Published in final edited form as:

Cornea. 2017 March ; 36(3): 367–371. doi:10.1097/ICO.0000000000001107.

Age-Stratified Analysis of Diabetes and Pseudophakia On Corneal Endothelial Cell Density: A Retrospective Eye Bank Study

Yicheng Chen, M.D.¹, Sean W. Tsao, M.D.¹, Moonseong Heo, Ph.D.², Patrick K. Gore, R.N.³, Mitchell D. McCarthy, Ph.D.³, Roy S. Chuck, M.D., Ph.D.¹, and Prabjot Channa, M.D.¹

¹Albert Einstein College of Medicine/Montefiore Medical Center Department of Ophthalmology and Visual Sciences. 3rd Floor, 3332 Rochambeau Ave, Bronx, New York 10467

²Department of Epidemiology and Population Health, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461

³Lions Eye Institute for Transplant and Research, Tampa, FL

Abstract

Purpose—To perform an age-stratified analysis of the effect of diabetes and pseudophakia on the corneal endothelial cell density.

Methods—A comparative analysis of donor characteristics from data supplied by the Lions Eye Institute for Transplant and Research on tissue harvested from July 1st, 2007 through May 23, 2014. The mixed-effects model was used to compare age-adjusted mean corneal endothelial cell density (ECD) between donors with and without diabetes.

Results—20,026 non-diabetic donor eyes and 13,617 diabetic donor eyes were included in this study. The ECD was 2,604 cells/mm² in non-diabetic corneas and 2,576 cells/mm² in diabetic corneas ($p < 0.001$). Among phakic patients, diabetic ECD was significantly less in the middle-age subgroups: -33 cells/mm² in the 21 to 40 year old subgroup ($p = 0.048$) and -25 cells/mm² in the 41 to 60 year old subgroup ($p = 0.009$). Among pseudophakic patients, diabetic ECD was significantly less only in the subgroup 61 years or older: -56 cells/mm² ($p = 0.026$). The magnitude of difference in ECD between phakic and pseudophakic donors was greater in diabetic patients in the subgroup 61 years or older ($p < 0.001$).

Conclusion—Donor eyes with a history of diabetes had a slightly lower ECD (-29 cells/mm²) than eyes without a history of diabetes. While this statistical relationship is consistent with our pathophysiologic understanding diabetes and the corneal endothelium, such a minor difference in ECD would be expected to have minimal clinical impact on overall corneal endothelial function.

Corresponding Author: Sean Tsao M.D. Mailing Address: 3rd Floor, 3332 Rochambeau Ave, Bronx, New York 10467. Office: 718-920-6665 sean.tsao@gmail.com. Requests for Reprints: Prabjot Channa M.D. Mailing Address: 3rd Floor, 3332 Rochambeau Ave, Bronx, New York 10467. Office: 718-920-6665 pchanna@montefiore.org.

This abstract was presented at the AAO Annual Conference 2016 in Chicago, IL.

Conflict of Interest: No conflicting relationships exist for any author.

Keywords

Diabetes; Pseudophakia; Corneal Endothelium

Introduction

Diabetes mellitus has long been known to have an effect on the function and morphology of the corneal endothelium.^{1,2} Numerous direct clinical observations of the diabetic cornea show variations in the endothelial cell microarchitecture. However, there is still no consensus on the effect of diabetes on the corneal endothelial cell density (ECD). Several reports show a decreased ECD in diabetic patients³⁻¹³, while some studies show no difference¹⁴⁻¹⁶ or even increased ECD.¹⁷

Furthermore, the effect of cataract surgery on the diabetic corneal endothelium is inconclusive, with some reports demonstrating increased vulnerability to surgery¹⁸⁻²¹, with others showing no difference.^{22,23}

While the majority of these investigations are single-center observational studies, none have yet reported on the data collected through an eye bank. To investigate the effect of diabetes and pseudophakia on ECD, we queried the large dataset available through the eye bank at the Lions Eye Institute in Tampa, Florida.

