AB044. Phototoxic stress induced in retinal pigmented epithelium cells by the synergy between polycyclic aromatic hydrocarbons and blue light

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Background: Lesion to the retinal pigment epithelium (RPE) is a crucial event in age-related macular degeneration (AMD) development. Although the pathogenesis of this complex disease is poorly understood, sunlight exposure and smoking are major environmental risk factors associated with AMD. High-energy visible blue light (HEV; 400–500 nm) is the most energetic and potentially harmful solar wavelengths reaching adults retina. On the other hand, RPE cells can be exposed to a large range of pollutants from cigarette smoke, with polycyclic aromatic hydrocarbons (PAH) being among the most toxic. Some PAH from cigarette smoke can absorb HEV light. This led us hypothesize that in RPE cells, the combination of PAH and HEV could synergize to exacerbate the stress caused by either factor alone. We thus investigate the combined effect of PAH and HEV light in RPE cells.

Methods: Confluent RPE immortalized cells (ARPE19) were exposed to nanomolar concentrations of benzo[a]pyrene (BaP) or indeno[1,2,3-cd]pyrene (IcdP). While IcdP efficiently absorbs HEV wavelengths, BaP, the most studied PAH, does not significantly absorb HEV light and was used as a control. BaP or IcdP contaminated ARPE19 were then irradiated with increasing sub-lethal doses of HEV light (150–500 J/cm^2) using a setup that mimics the light spectrum normally reaching the retina. Cytotoxicity, apoptosis and reactive oxygen species (ROS) generation were assessed in each condition.

Results: In presence of low concentrations of IcdP, sub-lethal amounts of HEV light trigger, in a dose-dependent way, up to 70% of apoptotic cell death. Co-exposure to IcdP and HEV also leads to a synergistic ROS generation in ARPE19 cells, thus inducing oxidative stress. None of these effects were observed with BaP. Efficient inhibition of ROS production by specific antioxidants only decreases death by 20% in cells simultaneously exposed to both IcdP and HEV light.

Conclusions: Low concentrations of IcdP synergize with HEV light to induce phototoxicity in ARPE19 cells. An increased oxidative stress results from the interaction between both agents and partially explains the enhanced HEV phototoxicity in IcdP contaminated ARPE19 cells. This suggests that another major mechanism is involved in the synergetic toxicity. For smokers, this synergy between HEV and PAH may accelerate RPE cells loss and contribute to their greater risk of developing AMD.

Keywords: Retinal pigment epithelium (RPE); polycyclic aromatic hydrocarbons (PAH); blue light; oxidative stress; age-related macular degeneration (AMD)

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