ABSTRACT

Purpose: Cataract is the major cause of blindness and is associated with oxidative damage of the lens. In the present study, the aim was to evaluate the protective effects of epigallocatechin gallate (EGCG) on sodium selenite-induced cataractogenesis in Sprague-Dawley rat pups.

Methods: Eight-day-old Sprague-Dawley rat pups were randomly divided into four treatment groups: (1) normal control (vehicle administration); (2) sodium selenite-treated; (3) sodium selenite + 100 mg/kg body weight EGCG; and (4) sodium selenite + 200 mg/kg body weight EGCG. Cataract was induced by a single subcutaneous injection of sodium selenite (2.46 mg/kg body weight) on postpartum day 10. Treatment groups received EGCG intraperitoneally from the 9th day upto the 14th day. On postpartum day 24, rat pups were examined for cataract formation and the lenses were isolated for further analysis of oxidative damage.

Results: Sodium selenite caused significant (p < 0.05) cataract formation, a reduction of the activities of SOD, catalase, and GSH level, and an increase of protein carbonyls level compared with the normal control group. In contrast, treatment with EGCG could significantly (p < 0.05) ameliorate cataract formation and oxidative damage in the lens. Moreover, EGCG administration significantly increased the GSH level and the activities of SOD and catalase, and declined the protein carbonyls in the lens when compared with the sodium selenite group.

Conclusions: Taken together, EGCG administrations demonstrate effective protective effects on sodium selenite-induced cataract and oxidative injury in mice.

Keywords: cataractogenesis; EGCG; sodium selenite; oxidative damage

REFERENCES


