RCMOS – Revista Científica Multidisciplinar O Saber. ISSN: 2675-9128. São Paulo-SP.

Ano VII, v.1 2026 | submissão: 07/11/2026 | aceito: 14/11/2026 | publicação: 19/11/2026

Predictors of insufficient response to myopia control in children and adolescents: a narrative review with a structured literature search

Preditores de resposta insuficiente ao controle da miopia em crianças e adolescentes: revisão narrativa com busca estruturada

Predictores de respuesta insuficiente al control de la miopía en niños y adolescentes: revisión narrativa con búsqueda estructurada

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Abstract:
Residual progression during myopia treatment is clinically important because it leaves some children exposed to a greater cumulative risk of high myopia despite the average efficacy of available interventions. This narrative review, supported by a structured literature search, discusses factors associated with insufficient response to myopia control in children and adolescents. Original cohort studies, secondary analyses of clinical trials, and prognostic-model studies addressing atropine, defocus lenses, orthokeratology, and repeated low-level red-light therapy were prioritized. Axial elongation was treated as the primary structural marker, with cycloplegic spherical-equivalent progression considered complementarily. Younger age at treatment initiation, faster pretreatment progression, and persistent axial elongation at early follow-up were the factors most consistently associated with residual progression. Baseline axial length and refraction, parental myopia, choroidal response, and adherence may add prognostic information, but their meaning varies across interventions and populations. No universal threshold defines treatment failure. Escalation should be based on the integration of baseline risk, individual trajectory, treatment adherence, and biometric evolution.

Keywords: Myopia; Axial Length, Eye; Risk Factors; Treatment Outcome; Child.

Resumo:
A progressão residual da miopia durante o tratamento é clinicamente importante porque expõe algumas crianças a maior risco cumulativo de miopia alta, apesar da eficácia média das intervenções disponíveis. Esta revisão narrativa, apoiada em busca estruturada da literatura, discute os fatores associados à resposta insuficiente ao controle da miopia em crianças e adolescentes. Foram priorizados estudos originais de coorte, análises secundárias de ensaios clínicos e pesquisas de modelos prognósticos sobre atropina, lentes com desfoco, ortoceratologia e terapia repetida com luz vermelha. A elongação axial foi tratada como o marcador estrutural principal; a progressão do equivalente esférico sob cicloplegia foi considerada de modo complementar. Menor idade no início do tratamento, velocidade prévia de progressão mais alta e persistência da elongação axial nas primeiras avaliações foram os sinais mais frequentemente associados à progressão residual. Comprimento axial e refração basais, miopia parental, resposta coroideana e adesão podem contribuir para a estimativa de risco, mas seu significado varia entre intervenções e populações. Não há um ponto de corte universal que defina falha terapêutica. A eventual intensificação do tratamento deve resultar da integração entre risco basal, trajetória individual, qualidade da adesão e evolução biométrica.

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Palavras-chave: Miopia; Comprimento Axial do Olho; Fatores de Risco; Resultado do Tratamento; Criança.

Resumen:
La progresión residual de la miopía durante el tratamiento es clínicamente importante porque expone a algunos niños a un mayor riesgo acumulado de miopía alta, pese a la eficacia media de las intervenciones disponibles. Esta revisión narrativa, apoyada en una búsqueda estructurada de la literatura, analiza los factores asociados a una respuesta insuficiente al control de la miopía en niños y adolescentes. Se priorizaron estudios originales de cohortes, análisis secundarios de ensayos clínicos y estudios de modelos pronósticos sobre atropina, lentes con desenfoque, ortoqueratología y terapia repetida con luz roja. La elongación axial se consideró el principal marcador estructural; la progresión del equivalente esférico bajo cicloplejía se evaluó de forma complementaria. La menor edad al iniciar el tratamiento, una mayor velocidad previa de progresión y la persistencia de elongación axial en las primeras evaluaciones fueron las señales más frecuentemente asociadas a progresión residual. La longitud axial y refracción basales, la miopía parental, la respuesta coroidea y la adherencia pueden contribuir a estimar el riesgo, pero su significado varía entre intervenciones y poblaciones. No existe un punto de corte universal que defina fracaso terapéutico. La eventual intensificación debe integrar riesgo basal, trayectoria individual, calidad de la adherencia y evolución biométrica.

