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A look at literature on myopia over the past 25 years: a personal review

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ABSTRACT

Over 100 years ago, Professor Foucher of Université Laval in Montreal (Canada) suggested that myopia was the result of an interaction between genetics and the visual environment, implying a Darwinian response to our changing world. His words are still relevant today. Over 30,000 articles have been published since he spoke then. Have his questions been answered? What has been learned from this body of research, particularly in the last 25 years? The purpose of this paper is to review the scientific evidence on myopia and to give clinical significance to the results and conclusions presented. It is therefore not a conventional review. More specifically, this work covers the major trends that have characterised myopia research, allowing us to refine our understanding of the mechanisms leading to the onset and development of myopia and to assess the effectiveness of optical and pharmacological methods for its treatment. This is a clinically oriented text that helps to understand why the strategies used to treat myopia produce certain results but also highlights their limitations. It opens up new perspectives. Science has indeed answered many questions about myopia. But it has also raised many more that need to be addressed in future research notably to facilitate the most accurate prediction of the evolution of a particular individual and his or her response to a given strategy.

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Introduction

In 1894, Professor Foucher, from Université Laval (Montreal) wrote: '*Is myopia an inevitable evil, offering little importance; and should we consider this breach of refraction, in the light of a Darwinian theory, as an adaptation of the eye, in conformity with the requirements of our civilization* ?'[1] The author was posing the very same unanswered questions which continue to persist one hundred years later. '*We have even been so far as to express the opinion that this error is not an accident of birth, but that we may have a certain hereditary predisposition to it; and that it appears only under the influence of labor, maintained and prolonged, and wrought in an insufficient or unfavorable light*'.[1]

It would be discouraging to think that all the research carried out since this statement has failed to provide answers to the questions raised. Instead, it is imperative to acknowledge that research conducted on myopia, particularly over the past 25 years, has contributed to improve the knowledge about the complexity and multifactorial nature of this refractive error, despite its apparent simplicity.

As for example, the National Academy of Science has recently classified myopia as a disease after a comprehensive review of its pathological consequences. This should be regarded as a paradigm shift, a real game changer both in terms of our comprehension of myopia and the obligation, for eyecare practitioners and all stakeholders, to prevent and treat it.

The aim of this review is to outline the results of research carried out over the last 25 years and, where appropriate, to translate them into clinical concepts.

Review methodology

Despite the fact that the present article has been inspired by traditional reviews, it is not written as a traditional one. Rather than providing a comprehensive overview of the extant literature on the subject, as it is available elsewhere, the objective is to translate for clinicians the knowledge that basic

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and clinical research brought to bear on myopia, its mechanisms and its impact. The selection and citation of articles was determined by the authors' editorial discretion, following an extensive process aimed at identifying potentially significant articles of various types from the literature published between 2000 and 2024. This identification process was conducted using the PubMed and Web of Science databases. The keyword 'myopia' was used in isolation and in conjunction with other key words, including 'retinal defocus', 'high', 'mechanism', 'genetics', 'pathologies', 'blur', 'spatial frequencies', 'contrast theory', 'contrast sensitivity', 'eye growth', 'management', 'control', 'optical devices', 'atropine', 'soft lenses', 'glasses', and 'red light therapy'.

Only articles published in English and in peer-reviewed journals were selected for inclusion in this manuscript. The 10 most cited ones, during the last 25 years, are listed in Table 1.

It is important to note that the authors deliberately chose not to report data on refraction and axial length related to the effectiveness of various myopia control strategies, as it was almost impossible to compare data between studies. The populations were not identical, and the conditions under which the studies were conducted varied. It is extremely difficult to make comparisons between control groups formed 10 years ago and those of today, not to mention that some studies were based on historical groups that may not be relevant now. As a result, the authors of the present article chose to use the conclusions and general trends from meta-analyses to translate them into clinical terms.

Genetics and environment

In the early 2000s, the primary focus of research was on understanding the genetic basis of myopia and its relation to environmental factors. Preliminary findings indicated that heredity exerted a more significant influence on refractive error onset compared to environmental factors.[2] The genetic approach was substantiated by the observation that the prevalence of myopia exhibited variation between ethnic groups and even within specific regions, such as Asia.

