CALLUS INDUCTION AND BUD FORMATION IN *FURCRAEA GIGANTEA* VENT., AMARYLLIDACEAE

ESTABLISHMENT of plantlets *in vitro* in monocotyledons is restricted to members of a few families like Gramineae, Orchidaceae, Liliaceae and Iridaceae, which have been ably summarised recently[1-3]. The comparative study of the totipotency in tissue explants and callus of some members of Liliaceae, Iridaceae and Amaryllidaceae show that the morphogenetic response of the latter was not encouraging.

The present investigation on *Furcraea gigantea* Vent., a fiber-yielding plant of Amaryllidaceae is to report the regenerating capability and totipotency of the cells for callus induction and bud differentiation *in vitro*.

Several bulbils having the potentialities to develop into independent plants were produced on the huge branched inflorescence axis. Each bulbil had a basal swollen part gradually tapering to the apex, ensheathed by three or four leaves. The basal part of the bulbil after proper sterilization was cut into four longitudinal sectors under aseptic conditions. The explants were grown on the Murashige and Skoog medium[4] supplemented with kinetin (1·2 mg/l), IAA (4 mg/l) and, 3% sucrose as carbohydrate source. The medium was jelled with 1% Bacto Difco Agar. Prior to autoclaving, the pH was adjusted to 5·8. Cultures were maintained at 28 ± 2°C and with an intensity of 1200 lux light for 8 hours in 24 hours period.

In 13 weeks, the explants regenerated into plantlets without callus formation. These regenerated plantlets were morphologically different from the bulbils, in having a thin basal part ensheathed by many narrow leaves. The basal part of the leaves from the regenerants were excised and transferred to Norstog and Rhamstine medium 59 (2)[5] supplemented with coconut milk (150 ml/l) and 2, 4 -D (0·5 mg/l) and with the omission of adenine sulphate and kinetin. It was jelled with 0·8% Bacto Difco Agar. Physical conditions were maintained as before.

The pale-yellow callus, developed after two weeks of inoculation, became quite profuse at the end of the 5th week and the condensed buds were differentiated. In 13 weeks, the buds developed further and produced many long and narrow leaves. However, rhizogenesis could not be observed even after a long continuous culture.

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POST-INFECTION CHANGES IN VITAMIN C CONTENTS OF BANANA FRUITS

THE deficiency of L-ascorbic acid is known to produce scurvy in human beings. It is required in several metabolic pathways and its biosynthesis in plants has been extensively reviewed by Mapson[5] and Isherwood and Mapson[7].

Helminthosporium speciferum Bain (Nicot) causes considerable rotting of barara fruits in local market and storage. Ascorbic acid contents in rotting 'alpan' bananas were determined by inoculating *H. speciferum*. Six replicates were used for each set of experiment. Inoculated fruits were incubated at 25° ± 1°C for 15 days and healthy and diseased tissues adjacent to the inoculated region were analysed at regular intervals of 5 days. Quantitative estimation in ascorbic acid content was made by Systronics colorimeter. Modified method suggested by Roe and Kuether[6] was employed and the results are presented in Table I.

TABLE I

Showing ascorbic acid (mg/100 gm. of fruit pulp) of controlled and infected fruits of banana

	Ascorbic acid				% of loss in ascorbic acid in 15 days of incubation
	Days of incubation				
	0	5	10	15	
Control	45·6	40·6	28·5	21·4	53·3%
Inoculated with *H. speciferum* ..	30·6	22·26	13·3		70·8%

Table I indicates a gradual loss in vitamin C in both the healthy and infected fruits. However, the fall was comparatively slower in control fruits. The percentage loss in fruits infected with *H. speciferum*

was 70·8% while corresponding loss in healthy fruits was 53·3% only. Many investigatiors[2,4,7] working with common Indian fruits and vegetables have made similar observations in fruits under pathogenesis. Ascorbic acid functions as one of the biologeical oxidation-reduction substances. It is easily oxidized to dehydro-L-ascorbic acid by the enzyme ascorbic acid oxidase or by certain other oxidative enzymes.

It seems, therefore, probable that the decline in the ascorbic acid is due to the production of ascorbic acid-degenerating enzymes either by the fungus itself or by the host-parasite interaction as postulated by Ghosh *et al.*[1].

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RADIATION-GENETIC STUDIES IN GARDEN PEA: TWO EARLY FLOWERING AND RIPENING INDUCED MUTANTS

ISOLATION of early flowering and ripening mutants is one of the most important goals in pea breeding. A few early mutants in *Pisum sativum*, the garden pea, have been reported. The earliness in these mutants is due to the formation of the first inflorescence at the nodes which are formed early in the development of these plants. The flowers in these mutants are produced in the axil of 4th–6th instead of 11th–13th leaf. In comparison to their respective initial lines, most of these mutants flower 10–14 days earlier, but their seed production gets significantly reduced[1].

In a radiation-genetic experiment, in which the seed of Bonneville variety of pea were utilized[5], early plants segregated in two M2 lines and they bred true in M3 and M4 generations. Crossing with the initial line indicated a monogenic recessive control of the earliness in these. Intercrossing between these two early mutants indicated that the two genes inducing earliness were non-allelic, as the F1 was not early unlike the parents. In 1975, 40 plants of each of these two mutants, the initial line, the control, the local line and Prof. Gottschalk's early mutant 46C were grown in a randomised block in 5 replications (each replicate comprised of 40 plants) at two locations, Kurukshetra (N. India) and Shillong (N.E. India); the plant to plant and row to row distance in each case was 10 and 30 cm, respectively. Critical difference for each trait was computed following the usual analysis of variance technique.

Yield and other agronomic characters of these mutants are given in Table I, a perusal of which reveals that EM1 and EM2 differ significantly in morphological traits from 46C. Unlike 46C, they also exhibit a perfectly normal floral structure and physiological earliness. At Kurukshetra, EM1 was significantly shorter and more productive than its initial line and 46C (Table I). However, at both the locations, 46C and EM1 were earlier in flowering and ripening than the initial and the local line. All the genotypes (except EM1) became dwarf, flowered and ripened later, and, yielded fewer seeds at Shillong than at Kurukshetra (Table I). This differential behaviour of the genotypes at these two locations could be an expression of "place or location effect". At Shullong, both the early mutants exhibited a significantly higher seed production than the initial and local line; highest seed production being in EM1. In fact, EM1 is the best productive early genotype at both the locations and represents one of the potential genotypes to be used for pea breeding in these two regions. Unfortunately, due to late sowing, the pea genotypes were attacked by *Erysiphae polygoni* DC. and *Perenosporapisi* Syd., at the fruiting stage at Shillong. This also reduced their total grain production.

While Snoad and Arthur[7] found that early flowering from the accumulation of dominant alleles, Rowlands[6] and Snoad and Arthur[7] reported the result to be due to an accumulation of recessive alleles. Watts *et al.*[9] regarded that the early flowering in garden pea was due to an accumulation of recessive alleles, and the genetic system was mainly additive in effect. However, their regression of Wr on Vr after Hayman[4] indicated the existence of full dominance. The dominance was lower in F2 than in F1. Therefore, the chances of recognising dominance were higher in F1 rather than in F2. Analysing the basic pea material used, it appears that while in the diallel crosses of Rowlands[6], Watts *et al.*[9], and Snoad and Arthur[8], only European and North American cultivars were utilised, out of 6 parents used by Snoad and Arthur[7], 4 were quite unrelated to European cultivars and these, therefore, exhibited differences in the system of genetic control of some traints. This is obvious