Methods

Our study protocol was deemed exempt by the Albert Einstein College of Medicine Institutional Review Board. Cornea donor data and donor tissue characteristics of all consecutive eyes collected from July 1, 2007 to May 23, 2014 were reported from the Lions Eye Institute in Tampa, Florida.

Information regarding the cornea donor, including age, gender, race, medical history, medication list, and cause of death were obtained from medical records, healthcare provider interviews, and family members. Diabetic status was determined by matching terms for diabetes in the medical history and querying for anti-hyperglycemic medications in the medication list. Further medical details, such as distinction between type 1 versus type 2 diabetes, duration of disease, hemoglobin A1C, presence and severity of concomitant diabetic retinopathy were not recorded as part of routine medical records collection by the eye bank and were thus unavailable for analysis.

The ECD was obtained using a specular microscope (Konan EB-10; Konan, Hyogo, Japan). The image quality was graded using a five-step rubric which defined endothelial morphology and image quality. A final ECD reading was obtained after technicians manually identified three to five micrographs with a minimum of 100 cell centers. Eye bank technicians were trained and certified in-house with periodic examinations to maintain standardization of readings between individuals. Phakic status was ascertained by technician at time of harvest. Data regarding pachymetry, endothelial cell polymorphism, polymegathism were not included in this database for analysis.

Both suitable and unsuitable donor tissue were included in the analysis for ECD as this study sought to address the overall effect of diabetes on the cornea. Corneas that were deemed unsuitable were either destroyed or designated for research. The three most commonly reported categories for tissue unsuitability were compared between diabetic and non-diabetic donors.

These categories were:

1. Positive serology for communicable disease (e.g. syphilis, HIV, Hepatitis C, etc.)
2. Abnormality with the corneal endothelium (e.g. ECD less than 1700 cells/mm² or other endothelial abnormality)
3. Other corneal abnormality excluding endothelial findings (e.g. stromal scar, infiltrate, foreign body, etc.)

SAS v9.3 (Cary, NC) was used for all statistical analyses. A Mann Whitney U test was used to compare age between diabetic and non-diabetic groups. For the outcome analysis, the unit of analysis was eye as opposed to individual. To take into account potential eye to fellow eye level correlations within individuals, we applied a mixed-effects linear regression model to test significance of diabetic status on ECD while adjusting for age. Specifically, subject-specific intercepts were considered random whereas all the other factors were considered fixed. The same mixed effects modeling approach was also used to test whether there was a significant interaction between diabetes and lens status on ECD. All reported p-values are two sided and statistical significance was declared if a p-value was less than 0.05.

Results

Baseline Characteristics

Among the 33,643 donor eyes reported by the eye bank, we had 20,026 (59.6%) non-diabetic eyes and 13,617 (40.4%) diabetic eyes. (Table 1) Our study population was predominantly male in both groups, 13,076 (65.2%) and 8,641 (63.4%) in the non-diabetic and diabetic groups respectively. There were 3,035 (9.0%) pseudophakic eyes, of which 2,560 eyes (84.3%) were from the group aged 61 years or older. There were insufficient pseudophakic eyes under age 41 years for meaningful analysis (n=5). The overall mean age of non-diabetic donors (54.8±0.1 years) was significantly younger than diabetic donors (59.2±0.1 years, $p < 0.001$). After stratifying by age groups, the mean age of non-diabetic donors was only younger in the middle aged subset (Age 41 to 60 years, $p < 0.001$).

The proportion of corneas deemed unsuitable for transplantation was higher in the diabetic group than the non-diabetic group (39.7% vs 37.5%, $p < 0.001$). Of the corneas unsuitable for transplantation, the diabetic group had a higher proportion in all three of the most frequently reported reasons for unsuitability: 1) positive serology for communicable disease (23.4% vs 20.2%, $p < 0.001$), 2) abnormality with the corneal endothelium (18.9% v 15.9%, $p < 0.001$) and 3) other corneal abnormality excluding endothelial findings (14.5% v 8.2%, $p < 0.001$).

