

Program#/Poster#: 2089/B639

Abstract Title: Dietary Superoxide Dismutase Protects Against Light-Induced Retinal Oxidative Stress in Young Senescence Accelerated Mice (SAM)

Presentation Start/End Time: Monday, May 01, 2006, 3:00 PM - 4:45 PM

Location: Hall B/C

Reviewing Code: 281 retina/RPE: oxidation, stress - RC

Author Block: P. Sicard¹, S. Amoureux¹, N. Acar², C. Joffre², A.M. Bron^{3,2}, M.-A. Maire², C. Vergely¹, C.P. Creuzot-Garcher^{3,2}, L. Bretilon², L. Rochette¹. ¹Laboratory of Experimental Cardiovascular Pathophysiology and Pharmacology, Faculties of Medicine and Pharmacy, Dijon, France; ²Eye and Nutrition Research Group, National Institute for Research on Agronomy, Dijon, France; ³Ophthalmology, University Hospital, Dijon, France.

Keywords: 673 retina, 620 oxidation/oxidative or free radical damage

Purpose: Oxidative stress from reactive oxygen species has been implicated in many diseases including age-related macular degeneration, in which the retinal pigment epithelium is a primary target. The aim of this study was to evaluate the protective effect of dietary supplementation in Superoxide Dismutase (SOD) on light-induced oxidative stress in a mouse model for aging, the senescence-accelerated mouse prone 8 (SAM P8).

Methods: Weaning SAM P8 and SAM resistant 1 (SAM R1, controls) were used. Animals were exposed 3 times to light (1900 lux for 7 hours) at 1, 2 and 3 months of age. At 3 months of age and before the last light exposure, animals were treated by gavage with SOD (**Glisodine**[®]: 10.8 mg/kg/day) or water during 7 days. The scotopic ERG was then recorded and animals were killed in order to measure 1) plasma antioxidant capacity by electron spin resonance using a spin probe CP-H (1-hydroxy-3-carboxy-pyrrolidine), 2) superoxide anion levels on retinal cryosections using an oxidative fluorescent probe (dihydroethidium, DHE, 10 μ M).

Results: Light-exposure did not alter the ERG response since a- and b-wave amplitudes were unchanged whatever the strain and the SOD supplementation. However, plasma antioxidant capacity was increased by 30 % in animals treated by SOD. Superoxide anion levels were increased up to 50% ($p < 0.01$) in the ganglion cell layer, and by 300 % in the outer nuclear layer ($p < 0.01$) in all light-exposed mice as compared to non-exposed animals. No differences were observed between SAM R1 and SAM P8. Within light-exposed animals, the SOD supplementation significantly reduced the superoxide anion levels ($p < 0.05$).

Conclusions: These results demonstrate that our light-exposure conditions promote retinal oxidative stress without inducing retinal degeneration. The similar results obtained in SAM R1 and SAM P8 animals may be explained by their young age. However, these data suggest that dietary SOD supplementation is efficient to limit retinal oxidative stress by increasing plasma antioxidant capacity.

Commercial Relationship: P. Sicard, None; S. Amoureux, None; N. Acar, None; C. Joffre, None; A.M. Bron, None; M. Maire, None; C. Vergely, None; C.P. Creuzot-Garcher, None; L. Bretilon, None; L. Rochette, None.

Support: None

©2006, Copyright by the Association for Research in Vision and Ophthalmology, Inc., all rights reserved. Go to www.iovs.org to access the version of record. For permission to reproduce any abstract, contact the ARVO Office at arvo@arvo.org.



-- Indicates International Multi-Country Collaboration

OASIS - Online Abstract Submission and Invitation System™ ©1996-2006, Coe-Truman Technologies, Inc.