Palabras clave: Miopía; Longitud axial del ojo; Factores de riesgo; Resultado del tratamiento; Niño.

Introduction

Myopia is a refractive error in which light focuses anterior to the retina, impairing distance vision. During childhood, its progression is predominantly related to anteroposterior growth of the eyeball, termed axial elongation. Axial length—the distance between the anterior and posterior portions of the eye—is therefore a key anatomical marker for monitoring myopia progression.

Beyond increasing dependence on optical correction, higher degrees of myopia increase the cumulative risk of myopic maculopathy, retinal detachment, glaucoma, and cataract. Epidemiological projections indicate that by 2050 nearly half the global population may have myopia and approximately 10% may have high myopia.[1] Reducing progression by 1 diopter, although seemingly modest, is associated with a clinically relevant reduction in lifetime risk of myopic maculopathy.[2]

Age at myopia onset modifies this trajectory. The earlier it develops, the more time is available for axial elongation before refractive stabilization. In a cohort followed for 12 years, the risk of high myopia in adulthood progressively decreased with later myopia onset.[3] This makes it particularly important to identify children whose progression is already rapid or who have a substantial familial predisposition.

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Low-concentration atropine, spectacle and contact lenses with defocus designs, orthokeratology, and repeated red-light therapy reduce average myopic progression. Group averages, however, do not answer the question encountered in clinical practice: which child will continue to show clinically relevant axial elongation during treatment? Children of the same age receiving the same therapeutic modality may have different trajectories, even when use appears adequate.[4]

In this context, “nonresponse” should not be understood as an absolute absence of benefit. The term describes residual progression substantial enough to prompt revision of follow-up or the therapeutic strategy. Thresholds vary because expected ocular growth depends on age, developmental stage, refraction, and observation period. The same annual elongation therefore has different implications in a young child and an adolescent.

Axial elongation is particularly useful here because it is less dependent on optical changes induced by some interventions. In orthokeratology, for example, corneal reshaping temporarily modifies refraction; isolated refractive change therefore does not necessarily reflect actual eye growth. Biometry and cycloplegic refraction should be interpreted together.

The literature has explored age, pretreatment progression rate, baseline axial length and refraction, parental myopia, choroidal parameters, lens variables, and adherence as potential predictors. There is consistent evidence of greater vulnerability in younger children receiving atropine, and early choroidal thickening was associated with less axial elongation in the LAMP cohort.[5,6] Other markers showed less consistent associations across therapeutic modalities.[7-12]

It is useful to distinguish a prognostic factor from an effect modifier. A prognostic factor identifies a higher likelihood of residual progression among treated patients. An effect modifier indicates that the magnitude of benefit from a particular intervention changes according to a patient characteristic and therefore requires comparison between treatments. Confusing these concepts may lead to selecting a specific therapy on the basis of a prognostic association that does not demonstrate comparative superiority.

This review discusses predictors of insufficient response to myopia control in children and adolescents, emphasizing axial elongation and refractive progression. It examines age, baseline axial length and refraction, prior progression rate, choroidal thickness, parental myopia, and adherence, as well as implications for monitoring and possible treatment escalation.

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Methods

This narrative review focuses on prognostic factors associated with continued clinically relevant myopic progression during treatment. The guiding question was: which clinical, biometric, and behavioral characteristics help identify children and adolescents more likely to maintain axial elongation or refractive worsening despite the intervention instituted?

The literature search prioritized MEDLINE/PubMed records updated through October 2026. Controlled descriptors and free-text terms related to myopia, atropine, orthokeratology, defocus lenses, repeated low-level red light, axial length, axial elongation, prognostic factors, treatment response, nonresponse, and treatment adherence were combined. Identification of relevant studies was supplemented by reviewing the references of key papers.

