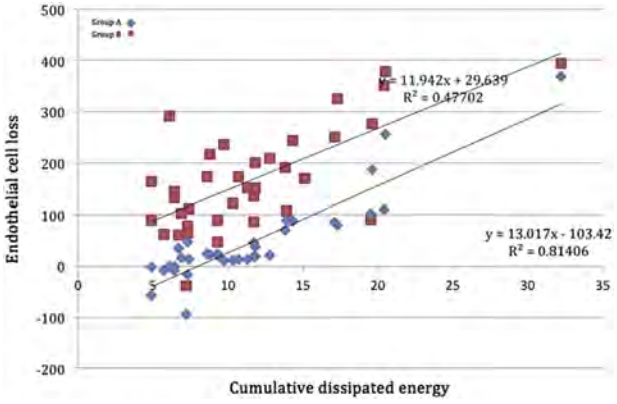

Figure 1. Correlation between CDE and endothelial cell loss.

postoperative corneal sensitivity; however, no significant changes were detected in the intergroup comparisons. (4) Group B had an 8.2% reduction in ECC, significantly higher than in the rest of the groups (Group A1: 4.8%; Group A2: 5.0%). (5) Endothelial cell loss showed linear correlation with CDE (Group B: $r^2 = 0.47$; Group A2: $r^2 = 0.81$; both $P < .01$) (Figure 1). (6) Group A2 patients had significantly worse scores on the subjective discomfort index.

The clinical significance of our study is that in energy-demanding cataracts, the combination of dispersive and cohesive OVDs seems to protect the endothelial cells more efficiently than the cohesive-only OVD (Visthesia). That is, for the same CDE, more endothelial cells were lost in the cohesive-only OVD patients. Moreover, the endothelial loss pattern in these patients was more random than the in the dispersive–cohesive OVD (Duovisc) patients. However, although statistically significant, the increased endothelial loss in the cohesive-only OVD patients did not interfere with postoperative visual acuity or corneal thickness. On the other hand, the subjective discomfort feeling was significantly more pronounced in the dispersive–cohesive OVD patients; therefore additional intracameral anesthesia is considered to be necessary. The intracameral anesthesia we used in our study provided anesthesia comparable to that of the cohesive-only OVD, with no evident impact on endothelial cells.

In summary, we consider that Visthesia and Duovisc offer comparable surgical outcomes in stage 3 cataract extraction surgery using torsional intelligent phaco technology. Despite the fact that the international literature provides conflicting comparative outcomes regarding the efficacy of the several commercially available OVDs,[1,3–6] our results suggest that Duovisc provides an additional significant protective effect on endothelial cells that should be taken into consideration, especially in cases with a low preoperative ECC.

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Effect of cigarette smoking on intraocular pressure

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Elevated intraocular pressure (IOP) is a major risk factor for glaucoma. Recently, corneal biomechanical properties have gained interest. There is conflicting epidemiological data on whether tobacco smoking affects IOP. Whereas some studies have found no association between smoking and IOP, others reported a relationship.[1] Interpretation of data is difficult because most studies categorized subjects as never smokers, ex-smokers, or current smokers. This categorization does not elucidate the chronic long-term habits of smokers; that is, the number of pack years. In contrast, clinical studies[2] have shown that cigarette smoking increases IOP. However, these studies exclusively examined the short-term effects after cigarette-smoke inhalation and nicotine uptake. Here, changes in the ophthalmic artery blood flow after nicotine administration and vasoconstriction of episcleral veins might lead to an inhibition of aqueous outflow from the trabecular meshwork.

We performed a study evaluating the effect of chronic smoking on corneal biomechanics using a dynamic bidirectional applanation device (Ocular Response Analyzer, Reichert Technologies). The study was a comparative age-matched case series including 117 eyes. Inclusion criteria for smokers were current smoking and a smoking history of at least 10 pack

Table 1. Differences in ocular response analyzer readings between nonsmokers and smokers.

Parameter	Mean (mm Hg) ± SD		*P* Value
	Nonsmokers (n = 66)	Smokers (n = 61)	
Goldmann-correlated IOP	16.3 ± 3.6	18.7 ± 3.6	.003*
Cornea-compensated IOP	16.2 ± 3.4	18.1 ± 3.9	.002*
Corneal resistance factor	11.1 ± 1.8	11.8 ± 1.5	.006*

IOP = intraocular pressure
*Statistically significant ($P < .05$, Student *t* test)

years. Only healthy subjects not taking systemic medication were included in the study. Nonsmokers were defined as those who had never smoked. Chronic smokers showed significant increases in the corneal resistance factor (CRF) and corneal hysteresis (CH) values. This increase in corneal stiffness might originate from compounds found in cigarette smoke that enhance corneal biomechanics; for example, formaldehyde crosslinks the cornea and experimentally increases the tissue's resistance to collagenases.[3,4]

The dynamic bidirectional applanation device also measures IOP, and it compensates the measured IOP (Goldmann-correlated IOP) for corneal thickness (corneal-compensated IOP). To determine a potential relationship between chronic smoking and IOP, we continued and extended our initial study (66 eyes of nonsmokers and 61 age-matched eyes of chronic smokers). Patient ages ranged from 20 to 71 years. The nonsmokers had a mean age of 45.8 years and a median age of 46.2 years. Smokers had a mean age of 44.9 years and a median age 43.9 years. Patients were recruited at the Institute for Refractive and Ophthalmic Surgery, Zurich, Switzerland. Institutional review board approval was obtained from the Ethical Committee of the Canton of Zurich.

Univariable and multivariable analyses were performed for factors related to IOP and to adjust for potential correlations. Because measurements in both eyes of the same subject are likely to correlate, generalized estimating equations with robust standard errors (Huber-White sandwich variance estimator) were used to account for the fact that both eyes of an individual were included in the analysis. Data analysis was performed using the Student *t* test.

In smokers, there were statistically significant increases and distinct increases (>2 mm Hg) in not only the Goldmann-correlated IOP but also in the corneal-compensated IOP ($P = .0003$ and $P = .002$, respectively) (Table 1). The CRF showed an increase similar to the one observed previously.[5] When adjusting for age, smoking status, CH, CRF, and corneal thickness, smoking had a statistically significant correlation with the Goldmann-correlated IOP ($R^2 = 1.925$, $P = .034$) and corneal-compensated IOP ($R^2 = 1.655$, $P = .034$). This finding might indicate that the increase in IOP in smokers is independent of the previously reported increase in biomechanical resistance.[5] One possible mechanism is an elevation in choroidal thickness caused by chronic smoking, which in return can cause an elevation in the episcleral venous pressure and IOP.[6,7]

We found a higher mean IOP in chronic long-term smokers than in nonsmokers. This increase seems to be independent of corneal biomechanical properties and of variations in central corneal thickness (CCT) because the corneal-compensated IOP has been repeatedly shown to independent of the CCT.[8] In light of an estimated 1.2 billion smokers worldwide in 2010, more extensive studies are needed to further investigate this relationship.

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Incidence of cystoid macular edema: Femtosecond laser–assisted cataract surgery versus manual cataract surgery

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One of the reported benefits of femtosecond laser–assisted cataract surgery has been the significant decrease in effective phacoemulsification time and cumulative dissipated energy that can be achieved by