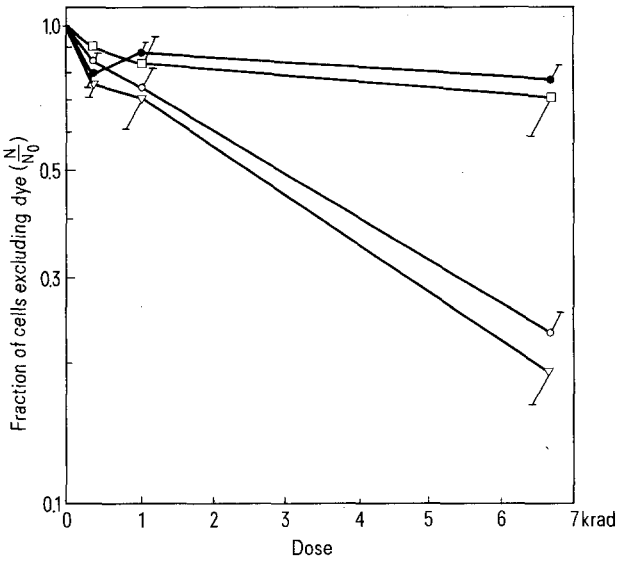
ed thymocytes. As this can be abolished by the specific blocker Propanolol, it is suggested that the protective action of Isoproterenol is mediated by the β-adrenergic receptor. To test whether c-AMP is involved in this action, we tested the effect of the c-AMP phosphodiesterase inhibitor Aminophyllin on the radiation response of thymocytes; this substance shows the same effect as Isoproterenol.

Since the first common biochemical effect of both substances tested is an elevation of c-AMP levels, it is suggested that c-AMP, or biochemical processes linked to c-AMP, interfere with the processes leading to radiation-induced loss of the capacity to exclude dye. This test is thought to reflect radiation-induced interphase death, where the membrane plays an important role probably connected with events taking place in the cytoplasm. Regarding the membrane role in the process, a recent observation has been reported where irradiation affects the structural immunoglobulins of lymphocyte plasma membranes[8]. The above statements, together with the high radiosensitivity reported for peroxidation of artificial phospholipid membranes[9], suggest that the cell membrane may be important as the primary target for some radiobiological effects on lymphocytes. Great differences in the oxygen enhancement ratios for cortisone sensitive and cortisone resistant thymocytes[10] may be interpreted, according to Alper[11], as a further indication of the cell membrane participation in radiobiological effects on lymphoid cells. In this context, the present experiments showing the correlation between induction of high c-AMP levels and radioresistance are in line with the participation of membranes, although they emphasize the interdependence of membrane and cytoplasm with regard to the cellular radiobiological response.



Fraction of thymocytes able to exclude Trypan blue after irradiation and incubation in medium containing Isoproterenol (□), Aminophyllin (●), Propanolol-Isoproterenol (△), Control without additions (○). All points are referred to a corresponding treatment without irradiation. These controls have a percentage of cells excluding dye of at least 85%.

1 Supported by International Atomic Energy Agency contract 2175/RB, PNUD Grant 102/77 and UACH Grant S-79-2.
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Presence of auxin protectors in *Eriophyes* induced *Zizyphus* stem galls

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Summary. 3 auxin protectors, which were o-dihydroxyphenols of high molecular weight, were isolated from *Zizyphus* gall tissues. An increase in polyphenol oxidase activity in the gall tissue was observed which probably led to the production of high levels of auxin protectors. This prevented IAA oxidation, resulting in hyperauxinity and auxin-autotrophy.

There are many conflicting reports regarding the reasons for abnormal growth in plants, and the responses they show, in terms of altered hormone metabolism[1]. However, one feature which is common to almost all tumors is the simplification of growth requirements and the presence of high levels of endogenous growth substances. This communication is aimed at suggesting that unregulated synthesis of auxin protectors is responsible for hyperauxinity and auxin-autotrophy in *Zizyphus* gall tissues. Auxin protectors were first reported in *Pharbitis nil*[2]. In the present studies 3 auxin

Polyphenol oxidase activity (assayed by the method of Ponting and Joslyn[8]) in normal and gall tissues

	Normal	Gall (days)				
		10	20	30	40	50
Polyphenol oxidase activity (A/min/g fresh wt)	2.0±0.01	2.2±0.02	2.8±0.02	3.2±0.01	2.6±0.02	2.2±0.02

Values are means ± SE.

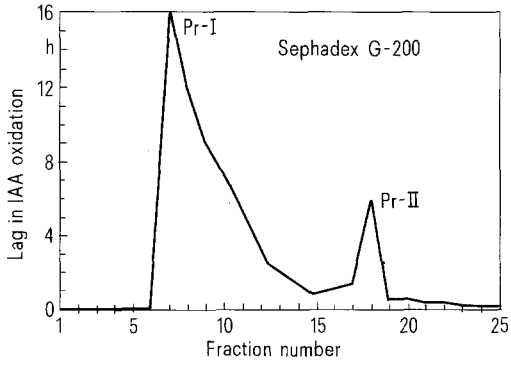

Fig. 1. Lag in IAA oxidation by various fractions obtained by filtration of gall tissue extract through Sephadex G-200. 1.0 ml of the gall extract (250 mg fresh wt/ml of potassium phosphate buffer pH 6.1) was loaded onto a Sephadex G-200 column and eluted with buffer. 5-ml fractions were collected. 0.2 ml of each fraction was incubated at 37 °C in 10 ml IAA reaction mixture (0.1 mM each of IAA, manganese chloride and 2,4-dichlorophenol, and 0.25 µg/ml of horse radish peroxidase). 0.5-ml samples were removed from the above reaction mixture at various time intervals and the amount of IAA oxidized was assayed by the method of Gordon and Weber[7].