Endothelial Cell Density

The age-adjusted mean ECD was $2,604 \pm 4$ cells/mm² for non-diabetic donor corneas and $2,576 \pm 5$ cells/mm² for diabetic donors corneas ($p < 0.001$). (Table 2) After stratifying for age, there was no difference in the age-adjusted mean ECD in the group 20 years or younger ($p = 0.874$). However, age-adjusted mean ECD remained significantly less in diabetic corneas in the age group between 41 to 60 years (-34 cells/mm², $p < 0.001$) and 61 years or older (-30 cells/mm², $p < 0.001$).

Amongst phakic donors, diabetic ECD was lower in the middle aged subgroups, between 21 to 40 years (-33 cells/mm², $p = 0.048$) and between 41 to 60 years (-25 cells/mm², $p = 0.009$). (Table 3) There was no difference in ECD for phakic corneas from the subset aged 61 years or older ($p = 0.547$).

Amongst pseudophakic donors, diabetic ECD was lower in the oldest subgroup, age 61 years or older (-56 cells/mm², $p = 0.026$). Diabetic ECD did not differ significantly in the younger group of pseudophakic eyes aged between 41 to 60 years ($p = 0.498$).

Comparison of Endothelial Cell Loss in Pseudophakia

For both diabetic and non-diabetic donors of all age groups, pseudophakic eyes had significantly lower age-adjusted mean ECD than phakic eyes (Supplemental Table 1). The magnitude of difference between phakic and pseudophakic eyes was greater in diabetic donors (309 ± 11 cells/mm²), compared to non-diabetic donors (251 ± 12 cells/mm², $p < 0.001$). When stratified by age, this interaction remained significant in the age group 61 years or older ($p < 0.001$), but not in the younger age group between 41 to 60 years ($p = 0.361$). (Supplemental Table 2)

Discussion

Our eye bank based study shows that donor corneas with a history of diabetes have fewer corneal endothelial cells than non-diabetic donors. The statistical relationship strengthened in older age subgroups from 41 years or above. Phakic diabetic corneas were most strongly affected in the middle aged subgroup between 41 to 60 years, while pseudophakic corneas were most affected in the age group 61 years or older. Our observations corroborate numerous other investigations that report a decreased ECD in diabetic eyes.³⁻¹² These observations are also consistent with animal based biochemical studies implicating the deleterious effect of the activated polyol pathway²⁴ and oxidative stress²⁵ on the corneal endothelium.

In a large population based study from rural India⁷, the age-adjusted mean ECD was 66 cells/mm² less in type 2 diabetics than in non-diabetics, which contrasts to our 28 cells/mm² difference for our entire population. Their study included only phakic type 2 diabetics, the majority of which (69.5%) fell between 40 to 60 years of age while our study included considerably more heterogeneity in age, diabetic and phakic status.

Interestingly, diabetes did not exert an effect on the age-adjusted mean ECD in the phakic subgroup analysis aged 61 years or more. Factors and patterns leading to donor expiration

could explain this effect. While the mean age between diabetics and non-diabetics was similar in the subgroup aged 61 years or more, the mean age of diabetics in the subgroup between 41 to 60 years was significantly younger than similarly aged non-diabetics. (Table 1) This points to a fundamental difference between the two subgroups: that the diabetics in the younger age group were expiring at a younger age, while the diabetics in the older age group were better able to survive their disease into later years. This may reflect differences in medical history (e.g. type 1 versus type 2 diabetes), disease severity or socioeconomic status which have a significant impact on mortality.²⁶

