Effect of taurine on biomarkers of oxidative stress in tissues of fructose-fed insulin-resistant rats

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ABSTRACT

Introduction: The present study was designed to investigate whether taurine mitigates fructose-induced oxidative stress in rat tissues such as heart and kidney.

Methods: Male Wistar rats of body weight 170-190g were divided into four groups containing six rats each. Control animals received the control diet containing starch while fructose-fed animals received a fructose-enriched diet (greater than 60 percent of total calories). Fructose and taurine rats received the fructose diet and two percent taurine solution to drink. Control and taurine rats received the control diet and two percent taurine solution. After the treatment period of 30 days, insulin resistance index, by homeostasis model assessment (HOMA) was determined. The levels of lipid peroxidation markers, the enzymatic and non-enzymatic antioxidants status in heart and kidney tissues were measured.

Results: Fructose rats showed high values of HOMA, increased lipid peroxidation and impaired antioxidant status. Taurine treatment to fructose rats attenuated the increased lipid peroxidation, enhanced the levels of antioxidants and improved insulin sensitivity.

Conclusion: Inhibition of peroxidation markers and upregulation of antioxidant activity in rat tissues by taurine signify the potential utility of taurine as an adjunct in treatment of insulin resistance.

Keywords: antioxidants, fructose diet, insulin, lipid peroxidation, taurine

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INTRODUCTION

Feeding rats with high fructose diet affects both glucose and lipid metabolism which result in a cluster of metabolic abnormalities such as glucose intolerance, hypertension, dyslipidemia and reduced insulin action[1]. Fructose-fed rats form a useful experimental model of insulin resistance. Fructose feeding is also reported to facilitate oxidative damage and has deleterious effects both due to reduction in antioxidant defence and enhanced free radical production[2]. A number of studies have found that the supplementation of antioxidants such as lipoic acid, glutathione, vitamin C and vitamin E improve insulin sensitivity in patients with insulin resistance, type 2 diabetes and/or cardiovascular disease[3,4].

Taurine (2-amino ethane sulphonic acid) is a sulphur-containing amino acid that is the most abundant free amino acid in excitable tissues and cells. It serves several physiological and metabolic functions and has been reviewed extensively[5]. Taurine comprises over 50% of the total free amino acid pool of the heart and has a positive inotropic action on cardiac tissue. The beneficial effects of taurine as an antioxidant in biological systems have been attributed to its ability to stabilise biomembranes, to scavenge reactive oxygen species, and to reduce the peroxidation of unsaturated membrane lipids[6].

Previous studies have shown that taurine reduces oxidative stress in liver of high fructose-fed rats[7]. In the present paper, we report the effects of taurine on whole body insulin sensitivity and oxidant-antioxidant balance in heart and kidney of fructose-loaded rats.

METHODS

Male adult Wistar rats of body weight ranging from 170g to 190g in were obtained from the Central Animal House, Rajah Muthiah Medical College, Annamalai University. They were housed two per cage under controlled conditions on a 12hr light/12hr dark cycle. They all received a standard pellet diet (Karnataka State Agro Corporation Ltd, Agro Feeds Division, Bangalore, India) and water *ad libitum*. The study protocol was approved by the Institutional Animal Ethical Committee, Rajah Muthiah Medical College, Annamalai University. Taurine was purchased from Sisco Research Laboratories, Mumbai, India. All other chemicals were of analytical grade procured from local commercial sources. After acclimatisation,

the animals were divided into the following groups consisting of 6 rats each.

Group 1 (CON) received the control diet and tap water *ad libitum*. The control diet contained corn starch (60%) as the sole source of carbohydrate, 20% casein, 0.7% methionine, 5% groundnut oil, 10.5% wheat bran and 3.5% salt mixture, and water *ad libitum*. Vitamin mixture (0.2ml) was added per kg feed. Group 2 animals (FRU) received a fructose-enriched diet and water *ad libitum*. The high fructose diet was similar in composition to the control diet except that starch was replaced by fructose. Group 3 (FRU+TAU) animals received the high fructose diet and were allowed to drink 2% taurine solution *ad libitum*. Group 4 (CON+TAU) animals received the control diet and were given 2% taurine solution *ad libitum*. The diets were prepared fresh daily.

