Effects of Taurine on Glutathione Peroxidase, Glutathione Reductase and Reduced Glutathione Levels in Rats

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Abstract: The aim of the present study is to investigate the effects of oral administration of taurine on endogenous glutathione peroxidase (GPx) and glutathione reductase (GR) activities and reduced glutathione (GSH) level in normal rats. Normal saline (Group I) or 5% taurine in normal saline was administered in dose of 50 mg (Group II), 250 mg (Group III) or 500 mg kg−1 of body weight (Group IV) through intragastric intubation for 60 days. GPx and GR enzyme activities and GSH and taurine levels were determined in liver, heart, stomach, kidney and plasma of normal Wistar rats. GPx activity showed an increase in liver, heart, stomach and plasma. GR activity increased in kidney and decreased in liver and plasma. GSH levels increased in liver, stomach and decreased in kidney. Liver showed an increase and heart, stomach and kidney a decrease in taurine level in taurine administered rats when compared to control rats. The results varied from organ to organ and the observed variations among organs might be related to their respective enzymatic, non-enzymatic antioxidant potential and its functions. From the present study it may be concluded that long term oral administration of taurine effects GPx, GR and GSH levels in normal rats.

Key words: Glutathione enzyme, free radical, taurine, redox-state, oxidative stress, prophylactic agents

INTRODUCTION

In tissues, the oxidation-reduction state also called the redox state depends on the delicate balance between oxidants and antioxidants status at any given time. The oxidants are the free radicals and reactive oxygen or nitrogen species (ROS and RNS, respectively) produced in the body as a result of regular intermediary metabolism and other oxidative processes like respiratory bursts seen in neutrophil during phagocytosis. Apart from their roles in the intermediary metabolism and body defence, the oxidants also play a major role as regulatory mediators in signalling processes (Seifried et al., 2007). However, at high concentration, oxidants can damage all major cellular constituents and is termed as oxidative stress (Valko et al., 2007). The oxidative stress is implicated in the pathogenesis of various disorders like cancer, diabetes, cardiovascular diseases, autoimmune diseases and neurodegenerative disorders (Nakagawa and Miyata, 2008; Mates et al., 1999; Gwarzo et al., 2010; Kayode et al., 2009; Vaghasia and Chanda, 2010). Potentially damaging oxidative stress is kept in check by endogenous cellular antioxidant mechanisms, which include antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) and nutrient derived antioxidant small molecules (vitamin C, vitamin E, carotenoids, flavonoids, glutathione, uric acid and taurine (Devasagayam et al., 2004).

Unfortunately, the public has become too familiar with the term antioxidant and equates it to a chemical that is good for our wellbeing. Antioxidant foods, drinks (often in parallel unhealthily rich in sugars and colourants), cosmetics and creams abound the market. As a result, numerous vitamins trace elements and micronutrients often with well-defined biochemical functions (such as ascorbate, a known cofactor for several enzymes) have been re-branded as universal antioxidants. This implies that because they are antioxidants, the more one consumes the better it is for your health (Gutteridge and Halliwell, 2010). Excessive intake of antioxidants could down-regulate important endogenous antioxidants (Halliwell and Gutteridge, 2007; Gutteridge, 1999). It is also found to depress parts of the immune system and increase microbial damage. The normal cellular protective responses to tissue damage may also be altered (Gutteridge, 1999; Ristow et al., 2009; Childs et al., 2001; Leufier and Royet, 2009; Ha et al., 2009).

In recent years, antioxidants have gained a lot of importance because several antioxidants like SOD, CAT, lycopene, co-enzyme Q10, vitamin C and vitamin E have

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been found to be pharmacologically active as prophylactic and therapeutic agents for various clinical disorders (Ratan et al., 2006). Taurine (2-aminoethane sulfoonic acid), a conditionally essential amino acid is a well-documented antioxidant agent. Clinically taurine has been used with varying degrees of success in the treatment of a wide variety of conditions including cardiovascular diseases, hypercholesterolemia, epilepsy and other seizure disorders, macular degeneration, Alzheimer’s disease, hepatic disorders, alcoholism and cystic fibrosis (Birdsell, 1998). Taurine was reported to be beneficial in preventing experimental diabetic neuropathy (Obrosova et al., 2001), lead-induced oxidative damage (Gurer et al., 2001), CCL-induced oxidative stress (Vohra and Hui, 2001), cerulein-induced acute pancreatitis (Ahn et al., 2001), nonylphenol-induced toxicity (Karafakioglou et al., 2011), aluminium-induced oxidative stress (Al-Kattani, 2010) and early changes in experimental diabetic kidney (Ha et al., 1999) through antioxidant mechanisms. Protective effects on lens (Devamohanarans et al., 1998) and modulating effects on human endothelial cell death are among its other effects (Wu et al., 1999).

