duction of dictyol D to a dihydrodehydroxy derivative (M^+/e 290) which was identical (GLC, MS and NMR) with 10,18-dihydropachydictyol A, prepared from authentic **1** by the same treatment. The ^{13}C chemical shifts assignment for **5** was straightforward. The upfield shifts of the resonances of carbons 7 and 9 (ca 3 and 5 ppm, respectively) in comparison with those of the corresponding carbon atoms in **1** are probably due to the crowded environment caused by the presence of the hydroxyl group at C-2, which constrains the 7-membered ring in a somewhat rigid conformation. The stereochemistry at C-2 was deduced from the ^1H-NMR-spectra after gradual addition of $Eu(fod)_3$: since 5-H experiences a paramagnetic shift larger than 1-H, it must be nearer to the hydroxyl group [$\Delta\delta\ ^{n=0.5}_{Eu}$ 3.23 (2-H), 2.33 (5-H), 1.68 (3-H), 1.51 (18′-H), 1.41 (1-H), 0.99 (6-H) and 0.81 (18″-H)]; as already observed for dictyol C, the complex formation at 6-OH is suppressed by steric hindrance.

Dictyol E (**6**), oily product, $[\alpha]_D$ + 26.8 °C (c 1, $CHCl_3$), has molecular formula $C_{20}H_{32}O_2$ (accurate mass measurement). In its mass spectrum diagnostically important peaks are seen at m/e 286 (21%, M^+-H_2O), 268 (5, M^+-$2H_2O$), 177 (6, M^+-side chain), 175 (11, M^+-2H-side chain), 157 (24, M^+-H_2O-side chain) and 155 (10, M^+-H_2O-2H-side chain). The salient difference between the ^1H-NMR-spectrum of **6** and that of **1** resides in the absence of the methyl doublet at δ 0.97, which is replaced by a 3H singlet at δ 1.23. This located the hydroxyl group in the side chain and led to formulation **6** for dictyol E. The ^{13}C-NMR-data (table 2) definitely confirmed the proposed structure and permitted the assignment of the stereochemistry at the chiral centres in the nucleus.

Effects of increased intracellular Ca^{2+} on cyclic nucleotides production by liver tissue[1]

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Summary. During hepatointoxication, the increase of intracellular Ca^{2+} is accompanied by an increase of cAMP. This reversible phenomenon suggests that the production of cAMP is likely to be a response of the cell in order to activate the exclusion of Ca^{2+}.

Calcium ion and cyclic 3′,5′-adenosine monophosphate (cAMP) are interrelated in the control of a variety of functions of the mammalian cell[3,4]. They are considered the second messenger which translates event at the cell surface into modified activity within the cell[5]. On the other hand, there is evidence that cyclic 3′,5′-guanosine monophosphate (cGMP) is also involved in the regulation of a number of metabolic and mitogenic processes[6].

A convenient model for the study of the in vivo effects of Ca^{2+} concentration on cAMP and cGMP syntheses is the acute hepatointoxication with thioacetamide (TAA) which produces a reversible manifold increase in intracellular Ca^{2+}[7]. On this basis, and in order to correlate the variations of intracellular liver calcium with the changes in rate of cAMP and cGMP syntheses, male Wistar rats (body weight: 200–240 g) were given orally 100 mg/kg b.wt of TAA as a 2% aqueous solution. 5-animal-groups were sacrificed at different times after TAA administration. The livers were sliced with scissors and aliquots of the sliced tissue were ashed at 700 °C, the ashes dissolved in 1 N HCl, and calcium and magnesium determined by atomic absorption spectrometry. In addition to this, 1 g samples of liver tissue were homogenized and deproteinized with trichloroacetic acid[8]. Cyclic AMP and cGMP were determined using radioimmunoassays kits obtained from The Radiochemical Centre, Amersham, England. Similar determinations were performed on liver tumors (cholangiocarcinomas) induced by prolonged feeding of rats with 4-dimethylaminoazobenzene[9].

The administration of TAA provoked a reversible increase of calcium in liver tissue with a maximum of approximately 24 times the normal value at 24 h after the administration of the hepatotoxic substance (figure 1). The concentration of cAMP showed a fairly similar pattern of increase, but its maximum value was observed after 48 h. At this moment, the concentration of Ca^{2+} was only 4

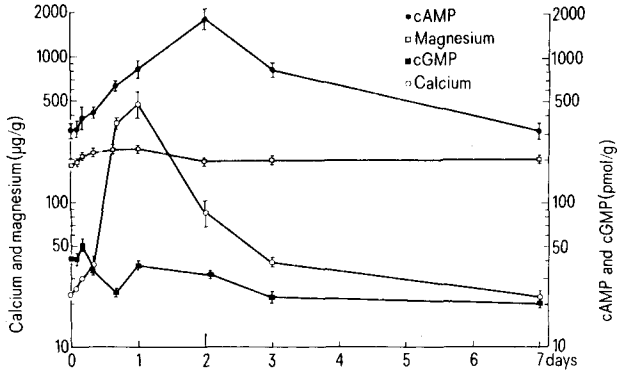

Fig. 1. Calcium, magnesium, cAMP and cGMP concentrations in liver of rats given thioacetamide. Each point corresponds to the mean value ± SE of a 5-animal-group.