The animals were maintained in their respective groups for 30 days. Food intake, fluid intake and body weight changes were measured regularly. At the end of the experimental period, the rats were sacrificed by cervical decapitation. An oral glucose tolerance test was carried out two days before the sacrifice of the animals. For this, the rats were fasted overnight and glucose (2g/kg body weight) was given from 30% solution orally. Blood samples were collected before glucose load and sequentially for every half an hour after glucose load up to 90 minutes and were immediately analysed for glucose.

Blood was collected and plasma was separated. Plasma insulin was assayed by enzyme-linked immunosorbent assay (ELISA) kit, using human insulin as standard. Plasma glucose levels were assayed by the method of Sasaki et al[8]. Homeostasis model assessment (HOMA) was used as an index to measure the degree of insulin resistance and was calculated by the formula: [insulin (μU/ml) x glucose (in mmol/L)/22.5][9].

The heart and kidney were removed and immediately rinsed in ice-cold saline. Homogenates were prepared from the tissues and were used for the analysis. Thiobarbituric acid reactive substances (TBARS), conjugated dienes, lipid hydroperoxides and lipofuscin were determined as described earlier[7]. In brief, the concentration of TBARS was estimated by measuring the pink- coloured chromophore upon reaction with thiobarbituric acid at 535nm. A standard curve was prepared using 1,1',3,3'-tetra methoxy propane as the standard. For conjugated dienes, the absorbance of tissue lipid extracts dissolved in cyclohexane was determined at 233nm. An extinction coefficient of 2.52 x $10^4 M^{-1}$ was used to calculate the concentration of conjugated dienes.

For lipofuscin measurement, 0.5 ml of tissue homogenate was suspended in 3ml of isopropanol and 2 ml of chloroform. This was allowed to stand for 30 min and centrifuged at 1800xg in a refrigerated centrifuge. The fluorescence was measured using a spectrofluorimeter with extinction at 360nm and emission at 440nm. Lipid hydroperoxides were measured in methanol-extracted tissue homogenates. 0.2 ml of lipid sample was mixed with 1.8 ml of the reagent, which contained 90 ml of methanol, 10 ml of 250 mM sulfuric acid, 88 mg butylated hydroxy toluene, 7.6 mg xylenol orange and 9.8 mg ferrous ammonium sulfate. The colour developed was read at 560 nm.

The activities of antioxidant enzymes superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione-S-transferase (GST) and glutathione reductase (GR) were assayed by methods described elsewhere[10]. SOD activity in the tissues was assayed, based on the inhibition of formation of NADH-phenazine methosulphate-nitro blue tetrazolium complex. CAT activity was assayed by quantifying the hydrogen peroxide after reacting with dichromate in acetic acid. For GPx activity, an aliquot of enzyme preparation was allowed to react with hydrogen peroxide (H_2O_2) and glutathione (GSH) for a specified time period. Then the GSH content remaining after the reaction was measured. Activity of GST was measured in the tissues by following the increase in absorbance at 340nm using 1-chloro-2,4, dinitro benzene (CDNB) as substrate. GR activity in tissues was assayed by measuring the rate of NADPH oxidation.

α-tocopherol was estimated by the method of Baker et al[11] by measuring the red-coloured complex upon reaction with 2, 2' dipyridyl at 520nm. Ascorbic acid was measured according to the method of Roe and Kuether[12] using 2,4 dinitro phenyl hydrazine. The levels of total, protein-bound and non-protein bound thiols were determined by the method of Sedlack and Lindsay[13] using dithionitrobenzoic acid (DTNB) as the colouring reagent. Taurine content was determined in plasma and tissues by high performance liquid chromatography after conversion to 3, 5-dinitrobenzoyl derivative by the method of Masouka et al[14].

