Old Questions – New Answers

How Does Diabetes Affect the Fatty Acid Composition of the Retina?

Hegde et al., Baltimore, Md., USA

Ophthalmic Res 2009;42:9–14

The authors utilized gas chromatography-mass spectrometry to determine the accumulation of lipids in the diabetic retina. As the major fatty acids in the retina palmitic, oleic, stearic, arachidonic and docosahexaenoic acids were found.

Interestingly, the content of all these fatty acids increased significantly in the diabetic retina. In particular major increases (~3 times the normal) were found in palmitic, oleic, stearic and arachidonic acids.

In addition, the relative percentages of fatty acids were affected. While the relative percentages of palmitic and oleic acids increased in the diabetic retina, there were insignificant changes in the percentages of stearic and arachidonic. Remarkably, the relative percentage of docosahexaenoic acid underwent a significant decrease, about 50% of the normal. These observations are consistent with the known increased mobilization of lipids from the adipose tissue and their accumulation in other tissues under diabetic conditions. Physiologically, the excessive accumulation of fatty acids in the diabetic retina correlates with the lipofuscinosis and neural dysfunction associated with this disease.

Can Experimental Full-Field ERG Be Used for Toxicological Assessments?

Yamashita et al., Saitama, Japan

Ophthalmic Res 2009;42:15–20

Full-field ERG, using contact lenses with built-in LEDs was performed on albino rats and used to evaluate the visual toxicity of sodium iodate (NaIO₃). In 2 subsequent experiments the authors determined the optimal conditions relating to stimulus intensity, background illumination and light adaptation period. In a second experiment they confirmed the usefulness of full-field ERG using rats with retinal toxicities induced by NaIO₃.

Whereas the rod response disappeared completely at 3 h, based on reduced oscillatory potentials, cone and flicker responses were still present at 8 h. All responses disappeared on the 7th day. These findings indicate that the retinal toxicity induced by NaIO₃ was expressed first in rods, followed by cones. Interestingly, there were no microscopical changes after 8 h of administration, although the RR shad completely disappeared by this time. The authors conclude that full-field ERG using an LED contact lens is useful for the evaluation of toxic effects on rods and inflammatory molecules such as TNF- α and iNOS.

How Does Alpha-Crystallin Protect Activated Retinal Microglia?

Wu et al., Chongqing, China

Ophthalmic Res 2009;42:21–28

Inhibition of microglial activation has become an important strategy to attenuate neurotoxic damage to the central nervous system. The authors evaluated the effects of α -crystallin on the production of cytokines in lipopolysaccharide and optic-nerve-injury-activated retinal microglia. Microglia were collected from retinas of newborn rats, cultured and treated with lipopolysaccharide in vitro. Microglia were also activated by an optic nerve crush in vivo. Pretreatments with and without α -crystallin were performed in cultured cells and by intravitreal injection in adult rats. Expressions of TNF- α , iNOS and NO were measured by RT-PCR, ELISA, Western blot and the nitrate reductase method.

As a result, activated microglia significantly upregulated TNF- α and iNOS mRNA expression and protein production in vitro. In addition, optic nerve crush also increased the expression of retinal iNOS and TNF- α protein, whereas treatment with α -crystallin in vitro and in vivo downregulated their expression.

The authors conclude that the protective effect of α -crystallin may be due to its effect on microglia via a downregulation in the expression and release of 2 key immune regulatory and inflammatory molecules, TNF- α and iNOS.

Uwe Pleyer, Editor-in-Chief