contact the surface of TiO_2, recombination of oxygen atoms occurred thus minimizing the rate of uptake of nitrogen and causing the increased evolution of oxygen.

In conclusion, it is suggested that molecular nitrogen reacts with atomic oxygen originating from hydrogen peroxide, as the interaction of molecular nitrogen with hydroxyl radicals is energetically very unfavourable[6]. The quantity of NO formed is directly related to the pressure of nitrogen as well as the concentration of hydrogen peroxide on the surface of TiO_2.

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ACTIVITIES OF ALDOLASE, LACTATE DEHYDROGENASE, GLUCOSE-6-PHOSPHATASE AND ARGINASE IN PERCHLORATE TOXICITY

P. SANGAN and D. B. MOTLAG
Department of Biochemistry, University of Madras, Madras 600 025, India.

PERCHLORATE is one of the toxic effluents in space research. Addition of perchlorate to the medium has been observed to reduce the growth rate of microorganisms[1,2]. Perchlorates as potassium or ammonium salt caused decreased food intake and loss of body weights in rats[3–5]. Ammonium perchlorate administered to chicks in their food was found to increase the glycolysis in erythrocytes[6]. Although these studies could prove the toxic effects of perchlorate, a study on the perchlorate toxicity in relation to carbohydrate metabolism and protein catabolism has not been carried out so far. It is, therefore, proposed to study the effect of perchlorate feeding to rats on the blood glucose level (a parameter of carbohydrate metabolism) and blood urea level (a parameter of protein catabolism). A few enzymes related to these parameters were also studied. In order to make a comparative study, potassium and ammonium perchlorates were used in the present investigation.

Weanling albino rats derived from the Wistar strain were purchased from Veterinary College, Madras. Glucose-6-phosphate, fructose-1,6-diphosphate and nicotinamide adenine dinucleotide were obtained from V.P. Chest Institute, Delhi. Bovine serum albumin was the product of Fluka Buchs, Switzerland. Sodium pyruvate was obtained from E. Merck, W. Germany. L-arginine monohydrochloride was purchased from Koch-Light, England. DL-glyceraldehyde was obtained from Sigma Chemical Co., St. Louis, USA. All other chemicals used were of analytical grade.

The animals were fed with commercial rat feed with paired feeding and water *ad libitum* along with oral administration (500 mg/kg body weight/day) of potassium or ammonium perchlorate for 45 days (chronic treatment). The dosage was selected based on the report of Spreca *et al*[7]. The animals were then sacrificed by stunning and decapitating and the blood was collected from jugular vein with potassium oxalate as an anticoagulant. Blood glucose was estimated by the method of Dubowski[8] modified by Sasaki and Matsui[9] and urea was estimated according to the method described by Geyer and Dabich[10]. The liver, kidney and intestine were dissected out immediately, washed with ice-cold saline and stored in ice. The appropriate amounts of the tissues were taken and homogenized in 0.1 M tris-HCl buffer pH 7.4. The homogenates were centrifuged at 2,500 rpm for 10 min at 4°C. The supernatants were used as the enzyme source. Aldolase and lactate dehydrogenase were assayed according to the methods prescribed by King[11]. Glucose-6-phosphatase and arginase were assayed by the methods of

Koide and Oda[12] and Herzfeld and Raper[13] respectively. The method of Lowry *et al*[14] was followed for protein estimation.

Table 1 shows the blood glucose and urea levels in perchlorate-treated and control rats. A significant decrease in the blood glucose level ($P < 0.001$) and an increase in the blood urea level were observed in perchlorate-treated rats when compared to these levels observed in control rats. The specific activities of aldolase, lactate dehydrogenase, glucose-6-phosphatase and arginase in both experimental and control rats are shown in table 2.

The specific activities of aldolase and lactate dehydrogenase were found to increase significantly in perchlorate-treated rats. The activity of the enzyme aldolase is a measure of the extent of glycolysis in a given tissue[15]. Ammonium perchlorate administered to chicks in their food increased the glycolysis in erythrocytes[6]. The increased specific activities of these two glycolytic enzymes indicate that the process of glycolysis is enhanced in the perchlorate-treated rats leading to a decreased blood glucose level. Glucose-6-phosphatase in the liver is one of the factors responsible for maintaining blood glucose. The glucose-6-phosphatase activity in perchlorate-treated rats was found to decrease significantly ($P < 0.001$) than that of control rats. The decreased glucose-6-phosphatase activity may be one of the reasons for the decreased blood glucose level in the perchlorate-treated rats.

