SUMMARY AND CONCLUSION

Cataract, loss of eye lens transparency, is the leading cause of blindness worldwide. The selenite cataract is an extremely rapidly-induced and convenient model of cataractogenesis. Alpha-crystallin, initially known as one of the major structural proteins of the eye lens, it is convincingly established now that alpha-crystallin functions like a chaperone and plays a decisive role in the maintenance of eye lens transparency. There is growing evidence that oxidative stress, mediated by active oxygen species (ROS) in the lens and lipid peroxides, produced in the crystalline lens, are responsible for a breakdown of lens homeostasis and decreasing the chaperone activity of α-crystallin leading to lens opacification.

In this study, a rat model of selenite-induced cataract were developed by injection with sodium selenite and Coenzyme Q10 was administered to rats by gastric intubation. After 21 day of injection, slit lamp examination was done to detect the occurrence of cataract. At the end of experiment, rats were taken and the following biochemical parameters determined:

1. GSH in blood and lens.
2. Malondialdehyde in blood and lens.
3. Catalase in blood and lens.
4. SOD in blood and lens.
5. Total soluble protein in lens.
7. Tryptophan and ANS fluorescence.
8. SDS electrophoresis of lens protein.
SUMMARY AND CONCLUSION

The observation and results obtained indicated the following:

1- A progressive increase in the activity of superoxide dismutase and decrease in catalase activity in the blood of model rats were observed but these changes were reversed after treating with Coenzyme Q10.

2- The activities of catalase and superoxide dismutase in lens of model rats were decreased significantly but these activities were reversed after treating with Coenzyme Q10.

3- The observed changes in the levels of reduced glutathione and malondialdehyde in blood and lenses of model rats were modified in case of rats treated with Coenzyme Q10. This could be due to its antioxidant activity.

4- UV spectra exhibited changes in lens proteins of selenite-induced cataract group that indicate alterations in tertiary structure of crystallins while these changes disappeared in rats treated with Coenzyme Q10.

5- Tryptophan fluorescence shows loss of intensity of intrinsic tryptophan fluorescence in model group (selenite-induced cataract) which confirms the altered tertiary structure of lens soluble protein while these changes disappeared in rats treated with Coenzyme Q10.

6- ANS fluorescence shows loss of intensity of ANS fluorescence in model group (selenite-induced cataract) which indicates conformational changes in the structure of soluble lens protein and confirms the loss of
chaperone activity in model group which increased in coenzyme Q10 treated group.

7- SDS electrophoresis of lens proteins of model rats showed obvious changes and crosslinking of protein that indicate the occurrence of opacifications. These changes didn’t appear in coenzyme Q10 treated rats. This observation manifests the beneficial role of this chemical chaperone in prevention of selenite cataract.

8- Slit lamp examination revealed the occurrence of mature cataract in model rats while treated group appeared with normal and clear lenses (85% of treated lenses were clear).

In conclusion, Coenzyme Q10 can be identified as a new intervention that can effectively inhibit oxidative stress involved in cataract. Coenzyme Q10 provides protection via attenuation of the oxidative stress. Although the precise mechanism should be explored in future studies and one must be cautious in applying animal models to human disease, these studies provide a theoretical basis for further study of the clinical prevention and treatment of cataract.