

Foreword



The field of myopia research is curiously different from research into the etiology of other medical conditions. Whereas for most conditions, research on animal models is universally held to be essential for understanding the etiology and promising treatment modalities, in the case of myopia there is little cross-over from laboratory to clinic, despite the dramatic findings that animals can be made myopic or hyperopic in compensation for defocus imposed by spectacle lenses — a result consistent with the prevalence of other diseases being associated with homeostatic mechanisms. Why this disconnect between animal and clinical studies of myopia? One likely cause is the strong association of the prevalence of myopia with educational level, making it seem to be a uniquely human disorder. This association has led to speculative conjectures about how myopia develops, but has not led to effective prophylaxis. As a result of this lack of therapeutic progress, some have rejected the possibility of arresting myopia by relatively non-invasive visual treatments. Despite several decades of experimental studies of myopia in animals, many clinicians continue to consider myopia as a particularly human condition, or as a consequence of one's genetic makeup, and regard the animal studies as only weakly

related to why humans become myopic. Given that every few years a new group of animals is added to the myopia zoo, all compensating for defocus imposed by spectacle lenses, how likely is it that humans are different?

Were the etiology of human myopia simple this controversy would have been resolved by now. But many human diseases, such as cardiovascular disease and diabetes, have a complex etiology, involving both genetic and behavioral components. How do they differ from myopia? One reason may be that these diseases can be effectively treated without fully understanding their underlying causes, whereas in the case of myopia, understanding the cause of the myopia may well be necessary because simply correcting the myopia may reinstate the conditions that caused the myopia in the first place, thereby setting in motion a positive feedback loop, resulting in an iatrogenic worsening of the myopia. Indeed, some of the possible treatments (scleral reinforcement, daily atropine administration) might be worse than the disease, at least for otherwise healthy children with mild myopia.

Only now are we beginning to appreciate and measure in children the parameters likely to be important indicators of the initiation and progression of the myopia development. For example, what is the refractive status of the retinal periphery and how is it affected by visual experience? Does the periphery become hyperopic relative to the fovea as a cause or consequence of the fovea becoming myopic? Does intensive reading first affect the central or peripheral refractions? How much of the variability in peripheral refractions is a function of eye-shape at birth vs the visual surroundings, and how do these interact? If medical students are more likely to become myopic than athletes, is this due to the total amount of reading or to the duration of episodes of reading? Or to the amount of time outdoors? If the latter, is the relevant factor the absence of hyperopic or myopic defocus at the fovea or in the periphery, or perhaps the enhanced stimulation of dopamine by bright light? These are issues that can be studied in both humans and animals, but require experimental manipulations more difficult than those that have been attempted to date.

An unfortunate consequence of the complicated, multifactorial, nature of the control of eye growth and development of refractive state is that the field of research has been nearly completely divided into those doing animal research and those doing human research. My dear friend and colleague, the late Sek-Jin Chew, was an exception. He studied muscarinic receptors in different ocular tissues to understand why atropine reduced myopic progression. He studied the blinking of chicks to understand whether brief pulses of increased intraocular pressure would affect ocular

elongation. And he raised mice wearing a diffuser over one eye under his bed to explore whether mice might be a useful animal model for myopia research because of the variety of genetic manipulations available. When he returned from New York to Singapore he initiated both epidemiological and animal research that led to Singapore becoming one of the world's leading centers of myopia research. We hope this volume will be a step in the direction of bringing together the fields of animal and epidemiological research into the etiology of myopia.

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