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LETTER TO THE EDITOR

Changes of extracellular matrix of the cornea in *diabetes mellitus*

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Dear Editor,

We have read the interesting article by Hager et al. [1] entitled "Changes of extracellular matrix of the cornea in diabetes mellitus" with great interest. The authors studied the corneal biomechanical changes in diabetes mellitus. They included a group of 484 eyes, including 99 eyes of diabetic patients. They found that corneal hysteresis (CH) was significantly higher (0.55 mmHg) in diabetic eyes than in healthy eyes. On the other hand, they showed that CH decreased with age (−0.036 mmHg/year, $p<0.01$).

There are very limited data regarding the biomechanical corneal changes in diabetes [1–3]. Diabetes is a multisystem disorder that affects various organ systems as well as eyes. Glucose can act as a collagen cross-linking agent as a result of Maillard reaction end-products. Similar to diabetes, aging causes cross-linking in the cornea and results in lower CH values. However, Hager et al. [1] found the contrary, and assumed that glycation of corneal proteoglycans contributed to an increase in CH. In contrast to their study, we found significantly lower CH values in diabetics than healthy subjects in our recently published study [2]. We believe that lower CH in diabetics may be explained by an alteration in the collagenous components due to collagen cross-linking. The lower CH in diabetics suggested that the dampening effects of the cornea decreased due to diabetes, and were induced to increase during the cross-linking of collagen fibrils.

Moreover, CRF deserves particular attention for this group of patients. We did not find any data in their study with regard to the differences in terms of corneal resistance factor (CRF) values between groups. Although the exact physiological properties of CH and CRF are still unclear, CH is thought to be an indicator of corneal viscous dampening, and CRF is thought to be a correction factor to reduce the effect of CCT on IOP measurement. In our study, CRF showed no significant difference to that of controls [2].

Diabetes was shown to have a protective effect against glaucoma. It might be hypothesized that glucose-mediated corneal collagen cross-linking may have been responsible for the IOP overestimation, and if we consider the possibility that the true IOP in these patients is statistically lower, then it is possible that the threshold for significant pressure-dependent damage to the optic nerve might have not been reached. If the impact of this altered corneal properties in diabetic patients is extrapolated with regard to IOP measurement, the results of these large epidemiological studies could certainly be looked at from a different perspective. From this point of view, we believe that identifying the impact of structural changes in diabetic corneas to IOP measurement errors is of utmost importance.

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