Main topic:
ASPECTS OF MEDICAL THERAPY IN GLAUCOMA

Glaucoma Now is a continuing medical education publication. Distributed worldwide, our goal is to educate and update general ophthalmologists, glaucoma specialists and ophthalmology residents. International leaders in the field of glaucoma are invited to contribute to this journal, sharing their most recent insights.

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Introduction
Elevated intraocular pressure (IOP) remains an important, treatable risk factor for the development and/or worsening of glaucomatous optic neuropathy. Epidemiologic studies have revealed an exponential relationship between increased IOP and open-angle glaucoma. Interventional studies support the role of IOP reduction in order to decrease the risk of development and progression of the disease. Recent studies have shed light on other potentially modifiable risk factors for the development and/or worsening of open-angle glaucomas. These may include lower ocular perfusion pressure, heavier caffeine use, and obstructive sleep apnea. Although these novel, potentially treatable risk factors are now under consideration in the care of glaucoma patients, IOP-lowering remains the mainstay of therapy.

Core Concepts
- Intraocular pressure lowering is the mainstay of treatment for open-angle glaucomas.
- Novel, potentially modifiable risk factors for open-angle glaucoma include lower ocular perfusion pressure, heavier caffeine use, and obstructive sleep apnea.
- A ‘goal’ or ‘target’ intraocular pressure may be based on a percentage reduction from baseline, a fixed threshold level according to stage of disease, and/or a calculation based on other risk factors for progression.
- Target intraocular pressure must be continually reassessed and readjusted when glaucomatous progression has occurred.
- Prostaglandin analogs, beta-blockers, carbonic anhydrase inhibitors, and alpha-agonists are all efficacious medical therapies for the treatment of ocular hypertension and glaucoma.
- Important considerations when choosing an initial agent for glaucoma medical therapy include likely efficacy, side effects, cost, and dosing schedule.
- Recent data suggests that the intraocular pressure-lowering efficacy of prostaglandin analogs and carbonic anhydrase inhibitors persists during the nocturnal period.

Setting a Treatment Goal
Once the clinician decides that medical therapy is warranted, the next step is to determine an IOP goal for that particular patient. This goal may be a specific IOP or a range of IOPs at which the clinician estimates that further glaucomatous damage is unlikely to occur. This ‘goal’ or ‘target’ IOP should be based on the stage of glaucomatous disease as well as the IOP level at which damage has occurred. Determination of target IOP may be based on a goal percentage reduction from baseline, a predetermined fixed level, and/or a calculation. Various advantages and disadvantages to each of these approaches. Several major controlled trials have used percentage reduction and this method will be discussed in the current article. Although our discussion will highlight strong evidence-based reasoning to determine an IOP goal based on severity of disease (Table 1), this goal remains only an estimate for low likelihood of disease progression. The clinician continually must reassess this ‘target’ IOP and have a low threshold to adjust the value or range if glaucoma progresses. Furthermore, the clinical use of target IOPs should take into account factors besides an individual patient’s baseline IOP, disease severity, and non-modifiable risk factors: also consider life expectancy and the tolerance for risk of an indicated IOP-lowering therapy. "Normal Tension Glaucoma"
The Collaborative Normal-Tension Glaucoma Study (CNTGS) randomized one eye of 140 patients with normal tension glaucoma deemed to be “high risk” (based on prior glaucomatous progression, visual field defects threatening fixation, or appearance of a new optic disc hemorrhage) to IOP lowering treatment with a goal minimum IOP reduction of 30% or to observation. Lowering IOP by at least 30% in the treatment group reduced the risk of glaucomatous progression compared with the control group (20% versus 60% rate of progression; p=0.0034) after 5 years of follow-up. This performance was only censored for the effect of cataract. Based on the CNTGS, goal IOP lowering should be at least 30% in patients with “normal-tension glaucoma”.

Table 1. Recommended IOP lowering goals based on the results of major randomized, controlled clinical trials.

