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*Original Article*

# A Study of Diabetic and Non-diabetic Human Cataract Lens by Scanning Electron Microscopy

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## ABSTRACT

**Background:** This research aimed to ascertain the differences in morphological manifestation in the human diabetic cataractous lens compared to a non-diabetic cataractous lens by Scanning Electron Microscopy (SEM). **Materials and Methods:** Lens fibers were prefixed in glutaraldehyde and subsequently post-fixed in a Hexamethyldisilazane and examined by SEM. The SEM images gave a comparative morphological impression of the ongoing structural alterations during the degeneration process of human non-diabetic cataracts and diabetic cataract lenses. **Results:** Diabetic cataractous lens fibers were identified by more chaotic longitudinal fiber splitting, porosity, and granulation of the lens fiber membrane and opening (distortion) of the lens fiber interdigitation system than their non-cataractous diabetic counterparts. Diabetic cataract lenses showed an increased rate of nucleus compaction compared to non-diabetic cataract lenses. **Conclusion:** The absence of Finger-like and flap projections and the disappearance of the ball and socket system is prominent in diabetic cataract lenses, suggesting the hyperglycaemic effect in the degeneration of lens fibers and further contributes to the early onset of cataracts in diabetes compared to non-diabetic.

**Keywords:** Cataract, Diabetic complications, Morphology, Scanning electron microscopy.

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## INTRODUCTION

Worldwide more than Two Hundred and Eight Five million individuals have agonized from diabetes mellitus. This integer is expected to augment to four hundred and thirty-nine million as per the International Diabetes Federation by 2030. Among the Type one and Type two diabetic patients, diabetic retinopathy is the main common impediment, measured as the most widespread reason for losing sight in the United States.[1] Diabetes mellitus can lead to pathologies in many tissues in the eye configuration, with both a systemic, persistent metabolic ailment and a microvascular character.[2]

A cataract is a primary reason for blindness in diabetic patients as the frequency and succession of cataracts are superior in patients with uncontrolled diabetes mellitus.[3-4] Clinical epidemiological and fundamental investigation studies have exposed the relationship between diabetes and cataract formation.

As of now, cataract surgical treatment is the most general surgical ophthalmic practice to alleviate the cataract globally successfully. However, there is a lack of clarification of pathomechanism and morphological manifestations of diabetic cataract lenses to a setback or thwart the progress of cataracts in diabetic patients. Hence the current research explores the morphological changes of human diabetic cataract lenses in contrast with non-diabetic cataract lenses by scanning electron microscope.

## MATERIALS AND METHODS

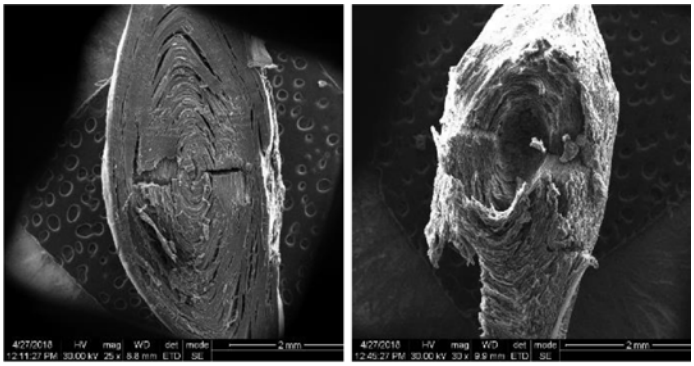
The age-matched diabetic cataract and non-diabetic cataract lenses were used in this study and approved by the Institutional Ethical Committee (IEC-NI/15/OCT49/61). The Cataract lens was obtained after Extracapsular cataract extraction. For fixation, the lenses were placed in 3% glutaraldehyde in phosphate buffer (pH 7). Then it was left overnight, approximately for eight hours, to ensure absolute infiltration. The sample was rinsed with fresh buffer three times in the fume hood. A succession of ethanol concentrations of 50, 60, 70, 80, 90, and 100 percent v/v of ethanol in distilled water was prepared for dehydration. The buffer solution was replaced with the lowest concentration of ethanol solution in the dehydration series and left for 15-20 min. Continued until the samples are in 100 percent ethanol, and the 100 percent ethanol step was repeated. Are at this stage, the samples are dehydrated for drying. A chemical drying agent, Hexamethyldisilazane (HMDS), was used.

The sample was transferred from 100% ethanol into a 1:2 solution of HMDS: 100% ethanol and left for 20 min. After that, the sample was moved to a fresh solution of 2:1 HMDS: ethanol for twenty minutes. Then the sample was transferred to a Hundred percent HMDS for twenty minutes and repeated the above step. While the sample was flooded in the final 100% HMDS solution, it was sheltered slackly in a fume hood all night. The HMDS has been evaporated, and the sample processed to the image by SEM makes use of sputter coating (Model: FEI Quanta FEG 200, FEI JERMAN).

