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INTERNATIONAL MYOPIA CONFERENCE PROCEEDINGS: CONFERENCE PAPER:

Understanding and Treating Myopia: What More We Need to Know and Future Research Priorities

Sally A. McFadden, PhD

Vision Sciences Group, School of Psychology, Faculty of Science and IT, and Hunter Medical
Research Institute, The University of Newcastle, Callaghan, New South Wales, Australia

Abstract

Tantalizing treatment options to limit further global increases in the prevalence of myopia are emerging. However, in order to design more effective interventions, we still need to learn more about the underlying causes of myopia and the associated biological changes. Based on the outcomes of the 2015 International Myopia Conference, this short article summarizes what more we still need to discover and suggests possible priorities for future research.

Keywords

myopia; myopia treatments; lens treatment; drug treatment; wavelength; retina; spatial vision;
research funding; future research

The final session at the International Myopia Conference (IMC, 2015) has raised a spectrum of areas and approaches that are being actively pursued by scientists and in some cases, through government policy, in pursuit of stemming the extraordinary levels of myopia and high myopia to address the associated impact on individual quality of life and economic costs and consequences. Particularly compelling were the statistics presented on the predicted numbers of people who will be affected by myopia and high myopia by 2050 if the current trends are not changed (Figure 1).^{1, 2}

Because of the urgency of finding suitable treatments, research on testing potential treatments in human clinical trials is paramount. Some of these show encouraging results, with the major ones being: low dose atropine,^{3, 4} orthokeratology or modified lens designs,⁵ and increasing time spent outdoors.⁶ However, each of these still appears to offer only partial or short term solutions, or still require replication, and each has major unanswered questions regarding their mode of action making it nearly impossible with our current state of knowledge, to design interventions that might be truly effective. Thus research on their mode(s) of action is urgently needed. Nevertheless, at least in the short term, research on combining treatments that appear to be partially effective should be one major priority.

At the same time, research priorities should address major unsolved mysteries. For example, knowing what retinal signals we need to target is fundamental. The apparent disagreement between species on the basic question as to whether and which wavelength exposures are important in changing ocular growth (for example blue light inhibits eye growth and red light enhances eye growth in chicks and guinea pigs; yet red filters inhibit eye growth in tree-shrews and some monkey studies),^{7, 8} is surprising given that most evidence suggests that the control of eye growth is phylogenetically conserved. Such findings raise the bar on including important species differences in retinal circuitry in generalizing any interpretations, as well as studying basic questions as to how defocus is processed by the retina, and what role temporal and spatial contrast and signal summation might play.

Many of our current treatment and research approaches related to abnormal eye growth assume that grow and stop signals are on opposite sides of a single continuum. However, it is still unclear if the pathway that makes the eye grow is different to the one that inhibits and/or fine tunes growth. Perhaps instead of focusing on myopia, we might be better off concentrating on signals that cause eye growth inhibition and axial hyperopia rather than those eliciting myopia.

Additionally, much of current research fails to differentiate between different stages of myopia. It is possible that the initiation and progression of myopia may each have distinct treatment needs. The effectiveness of outdoor activity on possibly retarding the onset of myopia more than its progression^{9, 10} and different effects on children of different ages¹¹ may be one such example.⁶

Finally, of fundamental importance is devising treatment strategies for the increasing numbers of individuals with high myopia (predicted to be 911 million by 2050)^{1, 2} whose progression is already in danger of advancing towards degenerative myopia. Indeed, how high myopia develops has received little attention from basic scientists, despite being the most serious cause of blindness and ocular disease with practically no treatment strategies.

Christine Wildsoet raised the issue of which ocular structure would be safest to target for pharmacological treatments and cautioned against targeting a complex structure like the retina.¹² For high myopia and certainly for advanced pathological myopia, the sclera is an obvious target, and research priority should be directed towards finding ways of counteracting the known remodeling changes that accompany long term myopia. To translate such work into useable treatments, we also need better predictors of progression rate and early clinical predictors of pathological changes.

Frank Schaeffel in his Sek Jin Chew Memorial Lecture at the 2015 IMC¹³ highlighted how many of the major hypotheses still unsolved today date back to similar suggestions proposed some 200 years ago. However, since that time, the myopia research community has grown to include a vast range of disciplines, from epidemiology to molecular biology with sophisticated non-invasive optical technologies now available. As has occurred in other epidemics that required urgent global attention, modern funding bodies and the myopia research community need to embrace better sharing and utilisation of interdisciplinary teams

and international consortiums and cooperation to rapidly advance towards finding useful treatments for this relentless epidemic the world is now facing.