<table>
<thead>
<tr>
<th>Glaucoma Stage</th>
<th>Recommended IOP Lowering From Baseline</th>
<th>Relevant Clinical Trial</th>
<th>Clinical Trial Highlights</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocular Hypertension</td>
<td>20%</td>
<td>OHTS1</td>
<td>20% IOP lowering reduced the risk of glaucoma onset from 9.5% to 4.4%</td>
</tr>
<tr>
<td>Early/Mild Glaucoma</td>
<td>≥ 30%</td>
<td>EMGT1, CIGTS1</td>
<td>An average of 37% IOP lowering associated with no visual field loss after 5 years</td>
</tr>
<tr>
<td>Moderate/Severe Glaucoma</td>
<td>40% - 50%</td>
<td>AGIS1</td>
<td>Patients maintained at an IOP &lt; 18 mm Hg (mean 12.3 mm Hg) at all study visits experienced no significant field loss before 5 years</td>
</tr>
<tr>
<td>Normal Tension Glaucoma</td>
<td>30%</td>
<td>CNTGS1</td>
<td>IOP reduction of 25% associated with 50% decreased risk of progression</td>
</tr>
</tbody>
</table>

Abbreviations: OHTS, Ocular Hypertension Treatment Study; EMGT, Early Manifest Glaucoma Trial; CIGTS, Collaborative Initial Glaucoma Treatment Study; AGIS, Advanced Glaucoma Interventional Study; CNTGS, Collaborative Normal-Tension Glaucoma Study.
Analogs are most often employed as daily dosing regimen, prostaglandin IOP in open-angle glaucoma and ocular hypertension. Because of their superior IOP-lowering efficacy and minimal risk of systemic side effects, and once daily dosing regimen, prostaglandin analogs are recommended as the drug of choice for initial therapy. The availability of generic latanoprost has decreased costs of this agent which is likely to extend its use as a first-line agent. A challenge posed by generic prostaglandin analogs is variability in bottle size and design between manufacturers. In a comparative economic analysis, Qum and colleagues determined that the number of doses per bottle and annual cost varied significantly between four manufacturer versions of generic latanoprost. Although prostaglandin analogs have been found to have minimal systemic side effects, a host of ocular side effects may occur. These include conjunctival hyperemia, periorbital skin alterations and nephrolithiasis. The clinician must determine whether visual field progression has occurred. Confirmed glaucomatous progression, by either structural or functional measures, necessitates additional IOP lowering and ongoing close monitoring.

**Nocturnal Intraocular Pressure Control**

Sleep laboratory studies and investigations with a novel contact-less IOP sensor suggest that not all medially attended glaucoma patients have an increased nocturnal IOP from baseline levels. Results from these studies should be evaluated cautiously. A substantial number of patients with seemingly adequate office-measured IOPs continue to demonstrate glaucomatous progression.

### Conclusion

Current available medical therapy is not effective to lower IOP in patients with open-angle glaucoma and ocular hypertension. Based on a paradigm shift, innovative intraocular pressure-lowering agents and devices are needed. Many other agents, marketed and off-label, are being used for glaucoma management. The availability of prostaglandin analogs 32,33 and carbonic anhydrase inhibitors 34 have demonstrated nocturnal IOP-lowering effect. However, timolol 35 and brimonidine 36 do not appear to reduce nocturnal IOP from baseline levels. Results from these studies should be evaluated cautiously. A substantial number of patients with seemingly adequate office-measured IOPs continue to demonstrate glaucomatous progression.

### Monitoring Outcomes

### References

The goal in glaucoma therapeutics is to maintain functional vision and improve patient outcomes. Treating a chronic disease like glaucoma can be challenging for patients and physicians alike. Monotherapy doesn’t work for most patients so additivity and complementary mechanisms play key roles in glaucoma treatment decisions. Patients for whom adherence is difficult and those who become refractory to current classes of compounds are in the greatest need of new therapeutic alternatives, be they novel mechanisms of action or improved delivery options.