The inheritance of refractive error was considered particularly true for higher myopes,[3] especially if associated with neurological issues.[4] By the mid-2000s, genome-wide association studies began revealing specific loci linked to myopia[5] and the importance of environment to favour their expression (nature vs nurture) began to emerge. It was a complex undertaking to achieve genetic mapping in myopia, but it became obvious that refractive errors are driven by genes affecting every component of the eye.[6]

However, as early as 2002, other authors suggested that the interrelationship between genes and the living environment was not dichotomous but complementary,[7] some others founding evidence that both factors are equally important[8] or may compensate for each other.[9] It has now been demonstrated that genetic components have the capacity to influence the manner in which patients respond to control strategies, which represents a modification of their visual environment.

The role of outdoor exposure[10] and sporting activities is contingent upon an individual's genetic profile.[11] CLEERE study[12] emphasised that the presence of myopic parents was not the sole factor that could account for the onset of myopia at an early age. Studies conducted in Singapore and in the United States showed that environmental risk factors explained around 20% of the variance in myopia.[13] Other risk factors identified were: prolonged near work,[14] increased education,[15] living in urban areas,[16] higher glycaemic index diets, body mass index, insulin resistance,[17] lighting levels, uncorrected refractive error during childhood and binocular vision dysbalances.[18] Social factors include births from older mothers, fertility treatment,[19] intrauterine growth retardation, smoking or exposure to smoke during pregnancy, and lower socioeconomic status.

More precisely, it was established that the growth of the eye is governed by a feedback mechanism involving image processing in the retina.[20] The image seen is influenced by its environment; its interpretation is influenced in fair part by the inherited characteristics of the eye. An exhaustive analysis of the gene-environment impact on all the layers of the retina has been the subject of a landmark publication in this field.[21]

As of today, myopia onset is now considered to be secondary to the presence of inadequate visual stimulation, genetically determined alterations in the feedback loops, and/or inappropriate responses of the target tissues.[22]

Table 1. Ten most cited articles in the field of myopia (2020–2025).

No. citations*	Authors	Title	Reference	Summary
3047	Holden BA, Fricke TR, Wilson DA et al	Global Prevalence of Myopia and High Myopia and Temporal Trends from 2000 through 2050.	Ophthalmology. 2016 May;123(5):1036–42	Current projections indicate that myopia and high myopia will account for approximately 50% of the global population by the year 2050, with approximately 1 billion individuals affected by high myopia. This substantial increase carries profound ramifications for the management and prevention of myopia-related ocular complications and vision loss.
1405	Nickla DL, Wallman J.	The multifunctional choroid.	Prog Retin Eye Res. 2010 Mar;29(2):144–68.	The choroid is a highly vascularised structure in the eye, characterised by distinctive features that modulate its thickness, regulate choroidal tone, and intrinsic neurons that contribute to the control of choroidal blood flow and ocular homoeostasis. These mechanisms are implicated in retinal function, ocular refractive adaptation, and the pathogenesis of myopia.
1385	Morgan IG, Ohno-Matsui K, Saw SM.	Myopia	Lancet. 2012 May 5;379(9827):1739–48.	Myopia has become a major public health issue in East Asia due to its high prevalence and associated pathologies. Lifestyle factors, reduced outdoor activity, and potential genetic contributions influence its development, while promising interventions for prevention and treatment are still under investigation.
884	Rose KA, Morgan IG, Ip J et al.	Outdoor activity reduces the prevalence of myopia in children	Ophthalmology. 2008 Aug;115(8):1279–85.	The present study has demonstrated that higher levels of outdoor activity, as opposed to indoor sport, were associated with a more hyperopic mean refraction and lower myopia prevalence in 12-year-old students. The most protective effects were observed in those with high outdoor activity and low near work, while no such associations were found in 6-year-olds.
799	Wallman J, Winawer J.	Homoeostasis of eye growth and the question of myopia	Neuron. 2004 Aug 19;43(4):447–68.	The present review examines the homoeostatic regulation of eye growth, focusing on the role of visual input in guiding growth, the mechanisms distinguishing ocular blur from myopia, the chemical signals involved in growth control, and the reasons why myopia persists in children despite this regulation.
793	Saw SM, Gazzard G, Shih-Yen EC, Chua WH	Myopia and associated pathological complications	Ophthalmic Physiol Opt. 2005 Sep;25(5):381–91.	Myopia, particularly high myopia, not only imposes economic and social burdens but also increases the risk of ocular complications such as cataracts, glaucoma, retinal detachment, and optic disc abnormalities, highlighting the need for early detection and intervention to prevent its progression and associated visual loss.
774	Morgan IG, French AN, Ashby RS et al.	The epidemics of myopia: Aetiology and prevention	Prog Retin Eye Res. 2018 Jan;62:134–149.	The myopia epidemic in East and Southeast Asia, characterised by high prevalence rates of myopia and high myopia in young adults, is driven by intensive education and limited outdoor activity. Potential solutions to prevent future vision loss include strategies such as increased outdoor time in schools and clinical methods to slow progression.