Taurine is one of the key ingredients in energy drinks which are widely available in the grocery stores and convenience stores for easy access (Smith et al., 2011). Taurine is marketed as a dietary supplement for promotion of biliary health, eye health and prevention and treatment of congestive heart failure (Babu et al., 2008). Little is known regarding the effects of high-dose or long-term taurine use in children and adolescents (Babu et al., 2008).

Despite its extensive use, clinical and animal studies conducted with various doses of taurine on normal endogenous antioxidant system are not reported in literature. In light of this context, the present study was designed to investigate the effects of oral administration of graded doses of taurine on endogenous glutathione peroxidase, glutathione reductase and reduced glutathione levels in plasma, liver, heart, stomach and kidney in normal Wistar rats.

MATERIALS AND METHODS

Experimental animals: Forty albino Wistar rats (150-200 g; 20 weeks old) of either sex were obtained from the Central Animal House, Rajah Muthiah Medical College, Annamalai University during February 2009 and August 2010. The animals were maintained with standard rat chow and tap water ad libitum under standard laboratory conditions. The experiment was conducted in accordance with the accepted principles and guidelines of the Committee for the purpose of control and supervision on experiments on animals (CPCSEA), India after obtaining necessary approval.

Chemicals and reagents: Taurine, homoserine, dowex resins, o-phthalaldehyde and mercaptopropanol were obtained from Sigma-Aldrich, St. Louis, MO. The assay kits for glutathione (GSH) (Cat. No. 703002), glutathione peroxidase (GPx) (Cat. No. 703102) and glutathione reductase (GR) (Cat. No. 703202) were purchased from Cayman chemicals company USA. Qualigens chemicals, India supplied the methanol (HPLC grade). All other chemicals used were of analytical grade.

Experimental design: Forty albino Wistar rats were divided into 4 groups of 10 rats each. Animals in Group 1 (Control) were administered 2.5 mL of 0.9% saline orally through intra-gastric intubation for 60 days. Animals in Group 2 (Low dose-Taurine), Group 3 (Medium dose-Taurine) and Group 4 (High dose-Taurine), were given 5% taurine in 0.9% saline orally at a dose level of 50, 250 and 500 mg kg⁻¹ of body weight, respectively through intra-gastric intubation for 60 days (Anand et al., 2010). At the end of 60 days blood samples were collected and liver, heart, stomach and kidney were dissected out for analysis.

Collection of blood samples: All the experimental procedures were performed between 08:00 and 10:00 h. following a fasting period of 12 h, blood samples were withdrawn from retro-orbital sinus using fine capillary tube into the heparinised tubes. The heparinized blood was centrifuged at 1000xg for 10 min at 4°C. The supernatant plasma was separated out and stored at -80°C until assay.

Tissue preparation: After collection of the blood, animals were sacrificed by cervical decapitation and liver, heart, stomach and kidney were quickly dissected out into ice-cold Phosphate Buffered Saline (PBS). Prior to removal of the tissues, they were rinsed with PBS (pH 7.4) containing 0.16 mg mL⁻¹ heparin to remove any blood clots. The tissues were homogenised and centrifuged as per the instructions from the suppliers of the kit. The resulting supernatant was removed and stored at -80°C until the assay was carried out.

Glutathione assay: GSH levels were measured following the procedure (Zhu et al., 2007) provided by the suppliers of the assay kit. Optimized enzymatic recycling method using GR is the basic principle of this assay kit.
Briefly, 100 µL of supernatant of tissue homogenate was added to an equal volume of the metaphosphoric acid and then centrifuged at 2,000g for 2 min to remove protein. Then 50 µL of 4 M triethanolamine per mL of the supernatant was added to increase the pH of the sample. For total GSH assay, 50 µL of sample was added to 150 µL of a reaction mixture containing 0.4 M 2-(N-morpholino)ethanesulfonic acid, 0.1 M phosphate (pH 6.0), 2 mM EDTA, 0.24 mM NADPH, 0.1 mM 5, 5'-dithiobis-2-nitrobenzoic acid (DTNB) and 0.1 unit GR. The absorbance was measured using a microplate reader at 5 min intervals for 30 min. Reduced glutathione (GSH) concentration were expressed as µmol g⁻¹ of wet tissue and µmol mL⁻¹ of plasma.