**Topical drops in the pipeline**

The trend in topical drop therapeutics is to have single daily dosing of combination compounds with multiple targets and mechanisms of action. Latanoprost bunod 0.024% (BOL-303259-X or LBN) is a nitric oxide (NO)-donating prostaglandin F2α agonist that is rapidly metabolized in situ to latanoprost acid and butanolenedimmonium, a NO-donating moiety. NO serves as a signaling molecule, using cyclic GMP as a second messenger, to initiate a series of events (e.g., inhibiting the Rho pathway perhaps via Rho kinase (ROCK) and a norepinephrine transporter (NET)). Inhibiting ROCK can enhance fluid outflow through the TM. This is the effector arm of the homeostatic mechanism; IOP, shear stress, cytokines, hormones are all stimuli, NO and perhaps adenosine are the mediators, the RK/MLCK pathways are further downstream, wound up with the cytoskeleton/cell adhesion/contactility changes that actually generate the physical changes that alter outflow resistance – the effector arm of the TM. NO seems to induce the production of aqueous humor. A third possible mechanism of action is based on rabbits where ROCK inhibition was shown to reduce episceral venous pressure (EVP). If it is hypothesized that a similar effect in EVP occurs in humans, based on subsets of normotensive subjects who attained post-treatment IOP reduction of 20% or more, and which appears to involve uveoscleral outflow. A trend in topical drop therapeutics is to have single daily dose combination of netarsudil and latanoprost. The addition of latanoprost, which increases uveoscleral outflow, adds another mechanism of action. In a Phase II study, the fixed-dose combination of AR-13324 0.02% and latanoprost 0.005% in PG24 Ophthalmic Solution provided superior IOP lowering relative to its individual active components at the same concentrations.1

**Aqueous adenosine levels** are positively correlated with IOP in ocular hypertensive individuals and adenosine could possibly serve as an endogenous modulator of IOP.2

In monkeys IOP is decreased and outflow facility increased following topical application of adenosine A1 receptor agonist.3 Investigators hypothesize that stimulating the A1 adenosine receptor in the TM results in an increase in an increase or upregulation of proteases, such as Protease A or MMP-2, that digest and remove accumulated proteins, which helps to open the conventional outflow pathway.4

**Core Concepts**

- The trend in topical drug therapeutics is combination compounds with multiple targets and mechanisms of action, with single daily dosing.
- New combination molecules target both, the trabecular and uveoscleral outflow pathways, aqueous humor formation and possibly episcleral venous pressure (EVP).
- Nitric oxide (NO) acts as a signaling molecule to regulate outflow facility via the trabecular meshwork (TM) by modulating (TM) contractility, cell adhesion and the cytoskeleton. An NO donating moiety can be attached to a latanoprost backbone to provide a dual action molecule that enhances both uveoscleral (latanoprost) and conventional outflow (NO).5
- Stimulating the A1 adenosine receptor in the (TM) results in an increase in an increase or upregulation of proteases, such as Protease A or MMP-2, that digest and remove accumulated proteins, which helps to open the conventional outflow pathway.
- Agonist-sensitive EP1, EP2, and EP4, but not EP3, receptors are present in TM cells, while all four EP receptor subtypes are active in Schlemm’s canal (SC) cells. EP2/4 receptors decrease cell stiffness in SC, but not in TM cells. Both receptors may mediate IOP lowering via changes in SC cell stiffness in the conventional outflow pathway.
- Drug delivery methods in development aim for steady, controlled stable delivery over periods of months with injectable or patient inserted devices.
- Alternative therapies that would offer long term IOP lowering while removing the patient from the drug offer long term IOP lowering while not replacing the patient resident. Alternative therapies that would offer long term IOP lowering while not replacing the patient resident.

**What’s New**

**What’s New in Medical Therapies:**

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**Advances in Drug delivery**

To have a truly global effect on preserving vision, longer duration of action therapeutic options are needed that take into account differences in availability of resources and disparities in access to ophthalmologists. Economically developed countries there can be one ophthalmologist for every 10,000 people; in India, this drops to approximately one for every 400,000 people; in Africa, one or less for every million people. Long-term therapies that do not require multiple visits to an ophthalmologist would be of ben-
A novel, intracamerally injected product is ENV515, a biodegradable proprietary PRINT™ nanoparticle with an extended-release formulation of travoprost. In a phase II trial a single dose of ENV515 achieved its primary efficacy endpoint, a change from baseline in diurnal IOP at Day 25, with comparable efficacy to once-daily Travaprost 0.04%. 18 Early studies indicate the formulation may have the potential to lower IOP for more than 6 months from a single dose. 21 The platform could also be used to deliver therapies for other ocular diseases.

An earlier stage product that would allow patient self-administration and also has potential for longer-term delivery of a variety of therapeutics is the Topical Ophthalmic Drug Delivery Device -TODDD™, a soft, flexible device that floats on the tear film completely concealed under the eyelid. It is easily inserted or replaced by clinic staff or the patients themselves. The delivered drug is polymerized into a customized matrix material or loaded into one or more multiple polymerized drug depots. The device has a large capacity and may be capable of delivering various drugs simultaneously and at different rates, over several months.

