

Intralenticular Hydrostatic Pressure Increases During Ciliary Muscle Contraction: A Finding Consistent With the Schachar Mechanism of Accommodation

Chen et al.¹ measured ex vivo mouse intralenticular hydrostatic pressure following the topical administration of either pilocarpine 0.2% or tropicamide 0.1%. They observed that intralenticular hydrostatic pressure increased with pilocarpine and decreased with tropicamide. In addition, the lens diameter after pilocarpine ($n = 8$) increased 80 μm , 3.8%, to 2.17 ± 0.12 mm compared to the lens diameter of 2.09 ± 0.07 mm that occurred following tropicamide ($n = 6$). The minimal 3.8% increase in lens diameter was not, and would not be expected to be, statistically significant in view of the authors' small sample size.

Chen et al.¹ also noted that the circumlental space between the ciliary processes and the lens equator decreased with pilocarpine to 124 ± 14 μm and increased with tropicamide to 174 ± 7 μm . Moreover, as one can observe from their images reproduced below, the valleys between the ciliary processes deepened and the distance between the lens equator and the sclera decreased with pilocarpine compared to that obtained with tropicamide (Fig.).

The authors found that during pilocarpine-induced ciliary muscle contraction intralenticular hydrostatic pressure increased. The authors did not have an explanation for this increased intralenticular hydrostatic pressure. Increased tension on the equatorial zonules during pilo-

carpine induced ciliary body activation would be expected to increase intralenticular hydrostatic pressure. This is consistent with the Schachar mechanism of accommodation, which predicts an increase in stress on the lens capsule during ciliary muscle contraction as occurs during human accommodation.³

According to the Schachar mechanism, during accommodation the anterior and posterior radial muscle fibers assist the anterior and posterior longitudinal muscle fibers in pulling on the scleral spur and pars plana, respectively, whereas the circular muscle fibers isometrically contract. This results in notching, outward movement of the anterior ciliary muscle^{2,5-8} with deepening of the valleys between the ciliary processes causing an increase in equatorial zonular tension, which minimally increases equatorial lens diameter, decreases the distance between the lens equator and sclera, and, counterintuitively, minimally increases central lens thickness with an associated large increase in central lens optical power.^{9,10} The forward movement of the ciliary processes decreases the circumlental space.

In fact, all of the authors' findings are consistent with the Schachar mechanism of accommodation.² Following the administration of pilocarpine, there was < 5% increase in equatorial lens diameter,⁴ the valleys between the ciliary processes deepened, the circumlenticular space decreased, the distance between the lens equator and the sclera decreased, and intralenticular pressure increased from equatorial zonular tension.

In addition to explaining the pilocarpine and tropicamide induced changes in intralenticular hydrostatic pressure,

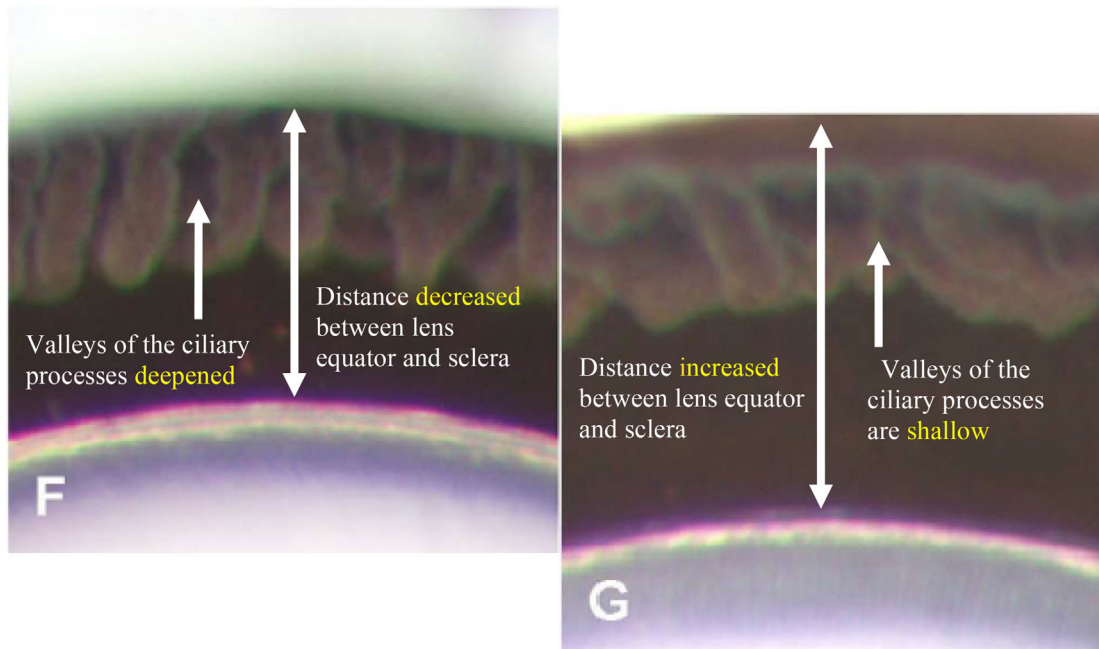


FIGURE. Reproduced from Chen et al.¹ Figs. 1F and 1G. Because the sclera does not significantly change during ciliary muscle contraction, Fig. 1G is shifted down to align the sclera of the two images. Annotations and arrows have been added. The valleys between the ciliary processes are *deeper* and the distance between lens equator and sclera is *decreased* following pilocarpine induced ciliary muscle contraction (F), compared to tropicamide induced ciliary muscle relaxation, where the valleys of the ciliary processes are *shallow* and the distance between the lens equator and sclera is *increased* (G).

ciliary body, and lens, the Schachar mechanism of accommodation elucidates the etiology for the reported lack of mouse accommodation. According to Schachar, vertebrates with lenses that have an aspect ratio (minor axis/major axis) > 0.6 have minimal accommodative amplitude.^{2,11} Therefore, the lack of accommodation in the mouse is **not** due to a ciliary muscle that is “smaller and less developed than the primate ciliary muscle,”¹ **but rather** related to the spherical shape of its lens.

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