SHORT COMMUNICATIONS

CATALYTIC DECOMPOSITION OF HYDROGEN PEROXIDE BY γ-IRRADIATED SALTS

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γ-Radiolysis of water or aqueous solutions of organic or inorganic substrates gives rise to hydrogen peroxide as one of the molecular species[1]. Many oxides with perovskite structures are known[2–4] to catalyze the decomposition of H_2O_2. These are heterogeneous catalysts and participate in oxidation reactions due to their favourable crystal structure permitting oxygen lability. However, the catalytic decomposition of hydrogen peroxide by γ-irradiated NaCl and Li_2SO_4, reported here appears to be interesting since it occurs in the homogeneous phase. Results of the kind do not seem to have been reported earlier.

A solution of H_2O_2 (5 ml) of about 0.74 M concentration was taken in a Warburg manometer flask. To this were added 0.5, 1.0 and 1.5 g γ-irradiated NaCl, or γ-irradiated Li_2SO_4 (exposed to a γ-dose of 100 kGy) at 24°C and the amount of oxygen evolved was measured at different time intervals by the standard procedure[5]. A blank was run every time using the same amount of H_2O_2 and unirradiated NaCl or Li_2SO_4 and the reading subtracted to obtain the contribution due to the *irradiated* salts. The volumes of the oxygen evolved were recorded after 30 min to eliminate the initial contraction of volume due to cooling effect following salt dissolution. The oxygen volume, corrected to STP, was proportional to the amount of H_2O_2 decomposed. The plot of μmol of H_2O_2 decomposed as a function of time was linear (figure 1). The computer calculated slope by the least square method gives the rate constant of the reaction. The results shown in table 1 are the average of three experiments for each sample.

The results of figure 1 (and table 1) show that the rate of decomposition of H_2O_2 is a zero-order reaction with the rate constant varying over the range 0.5 to 7×10^{-4} μmol sec^{-1}. It may be further noted that the salts irradiated to a higher dose give a larger rate constant. It was further observed that the reaction rate (R) depended both on the amount of the catalyst (irradiated salt) and the radiation dose (D) given to it. These results lead to the rate expression.

$$R = k\,[\text{catalyst}]\,D. \qquad (1)$$

As suggested by Ahnström[6], and confirmed subsequently[7,8] NaCl crystals on dissolution produce hydrated electrons from F centres and $Cl^{\bullet}$ from hole centres. A part of the $Cl^{\bullet}$ radicals combine to give Cl_2, rather the unstable species with the distended bond Cl....Cl as envisaged by Haward *et al*[9] and

