Myopia has become such an epidemic that the World Health Organization describes it as a key risk factor for ocular pathologies that can lead to blindness. As eyecare professionals and public health authorities respond to the crisis, this article offers a strategic approach with effective mechanisms for myopia control. This approach is based on three pillars of intervention: managing the environments of young people with myopia, managing their ametropia and peripheral defocus, and treating anomalies in binocular vision.

In a report from 2015, the World Health Organization stated that myopia should be considered a key risk factor for ocular pathologies that can lead to blindness. Myopia is therefore a public health imperative that concerns all healthcare professionals and the competent authorities. The report echoes the increase observed over the past two decades in the prevalence of myopia around the world, and in particular the spike in cases of high myopia (>5D), which is associated with the most harmful effects in terms of ocular health. Nearly 90% of the populations of certain Asian countries are already myopic, and Europe and North America are not far behind – nearly 50% of their populations will be myopic by 2050, double the prevalence observed in the 1970s and 1980s. Worldwide, the rate of high myopia will soon reach 10%, a three-fold increase compared to the same reference period.

In light of these facts, it is only natural that eyecare professionals and other industry players around the world are doing their best to halt the unprecedented growth of pathological myopia. A recent series of eight articles presents what we currently know about the identifiable causes of this boom in myopia, both genetic and epigenetic, as well as proven interventions and potential new approaches suggested by the available data. Although many of the questions raised by the research remain unanswered, we can already identify some promising intervention options and predict the ones that will allow us to stem the myopia epidemic.

**THE THREE PILLARS OF MYOPIA CONTROL IN PRACTICE**

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**KEYWORDS**
Myopia control, ocular pathologies, high myopia, ocular health
Who and when?

Before we can begin to discuss intervention strategies, we must identify the clinical population that should be offered myopia control and also determine the right time to begin it.

As trends have shown, any child under the age of 10 who has myopia (-0.50 D and up, with cycloplegic refraction) has a significant risk of their ametropia progressing. Adolescents who become myopic after this age are also considered to be at risk of rapid progression, although at a slower rate. Myopic patients aged 3 to 18 have therefore traditionally been the target population. But due to the greater visual demands imposed upon young adults today, in particular now that post-secondary studies tend to last longer, myopic development often continues past age 18.

For this reason, the clinical population can now be expanded to include individuals up to the age of 25. Since Flitcroft’s research in 2012 on myopia aetiology, it has been proven that no one who has myopia is free from risk when it comes to the development of pathologies leading to blindness. However, the risk is obviously proportional to the degree of myopia reached once the patient’s condition has stabilised. A 2016 study conducted in the Netherlands established more specific criteria for when the impact of myopia on eye health is significant.

The intervention chosen for a myopia patient should therefore make it possible to maintain their ametropia under 6 D or their axial length under 26 mm. If the patient goes beyond these thresholds, the risks of vision impairment increase significantly after 65. We can therefore use them along with growth curves now available to determine whether a patient needs care. More importantly, this information allows us to pinpoint exactly what intensity of control they need and establish the type of intervention.

In the typical patient, we see an average annual progression of 0.50 D and 0.2 mm in axial length. The rate of increase is higher for those who become myopic before 10, patients with high myopia and Asians, whose myopia tends to progress more rapidly. Given these facts, it is possible to predict a young myopia patient’s stabilisation level even though their ametropia progression will not be linear over time (This approach is advocated by the University of Montreal’s clinic, IRLCM).

For example, an eight-year-old first-time patient whose myopia (confirmed with cycloplegic refraction) is -2.00 D will undergo a progression of 5.00 D by the age of 18, reaching a level (-7.00 D) higher than the previously established target. More importantly, since the pathology is related to an elongation of the eye, the axial length, initially measured at 24.5 mm, will increase to 26.5 mm.

But whether in terms of refraction or axial length, numbers exceeding the targets are what confirm the need for intervention.