**Glutathione peroxidase assay:** GPx activity was measured following the procedure (Zhu et al., 2007) provided by the suppliers of the assay kit. This assay kit measures GPx activity indirectly by a coupled reaction with GR. Oxidised glutathione (GSSG) produced upon reduction of hydroperoxide by GPx is recycled to its reduced state by GR in the presence of NADPH. The oxidation of NADPH to NADP⁺ is accompanied by a decrease in absorbance at 340 nm and is directly proportional to the GPx activity in the sample. The decrease in absorbance was read with microplate reader and the activity is reported as nmol/min/mL of plasma or nmol/min/g of wet tissue.

**Glutathione reductase assay:** GR activity was measured following the procedure (Zhu et al., 2007) provided by the suppliers of the assay kit. GR activity was assayed spectrophotometrically by monitoring the oxidation of NADPH to NADP⁺ by GR at 340 nm. Briefly, 200 µL of reaction mixture contained 50 mM potassium phosphate (pH 7.5), 1 mM EDTA, 1 mM GSSG and 0.1 mM NADPH. The reaction was initiated by addition of 20 µL of supernatant of tissue homogenate. The decrease in absorbance at 340 nm was recorded at 60 sec intervals for 6 min. Each assay was performed in duplicate and enzyme units were reported as nmol NADPH oxidized/min/g of wet tissue or mL of plasma.

**Estimation of taurine levels:** The 0.45 gram of tissue was weighed and stored at -80°C until the assay. Taurine was assayed by the method described by Waterfield (1994) using high performance liquid chromatography with fluorimetric detection.

**Statistical analysis:** Differences among control and experimental groups were tested using one-way ANOVA with Scheffe multiple comparisons test (for significant ANOVA) after logarithmic transformation of the data to correct for normality (Rosner, 2000; Hair, 1998). Differences of p<0.05 were considered statistically significant.

**RESULTS**

**Effects on reduced glutathione (GSH) levels:** The GSH level is estimated in µM g⁻¹ of wet tissue or µM mL⁻¹ of plasma. Oral administration of taurine in rats (Group II, III and IV) resulted in a significant increase in liver GSH level compared with control rats at p<0.05 level. A significant dose dependent increase was observed in the stomach GSH level of the taurine administered rats (Group II, III and IV) compared to control rats (Group I) at p<0.05. In kidney the level of GSH was significantly decreased in the taurine administered groups (Group II, III and IV) compared to control group (Group I) at p<0.05. There was no significant difference in the heart or plasma GSH levels between the taurine administered rats (Group II, III and IV) and the control rats (Group I) (Table 1).

**Effects on glutathione peroxidase (GPx) activity:** The GPx activity is expressed as µM/min/g of wet tissue or µM/min/mL of plasma. GPx activity in liver showed a significant difference between Group I (Control) and Group III and IV (Medium dose-taurine and High dose-taurine) but not Group II (Low dose-taurine) The activity of GPx in the heart showed a significant increase in the taurine administered groups (Group II, III and IV) when compared to the control group (Group I) at p<0.05. A significant increase was observed in stomach GPx activity of the taurine administered groups (Group II, III and IV) compared to control rats (Group I) at p<0.05. A significant increase in the plasma GPx activity was observed in the Group III (Medium dose-taurine) and Group IV (High dose-taurine) compared to the Group I and II at p<0.05. There was no significant difference in the kidney GPx activity between the taurine administered rats (Group II, III and IV) and the control rats (Group I) (Table 2).

