Taurine Prevents Oxidative Damage of High Glucose-Induced Cataractogenesis in Isolated Rat Lenses

Hee-Young Son¹, Harriet Kim¹ and Young H Kwon¹².*

¹ Department of Food and Nutrition, and ²Research Institute of Human Ecology, Seoul National University, Seoul, 151–742, Korea

(Received September 19, 2006)

Summary Diabetic cataract is an ocular disease represented as blindness by lens opacification. Oxidative as well as osmotic stress caused by accumulation of polyols within the lens has been shown to be associated with glucose-induced cataractogenesis. Taurine has an antioxidant capacity and its level in diabetic cataractous lens is markedly decreased. Therefore, we investigated whether taurine is a part of antioxidative defense mechanism involved in protecting the lens against high glucose-induced oxidative stress and tissue damage. Lenses were isolated from male Sprague-Dawley rats weighing about 180–200 g and cultured in high glucose medium (55.6 mM) for 6 d as a model of high glucose-induced cataractogenesis. To investigate the antioxidative effect of taurine, 30 mM taurine was added in normal medium for 2 d before the addition of high glucose. The culture of lenses in high glucose medium increased the weight and opacity of lenses of and the carbonylated protein level, and decreased glutathione (GSH) content. Although there were no significant effects of taurine on the weight or opacity of lenses, pretreatment of lenses with 30 mM taurine significantly reversed the level of protein carbonylation and GSH to those of controls. Therefore, taurine might spare GSH and protect the lens from oxidative stress induced by a high concentration of glucose.

Key Words cataract, oxidative stress, glucose, taurine, GSH

Diabetic cataract, one of the secondary complications of diabetes, is the major cause of blindness due to the opacification of the lens. The lens is the only transparent organ in the body and focuses light on the retina. The main components of the lens are about 63% of water and 35% of protein which is composed of over 90% of crystallins (1). Because the turnover rate of crystallins is very slow compared to many other organs, the reversal of protein modification by several factors is slow and it can be thought to induce cataract (2). Oxidative as well as osmotic stress caused by accumulation of polyols within the lens has been shown to be associated with glucose-induced cataractogenesis. Taurine has an antioxidant capacity and its level in diabetic cataractous lens is markedly decreased. Therefore, we investigated whether taurine is a part of antioxidative defense mechanism involved in protecting the lens against high glucose-induced oxidative stress and tissue damage. Lenses were isolated from male Sprague-Dawley rats weighing about 180–200 g and cultured in high glucose medium (55.6 mM) for 6 d as a model of high glucose-induced cataractogenesis. To investigate the antioxidative effect of taurine, 30 mM taurine was added in normal medium for 2 d before the addition of high glucose. The culture of lenses in high glucose medium increased the weight and opacity of lenses of and the carbonylated protein level, and decreased glutathione (GSH) content. Although there were no significant effects of taurine on the weight or opacity of lenses, pretreatment of lenses with 30 mM taurine significantly reversed the level of protein carbonylation and GSH to those of controls. Therefore, taurine might spare GSH and protect the lens from oxidative stress induced by a high concentration of glucose.

MATERIALS AND METHODS

Chemicals. Most reagents were obtained from Sigma...
Chemical Co. (St. Louis, MO, USA), unless otherwise stated. Medium 199 (M199), glutamine, gentamycin, and fungizone were purchased from Invitrogen (Carlsbad, CA, USA).

**Lens organ culture.** Male Sprague-Dawley rats weighing about 180–200 g were killed by CO₂ inhalation and the eyes were extracted. The rats were maintained and handled according to the policies and procedures of the Institutional Animal Care and Use Committee of the Seoul National University. The lenses were carefully removed by a posterior approach and immediately incubated in 24-well cluster plates containing Medium 199 based on the method of Spector et al. (19). The final preparation had an osmolarity of 290–300 mOsm. Lenses culture was maintained at 37°C with an atmosphere of 95% air/5% CO₂. No metal instruments that could cause damage were used to handle the lenses. Damaged lenses which rapidly lost their transparency, were excluded from the experiments and lens viability was also checked by measuring protein leakage in incubation media after 24 h of incubation (20). Lenses were divided randomly into four groups. The lenses from the first group incubated in a normal medium (M199) served as a control (n=13). The second group of lens was incubated in a normal medium for 6 d after being incubated in a medium containing 30 mM taurine for 2 d (n=12). The third group was incubated in a normal medium for 2 d and then in a medium containing 55.6 mM glucose for 6 d (n=12). The fourth group was incubated in a medium containing 30 mM taurine for 2 d and then in a medium containing 55.6 mM glucose for 6 d (n=13). The medium was changed every 24 h. Glucose-treated lenses turned opaque within 6 d. After culture, lenses of each group were divided into three sets (n=3–4 each set) and were used for biochemical measurements.

