# AB024. Photo-oxidation of N-retinylidene-N-retinylethanolamine *in vitro* by high-energy visible light

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**Background:** Age-related macular degeneration (AMD) is the second Canadian cause in visual deficiency. AMD is characterized by the death of photoreceptors and retinal pigmented epithelium (RPE) in the macular region of the retina, leading to the loss of central vision. Epidemiologic studies suggest an association between lifetime sun exposure and the probability to develop AMD even though mechanisms are unknown. Sunlight is made of about 30% of high-energy visible (HEV) light (blue light), the most energetic wavelength reaching the retina. These wavelengths can be absorbed by lipofuscin, an age pigment accumulating in RPE cells. Lipofuscin principal component is N-retinylidene-N-retinylethanolamine (A2E). Many research teams showed that absorption of HEV light by A2E in RPE cells at non-physiological doses produces free radicals and leads to cell death. Our earlier work shows that when A2E-loaded RPE cells are irradiated with HEV light at physiological doses, the same light does not lead to oxidative stress as measured by telomere and mitochondrial integrity. Our hypothesis is that HEV light, at physiological doses, modify or convert A2E in derived produces, inhibiting its photo-oxidant effect.

**Methods:** *In vitro*, we irradiated A2E with HEV light with or without antioxidants and with varying irradiation regimen to observe the UV-Visible spectrum of A2E. In cellulo, we loaded ARPE-19 cells with A2E and irradiated cells at physiological levels for 4 consecutive days. We then observed A2E fluorescence using a fluorescence microscope with nucleus counterstaining with DRAQ5.

**Results:** HEV light leads to the disappearance of A2E characteristic UV-Visible spectrum and the apparition of a new product suggesting that HEV light modifies A2E. Nor oxidation and irradiation regimen seem to have an impact in A2E's conversion by HEV light. We observed and progressive diminution of A2E fluorescence in cellulo during physiological irradiations.

**Conclusions:** The loss of A2E photo-oxidation capacities by HEV light seems to be caused by its conversion by HEV light. We suggest that HEV light, at physiological doses, may be protective rather than photo-toxic. The next step would be to identify A2E' derived produces and their cell toxicity.

**Keywords:** Age-related macular degeneration (AMD); A2E degradation; blue light; retinal pigmented epithelium (RPE); cellular toxicity

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