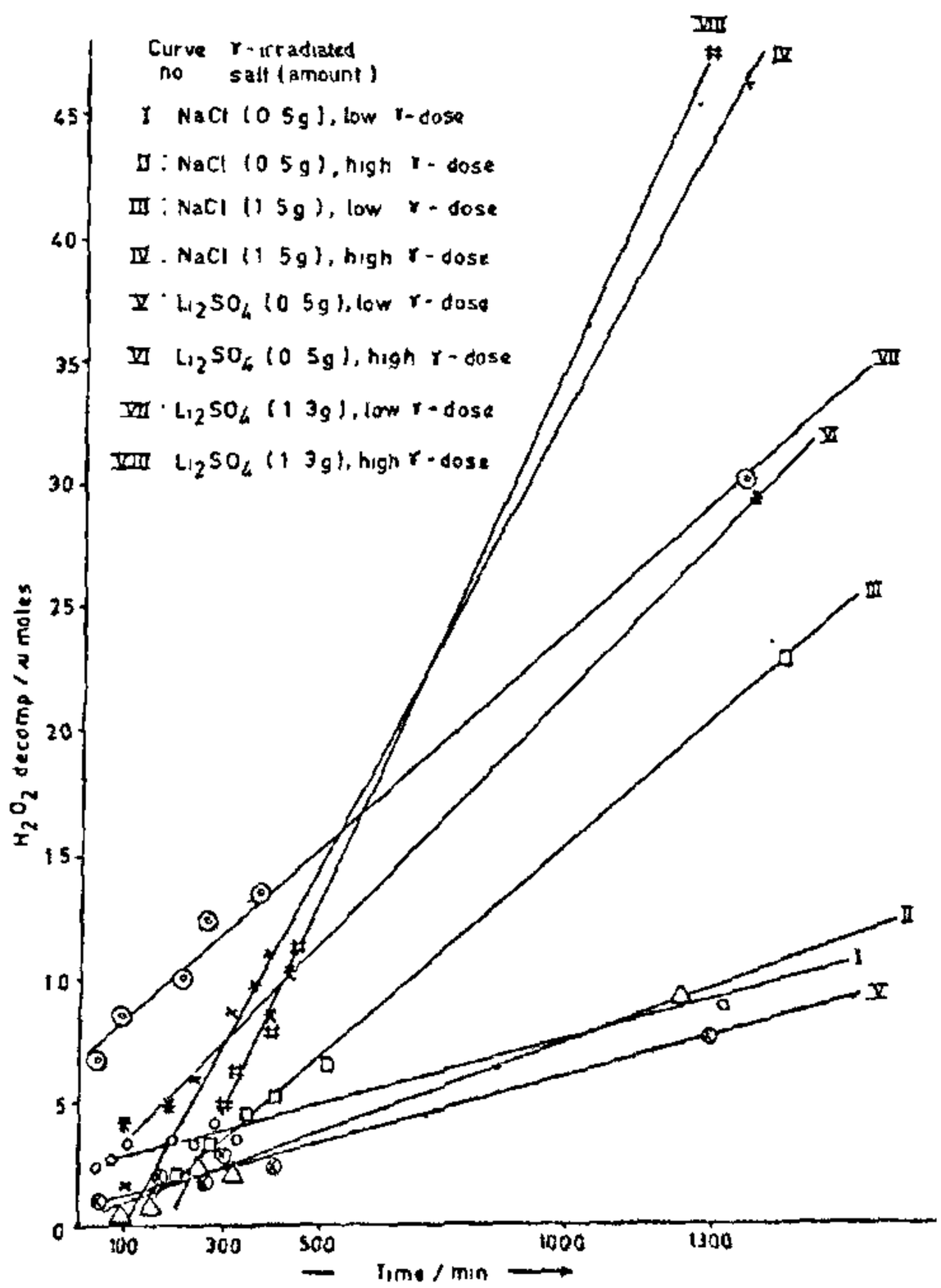

Figure 1. Plot of H_2O_2 decomposed (μmol) vs time (min).

Table 1 *Reaction constant of the decomposition of H_2O_2 by γ-irradiated salts*

Amount of γ-irradiated salt/g	γ-irradiated NaCl		γ-irradiated Li_2SO_4	
	~50 kGy γ-dose	~100 kGy γ-dose	~50 kGy γ-dose	~100 kGy γ-dose
0.5	0.75	1.46	0.79	3.21
1.0	2.09	5.60	1.19	3.54
1.5	3.16	5.70	3.17	7.07

Will ar[illegible]. This species reacts with H_2O_2 liberating oxygen according to the mechanism reported earlier[11].

$$Cl\ldots Cl + H_2O_2 \longrightarrow HCl + H\text{–}O\text{–}O\text{–}Cl \quad (2)$$

$$H\text{-}O\ O\text{-}Cl \longrightarrow HCl + O_2. \quad (3)$$

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INHIBITION OF RAT BRAIN SUCCINATE DEHYDROGENASE BY CARBAMATE AND ORGANOPHOSPHATE PESTICIDES

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ORAL administration of carbaryl and bavistin (Carbamate pesticides) and phosalone and elsan (organophosphate pesticides) has been found to inhibit rat brain succinate dehydrogenase activity significantly.

For acute effects

Normal, adult albino rats weighing 150–250 g were fed single dose of these pesticides orally along with groundnut oil at 50% of LD_{50}, (calculated according to body weight) and were sacrificed after 1 hr. The reported LD_{50} values for the pesticides are: carbaryl: 400–500 mg; bavistin: 6400 mg, phosalone. 120 mg and elsan: 350 mg per kg body weight[1–3].

For chronic effects

Weanling albino rats weighing 50–70 g were daily given pesticides at 5% of LD_{50} orally along with groundnut oil. Administration of pesticide was continued separately in four groups for 15 days and in the remaining four groups for 60 days and thereafter these were sacrificed.

Control groups were run simultaneously for each study. All the animals were maintained on Hindustan Gold Mohr rat feed.

Brain was immediately removed after sacrifice, washed in chilled normal saline and homogenized in isotonic solution (0.25 M sucrose, 0.01 M TRIS, 0.001 M EDTA) to form 10% homogenate. The cellular debris was removed by centrifugation at $700 \times g$ for 5 min. Succinate dehydrogenase (SD) activity was estimated in the supernatant according to Kun and Abood[4]. Protein in the supernatant was estimated according to Lowry[5].

The specific activity of brain SD in weanling rats was found to increase with age; however, in adult rats it showed decline (table 1). At the end of 1 hr after administration of 50% of LD_{50} of each pesticide, both the organophosphate pesticides (elsan and phosalone) inhibited brain SD significantly (15 and 27.3% respectively, $P < 0.05$). Carbamates (carbaryl and bavistin) were also found to exhibit inhibitory trend when fed at the same levels but it was not significant (7.6 and 14.7% respectively, $P > 0.05$).

Regular administration of these pesticides at one-tenth level of LD_{50} for 15 days (chronic administration) revealed significant inhibition of SD by phosalone (17.8%, $P < 0.01$) and carbaryl (14.1%, $P < 0.05$). Chronic administration of elsan and bavistin for the same period did not show much effect.

Chronic administration of all the four pesticides up to 60 days exhibited highly significant inhibition of SD ranging from 27.3 to 47.5%. Carbaryl was found to exert only 20.3% inhibition (table 1).

Inhibition of SD which is a very important enzyme of TCA-cycle and forms one of the steps of energy production site is bound to inhibit the whole cycle in the brain tissue and thus lower the energy output. This ought to inhibit the operation of electrochemical changes which all are endergonic reactions, making