Early animal testing and preliminary human trials indicate safety and efficacy. 19 Another device in early development is the iDose - an intracanalular implant that is injected through a clear corneal incision and secured in the anterior chamber. The iDose is designed to continuously elute therapeutic levels of travoprost for extended periods of time. Interestingly, such novel delivery systems are not currently in play for latanoprost, the oldest of the widely-used PG analogues.

Alternative therapies that would offer longer term IOP lowering while removing the patient from the drug delivery system include gene therapy and stem cell strategies, and a variety of formulations and devices.

Clinical decision-making will continue to be complex, and likely with an increase in specificity for the individual patient: their type of glaucoma; underlying mechanisms; genetic make-up; co-morbid conditions; patient characteristics and need for progression. Advances in medical therapy and drug delivery methods hold much promise for both patients and physicians.

References
other studies have reported lower rates of non-persistence. Some studies have reported worse visual outcomes (greater intracocular pressure fluctuations and visual field defects) among non-adherent participants, although others have failed to demonstrate that interventions which increase adherence improve visual outcomes.

Physicians seem to be poor judges of which patients fail to adhere. Younger age and African-American race repeatedly have been shown to be risk factors for non-adherence. Chang et al. reported a scoring system for estimating the probability of non-adherence which included age and race in addition to worse self-reported general health, shorter duration since commencement of glaucoma medication, lower self-reported adherence over the previous month and being less likely to follow doctor’s orders exactly (Figure 1). Other studies have also reported higher initial medication copayment costs, increased number of eye care visits, lower annual income and not using mail-order prescription delivery systems as factors associated with lower adherence. Very few studies have reported long-term patterns of glaucoma medication adherence; the most recent study found that adherence patterns in the first year were indicative of adherence over the previous month and being less likely to follow doctor’s orders exactly (Figure 1). Other studies have also reported higher initial medication copayment costs, increased number of eye care visits, lower annual income and not using mail-order prescription delivery systems as factors associated with lower adherence. Very few studies have reported long-term patterns of glaucoma medication adherence; the most recent study found that adherence patterns in the first year were indicative of adherence over the previous month and being less likely to follow doctor’s orders exactly (Figure 1).

Broadly, interventions for increasing adherence to glaucoma medications fall into two main categories, those that use educational and individualized care plan interventions and those that simplify glaucoma medication regimens. While topical administration remains the standard, sustained release of topical glaucoma medications via contact lenses, nanoparticles, bioadhesive matrix polymers and ocular implants are all under investigation. One study reported almost three-quarters of judges of which patients fail to adhere. Older age and African-American race repeatedly have been shown to be risk factors for non-adherence. Chang et al. reported a scoring system for estimating the probability of non-adherence which included age and race in addition to worse self-reported general health, shorter duration since commencement of glaucoma medication, lower self-reported adherence over the previous month and being less likely to follow doctor’s orders exactly (Figure 1). Other studies have also reported higher initial medication copayment costs, increased number of eye care visits, lower annual income and not using mail-order prescription delivery systems as factors associated with lower adherence. Very few studies have reported long-term patterns of glaucoma medication adherence; the most recent study found that adherence patterns in the first year were indicative of adherence over the previous month and being less likely to follow doctor’s orders exactly (Figure 1).

Newman-Cassey et al. created a conceptual framework of glaucoma medication adherence based on Health Behaviour Theory (which is recommended by the US National Institute of Health as the basis for all new educational interventions). The authors then performed a systematic review of literature surrounding educational interventions, and found that only two out of eight reviewed studies formally defined how the studied educational intervention applied Health Behaviour Theory. Five out of the eight reviewed studies reported statistically significant improvement in adherence after educational interventions, all of these focused on improving patient knowledge about glaucoma and the majority included a personalized format of education for participants. As the authors noted, other chronic disease literature shows a diminishing effect of educational interventions over time; thus the intervention should include strategies for sustainability, although this has not been studied in glaucoma educational interventions. Given the burden of implementing individualized educational interventions for adherence, as well as the possibility of the need for repeated interventions to maintain the effect, educational interventions may have practical drawbacks that limit their use.

