds are not required. The present paper reports some initial observations on mitotic chromosome

aberrations induced by mangiferine treatment on dormant seeds





Figures 1–3. Abnormal root-tip cells of mangiferine-treated seeds of *Solanum incanum* (×600). **1,** stickiness of chromosomes at metaphase; **2,** sticky bridge; **3,** lagging chromosomes at anaphase in one cell, and completed cytokinesis in the other.

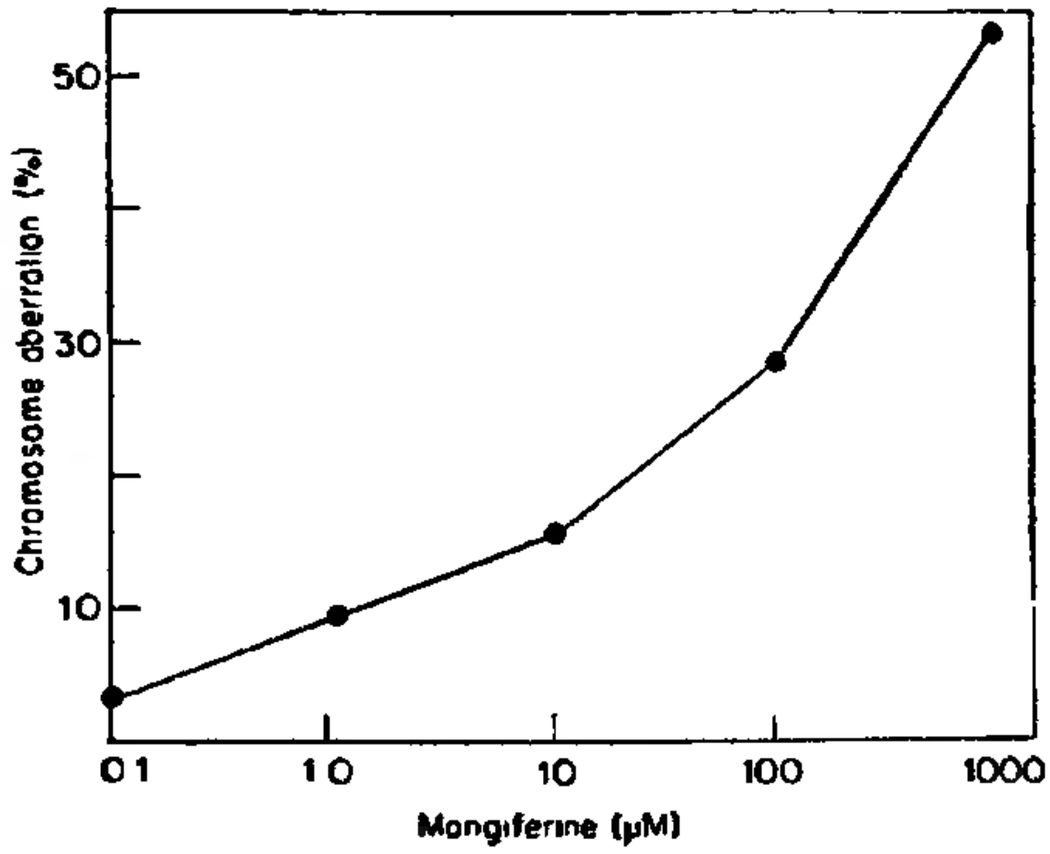


Figure 4. Frequency of chromosome aberrations in *Solanum incanum* with increasing concentration of mangiferine.

Genetically pure, healthy seeds of wild *S. incanum* were treated with different concentrations of mangiferine (molar concentration prepared in phosphate buffer, pH 7) ranging from 0.1 to 1000 μM for 6 h, washed with tap-water and grown in a sand-bed in a greenhouse. The emerging root tips were fixed in Carnoy's solution (acetic acid : alcohol, 1 : 3) for 24 h and squashed in 1% acetoorcein. About 300 root-tip cells were analysed for each kind of chromosomal abnormality.

Cytological analysis of root-tip cells revealed several kinds of chromosome anomalies such as stickiness of chromosomes, bridges, laggards or sticky bridges and fragments (figures 1–3). These abnormalities increased with concentration of mangiferine (figure 4). At 10 mM mangiferine caused necrosis at the root apex; shoots also turned pale and failed to survive. Lower concentrations (10 μM to 100 μM) also induced pale seedlings at low frequency but the seedlings later turned green. The correlation coefficient between concentration of mangiferine and induced chromosome aberrations was highly significant ($r = 0.901$). Thus the present observations suggest that mangiferine is a mutagenic chemical. Perhaps it interacts with nucleoprotein to cause damage to chromosomes. Chromatid bridges with lagging chromosomes appear owing to breakage and reunion of chromosomes[6]. Mangiferine may also prevent spindle formation.

