UVR-B induced cataract is linked to an increased expression of inflammatory cytokines in the lens epithelium *in vivo*

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Background & Purpose

- Cataract is the most common cause of blindness in the world and there are currently no prevention strategies (McCarty and Taylor 2002).
- Experimental studies showed that UVR-B induced cataractogenesis may be associated with an inflammatory reaction (Meyer, Lofgren et al. 2013).
- There has been some evidence for the neuropeptides monocyte chemotactic protein-1 (MCP-1) and neurokinin receptor-1 (NKR-1) to modulate ocular inflammation processes (Zhu, Wolff et al. 2015; Casini, Dal Monte et al. 2004).
- The aim of this study was to investigate the influence of UVR-B exposure on the expression of NKR-1 and MCP-1 in the lens epithelium in vivo and thereby to examine if these neuropeptides are involved in inflammatory responses in cataract development.

Methods

- C57Bl/6 mice were exposed *in vivo* to UVR-B irradiation using a Bio Spectra system (Vilber Lourmat, Germany).
- In one eye the mice received a five-fold cataract threshold equivalent dose (1,45 kJ/m²; 300-nm wavelength region) while the other eye was completely shielded.
- Three and seven days after UVR-B exposure cataract formation was assessed with a Leica dark-field microscope camera system and quantified with a software measuring pixel intensities.
- NKR-1 and MCP-1 levels in lens epithelium lysates were analyzed with ELISA (enzyme-linked immunosorbent assay) for the exposed and also for the contralateral non-exposed eye.

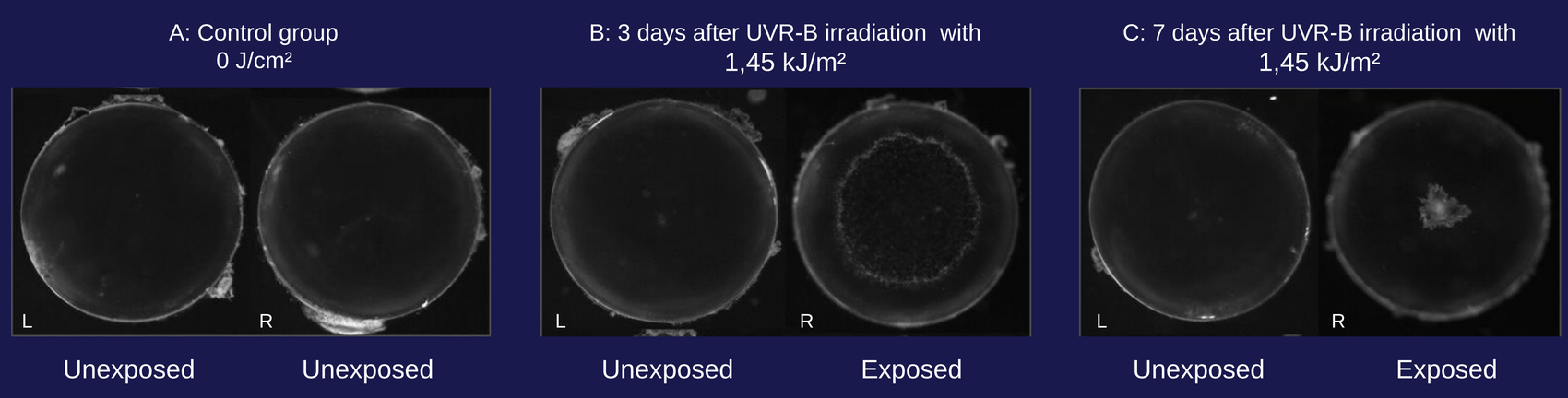

Fig.1: Cataract morphology of lenses three and seven days after *in vivo* exposure to a five-fold threshold dose (1,45 kJ/m²) UVR-B of 312nm. Comparison of the exposed lenses to the unexposed control lenses.
A: Unexposed control lenses are clear.
B: In the exposed eye subcapsular cataract developed in form of a wide area with obscure granules.
C: The anterior subcapsular cataract in the exposed eye shrank to a smaller triangled area.

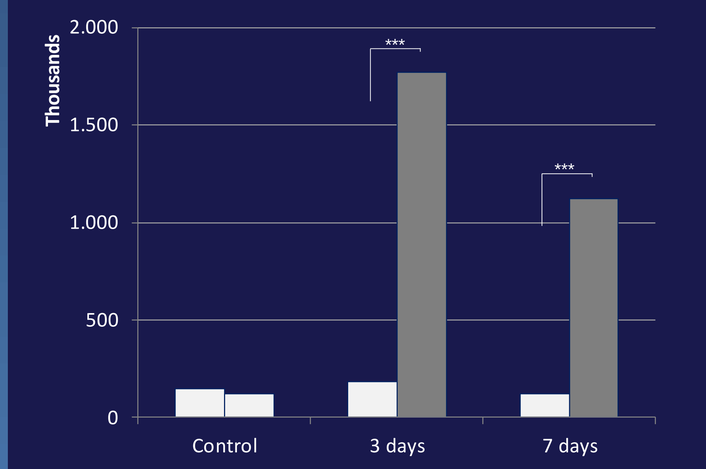

Fig.2: Pixel intensity in the exposed and non-exposed lenses three and seven days after exposition in comparison with the control group.