## RESULTS

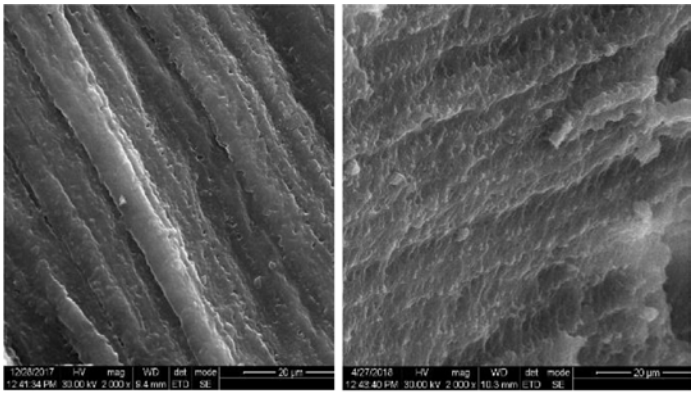
The SEM images of the longitudinal sections of the diabetic and non-diabetic cataract lens are shown in Figure 1. In the non-diabetic cataract lenses, the Capsule, cortex, and nuclear regions are visible, and sutures can be seen clearly from the lens. A diabetic cataract lens shows a more disordered splitting of longitudinal fibers than a non-diabetic lens.

The longitudinal sections of the nuclear part of diabetic and non-diabetic cataract lens are shown in Figure 2. A higher degree of compaction



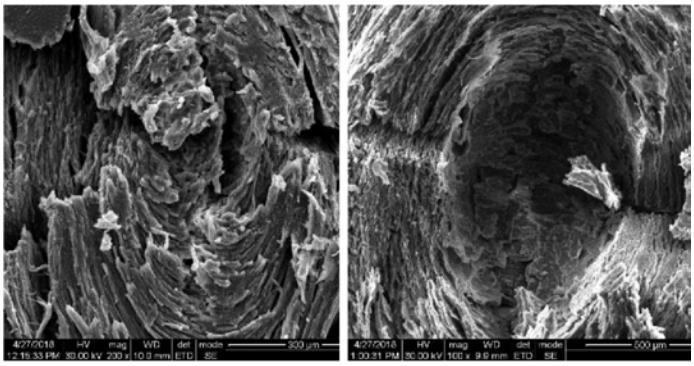
Non-diabetic Lens Diabetic Lens

**Figure 1:** Longitudinal sections of the diabetic and non-diabetic cataract lens by SEM imaging.



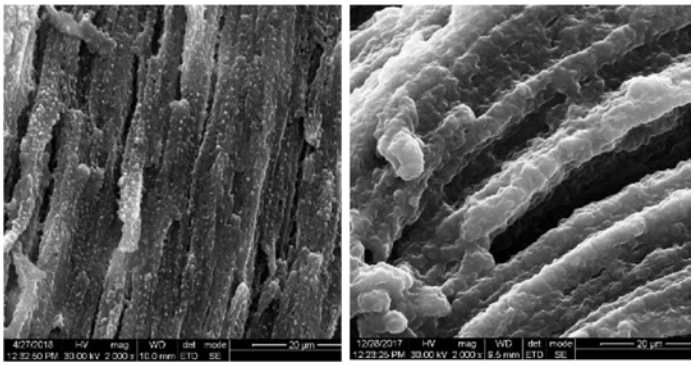
Non-diabetic Lens Diabetic Lens

**Figure 3:** The longitudinal sections of the mid cortex region of diabetic and non-diabetic cataract lens by SEM imaging.



Non-diabetic Lens Diabetic Lens

**Figure 2:** The longitudinal sections of nuclear part of diabetic and non-diabetic cataract lens by SEM imaging.



Non-Diabetic Lens Diabetic Lens

**Figure 4:** The longitudinal sections of the deep cortex region of diabetic and non-diabetic cataract lens by SEM imaging.

in nucleus fiber cells was seen in diabetic lenses than in non-diabetic cataract lenses. The longitudinal sections of the mid cortex region of diabetic and non-diabetic cataract lenses are shown in Figure 3. The mid cortex demonstrates the regular hexagonal close packing of fibers with shrunken fingerlike projections and flap projections in non-diabetic cataract lenses but not in diabetic cataract lenses.

The SEM images of longitudinal sections of the deep cortex region of diabetic and non-diabetic cataract lenses are shown in Figure 4. The deep cortex or most superficial nuclear regions shows the interconnecting system, also called a ball and socket joints which are still visible in non-diabetic cataract lenses with disintegrated lens fibers. The ball and socket joints almost disappeared or appeared to be more rounded in the diabetic cataract lens.

