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## Effect of dietary taurine supplementation on GSH and NAD(P)-redox status, lipid peroxidation, and energy metabolism in diabetic precataractous lens.

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## Source

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## Abstract

**PURPOSE:** To evaluate changes in glutathione and NAD(P)-redox status, taurine and malondialdehyde (MDA) levels, glucose utilization, and energy metabolism in diabetic precataractous lenses and to assess whether these changes can be prevented with dietary taurine supplementation.

**METHODS:** The experimental groups included control and streptozotocin-diabetic rats with a 3-week duration of diabetes fed unsupplemented or taurine (1% or 5%)-supplemented diets. The levels of glucose, sorbitol, fructose, myo-inositol, oxidized glutathione (GSSG), glycolytic intermediates, malate, alpha-glycerophosphate, and adenine nucleotides were assayed in individual lenses spectrofluorometrically by enzymatic methods, reduced glutathione (GSH) spectrofluorometrically with O-phthaldialdehyde, MDA colorimetrically with N-methyl-2-phenylindole, and taurine by high-performance liquid chromatography. Free cytosolic NAD+/NADH and NADP+/NADPH ratios were calculated from the lactate dehydrogenase and malic enzyme systems.

**RESULTS:** Sorbitol pathway metabolites and MDA were increased, and GSH and taurine levels were reduced in diabetic rats versus controls. The profile of glycolytic intermediates (an increase in glucose 6-phosphate, no change in fructose 6-phosphate and fructose 1,6-diphosphate, an increase in dihydroxyacetone phosphate, a decrease in 3-phosphoglycerate, phosphoenolpyruvate, and pyruvate, and no change in lactate), and a 9.2-fold increase in alpha-glycerophosphate suggest diabetes-induced inhibition of glycolysis. Free cytosolic NAD+/NADH ratios, ATP levels, ATP/ADP, and adenylate charge were reduced, whereas free cytosolic NAD+/NADH ratios were elevated. Lens taurine levels in diabetic rats were not affected by supplementation with 1% taurine. With 5% taurine supplementation, they were increased approximately 2.2-fold higher than those in untreated diabetics but remained 3.4-fold lower than in controls. Lens GSH levels were similar in diabetic rats fed unsupplemented and 5% taurine-supplemented diets, whereas GSSG and MDA levels and GSSG/GSH ratios were reduced by 5%

taurine supplementation. The decrease in free cytosolic NAD+/NADH, ATP/ADP, and adenylate energy charge were ameliorated by 5% taurine supplementation, whereas accumulation of sorbitol pathway intermediates, depletion of myoinositol, inhibition of glycolysis, a decrease in ATP and total adenine nucleotide, and an increase in free cytosolic NADP+/NADPH were not prevented.

**CONCLUSIONS:** Dietary taurine supplementation ameliorates MDA levels, GSSG/GSH, and NAD+/NADH and fails to prevent the osmotically mediated depletion of GSH and taurine and the decrease in glucose utilization and ATP levels in diabetic precataractous lens. Dietary taurine supplementation cannot be regarded as an alternative to aldose reductase inhibition in eliminating antioxidant and metabolic deficits contributing to diabetes-associated cataractogenesis.

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