(*Continued*)

Table 1. (Continued).

No. citations*	Authors	Title	Reference	Summary
694	He M, Xiang F, Zeng Y, Mai J et al.	Effect of Time Spent Outdoors at School on the Development of Myopia Among Children in China A Randomized Clinical Trial	JAMA. 2015 Sep 15;314(11):1142–8	A study of 6-year-old children in Guangzhou, China, revealed that incorporating 40 minutes of outdoor activity into their school curriculum resulted in a 9.1% reduction in the incidence of myopia over a period of three years, when compared to the control group. This finding was accompanied by a significant difference in spherical equivalent refraction, but no significant difference in axial length elongation. These observations underscore the potential benefits of augmenting outdoor time as a strategy for myopia prevention.
679	Lin LL, Shih YF, Hsiao CK, Chen CJ	Prevalence of myopia in Taiwanese schoolchildren: 1983 to 2000	Ann Acad Med Singap. 2004 Jan;33(1):27–33.	A review of nationwide myopia surveys in Taiwan revealed a significant increase in the prevalence and severity of myopia among children, with earlier onset ages. This suggests that addressing eye care for preschool children is crucial to reduce the growing prevalence and severity of myopia.
640	Dolgin E.	The myopia boom	Nature. 2015 Mar 19;519(7543):276–8.	Over a century ago, renowned British eye surgeon Henry Edward Juler recommended that a change of air, such as a sea voyage, be prescribed once myopia became stationary, reflecting ideas that, as modern scientist notes, have taken a century to be revisited.

*As of May 11, 2025. Web of Science database.

Retinal response to visual signals

After these first steps, researchers found that this response appeared to influence eye growth beyond the phenomenon of emmetropization. Wallman and Winawer[23] introduced the concept of retinal homoeostasis. Rozema[24] has detailed that ocular growth, between the ages of 18 months and 17 years must be accompanied by a delicate balance between its various components in order to maintain clear vision at all distances. During this period of retinal homoeostasis, hyperopia initially increases marginally in order to establish a reserve,[25] corresponding with the natural tonus of the accommodative system. Subsequently, during the second phase, the reserve is depleted, ideally by adulthood. The equilibrium is tenuous, with a constant struggle between the growth of the eye and its ability to adapt its power, subject to disruption at any moment. This is why Flitcroft[26] consider myopia onset as a failure in homoeostasis.

Models

Three different models were identified. The first one targeted the choroidal reaction to visual stimulation, in effecting ocular and refractive change.[27] Thinning of the choroid induced by visual stimulation is linked to eye elongation while factors associated with slowing eye growth typically cause short-term choroidal thickening. This is why choroid is now considered a significant biomarker of eye growth.[28] A second model identified scleral changes that are associated with growth adaptation.[29] The relay between the choroid and the sclera is provided by the retinal pigment epithelium. It is also hypothesised that atropine may enhance this function.

A considerable body of research[30] has indicated the involvement of biomodulators in myopia development.[31] Of particular interest is the role of scleral extracellular matrix proteins and developmental genes of the eye. The alterations in retinal dopamine metabolism had been observed and were considered consistent with a hypothesised role for dopaminergic amacrine cells in the visual regulation of eye growth.

The emerging picture is one of complex interaction. Mutations in several genes likely act to dysregulate natural eye growth balance as a response to its environment and visual stimulation.