An increase in blood urea levels was observed in the case of rats treated with perchlorate (table 1). The perchlorate treatment was also found to increase the activity of liver arginase ($P < 0.001$)

Table 1 *Blood glucose and urea levels in control and perchlorate-treated rats. The values are given as mean ± S.D. for six animals in each group*

Constituents	Control	Potassium perchlorate treated	Ammonium perchlorate treated
Glucose (mg/dl)	89.45 ± 3.83	61.87 ± 4.39[a]	53.1 ± 7.85[a]
Urea (mg/dl)	29.6 ± 3.5	39.0 ± 2.81[a]	36.2 ± 3.1[b]

[a] $P < 0.001$; [b] $P < 0.01$

Table 2 *Enzymes levels in control and perchlorate-treated rats. Aldolase and lactate dehydrogenase activities are expressed as nanomoles and glucose-6-phosphatase and arginase activities are expressed as micromoles of product liberated per mg of protein per hour under incubation condition. The values are given as mean ± S.D. for six animals in each group*

Enzyme	Organ	Control	Potassium perchlorate treated	Ammonium perchlorate treated
Aldolase	Liver	622.86 ± 5.60	659.95 ± 11.31[a]	661.0 ± 15.05[a]
	Kidney	637.74 ± 8.30	674.38 ± 8.3[a]	688.0 ± 17.23[a]
	Intestine	219.1 ± 4.62	248.85 ± 5.43[a]	246.0 ± 6.20[a]
LDH	Liver	801.8 ± 4.41	839.04 ± 8.48[a]	845.48 ± 8.48[a]
	Kidney	720.88 ± 8.53	754.52 ± 11.58[a]	762.14 ± 11.04[a]
	Intestine	709.04 ± 17.02	733.6 ± 15.09[b]	735.86 ± 14.9[b]
G-6-Pase	Liver	2.232 ± 0.062	1.875 ± 0.036[a]	1.729 ± 0.034[a]
	Kidney	1.302 ± 0.023	1.334 ± 0.029[a]	1.297 ± 0.030[a]
	Intestine	0.404 ± 0.010	0.237 ± 0.011[a]	0.229 ± 0.008[a]
Arginase	Liver	8.389 ± 0.098	11.600 ± 0.290[a]	10.478 ± 0.110[a]

[a] $P < 0.001$; [b] $P < 0.05$

(table 2). Thus increased arginase levels may be the principal cause for the increased blood urea levels in perchlorate-treated rats.

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LATE HOLOCENE EVIDENCE OF NEOTECTONICS IN THE UPPER VASHISHTHI VALLEY (WESTERN MAHARASHTRA)

V. S. KALE, S. N. RAJAGURU* and G. RAJAGOPALAN**
Department of Geography, University of Poona, Pune 411 007, India.
**Department of Archaeology, Deccan College, Pune 411 006, India.*
*** Birbal Sahni Institute of Palaeobotany, Lucknow 226 007, India.*

THE Koyna-Pophali region, coinciding with the headwaters of the Vashishthi river, is a known earthquake prone region. Gravity data indicate possible fault to the west of Koyna, in Pophali area and between Rampur and Guhagar, indicating a tectonic sag in the Koyna area[1]. A number of thermal springs and strong negative anomalies in the Western Ghat section and more strong positive anomalies in the coastal tract corraborate this inference and suggest a zone of tectonic impress. The seismo-tectonic status of the area is also reflected in the anomalous channel characteristics of the Vashishthi river.

The channel of upper Vashishthi exhibits a braided pattern, with bars in the upstream and strongly braided channel in the downstream. The width/depth ratio near Pophali, is > 49 and the channel actively cuts its banks. Despite the braided planform, the main feature of the channel is incision and terrace formation. The lithosection (figure 1) observed at the site reveals, the predominance of ungraded bouldery-cobbly-pebbly gravel (1.8–2.1 m in thickness and > 200 m width) which is in fact, a misfit in the present humid morphogenetic environment. The textural and the sedimentological properties of the gravel suggest excessive local aggradation from the Ghat slopes as well as the incompetence of the river to transport the sediments downstream[2].

The great influx of cobbly-pebbly sediments indicates an adjustment in geomorphic regimen from three possible sources, namely catastropic floods, landslides, and tectonic movements. Floods of unusual magnitude in the Vashishthi headwaters will cause vigourous erosion and incision in such a hilly terrain[3]. Similarly, a pluvial phase will result in accelerated chemical weathering and increased supply of finer sediments. In response to such a change in the load, the river will be transformed