**Measurement of the lenticular opacity.** Photographs of the lens were taken using a dark-field set up with illumination from a circular light situated above the lens by microscope equipped with a CCD camera (Olympus, Japan). The outline of the lens image was determined by selecting 4 points on the image, and then the transparent area within the outline and thread level were set automatically by the software (21). The total area of opacity, in pixels, was analyzed by a computer using Image Pro Plus system (Media Cybernetics, USA). The lenses were classified into 6 grades according to the density of opacity, which is expressed as an arbitrary unit of pixels (Grade I: <20, Grade II: 20–30, Grade III: 30–40, Grade IV: 40–50, Grade V: 50–60, Grade VI: >60).

**Determination of the lenticular GSH level.** The GSH level in the lens was determined by Ellman’s reaction (22). Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0. Proteins was precipitated by centrifugation after the addition of an equal volume of a 20% TCA solution. The supernatant was then mixed with eight-times volume of 0.3 M sodium phosphate and an equal volume of 5,5′-dithiobis(2-nitrobenzoic acid) (DTNB) solution prepared by dissolving 4 mg of DTNB in 10 mL of 1% trisodium citrate solution. The absorbance was measured spectrophotometrically at 410 nm and GSH level was calculated with reference to the standards.

**Determination of the lenticular taurine level.** The level of taurine in the lens was determined by HPLC. Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0 and homogenate was centrifuged at 10,000 ×g for 5 min. Proteins were precipitated by centrifugation after the addition of acetonitrile. The supernatant was incubated with dabsyl chloride, a derivatizer, at 70°C for 10 min and then filtered. The column was Waters symmetry C18 (3.9×150 mm) and mobile phases were composed of solution A (20 mM sodium acetate, 0.05% triethylamine, pH 6.5) and solution B (acetonitril) (73:27). The mobile rate was 1.0 mL/min.

**Determination of glutathione peroxidase activity.** The glutathione peroxidase activity was determined by the procedure of Tappel (23). Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0 and homogenate was centrifuged at 10,000 ×g for 20 min. Twenty microliters of the supernatant were then mixed with 310 µL of the assay mixture (50 mM Tris, pH 7.6, 0.1 mM EDTA, 0.25 mM GSH, 0.12 mM NADPH, and 1 U/mL glutathione reductase). The reaction was initiated by addition of 10 µL of cumene hydroperoxide and absorbance at 340 nm was recorded. The activity was calculated using a molar extinction coefficient for NADPH of 6.22 µmol⁻¹cm⁻¹ at 340 nm and expressed in units per milligram of protein.

**Determination of glutathione reductase activity.** The glutathione reductase activity was determined by the procedure of Carlberg and Mannervik (24). Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0 and the homogenate was centrifuged at 10,000 ×g for 20 min. Twenty microliters of the supernatant were then mixed with 400 µL of the assay mixture (0.1 M potassium phosphate buffer, pH 7.0, 1 mM EDTA, 0.1 mM NADPH, and 1 mM oxidized glutathione) and incubated at 30°C. The absorbance at 340 nm was recorded and the activity was expressed in units per milligram of protein. To calculate the specific enzyme activity, protein in each sample was measured by the method of Lowry et al. (25).

**Determination of carbonylated protein level.** Protein carbonyl content in the lens was analyzed as described by Levine et al. (26). Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0 and were further diluted to 0.5 mg protein/mL in 0.1 M potassium phosphate buffer, pH 7.0. Proteins were precipitated by centrifugation after the addition of an equal volume of a 20% TCA solution and the pellet was mixed with 1 mL of 10 mM 2,4-dinitrophenylhydrazine (DNPH) in 2 N HCl and kept at room temperature for 1 h with vortexing every 10 min. Five hundred microliters of 20% TCA was added and the precipitated proteins were subsequently washed three times with ethanol : ethylacetate (1:1). The final pellet was redi-
solved in 1 mL of 6 M guanidine solution, pH 2.3. After removing any insoluble material by centrifugation at 10,000 × g for 10 min, the absorption of the supernatant was measured spectrophotometrically at 370 nm. The blank of each sample was prepared by treatment with 2 N HCl instead of 10 mM 2,4-DNPH in 2 N HCl.