For a 12-year-old boy with myopia of -5.00 D and an axial length of 25.5 mm, the projection by the age of 18 is -8.00 D and 28.5 mm, levels widely associated with a higher risk of ocular pathology. But if his 14-year-old brother has low myopia (-1.00 D) and a short axial length (23.7 mm), he has a lower risk; his expected progression will result in an average level of myopia (-3.00 D) and an unproblematic eye elongation (24.0 mm).

Choosing an intervention

The first pillar of clinical intervention concerns the myopic patient’s environment. More specifically, the length of time spent exposed to outdoor daylight each day, working distance, ambient and accent lighting, and the use of electronic devices must all be taken into account.

When children spend time outdoors, they are exposed to a light level of about 100,000 lux, which is 10 times brighter than indoor lighting. This higher light level is recognised as a factor that reduces the risk of the onset of myopia, although its beneficial effects are less perceptible once ametropia is present.

For this reason, children should be exposed to outdoor light at an early age, the recommendation being at least 90 minutes per day, without reading or playing video games during that time. A higher dopamine level and exposure to violet light between 370 to 400 nm have been suggested as additional factors protecting against the onset of myopia, although their exact mechanisms remain to be determined and a consensus still needs to be reached on the latter point.

Physical exercise in such an environment can also be beneficial. Indeed, recent data indicates that childhood obesity is linked to different ocular biomechanics: the eyes of overweight children are more deformable and therefore likely to be stretched when they become myopic.

Research on the impact of near work has yielded contradictory results, with some studies suggesting prolonged reading or near work has no effect on the development of myopia. But others confirm that children who read more and at a closer range are more likely to develop myopia or to see a more dramatic progression in their ametropia.

This may be influenced by ambient lighting – some authors believe LED lights have the most harmful effect, followed by fluorescent lights, while incandescent lights seem to cause the least harm. The distribution of the light spectrum, in particular spikes in blue light, is thought to be responsible for these differences. Similarly, it has been suggested the use of electronic tablets and smartphones is conducive to myopia; especially if exposure to these technologies takes place before the age of three. This is due to the chromatic aberrations that are generated. These screens emit both long and short wavelength light, but the...
brain seems to prioritise focus on the longest wavelengths. With blue light, the focus is located in front of the retina, and this is thought to send a defocus signal leading to myopisation of the eye. These conclusions, observed on an animal model, have yet to be validated in a range of human populations and are still subject to debate. But it should also be remembered that screen use is associated with a viewing distance (18 cm) that is only half the normal reading distance (40 cm). This closer proximity has a direct impact on the development of myopia, namely when there is an anomaly in binocular vision, which we will address further on. Moreover, content viewed on electronic devices is renewed continuously due to the absence of indicators for interruption. This leads to long periods of near focus with no visual pauses, unlike when text is read from a printed page.

The second pillar of intervention involves optimising focus in both central and peripheral vision. Any central defocus will be interpreted by the brain as sensory deprivation, which is considered to be a strong trigger for myopisation. It is therefore essential to fully correct ametropia at all times. To this end, cycloplegic refraction is recommended, and the patient’s condition should also be monitored closely and the prescription updated frequently. Furthermore, an undercorrection should never be prescribed since it is equivalent to sensory deprivation and only encourages a more rapid development of the myopia. If central focus is to be prioritised, peripheral refraction with hypermetropic or myopic defocus, which is considered to be a strong trigger for myopisation, has a significant impact on establishing emmetropization mechanisms and controlling the axial length. The local retinal mechanism may detect contradictory signals (hypermetropic or myopic defocus), and if they are of equal intensity, it will choose the signal that slows down eye elongation. Moreover, the retina reacts differently among its various quadrants (nasal, temporal, superior and inferior). Given these facts, the optical control strategy should be oriented towards creating myopic defocus, ideally regulated by quadrant and limited around the macula. It should be remembered that with monofocal Eyeglasses or conventional contact lenses, peripheral refraction is characterised by hyperopic defocus, which is assumed to be an important factor in stimulating growth in axial length.

For this reason, it is essential to create an area of convex power surrounding the area that corrects central vision. This will modify visual input in the retinal periphery and thus reduce the optical factor that promotes progression of the myopia.

