

Chapter 23. Lutein and Oxidative Stress-Mediated Retinal Neurodegeneration in Diabetes



DIABETES

Oxidative Stress and Dietary Antioxidants

Edited by
VICTOR R. PREEDY



Diabetes Chapter 23 Lutein And Oxidative Stress Mediated Retinal Neurodegeneration In Diabetes

Yoko Ozawa, Mariko Sasaki



Diabetes Chapter 23 Lutein And Oxidative Stress Mediated Retinal Neurodegeneration In Diabetes:

Diabetes Yoko Ozawa, Mariko Sasaki, 2013-10-29 Although many details remain elusive recent progress in molecular biology has elucidated the underlying mechanisms of many disease pathogeneses including some fundamental concepts One such concept is that oxidative stress is involved in the age related progression of diseases as observed in diabetic complications and neurodegenerative diseases Concordantly oxidative stress accumulates with time and can eventually overcome the biological defense systems Therefore the progression of the diseases might be prevented or slowed by suppressing oxidative stress Here we focus on lutein a plant derived carotenoid and food factor that can act as an antioxidant in the mammalian retina In an animal model of diabetes the continuous intake of lutein suppresses the diabetes related reduction of synaptophysin protein and brain derived neurotrophic factor in the retina and the subsequent visual function impairment This mechanism overlaps with the renin angiotensin system Here we review the involvement of oxidative stress in diabetes associated retinal neurodegeneration and discuss lutein s neuroprotective effects

Diabetes Victor R Preedy, 2020-04-24 Diabetes Oxidative Stress and Dietary Antioxidants Second Edition builds on the success of the first edition covering updated research on the science of oxidative stress in diabetes and the potentially therapeutic usage of natural antioxidants in the diet and food matrix The processes within the science of oxidative stress are not described in isolation but rather in concert with other processes such as apoptosis cell signaling and receptor mediated responses This approach recognizes that diseases are often multifactorial and oxidative stress is a single component of this Since the publication of the first edition the science of oxidative stress and free radical biology continues to rapidly advance with thousands of the research articles on the topic New sections in this update cover the role of dietary advanced glycation end products AGEs in causing OS in diabetes oxidative stress and diabetes induced bone metabolism and oxidative stress and diabetic foot ulcer Saves clinicians and researchers time in quickly accessing the very latest details on a broad range of diabetes and oxidation issues Combines the science of oxidative stress and the putative therapeutic usage of natural antioxidants in the diet its food matrix or plant Includes preclinical clinical and population studies to help endocrinologists diabetologists nutritionists dieticians and clinicians map out key areas for research and further clinical recommendations

Oxidative Stress in Diabetic Retinopathy Ángel Luis Ortega, 2021-03-09 The combination of an increasing prevalence of diabetes and the aging of populations enables the appearance of a greater number of associated complications such as diabetic retinopathy Diabetic retinopathy is the leading cause of preventable vision loss in working age adults The objective of this Special Issue is to highlight the existing evidence regarding the relationship between oxidative stress and low grade chronic inflammation induced by hyperglycemia with the development and progression of diabetic retinopathy with an emphasis on the importance of early diagnosis and the use of antioxidant and anti inflammatory approaches to prevent or delay the harmful effects of diabetes on retinal tissue

Diabetes Jose Javier Garcia-Medina, null Monica

del-Rio-Vellosillo,Manuel Garcia-Medina,Vicente Zanon-Moreno,Roberto Gallego-Pinazo,Maria Dolores Pinazo-Duran,2013-10-29 Diabetic retinopathy DR is the leading cause of acquired blindness in working age adults worldwide Biochemical changes in DR contribute to both the microscopic structural and functional changes in the retina All these alterations result in macroscopic retinal damage that can be assessed by funduscopy The overproduction of reactive oxygen species ROS in the mitochondria is considered a causal link between elevated glucose and biochemical abnormalities in the pathophysiology of DR Moreover oxidatively induced pathways also seem to provide positive feedback to ROS production resulting in a vicious cycle ROS can directly damage lipids proteins and DNA leading to cell death To make the situation worse antioxidant defenses are also impaired in diabetes Otherwise ROS are involved in the production of inflammatory cytokines and growth factors such as interleukins or vascular endothelial growth factor VEGF In addition ROS not only contribute to the development of DR but also play a role in its progression when the hyperglycemic insult is controlled This phenomenon is called metabolic memory

