Table 1 *Effect of salinity stress on levels of phenols, quinones and ascorbic acid and activity of polyphenol oxidase in groundnut leaves*

Parameter	Control	Stress
Total phenols (mmol g)	3 96 ± 0 13	5 48 ± 0 26
o-Dihydric phenols (mmol g)	2 83 ± 0 05	4.11 ± 0 16
Quinones (mmol g)	1 64 ± 0 06	0.062 ± 0 003
Ascorbic acid (mg% o)	23 86 ± 0.84	9.75 ± 0.33
Polyphenol oxidase (ΔOD 30 sec/g)	4 43 ± 0.16	2 51 ± 0 08

Values are mean ± SE ($n = 6$).

from leaves was extracted with 4% oxalic acid and determined by the method of Roe[9]. Polyphenol oxidase was extracted according to Azhar and Krishnamurthy[10] and assayed according to Mayer *et al*[11].

The results obtained are given in table 1. The total phenols and o-dihydric phenols were increased in the test group. Quinone and ascorbic acid were decreased. Polyphenol oxidase activity in the saline group also was lower compared to that in the control group. The higher levels of phenols and lower levels of quinones in the leaves of the salinity-stressed plants can be explained on the basis of the lower activity of polyphenol oxidase[12]. Ascorbic acid has been implicated in the reduction of quinones to phenols[13]. The present studies indicate that the role of ascorbie acid in phenol formation may be minimal. It has been reported that, under stress conditions, the metabolism of a significant portion of carbohydrates switches from glycolytic to the pentose phosphate pathway mode, thereby leading to accumulation of phenols synthesized via the shikimate pathway[14]. The utilization of phenols for lignin formation involves the enzyme peroxidase[15]. Studies in this laboratory have shown that this enzyme is decreased in groundnut leaves and cotyledons during stress[16,17]. Thus the protecting accumulation of phenols may be due to the combined operation of three factors—decreased conversion of phenols to quinones by polyphenol oxidase, increased oxidation of carbohydrates via pentose phosphate pathway and decreased utilization for lignin formation. The accumulation of phenols may also compensate for the decrease in ascorbic acid, which also has a protective role[2]. The reduced activity of polyphenol oxidase, in addition to allowing phenols to accumulate, has the additional effect of keeping the level of quinones in check. Quinones, while possessing antimicrobial properties like phenols, have been shown to be toxic to the plant cells as well[14].

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ADVENTITIOUS BUD FORMATION FROM LEAF CULTURES OF CASTOR (*RICINUS COMMUNIS* L.)

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SUCCESS in regeneration and propagation *in vitro* has been achieved in several oil crops. However, very

little is known about the tissue culture response of castor despite the fact that it is one of the major oil yielding crops of the world and has assumed great importance as a source of vegetable, medicinal and industrial oil, widely used in different sectors of the international economy[1]. Earlier reports on tissue culture work with castor is limited to callus induction and organogenesis[2–6]. With this in view, work was initiated for developing cell and tissue culture system for castor and recently reported morphogenesis from callus cultures[7] which is a prerequisite for the development of an *in vitro* system for castor. In the present study we report induction of adventitious buds and their differentiation from leaf cultures.

