Bright Light Blocks the Development of Form Deprivation Myopia in Mice, Acting on D1 Dopamine Receptors

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Chen et al.[1] provide more evidence, using the mouse myopia model, to support the idea that bright light suppresses the development of myopia via increased dopamine release. They show that the cells activated by bright light, delineated by increased expression of phosphorylated tyrosine hydroxylase and c-fos, are predominantly ON-bipolar cells, which are known to stimulate the release of dopamine from dopaminergic amacrine cells.[2] The protective effects of bright light against form-deprivation myopia seen in nonhuman primates[3] make it likely that similar dopamine-mediated effects will be seen in children, where it is known that more time outdoors, where light is much brighter than indoors during the day, slows the onset of myopia. However, the results of Chen et al.[1] are unusual in that the protective effect of light appears to be mediated by D1 dopamine receptors, with the protective effect blocked by the relatively selective D1 receptor antagonist SCH39166. Most other studies have suggested that D2 dopamine receptors are involved, although the pharmacologic characterization of receptors is very limited. The possibility of interactions between the D1 and D2 pathways has also been raised (see Feldkaemper and Schaeffel[4] for a review). Given the increasing use of increased time outdoors to control the development of myopia in children, the receptors involved now require more detailed pharmacologic characterization, particularly if the use of dopamine agonists for myopia control is to be considered. One further issue that needs resolution is whether the involvement of D1 dopamine receptors is a feature of mammalian retinas, or of retinas adapted for living in low light rather than bright light conditions or a more general feature of control of eye growth.

References

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