analyzed, but their breakdown of ages totals 63—and the lack of a detailed description of Figure 1.

As to the research itself, the authors used an infrared eye-tracking system with an accuracy of no better than 0.5°, and that varies with gaze angle. Yet, they reported a single mean amplitude of nystagmus of 0.22° across all gaze angles. In any case, a measuring system should be at least twice as sensitive as the effect that one intends to measure. Consequently, one can have no confidence in any reported nystagmus with amplitude of 1° or less. In addition, the authors provide no information about the parameters of the targets that the subjects fixated.

With regard to data analysis, reporting of means and standard deviations implies that the data are normally distributed. The reported mean amplitude of nystagmus is an absolute value, and a negative amplitude has no meaning. Consequently, the SD 0.33° gives a 2-SD range (that should include approximately 95% of the sample) of +0.88° to −0.44°. Thus, the data are not normally distributed.

Finally, and most important, is the issue of the authors’ conclusion that their findings somehow relate to the HGN test conducted by police officers. The authors state that they did not visually observe the nystagmus that was being recorded. Yet the HGN test is one of visual observation. Even the most experienced clinician under ideal conditions can recognize an eye movement of no less than approximately 2 prism diopters, or approximately 1°. The only conclusion that the authors can make is that they measured small-amplitude physiologic nystagmus using a non–visually observed method. This method has nothing to do with the HGN test.

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Citation: Invest Ophtalmol Vis Sci. 2010;51:6900-6901. doi:10.1167/iovs.10-5889

Author Response: GEN Is Not HGN

We are sorry that Dr. Citek is “deeply troubled” by the peer review of our paper.1 The review was conducted by two reviewers, both of whom took time to evaluate four drafts of the paper before the final acceptance.

The title of Dr. Citek’s letter “GEN Is Not HGN” suggests that his difficulties with our paper on physiologic gaze-evoked nystagmus had more to do with his involvement with the HGN test than with our paper itself. There is no physiological difference between horizontal gaze-evoked nystagmus and horizontal gaze nystagmus.

The erroneous number of subjects per decade appeared in the online prepublication version of the article and was corrected in the print version.

This study was performed specifically to evaluate the incidence of horizontal gaze nystagmus in normal subjects of different ages. We found that there was clear recordable nystagmus at smaller gaze angles, with amplitudes well within the recording limits of our system in a significant number of normal subjects. This finding suggested to the reviewers that a more detailed look at physiological gaze-evoked nystagmus at these angles would be important in judging the limitations of the HGN test. We concurred.

Dr. Citek comments that we made no reference to any “accepted peer-reviewed literature that supports the horizontal gaze nystagmus test.” We also did not reference any of the ample literature that documents the high false-positive rate of the test. Had we done either, the reviewers, we suspect, would have rightfully noted that these references should be removed, as they don’t have anything to do with the study itself.

We agree that the nystagmus amplitudes quoted in the paper are small compared with those in other studies. As noted in the paper, the method used to calculate the amplitude would result in smaller values than other methods. The program written to identify the beats of nystagmus looked at velocities of the fast and slow phases. The algorithm clipped the extremes of the movements, to get the best estimate of the velocities of both the slow phases and the quick phases. We did not write an additional program to best determine the true amplitudes of nystagmus beats, as it was not critical to the original purpose of the project.

Clearly, this article does not prove that the HGN test is not valuable. As Dr. Citek noted, we did not evaluate the incidence of visually detectable nystagmus at different gaze angles. Our findings suggest that such a study would be worthwhile.

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Citation: Invest Ophtalmol Vis Sci. 2010;51:6901. doi:10.1167/iovs.10-6537

Eu-estrogenemia and Retinal Blood Flow

Deschênes et al.1 reported remarkable news in their study, “Postmenopausal Hormone Therapy Increases Retinal Blood Flow and Protects the Retinal Nerve Fiber Layer,” which appeared in the May 2010 issue of the journal. For those women who are using hormone replacement therapy (HRT) for specific indications, such as vasomotor instability, this serendipitous benefit can provide additional reassurance that the holistic effect of HRT improves the quality of life for many women.
Three to four decades ago, the chronic hypoestrogenemia of menopause was considered a pathologic state. Our sophisticated evidence-based clinical trials and basic science evaluation of estrogen receptor function are giving us an amazing understanding of the role of eu-estrogenemia.1,2

A large number of menopausal women are following the recommendation of ‘lowest dose for the shortest duration,’ to treat vasomotor instability and urogenital atrophy. Also, millions of women abruptly stopped HRT in 2002 on the recommendations of Jacques Rossouw, the head of Women’s Health Initiative (WHI) at the National Institutes of Health (NIH). We have named this epidemiologic phenomenon, Rossouw’s cohort.3 As clinical gynecologists and fellow ophthalmologists, we have another three decades in which we will be observing our patients’ health, well-being, and quality of life. We warily consider what suboptimal retinal blood flow will mean for our patients who are chronically hypoestrogenemic.

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Citation: Invest Ophthalmol Vis Sci. 2010;51:6901–6902. doi:10.1167/iovs.10-5923

Author Response: Eu-estrogenemia and Retinal Blood Flow

We thank Drs. Turner and Kerber for their supportive letter in the importance of the findings of our study published in the May issue of the journal, “Postmenopausal Hormone Therapy Increases Retinal Blood Flow and Protects the Retinal Nerve Fiber Layer.”1 This study was designed to bring to light the significant beneficial role that estrogens play by increasing retinal blood flow and protecting the retinal nerve fiber layer in women. There is a large body of evidence indicating that estrogens have beneficial vasomotor effects in several vascular beds, and there is a growing body of evidence that they have protective and trophic effects on the neurons in the brain. However, our understanding of the role of estrogens in the retina and optic nerve is limited, and we are just seeing the tip of the iceberg.

We are pleased that Drs. Turner and Kerber have called gynecologists’ and ophthalmologists’ attention to the visual function of women who are chronically hypoestrogenemic. Particularly, we believe that this hypoestrogenemic state could be important in women who experience early menopause onset, premature ovarian failure caused by chemotherapy and radiotherapy, genetic disorders, and hypopituitarism, and in women undergoing aromatase inhibitor or selective estrogen receptor modulator therapy used for treating or preventing the recurrence of breast cancer.3 Women who are chronically hypoestrogenemic may be susceptible to impaired ocular blood flow, which is hypothesized to be a contributing factor in the etiology and progression of age-related macular degeneration4 and glaucomas5 and to the thinning of the retinal nerve fiber layer, which is a clinical feature of glaucoma.

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Problems with Monotherapy for Bacterial Keratitis

I read with interest the article by Kaye et al.1 in the January issue, “Bacterial Susceptibility to Topical Antimicrobials and Clinical Outcome in Bacterial Keratitis.” The authors found a fairly high rate of failure of monotherapy for bacterial keratitis with either ciprofloxacin or ofloxacin (5%), particularly due to resistance from Streptococcus pneumoniae and other Streptococcus species. Based on my October 1998 publication in the journal,2 I was not surprised. What did surprise me is that the authors did not add topical fortified gentamicin when faced with a clinical failure due to Streptococcus sp. My experimental data and my anecdotal clinical experience suggest that this treatment would have been beneficial.

The authors are to be commended, however, for showing the lack of correlation between the MICs (minimum inhibitory concentrations) of fluoroquinolones and the clinical response of streptococcal keratitis. This finding is important because the newer fluoroquinolones (e.g., moxifloxacin, gatifloxacin) have been said to have improved efficacy in the Gram-positive spec-