

INFLUENCE OF SIMULTANEOUS SUPPLEMENTATION OF JAGGERY ON LEAD INTOXICATION IN RATS

S.J.S. Flora* and Surendra Singh

Industrial Toxicology Research Centre, P.O. Box 80, Lucknow-226001, India

(Received September 23, 1988; revised November 13, 1988)

Beneficial effects of gur (Jaggery), a solidified product of sugarcane juice, on lead induced biochemical changes in blood and urine and on lead absorption in blood and tissues were investigated in rats. A daily single oral administration of gur along with lead for six weeks restored the inhibited activity of δ -aminolevulinic acid dehydratase, mean cell hemoglobin content, elevation of urinary δ -aminolevulinic acid excretion, blood zinc protoporphyrin and reduced the uptake of lead in liver, kidney and blood. The results suggest that many adverse effects of lead can be favourably reduced by simultaneous supplementation of gur (Jaggery).

Key words: Jaggery (Gur), Lead toxicity, δ -aminolevulinic acid dehydratase.

INTRODUCTION

Absorption and retention of toxic metals depends on dietary factors. The evidence for effects of mineral, amino acids, vitamins, fats, and protein on the absorption and retention of lead in experimental animals has been particularly well documented [1].

Jaggery or gur, has been known for its therapeutic properties in the Ayurvedic system of Indian medicine. Regular consumption of gur is said to confer symptomatic relief in industrial and mine workers in India, gur is therefore provided regularly to workers engaged in mining and industrial establishments. In the present study, beneficial effect of simultaneous supplementation of gur (Jaggery) on the uptake of lead and some lead sensitive biochemical variables in blood and urine were determined in rats.

MATERIALS AND METHODS

Gur or Jaggery. Jaggery is inspissated juice expressed from sugarcane. Sugarcane juice contains sugars and other carbohydrates, proteins, minerals, traces of heavy metals and vitamins [2-4].

Experimental. Twenty four male albino rats (150 \pm 10 g) of Industrial Toxicology Research Centre's Colony maintained on standard pellet diet (Hindustan Lever Ltd)[†] and water *ad libitum*, were divided equally into four groups and treated 6 days/week for six weeks as follows :

- Group I— No treatment (Normal animals)
- Group II— Gur, 200 mg/kg/2 ml of sterile distilled water, given orally/once.
- Group III— Lead, 10 mg/kg, has lead acetate, orally, once.
- Group IV— Lead, 10 mg/kg, orally, once + Gur, 200 mg/kg, orally, once.

The dose of gur in rats was based on half of its average daily consumption by mine workers in India.

After the last administration each animal was placed in a separate metabolic cage for the collection of 24 hr urine sample. The animals were thereafter sacrificed by decapitation, kidney, liver, brain removed and blood collected in heparinized vials. The tissues were cleaned free of extraneous material and weighed.

Standard procedures were used to determine the activity of blood δ -aminolevulinic acid dehydratase (ALAD) [5] zinc protoporphyrin (ZPP) [6] mean cell volume (MCV), mean hemoglobin contents (MCH), mean cell hemoglobin concentration (MCHC) and urinary δ -aminolevulinic acid (ALA) excretion [7]. The lead contents of kidney, liver, brain [8] and of blood [9] were estimated using atomic absorption spectrophotometer (Perkin Elmer 5000) after wet acid digestion with HNO_3 .

RESULTS AND DISCUSSION

The exposure to lead alone significantly decreased the activity of blood ALAD, MCH contents, increased the blood ZPP, MCHC levels and urinary excretion of ALA. The simultaneous administration of gur significantly reduced these effects (Table 1).

*To whom all correspondence should be addressed.

[†]Metal content of diet (ppm dry weight) Cu 10.0, Mn 55.0, Co 5.0, Fe 70.0, Zn 45.0.

Table 1. Influence of Jaggery on some blood and urine variables in lead intoxicated rats.

	Blood				Urine	
	ALAD (n mol/min/ml erythrocyte)	ZPP ($\mu\text{g/g Hb}$)	MCV (μ^3)	MCH (pg Hb)	MCHC (g/100 ml)	ALA (mg/100 ml)
Normal animals	6.18 \pm 0.25	1.01 \pm 0.06	54.86 \pm 0.48	16.40 \pm 0.65	29.85 \pm 1.03	0.085 \pm 0.007
Jaggery	5.61 \pm 0.46	1.10 \pm 0.10	55.48 \pm 0.67	19.39 \pm 0.54 ^b	26.14 \pm 1.20	0.082 \pm 0.03
Lead	1.91 \pm 0.26 ^a	2.43 \pm 0.18 ^a	54.35 \pm 0.80	14.18 \pm 0.62 ^c	34.99 \pm 1.17 ^c	0.51 \pm 0.01 ^a
Lead + Jaggery	5.13 \pm 0.33 [*]	1.85 \pm 0.15 ^{a***}	53.84 \pm 0.77	18.67 \pm 0.81 ^{c**}	35.25 \pm 1.25 ^c	0.41 \pm 0.02 ^{a**}

Each value is mean \pm SE; N=6; ^ap < 0.001, ^bp < 0.01, ^cp < 0.05 compared to normal animals, *p < 0.001, **p < 0.01, ***p < 0.05 compared to lead treated groups as evaluated by the student's 't'-test.