All the values were expressed as means ± SD of six rats from each group and statistically evaluated by two-way analysis of variance considering diet and treatment as two factors. When significance was found, the means were tested for significance by Tukey's test for multiple comparisons[15]. A value of $p<0.05$ was considered significant.

Table I. Levels of plasma glucose, insulin and insulin sensitivity index of control and experimental animals.

					ANOVA[2]		
Parameters	CON	FRU	FRU+TAU	CON+TAU	FRU	TAU	Interaction
Glucose (mg/dL)	81.22 ± 7.79	97.35 ± 2.37[a]*	87.52 ± 6.22[b]*	84.99 ± 6.62	0.05	0.05	NS
Insulin (μU/ml)	54.21 ± 4.03	90.70 ± 4.00[a]*	59.63 ± 5.49[b]*	57.40 ± 8.04	0.05	0.05	0.05
Insulin resistance index (HOMA)[#]	12.52 ± 1.24	21.59 ± 1.08[a]*	13.56 ± 0.67[b]*	12.7 ± 1.56	0.05	0.05	0.05

Values are means ± SD from 6 animals in each group

[a] – compared with CON; [b] – compared with FRU * $p<0.05$

(ANOVA followed by Tukey's test) NS – not significant

$$^{\#}\text{HOMA} = \frac{\text{Insulin (}\mu\text{U/L) x glucose (mmol/L)}}{22.5}$$

Table II. Lipid peroxidation products in the hearts and kidneys of control and experimental animals.

					ANOVA[2]		
Parameters	CON	FRU	FRU+TAU	CON+TAU	FRU	TAU	Interaction
TBARS (nmoles/mg protein)							
Heart	1.00 ± 0.09	1.41 ± 0.12[a]*	1.11 ± 0.08[b]*	1.01 ± 0.06	NS	0.05	NS
Kidney	1.64 ± 0.04	2.15 ± 0.10[a]*	1.66 ± 0.09[b]*	1.66 ± 0.04	0.05	0.05	0.05
Conjugated dienes (A 233/215)							
Heart	0.52 ± 0.02	0.68 ± 0.03[a]*	0.59 ± 0.05[b]*	0.54 ± 0.03	0.05	0.05	0.05
Kidney	0.62 ± 0.14	0.81 ± 0.12[a]*	0.62 ± 0.04[b]*	0.62 ± 0.09	NS	NS	NS
Lipofuscin (relative fluorescence)							
Heart	22.8 ± 1.50	25.8 ± 1.90[a]*	23.0 ± 1.10[b]*	22.5 ± 1.40	NS	NS	0.05
Kidney	21.4 ± 1.21	24.8 ± 1.15[a]*	20.4 ± 1.16[b]*	20.9 ± 1.04	0.05	0.05	0.05
Hydroperoxides (μmoles/mg protein)							
Heart	1.20 ± 0.10	1.48 ± 0.17[a]*	1.26 ± 0.15[b]*	1.29 ± 0.10	NS	0.05	NS
Kidney	1.66 ± 0.15	2.17 ± 0.24[a]*	1.77 ± 0.09[b]*	1.70 ± 0.11	0.05	0.05	0.05

Values are means ± SD from 6 animals in each group

[a] – compared with CON; [b] – compared with FRU * $p<0.05$

(ANOVA followed by Tukey's test) NS – not significant

RESULTS

The levels of plasma glucose, insulin and insulin resistance index of control and experimental animals are listed in Table I. The levels were significantly elevated in fructose-fed rats as compared to control rats. Taurine treatment to fructose-fed rats prevented the increase. The degree of insulin resistance as measured by HOMA was higher in fructose-fed rats while in taurine treated rats the values were normal.