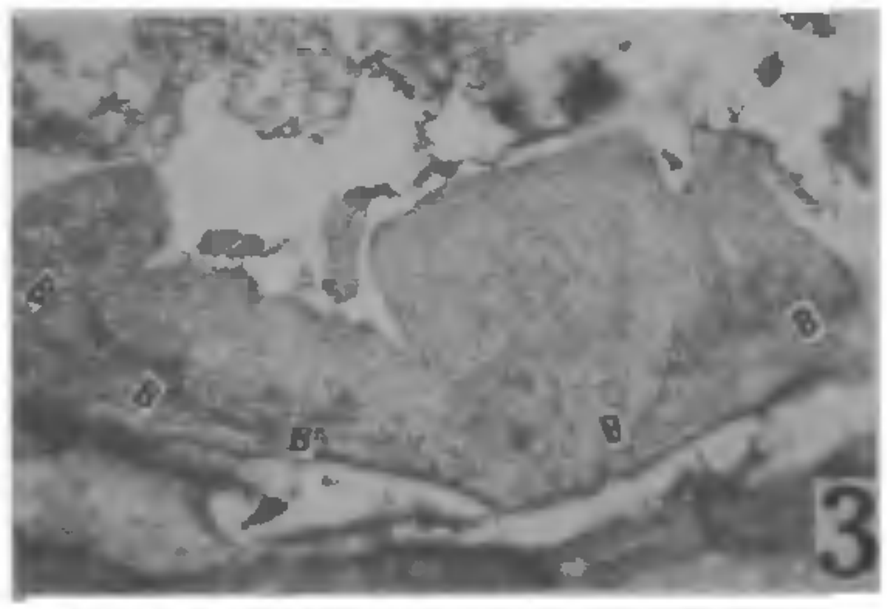
Castor (*Ricinus communis* L. cv. GAUCH-I) seeds obtained through the courtesy of the Directorate of Oil Seed Research, Hyderabad, were germinated in earthen pots. Young leaves collected from 12-day-old seedlings were surface-sterilized with 0.1% $HgCl_2$ solution for 4 min, thoroughly washed with distilled water and cultured on Murashige and Skoog[8] basal medium supplemented with kinetin (KN), naphthalene aceticacid (NAA), and indole aceticacid (IAA) alone and in different combinations and concentrations. The cultures were maintained at 25±2°C under continuous light (2000 lux). For histological studies the cultured explants were periodically fixed in FAA (90:5:5; farmalin : acetic-acid : alcohol) and processed in a teritiary butyl alcohol series and embedded in paraffin. Serial sections of 6–8 μm were cut and stained with amidoblack[9].

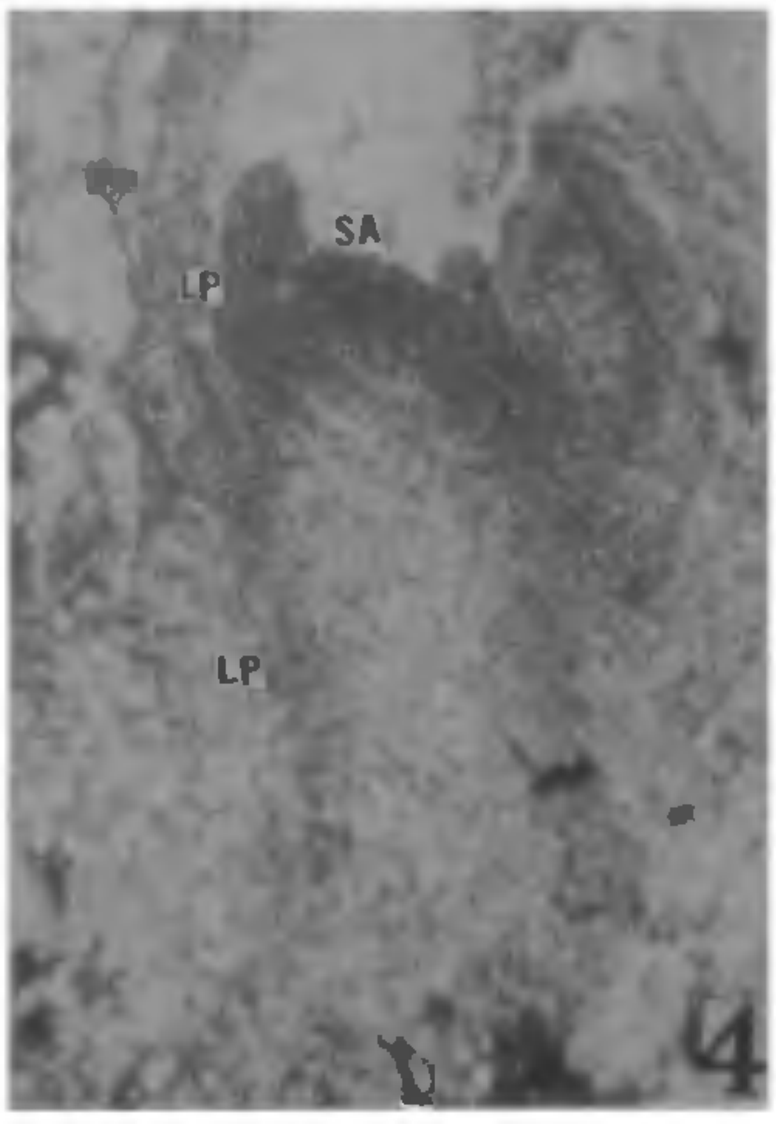
After two weeks of incubation shoot buds (3–4) and leafy shoots accompanied by varying amounts of callus formation arose from cut ends of the explants on medium supplemented with KN 2 mg/l and IAA 1 mg/l (figure 1). Media containing KN, NAA, IAA alone in the range of 0.5 mg/l to 2 mg/l callus were produced. Roots were the only organs formed on explants treated with combinations of KN at 0.5 mg/l and NAA 2 mg/l.