**Determination of TBARS level.** The level of thiobarbituric acid reactive substances (TBARS) in the lens homogenate was analyzed according to the method of Ohkawa et al. (27). Briefly, lenses were homogenized in 10 volumes of 0.1 M potassium phosphate buffer, pH 7.0 and were further diluted to 2 mg protein/mL in 0.1 M potassium phosphate buffer, pH 7.0. Five hundred microliters of homogenate were incubated at 37°C for 1 h, and then 500 μL of 10% (w/v) TCA and 500 μL of 0.67% thiobarbituric acid were added to the reaction mixture. After vortexing vigorously for 30 s and the reaction mixture was boiled for 15 min, cooled, and then centrifuged at 2,500 × g for 15 min. Finally, the absorption of the supernatant was measured spectrophotometrically at 535 nm. As a standard, 1,1,3,3-tetraethoxypropane in methanol was run simultaneously.

**Statistical method.** Statistical analyses were performed using SAS statistical programs. Values were expressed as the mean±SE. Analysis of variance coupled with Duncan’s new multiple-range test was used to detect the mean differences among groups. A value of p<0.05 was considered statistically significant.

**RESULTS**

Rat lenses were pre-cultured in 30 mM of taurine in this study and the antioxidative effect of 30 mM taurine measured by chemiluminescence assay using an ABEL-microperoxidase-H2O2 system showed 34.8% inhibition. The wet weights of lenses cultured in the different media were measured (Fig. 1). It has been known that the wet weight of lenses increases progressively with the stage of maturation of cataract (28). The weight of lenses incubated in high glucose medium was significantly increased compared to that of control lenses (p<0.05). After being cultured in a medium with taurine, high glucose-treated lenses were still significantly heavier than those of the control group (p<0.05). This result showed that the high concentration of glucose increased the weight of the lenses and taurine had no effect on decreasing that weight.

To examine whether there is a change in the lens opacity from a high concentration of glucose and the addition of taurine, the opacity of the lenses was measured individually using the Image pro analysis system (Fig. 2). An increase in grade indicates an increase in the opacity of lenses. The control group was distributed at II–III, while the high glucose group without taurine was at III–V, showing opacity of the glucose-treated lenses increased markedly. The level of opacity of high glucose group with 30 mM taurine was at II–V. With the change of the lenticular weight, the opacity of the lens clearly increased with a high concentration of glucose, but taurine did not interfere with this increase of the opacity of the lens.

**Fig. 1.** Wet weight of rat lenses cultured in the presence of 30 mM taurine and/or high (55.6 mM) glucose medium. Lenses were cultured in the media as follows. CON: Lenses were cultured in 5.6 mM glucose medium (M199) for 8 d; CON+TAU: Lenses were cultured in M199 for 6 d after being cultured in 30 mM taurine-supplemented M199 for 2 d; HGC: Lenses were cultured in 55.6 mM glucose medium (HGC) for 6 d after being cultured in M199 for 2 d; HGC+TAU: Lenses were cultured in HGC for 6 d after being cultured in 30 mM taurine-supplemented M199 for 2 d. Values are mean±SE (n = 3–4). Means with different superscripts are significantly different at p<0.05.

**Fig. 2.** The distribution of lens opacity cultured in the presence of 30 mM taurine and/or high (55.6 mM) glucose medium. The figure shows the percentage of rat lenses with different grades of opacity as described in “Materials and Methods.” The experiment group is described in Fig. 1.

The lens has several enzymatic and non-enzymatic antioxidants to protect it against ROS-mediated damage (14). The anticaataract effect of a variety of natural and synthetic compounds has been attributed to their antioxidant properties (29); therefore, we measured several biomarkers of oxidative stress in the lens. Figure 3A presents the GSH levels of lenses after being cultured in different media for 8 d. The result showed that the GSH level in the high glucose only group was significantly decreased compared to that in the control group. The value of the taurine-supplemented group was significantly higher than the value of the group treated with glucose only, indicating that the addition of taurine to the medium of the high-glucose group reversed the
Antioxidativity of Taurine in High Glucose-Induced Cataractogenesis

To determine the fate of taurine after exhibiting antioxidative activity, we measured the level of lenticular taurine (Fig. 3B). Taurine concentration in glucose-treated lenses decreased distinctly compared to the control group ($p<0.05$). The level of taurine in the taurine-pretreated group was also significantly decreased compared to that of the control group ($p<0.05$) and was not different from that of the high glucose group without taurine. Therefore, GSH is thought to be an important factor in cellular function.