Table II shows the influence of high fructose diet and taurine on the levels of TBARS, lipid hydroperoxides, lipofuscin and conjugated dienes in the hearts and kidneys of control and experimental animals. Fructose rats showed significantly higher peroxidation as compared to control rats. Taurine treatment of fructose-fed rats reduced the levels of lipid peroxidation markers. No significant changes were observed in control rats treated with taurine as compared to control rats.

Table III shows the activities of enzymatic antioxidants SOD, CAT, GPx, GST and GR in the hearts and kidneys of control and experimental animals. The activities of these enzymes were significantly lower in fructose-fed rats than normal rats. On taurine treatment, the activities of all these enzymes were significantly higher as compared to fructose rats. The levels were near normal in control rats treated with taurine except for GR in heart and kidney.

Table IV shows the concentrations of non-enzymatic antioxidants vitamin C, vitamin E and total thiol, non-protein thiol, protein bound thiols in

Table III. Activities of antioxidant enzymes in the hearts and kidneys of control and experimental animals.

					ANOVA[2]		
Parameters	CON	FRU	FRU+TAU	CON+TAU	FRU	TAU	Interaction
Superoxide dismutase (Units- 50% of NBT reduction/min/mg protein)							
Heart	3.60 ± 0.61	3.06 ± 0.77[a]*	4.02 ± 0.58[b]*	4.12 ± 0.48	0.05	NS	NS
Kidney	4.76 ± 0.58	4.02 ± 0.46[a]*	4.96 ± 0.56[b]*	4.68 ± 0.51	NS	NS	0.05
Catalase (μmoles of H_2O_2 consumed/min/mg protein)							
Heart	54.50 ± 5.28	48.30 ± 3.81[a]*	56.85 ± 3.45[b]*	54.01 ± 3.55	NS	NS	NS
Kidney	57.43 ± 4.00	44.20 ± 3.76[a]*	54.00 ± 3.76[b]*	57.20 ± 3.66	0.05	0.05	0.05
Glutathione peroxidase (μmoles of GSH oxidised/min/mg protein)							
Heart	5.23 ± 0.68	4.39 ± 0.32[a]*	5.82 ± 0.37[b]*	6.01 ± 0.67	0.05	0.05	NS
Kidney	5.01 ± 0.28	4.64 ± 0.25[a]*	5.49 ± 0.45[b]*	6.01 ± 0.48	0.05	0.05	NS
Glutathione reductase (μmoles NADPH oxidised/min/mg protein)							
Heart	18.92 ± 1.83	15.30 ± 1.68[a]*	24.48 ± 0.78[b]*	26.05 ± 1.25	0.05	0.05	NS
Kidney	22.13 ± 1.45	18.77 ± 0.74[a]*	25.06 ± 0.95[b]*	28.75 ± 1.26	0.05	0.05	NS
Glutathione-S-transferase (mmoles of glutathione – CDNB/conjugate formed/min/mg protein)							
Heart	4.32 ± 0.41	4.01 ± 0.32[a]*	4.85 ± 0.42[b]*	4.90 ± 0.62	0.05	NS	NS
Kidney	5.23 ± 0.39	4.03 ± 0.23[a]*	5.40 ± 0.27[b]*	5.81 ± 0.48	0.05	0.05	0.05

Values are means ± SD from 6 animals in each group

[a] – compared with CON; [b] – compared with FRU * $p<0.05$

(ANOVA followed by Tukey's test) NS – not significant

Table IV. Concentrations of non-enzymatic antioxidants in the hearts and kidneys of control and experimental animals.