**Effects on glutathione reductase (GR) activity:** The GR activity is expressed as µM/min/g of wet tissue or µM/min/mL of plasma. Oral administration of taurine resulted in a significant decrease in the liver GR activity for the taurine administered rats (Group II, III and IV) compared to the control rats (Group I) at p<0.05. The GR activity in the kidney was significantly increased in the taurine groups (Group II, III and IV) compared to control rats at p<0.05. In plasma a significant decrease in GR activity was observed between the control (Group I) and taurine administered groups (Group II, III and IV) at
Table 1: Effects of exogenous taurine on GSH level in tissue and plasma

<table>
<thead>
<tr>
<th>Tissue/plasma</th>
<th>Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver (μmol/g of wet tissue)</td>
<td>50.51±2.52</td>
<td>103.02±11.49</td>
<td>105.78±14.69</td>
<td>107.86±10.29</td>
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<tr>
<td>Heart (μmol/g of wet tissue)</td>
<td>53.36±27.26</td>
<td>80.18±15.4</td>
<td>90.55±17.60</td>
<td>108.66±14.34</td>
<td></td>
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<tr>
<td>Stomach (μmol/g of wet tissue)</td>
<td>26.81±11.54</td>
<td>123.19±7.49</td>
<td>147.10±17.27</td>
<td>180.20±38.50</td>
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<tr>
<td>Kidney (μmol/g of wet tissue)</td>
<td>48.88±2.79</td>
<td>1.96±1.49</td>
<td>6.02±6.46</td>
<td>6.46±6.39</td>
<td></td>
</tr>
<tr>
<td>Plasma (μmol/mL of plasma)</td>
<td>1.48±0.97</td>
<td>1.58±0.52</td>
<td>1.55±0.24</td>
<td>1.35±0.01</td>
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</table>

Group I: Control; Group II: Low dose taurine; Group III: Medium dose taurine; Group IV: High dose taurine; a-Group I vs Group II; b-Group I vs Group III; c-Group I vs Group IV; d-Group II vs Group III; e-Group II vs Group IV; f-Group III vs Group IV; a to f represents the significant difference at p<0.05

Table 2: Effect of exogenous taurine on GPx activity in tissue and plasma

<table>
<thead>
<tr>
<th>Tissue/plasma</th>
<th>Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver (μmol/min/g of wet tissue)</td>
<td>11.06±3.87</td>
<td>14.85±2.47</td>
<td>16.79±1.81</td>
<td>18.85±5.79</td>
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<tr>
<td>Heart (μmol/min/g of wet tissue)</td>
<td>3.06±5.08</td>
<td>32.31±25.02</td>
<td>41.15±20.32</td>
<td>65.00±41.39</td>
<td></td>
</tr>
<tr>
<td>Stomach (μmol/min/g of wet tissue)</td>
<td>6.75±2.26</td>
<td>12.39±3.48</td>
<td>36.71±2.09</td>
<td>74.08±20.35</td>
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</tr>
<tr>
<td>Kidney (μmol/min/g of wet tissue)</td>
<td>11.24±9.04</td>
<td>10.41±5.51</td>
<td>11.28±3.83</td>
<td>15.06±5.94</td>
<td></td>
</tr>
<tr>
<td>Plasma (μmol/min/mL of plasma)</td>
<td>2.84±5.00</td>
<td>2.45±1.88</td>
<td>7.18±4.69</td>
<td>19.76±9.48</td>
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</tbody>
</table>

Group I: Control; Group II: Low dose taurine; Group III: Medium dose taurine; Group IV: High dose taurine; a-Group I vs Group II; b-Group I vs Group III; c-Group I vs Group IV; d-Group II vs Group III; e-Group II vs Group IV; f-Group III vs Group IV; a to f represents the significant difference at p<0.05

Table 3: Effect of exogenous taurine on GR activity in tissue and plasma

<table>
<thead>
<tr>
<th>Tissue/plasma</th>
<th>Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver (μmol/min/g of wet tissue)</td>
<td>58.62±29.15</td>
<td>12.79±5.56</td>
<td>12.40±1.59</td>
<td>14.09±10.01</td>
<td></td>
</tr>
<tr>
<td>Heart (μmol/min/g of wet tissue)</td>
<td>57.79±11.85</td>
<td>55.86±22.55</td>
<td>55.82±21.00</td>
<td>51.36±15.47</td>
<td></td>
</tr>
<tr>
<td>Stomach (μmol/min/g of wet tissue)</td>
<td>7.37±3.90</td>
<td>6.64±3.94</td>
<td>6.76±3.53</td>
<td>6.30±4.27</td>
<td></td>
</tr>
<tr>
<td>Kidney (μmol/min/g of wet tissue)</td>
<td>2.57±0.64</td>
<td>5.24±1.34</td>
<td>6.12±2.12</td>
<td>20.22±2.95</td>
<td></td>
</tr>
<tr>
<td>Plasma (μmol/min/mL of plasma)</td>
<td>4.83±1.75</td>
<td>3.00±0.87</td>
<td>3.23±0.90</td>
<td>3.30±1.49</td>
<td></td>
</tr>
</tbody>
</table>

