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EDITORIAL

The Ethnicity Blind Spot: Why Race-Neutral Myopia Guidelines Are Failing South and East Asian Children

Shibal Bhartiya[1], Syril Dorairaj[2]

[1]Clinical Director, Ophthalmology & Community Outreach, Marengo Asia Hospitals, Gurugram, Haryana, India; Research Collaborator, Mayo Clinic, Jacksonville, Florida, USA; Executive Editor, Journal of Current Glaucoma Practice;
[2]Professor of Ophthalmology, Mayo College of Medicine and Science; Consultant, Department of Ophthalmology, Mayo Clinic Jacksonville, Florida.

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Address for correspondence:
Shibal Bhartiya,
Marengo Asia Hospitals, Sector 56,
Gurugram 122011, Haryana, India.
E-mail: shibalbhartiya@gmail.com

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Abstract

Myopia is increasing globally, yet ethnic variability in disease patterns remains underrecognized. Differences in prevalence, progression, and treatment response across populations highlight the limitations of a one-size-fits-all approach. This article explores the role of ethnicity in shaping myopia risk and outcomes, emphasizing the need for inclusive research and personalized care strategies. Addressing this "ethnicity blind spot" is critical for improving visual health outcomes worldwide.

Introduction

The myopia epidemic has numbers that are difficult to look at steadily. In urban East Asian countries, myopia now affects 80 to 90 percent of young adults, with high myopia, defined as spherical equivalent of −5.00 dioptres or worse, reaching approximately 20 percent.[1-4] In urban Indian children aged 5 to 15 years, prevalence rose from 4.44 percent in 1999 to 21.15 percent in 2019, with projections suggesting it will approach 48 percent by 2050 unless active intervention is instigated.[4] Globally, the pooled prevalence of myopia in children and adolescents has risen from approximately 24 percent in 1990 to nearly 36 percent in 2023, with East Asian children disproportionately represented.[2]

These numbers are alarming. They are also, by themselves, insufficient to drive the right clinical response. Because the guidelines used to manage myopia progression in Asian children were largely built on evidence generated in non-Asian cohorts, they assume a biological and environmental uniformity that does not exist.

This editorial argues that the most consequential gap in paediatric myopia care today is not the absence of effective interventions, we have several, but the absence of ethnicity-stratified risk stratification. A child entering a Gurugram school at age six faces a meaningfully different trajectory than a child entering a school in Amsterdam at the same age. We do not yet treat them accordingly.

The Divergence Cannot Be Explained by Behaviour Alone

The standard public health narrative attributes myopia progression to near work, screen time, and reduced outdoor activity. These factors are real, and their role is not disputed. What is disputed, or more precisely, what is underexamined, is whether they are sufficient to explain the magnitude of the ethnic divergence.

A systematic review of 143 population-based surveys across 42 countries found that East Asian children showed the highest myopia prevalence, reaching 69 percent by age 15, compared to approximately 20 percent in white European children of the same age, and 5.5 percent in Black African children. Crucially, the time trends over the last decade showed a 23 percent increase in East Asians, with only a weaker increase among South Asians and small changes among western populations.[5] If near work and screen time were the primary drivers, and if these behaviours

have converged globally across affluent urban populations, the prevalence gap should be narrowing. It is not narrowing. It is widening.[6-8]

Genetic susceptibility contributes, but incomplete heritability estimates and the rapid pace of the epidemic, too fast for genetic drift, confirm that gene-environment interaction is the operative framework, not genetics alone.

What this means clinically is that the same environmental exposure produces different outcomes in children of different ethnic backgrounds. A South Asian or East Asian child spending eight hours in a classroom is not experiencing the same biological risk as a European child in an identical classroom. The myopia risk embedded in that environment may be ethnicity-modulated.[9-14]

The Light Deprivation Layer

Layered onto the ethnic risk gradient is a mechanism that clinical guidelines have been slow to operationalise: the role of ambient light intensity in axial elongation.

