enzymes was as follows: for PMG 5 ml of 1% pectin (pH 5·5), 1·5 ml of phosphate citrate buffer (pH 5·5) and 1 ml of distillel water; for PG 5 ml of sodium polypectate (pH 4·5), 1·5 ml of phosphate citrate buffer (pH 4·5) and 1 ml of distilled water; for PMTE 5 ml of 1% pectin solution (pH 8·5), 1·5 ml of tris HCl buffer (pH 8·5) and 1 ml of distilled water, and for PGTE 5 ml of sodium polypectate (pH 8·5), 1·5 ml of tris HCl buffer (pH 8·5) and 1 ml of distilled water.

PME, PG, PMG, PMTE and PGTE were produced *in vitro* on glucose pottassium nitrate medium which indicates that the pathogen produces these enzymes constitutively. PME was detected on glucose potassium nitrate medium on the 12th day but 1% pectin and CMC induced early production of this enzyme. PMC activity was maximum on glucose potassium nitrate medium (6 days incubation) but the addition of pectin and CMC reduced the activity considerably. Production of PMTE and PGTE was higher on the third day of incubation in the medium with pectin. However, addition of 1% CMC did not improve enzyme secretion as compared to the basal medium. It has been observed that the culture medium and the length of the incubation period greatly influenced the type and activity of enzymes.

Only PME, PG, and PMTE were detected *in vivo* in diseased tissue extracts. It should be noted that plant tissues also produce pectic enzymes but in the present case no enzyme could be detected in the healthy tissues of papaya fruits. A complicating factor in the detection of extracellular enzymes in tissues of certain host plants, whether healthy or diseased, is the susceptibility of these enzymes to inactivation by oxidized host constituents of which polyphenols have been particularly implicated (Byrde *et al.*[4]). PMG and PGTE could not be detected in diseasel tissues. It is known that plant cell walls contain proteins which can specifically and effectively inhibit polygalacturonases of fungal origin (Albersheim and Anderson[5]). Self-inhibitory action of the pathogens also results in the rapid disappearance of the pectic enzymes in the invaded tissues (Sadasivan *et al.*[6]).

The principal pectic enzymes involved in pathogenesis in the present case appear to be polygalacturonase and pectin methyl transeliminase as they are produced both *in vivo* and *in vitro*. All the enzymes detected *in vivo* during pathogenesis appear to be of fungal origin as none could be detected in healthy tissues.

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EFFECT OF GIBBERELLIC ACID AND COUMARIN ON THE CAROTENOID CONTENT OF CARROT (*DAUCUS CAROTA* L.) ROOT

IN animals, the carotenoids are the precursors of the vitamin A group. Carrot is a very good source for carotenoids, particularly carotene. So, it is of practical interest to see the effect of growth substances, which are used for controlling growth behaviour of plants, on the carotenoid content. The present note reports the results of investigation on the effects of GA_3 and coumarin on the carotenoid content of pre and post-treated carrot (*Daucus carota* L. cv. long orange).

In case of the pre-treatment, seeds were treated for 48 h in the GA_3 and coumarin solutions. The concentrations of GA_3 were 0, 10, 100, 250, 500 and 1,000 ppm and that of coumarin were 0, 10, 100, 250 and 500 ppm. After the soaking treatment the seeds were sown in different plots of lands. For the post-treatment untreated seeds were sown and 27 day old seedlings were selected for the application of the chemicals. Here GA_3 was applied to the individual plant in such a way that each plant of different plots received 0, 1, 5, 10, 50 and 100 μg GA_3 respectively. But the plots kept for coumarin treatments were sprayed with equal amounts of the solutions of different concentrations, *viz.*, 0, 10, 100, 250 and 500 ppm respectively. For each treatment there were three replicate plots. Roots were harvested after 75 days of sowing.

In the quantitative estimation of carotenoids the formula given by Goodwin[1] was used.

Results and Discussion

Carotenoid content of GA_3 pre-treated plants increased with the increase of GA_3 concentration up

to 250 ppm, but beyond that concentration it decreased. At the control, 250 ppm and 1,000 ppm carotenoid contents were 43·2, 61·0 and 40·0 μg/g fresh tissue respectively. In the GA_3-post-treated plants there was maximum increase in the amount at 10 μg/GA_3/plant; but higher concentrations than that resulted in a decrease of carotenoid (Table I). Increase of cartenoids at the pre-treatment concentration of 250 and the decrease of the same at 100 μg/plant were statistically significant.

TABLE I

Effect of GA_3 on the carotenoial content of carrot root

Seed pre-soaking treatment		Post-treatment	
Conc. of GA_3 ppm	Carotenoid content (μg/g fresh root* ± SEM)	Conc. of GA_3 μg/plant	Carotenoid content (μg/g fresh root* ± SEM)
0	43·2±0·57	0	43·2±0·57
10	43·8±0·11	1	43·0±1·15
100	44·6±1·01	5	44·8±0·87
250	61·0±1·21**	10	45·6±0·50
500	40·8±0·83	50	44·8±1·56
1000	40·0±0·72	100	32·8±0·41**

* Each value is the mean of three replicates.

** Significant at 1% level of probability as compared to the control.

TABLE II

Effect of coumarin on the carotenoid content of carrot root

Seed-pre-soaking treatment		Post-treatment	
Coumarin conc. ppm	Carotenoid content (μg/g fresh root* ± SEM)	Coumarin conc. ppm	Carotenoid content (μg/g fresh root ± SEM)
0	43·2±0·57	0	43·2±0·57
10	52·8+2·55	10	40·8±0·61
100	45·6±1·02	100	40·8±1·16
250	43·2±0·57	250	38·4±1·13
500	43·1±0·49	500	38·2±1·10

* Each value is the mean of three replicates.

In case of the coumarin pre-treated plants, the maximum amount of carotenoids obtained was 52·8 μg/g fresh tissue at 10 ppm. At the control the value was 43·2 μg/g. But in the coumarin post-treated plants there was gradual decrease of carotenoid content with the increase of concentration. (Table II). Increase or decrease of the carotenoid content as a result of coumarin application was not significant.

At lower concentrations of GA_3 carotenoid content increased. So, there is a possibility that with the application of GA_3 at an optimum concentration, the quality of carrots may be improved through the increase of carotenoids. Banga and De Bruyn[2] reported that carotenoid content in carrot roots primarily depended upon adequate photosynthesis. It has also been known that GA_3 is responsible for chlorosis of plants[3,4,5]. Hence, higher concentration of GA_3 may reduce the amount of carotenoids through reduction of photosynthesis as a result of degradation of chlorophyll pigment.

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GAMMA RAY INDUCED SEMI-DWARF MUTANT WITH A NEW TYPE OF LEAF ARRANGEMENT IN RICE

THE leaves in rice (*Oryza sativa*) are arranged in an alternate fashion (distichous) on the stem (culm) and each leaf has long leaf-sheath which wraps the culm partially or completely. In tall *indica* varieties, leaves are longer and drooping and in semi-dwarf, the leaves are erect, or semi-erect, but the distichous phyllotaxy is always maintained.

An induced mutant showed entirely a new pattern of phyllotaxy (Fig. 1). A comparative morphological characteristics of the mutant with semi-dwarf variety and its parent are reported here.