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Letter to the Editor

Comments to paper entitled: Predicting scleral GP lens entrapped tear layer oxygen tensions



Dear Editor,

We have read with interest the article authored by Jaynes, J.M.; Edrington, T.B.; Weissman, B.A. recently published in Cont. Lens Anterior Eye, (2015; 38: 44–47) entitled **"Predicting scleral GP lens entrapped tear layer oxygen tensions**" [1].

We found the topic very interesting. However, we think that the conclusion of the authors is not fully supported by their results. The authors alert to the fact that the cornea would suffer hypoxia for any combination under the best scenario they evaluated theoretically (300 microns lens with 140 barrer Dk and 50 microns corneal clearance). The authors indirectly infer that the cornea would undergo physiological effects. Similar results had already been found by Michaud et al. [2] and hypothesized that 250 microns lens with a Dk higher than 150 barrer and up to 200 microns of corneal clearance should be used to minimize corneal edema. However, none of these two studies provided clinical measures of edema.

Having been working in this topic for the last 4 years we admit that this is at a first glance a logical thought. Indeed, when we first tried to publish our results back in 2011, reviewers raised the question whether such hypothesis would be verified considering the apparent absence of hypoxia in the clinical setting. Then we started a clinical pilot study to demonstrate the relationship between post-lens tear film thickness and physiological stress.

Meanwhile, Michaud et al. [2] published their results, who already anticipated the results shown in the present study [1]. Both used constant oxygen consumption for the whole cornea which does not allow to estimate the accurate concentration of oxygen at different levels within the eye. The readers could find these estimations in our previous work [3]. Looking for a direct comparison between the three studies we can report the partial pressure of oxygen for 300–350 lens thickness (Dk = 100 barrer) and post-lens tear film of 300–350 microns. Michaud's results would provide about 15 mmHg while Compan's would predict about 23 mmHg. Extrapolated data from Jaynes results would render about 24 mmHg very close to our previous predictions.

The outcomes from our clinical pilot study confirmed that edema (as an indirect sign of hypoxia stress) would indeed be inversely related with the post-lens tear film. Our clinical data showed that edema would raise from 2.5 to 5% which might be considered clinically acceptable. Several mechanisms that are beyond the scope of this letter could result in a lower edema response than that expected from the theoretically predicted distribution of oxygen through the cornea.

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