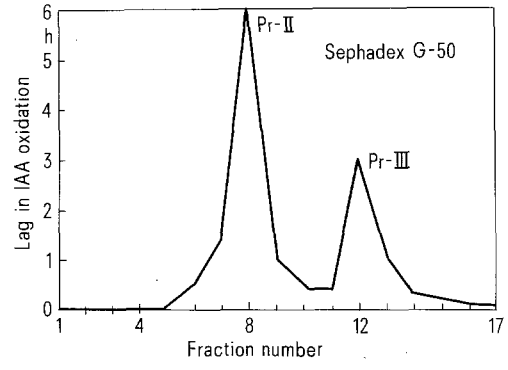

Fig. 2. Same as in figure 1, except that Sephadex G-50 column was used.

protectors were isolated from *Zizyphus jujuba* Lamk. stem galls, incited by a plant mite, *Eriophyes cernuus* Massee. One of these, Pr-I, had a mol.wt exceeding 200,000 daltons, and of the other two, Pr-II had a mol.wt of 10,000 and Pr-III of 2000 daltons approximately. It is believed that these substances may be polymers or oligomers of a small mol.wt substance[3]. The auxin protectors prevented the destruction of indole-3-acetic acid (IAA) by the enzymes normally occurring in the tissues (figures 1 and 2). The protectors were oxidized first; during this period a lag in IAA-oxidation was observed. Only when the concentration of the protector dropped was IAA oxidized at a rate similar to that in the control lacking protectors (figure 3). The possible mechanisms of action of the protectors could be that a) they donate electrons more rapidly than IAA or replenish electrons lost by IAA, thus converting themselves in to an oxidized state[3], or b) they react preferentially with spectroscopically distinct forms of peroxidase known as compound II and compound III involved in IAA oxidation by peroxidase[4].

The auxin protectors from *Zizyphus* gall tissue were considered to be o-dihydroxyphenols, as also reported in *Pharbitis* shoots[3], based on the following evidence: 1. the chromatography of the protectors showed a blue fluorescing spot (R_f 0.43) in UV-light. On spraying the spots with sucrose suspension the spots gave a light brown colour characteristic of diphenols[5]; 2. they were rendered ineffective when incubated with polyphenol oxidase (figure 3); 3. the lag period prior to IAA oxidation was mimicked by diphenols (figure 3), and 4. the UV absorption spectra of both the oxidized auxin protectors and diphenols were similar and showed a loss in UV absorbing chromophores.

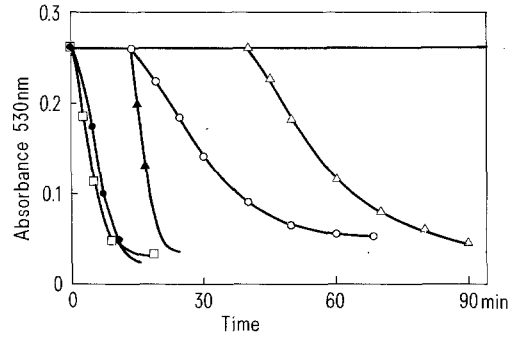

Fig. 3. Effect of PPO on Pr-III and catechol induced lags in IAA oxidation. Oxidation of IAA by horse radish peroxidase (□), and in the presence of Pr-III (○) and catechol (△). Respective solid symbols represent the samples incubated with PPO (100 units of PPO per ml of protector or 50 mg/ml of catechol in phosphate buffer were incubated at 25 °C for 1 h and then filtered through a Sephadex G-50 column; 0.5 ml of eluate was used for assay).

It is well known that synthesis of phenolic compounds is regulated by the enzyme polyphenol oxidase E.C. 1.10.3.1 (PPO) which is able to convert monophenols (electron acceptors) to o-diphenols (electron donors) and the latter to o-quinones (strong electron acceptors). As shown in the table, the PPO activity increased up to the 30th day of gall growth, which presumably resulted in increased production of auxin protectors which in turn prevented auxin destruction. Once the growth autonomy of the gall tissue was accomplished the PPO activity declined from the 30th day onward, probably to prevent conversion of diphenols to quinones. It is therefore suggested that in abnormal growth PPO is the key enzyme which regulates the monophenol-diphenol-peroxidase system responsible for auxin destruction.

In our earlier communication[6] it was reported that high concentrations of α-naphthalene acetic acid, gall tissue extract, and gall callus graft induced 'galls' on *Zizyphus* stem segments cultured on MS medium. It seems likely that the increased synthesis of auxin protectors in the tissue can result in hyperauxinity and auxin-autotrophy.

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