					ANOVA[2]		
Parameters	CON	FRU	FRU+TAU	CON+TAU	FRU	TAU	Interaction
Ascorbic acid (mg/mg protein)							
Heart	0.59 ± 0.09	0.48 ± 0.06[a]*	0.56 ± 0.05[b]*	0.60 ± 0.05	NS	0.05	0.05
Kidney	0.79 ± 0.09	0.69 ± 0.07[a]*	0.78 ± 0.05[b]*	0.85 ± 0.06	0.05	0.05	0.05
Vitamin E (mg/mg protein)							
Heart	0.95 ± 0.07	0.84 ± 0.07[a]*	0.95 ± 0.09[b]*	0.85 ± 0.09	NS	NS	NS
Kidney	0.93 ± 0.05	0.84 ± 0.04[a]*	0.90 ± 0.05[b]*	0.87 ± 0.06	NS	NS	NS
Total thiol (μg/mg protein)							
Heart	12.73 ± 0.55	11.88 ± 0.40[a]*	15.54 ± 0.46[b]*	16.42 ± 0.38	0.05	0.05	NS
Kidney	13.79 ± 0.20	13.25 ± 0.26[a]*	15.05 ± 0.43[b]*	15.32 ± 0.49	0.05	0.05	NS
Non-protein thiol (μg/mg protein)							
Heart	5.22 ± 0.37	4.65 ± 0.50[a]*	5.89 ± 0.49[b]*	6.52 ± 0.28	0.05	0.05	NS
Kidney	5.19 ± 0.22	4.71 ± 0.31[a]*	5.61 ± 0.19[b]*	5.81 ± 0.21	0.05	NS	NS
Protein-bound thiol (μg/mg protein)							
Heart	7.51 ± 0.33	7.03 ± 0.27[a]*	7.65 ± 0.34[b]*	9.90 ± 0.36	0.05	0.05	0.05
Kidney	8.80 ± 0.52	8.14 ± 0.48[a]*	9.44 ± 0.37[b]*	9.51 ± 0.27	0.05	NS	NS

Values are means ± SD from 6 animals in each group

[a] – compared with CON; [b] – compared with FRU * $p<0.05$

(ANOVA followed by Tukey's test) NS – not significant

Table V. Taurine content in plasma (μmol/L) and tissues (μmol/mg protein) in control and experimental animals.

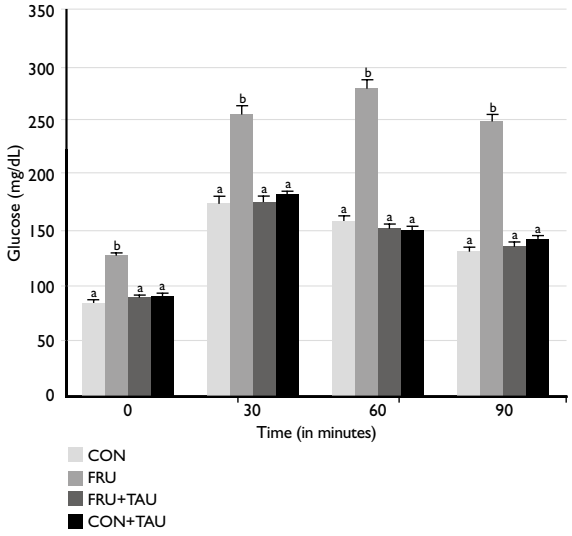
					ANOVA[2]		
Parameters	CON	FRU	FRU+TAU	CON+TAU	FRU	TAU	Interaction
Plasma	102.1 ± 4.17	92.0 ± 8.2[a*]	143.0 ± 5.8[b*]	158.0 ± 10.6[ab*]	NS	0.05	0.05
Heart	889.9 ± 28.1	843.8 ± 34.8[a*]	891.0 ± 38.5[b*]	944.0 ± 33.6[ab*]	NS	NS	NS
Kidney	498.2 ± 28.2	444.2 ± 18.1[a*]	562.3 ± 24.6[b*]	527.3 ± 25.0[ab*]	0.05	NS	0.05

Values are means ± SD from 6 animals in each group

[a] – compared with CON; [b] – compared with FRU * $p<0.05$

(ANOVA followed by Tukey's test) NS – not significant

Fig. 1 Oral glucose tolerance test.



the hearts and kidneys of control and experimental animals. Significant reductions were observed in the levels of the antioxidants in tissues of fructose-fed rats. Simultaneous treatment of fructose rats with taurine brought the levels to near normal values and the levels were not significantly different from those of control rats. Taurine did not have any significant effect on the non-enzymic antioxidant levels in animals fed control diet.