A third model would emerge gradually, with the detection of defocus by photoreceptors serving as a trigger for the first two mechanisms that had been identified. The modification of the on and off pathway had been identified as a contributing factor to the development of myopia. This alteration may be pharmacologically induced; however, spectral and temporal properties of light may also influence eye growth.[32] For instance, exposure to low frequencies may also alter the on and off pathway response. It was found therefore imperative that spatial frequency contents of the visual landscape/scenery and its contrast are incorporated into the factors of the environment, impacting the quality of the visual signal and, in turn, that of the retinal response. In clinical terms, this means that practitioners must not only deal with the ocularenvironment but must also make recommendations to expose the individual to the most favourable environment (open, green spaces, involving a spectrum of high and low frequencies, and a balance of contrasts).

The concept of blur

The concept of blur had been extensively studied, both in terms of the central image reaching the retina and that affecting the periphery. Research has demonstrated that the retina possesses the capacity to discern whether such blur is situated in front of (myopic defocus) or behind the tissue (hyperopic).[33] Both signals can be recognised at the same time. This explains why orthokeratology and soft multifocal lenses can alter eye growth.

Interpretation of blur is based, in part, on the recognition of optical aberrations (chromatic, spherical).[34] The absence of aberrations perception could potentially result in an incapacity to compensate ocular growth, owing to a deficiency in directional cues.

Smith et al. demonstrated that myopia can be induced by form deprivation stimulation,[35] which is a consequence of a different mechanism. It is believed that form deprivation is interpreted by the retina as an extreme hyperopia. [24] He also demonstrated that the impact of optical defocus is local, regional, and is predominantly characterised by mechanisms that integrate visual signals in a spatially constrained manner.[36]

Central vision exerts minimal influence on the signals that dictate eye growth.[37] In contrast, the peripheral retina plays a dominant role in this process, whether the signal is located in front of or behind the retina. Local operation would result from the presence of a retinal mechanism which quantifies the loss of image contrast at specific spatial frequencies, attributable to diffusers or defocus.[38]

Following these findings, a paradigm shift in the interpretation of refractive errors was imminent. This new paradigm would necessitate the consideration of the retinal surface in three dimensions, bringing a new level of complexity to the model of emmetropization and eye growth. A model revisited should then take into account the inherent intricacy and interplay between the environment, ocular optics and ocular shape. In this regard, it should be noted that the elongated and more prolate retina of highly myopic individuals may not respond in the same way as the retina of individuals with mild to moderate myopia. Indeed, the misalignment and change in the density of the photoreceptors, the vascular changes in the superior and inferior retina and the thinning of the choroid may minimise the potential of these eyes to resist elongation.[39]

Hung and Ciufredda suggested the Incremental Retinal Defocus Theory,[40] which suggests that the change in retinal defocus magnitude during an increment of genetically programmed ocular growth provides the requisite signal for the appropriate alteration in subsequent environmentally induced ocular growth. Moreover, the rate of change of blur size provides the direction for blur-driven regulation. This theory also involves the concept of duration of exposure to the stimulus as a variable to be considered. Short-term exposure to negative stimulation generates a recovery action predicated on an interpretation of the newly generated blur.[41] This protective reaction happens rapidly after removal of the stimulation.[42] To the opposite, recurrent cycles of near work engender repeated periods of decreased retinal-image defocus. This long-term exposure is hypothesised to result in an increase in axial growth, thereby leading to the development of permanent myopia.[43] Another element to add to this is the extent of the field exposed to the defocus. The most effective treatment strategies would be those that influence visual signals over a large area of the retina.[44] It was confirmed recently as the area covering 6 to 10° from the fovea.[45] which means to

extend slightly over the macula (1 deg = 0.3 mm). This helps to explain why, recently, smaller treatment areas in orthokeratology (OK) were proven more effective, as they increase the defocus in the sensitive area. Similarly, by dilating the pupil, atropine allows more defocus to reach the target and increases the dose.

Managing myopia

It is essential to manage every myopic kid, as there is no safe level of myopia.[46] Just one dioptre saved helps to reduce the risk of myopic maculopathy by 40%.[47]

Cho[48] laid the foundations for the significant contribution of OK in myopia management. It was determined later that these lenses could influence peripheral refraction, generating aberrations and favourable myopic defocus, thus clinically confirming the theoretical bases put forward by Earl Smih III

The COMET study[49] was another seminal piece of research in the field. At the time, the prevailing opinion was that the development of myopia was primarily caused by excessive effort in near vision. It was demonstrated by COMET authors that progressive/bifocal devices had no significant impact, including the exception of a niche group of patients who exhibited a high lag of accommodation and an esodeviation at near.