We acknowledge that there may be a significant portion of undiagnosed diabetes in our control group, since nearly 30% of diabetes mellitus remains undiagnosed in the American population.²⁷ Collection of data on hemoglobin A1C and serologic screening of donors is not included in the standard protocol for tissue harvest at the Lions Eye Institute and thus is unavailable for our study. Presumably, patients with undiagnosed diabetes would have milder or early manifestations of disease and would thus have an ECD similar to that of a non-diabetic individual. If these individuals with undiagnosed diabetes were moved to the diabetic group, it is possible that the difference in ECD between groups will be further diluted. However, our eye bank data is representative of the information available to the corneal surgeon, and would still remain clinically applicable in setting the surgeon's expectation of ECD in diabetic patients.

Our proportion of diabetic donors (40%) is higher than that reported in the literature. The Cornea Donor Study and Cornea Preservation Time Study had a lower number of diabetic donors (18% and 27% respectively) because both prospective studies followed grafts that were suitable for transplantation and thus was biased to exclude corneas unsuitable for transplantation.^{28,29} The question in our study is fundamentally different from that in the CDS and CPTS, as we chose to include all corneas, even those unsuitable for transplantation, in order to study the overall effect of diabetes on ECD.

Many studies have shown that duration and severity of diabetes has an adverse effect on the ECD.^{4,5,8-11,15,17} Furthermore, type 1 diabetics are thought to experience accelerated loss of the ECD compared to type 2 diabetics.^{13,16} Unfortunately, our study was not equipped to validate these observations as these details of the medical history are not gathered as part of the eye bank protocol. This data may become increasingly relevant to eye banks as our understanding of the effect of diabetes on the cornea continues to evolve.

Our data suggests that the corneal endothelium suffers greater attrition after cataract surgery in diabetics when compared to non-diabetics of a similar age. This is consistent with other reports that suggest increased vulnerability of the corneal endothelium to cataract surgery.¹⁹⁻²¹ While this relationship was significant in the oldest age subgroup 61 years or older, it did not differ significantly in the middle-aged subgroup between 41 to 60 years old. This effect could reflect a resiliency to surgical stress enjoyed by a younger corneal endothelium, or alternatively, reflect an increased susceptibility to damage in older diabetic patients. We do recommend restraint when interpreting this finding, as neither preoperative ECD nor intraoperative phacoemulsification settings available for our study - both variables have been known to the outcome of postoperative ECD.²³

Our database shows that diabetic donors have a higher proportion of tissue that are graded unsuitable for transplantation than non-diabetic tissue. This finding is not entirely surprising as presence of diabetes mellitus may serve as a surrogate marker of poor overall health status, leading to an unfavorable grade for transplantation. However, we recommend a cautious interpretation of this result because our database does not fully encompass all of the variables necessary to judge suitability for tissue transplantation (e.g. pachymetry, pleomorphism, polymegathism, etc.) Furthermore, the most frequently reported reason for unsuitability (positive serology for communicable disease) has no known association with diabetic pathophysiology. Our study expands upon the previously known relationship between diabetes and ECD - the broader question of whether diabetes has an impact on graft suitability lies beyond the scope of our investigation.

The impact of the donor diabetic status on corneal transplantation is probably minimal. Two separate studies arising from the Cornea Donor Study showed that neither a history of diabetes in the donor nor recipient increased the graft failure rate for penetrating keratoplasty.^{28,30} A recent study looking at both Descemet's stripping automated endothelial keratoplasty and penetrating keratoplasty showed no difference in graft failure when using tissue from donors with a history of diabetes.³¹ However, two longitudinal studies on penetrating keratoplasty did find higher risk of graft failure in recipients with a history of diabetes.^{32,33} Our study suggests that diabetic donor have fewer corneal endothelial cells, but this may not necessarily correlate to a higher risk of transplant failure.