Priority was given to cohorts, secondary analyses of clinical trials, and prognostic-model studies involving myopic participants aged up to 18 years receiving an intervention to control progression. Adult studies, case reports, case series, research on prevention of myopia incidence, and secondary myopia were not included in the central synthesis. Consensus documents and reviews were used only to contextualize the topic.

The synthesis was organized by prognostic variable and therapeutic modality. Outcomes considered were axial elongation, change in cycloplegic spherical equivalent, and the definitions of insufficient response adopted.

In orthokeratology, temporary changes in corneal curvature may alter refraction without necessarily corresponding to less axial growth. The two outcomes were therefore interpreted as complementary.

No formal standardized risk-of-bias assessment or quantitative comparison between studies was performed. Findings are therefore presented interpretively, taking account of differences in populations, interventions, and follow-up duration.

Evidence synthesis and discussion

The key studies are presented in Table 1. Most prognostic evidence comes from retrospective cohorts, secondary analyses of clinical trials, and prediction models, particularly involving low-concentration atropine and orthokeratology. Myopia-control spectacle lenses have fewer studies specifically addressing determinants of individual response. The predominance of Asian populations and retrospective data limits direct extrapolation of thresholds and models to other clinical settings.[5-16]

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Table 1 – Key studies on predictors of response to myopia control.

Study	Design and sample	Intervention and follow-up	Main prognostic finding
Li et al., 2021 [5]	Secondary clinical-trial analysis; 350 children, 4–12 years	0.01%, 0.025%, or 0.05% atropine; 2 years	Younger age was associated with greater axial elongation and refractive worsening.
Yam et al., 2022 [6]	Double-blind clinical trial; 314 children with adequate choroidal data	0.01%, 0.025%, or 0.05% atropine; 2 years	Greater choroidal thickening was associated with less axial elongation and refractive progression.
Cho et al., 2022 [7]	Retrospective cohort; 109 children, 4–13 years	0.05% atropine; mean follow-up 25.4 months	Older age and initial axial shortening were associated with less axial growth.
Tang et al., 2025 [8]	Retrospective multicenter model; 1,183 children, externally tested in 405	Orthokeratology; outcome at 1 year	Age, spherical equivalent, axial length, pupil diameter, corneal asymmetry, and parental myopia; external AUC 0.817.
Wang et al., 2017 [9]	Retrospective cohort; 249 children	Orthokeratology; mean follow-up 2.5 years	Older age and greater baseline myopia were associated with less axial elongation.
Fu X et al., 2025 [10]	Retrospective model; 439 children (training 308; test 131)	Orthokeratology	Age, parental myopia, white-to-white distance, and spherical refraction formed the prediction; test AUC 0.820.
Najji et al., 2026 [11]	Retrospective longitudinal cohort; 723 children, 4–14 years	DIMS or HAL spectacles; 3 years	Faster pretreatment progression predicted a persistently progressive trajectory.
Fu A et al., 2021 [13]	Random-allocation study; 328 children	0.01% or 0.02% atropine; 1 year	Younger age, 0.01% atropine, shorter axial length, and smaller pupillary response were associated with rapid elongation.
Deng et al., 2025 [15]	Retrospective cohort; 300 children (542 eyes)	Orthokeratology	Younger age, less myopia, and high myopia in both parents were associated with greater elongation; regular follow-up and greater decentration with less elongation.
Chen et al., 2022 [16]	Retrospective cohort; 73 children with elongation ≥0.30 mm in the first year	Orthokeratology with or without 0.01% atropine; 3 years	Adding 0.01% atropine did not reduce cumulative axial elongation compared with orthokeratology alone.

