Commentary

Inhibiting Myopia by (Nearly) Invisible Light?

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ARTICLE INFO

Article history:
Received 11 January 2017
Accepted 11 January 2017
Available online 16 January 2017

Torii et al. (2017) propose that the absence of “violet” light (VL, near ultraviolet (UV), below 400 nm) in our industrialized world is a major contributing factor for the recent epidemic increase in myopia. They conclude from their study that artificial lighting should be revised to include a UV component, as in sunlight. The proposal is provocative since short wavelength light exposure has been a topic of research for decades and has been shown to induce photo-oxidation and retinal degeneration.

Their conclusion is based on three lines of studies (1) experiments in chickens showing that form deprivation and negative lens induced myopia were reduced when chicks were exposed to light containing near UV and that transcription of retinal genes involved in myopia inhibition was stimulated, (2) measurements of gene expression in mouse photoreceptor cell cultures with and without UV exposure, and (3) analyses of myopia progression in students and children who wore either UV blocking or UV transmitting correcting lenses.

We believe that important limitations of the study should be kept in mind.

(1) The Authors use two strategies to experimentally induced myopia in chicks. It is well established that imposed hyperopic defocus and form deprivation produced by diffusers can produce axial myopia in chickens. However, details concerning the rearing regimen are lacking. It appears that the treatment lenses were glued in place. This is problematic for the experiments involving −9 D glass lenses, because it is not usually possible to remove glued lenses for routine daily cleaning. As a consequence, the defocusing effects of the treatment lenses can be severely compromised by moisture on the inside of the lenses and debris (as the Authors mention in their discussion). Previous experiments have shown that chicks can completely compensate for moderate powered negative lenses in a relatively short period of time. The fact that the treated eyes in neither of the light groups (VL− or VL+) exhibited accurate compensation to the −9 D lenses is in agreement with the idea that the optics of the lenses were compromised. Unfortunately, zero powered lenses were not fitted in front of the control eyes so that it is not known whether this was a factor.

The Authors observed systematic differences in the degree of axial elongation in the chickens reared with the plastic lenses. The Authors argue that these differences are due to the variation in the transmission of violet light between the different goggles. However, it appears that the differences in the transmission of violet light was produced by “opacifying” the lenses, as illustrated in Fig. S2. However, that would result in substantial differences in the degree of image degradation produced by the “translucent” versus the “opacified” goggles. It is well established in several species, including chickens, that the degree of axial myopia is directly related to the degree of image degradation produced by diffuser lenses.

(2) In contrast to humans, chickens have UV light-transmitting ocular media down to at least 350 nm and a UV receptor (Schaeffel et al., 1991). It is therefore not unexpected that light-regulated genes (like ZENK) are up-regulated.

(3) Studies in mouse photoreceptor cell lines permit only limited extrapolation to humans because spectral filtering by the ocular media is excluded and because mice have also UV vision.

(4) The literature agrees that the ocular media of phakic human subjects effectively blocks light below 400 nm (i.e. Artigas et al., 2012) so that only low levels can reach the retina (at least 2 log units attenuation). Therefore, it is not clear how much violet light would reach the retina in their study. UV exposure of the skin was not different when different optical corrections were used. Therefore, in agreement with this and other studies (reviewed by Rose et al., 2016), vitamin D stimulation by UV light can be excluded as a contributing factor. The question is then how reduced myopia progression in human subjects can be explained when they wear optical corrections that transmit near UV light. It appears likely that their retrospective analysis was confounded by variables that are known to influence the rate of myopia progression. In particular in both human studies, the subjects who were wearing lenses that blocked short wavelength light were younger (by >1.5 years in the spectacle lens...
study and by 0.5 years in the contact lens trial) and they were more myopic. It is very reasonable to argue that the onset of myopia was earlier and was progressing faster in the “experimental” groups. Thus, it may not be surprising that they showed slightly greater increases in axial dimensions. A randomized controlled trial (RCT) would be the gold standard for these kinds of analyses.

Nevertheless, the effects of the spectral distribution of light on myopia development remain an important topic. Long wavelength light (>650 nm, red (not green, as the Authors state)) has been shown to act as a strong inhibitor of eye growth in rhesus monkeys (Smith et al., 2015) and tree shrews (Gawne et al., 2016) - an unexpected effect because the opposite was found in chickens (Seidemann and Schaeffel, 2002; Foulds et al., 2013). We believe that the proposed role of non-visible UV light in myopia is not (yet?) convincing and more studies in humans or monkeys are necessary to justify a re-design of artificial light sources to include near-UV.

Disclosure

The authors declared no conflicts of interest.

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