Table 2. Influence of Jaggery on the blood and tissue lead contents in exposed rats.

	Blood ($\mu\text{g}/100 \text{ ml}$)	Liver ($\mu\text{g}/\text{g}$)	Kidney ($\mu\text{g}/\text{g}$)	Brain ($\mu\text{g}/\text{g}$)
Normal animals	8.10 \pm 0.60	2.30 \pm 0.39	6.89 \pm 0.74	1.74 \pm 0.16
Jaggery	8.08 \pm 0.70	3.08 \pm 0.49	3.81 \pm 0.44	2.04 \pm 0.38
Lead	69.22 \pm 4.42 ^a	13.37 \pm 1.35 ^a	22.01 \pm 1.09 ^a	3.10 \pm 0.39 ^a
Lead + Jaggery	43.06 \pm 3.51 ^{a*}	7.98 \pm 0.62 ^{a***}	11.01 \pm 0.63 ^{a*}	3.30 \pm 0.83 ^a

Each value is mean \pm SE; N=6; ^ap < 0.001 compared to normal animals; *p < 0.001, **p < 0.01 compared to lead treated group as evaluated by the student's 't'-test.

The accumulation of lead in kidney, liver, brain and blood increased significantly on exposure to lead alone. The uptake of lead in these tissues (except brain) reduced significantly in animals when gur was administered concomitantly (Table 2).

There has been a local traditional belief and practice among workers engaged in various occupations in our country to consume jaggery (gur) daily, for alleviating the occupation related toxic effects and such a practice appears to keep them, to a greater extent symptoms free. The results of the present study showed significant recovery in the lead induced inhibition in the activity of blood ALAD, decrease in MCH contents and elevation in blood ZPP level and urinary ALA excretion. Beside biochemical alterations lead contents of tissues and blood were greatly reduced by gur treatment. These findings indicate that gur treatment presumably increased the physiological status of almost every important organ and cell of the body suggesting that lead clearance from blood and tissues could be by an activated biochemical pathway. The present results suggest that gur or its microingredient(s) have potential properties which play a protective role against lead toxicity. Thiamine, riboflavine, and vitamin C, the three major

constituents of gur have been evaluated as potential therapeutic agents for lead toxicity [10,11]. Gur is reported to increase hemoglobin and sulphhydryl contents [12] which suggest that gur or its microingredient(s) may play some protective role in the removal of toxic biologically active metal from the body.

Acknowledgement. Dr. S.J.S. Flora is grateful to Indian Council of Medical Research for the award of a supernumery position (Senior Research Officer). Thanks are due to Mr. Ashok Kumar for the technical assistance and to Mr. A.K. Nigam for secretarial assistance.

REFERENCES

1. K.R. Mahaffey and I.A. Michaelson, Interactions Between Lead and Nutrition. In: *Low Level Lead Exposure: The Clinical Implications of Current Research*, H.L. Needleman, ed. (Raven Press, New York, 1980), pp. 159-200.
2. *Gur, The Wealth of India, Industrial Product Part IV: F-H* (Council of Scientific and Industrial Research, New Delhi 1957), p.182.
3. Monograph on the Gur Industry of India, S.C. Roy,

- ed. (Indian Central Sugarcane Committee, Indian Institute of Sugar Technology, Kanpur, 1951), p. 285.
4. Meade-Chen, *Sugar Hand Book* (John Wiley and Sons, 1977), 10th ed., p. 15.
 5. A. Berlin and K.H. Schaller, *Z. Klin. Chem. Klin. Biochem.*, **12**, 389 (1974).
 6. P. Grandjean, *Bril. J. Indust. Med.*, **36**, 52, (1979).
 7. J.R. Davis, R.H. Abrahams, W.I. Fishbein and E.A. Fabrega, *Arch. Environ. Health*, **17**, 164, (1968).
 8. D.W. Yeager, J. Cholak and E.W. Henderson, *Environ. Sci. Technol.*, **5**, 1020 (1971).
 9. D.W. Hessel, *Atomic Absorp. Newslett.*, **7**, 55, (1968).
 10. S.K. Tandon, S.J.S. Flora and S. Singh, *Pharmacol. Toxicol.*, **60**, 62 (1987).
 11. S.J.S. Flora and S.K. Tandon, *Acta Pharmacol. et Toxicol.*, **58**, 374 (1986).
 12. A.P. Sahu, R.K. Upreti, A.K. Saxena and R. Shanker, *Ind. J. Exp. Biol.*, **26**, 112 (1988).