Table 1. The activities of glutathione peroxidase and glutathione reductase of rat lenses cultured in M199 in the presence of 30 mM taurine and/or high (55.6 mM) glucose medium.

<table>
<thead>
<tr>
<th></th>
<th>Glutathione peroxidase (μmol NADPH oxidized/min/mg protein)</th>
<th>Glutathione reductase (μmol NADPH oxidized/min/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>56.40±2.37NS</td>
<td>14.93±0.72NS</td>
</tr>
<tr>
<td>Control+taurine</td>
<td>60.12±1.88</td>
<td>12.08±1.43</td>
</tr>
<tr>
<td>High glucose</td>
<td>56.18±5.57</td>
<td>12.88±3.05</td>
</tr>
<tr>
<td>High glucose+taurine</td>
<td>61.86±3.04</td>
<td>15.72±1.11</td>
</tr>
</tbody>
</table>

Lenses were cultured in the media as follows. Control: Lenses were cultured in 5.6 mM glucose medium (M199) for 8 d; Control+taurine: Lenses were cultured in M199 for 6 d after being cultured in 30 mM taurine-supplemented M199 for 2 d; High glucose: Lenses were cultured in 55.6 mM glucose medium (HGC) for 6 d after being cultured in M199 for 2 d; High glucose+taurine: Lenses were cultured in HGC for 6 d after being cultured in 30 mM taurine-supplemented M199 for 2 d. Values are mean±SE ($n=3–4$). NS: Means are not significantly different.
and defense against oxidative stress. We also measured total protein content per lens to determine the possibility of protein leakage due to the loss of cell viability. Although the levels of GSH and taurine were significantly decreased, there was no significant decrease in total protein content in the high-glucose treated group (data not shown), suggesting the cultured lenses retained their viability. The activities of the antioxidative enzymes, glutathione peroxidase and glutathione reductase, were not significantly changed by the addition of either glucose or taurine (Table 1).

The levels of carbonylated protein (Fig. 4A) and TBARS (Fig. 4B) were measured to investigate whether lenses were damaged by free radicals or not. A carbonyl group can be introduced into proteins by glycation and glycoxidation reactions. Therefore, protein carbonyl groups provide a reasonable marker for free radical-induced protein oxidation (30). The level of carbonylated protein in the high glucose with taurine group was significantly lower than that in the high glucose without taurine group and was reversed to that of the control group (p<0.05). Although the TBARS level was not significantly changed by the treatment of high glucose or taurine supplementation, protein peroxidation and lipid peroxidation progressed together with a highly positive correlation (r=0.8086, p=0.0001), suggesting that oxidative stress induced by the high concentration of glucose was decreased by taurine.

**DISCUSSION**

Here, we show that taurine prevents oxidative damage of high glucose-induced cataractogenesis in isolated rat lenses. Oxidative stress has been suggested as a common underlying mechanism of cataractogenesis, and augmentation of the antioxidant defenses of the lens has been shown to prevent or delay cataract. The ubiquitously found $\beta$-amino acid taurine has been shown to possess a number of cytoprotective properties through its actions as an antioxidant, osmoregulator, and intracellular Ca$^{2+}$ flux regulator (31). Although taurine is considered as an inert compound chemically, taurine is capable of converting reactive hypochlorous acid into...
In the present study, the presence of high glucose in the medium induced a significant physiological stress to lenses, as evidenced by the increase in lens weight and opacity. This result is consistent with previous studies in which the lens was cultured in 30 mM xylose (33) or 30 mM galactose medium (34). These changes in lenses are thought to be due to the water absorption into the cells because the lenticular membrane was damaged by oxidative stress, and osmotic imbalance was caused by polyol pathway activity (28). However, in the present study, the pretreatment of lenses with 30 mM taurine could not reverse the changes in lenticular weight or opacity induced by cataract development.

