



# Light, literacy and the absence of ultraviolet radiation in the development of myopia

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**Summary** As the prevalence of myopia steadily increases, reaching as high as 90% in some populations, investigators continue to look for causative factors other than family history. Most current research suggests an association of axial myopia with reading or either the presence or absence of light. Even though these studies are frequently inconsistent, non-reproducible or contradictory, many clinicians utilize them in recommending treatments for children, such as bifocals or atropine. By reviewing the biologic effects of non-ionizing electromagnetic fields, we may gain insight into these discrepancies as well as unify the combined role of literacy and light in the pathogenesis of myopia. These biologic effects are wavelength specific. The wavelength of artificial (either incandescent or fluorescent) light is primarily 700–400 nm, while the wavelength of natural light is 700–200 nm, inclusive of the ultraviolet spectrum. So the opposite findings of myopia resulting from either accommodation under continuous light or under darkness (form deprivation) can be reconciled by restating it:

*Close focusing in the absence of UV light may provoke axial myopia.*

Experimental evidence exhibiting both scleral remodeling under accommodation as well as the inhibition of scleral remodeling by the hardening of collagen under ultraviolet exposure may support this concept. Perhaps new research can look into the role of the presence or absence of UV light in animal models of myopia.

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Eight years ago, *Nature* published an article by Graham Quinn and his associates of the Department of Ophthalmology at the University of Pennsylvania [1] finding a strong association between night-lights and the development of myopia. This article generated an immediate backlash from several respected ophthalmic and optometric sources that ultimately resulted in the retraction of the original article 10 months later [2]. However, none of the

criticisms disproved the assumption, but rather maintained that the evidence did not support the conclusion.

Over the ensuing years, myopia's prevalence has steadily increased, and the search for possible explanations (in addition to a genetic predisposition) continues. And once again articles implicating continuous lighting as a cause are appearing in the literature [3]. I believe it is now time to revisit this topic.

Due to the morbidity of progressive myopia, efforts to understand its causes are ongoing. In

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addition to the possible roles of the presence or absence of light, an association of myopia with literacy has long been suspected [4].

The prevalence of myopia in literate societies is based on the assumption that in certain children and young adults the close focusing needed to read (either directly or indirectly involving accommodation) provokes axial lengthening of the eye, which results in myopia. However, some studies do not find this association [5]. Additionally, non-literate societies engage in close work with such tasks as weaving and tool making, yet no association of myopia with these activities has been found [6].

Support for the role of reading in the development of myopia has led to several modes of treatment in both the optometric and ophthalmic communities. These include the pharmacologic block of accommodation (cycloplegia) by antimuscarinic drugs such as atropine and pirenzepine in children [7]. Such treatments are not without morbidity and complications themselves. Those advocating atropine, which also causes mydriasis (pupillary dilatation), recommend its long-term use in adolescents. However, light itself is thought to cause many of the diseases of the aging eye [8], and what effect years of atropine treatment will have on this problem has not been addressed.

The role of light in the development of myopia continues to be a controversial topic with many studies finding seemingly opposite conclusions. While studies like the Quinn article suggest light as a causative agent, other studies and clinical experiences claim the opposite, that darkness may provoke myopia [9].

We can reconcile the seemingly contradictory role of light and dark in the pathogenesis of myopia by restating it as:

*Close focusing in the absence of UV light may provoke axial myopia.*

A review of the known effects of non-ionizing electromagnetic radiation (emr) may give us insight as well as unify the combined role of literacy and light in the pathogenesis of myopia. Non-ionizing emr includes all wavelengths longer than 200 nm, including frequencies in the visible and ultraviolet spectrums. Before addressing specific biologic effects, we must remember that emr fields are omnipresent, whether in the visible spectrum or not. Indeed, all life itself is dependent on the presence of emr fields, from photosynthesis to heat.

The biologic effects of emr are extensive. These effects are frequency specific, dose dependent and delivery sensitive. For example, leukemia has been associated with the presence of long wavelength electric power fields, but not shorter wavelength

radio waves [10]. Dose dependency is readily demonstrated by sunburn, and even if the wavelength and the dose are equal, the biologic effect can be different depending on whether the emr is pulsed or continuous [11].

If we look specifically at the effects of emr on the eye, we find many examples. UV light is implicated in many ophthalmic diseases [12], and visible light, especially blue, is seen as a causative factor in macular degeneration. Additionally, we cannot forget that vision itself is dependent on the visible light emr field.

We can now return to the contradictory biologic effects of emr on myopia. Many studies show that form deprivation (darkness) leads to axial myopia [13]. In contrast, we again find recent studies implicating continuous light as a cause of progressive myopia [14]. Some of these studies suggest it may not be the light alone that is necessary, but the act of accommodation during continuous light [15].

In summary, a review of the current literature shows that both darkness as well as continuous light can each provoke progressive axial myopia. But we can reconcile this contradiction by returning to the specific emr wavelengths encountered in light, which all have unique biologic effects. The wavelength of natural light is 700–200 nm, from red to violet and into ultraviolet. However, the wavelengths of incandescent and fluorescent lights are 700–400 nm with no UV frequencies in incandescent light and only very little UVA frequencies in fluorescent light. There is experimental evidence to support the claim that ultraviolet frequencies may prevent axial lengthening of the eye. Work by Bryant and McDonnell [16] shows scleral remodeling occurring with accommodation, and work by Wollensak et al. [17] shows that UVA hardens collagen and blocks scleral remodeling.

The discrepancies of the past studies – that is, the increased myopia in some, but not all literate societies, and the absence of myopia in accommodating non literate peoples – may be due to the type of light present. I believe the increase in myopia seen in the last 125 years may not be due the advent of literacy, but rather may due to the advent of artificial light. If so, Quinn et al could have collected accurate data but reached the wrong conclusion. Indeed, the Old Wives' Tale about damaging your eyes by reading in poor light may have a factual basis. And that basis may not be dim light, but UV free light. Perhaps the universally feared wavelength of 200–400 nm is harmful in its absence.

As a clinical pediatric ophthalmologist I continually hear parents of young myopic children ask, "What can we do to keep this from progressing?"

Perhaps researchers can look into the role of UV light on myopia models, and see if an answer lies there.

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