Statistical analysis was carried out by using SPSS Statistics 22. The significance level was set to 0.05.

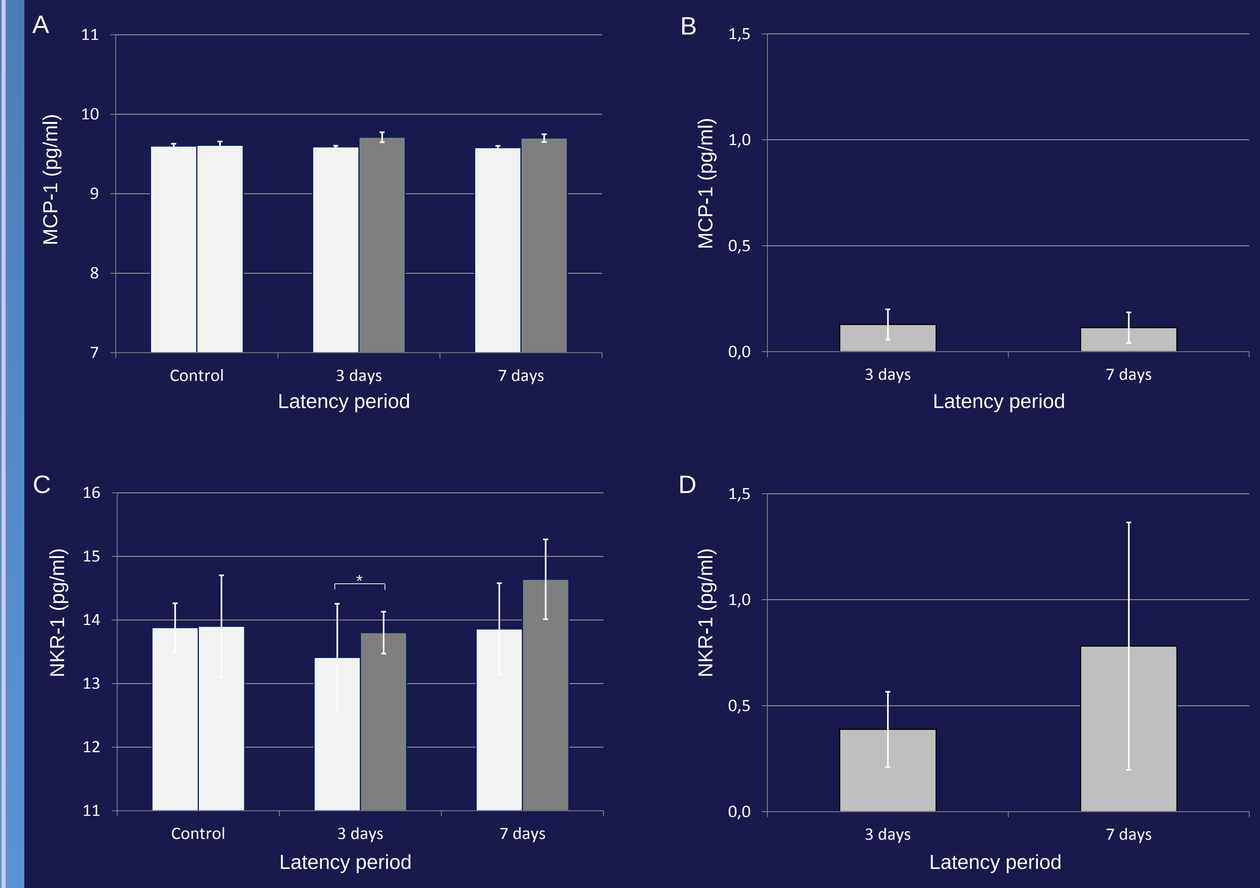

Fig.3: ELISA (enzyme-linked immunosorbent assay) for MCP-1 and NKR-1 in the lens epithelium.

A: Absolute value of MCP-1 in the lens epithelium with Standard Deviation. MCP-1 levels increased following UVR-B exposure at latency periods three and seven days.

B + D: 95% Confidence intervals for the mean differences between the exposed and contralateral eye for latency periods of three days and seven days.

C: Absolute value of NKR-1 in the lens epithelium with Standard Deviation. NKR-1 levels increased following UVR-B exposure at the latency period of seven days.
A significant difference was found for NKR-1 levels between the exposed and the contralateral eye three days after exposure.

Results

- All UVR-B exposed mice developed cataracts in the exposed eye.
- Pixel intensity was significantly higher in the exposed eye after three and seven days (17Mio. Pixel/ 11Mio. Pixel) compared to the non-exposed eye (<200.000 Pixel).
- MCP-1 levels in the exposed lens epithelium increased following UVR-B exposure at latency periods three (9,71 pg/ml) and seven days (9,70 pg/ml). MCP-1 levels in the unexposed eyes did not increase compared to the control group (9,58 pg/ml).
- A significant difference was found for NKR-1 levels between the exposed eye (13,80 pg/ml) and the contralateral eye (13,41 pg/ml) three days after exposure.

Conclusions

- UVR-B induces cataract three and seven days after exposure *in vivo*.
- Further research on the influence of inflammatory cytokines is needed and it should be explored if possible anti-inflammatory treatments may counteract UVR-B induced cataract development.

Financial Disclosures
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