In summary, we show that donors with a history of diabetes have an age-adjusted ECD that is 28 cells/mm² fewer than donors without diabetes. This difference rose to 56 cells/mm² when comparing pseudophakic donors with and without diabetes aged 61 years or older, suggesting increased vulnerability to cataract surgery. We emphasize that, while statistically significant and consistent with our pathophysiologic understanding of diabetic changes in the corneal endothelium, these overall differences in ECD are small and probably do not have an impact on overall corneal endothelial function.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The study was supported in part by the NIH/NCATS award to Einstein-Montefiore CTSA grant KL2 RR025749 and KL2 TR000088.

References

1. Lass JH, Spurney RV, Dutt RM, et al. A morphologic and fluorophotometric analysis of the corneal endothelium in type I diabetes mellitus and cystic fibrosis. *Am J Ophthalmol.* 1985; 100:783–788. [PubMed: 4073174]
2. Janiec S, Rzendkowski M, Bolek S. The relation between corneal autofluorescence, endothelial cell count and severity of the diabetic retinopathy. *Int Ophthalmol.* 1994; 18:205–209. [PubMed: 7797383]
3. Roszkowska AM, Tringali CG, Colosi P, et al. Corneal endothelium evaluation in type I and type II diabetes mellitus. *Ophthalmologica.* 1999; 213:258–261. [PubMed: 10420110]

4. Inoue K, Kato S, Inoue Y, et al. The corneal endothelium and thickness in type II diabetes mellitus. *Jpn J Ophthalmol.* 2002; 46:65–69. [PubMed: 11853716]
5. Lee JS, Oum BS, Choi HY, et al. Differences in corneal thickness and corneal endothelium related to duration in diabetes. *Eye (Lond).* 2006; 20:315–318. [PubMed: 15832184]
6. Choo M, Prakash K, Samsudin A, et al. Corneal changes in type II diabetes mellitus in Malaysia. *Int J Ophthalmol.* 2010; 3:234–236. [PubMed: 22553561]
7. Sudhir RR, Raman R, Sharma T. Changes in the corneal endothelial cell density and morphology in patients with type 2 diabetes mellitus: a population-based study, Sankara Nethralaya Diabetic Retinopathy and Molecular Genetics Study (SN-DREAMS, Report 23). *Cornea.* 2012; 31:1119–1122. [PubMed: 22357387]
8. Urban B, Raczyńska D, Bakunowicz-Lazarczyk A, et al. Evaluation of corneal endothelium in children and adolescents with type 1 diabetes mellitus. *Mediators Inflamm.* 2013; 2013:913754. [PubMed: 24381412]
9. Storr-Paulsen A, Singh A, Jeppesen H, et al. Corneal endothelial morphology and central thickness in patients with type II diabetes mellitus. *Acta Ophthalmol.* 2014; 92:158–160. [PubMed: 23387877]
10. Briggs S, Osuagwu UL, AlHarthi EM. Manifestations of type 2 diabetes in corneal endothelial cell density, corneal thickness and intraocular pressure. *J Biomed Res.* 2015; 30
11. Calvo-Maroto AM, Cervino A, Perez-Cambrodi RJ, et al. Quantitative corneal anatomy: evaluation of the effect of diabetes duration on the endothelial cell density and corneal thickness. *Ophthalmic Physiol Opt.* 2015; 35:293–298. [PubMed: 25545996]
12. Leelawongtawun W, Suphachearaphan W, Kampitak K, et al. A comparative study of corneal endothelial structure between diabetes and non-diabetes. *J Med Assoc Thai.* 2015; 98:484–488. [PubMed: 26058277]
13. Schultz RO, Matsuda M, Yee RW, et al. Corneal endothelial changes in type I and type II diabetes mellitus. *Am J Ophthalmol.* 1984; 98:401–410. [PubMed: 6486211]
14. Busted N, Olsen T, Schmitz O. Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus. *Br J Ophthalmol.* 1981; 65:687–690. [PubMed: 7317320]
15. Matsuda M, Ohguro N, Ishimoto I, et al. Relationship of corneal endothelial morphology to diabetic retinopathy, duration of diabetes and glycemic control. *Jpn J Ophthalmol.* 1990; 34:53–56. [PubMed: 2362374]
16. Larsson LI, Bourne WM, Pach JM, et al. Structure and function of the corneal endothelium in diabetes mellitus type I and type II. *Arch Ophthalmol.* 1996; 114:9–14. [PubMed: 8540858]
17. Siribunkum J, Kosirukvongs P, Singalavanija A. Corneal abnormalities in diabetes. *J Med Assoc Thai.* 2001; 84:1075–1083. [PubMed: 11758839]
18. Lee JS, Lee JE, Choi HY, et al. Corneal endothelial cell change after phacoemulsification relative to the severity of diabetic retinopathy. *J Cataract Refract Surg.* 2005; 31:742–749. [PubMed: 15899451]
19. Mathew PT, David S, Thomas N. Endothelial cell loss and central corneal thickness in patients with and without diabetes after manual small incision cataract surgery. *Cornea.* 2011; 30:424–428. [PubMed: 20885307]
20. Morikubo S, Takamura Y, Kubo E, et al. Corneal changes after small-incision cataract surgery in patients with diabetes mellitus. *Arch Ophthalmol.* 2004; 122:966–969. [PubMed: 15249359]
21. Hugod M, Storr-Paulsen A, Norregaard JC, et al. Corneal endothelial cell changes associated with cataract surgery in patients with type 2 diabetes mellitus. *Cornea.* 2011; 30:749–753. [PubMed: 21317781]
22. Inoue K, Tokuda Y, Inoue Y, et al. Corneal endothelial cell morphology in patients undergoing cataract surgery. *Cornea.* 2002; 21:360–363. [PubMed: 11973383]
23. Tsai CC, Kao SC, Kau HC, et al. Risk factors for corneal endothelial injury during small-incision cataract surgery in patients with diabetes mellitus. *Arch Ophthalmol.* 2005; 123:1020–1021. author reply 1021. [PubMed: 16009855]
24. Meyer LA, Ubels JL, Edelhauser HF. Corneal endothelial morphology in the rat. Effects of aging, diabetes, and topical aldose reductase inhibitor treatment. *Invest Ophthalmol Vis Sci.* 1988; 29:940–948. [PubMed: 2967259]