In 2020, a new Cochrane review[50] was published. From this review, the body of evidence indicates that the strategy of under-correction is ill-advised and results in increased axial length evolution. With regard to spectacle lenses, the analysis confirmed the earlier disappointing results concerning the use of progressive/bifocal lenses to manage myopia progression. Peripheral defocused spectacles were emerging, yielding encouraging but still preliminary results. In the field of contact lenses, OK and multifocal soft lenses have been demonstrated to be more efficacious than single vision lenses (SVL) in retarding the progression of axial length. As for the pharmacological approach, atropine was substantiated as the only molecule considered efficient and commercially available for myopia management.

At the same time, it was demonstrated that the use of single vision contact lenses may be associated with faster progression. Conversely, multifocal lenses and those designed with a more convex peripheral power have demonstrated comparable efficacy to OK. There are numerous potential explanations for the observed performance: reduction of the accommodative lag, of the hyperopic defocus in the retinal periphery, imposition of a myopic defocus across the retinal surface, and compensation of the negative spherical aberrations generated during accommodation.[51]

Comparing efficacies

In 2019, the International Myopia Institute[52] published a white paper on the management of myopia. This publication looked at various aspects of myopia management, including the nature and type of strategy proposed for myopia management. From this paper, many factors must be considered before making a final selection such as current age and age at onset of myopia, previous progression, degree of refractive error, presence of astigmatism, binocular vision status, pupil size, corneal parameters and profile, axial length vs risk of high myopia, reading/screen habits, safety of the devices, etc. Parental expectations and child behaviour, potential compliance also play a role. Resources such as the cost of the goods and their availability are certainly other important elements that may limit the options offered. The relative effectiveness of each approach must also be considered.

Based on a comprehensive review of the literature at the time, the authors of this white paper showed that the strategies used seemed to be all equivalent in terms of their average effectiveness. The selection of the strategy may be rationalise using decision trees.[53–55] However, it is crucial to remember that the patient in the exam chair is unique and may diverge from average results or normal curves for cohorts of the same age, ethnicity or refraction. Myopia management must be customised, one child at a time.

Specific strategies

Orthokeratology (OK)

In 2005, Loric study[48] was the first suggesting that OK may be influencing the development of myopia beyond its known effect of correction. Ten years later, a meta-analysis of eight randomised clinical trials showed that OK reduced axial length significantly compared to single vision lenses.[56]

It is important to remember the basic principles to understand such results. Corneal moulding produces a huge increase in positive spherical aberration (on-axis), and a reduction in contrast sensitivity, which are considered key factors in influencing the retina's response to visual signals.[57] Coma is also present, in the peripheral retina, contributing to the results.

To optimise these results OK lens design evolved since the early works of Jessen in 1962. The most significant changes have been the use of a reverse curve (Wlodyga-Stoyan design), the use of toric peripheral curves, to help with lens centration and stabilisation, the increase in the overall diameter to cover 95% of the corneal surface. A smaller central zone helps also to generate more defocus in the pupil and increase the dose needed to control eye elongation.[58] An optimal coverage of the pupil by the optical defocus (Plus Power Ratio) must be reached to be effective.[59] Any change in lens design must be evaluated with a topography. Analysing the topographic maps before and after fitting is the only way to understand the optical changes caused by the corneal reshaping.[60]

OK does not generate major changes in the binocular vision.[61] The patient's quality of life is improved as it can be assessed using a dedicated questionnaire.[62]

The use of OK lenses may be limited by the fact that they were associated with a higher risk of infections and keratitis caused by several organisms.[63] By using modern lens designs, high-permeability materials and insisting on hygiene and proper lens care, OK lens wear is nowadays considered safe and efficient for myopia management.[64]

Soft lenses

For soft lenses, it is suggested to use the highest peripheral convex power (add), considering that there is a dose-response relationship at the retinal level[65]; the dose of defocus being defined by the lens power and the area of its impact on the retina. This was confirmed during a 3-years longitudinal study.[66] It has also recently been shown that the effect of fixed amount of defocus (same add for every distance vision correction) varies at the retinal level as the eye is stretched.[67] This may suggest that a higher dose may be required for higher levels of myopia.[68]

Many of the lenses used to manage myopia are based on designs intended to correct presbyopia. In the former case, it is crucial to significantly increase the level of peripheral high order aberrations to influence the retinal response. In presbyopia, these aberrations must be minimised to maintain visual quality at all distances. It is not logical to use the same design for both purposes.

