Journal of Clinical Research and Ophthalmology

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Dates: **Received:** 08 December, 2015; **Accepted:** 08 December, 2015; **Published:** 10 December, 2015

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www.peertechz.com

Keywords: Cornea; Diabetes; *In vitro; In vivo*

ISSN: 2455-1414

Editorial

Corneal Diabetes: Where to Next?

Editorial

Diabetes mellitus (DM) or better known as simply diabetes is a group of metabolic diseases in which high blood sugar levels are maintained over a prolonged period. Long term complications include but not limited to heart disease, stroke, kidney failure, and ocular damage. There are two main types of diabetes: Type I (T1DM) and Type II (T2DM). In 2013, an estimated 382 million people were diagnosed with diabetes with type 2 accounting for 90% of the cases. Unfortunately, to date, despite significant amount of research there is no known cure except in very specific cases.

DM was one of the first diseases described [1], with the first described cases believed to be T1DM. Originally, the disease was classified as "madhumeha" or "honey urine"[1], and was noted that urine attracts ants. The term diabetes was first used in 230 BCE and was considered as a rare disease. T1DM and T2DM were described and identified in 400-500 by Sushruta and Charaka [1], who associated T1DM with youth and T2DM with being overweight [1]. Today we know that T1DM is characterized by loss of the insulin-producing beta cells of the islets of Langerhans in the pancreas [2,3]. T2DM, on the other hand, is known for insulin resistance and it is the more common of the two [4,5].

With regards to ophthalmic complications as a result of DM, most profound effects are seen in cornea and retina. In fact, of the 382 million people diagnosed with DM worldwide approximately 70% of them suffer from some kind of corneal complications collectively and commonly known as diabetic keratopathy [6-10]. Common dysfunctions that may lead to impaired vision or blindness include decreased wound healing, corneal edema, and altered epithelial basement membrane. Clinically, we have no preventive measure for T1DM, while T2DM can be managed by means of physical exercise, weight control, and diet. Even then, the effect on the cornea will depend on the severity of the disease and the stage at which it was diagnosed. Unfortunately, DM is a chronic disease and corneal impairments are almost inevitable, mainly due to the fact that the eye has been exposed to hyperglycemia long-term and the basement membrane has accumulated enough toxic end products to lead to cell death, opacity, and eventually vision impairment.

In terms of research, in vivo, scientists have concentrated for years on animal studies and developed a variety of animal models both for T1DM [15-19] and T2DM [20-28], as recently reviewed by King [29]. However, there is a significant lack of reproducible paradigms of human DM complications and rather disappointing results when rodents' treatments are tested on humans. In vitro, there are quite a large number of studies looking into nerve pathologies and corneal sensitivity [9,11-14,30,31]. Perhaps the most advanced model is the organotypic cultures, developed by Ljubimov and co-authors [32,33], for the identification of epithelial defects in DM. Even still, we are way far from our ultimate goal which is to treat and prevent corneal damage and vision impairment.

In summary DM is a multifactorial disease and when it affects the human cornea there is a variety of factors that we have to consider if we are going to treat any defects. Clearly, both in vivo and *in vitro* studies are necessary and huge advancements have been made over the last ten years, but we need further and greater understanding of the molecular events at the initial stages of the disease.

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Citation: *Dimitrios K (2015) Corneal Diabetes: Where to Next? J Clin Res Ophthalmol 2(4): 065-066. DOI: 10.17352/2455-1414.000024*