Commentary

New Research Routes to Fight Myopia

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Myopia can be defined optically as a failure in the ability of the eye to focus images of distance objects on the photosensitive internal layer, the retina. This can be due to excessive power of the cornea and crystalline lens, or more commonly due to an excessive axial growth of the eyeball. In the last years, we have assisted to an increased rate of myopia incidence and myopia prevalence within the population. Recent estimates suggest that by 2050, about half (about 5 billion) of the world population will be myopic (Holden et al., 2016). Far from a futuristic and alarming prediction, there is already evidence that myopia prevalence has been increasing over the last decades, not only in Asia, but also in the USA (Vitale et al., 2009) and Europe (Williams et al., 2015).

With a layered structure of internal photosensitive tissue (retina), a medial vascular tissue (choroid) and an external structural cover (sclera), excessive eye growth can lead to several pathologies derived from the stretching and tension within and between those layers. Thinning, vascular anomalies and detachments can potentially cause severe visual impairment and blindness. Indeed, the risk increases exponentially with the degree of myopia (Flitcroft, 2012). Myopia development can be quite different in presentation and development, but it is generally accepted that the younger the age at onset, the faster the progression rate and the higher the final myopia degree. Therefore, there is a growing interest in treating myopia in children using minimally invasive treatments, frequently involving the use of specially designed contact lenses (González-Méijome et al., 2016).

The work by Torii et al. (2017) recently published in EBioMedicine presents a possible route to explain the complex interaction between genetics and environment through the exposure to violet light (VL) within a narrow band of 360–400 nm. After the earlier reports on the potential role of the transcription factor Erg-1 in myopia protection in mice by Brand et al. (2007) and by others in chickens and mammals, the results of Torii et al. (2017) suggest that might also be relevant in humans. Whether there is a cause and effect between the exposure to VL and axial elongation of the eye due to expression of this factor is yet to be confirmed in larger controlled trials and, the link between VL exposure and Erg-1 activation is still to be elucidated. Therefore, future research efforts addressing this question should consider prospective controlled and randomized clinical trials involving larger sample sizes in order to overcome the limitations of the present study. Considering different dose of exposure to VL might also help to strength the potential relationship between the genetic factors and the anatomic changes, thus eventually strengthening the confirmatory evidence of such potential link. Furthermore, the results presented by the authors showing minor role of vitamin D in favor of shorter wavelength light exposure seem to provide a basis for the interpretation of a large scale study recently published by European researchers from a large scale study (Williams et al., 2017).

The search for causes of myopia onset and progression has been elusive due to its multifactorial nature (Flitcroft, 2012). Several genetic factors have been suggested to have a role in myopia occurrence, though the role attributed to such factors is usually reduced when compared to other “environmental” factors (Morgan & Rose, 2005). Intensive near work has been also associated with increased risk of myopia. The sustained effort by the accommodative system responsible for increasing the power of the internal crystalline lens to focus near objects might be a source of biomechanical stress within the eye ultimately resulting in eye elongation. Though previous research have linked higher educational levels achieved with myopia development the link between near work and myopia development is not yet well understood. The association between genetics and education has been object of recent study by Verhoeven et al. (2013) in the context of the Consortium for Refractive Error and Myopia concluding that those individuals with higher genetic risk were more susceptible to develop myopia when exposed to higher education.

The results of Torii et al. (2017) are of paramount relevance in the context of the current uncertainty about the role of different factors in ocular growth and myopia development. Considering the feasibility of customizing the transmittance of myopia correction optical devices (i.e. contact lenses, spectacle lenses or intra-ocular lenses), if the authors’ hypothesis is confirmed in prospective controlled clinical trials, immediate treatments can be implemented with obvious benefits for myopic individuals by reducing their rate of progression. Such treatments might be also considered as prophylactic methods to prevent myopia onset in those at risk of myopia development considering the concurrence of genetic, environmental or other risk factors.
Disclosure

The author is involved in research projects, proposals and clinical trials related with myopia and myopia progression. The author has been consultant over the past 5 years to companies with financial interests in myopia treatment. The author does not have other conflicts of interest.

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