The size of the convex area and its location compared to the pupil have been the subject of debate. While some authors have suggested that regardless of location any convex power is beneficial for halting myopia, the most recent data indicates this convex power yields more significant results if the added convex power is greater than +3.00 and located inside the pupil’s diameter at the periphery of the correction targeting the ametropia. In this way it generates more positive spherical aberrations, which are considered to be protective. This would mean the response is proportional to the dose, at least in the animal model. In theory, this suggests the higher the added convex power, the more effectively the visual system responds in terms of halting the myopia.

The above elements are complemented by the third pillar, binocular vision. Any myopic patient for whom an ametropia control strategy is being considered should be given an appropriate binocular vision assessment, analysing the vergence phase (i.e. screen test and near-point of convergence), the accommodation phase (i.e. flexibility, lag, NRA and PRA) and the refractive phase (i.e. AC/A ratio). This becomes especially important when the use of smartphones and tablets is taken into consideration. As mentioned above, because the viewing distance with these devices is reduced by over 50% from 40 cm to a mere 18 cm, the patient must accommodate and converge more than when reading a print text. In an ideal world, orthoptic assessment tests would be done at this reduced distance, comparing the results of a straight gaze and the downward gaze associated with smartphone use.

Any accommodation anomaly – and in particular a lag of over +1.00 D – will be considered a precipitating factor for myopia onset, as if the eye becomes myopic in part to compensate for an accommodation deficit (this is especially the case in myopia patients with anisometropia). Furthermore, no optical myopia control strategy can be effective without a normal accommodative capacity that is unaltered by the wearing of eyeglasses or contact lenses. Excess convergence is associated with more rapid myopia.

Optical methods of myopia control include anti-myopia eyeglasses, multifocal soft contact lenses and rigid orthokeratology contact lenses. This arsenal can be rounded out by a pharmacological approach involving low doses of atropine.

What is the best strategy?

Optical methods of myopia control include anti-myopia eyeglasses, multifocal soft contact lenses and rigid orthokeratology contact lenses. This arsenal can be rounded out by a pharmacological approach involving low doses of atropine.

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Figure 1: A well fitted ortho-k lens on an eye.
The results obtained vary according to product design and the patient’s characteristics (e.g. age, gender, ethnicity, orthoptic assessment, initial degree of myopia). It can be difficult to compare all the published studies that report clinical results, particularly when orthokeratology lenses are involved, since authors only rarely specify the exact design of the lenses studied. This is a major limitation and accounts for the great variability in the results obtained. To be credible, a study must include all the relevant details readers will need if they decide to assess the optical effects associated with the suggested design.47

**Axial length: a real parameter of progression**

Any control method’s effectiveness should be evaluated not only according to changes in ocular dioptries but above all according to progression in the eye’s axial length.8 The axial length should be measured periodically in children prior to myopia onset because its rapid progression during childhood is recognised as a reliable predictor of myopia onset.11 For children with a risk of myopia, it is therefore essential to follow the recommendations for environmental control to delay the onset of ametropia and thus minimise the potential risks of ocular pathology.

The goal of optical correction is to slow myopia progression by influencing peripheral refraction48 to generate myopic defocus.41 This strategy can be applied simultaneously (lenses with concentric powers) or in alternation (lenses with distinct areas).

**Anti-myopia eyeglasses**

Regular eyeglasses with progressive addition or visible ex­ecutive-style lenses aim mainly to correct this accommodative lag and thereby halt myopia progression. Because they are associated with a lower effectiveness,38 eyeglasses with lenses designed specifically for myopia control are preferable. With myopia control lenses, the effectiveness varies between 35% and 50%, which is comparable to certain contact lenses.49 Anti-myopia eyeglasses are recommended for any myopic patients who do not wish to wear contact lenses or simply cannot due to either their young age (under seven) or a particular condition. Patients with insufficient convergence in spite of orthoptic treatment will also do better with eyeglasses than contact lenses, especially if they use anti-myopia eyeglasses with an internal prism.

