

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.^{2,3}

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.⁴ 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.

Ralph J. Turner¹
Iruin J. Kerber²

¹Department of Surgery, University of Texas Health Science Center, Tyler, Texas; and ²Department of Obstetrics and Gynecology, University of Texas Southwestern Medical School, Dallas, Texas.

E-mail: rjturnertx@aol.com

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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 hypopitu-

itarism,² 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 glaucoma⁵ and to the thinning of the retinal nerve fiber layer, which is a clinical feature of glaucoma.

Micheline C. Deschênes^{1,2}
Denise Descovich²
Michèle Moreau³
George A. Kuchel⁴
Elvire Vaucher⁵
Mark R. Lesk^{1,2}

Departments of ¹Ophthalmology and ³Family Medicine, Faculty of Medicine, and ⁵School of Optometry, University of Montréal, Montréal, Québec, Canada; the ²Maisonneuve-Rosemont Hospital Research Center, Montréal, Québec, Canada; and the ⁴UConn Center on Aging, University of Connecticut Health Center, Farmington, Connecticut.
E-mail: lesk@videotron.ca

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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 (9%), 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-