Diabetes Christine Lee,Amanda I. Adler,2013-10-29 Retinopathy is a serious microvascular complication of diabetes These retinal vascular abnormalities are one of the leading causes of blindness in diabetes Oxidative stress in the retina contributes to the development of diabetic retinopathy Micronutrients including vitamin A vitamin C and carotenoids possess antioxidant properties and may be used as an adjunct therapy to slow the development and the progression of retinopathy This chapter reviews the epidemiologic findings relating these antioxidant related micronutrients to diabetic retinopathy Although some evidence suggests an inverse association of the carotenoids lycopene lutein and zeaxanthin with diabetic retinopathy there is inadequate evidence to support a role for vitamin C and vitamin E Further epidemiologic studies are needed to characterize the nature of this association and to justify randomized controlled trials

Oxidative Stress in Diabetic Retinopathy Ángel Luis Ortega,2021 The combination of an increasing prevalence of diabetes and the aging of populations enables the appearance of a greater number of associated complications such as diabetic retinopathy Diabetic retinopathy is the leading cause of preventable vision loss in working age adults The objective of this Special Issue is to highlight the existing evidence regarding the relationship between oxidative stress and low grade chronic inflammation induced by hyperglycemia with the development and progression of diabetic retinopathy with an emphasis on the importance of early diagnosis and the use of antioxidant and anti inflammatory approaches to prevent or delay the harmful effects of diabetes on retinal tissue

Diabetes Jose Javier Garcia-Medina,Monica del-Rio-Vellosillo,Vicente Zanon-Moreno,Manuel Garcia-Medina,Maria Dolores Pinazo-Duran,Roberto Gallego-Pinazo,2013-10-29 Conventional treatments for diabetic retinopathy DR includes glycemic control laser photocoagulation vitrectomy intravitreal triamcinolone and intravitreal anti vascular endothelial growth factor agents However these strategies have not proved capable of halting the progression of this disease in all cases The mechanisms leading to DR are not fully understood but there is a growing body of evidence showing that oxidative stress plays a pivotal

role in the development of this diabetic complication. In fact, it has been proposed that oxidative stress is the initial and maintaining event that triggers and provides feedback to the other pathophysiological pathways related to DR. The experimental data discussed in this review show that different antioxidant agents may prevent various biochemical and structural alterations in retinal cells cultured under hyperglycemic conditions and also in experimental diabetic animals. Although the results of human trials are somewhat variable, some clinical studies have provided promising results.

Diabetic Retinopathy Elia Duh, 2008-10-08 Diabetic retinopathy is the most common microvascular complication of diabetes. It remains a major cause of new onset visual loss in the United States and other industrialized nations. In *Diabetic Retinopathy*, Elia Duh and a panel of internationally recognized experts comprehensively assess the current state of knowledge regarding the clinical management of DR as well as its underlying mechanisms. The authors outline the current understanding of diabetic retinopathy from the perspective of clinical practice while reviewing the multifactorial pathogenesis and pathophysiology of DR from the standpoint of biomedical research. Also included is a discussion of emerging concepts relating to the management and treatment of DR. Informative and highly practical, *Diabetic Retinopathy* provides ophthalmologists, diabetologists, endocrinologists, and internists with a highly readable guide not only to understanding diabetic retinopathy but also to its optimal clinical management.