Group I: Control; Group II: Low dose taurine; Group III: Medium dose taurine; Group IV: High dose taurine; a-Group I vs Group II; b-Group I vs Group III; c-Group I vs Group IV; d-Group II vs Group III; e-Group II vs Group IV; f-Group III vs Group IV; a to f represents the significant difference at p<0.05

Table 4: Effect of exogenous taurine on tissue level in tissue and plasma

<table>
<thead>
<tr>
<th>Tissue/plasma</th>
<th>Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver (μmol g⁻¹ of wet tissue)</td>
<td>23.62±0.54</td>
<td>104.3±80.98</td>
<td>334.8±122.5</td>
<td>695.8±105.3</td>
<td></td>
</tr>
<tr>
<td>Heart (μmol g⁻¹ of wet tissue)</td>
<td>629.3±388.39</td>
<td>524.7±111.68</td>
<td>506.7±54.13</td>
<td>148.31±46.24</td>
<td></td>
</tr>
<tr>
<td>Stomach (μmol g⁻¹ of wet tissue)</td>
<td>357.2±190.46</td>
<td>332.0±157.94</td>
<td>233.4±162.2</td>
<td>122.9±42.73</td>
<td></td>
</tr>
<tr>
<td>Kidney (μmol g⁻¹ of wet tissue)</td>
<td>381.9±35.47</td>
<td>52.89±18.39</td>
<td>71.91±22.3</td>
<td>134.9±9.61</td>
<td></td>
</tr>
<tr>
<td>Plasma (μmol mL⁻¹ of plasma)</td>
<td>5.31±0.36</td>
<td>5.45±0.32</td>
<td>5.40±0.28</td>
<td>5.40±0.41</td>
<td></td>
</tr>
</tbody>
</table>

Group I: Control; Group II: Low dose taurine; Group III: Medium dose taurine; Group IV: High dose taurine; a-Group I vs Group II; b-Group I vs Group III; c-Group I vs Group IV; d-Group II vs Group III; e-Group II vs Group IV; f-Group III vs Group IV; a to f represents the significant difference at p<0.05

p<0.05. There was no significant difference in the heart and stomach GR activity between the taurine administered rats (Group II, III and IV) and the control rats (Group I) (Table 3).

Effects on taurine levels in tissue and plasma: The taurine levels in the tissue and plasma were expressed as nM g⁻¹ of wet tissue and nM mL⁻¹ of plasma, respectively. In liver taurine level a significant dose dependent increase was observed in the taurine administered groups (Group II, III and IV) compared to control rats at p<0.05. A significant decrease in the levels of taurine in the heart was observed in the group IV (High dose-taurine) rats when compared to control rats (Group I) at p<0.05. The taurine levels in stomach was significantly lowered in taurine administered groups when compared to the control group (Group I) at p<0.05. The taurine level in the kidney was significantly decreased in the taurine administered groups than control at p<0.05. There was no significant difference in the plasma taurine level between the taurine administered rats (Group II, III and IV) and control rats (Group I) (Table 4).

DISCUSSION

In the present study a dose dependent increase in GSH levels following taurine administration was observed in liver, heart and stomach. GSH levels in the tissue are primarily dependent upon the rates of biosynthesis and utilization in oxidation/reduction reactions (Rebrin et al., 2005). Cetiner et al., (2005) studied on effect of taurine on methotrexate induced toxicity in rat livers and reported that taurine does not have a known direct effect on GSH biosynthesis but it reduces the consumption of GSH by...
inhibiting the LPO reactions, a marker for free radical induced damage. Similarly, Miyazaki et al. (2004) also reported that administration of taurine (TAU) increased the GSH levels in rats due to the inhibition of GSH oxidation to prolong the exercise performance. TAU and GSH biosynthesis have the common precursor cysteine (Rebrin et al., 2005; Dawson et al., 2002). In a study on sulfur containing amino acids on ageing (Benedetti et al., 1991) and in another study on cyclosporine-A induced oxidative stress and hepatotoxicity in rats (Hagar, 2004) it has been pointed out that TAU increased the GSH levels as a result of directing more amount of cysteine into GSH biosynthesis. Schaffer et al. (2003) also agreed to the above statement in their review on cytoprotective role of taurine. However, in kidney, GSH level was significantly decreased in the taurine administered animals and plasma GSH level remained unchanged. Harris (1992) reviewed on regulation of antioxidant enzymes had pointed out that the antioxidant enzymes levels also depend on factors such as organ specificity, age of the organism etc. The decrease in kidney GSH contrary to the increase observed in liver, heart and stomach in the present study could be due to the organ specificity and presently the mechanism for this decrease is not known.

