Author Response: Light Levels and the Development of Deprivation Myopia

We thank Galvis et al.[1] for their comments regarding the possible role of ultraviolet (UV) exposure in the regulation of ocular growth. As we noted in our report,[2] although much still is unknown, results from animal studies suggest that UV light is not critical for the regulation of ocular growth during experimentally induced changes in scleral growth rates, and more specifically, does not underlie the ability of bright light to retard the development of experimental myopia. As discussed in our study,[2] and as noted by the authors, the protection provided by bright light against the development of deprivation myopia has been obtained using UV-free lighting systems in all animal models tested (chicks,[2–5] tree shrews,[6] and rhesus monkeys[7]). Normal emmetropization also is modifiable in chicks by alterations in light intensity, again using UV-free systems.[8,9] Therefore, UV exposure does not underlie the ability of bright light to retard the development of deprivation-myopia, or the ability of bright light to maintain normal untreated eyes in a hyperopic state. However, we have not tested whether broadening the spectral output of our lighting system to include UV output can induce an even greater protective effect against the development of myopia. This seems unlikely, as the development of deprivation myopia can be abolished in rhesus monkeys (20,000 lux)[7] and chicks (40,000 lux)[1] by bright light alone; therefore, UV exposure seems unnecessary. Instead, our data suggest that the ability of light to retard the development of deprivation myopia is driven by intensity-dependent increases in retinal dopamine release,[4] although the role of spectral composition, in the visible range, is an area of interest (for review see the study of Rucker[10]).

In terms of experiments directly testing the role of UV exposure, we noted the report of Hammond et al.[11] In this study, the investigators observed no difference in compensation to −10 diopter (D) or −20 D lenses under white light or UV light of matching illuminance. This suggests that the presence or absence of UV light does not modify compensation to negative lens wear and, hence, the emmetropization process. However, we agree with the authors that it would be interesting to see if greater intensities of UV exposure could alter the rate of compensation to optical defocus. The mechanism by which UV exposure may inhibit ocular growth has been postulated to involve elevated levels of vitamin D. Again, animal studies do not support a role for vitamin D in the regulation of ocular growth. Specifically, vitamin D_3 supplementation does not affect the development of FDM or LIM in the tree shrew (Siegwart JT, et al. *IOVS* 2011;52:ARVO E-Abstract 6298).

Whether UV exposure, and with it vitamin D levels, has a role in the protective effects of time outdoors still is unclear. An association between vitamin D receptor polymorphisms and myopia has been observed.[12] Epidemiologic analysis has shown that increasing time spent outdoors is paralleled with increasing vitamin D levels, both of which show a negative correlation to incident myopia.[13–16] However, survival analysis has indicated that the critical factor for incident myopia is time spent outdoors, rather than vitamin D levels.[16] In summary, the combination of animal experiments and survival analysis from epidemiological studies show that increased time outdoors and increased light exposure can prevent the development of myopia, but there is limited evidence that UV exposure or vitamin D has a causal role. Caution must be taken when interpreting any correlation between UV exposure, vitamin D levels, and myopia, as it is difficult to distinguish between a causative role and a simple biomarker for time spent outdoors.

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