are used for both the inlet and the outlet for electrons and the analyser is used in the dispersion mode. The entire analyser assembly with a copper Faraday cup electron collector and the helium lamp are mounted on a 8½" dia aluminium flange fitted on to the spectrometer chamber. The spectrometer chamber can be pumped to a base vacuum of 10^{-6} torr. The spectrometer chamber region has been shielded from Earth's magnetic field using a set of Helmholtz coils.

The gas under investigation is introduced into the photoionization chamber through a fine needle valve. By slowly increasing the dispersion voltage applied between the analyser plates, the photoelectron signal obtained can be read off a ECIL electrometer amplifier. The signal can be maximised with respect to variation of the helium gas pressure, reaction gas pressure and also rotation of the inlet slit provided on the reaction chamber. We have obtained the photoelectron spectrum of argon using this spectrometer. The resolution obtained is about 0·1 eV with a count rate of 10^7 electron/sec. By reducing the slit with and employing a charge sensitive amplifier both the sensitivity and resolution can be improved. A double 127° sector analyser system can also be mounted in the same chamber to improve the resolution. A 180° spherical sector analyser[3] has been fabricated and the performance of this is now being tested. In order to satisfactorily record the UV photoelectron spectrum of a gas or vapour, we are using a channeltron electron multiplier and a locally fabricated rate meter. Details of the design of the spectrometer will be published elsewhere in the near future.

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PRODUCTION OF PECTIC ENZYMES BY *CORYNESPORA CASSIICOLA* CAUSING FRUIT ROT OF PAPAYA

The importance of pectic enzymes produced by pathogenic microorganisms has received special emphasis in studies on physiology of parasitism in plants. Pectic enzymes are implicated almost routinely as a feature of host-pathogen interaction and it is more or less accepted that pectic enzymes are responsible for both maceration and killing effects during disease development. The principle involved in maceration was at an early stage equated with protopectinase (PP) complex. But now it has been resolved into several enzymes like PME (Pectin methylesterase), PG (Polygalacturonase), PMG (Polymethylgalacturonase), PMTE (Pectin methyl transeliminase) and PGTE (Polygalacturonase transeliminase). Studies on the production of pectolytic enzymes *in vitro* have largely been done by the investigators because such studies provide information on the capacity of a pathogen to produce these enzymes. Hence pectic enzymes of *Corynespora cassiicola*, a pathogen observed to be quite virulent on papaya fruits at Jabalpur, have been investigated *in vitro* as well as *in vivo*.

The pathogen was grown on three different media at 28° C (Table I). The mycelial mats were harvested after the incubation period of 3, 6, 9, 12 and 15 days. The filtrates obtained were centrifuged at 4000 r.p.m. for 20 minutes. The clear supernatant was used as the crude enzyme. The *in vivo* studies were carried out with extracts of healthy and inoculated fruits. The crude enzyme sample was prepared by grinding 5 g of rotted/healthy fruit tissue in pestle and mortar in distilled water (15 ml) with 0·5 N NaCl (15 ml). The ground tissue extract was strained through several layers of muslin cloth and squeezed. The filtrate was centrifuged at 4000 r.p.m. for 20 minutes. The supernatant was taken as the enzyme sample.

The enzyme preparations were assayed for the presence and activity of pectic enzymes (PME, PG, PMG, PMTE and PGTE). The activity of PME was measured in terms of increase in the activity of hydrolysis of pectin which has been expressed as microequivalent of methoxyl group removed by the enzyme sample (Hancock *et al.*[1]; Agarwal *et al.*[2]). The activity of other enzyme samples was assayed by determining the loss in viscosity of the reaction mixture immediately and after 2 hrs. at 30° C following the method of Bell *et al.*[3].

The per cent enzyme actively was calculated as $V_1 - V_2/V_1 - V_w \times 100$ where V_w is the viscosity of distilled water. The composition of the reaction mixtures for 2 ml of enzyme sample for the different

TABLE I

Percentage activity of pectic enzymes in vitro *and* in vivo *secreted by* Corysnespora cassiicola *pathogenic on papaya fruits on different days of incubation*

Days of incubation	*Pectin methylestease					Polygalacturonase				
	3	6	9	12	15	3	6	9	12	15
In vitro										
Glucose potassium nitrate medium**	..	..	..	10	5	7·96	9·17	11·87	17·46	30·14
Glucose potassium nitrate with 1% pectin	10	10	15	15	10	..	..	..	3·29	..
Glucose potassium nitrate with 1% CMC	10	10	15	15	10	..	..	..	11·15	12·28
In vivo										
Healthy tissue	(No enzyme detected)									
Diseased tissue	..	..	..	5	10	10·3	35·36	45·02	40·3	18·6

Days of incubation	Polymethylgalacturonase					Pectin methyl transeliminase				
	3	6	9	12	15	3	6	9	12	15
In vitro										
Glucose potassium nitrate medium**	32·6	74·81	45	29·11	3·70	45	70·94	89·78	42·26	27·21
Glucose potassium nitrate with 1% pectin	21	27·35	8·55	6·88	5·40	98·14	60·67	48·65	47·78	40·45
Glucose potassium nitrate with 1% CMC	17·1	18·57	19·93	28·39	21·17	47·74	48·51	77·23	51·57	45
In vivo										
Healthy tissue	(No enzyme detected)									
Diseased tissue	..	..	..	..	..	3·2	18·3	39·06	33·5	6·5

Days of incubation	Polygalacturonase transeliminase				
	3	6	9	12	15
In vitro					
Glucose potassium nitrate medium **	43·66	39·3	34	14·75	11·14
Glucose potassium nitrate with 1% pectin	58·3	25·35	20·61	15·24	3·57
Glucose potassium nitrate with 1% CMC	38·0	29·8	26·9	20	15·5
In vivo					
Healthy tissue	(No enzyme detected)				
Diseased tissue	..	..	..	..	..

* denotes μ mg methoxyl groups removed by 2 ml enzyme preparation in 2 hours.

** Glucose 5 gm KNO_3 3·5 gm; KH_2PO_4 1·75 gm; Mg $SO_4.H_2O$ 0·75 gm; distilled water; 1000 ml.

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