Opinion
Volume 7:6, 2022

Journal of Diabetic Complications and Medicine

ISSN: 2475-3211 Open Access

In the field of Diabetes, Thirty Years of Fruitful Collaborations between a Physician and Mass Spectrometricists

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Introduction

Long-term diabetes complications are caused by a process called nonenzymatic protein glycation, which results in the formation of advanced glycation end products. In this context, the 30 years of collaboration between my research group at Padua University's DPT of Medicine and the mass spectrometric group at Padua University's CNR are described and discussed. Beginning with applications on small molecules that are responsible for the browning that is observed in the interactions between sugars and proteins, and progressing all the way up to intact proteins like albumin, immunoglobulin, hemoglobin, and so forth, with the determination of their glycation levels as well as their glycation sites, new mass spectrometric techniques have made it possible to conduct more in-depth research. The role of advanced glycation end products in the development of diabetes's chronic complications has been better understood thanks to this study. This review focuses primarily on the findings that were obtained from placenta samples of patients with gestational diabetes, diabetic cardiovascular disease, and diabetic nephropathy. [1].

Description

Macrovascular complications of diabetes (MCD)—basically ischaemic heart disease, cerebrovascular disease, and peripheral vascular disease—develop in over half of the diabetic population, resulting in high morbidity and mortality. This poses a serious threat to global public health. Diabetes is predicted to be the seventh leading cause of death worldwide by 20301. Diabetic patients are still at risk of developing MCD despite successfully controlling hypertension, hyperlipidemia, and hyperglycemia.4 As a result, it is essential to identify more viable drug targets and develop more efficient approaches in order to prevent or slow down the pathogenesis and progression of MCD. In diabetes, excessive reactive oxygen species (ROS) are produced, resulting in detrimental cellular events such as the formation of advanced glycation end products (AGEs) and the overexpression of receptor for AGEs (RAGE), as well as activation of the polyol pathway, hexosamine pathway, and protein kinase C (PKC).5, 6 These events contribute to the pathogenesis and progression of MCD [2].

Numerous antioxidant genes are activated by the transcription factor nuclear factor (erythroid-derived 2)-like 2 (NRF2), which in turn produces cellular antioxidants 7 that act as free radical scavengers and prevent the pathogenesis of MCD that is caused by oxidative stress. In the cytoplasm, Kelch-like ECH-associated protein 1 (KEAP1) has a negative effect on NRF2. Small molecule-induced structural inhibition of KEAP1 protein—a canonical way to activate NRF2—has become a research hotspot in the past two decades, with the protective effect confirmed in animal models of MCD. KEAP1 prevents NRF2 from nuclear translocation on the one hand and facilitates NRF2's proteasomal degradation on the other. In MCD, other strategies have been used to activate NRF2 in addition to structurally inhibiting the KEAP1 protein. These include the activation of the transcription of the Nfe2l2 gene, the reduction in the level of KEAP1 protein caused by the microRNA-induced degradation of Keap1 mRNA, the prevention of NRF2 protein degradation by proteasomes, and the modulation of other NRF2 upstream regulators. These investigations have revealed novel targets upstream of NRF2 for the intervention of MCD as well as alternative strategies for activating NRF2. With the intention of providing insight into future management of MCD, we summarize and discuss the various strategies developed to activate NRF2 and their results in MCD [3-5].

Conclusion

MicroRNA-induced inhibition of KEPA1 production, inhibition of NRF2's proteasomal degradation, inhibition of NRF2's AKT/GSK-3/Fyn-mediated nuclear export, and modulation of HDAC/P300-controlled Nfe2l2 gene transcription are some of the newer strategies for NRF2 activation that have emerged in recent years (Figure 2, red characters). Epigenetic mechanisms like microRNAs45, 48 and histone modifications28, 47 have recently emerged as effective approaches in these novel strategies for NRF2 activation with potential for MCD intervention. In addition, NRF2 antioxidant signaling is regulated by a number of other epigenetic mechanisms, including DNA methylation, circular RNAs, and long non-coding RNAs.99, 100. These epigenetic mechanisms should provide new information for future research on NRF2 activation in MCD.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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Received: 02 November, 2022, Manuscript No. jdcm-23-86185; **Editor Assigned:** 04 November, 2022, PreQC No. P-86185; **Reviewed:** 16 November, 2022, QC No. Q-86185; **Revised:** 22 November, 2022, Manuscript No. R-86185; **Published:** 28 November, 2022, DOI: 10.37421/2475-3211-2022.7.191

How to cite this article: Khoei, Mahsa Khayat. “In the field of Diabetes, Thirty Years of Fruitful Collaborations between a Physician and Mass Spectrometricists." J Diabetic Complications Med 7 (2022): 191.