Similarly, a dose dependent increase in GPx activity of liver, heart, stomach and plasma in the taurine administered rats is seen. Sinha et al. (2008) have shown that taurine increased the GSH levels, GPx and GR activity of erythrocytes in cadmium induced toxicity rats by inhibiting the lipid peroxidation. Yildirim et al. (2007) have reported that taurine is effective in decreasing liver MDA levels and increasing GSH content and GPx activity when given to 13-14 months old rats at a dose of 200 mg kg/day ip for a week. An increase in GPx activity and GSH level following taurine administration similar to our study is reported by Conthair et al. (1999) in individuals with chronic beryllium disease and suggested that the increase in GPx may be mediated through redox mechanisms which lead to increase in GSH levels. Similarly, Sudheesh et al. (2010) reported that treatment with Palladium \(\alpha\)-lipoic acid formulation (POLY-MVA) significantly increased the heart mitochondrial GSH level and GPx activity and pointed out that the enhanced activity of GPx can be attributed with the high level of GSH.

In the present study, GR activity showed a significant decrease in liver and plasma. Ulusu et al. (2003) in their study using streptozotocin induced diabetic rats have pointed out that GR is responsible for the regeneration of GSH and the decrease in GR activity in brain and heart may be due to the decline in production and availability of GSH to overcome \(\text{H}_2\text{O}_2\). In kidney, GR activity was significantly increased and remained unchanged in heart and stomach of the taurine administered rats. Mallikarjuna et al. (2008) has reported that the increased GR activity is an indicative of increased GSH concentration via GSSG reaction at cellular level. Antioxidant enzyme activity is tissue dependent and varies from tissue to tissue and the duration and severity of the pathological conditions (Ulusu et al., 2003). In alloxan induced diabetic rabbits, Winiarska et al. (2009) also showed that the effect of taurine on the activity of the enzymes of glutathione metabolism appear to be tissue dependent. In our study also GR activity varied from tissue to tissue and this might be due to the tissue specificity or glutathione availability. The kidney is the organ that excretes any excessive intake of the taurine (Sved et al., 2007). The high intake of taurine may act as a stimulus for the greater excretion of taurine by the kidney resulting in low levels of taurine in kidney (Table 4). The low level of taurine’s antioxidant activity is probably compensated by the GSH resulting in a decreased GSH level in kidney (Table 1). The low level of GSH may stimulate the GR activity. The threshold for maximum stimulation may be reached at the concentration administered in high dose group and hence responsible for the sharp rise in GR activity seen in Group IV (Table 3).

Liver taurine levels were significantly increased in a dose dependent manner in taurine administered animals. Parikkar-Karpuzoglu et al. (2007) and Parikdar et al. (2008) observed that administration of taurine in old and young rats depleted of taurine using \(\beta\)-alanine pre-treatment resulted in significant increase in liver and heart taurine levels. In the study of Eppler and Dawson (2001), 18 month old male Fischer rats were treated with taurine (1.5% in drinking water) for 8 months and reported that taurine concentration in liver and kidney was significantly higher in supplemented rats. Dawson et al. (1999) observed that TAU supplementation (1.5% of TAU in drinking tap water) in old rats can significantly increase TAU content in liver and kidney of aged male Fischer rats. Similarly, in the present study we observed that taurine levels in the kidney were significantly increased in the taurine administered animals.

CONCLUSION

The present study suggests that long term oral administration of taurine may affect the GSH level, GPx and GR activity of organs and the effects vary from organ to organ. Apparently, more studies are necessary to establish the role of oral administration of antioxidants like taurine in normal rats on physiological functions.

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REFERENCES