25. Shimazaki J, Tsubota K, Yoshida A, et al. Changes of corneal redox state in diabetic animal models. *Cornea*. 1995; 14:196–201. [PubMed: 7743804]
26. Saydah SH, Imperatore G, Beckles GL. Socioeconomic status and mortality: contribution of health care access and psychological distress among U.S. adults with diagnosed diabetes. *Diabetes Care*. 2013; 36:49–55. [PubMed: 22933434]
27. Centers for Disease Control and Prevention. National Diabetes Statistics Report: Estimates of Diabetes and Its Burden in the United States. Atlanta, GA: U.S. Department of Health and Human Services; 2014.
28. Lass JH, Riddleworth TD, Gal RL, et al. The effect of donor diabetes history on graft failure and endothelial cell density 10 years after penetrating keratoplasty. *Ophthalmology*. 2015; 122:448–456. [PubMed: 25439611]
29. Lass JH, Szczotka-Flynn LB, Ayala AR, et al. Cornea preservation time study: methods and potential impact on the cornea donor pool in the United States. *Cornea*. 2015; 34:601–608. [PubMed: 25850706]
30. Sugar A, Tanner JP, Dontchev M, et al. Recipient risk factors for graft failure in the cornea donor study. *Ophthalmology*. 2009; 116:1023–1028. [PubMed: 19395036]
31. Vislisel JM, Liaboe CA, Wagoner MD, et al. Graft survival of diabetic versus nondiabetic donor tissue after initial keratoplasty. *Cornea*. 2015; 34:370–374. [PubMed: 25642643]
32. Price MO, Thompson RW Jr, Price FW Jr. Risk factors for various causes of failure in initial corneal grafts. *Arch Ophthalmol*. 2003; 121:1087–1092. [PubMed: 12912684]
33. Yu AL, Kaiser M, Schaumberger M, et al. Donor-related risk factors and preoperative recipient-related risk factors for graft failure. *Cornea*. 2014; 33:1149–1156. [PubMed: 25170580]