Definitions of insufficient response are not uniform. In the model by Tang et al., axial elongation of at least 0.20 mm during the first year of orthokeratology defined insufficient response.[8] Fu et al. classified axial growth greater than 0.36 mm/year as rapid among users of 0.01% or 0.02% atropine.[13] Chen et al. selected children with elongation of at least 0.30

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mm during the first year of orthokeratology.[16] In the French DIMS and HAL spectacle cohort, identification was based on refractive trajectories rather than a single biometric threshold.[11]

The practical implication is straightforward: a single cutoff is insufficient. The same annual elongation should be interpreted according to age, baseline axial length and refraction, documented pretreatment progression, and the intervention used. “Nonresponse” therefore describes residual progression requiring reassessment rather than an absolute absence of effect.

Age and prior progression rate

Age at treatment initiation was the most frequently replicated finding. In the secondary LAMP analysis involving 350 children treated with 0.01%, 0.025%, or 0.05% atropine, younger age was the only predictor consistently associated with greater axial elongation and refractive worsening across groups.[5] In a cohort of 109 children treated with 0.05% atropine, older age and initial axial shortening were associated with less subsequent axial growth.[7] Fu et al. also identified a higher risk of rapid elongation in younger children, particularly with 0.01% atropine.[13]

These findings do not mean that young age represents treatment failure. Rather, it indicates greater expected residual ocular growth and therefore leaves less room to passively interpret continued progression. In young children, dose, regularity of use, and the rate of axial-length change warrant attention from the start of follow-up.

The progression rate documented before intervention adds information that age alone cannot provide. In the French cohort of 723 children using DIMS or HAL spectacles, faster refractive progression before prescription was the main marker of remaining in the persistent-progression group, whose progression was approximately −0.58 diopters/year; age and baseline myopic degree were not independently associated.[11]

Retrieving previous prescriptions and, where available, serial biometric measurements distinguishes children with the same current refraction but different risk trajectories. A young child progressing by −1.00 diopter/year before treatment should not be monitored in the same way as another child of similar age and refraction with slow progression.

Axial length, baseline severity, and early biometric response

Baseline axial length and refraction should be treated as part of the risk profile rather than isolated classifiers. Among children receiving atropine, shorter initial axial length was associated with rapid elongation in a cohort of 328 participants.[13] Among orthokeratology

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users, younger age and less baseline myopia were associated with greater elongation in some cohorts.[9,15] In contrast, a post hoc clinical-trial analysis found greater progression in younger children with more baseline myopia.[14]

This variable direction probably reflects differences in age, myopia duration, and intervention rather than invalidating biometric data. The most useful information is the trajectory: serial measurements obtained with the same method at comparable intervals help identify growth remaining above that expected for the child. In orthokeratology, cycloplegic refraction should be interpreted alongside axial length because corneal reshaping transiently changes refraction.

Among dynamic markers, choroidal response is of interest. In LAMP, a greater increase in subfoveal choroidal thickness during two years of atropine was associated with less axial elongation and refractive progression.[6] This finding is particularly useful for monitoring the initial response; evidence is insufficient to use it alone to select therapy before treatment begins.

Initial axial-length shortening described with 0.05% atropine was also related to less subsequent elongation.[7] It should be viewed as a possible early response signal rather than definitive anatomical reversal or a sole criterion for success.

Parental myopia, adherence, and orthokeratology-specific factors

Parental myopia appeared in both orthokeratology prognostic models analyzed but should not be understood solely as a genetic variable. In the multicenter model by Tang et al., it contributed, alongside age, baseline spherical equivalent, axial length, pupil diameter, and the corneal surface asymmetry index, to predicting elongation of at least 0.20 mm in the first year.[8] In another cohort, high myopia in both parents was associated with greater axial elongation.[15] Family history gains value when interpreted together with the child's trajectory and follow-up conditions.

Adherence is an indispensable clinical variable, although often measured imprecisely. In a European atropine cohort, reported adherence was not significantly associated with axial elongation.[12] This does not establish that it is irrelevant: irregular eye-drop use, insufficient lens wear, widely spaced visits, or centration problems can produce progression resembling pharmacological or optical failure. Before changing the intervention, it is necessary to verify that treatment exposure was sufficient.