Overall, the busy clinician should be aware of the risk factors associated with poor adherence, such as younger age and non-white race. Formal scoring systems for estimating probability of non-adherence can also be used. Educational interventions and simple medication regimens may sense in theory, but there are no compelling studies of these interventions for glaucoma adherence to support their use. Automated reminder systems are less resource intensive and may improve adherence by more than 10%, and EMR systems are well-positioned to allow for streamlined implementation of such reminder systems. Although plausible, studies have yet to show that improved adherence to glaucoma medications leads to improved visual outcomes.

References


Figure 1. Scoring system for estimating the probability of nonadherence to once daily prostaglandin therapy, taken from Chang et al. [1].

Figure 2. Screen capture of automated reminder system used to improve glaucoma medication adherence, taken from Boland et al. [7].
Practical Tips: How to Provide the Best Possible Care for Patients with Glaucoma: Make it Personal!
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There are two overarching principles that apply to caring for patients with glaucoma:
1) Always direct attention towards improving, restoring, or maintaining the overall health (function plus quality of life) of each person as an individual.
2) Always personalize care.

There is no other generally appropriate guideline. Specifically, there is NO diagnostic procedure of choice for glaucoma, NO drug of choice for glaucoma, NO surgical procedure of choice for glaucoma. But, there is a diagnostic procedure of choice, a drug of choice and a surgical procedure of choice for each individual person.

Regarding principle number 1, be aware of substituting surrogates for the relevant outcome. Lowering intraocular pressure (IOP) and preserving visual field are not the relevant outcomes. They are only some of the mechanisms that a good physician uses to try to achieve the real goal, maximizing health.

Regarding principle number 2, personalization, five things have to be known for each person:

a) The mechanism of the glaucoma (gonioscopy answers this)
b) The stage of the glaucoma (ophthalmoscopy answers this)
c) The rate of change of the stage (unbiased evaluation of serial, valid data points answers this)
d) The duration the glaucomatous process will continue (usually the same as Estimated Remaining Years (ERY))
e) The ability of the patient to care for himself/herself

To provide the best possible care, the physician knows how to gonioscope, do so before reviewing the previous examination. Knowing the number of functional retinal ganglion cells would probably be the best method, but that is not possible currently. In this regard, when obtaining data and drawing conclusions are being made.

a) Mechanism
There is no substitute for gonioscopy at the slit lamp; the assessment of the anterior chamber angle with instruments other than the gonioscope is expensive, time-consuming, and almost always unnecessary if the examining physician knows how to gonioscope properly. Anterior Segment OCT is not an adequate substitute, as it does not allow understanding the CHARACTER of the angle: the color, location and amount of pigmentation, the presence of a Sampaio/Eline, the appearance of peripheral anterior synechiae, the presence of a microphlema, etc.

b) Stage
Get a valid estimate of the stage of the person’s glaucoma. The best indicator in this regard is the nature of the optic disc, not the visual field or the IOP. The thickness of the retinal nerve fiber layer is not sufficient. Cup/disc ratios are a rough approximation, but are unsatisfactory. Because they do not take into account the position of the cup or the size of the disc they do not always relate validly to the results of field examination. Knowing the number of functional retinal ganglion cells would probably be the best method, but that is not possible currently.

The Disc Damage Likelihood Scale (DDLS) is an easy and accurate way to estimate the amount of damage. There is no other generally appropriate substitute, as it does not allow understanding the CHARACTER of the angle: the color, location and amount of pigmentation, the presence of a Sampaio/Eline, the appearance of peripheral anterior synechiae, the presence of a microphlema, etc.

Establishing staging, rate of change and duration is facilitated by using the Glaucoma Graph (Figure 1).

c) Rate of Change
Patients want to know and doctors need to know what will likely be happening in the future. Establishing this requires valid data points. It is essential to develop a method of establishing as best as possible what is going to happen in the future. Obtaining valid data points regarding glaucoma is difficult and requires great skill, minimization of bias, and willingness to ignore what may be misinformation. Invalid entries on the record are worse than having no entry, as wrong information may lead to wrong decisions. It is far better to say, “I do not know, and we need better information,” then it is to give advice based on faulty information.

The practical points are: Demand excellent performance from technicians and time consuming. The more data points the more false-positive interpretations will occur, leading to incorrect treatment. As trends are more likely to be accurate when data points are averaged, obtain two or three good data points and use the average of them as the basis for comparison. This system works better than obtaining four to six separate data points. However, always from when the disease starts until the person living with the disease dies. Having a reasonable idea of how long the person will live is essential for care to be rational and appropriately personal. This is how Estimated Remaining Years (ERY) comes in.

