Re: Economides et al.: Capturing the moment of fusion loss in intermittent exotropia (Ophthalmology. 2017;124;496-504)

TO THE EDITOR: Based on the eye tracker recording data presented, I can agree with the conclusion on page 503 in the discussion section that states “once the ocular axes separate, visual feedback has no impact on the subsequent outward movement of the deviating eye to its final position.” However, the abstract conclusion implies something different, stating that the process of fusion loss itself in patients with intermittent exotropia is not influenced by visual feedback. Semantics perhaps, but as a clinician who has examined 20 or 30 of these patients weekly for the past 17 years, how else to explain the consistently reported symptom of unilateral lid squinting in bright, outdoor sunlight, almost universally accepted by clinicians as natural history, a powerful, nonrandom dissociation to the tropic phase inducing blur or diplopia, and hence the observed compensatory lid closure?

ALEX CHRISTOFF, BS, CO
The Wilmer Eye Institute at Johns Hopkins Hospital, Baltimore, Maryland

Financial Disclosures: The author has no proprietary or commercial interest in any materials discussed in this article.

Available online: November 30, 2017.

Correspondence:
Alex Christoff, BS, CO, The Wilmer Eye Institute at Johns Hopkins Hospital, Baltimore, MD 21286. E-mail: achr15@jhmi.edu.

Reference


Reply: In the abstract conclusion, we are referring to the eye movement occurring after loss of fusion. Of course, maintenance of fusion in intermittent exotropia relies on visual feedback.

JOHN R. ECONOMIDES, PHD
DANIEL L. ADAMS, PHD
JONATHAN C. HORTON, MD, PHD

1Department of Ophthalmology, University of California, San Francisco, San Francisco, California; 2Center for Mind/Brain Sciences, The University of Trento, Trento, Italy

Financial Disclosures: The authors made the following disclosures: J.C.H.: Supported by grants EY10217, EY02162 (Beckman Vision Center) from the National Eye Institute and a Physician-Scientist Award from Research to Prevent Blindness.

Available online: November 30, 2017.

Correspondence:
Jonathan C. Horton, MD, PhD, Beckman Vision Center, University of California, San Francisco, 10 Koret Way, San Francisco, CA 94143-0730. E-mail: hortonj@vision.ucsf.edu.

Re: Keel et al.: The prevalence of diabetic retinopathy in Australian adults with self-reported diabetes: The National Eye Health Survey (Ophthalmology. 2017;124;977-984)

TO THE EDITOR: We congratulate Keel et al1 for their interesting paper on estimating the prevalence of diabetic retinopathy (DR) in non-Indigenous Australians and Indigenous Australians with self-reported diabetes. Collectively, these findings will be greatly useful in the future planning of healthcare resource allocation.

We are intrigued by a few interesting areas stated in this study. First, the authors revealed that the odds ratio (OR) for age was 0.97 (95% confidence interval [CI], 0.94–0.99) for any DR among non-Indigenous Australian participants. There is still a debate regarding age as a risk factor in this aspect. Similarly, the Singapore Malay Eye Study reported that older age (OR, 0.73; 95% CI, 0.57–0.93) was protective of any DR.2 However, another cross-sectional survey in Gegharkunik, Armenia, found that older age (OR, 1.05; 95% CI, 1.02–1.08) was an independent risk factor for DR.3 Could this be owing to differences in population characteristics and even study methodology? What could be the plausible reasons to explain this observation? We would greatly appreciate if the authors could further comment on this.

Second, in this article, higher educational attainment was found to be associated with vision-threatening DR in non-Indigenous Australian participants. We are wondering whether this finding could be potentially confounded by income status that was not accounted for in the model presented by the authors. If income information is available in this study, perhaps the authors can further evaluate this aspect?

In Table 4, among indigenous participants, it was observed that longer duration of diabetes was a significant risk factor for any DR (OR, 1.69 per year). In addition, the interaction term of age x duration of diabetes was also significantly associated with any DR but with the effect estimate pointing at the protective direction (OR, 0.99; P = 0.005). In view of the conflicting directions, how should this be interpreted? We would greatly appreciate the authors’ clarification on this.

Last, as acknowledged by the authors in the Discussion section, other relevant confounders and indicator of diabetic control (i.e., hemoglobin A1c, body mass index) were not accounted for in this study. Therefore, we humbly opine that the current risk factor-related findings should be evaluated with caution.