Melanin Protects Choroidal Blood Vessels against Light Toxicity

Swaantje Peters[a,*], Thomas Lamah[b], Despina Kokkinou[a],
Karl-Ulrich Bartz-Schmidt[a], and Ulrich Schraermeyer[a]

[a] Center of Ophthalmology, University of Tuebingen, Schleichstr. 12, D-72076 Tuebingen, Germany. Fax: +49 7071 29 45 54. E-mail: Swaantje.Peters@med.uni-tuebingen.de
[b] Department of Anatomy I, University of Cologne, Joseph Stelzmann Str. 9, D-50931 Cologne, Germany

* Author for correspondence and reprint requests

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Low ocular pigmentation and high long-term exposure to bright light are believed to increase the risk of developing age-related macular degeneration (ARMD). To investigate the role of pigmentation during bright light exposure, cell damage in retinae and choroids of pigmented and non-pigmented rats were compared. Pigmented Long Evans (LE) rats and non-pigmented (albino) Wistar rats were exposed to high intensity visible light from a cold light source with 140,000 lux for 30 min. Control animals of both strains were not irradiated. The animals had their pupils dilated to prevent light absorbance by iris pigmentation. 22 h after irradiation, the rats were sacrificed and their eyes enucleated. Posterior segments, containing retina and choroid, were prepared for light and electron microscopy. Twenty different sections of specified and equal areas were examined in every eye. In albino rats severe retinal damage was observed after light exposure, rod outer segments (ROS) were shortened and the thickness of the outer nuclear layer (ONL) was significantly diminished. Choriocapillaris blood vessels were obstructed. In wide areas the retinal pigment epithelium (RPE) was absent in albino rats after irradiation. In contrast, LE rats presented much less cell damage in the RPE and retina after bright light exposure, although intra-individual differences were observed. The thickness of the ONL was almost unchanged compared to controls. ROS were shortened in LE rats, but the effect was considerably less than that seen in the albinos. Only minimal changes were found in choroidal blood vessels of pigmented rats. The RPE showed certain toxic damage, but cells were not destroyed as in the non-pigmented animals. The number of melanin granules in the RPE of LE rats was reduced after irradiation. Ocular melanin protects the retina and choroid of pigmented eyes against light-induced cell toxicity. Physical protection of iris melanin, as possible in eyes with non-dilated pupils, does not seem to play a major role in our setup. Biochemical mechanisms, like reducing oxidative intracellular stress, are more likely to be responsible for melanin-related light protection in eyes with dilated lens aperture.

Key words: Melanin, RPE, Light Toxicity