ACTIVITIES OF GLUCOSE-6-PHOSPHATE DEHYDROGENASE AND PHOSPHO GLUCOSE ISOMERASE IN IRON DEFICIENCY IN RATS

D. C. SHARMA* AND M. M. SIMLOT
Department of Biochemistry, R. N. T. Medical College, & Rajasthan College of Agriculture, Udaipur 313 001, India.
*Present address: Department of Biochemistry, J. L. N. Medical College, Ajmer 305 001, India.

THE activities of enzymes glucose-6-phosphate dehydrogenase (G-6-PD; E. C. 1.1.1.49) and phospho glucose isomerase (PGI; E. C. 5.3.1.9) of carbohydrate metabolism may determine the utilisation of available glucose-6-phosphate either through Embden-Meyerhof-Parnas (EMP) pathway or hexose mono phosphate (HMP) shunt[1]. Complete inhibition of the G-6-PD activity has been reported[2,3] in iron-deficient litters of rats. They had also reported a slight decrease in the activity of PGI[2]. The present study in which dietary iron deficiency was produced in normal adult rats by a method different from that reported earlier[2], has shown a decrease in the G-6-PD activity and an increase in the PGI activity.

Male albino rats (4 weeks old) of Norway strain (weighing 30-40 g) were obtained from Haffkine Institute, Bombay. Severe iron deficiency anemia was produced in them in 8 weeks by feeding iron-deficient synthetic diet[4] and was confirmed by blood haemoglobin and plasma iron determination[5]. Fifteen iron-deficient anemic rats, together with an equal number of litter-mates (control group), were then starved for 12 hr, anesthetized by intraperitoneal injection of nembutal, the liver was removed, chilled in crushed ice, blotted and homogenized in 0.14M KCl in a Potter-Elvehjem homogeniser. The homogenate was centrifuged in cold for 10 minutes at 3000 rpm. The activities of G-6-PD and PGI were determined in the supernatant by standard methods[6,7]

The results are shown in table 1, which reveal that in iron deficiency the average activity of G-6-PD decreased by 77% while that of PGI increased by 15%. These results vary with those of Srivastava *et al*[2,3], who observed a complete loss of activity of G-6-PD. It may be due to difference in the severety of iron deficiency induced. Of the two pathways of glucose utilisation, it has been reported that HMP shunt consumes 55% of the available glucose in normal rat liver[8]. Thus any imbalance or deviation from this ratio should proportionally adjust the utilisation by the other pathway if glucose consumption remains unaffected as carbohydrate absorption had remained unaltered in our iron deficient rats[9]. Considerable loss of G-6-PD activity may also affect the availability of pentoses for nucleic acid synthesis as has been observed in the lowering of DNA content of the tissues in iron deficient rats[10].

TABLE 1

Hepatic glucose-6-phosphate dehydrogenase and phospho glucose isomerase activities in iron deficient rats.

	G-6-PD[a]	PGI[b]
Control group	469 ± 20	62 ± 3.9
Fe-deficient group	107 ± 13	71.6 ± 7.9
% of control	23	115
P	<0.001	<0.001

[a] nmol NADPH formed/min at 20° C per 1 g of fresh liver.
[b] μmol of fructose-6-phosphate formed in 30 min at 37° C per 1 g of fresh liver.
The values of enzyme activities are mean ± S.E. of 15 determinations on different rats.

6 March 1982; Revised 10 September 1982

1. Noltman, E. A. and Kuby, S. A., In *Enzymes*, Vol. 7, 2nd ed., (Eds. P. D. Boyer, H. Lardy and K. Myrback), Academic Press, New York, 1963, p. 238.
2. Srivastava, S. K., Sanwal, G. G. and Tewari, K. K., *Indian J. Biochem. Biophys.* 1965, **2**, 257.
3. Srivastava, S. K., Zaheer, N. and Krishnan, P. S., *Arch. Biochem. Biophys.*, 1964, **105**, 446.
4. Sharma, D. C., Singh, P. P. and Simlot, M. M., *Br. J. Nutr.*, 1972, **28**, 201.
5. Sharma, D. C., Singh P. P. and Khalsa, J. K., *Clin. Biochem.*, 1969, **2**, 439.
6. Glock, G. E. and McLean, P., *Biochem. J.*, 1953, **55**, 400.
7. Bodansky, O. and Calitri, D., *Cancer*, 1954, **7**, 1191.
8. Murphy, J. R. and Muntz, J. A., *J. Biol. Chem.*, 1957, **224**, 987.
9. Sharma, D. C., Singh, P. P. and Simlot, M. M., *Am. J. Dig. Dis.*, 1973, **18**, 73.
10. Canale, V. and Lanzkowsky, P., *Br. J. Haemat.*, 1970, **19**, 579.