Dual focus lenses are the most studied soft lenses for myopia management.[69] Overall, lens wear reduced the progression of myopia and axial length significantly and on a sustained basis for 6 years. Older participants performed better. There was no benefit to combine with atropine low-dose (0.01%). Visual acuity may be reduced compared to SVL on some patients,[70] but accommodation was not altered by the wear of these lenses.[71] There was no rebound effect at the end of the treatment.[72]

The concept of extended depth of focus had been introduced in recent years. Depth of focus is the area of clear vision along the axis over which an image can be focused and perceived as clear. A single vision lens has a single focal point and consequently a short depth of focus. Bifocals lenses have habitually two focal points. Each focal point is associated with a short depth of focus and around each of those is a 'halo' created by the alternative focal point. Intermediate vision may be blurred as a consequence. The extension of this depth of focus can be achieved through the manipulation of lens design, generating a non-monotonic, aperiodic profile characterised by a varying power distribution, with no discrete zones. Consequently, contrast sensitivity potentially declines, concomitant with a deterioration in the retinal optical quality of the image. This loss of contrast may contribute to myopia management. Their efficiency for myopia management was confirmed, although limited in comparison with other modalities.[73]

One similar design is based on catenary optics. The centre of the lens provides distance vision correction but within 5 microns from the optical centre, more convex power is introduced and increases rapidly to reach +3D at the optic zone limit. This virtual pinhole effect mimics extended depth of focus design although not adopting an aperiodic power profile. Preliminary results, obtained after a 1-year period, demonstrate a satisfactory level of efficacy. Nevertheless, it is imperative to await the results of a more extended period in order to provide a comprehensive evaluation of this option.[74]

Wearing contact lenses not only helps to manage myopia but also has a positive effect on self-esteem of the young patients, perception of their physical appearance, athletic ability and social acceptance.

Despite these positive elements, some practitioners may be reluctant to fit children with contact lenses, citing safety issues or the difficulty of teaching them how to handle and care for their lenses. Otherwise, Gifford showed that the risk associated with contact lens wear is much lower than that of visual impairment associated with myopia progression.[75] It has also been shown that children can safely wear contact lenses, and that the time required to teach them to handle and care for lenses exceeds that required for adults by no more than 15 min.

Peripheral defocus glasses

The first introduction of a dedicated design in glasses was in 2020 with the launch of Defocus Incorporated Multiple Segments lenses in the market. It is now supported by many years of data collected from a randomised clinical trial. The design of the lens is made to provide a significant level of defocus (+3.50). The optical effect effectively impacts the 'sweet spot' of the retina.[76] On the long term, participants who wore these lenses for 6 years showed the least change over time. No rebound effect was observed at the end of treatment.

The Highly Aspheric Lenslet technology is the second design that has been studied in a rigorous long-term clinical trial.[77] After 5 years, the results are showing a significant reduction of myopia and axial length evolution. This lens is delivering a high volume of peripheral defocus, always the same, regardless of distance correction. This implies that the power and distribution of lenslets vary according to the refractive error, respecting dose-response principle more than any other design in the field.

Contrast theory

The Diffusion Optics Technology lenses are designed under the concept of reducing contrast sensitivity and were studied for 4 years.[78] Such reduced visual signal is known as altering the on-off pathway, enhancing the on response and consequently its positive impact on axial length evolution.[79] It should be noted here that other designs relying on peripheral defocus also result in a reduction in contrast.

After 4 years, Diffusion Optics Technology lenses were associated with a reduced progression of myopia and in a slower progression of axial length. These figures may seem lower than other designs, but the trial continued during the COVID and lockdown periods. Compliance and follow-up of participants were greatly affected.

Several other designs have also appeared in recent years, with varying degrees of success. Among these, one manufacturer offers two versions of lenses with cylindrical rings for peripheral defocus. The power of the rings is higher for younger patients, who are considered fast progressors until proven otherwise. This type of treatment is in line with the principles of dose-response and personalised treatment. The results after 1 year seem to indicate that this approach is effective.