Substantial evidence from animal models shows that light characteristics, including intensity, chromaticity, and photoperiod, influence ocular development and refractive error regulation, possibly via modulation of retinal dopamine. Bright light exposure increases dopamine release and acts as a regulatory brake to slow excessive axial elongation.[15] Outdoor lux levels of 30,000 to 130,000 are typically encountered outside, compared to less than 500 lux indoors. Animal studies have found that light levels in the range of 15,000 to 25,000 lux reduce the myopia and axial elongation produced by form deprivation, and that administering a dopamine D2 receptor antagonist intravitreally abolishes this protective effect.[16]

The clinical implication is not trivial. In a cluster randomised trial, children who spent less time outdoors but were exposed to high light intensity of 10,000 lux or greater also showed a protective effect against myopia, suggesting that light intensity itself, not merely physical activity or viewing distance, is a meaningful variable.[17]

This is where the ethnic and environmental threads converge in a way that should concern anyone practising in South or East Asia. Urban schooling cultures in these regions characteristically combine high academic intensity, long indoor hours, and limited structured outdoor time: often less than 30 minutes per day. The retinal dopaminergic system of a genetically susceptible child is not receiving the luminance signal that, in evolutionary terms, should be calibrating axial elongation. It is receiving, instead, hours of sub-500 lux fluorescent illumination and near-focal visual demand. This is not simply a lifestyle problem. It is a photobiological problem operating within a genetically sensitised substrate.

The Guideline Gap

Against this backdrop, it is worth examining what most clinical guidelines currently recommend. Atropine, orthokeratology, and defocus-incorporated spectacle lenses have level-one evidence for myopia control. They work.

But the major trial data underpinning dosing recommendations, including the Low-Concentration Atropine for Myopia Progression (LAMP) studies and the seminal atropine dose-response work from Singapore, were conducted in predominantly East Asian populations. The extrapolation of these findings to South Asian and Indian children, who carry overlapping but not identical genetic and environmental risk profiles, is assumed rather than demonstrated.[1-18]

More concerning is the default clinical encounter: a child presents with −1.00 dioptres, is given spectacles with full correction, and sent home. Myopia control, the active, evidence-based effort to slow axial elongation, remains an add-on consideration in most South Asian clinical settings, offered selectively rather than as the default standard of care for a high-risk population.

Children from urban environments have 2.6 times the odds of myopia compared to those from rural environments.[5] Every child presenting to an urban clinic in South East Asia is already in the highest-risk demographic stratum. Treating them as if they face average risk is not conservative. In hindsight, it just may be negligent by omission.

What Ethnicity-Stratified Care Would Look Like

The argument here is not that a new drug or device is needed. The argument is that a new clinical decision framework is needed. One that treats ethnic background and urban geography as formal risk inputs at the point of first presentation, not as background context.

Concretely, this would mean: any child of South or East Asian descent presenting in an urban environment with first documented myopia, regardless of magnitude, should be considered high-risk for rapid progression and offered myopia control as the default rather than the exception.[9-17] Axial length measurement should be routine, not optional, not because the number alone is actionable but because serial axial length provides the only objective measure of whether an intervention is working.

Outdoor light exposure should be prescribed with the same specificity as medication, not "spend more time outside" but a structured recommendation of a minimum of 90 minutes daily in ambient light exceeding 1,000 lux, consistent with the evidence base.

Finally, the conversation about risk should happen at first presentation, with parents, in culturally relevant language that does not frame myopia as a minor inconvenience but as a progressive condition carrying long-term risk of glaucoma, retinal detachment, and myopic maculopathy, risks that scale with axial length and that are, to a meaningful degree, modifiable.[18]

A Call for South Asian Cohort Data

There is a specific research gap that this journal is well-positioned to highlight. High-quality, prospectively designed myopia progression studies with axial length as the primary endpoint, conducted in South Asian urban children, with ethnic background and documented light exposure as covariates, are scarce.

The Indian subcontinent is on the same trajectory as East Asia was two decades ago. The current myopia epidemic in India, unless countered by active anti-myopia strategies and lifestyle changes, is projected to follow the East Asian pattern within a few decades. We have the epidemiological warning and the mechanistic understanding. What we lack is the cohort-level data to build ethnicity-specific intervention protocols. That work needs to begin now, and it needs to begin in our own institutions, with our own patients.

The myopia epidemic will not be resolved by applying Western guidelines to Asian children and hoping the biology cooperates. It will require acknowledging that the child in front of us is not a generic patient, and that the guidelines we reach for were not written with her in mind.

Conclusion

Ethnicity plays a crucial yet underrecognized role in myopia. Integrating ethnic variability into research and clinical decision-making is essential for equitable and effective eye care.

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