At the end of the experimental period, the contents of taurine were significantly decreased in plasma and tissues of the fructose-fed rats as compared to control rats (Table V). Taurine supplementation resulted in higher levels of taurine in plasma and tissues as compared to control rats. The results of the oral glucose tolerance test in experimental animals are depicted in Fig. 1. The fasting glucose level was higher in fructose-fed rats as compared to control rats, and the level was significantly lower in fructose-fed rats treated with taurine than the untreated fructose-fed rats. Significant elevations were observed in the glucose levels after the oral glucose load at all the time points in fructose-fed rats. The response was normal in taurine-treated rats.

DISCUSSION

Insulin resistance in fructose-fed rat model has been attributed to a low level of insulin-stimulated glucose oxidation due to modifications in the post-receptor cascade of insulin action[16]. High levels of dietary fructose and severe hyperglycemia may have interactive effects, which contribute to the progression and development of pathology. Fructose feeding can induce free radical formation by down regulation of HMP shunt enzymes that generate reduced environment in the form of NADPH and NADH[17]. Further, an increase in catabolism of fructose would result in energy depletion in cells, making them more susceptible to peroxidation.

Enhanced lipid peroxidation in fructose-fed rats could be associated with high circulating glucose, which enhances free radical production from glucose autoxidation and protein glycation. Prolonged exposure of rats to hyperglycemic condition reduces the activities of SOD and other antioxidant enzymes. Inactivation of Cu, Zn- SOD by glycation of specific lysine residues has been reported by Oda et al[18]. Reactive oxygen species (ROS) can themselves reduce the activity of antioxidant enzymes such as CAT and GPx[19]. Reduction in the activities of GST and GR are suggestive of reduced scavenging potential in insulin resistant rats.

Taurine supplementation could have reduced lipid peroxidation by causing reduction in blood glucose levels and by the attenuation of hyperinsulinemia. Previously it was shown that taurine normalises glucose metabolism and attenuates hyperinsulinemia in high fructose-fed rats[20]. Administration of taurine may attenuate tissue lipid peroxidation either by inhibition of ROS formation or by binding Fe^{2+} like a chelator[21]. Although taurine is a poor scavenger of ROS, complex formation between sulphonic acid group (SO_3^-) to free metal ion species such as Fe^{2+}, Cu^+ or oxidant metalloproteins has been reported[22].

Taurine may also inhibit lipid peroxidation by inducing GPx and SOD. Taurine could protect tissues against reduced glutathione pool depletion by preventing a decrease of glutathione reductase activity.

Ogasawara et al[23] reported that taurine can react with glucose and other aldehydes such as acetaldehyde and malondialdehyde and has higher reactivity with these aldehydes than other amino acids such as glycine, α- and β-alanine. Further, the taurine-glucose reaction product showed an antioxidative effect on lipid peroxidation of constituted liposomes. The observed effects of taurine could also be attributed to its ability to resist cell damage in a non-specific way by membrane stabilisation and by osmoregulation[24].

A wealth of data is available on the role of taurine in diabetes. Koya et al[25] suggested that taurine improves oxidative stress in the glomeruli of the diabetic rats. In another study, taurine supplementation ameliorated biochemical retinal abnormalities caused by diabetes[26]. Taurine is synthesised from cysteine, which is the precursor of GSH. In fructose-induced insulin-resistant rats, depletion of GSH and decreased activities of antioxidant enzymes are observed. Thus, in fructose-treated rats, decreased tissue levels of taurine may be due to decreased availability of cysteine. Hence, taurine supplementation to these animals may spare cysteine, which increases the tissue levels of GSH. A parallel improvement of insulin sensitivity and attenuation of oxidative stress by taurine in this nutritional model of insulin resistance suggest the possible utility of taurine as a therapeutic adjunct in a metabolic state of insulin resistance.

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