REFERENCES

1. Holden BA, Fricke TR, Wilson DA, Jong M, Naidoo KS, Sankaridurg P, Wong TY, Naduvilath TJ, Resnikoff S. Global prevalence of myopia and high myopia and temporal trends from 2000 through 2050. *Ophthalmology*. 2016; 123:1036–42. [PubMed: 26875007]
2. Sankaridurg, P.; Holden, BA.; Fricke, TR.; Wilson, DA.; Jong, M.; Naidoo, KS.; Wong, TY.; Naduvilath, TJ.; Resnikoff, S. Special Session: Brien Holden Memorial Symposium. Global prevalence of myopia, high myopia and temporal trends from 2000 to 2050.. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
3. Wildsoet, C. Final session. Is 'light' a panacea for myopia? Question 4: is there a role for drug—treatments in human myopia—prevention/control? [Schaeffel F: discussant].. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
4. Chia A, Chua WH, Wen L, Fong A, Goon YY, Tan D. Atropine for the treatment of childhood myopia: changes after stopping atropine 0.01%, 0.1% and 0.5%. *Am J Ophthalmol*. 2014; 157:451–7. e1. [PubMed: 24315293]
5. Atchison, D. Final session. Is 'light' a panacea for myopia? Question 3: is there a role for lens—treatments in human myopia—prevention/control? [Troilo D, discussant].. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
6. Rose, K. Final session. Is 'light' a panacea for myopia? Question 1: do human studies 'prove' that (i) outdoor activity is protective, (ii) light is the agent? [Flitcroft I, discussant].. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
7. Norton, T. Final Session: Is 'Light' A Panacea For Myopia? Question 2: what do animal studies tell us about the mechanism of myopia—protection by light? [Ashby R, discussant].. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
8. Smith, E, 3rd. Final Session: Is 'light' a panacea for myopia? Question 5: what more do we need to know? What kind of research to support? [McFadden S, discussant].. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
9. Rose K, French A, Mitchell P, Morgan I. The role of time outdoors on the progression of refractive error in Australian children. *Invest Ophthalmol Vis Sci*. 2013;54. E-Abstract 5959.
10. Jones-Jordan LA, Sinnott LT, Cotter SA, Kleinstein RN, Manny RE, Mutti DO, Twelker JD, Zadnik K, Group CS. Time outdoors, visual activity, and myopia progression in juvenile-onset myopes. *Invest Ophthalmol Vis Sci*. 2012; 53:7169–75. [PubMed: 22977132]
11. Jin JX, Hua WJ, Jiang X, Wu XY, Yang JW, Gao GP, Fang Y, Pei CL, Wang S, Zhang JZ, Tao LM, Tao FB. Effect of outdoor activity on myopia onset and progression in school-aged children in northeast China: the Sujiatun Eye Care Study. *BMC Ophthalmol*. 2015; 15:73. [PubMed: 26152123]
12. Wildsoet, C. Topic 5: Regulation of Eye Growth - Roles of Retinal Pigment Epithelium (RPE) & Choroid. Evidence & possibilities for local ocular growth regulating signal pathways.. Presentation given at the 15th International Myopia Conference; Wenzhou, China. September 24-27, 2015;
13. Schaeffel F. Myopia - what is old and what is new? *Optom Vis Sci*. 2016; 93:XXX–XX.

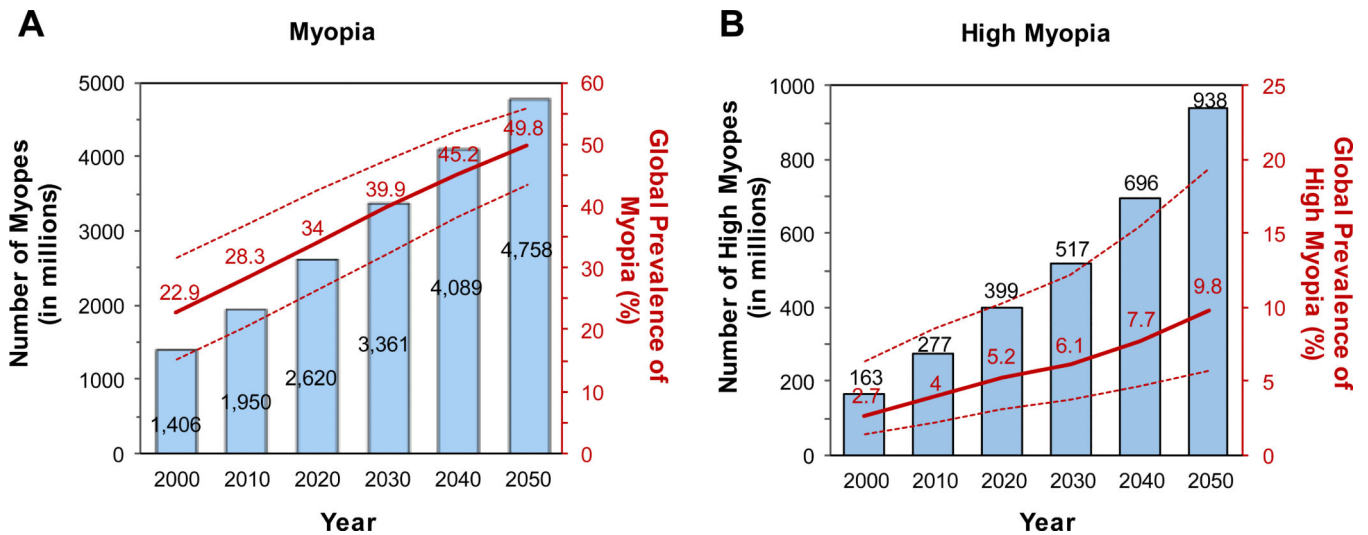


Figure 1.

Global numbers affected (grey bars) and percent prevalence (red lines) in the world population for A. Myopia, defined as -0.500 DS, and B. High Myopia, defined as -5.000 DS. Predicted data is based on a Comprehensive Meta-analysis of existing data. Adapted with permission from IMC 2015 presentation by P. Sankaridurg.² Dotted lines show 95% confidence limits calculated in the meta-analysis of prevalence data, and includes uncertainty in future population projections as estimated by the high and low fertility population projections from United Nations data. For further details of methods see article by Brien Holden and colleagues.¹