## DISCUSSION

Focusing light rays onto the retina is the critical function of the human lens. It consists of decidedly dedicated three essential parts, including lens capsule, lens epithelium, and lens fibers. Lens capsules form the furthest layer of the lens, and the interior of the lens is formed by lens fibers and the lens epithelium, located between the lens capsule and the outermost layer of lens fibers.[5] Firmly packed layers of lens fibers are referred to as laminae. The lens fibers are linked mutually through gap junctions and interdigitations of the cells that resemble "ball and socket" forms.[6]

Lens fibers also have a very wide cytoskeleton that preserves the specific shape and packing of the lens fibers, and disruption or mutations in certain cytoskeletal elements can lead to the loss of lens transparency.[7] Diabetic Mellitus is coupled with a fivefold higher incidence of cataracts, which remains the most critical reason for blindness around the globe. A characteristic diabetic cataract includes cortical and posterior subcapsular and nuclear opacities. The duration of diabetes and quality of glycaemic control is the most critical risk factors for diabetic cataract formation.[8]

The following mechanisms are assumed to be the reason for the development of cataracts in untreated diabetes. The first proposed mechanism is the polyol pathway. Lack of insulin causes glucose accumulation in blood and the aqueous humor and the lens. Glucose reduction to sorbitol occurs inside the lens through the aldose reductase pathway. Furthermore, the rate of production of sorbitol in diabetic patients takes place more quickly than in non-diabetic patients, and the produced sorbitol can be converted into fructose by the enzyme sorbitol dehydrogenase more slowly compared to the production rate. Further, sorbitol metabolism proceeds very slowly; the resulting sorbitol accumulation is followed by an osmotic swelling of the lens fibers. Characteristic findings of a completely developed diabetic cataract are snowflake-like opacifications of the anterior and posterior lens cortex.[9-10]

Another proposed mechanism is non-enzymatic glycation. Advanced glycation arises during normal aging but to a greater degree in diabetic patients, contributing to lens opacity formation. Advanced glycation is produced by a non-enzymatically reaction between the piece of the excess

glucose and proteins, which may lead to the production of superoxide radicals and advanced glycation end-product formation. Excessive accumulation of advanced glycation end product in the crystalline lens of diabetic patients plays an indispensable role in cataractogenesis.

Increased oxidative stress is another proposed mechanism of diabetic cataracts. In diabetic eyes, antioxidant capacity is reduced free radical load is increased, which increases the susceptibility of the crystalline lens to oxidative damage. The decrease in antioxidant capacity is facilitated by advanced glycation and defects of antioxidant enzyme activity. Chronic hyperglycemia may increase the oxidant load and facilitate the onset of senile cataracts. This study indicates that some diabetic cataract lens nuclei areas are more likely affected than non-diabetic cataract lenses. A specifically higher degree of compaction in nucleus fiber cells was observed in diabetic cataracts. These findings are consistent with,[11] which analyzed SEM images of transparent diabetic and cataractous diabetics and explored a higher nuclear compaction rate in diabetic cataract lenses.

The overall shape of the diabetic cataract lens fibers remained the same, but the socket interconnections were unlike the normal cataract lens. The diabetic cataract lens had more chaotic longitudinal fiber splitting around the place. More Shrinkage of the originally hexagonally-shaped lens fibers with a wrinkled surface and more granular material was observed, which could account for a considerable reduction in vision due to a substantial increase in light scattering through the lens. Our study is consistent with,[12] who reported an elevated proportion of cortical cloudiness in people with diabetes, as recognized by Scheimpflug photography and densitometric analysis. In addition, they confirmed correspondence between type two diabetes mellitus and cortical lens cloudiness.

The dry lens matter is mostly of crystalline protein, which changes very slowly during the life of a normal human eye. If exposed to free radicals, lens transparency is affected due to carbonylation and glycation in the lens protein. This process is very fast in diabetic patients with high plasma glucose levels leading to more damage in lens fibers than in non-diabetic, which may be a reason for the early onset of cataracts in diabetic patients. It has been reported that ultrastructural studies of lenses from mature onset diabetes subjects exposed to declined epithelial cell density and extensive cellular damage to the outer region of the lens nuclei, particularly at the cortical, nuclear boundary.[13]

## CONCLUSION

The unique morphological structure of the human lens was more intensively denatured in diabetic cataracts than in non-diabetic cataracts. Uncontrolled plasma glucose levels lead to a lack of antioxidant protection for the lens cells putting forward the possible reason for the early onset of cataracts in diabetes. In conclusion, maintaining the proper control of plasma glucose levels is the only solution to delay the early onset of cataracts in diabetic patients.

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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