There is another lens which design (perifocal) has been reported as efficient in one publication.[80] This study is polluted by many flaws, the paucity of information regarding the population tested and the improvement in results over time which are at odds with the habitual trend observed in more robust studies examining peripheral defocus glasses. Not surprisingly, recent independent studies has shown that this design is less efficient than other devices.[81]

Clinicians need to understand that the products they recommend must be supported by rigorous studies, and if possible, with long-term results.

Performance of spectacles with peripheral defocus designs is highly correlated with compliance. As with other optical devices, patients who did not follow the required wearing times experienced greater progression than others.[82] For this reason, it is recommended that myopia control spectacle lenses be worn consistently, even when reading or playing outdoors.

Low-dose atropine

For decades, the muscarinic cholinergic antagonist atropine has been the primary pharmacological treatment for myopia. This all started with the publication of the Atom 1[83] and Atom 2 studies.[84] However, its mechanism of action is still not fully understood.

The nature of atropine as an antagonist of muscarinic cholinergic receptors was an obvious hypothesis, but the use of other drugs with similar actions did not produce the same results. More promising, authors looked at serotonin as it appears to become elevated when myopia is induced. Atropine has been shown to bind to this serotonergic receptor and antagonise the signalling. Such an effect delayed myopia in chickens.[85] Consequently, atropine mode may be linked to its action on non-cholinergic receptors- like serotonin ones.

Most reviews or meta-analyses showed a dose-response effect for the potential to treat myopia.[86] Atropine's side effects have also been shown to be dose dependent. There is also a rebound effect on discontinuation of treatment, more pronounced at higher doses.[87]

Based on the results of many studies, in particular the LAMP longitudinal study,[88] the use of low-dose atropine has become the standard of care. Originally, a concentration of 0.01% was suggested, but it was demonstrated over time that it may stabilise refraction but not axial length.[89] This is why 0.05% emerges as an optimal dosage balancing efficacy vs side effects,[90] especially if used as an adjunct therapy.

Atropine as monotherapy may be recommended differently. The Dutch approach[91] suggests to rely on a growth chart[92] to determine the dosage needed. Above the 75th percentile, 0.5% is prescribed, along with photochromic progressive spectacles to compensate for side effects. Below the 75th percentile, 0.05% is recommended. Atropine is maintained until the patient shows stability (<0.05 mm AL elongation/year × 2 years in a row). Stopping the treatment too soon is not recommended and may not influence the final outcome, many years later.[93] At higher doses, it is necessary to taper the drug over time at cessation of the treatment. There is no consensus on how and for how long to taper atropine to reduce the rebound effect.

Non -responders

It is also known that there are between 10% and 20% of non-responders regardless of the prescribed myopia management strategy.[94] Part of the non-response is certainly explained by non-compliance, which is also relatively similar across strategies. Some patients may also respond more slowly than others to the same therapy. Frequent patient monitoring is important to monitor compliance and progress. A re-evaluation of the condition, in combination with the addition of low dose atropine to optical strategies, may represent a valid option when monotherapy appears to be ineffective.

Red light therapy

Since 2021–22, several articles have reported on the apparent efficacy of high-intensity red light therapy. Short-term results indicated that not only myopia could be controlled using this strategy, but that axial length also appeared to be reduced in several cases.[95] More recently, some concerns have arisen, notably following a case report describing retinal pathology post-treatment,[96] significant rebound effects,[97] or abandonment rates due to photosensitivity or after-images generated. Some authors[98] have expressed serious doubts about the long-term effect of this exposure to major energy doses, exceeding the norms.[99] Recently, a significant loss of photoreceptors has been linked to this technology.[100] This is of particular concern, given that, as is the case with other therapeutic interventions, a dose-response relationship is present. It is imperative that the utmost caution is exercised until such time as new evidence establishes beyond any doubt that these devices will not endanger children's ocular health in the future.

Conclusion

The 33,000 articles published since the original publication of professor Foucher have confirmed that environment and genetics play a synergic role in myopia onset and evolution. However, many unknowns remain As researchers and clinicians, there is an evident opportunity to enhance our efforts in this area. It is evident that the environment within which we function is undergoing a period of rapid and considerable change. It is imperative that research in this field, and most crucially, the practical implementation of care for individuals with myopia, undergoes a corresponding evolution. The dynamics of myopia management becomes truly fascinating.

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