Among the systems that defend against ROS in the lens are a high level of GSH, abundant antioxidant enzymes, and the chaperone-like functions of crystallins. The oxidative stress response of the lens is characterized by a diminishing level of GSH and reduced activities of antioxidant enzymes (35, 36). The GSH concentration in the high glucose group decreased significantly compared to that in the control group as shown in previous studies (16, 37). The decrease of GSH in the high glucose group is thought to be due to the faster GSH efflux under hyperosmotic conditions caused by an accumulation of sorbitol. In addition, the synthesis of GSH in the lens could be also decreased because the concentration of amino acids in the lens decreased as a function of osmotic pressure (15, 37). GSH is synthesized by a chain of processes from amino acids absorbed through the lens membrane (38). GSH in the cultured lenses was about 40% lower than that in non-cultured lenses within 24 h and 60% lower within 72 h, indicating that GSH concentration was sensitive to oxidative stress (39). The activities of glutathione peroxidase and glutathione reductase were not changed significantly by the treatment with glucose and/or taurine in our study. Lou et al. (37) found that under hyperglycemic conditions, there was no change in the enzyme glutathione reductase activity. They concluded that the decreased membrane transport of amino acids which are needed for GSH biosynthesis and the simultaneous loss of GSH through leaky membranes, initiated by the polyol pathway, can be responsible for the drastic GSH depletion. In a recent study using streptozotocin-induced diabetic rats, the levels of gamma-glutamylcysteine synthetase mRNA and protein were not reduced in the diabetic rats (40), suggesting that the availability of substrate may have an important role in the synthesis of GSH, rather than the level of involved enzyme.

The level of GSH in a taurine-treated high glucose group increased significantly compared to the high glucose group without taurine and was similar to the control group, indicating that taurine is involved in the action of GSH. It has been shown that the addition of taurine increased the level of GSH in the lens oxidatively stressed by menadione (14), suggesting that taurine could spare GSH. In fact, we measured the lenticular taurine content after culturing for 8 d and found that taurine content in the glucose-treated group with or without taurine decreased significantly compared to that in the control. But the taurine content of the lenses cultured in taurine-supplemented medium for 2 d was higher than that of the control lenses, indicating that taurine added to the high-glucose medium may be used in protecting the lens against oxidative stress. Other studies also suggested that the decrease of taurine in diabetic cataractous lenses might be directly due to the oxidative stress (15, 34). In addition, intracellular accumulation of sorbitol is most likely to cause depletion of taurine, since specific transporter systems exist for taurine, not for sorbitol (32).

To investigate the antioxidant activity of taurine, levels of TBARS and carbonylated protein were also measured. It is thought that the lens is sensitive to oxidative stress and the increase of oxidative stress by high glucose could be a major cause of cataract. Many studies have reported the increase of MDA concentration in cataractous lens. MDA concentration of rabbit lenses treated in 30 mM galactose for 3 d increased significantly (34). It was also reported that the concentration of carbonylated protein in diabetic lenses increased compared to normal lenses (41, 42). Taurine also functions as a modulator of intracellular Ca²⁺, which is also involved in oxidative stress-mediated cell injury (31). A recent study also showed that inhibition of peroxidation markers and upregulation of antioxidant activity in rat tissues by taurine signify the potential utility of taurine as an adjunct in the treatment of insulin resistance (43).

In the present study, taurine reversed the levels of oxidative stress and kept the GSH level high, although the physiological characteristics of the cataracts were not changed by the addition of taurine. Therefore, taurine seems to act as an antioxidant with GSH in high glucose-induced cataracts and further studies on the significance of taurine in lens physiology and cataract formation will be needed.