Table 1
General Characteristics of the Eye Bank Population

All Age Groups	Non-Diabetic	Diabetic	p value
Number of Eyes	20036 (59.5%)	13616 (40.4%)	
Gender (M)	13,076 (65.2%)	8,631 (63.4%)	
Pseudophakic	1,302 (6.5%)	1,733 (12.7%)	
Mean Age (years)	59.2 ± 0.1	54.8 ± 0.1	< 0.001
By Age Subgroup			
Age 20 years or younger			
Number of Eyes	246	814	
Pseudophakic	NA	NA	
Mean Age (years)	15.4 ± 4.9	15.3 ± 5.3	0.648
Age between 21 to 40 years			
Number of Eyes	984	2,896	
Pseudophakic	NA	NA	
Mean Age (years)	31.7 ± 6.2	30.9 ± 5.9	0.140
Age between 41 to 60 years			
Number of Eyes	4,792	7,788	
Pseudophakic	172 (2.2%)	293 (6.5%)	
Mean Age (years)	52.9 ± 5.3	52.1 ± 5.4	< 0.001
Age 61 years or older			
Number of Eyes	7,594	8,538	
Pseudophakic	1128 (15.2%)	1432 (23.2%)	
Mean Age (years)	68.1 ± 4.6	68.1 ± 4.6	0.386

Abbreviations: NA (Not available)

Percentages are shown in reference to the entire cohort.

Table 2
Comparison of Mean Corneal Endothelial Cell Count between Diabetic and Non-Diabetic Donor Eyes

All Age Groups	n	Mean Endothelial Cell Count (cells/mm2)(±SEM)	p value
Non-Diabetic	20,036	2,604 ± 4	< 0.001
Diabetic	13,616	2,576 ± 5	
By Age Subgroup			
Age 20 years or younger			
Non-Diabetic	246	3,255 ± 17	0.874
Diabetic	814	3,260 ± 30	
Age between 21 to 40 years			
Non-Diabetic	984	2,850 ± 8	0.044
Diabetic	2,896	2,816 ± 14	
Age between 41 to 60 years			
Non-Diabetic	4,792	2,609 ± 6	< 0.001
Diabetic	7,788	2,575 ± 7	
Age 61 years or older			
Non-Diabetic	7,594	2,500 ± 6	< 0.001
Diabetic	8,538	2,470 ± 7	

Age-adjusted mean and p values are shown. SEM Standard Error of the Mean

Table 3
Comparison of Corneal Endothelial Cell Count Between Non-Diabetic and Diabetic Donors based on Phakic Status

Mean Endothelial Cell Count (cells/mm ²)(±SEM)					
	n	Phakic	p value	Pseudophakic [*]	p value
Age 20 years or younger					
Non-Diabetic	814	3,254 ± 16			
Diabetic	246	3,273 ± 30	0.586		
Age between 21 to 40 years					
Non-Diabetic	2,894	2,850 ± 8			
Diabetic	978	2,817 ± 14	0.048		
Age between 41 to 60 years					
Non-Diabetic	7,613	2,616 ± 6		172	2,338 ± 47
Diabetic	4,499	2,591 ± 7	0.009	293	2,298 ± 36
Age 61 years or older					
Non-Diabetic	7,403	2,538 ± 6		1,128	2,258 ± 19
Diabetic	6,161	2,532 ± 6	0.547	1,428	2,202 ± 17

Age adjusted means and p values are shown.

^{*} Insufficient number of pseudophakic subjects under age 41 for meaningful analysis