**Contact lenses**

The vast majority of young people with myopia should nevertheless switch to contact lenses as soon as possible since most of the designs used are associated with more dramatic results in terms of halting myopia progression. Contact lenses can thus generally achieve the effectiveness required to control the ametropia,50 especially when the parameters of the prescribed lenses can be customised. Generally speaking, patients with pupils measuring less than 5 mm under photopic conditions and those whose ametropia is less than 2.00 D will enjoy better control of their condition if they wear multifocal soft contact lenses.12 The lens design must be centred for distance correc­tion.39 In these cases, concentric designs with high addition will offer the best results in terms of halting myopia progression (50% to 60%), while aspheric designs will be less effective.51 A lens recently designed specifically to slow myopia progression, using concentric alternating powers, makes it possible to achieve a 52% slowdown rate in axial growth.52 But lenses designed to increase depth of focus have yielded disappointing performances (25% to 40%) when compared to the others.53 In all of these cases, best results are obtained when compliance is high.54 The lenses should be worn at least 10 hours per day, six days a week. The patient’s reaction also depends on the degree of exposure to peripheral myopic defocus (the dose).

Patients with rapid progression – i.e. all patients whose myopia onset occurred before the age of 10 – whose myopia was more severe at the beginning (>–2.00 D) or who have large pupils (>5 mm) will benefit from orthokeratology lenses. These lenses can generate a high level of convex peripheral power, which is desirable for most patients. There are several designs on the market, but few are designed to control myopia and there is a significant difference between myopia correction and myopia control. They should cover a larger area of the cornea (95%), with more significant bonding, but also have a smaller central treatment area to maximise convex power on the pupil.55 Software programmes can be used to personalise the design type to a patient’s individual parameters and achieve control of 90%.56

![Figure 2: Tangential topo map showing optimization of the ortho-k lens design, with convex ring power inside of the pupil area.](image-url)

For smaller pupils, the effect of orthokeratology lenses can be enhanced by combining this strategy with low doses (0.025%) of atropine. This medication causes a slight dilatation of the pupils (1 mm to 2 mm), which allows for greater exposure to the convex powers generated by the orthokeratology lens. Peripheral refraction is therefore influenced to a greater extent by the convex power generated. Atropine can also be used as a single treatment, without the use of contact lenses. The concentration must be
higher (0.05%) in such a case to achieve good dioptic control, and above all control of eye elongation. At a lower dose (0.01%), although the ametropia would be stabilised, it would not be unusual to see the axial length continue to increase in the absence of a control strategy.**

**CONCLUSION**

All young myopic patients need a detailed assessment of their condition and a strategy for controlling it. Simple monofocal concave lenses should no longer be considered a valid option for correcting myopia in at-risk young people. The control strategy should be designed according to the slowdown rate needed, using a prediction of the degree of myopia in adulthood in the absence of intervention and integrating consideration of the selected treatment methods. With available options today, contact lenses should be chosen as an initial approach, although anti-myopia eyeglasses are also a good and valid option, especially in cases with binocular vision problems or when contact lenses cannot be worn. In more complex cases, contact lenses or anti-myopia eyeglasses can be combined with the use of low-dose atropine (0.025% or 0.05%). For optimum long-term results, it is important to customise the treatment to each patient and to provide regular follow-up care. The constant innovation in products on the market along with a better understanding of the mechanisms at stake will lead to better clinical practice in the future. What’s more, interventions to control myopia and axial length progression will become more effective. As practitioners, we must not hesitate to take an interest in these advances and apply them in our daily work, whether it’s for children or young adults.

**KEY TAKE AWAYS**

- High myopia is becoming epidemic but can be prevented.
- Eyecare practitioners must consider to implement myopia control strategy on any myopic kid entering their office.
- There is not a single option to control myopia: optimal strategy must be personalized.
- An effective myopia control strategy is composed with 3 pillars: control of the environment, presence of a normal binocular function, and control of the blur (central and peripheral) with appropriate optical devices.
- Young adults can be considered for myopia control as kids, because myopia may still progress over 18 years old.

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