**REFERENCES**

6) Linklater HA, Dzialoszynski T, McLeod HL, Sanford SE.
Trevisich JK. 1990. Modelling cortical cataractogene-
sis XI: Vitamin C reduces gamma-crystallin leakage
7) Jain A, Lim G, Langford M, Jain S. 2002. Effect of high-
glucose levels on protein oxidation in cultured lens cells,
and in crystalline and albumin solution and its inhibi-
tion by vitamin B6 and N-acetylcysteine: its possible rel-
ance to cataract formation in diabetes. *Free Radic Biol
8) Mitton KP, Linklater HA, Dzialoszynski T, Sanford SE,
Starkey K, Trevisich JK. 1999. Modelling cortical cata-
racogenesis 21: In diabetic rat lenses taurine supple-
mentation partially reduces damage resulting from
osmotic compensation leading to osmolyte loss and
9) Mitton KP, Dean PW, Dzialoszynski T, Xiong H, Sanford
SE, Trevisich JK. 1993. Modelling cortical cataracto-
genesis XIII: Early effects of lens ATP/ADP and glu-
tathione in the streptozotocin rat model of the diabetic
food factors in oxidative stress caused by hyperglycemia.
11) Wolff SP, Dean RT. 1987. Glucose autoxidation and pro-
tein modification. The potential role of ‘autoxidative gly-
*Physiol Rev* 72: 101–163.
13) Aruoma O, Halliwell B, Hoey B, Butler J. 1988. The anti-
oxidant action of taurine, hypotaurine and their meta-
stress to rat lens in vitro: protection by taurine. *Free
Modelling cortical cataractogenesis 22: Is in vitro red-
uction of damage in model diabetic rat cataract by
taurine due to its antioxidative activity? *Exp Eye Res*
16) Obrosova I, Cao X, Greene DA, Stevens MJ. 1998. Diabe-
tes-induced changes in lens antioxidant status, glucose
utilization and energy metabolism: effect of D.L-α-lipoic
supplementation on GSH and NAD(P)-redox status, lipid
peroxidation, and energy metabolism in diabetic precat-
18) Di Leo M, Santini S, Silveri N, Giardina B, Franco
ci E, Ghirlanda G. 2004. Long-term taurine supplemen-
tation reduces mortality rate in streptozotocin-induced
1993. The prevention of cataract caused by oxidative
stress in cultured rat lenses I: H2O2 and photochemi-
20) Tumminia SJ, Qin C, Zigler JS, Russell P. 1994. The inte-
grity of mammalian lenses in organ culture. *Exp Eye
21) Nagai N, Ito Y, Inomata M, Shumiyama S, Tae H, Hata-
guchi Y, Nakagawa K. 2006. Delay of cataract develop-
ment in the shumiya cataract rat by the administration of drinking
water containing high concentration of magnesium
22) Ellman GL. 1959. Tissue sulfhydryl groups. *Arch Bio-
chem Biophys* 82: 70–77.
23) Tappel AL. 1978. Glutathione peroxidase and hydroper-
25) Lowry O, Roseborough N, Farr A, Randall R. 1951. Pro-
tein measurement with the Folin phenol reagent. *J Biol
26) Levine R, Garland D, Oliver C, Amici A, Climenti L, Lenz
27) Ohkawa H, Ohishi N, Yagi K. 1979. Assay for lipid perox-
idation in animal tissues by thiobarbituric acid reaction.
28) Beyer-Mears A, Kelly K, Cruz E. 1985. Synergism of sor-
binal and normal diet on reversal of stage-II sugar cata-
29) Seddon J. 2007. Multivitamin-multimineral supple-
ments and eye disease: age-related macular degenera-
30) Moskovitz J, Yin M, Chock P. 2002. Free radicals and
31) Franco
ci D, Lio M, Bennardini F, Ghirlanda G. 2004. Is tau-
32) Hansen S. 2001. The role of taurine in diabetes and the
development of diabetic complications. *Diabetes Metab
33) Desouky MA, Geller AM, Jernigan HMJ. 1992. Effect of
osmotic stress on phosphorylcholine efflux and turn-
34) Malone JI, Benford SA, Malone JI. 1993. Taurine pre-
vents galactose induced cataracts. *J Diabetes Complica-
36) Ganea E, Harding J. 2006. Glutathione-related enzymes
37) Lou MP, Dickerson JEJ, Garadi R, York BMJ. 1988. Glu-
tathione depletion in the lens of galactosemic and dia-
38) Reddy V. 1979. Dynamics of transport systems in the
eye. Friedenwald Lecture. *Invest Ophthalmol Vis Sci* 18:
1000–1018.
Investigations into the loss of glutathione from lenses in
40) Patriarca S, Sufaro A, Domenicotti C, Oddeti P, Cotta-
laso D, Marinari U, Pronzato M, Traverso N. 2005. Sup-
plementation with N-acetylcysteine and taurine failed
to restore glutathione content in liver of streptozotocin-
induced diabetic rats but protected from oxidative stress.
*Biochim Biophys Acta* 1741: 48–54.
41) Suryanarayana P, Saraswat M, Mrudula T, Krishna T,
Krishnaswamy K, Reddy G. 2005. Curcumin and tur-
meric delay streptozotocin-induced diabetic cataract in
42) Kyselova Z, Garcia S, Gajdosikova A, Gajdosik A, Stefek
oxidation and cataract development in streptozotocin-
43) Nandhini A, Thirunavukkarasu V, Ravichandran M,
Anuradha C. 2005. Effect of taurine on biomarkers of
oxidative stress in tissues of fructose-fed insulin-resist-