The author thanks Prof. S. Ghosal, Department of Pharmaceutics, Institute of Technology, BHU, Varanasi, for providing mangiferine and the Council

of Science and Technology, Lucknow, for financial support.

22 November 1988

1. Chattopadhyay, S., Chattopadhyay, U., Shukla, S. P. and Ghosal, S., *Pharm. Res.*, 1984, **61**, 237.
2. Chaudhuri, R. K. and Ghosal, S., *Phytochemistry*, 1971, **10**, 2425.
3. Ghosal, S., Biswas, K. and Chattopadhyay, B. K., *Phytochemistry*, 1978, **17**, 689.
4. Ghosal, S., Srivastava, A. K., Srivastava, R. S., Chattopadhyay, S. and Maitra, M., *Planta Med.*, 1981, **42**, 279.
5. Farns Worth, N. R., Bingel, A. S., Cordell, G. A., Crane, F. A. and Fong, H. H. S., *J. Pharm. Sci.*, 1975, **64**, 535.
6. Kihlman, P. P., *Adv. Cell. Mol. Biol.*, 1971, **1**, 59.

CALLUS INITIATION AND REGENERATION POTENTIAL IN DIFFERENT GENOTYPES OF *ERUCA SATIVA*

SURYA-PARKASH, J. B. CHOWDHURY and R. K. JAIN
Department of Genetics, Haryana Agricultural University, Hisar 125 004, India

ERUCA SATIVA Mill. 'taramira' ($n=11$, genome formula–EE) is highly resistant to aphids (*Lipaphis pseudobrassicae*) and thrives well under both rainfed and drought conditions[1]. Besides, its oil is rich in erucic acid[2], an industrially useful compound. Direct utilization of *E. sativa* in a breeding programme has been limited owing to the presence of strong incompatibility barriers with other *Brassica* species. Attempts are now being made to use *in vitro* techniques for the genetic amelioration of this crop species[3,4]. The present study was undertaken to identify genotypes responsive to *in vitro* culture and to develop regeneration protocols from cotyledonary explants of *E. sativa*.

Seeds of *E. sativa* cvs. T-27, TMH-46 and TMH-48 were surface-sterilized for 20 sec with 90% ethanol and for 7 min with $HgCl_2$ solution, washed thoroughly in sterilized distilled water, and germinated on agar (0.8%)/sucrose (2.0%) medium under a light and dark cycle of 16/8 h at 25 ± 1°C. After 7–8 days, the cotyledonary leaves were excised and cultured on MS[5] medium supplemented with cytokinins and/or auxins (table 1). About 100 cotyledons were cultured per medium/genotype tested. The number of explants regenerating shoots and the number of shoots per regenerating explant were recorded after 30 days of culture. All the cultures were kept under continuous fluorescent light of 5000 lux intensity at 25 ± 1°C.

Cotyledonary explants enlarged significantly within a week of culture and exhibited three kinds of response: callus, root formation and shoot formation (table 1). Callusing was observed at the cut ends of the explants in all the cultivars and on all the media, although with different efficiencies. Callus proliferation could be increased by causing multiple injuries

Table 1 *Effect of different media on callus induction and root and shoot regeneration in cotyledonary explants of different cultivars of Eruca sativa*

Medium	Cultivar	Cotyledons (%) forming calli	roots	shoots
MS + 0.05 mg/l NAA	T-27	64.6	89.6	2.1 (0 1)*
	TMH-46	57.8	85.6	1.1 (0 1)
	TMH-48	60.0	90.8	1.5 (0 1)
MS + 0.5 mg/l NAA + 2 mg/l BAP	T-27	87.1	77.0	20.7 (0 10)
	TMH-46	90.0	84.5	15.6 (0 10)
	TMH-48	93.3	85.0	8.3 (0 5)
MS + 0.2 mg/l NAA + 2.0 mg/l kinetin	T-27	95.8	91.5	15.3 (0 10)
	TMH-46	93.1	85.4	10.4 (0 5)
	TMH-48	91.4	81.4	12.9 (0 6)
MS + 2 mg/l NAA + 10 mg/l kinetin	T-27	3.9	11.8	1.0 (0 1)
	TMH-46	10.7	14.6	1.0 (0 1)
	TMH-48	33.4	36.2	1.0 (0 1)

*Number of shoots per regenerating explant.