In orthokeratology, some predictors relate to lens fitting itself. In the study by Deng et al., greater treatment-zone decentration, provided it caused no visual impairment or

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complications, and regular attendance at follow-up visits were associated with less axial elongation.[15] Because these variables depend on lens design, fitting, and follow-up, they cannot be transferred directly to atropine or spectacle lenses.

Prognostic models and clinical use

Multivariable models help combine variables that individually have limited value. The model by Tang et al. was derived from 1,183 children, divided into development (n=831) and internal validation (n=352), and externally tested in 405 children. Areas under the curve were 0.871, 0.863, and 0.817, respectively.[8] Although external performance is encouraging, the study involved a Chinese population and requires validation in other care systems.

In another orthokeratology model developed from 439 children, age, parental myopia, white-to-white distance, and spherical refraction contributed to predicting persistent elongation; areas under the curve were 0.831 in the training set and 0.820 in the test set.[10] Both studies demonstrate discrimination but do not prove that model use improves outcomes or replaces clinical assessment.

Routine incorporation of these tools requires prospective studies, external validation, and evidence that estimates usefully change management. Until then, they are better suited to supporting risk stratification than to serving as prescriptive algorithms.

Residual progression and treatment escalation

Residual progression does not mandate combination therapy. Chen et al. followed 73 children with axial elongation of at least 0.30 mm during the first year of orthokeratology. Over the next two years, 37 received 0.01% atropine in addition to the lens and 36 continued orthokeratology alone; after three years, cumulative elongation was similar between groups (0.91 ± 0.30 mm and 0.91 ± 0.24 mm, respectively).[16]

This finding does not invalidate combination treatment in every circumstance but shows that decisions should not depend solely on an axial-elongation threshold. Reviewing adherence, instillation technique, lens fitting and centration, biometric measurement reliability, and previous progression reduces the risk of attributing a failure with another cause to patient biology. Any escalation requires a clinical hypothesis consistent with the intervention used.

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Evidence limitations and clinical implications

The literature has important limitations. Definitions of insufficient response, follow-up duration, and therapeutic modalities are not uniform; furthermore, many studies are retrospective and vulnerable to confounding by indication. Children perceived as higher-risk may receive more intensive treatment, making it difficult to attribute their course exclusively to the dose or modality used.

The predominance of Asian cohorts also restricts automatic application of thresholds and models to Brazilian children. Age at myopia onset, school routines, outdoor exposure, screen-use patterns, access to care, and family adherence differ between settings and may modify predictor performance.

In clinical monitoring, a useful initial assessment includes age, cycloplegic refraction, axial length, previous progression records, family history, and actual adherence conditions. Follow-up should prioritize the individual axial-length trend rather than interpreting the current prescription in isolation. Younger children, those with rapid pretreatment progression, or those maintaining elongation disproportionate to the expected trajectory warrant targeted reassessment.

To reduce current uncertainty, prospective studies should use standardized axial-length measurements, explicit definitions of residual progression, more objective adherence measures, and external model validation. The clinical aim is not to label a child prematurely as a “nonresponder” but to identify promptly those whose course justifies adjustment of monitoring or the therapeutic strategy.

Final considerations

No single axial-elongation threshold defines insufficient response for every child receiving treatment. Younger age at therapy initiation, documented pretreatment progression rate, and persistent axial growth during early follow-up were the elements most consistently associated with residual progression. Baseline axial length and refraction, parental myopia, choroidal parameters, and adherence add information but should not be applied in isolation.

In clinical practice, decisions concerning residual progression should begin with serial comparable biometric measurements, adherence review, and confirmation that the intervention is appropriately applied. Combining treatments may be suitable in selected cases but is not justified solely by exceeding a fixed axial-elongation value.

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Broad applicability of current models is limited by heterogeneous definitions, predominance of retrospective studies, and concentration of data in Asian populations. Prospective, multicenter, ethnically diverse cohorts with external validation and objective adherence measures are needed before prediction models guide routine decisions.

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