The transfer of shoot buds (1–2 cm height) to the rooting medium, which consisted of half strength MS salts with KN 0.5 mg/l and NAA 2 mg/l, delayed root initiation by 15–20 days. However, the growth of the roots was rapid after its initiation (figure 2). Roots formed *in vitro* are generally normal though stout and fleshy roots were also observed.

Histological studies on the explants reveal that the callus originated from the cut ends and rarely from sub-epidermal tissue. After 15 days of incubation

Figures 1 and 2. 1. Adventitious shoot buds and leafy shoots from leaf explants; 2. Plantlet with prominant roots.





Figures 3 and 4. 3. A row of adventitious shoot buds originating from cut ends of the leaf explants; 4. Adventitious bud showing complete tissue organization. (LP, Leaf primorida, SA, Shoot apex; PC, Procambium; B, Adventitious shoot buds.)

protrusion of adventitious buds primordia appear from the cut ends of the explant, either singly or in rows (figure 3). After 20 days these progressively develop into adventitious buds possessing a clear dome-shaped shoot apex and leaf primordia with conspicuous vascular cambium (figure 4).

The major constraint in the present work is the low rate of plant differentiation from adventitious buds. This is due to retarded growth and the presence of callus at every stage of organogenesis studied. Further work is in progress to overcome this difficulty.

These findings, though preliminary, suggest the possibility of *in vitro* regeneration and micropropagation of castor, a project which has not yet been reported.

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ISOLATION OF NEWCASTLE DISEASE VIRUS (AVIAN PARAMYXOVIRUS-1) FROM PIGEONS (*COLUMBA LIVIA*) IN INDIA

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AVIAN paramyxoviruses of serotype 1 have been isolated from racing pigeons during the widespread outbreaks of a severe viral disease that spread from Italy through Europe in 1981 and 1982 and to Great Britain in 1983[1–4]. The disease presented a clinical picture resembling the neurotropic form of Newcastle disease in poultry. Outbreaks of the disease and virus involved in Japan have recently been reported[5]. In March 1988, outbreaks of similar disease were observed in racing pigeons in parts of Punjab and in the Ambala city of Haryana.

Samples of pooled brain and viscera from sick pigeons were inoculated into the allantoic cavity of 9 to 10-day-old chicken embryos. Two viral isolates were recovered. With one viral isolate, initially there was no haemagglutination of chicken red blood cells; however, both the isolates were further passaged in eggs for identification. The passaged viral isolates were tested in haemagglutination inhibition (HI) tests as described[6], using chicken anti-Newcastle disease virus (NDV) 'F' strain serum and convalescent serum from pigeons. Both the isolates were found to possess haemagglutinating antigens inhibited by the NDV antiserum and the convalescent serum from pigeons, thus identifying the agents as Newcastle disease virus. It appears to be the first report on the isolation of Newcastle disease virus, from pigeons, in India.

The results of HI tests conducted on six affected pigeons are given in table 1. Only two of the six paired sera showed a conversion from negative to

Table 1 *Results of HI test on paired sera of six pigeons from Ludhiana*

Serum specimen No.	Antigens**	
	NDV 'F' strain	54/88 virus (pigeon isolate)
*8811–1	<2	<2
8811–2	4	16***
8813–1	<2	<2
8813–2	8	16
8818–1	4	32
8818–2	8	64
8822–1	2	32
8822–2	2	16
8825–1	8	32
8825–2	4	32
8827–1	8	16
8827–2	8	64

*The interval between the first and the second specimens varied from 7 to 10 days; **Four haemagglutinating units were used in the HI test proper; ***Titres were expressed as reciprocals of serum dilutions.