# Atropine and Myopia Management: Current Evidence, Mechanisms, and Clinical Implications

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**Abstract**

Myopia, especially in children and young adults, has become a serious global public health issue. The increasing prevalence of high myopia and its associated ocular complications has prompted interest in pharmacological interventions aimed at slowing axial elongation. Of these, the non-selective muscarinic antagonist atropine has shown notable effectiveness in slowing the progression of myopia. This review summarizes current evidence on the use of low-dose atropine in myopia management, elucidates its proposed mechanisms of action, evaluates dosage-dependent outcomes, and discusses clinical implications for optometric practice. Low-dose atropine (0.01–0.05%) has been proven to be a safe and effective treatment option with few adverse effects by recent large-scale studies like the ATOM and LAMP trials. Despite ongoing debates regarding the optimal concentration, rebound effects, and long-term safety, atropine remains the most widely studied pharmacologic agent for myopia control. Understanding its clinical application, pharmacodynamics, and integration with optical interventions offers valuable insight for eye care practitioners striving to combat the global myopia epidemic.

**Keywords:**Myopia, Atropine for the Treatment of Myopia, accommodative stress, cycloplegia, mydriasis.

## Introduction

Myopia, or nearsightedness, has emerged as one of the most frequent vision issues globally. Its numbers have, in recent decades, soared alarmingly everywhere from East and Southeast Asia [1]. If current trends persist, close to half the world's population could be nearsighted by 2050. If there is a greater concern shared by the clinicians, it is the increasing number of children at risk of higher myopia, which may increase risks for potentially sight-threatening complications such as retinal detachment, glaucoma or myopic maculopathy [2]. In addition to the clinical burden, families are also financially stressed with lifelong dependence on spectacles/contact lenses and regular eye care.

Conventional optical interventions can correct blurry vision, but they do almost nothing to keep the eye from growing longer, which is the underlying determinant of myopia progression [2,3]. This space has led researchers and clinicians to look at other avenues. Among pharmacologic modalities, the antimuscarinic drug atropine, a relic of ancient medicine, has gained the most interest. Originally employed to dilate the pupil and paralyse accommodation, atropine has made a comeback as one of the first-performing pharmacological agents in

myopia control [4]. In this update, I incorporate the most recent evidence on what atropine is, what clinical studies tell us and where it fits into contemporary Optometric practice.

**Pathophysiology of Myopia Progression**

Myopia occurs primarily when the eye becomes too long, resulting in light focusing in front of the retina rather than directly on it. The causes of this lengthening are not due to one single factor, but rather a combination of genetics, environment and biochemical signaling. Children who partake of long periods of near work and those with low outdoor exposure are likely to develop progressive myopia [1].

Biologically, from communicating simple processes of the retina and choroid to even defining the complicated loop feedback systems that exist between these three structures. When these signals go awry, the sclera starts to re-arrange its design such that it would make the eye longer and easier. Neurotransmitters, such as dopamine, which help in controlling eye growth, also appear to be important. Decreased retinal dopamine levels have been correlated with longer axial length. Understanding these paths may also clarify how some drugs, like atropine, can slow the process [4].

**Pharmacology of Atropine**

Atropine acts by inhibiting the effects of the neurotransmitter acetylcholine on muscarinic receptors (M1–M5). In ophthalmology, its classical role has been to achieve cycloplegia and mydriasis [6]. What is equally interesting is that it seems not entirely to be about accommodation being slowed down. Rather, it is likely to affect growth signals within the retina and sclera. Studies indicate that atropine may have the potential to influence choroidal thickness, scleral remodelling, and biochemical cascades for ocular elongation [5,6].

When instilled topically, atropine is absorbed via the cornea and conjunctiva. They gain useful intraocular levels in low concentrations, with minimal systemic penetration, and are therefore safe for long-term use even in small children.

**Evidence from Clinical Trials**

Some of the best-known studies supporting atropine come from Singapore. The trial ATOM1 demonstrated that 1% atropine significantly retarded myopia progression, but was associated with unpleasant side effects such as photophobia and binocular near vision difficulties. To counter this, lower doses were trialled in the ATOM2 study. This time, 0.01% atropine was the most effective of them all, while the others had fewer side effects, too.

The LAMP trial from Hong Kong provided more weight to the low-dose atropine argument. For 0.05%, 0.025%, and 0.01%, a clear dose response emerged, with the strongest effect on both refractive error and axial length for 0.05% [6].

Low-dose atropine (0.01–0.05%) consistently slowed myopia progression on average by ~30–60% in various large population-based studies and meta-analyses from some developed countries [6]. These results have determined the current clinical practice and have impacted guidelines globally.

**Atropine Action Mechanisms in Controlling Myopia**

While researchers are still working to determine the exact mechanism, most scientists have come around to atropine's benefits not primarily through controlling accommodation. Instead, atropine seems to interact directly with ocular growth mechanisms [7]. It may change the muscarinic activity of retinal and scleral tissues, slow down the propulsive activity of scleral fibroblasts, and inhibit extracellular matrix changes, which are used to promote ocular growth [2].

Some studies even show that atropine boosts the release of dopamine, a naturally occurring inhibitor of eye growth. Others have cited changes in choroidal thickness that may alter biomechanical forces within the eye. In sum, these effects suggest that atropine impacts the underlying biological triggers of myopia development.

**Clinical Implications for Optometric Practice**

For any Whitby optometrist, atropine treatment is an invaluable aspect of childhood myopia management. Most doctors start with 0.01% to 0.05%, used once nightly, she said [6]. Kids ages 5 to 12 with early or progressing nearsightedness are likely to get the most benefit.

Follow-up every 3-6 months is required to monitor refraction, axial length and any side effect - usually mild photophobia or slight blur at near [8]. These are so far well corrected by photochromic lenses or suitable spectacle designs. Parents should also be advised on potential rebound following treatment cessation, in particular when using higher concentrations.

Atropine with orthokeratology or multifocal contact lenses is increasingly used with more efficacy than a single treatment alone [9].

**Limitations and Challenges**

Atropine treatment does, however, have certain disadvantages. Rebound can also develop, especially if higher doses are abruptly halted. All kids don't react the same way. Your ethnicity, your genes and your lifestyle may all affect how you do.

There is also a further practical problem of availability: in many regions, low-dose atropine cannot be obtained like other medications, which are commercially standardised drugs; the optometrist has to prepare it by an external compounding pharmacy [10]. Long-term safety data beyond 5 years are still developing, and compliance can be challenging for some families [11]. Better-coordinated long-term research will help to address these concerns.

**Future Perspectives**

Such approaches are coming to develop more selective therapies, like M3 antagonists that target specific receptor subtypes and have fewer side effects. Sustained-release forms such as inserts or nanoparticle-mediated delivery systems could further enhance ease of use and compliance.

The combination of atropine with optical interventions and digital-monitoring solutions could generate more individualized treatment strategies. Cross-disciplinary work between eye-care professions and research disciplines will be critical to advance this work.

**Conclusion**

Myopia treatment and atropine have made the management of myopia extremely different for clinicians. Low-dose formulations, especially 0.01% to 0.05%, have been shown to retard progression well with little associated side effects [12]. Challenges continue, however, including rebound effects and limited regulatory approval in some countries as well as the need for long-term data [13]. However, atropine still remains as one of the most reproducible drugs there is on the market nowadays. It is important for optometrists to closely follow the science behind its operation, evidence and practical application in achieving full care of children with progressive myopia.

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