Two practical points: 1) It is possible with a reasonable level of accuracy to predict the likelihood of a person living five to ten more years, and 2) It is not always possible to predict accu-


the basis of the age of the person.

A second important consideration relates to general health, which is a Gestalt impression that can be usually given accurately by the patient; asking about the person’s level of energy is a good clue in this regard. A third criterion relates to the person’s weight, a fourth to whether they smoke cigarettes, a fifth to the age at which family members died, and a sixth to the person’s age. The number of ERY can be estimated, using the information in Table 1. Recall, current age is not a good indication of estimated remaining years (ERY).

To estimate a trend validly, not only must the data points be valid, but the system must also be stable. This is often not the case in a person with a chronic disease. For example, the ERY is always merely an estimate; this needs to be remembered by both doctor and patient. The ERY is similar to estimating a target intracranial pressure. The specific target pressure needs to be adjusted constantly in terms of the additional information that comes from each follow-up. Likewise, an ERY could be 10 when first calculated, but at the next visit needs to be changed to 2 because of the development of a major illness. Nevertheless, an estimate of the duration the glaucoma is expected to remain active is essential in making a decision regarding the need for treatment. The scheme suggested above has not been validated, and is based on personal experience. Other validated methods have been suggested: the Charlson index and the Elixhauser score, for example, are two of the most commonly used comorbidity measures, and predict long-term mortality for any individual by assigning weights to medical conditions based on severity. Both measures have calculators readily available for users. As most patients in whom progression of glaucoma will be examined at least once every six months, it is easy to modify the ERY at each visit using any of the methods outlined above. Each visit will allow increasing accuracy with the estimate. Finally, new treatments may become available or current treatments may stop working. System instability is the rule. This makes it ESSENTIAL to revisit the original baseline data as the foundation for deciding on the nature of any change that may have occurred.

II. Ability to care for one’s self

It is not difficult to get a good idea of how well people can care for themselves. Providing appropriate person-tailored care requires having a good understanding of how well or poorly the patient can care for himself or herself. It is unrealistic to expect patients who are biologically, psychologically or emotionally unable to comprehend exam findings or to articulate concerns, to adhere to a treatment plan, or to continue to follow instructions well, or to report changes accurately.

In summary:

1) Always direct attention towards restoring, maintaining or improving the overall health of each individual person.

2) Avoid generalizing from a group to an individual; since each person is unique, direct treatment not towards the form of glaucoma, but the individual with glaucoma; strive to personalize diagnosis and care at all times.

References


STATEMENT OF NEED AND PROGRAM DESCRIPTION

Recent months and years have seen significant advances in our understanding of glaucoma. Much has been learned, not only about damage mechanisms and pathogenesis, but also about diagnosis and management. Treatment options – both medical and surgical – continue to expand. This program will review this new knowledge with an emphasis on incorporating recent insights into day-to-day practice.

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Participants have an implied responsibility to use newly acquired information to enhance patient outcomes and professional development.

The information presented in this activity is not meant to serve as a guideline for patient care.

Any procedures, medications, or other courses of diagnosis or treatment discussed or suggested in this activity should not be used by clinicians without evaluation of their patient’s conditions and possible contraindications or dangers in use, applicable manufacturer’s product information, and comparison with recommendations of other authorities.

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Paul L. Kaufman, MD, PhD is Peter A. Duehr Professor of Ocular Pharmacology at the Departments of Ophthalmology & Visual Sciences and Animal Health & Biomedical Sciences at the University of Wisconsin, Madison, Wisconsin, USA. He has acted as a consultant to Advanced Genetic Technology Corp., Alcon, Allergan, Bausch & Lomb, Amakem Therapeutics, Refocus Group Inc, Suncap Pharma and Valeant Pharmaceuticals. He received grants from Lens AR Inc, WARF, Z Lens LLC, Vista Ocular and ReGen Eye. He has received honoraria from Advanced Genetic Technology Corp., Alcon, Allergan, Bausch & Lomb, Amakem Therapeutics, Refocus Group Inc, Suncap Pharma and Valeant Pharmaceuticals. He also receives royalties from WARF and has obtained travel support from Alcon. Carol A. Rasmussen, MS is his associate researcher. She has acted as a consultant within the last 3 years to Ocular Services on Demand.

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