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times higher than the normal one. The level of cGMP was slightly affected by the increase in Ca^{2+} concentration and presented an almost steady decrease throughout the experiment. In addition, the concentration of Mg^{2+} showed a small increase which was coincidental with the maximal increase in calcium content. The assayed liver tumors have shown increased Ca^{2+} and cAMP concentrations while Mg^{2+} and cGMP concentrations were equal or lower than the corresponding value in normal liver tissue (figure 2).

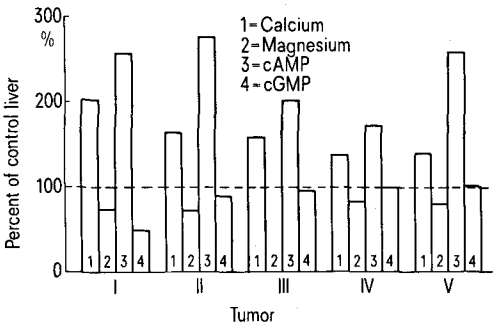

Fig. 2. Calcium, magnesium, cAMP and cGMP concentrations (as percent of the corresponding value in normal liver tissue) in 5 primary liver tumors (cholangiocarcinoma) induced in rats by 4-dimethylaminoazobenzene feeding.

These results appear to indicate a direct relationship between Ca^{2+} concentration and cAMP synthesis. There is evidence that TAA produces a change in cell membrane permeability leading to increased influx of Ca^{2+}[10]. As shown in figure 1, during the first 8 h after TAA administration, the concentrations of Ca^{2+} and cAMP increase at a fairly similar rate. This seems to indicate an increase in both Ca^{2+} permeability and adenylate cyclase activity. The observation that after that point the concentration of Ca^{2+} decreases faster than the concentration of cAMP appears related to the suggested role of cAMP in the regulation of the changes in Ca^{2+} concentration in the cytoplasm[3] by increasing the rate of Ca^{2+} efflux from the cells[5,11]. The observation that cAMP-dependent protein kinase modulates calcium transport by the cardiac sarcoplasmic reticulum[12] suppors that hypothesis.

It seems likely that under these experimental conditions the buildup of cAMP forms part of a cellular mechanism, preventing the uptake of Ca^{2+} into intracellular pools which could provoke a permanent cell damage. It is worth noting that in primary liver tumors a similar Ca^{2+}-cAMP interrelation was observed.

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Occurrence of oxalyl-CoA synthetase in Indian pulses

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Summary. The presence of oxalyl-CoA synthetase was observed in common edible pulses. Excepting in chick pea, the changes in oxalyl-CoA synthetase activity of winter pulses proceeded in stages. The enzyme remained more active in late strains than in early strains of winter pulses. Unlike the activity of the enzyme in winter pulses, that in summer pulses behaved differently.

ATP- and CoA-dependent decarboxylation of oxalate to formate by extracts of a number of plant tissues was first reported by Giovanelli and Tobin[1]. The enzyme catalyzing the reaction – oxalate → oxalyl-CoA – was partially purified (6fold) from pea seeds and its properties were studied. This enzyme was referred to oxalyl-CoA synthetase by the trivial name (or by the systematic name of oxalate-CoA ligase (AMP). Although the pea seeds yielded the most active preparations, the enzyme was also detected in the seeds of lupin, pumkin and in wheat germs[2]. Subsequently, the enzyme was found to be very active in the crude extracts of Lathyrus sativus seeds[3] and was shown to be responsible for the synthesis of β-N-oxalyl, L-α,β-diamino propionic acid (the L. sativus neurotoxin) in condensation with L-α,β-diamino propionic acid in the presence of ODAP synthase[4]. The present communication relates to the occurrence and the activity of oxalyl-CoA synthetase in common Indian pulses with the aging of the seedling.

Materials and methods. Early and late strains of pulses grown in winter, i.e. as post monsoon crops (chick pea, pea, lentil and chickling pea), and those grown in summer, i.e. as pre-monsoon crops (pigean pea, green gram, cowpea and soybean), were collected from the Division of Plant Introduction, IARI, New Delhi-12 (India). Germination of the seeds and the narturing of seedlings were carried out as described by Barat et al.[5]. Samplings collected after the desired period of time were washed with distilled water, pressed softly between 2 filterpaper sheets and then chilled in ice. They were then used as experimental materials. Procedure reported by Malathi et al.[3] was followed for the assay of the enzyme. Protein was estimated by the Lowry et al. method[6]. The activity was expressed in terms of μmoles of acetyl phosphate/mg of protein.

Results and discussion. The activity of oxalyl-CoA synthetase in winter and summer pulses is presented in the table. Data reveal that the presence of oxalyl-CoA synthetase was detected in winter-(chickling pea, pea, lentil and chick pea) as well as in summer-(pigean pea, soybean, green gram and cowpea) pulses. Irrespective of pulse crops and

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