The Role Of Regulated In Development and DNA Damage Response 1 (REDD1) in Diabetes-Induced Retinal Pathology and Visual Dysfunction William Miller, 2022

Diabetic Retinopathy (DR) is a major cause of visual dysfunction, yet much remains unknown regarding the specific molecular events that contribute to diabetes-induced retinal pathophysiology. Herein, we explored a role for the stress response protein regulated in development and DNA damage 1 (REDD1) in the development of diabetes-induced oxidative stress and functional defects in vision. It is well established that REDD1 is associated with the development of oxidative stress in a number of cell types and tissues, and that REDD1 protein content and oxidative stress are both increased in the retina of diabetic mice. However, prior to the work detailed in this dissertation, little was known surrounding the mechanism by which the diabetic metabolic environment promotes retinal REDD1 accumulation and if that increase in REDD1 content contributes to the development of oxidative stress and retinal defects. The overarching goal of this dissertation was to test the hypothesis that diabetes-induced REDD1 contributes to the development of retinal pathology by exacerbating oxidative stress. To test the hypothesis, we first assessed the role of REDD1 in mitochondrial reactive oxygen species (ROS) production. In the retina of streptozotocin (STZ)-induced diabetic mice, REDD1 protein expression and ROS levels were increased. In rat retinal R28 cell cultures, hyperglycemic conditions enhanced REDD1 protein expression, ROS levels, and the mitochondrial membrane potential. However, similar effects were not observed in the retina of diabetic mice or R28 cells lacking REDD1. In the retina of diabetic mice and cells exposed to hyperglycemic conditions, the antioxidant N-acetyl-L-cysteine (NAC) normalized ROS levels and prevented an increase in REDD1 expression. Diabetic mice treated with NAC also exhibited improved contrast sensitivity.

as compared to diabetic controls Hydrogen peroxide H₂O₂ addition to culture medium increased REDD1 expression in R28 cells and attenuated Akt Glycogen Synthase Kinase 3 GSK3 phosphorylation in a REDD1 dependent manner In REDD1 deficient R28 cells exposed to hyperglycemic conditions expression of a dominant negative Akt or constitutively active GSK3 increased the mitochondrial membrane potential and promoted ROS levels The findings provided new insight into the mechanisms whereby diabetes induced hyperglycemia causes oxidative stress and visual dysfunction Specifically the findings are consistent with a model wherein hyperglycemia induced REDD1 activated a ROS generating feedback loop that included Akt GSK3 The transcription factor Nuclear factor erythroid 2 related factor 2 Nrf2 plays a critical role in preventing oxidative stress by promoting the expression of antioxidant genes The retinal antioxidant response to diabetes is insufficient to prevent oxidative stress due at least in part to blunted activation of Nrf2 In a second series of studies we tested the hypothesis that diabetes induced REDD1 promotes oxidative stress in the retina by suppressing Nrf2 activity We found that REDD1 ablation enhanced Nrf2 DNA binding activity in the retina and that the suppressive effect of STZ diabetes on Nrf2 activity was absent in the retina of REDD1 deficient mice compared with wild type WT In human MIO M1 retinal Müller cell cultures REDD1 deletion prevented oxidative stress in response to hyperglycemic conditions and this protective effect required Nrf2 REDD1 suppressed Nrf2 stability by promoting its proteasomal degradation independently of Nrf2's interaction with Kelch like ECH associated protein 1 Keap1 but REDD1 mediated Nrf2 degradation required GSK3 activity and Ser 351 Ser 356 of Nrf2 Diabetes diminished inhibitory phosphorylation of the beta isoform of GSK3 GSK3 beta at Ser 9 in the retina of WT mice but not in REDD1 deficient mice Pharmacological inhibition of GSK3 enhanced retinal Nrf2 activity and prevented oxidative stress in the retina of diabetic mice The findings support a model wherein hyperglycemia induced REDD1 blunts the Nrf2 antioxidant response to diabetes by activating GSK3 which in turn phosphorylates Nrf2 to promote its degradation Finally we investigated the hypothesis that diabetes promotes REDD1 expression by reducing the rate of REDD1 degradation In the retina of STZ diabetic mice REDD1 protein content was increased in the absence of a change in REDD1 mRNA In human MIO M1 retinal Müller cells the rate of REDD1 degradation was reduced upon exposure to hyperglycemic conditions or the oxidant H₂O₂ Antioxidant addition to culture medium prevented both the increase in oxidative stress and the reduced rate of REDD1 degradation in cells exposed to hyperglycemic conditions Surprisingly the suppressive effect of oxidative stress on REDD1 degradation was independent of the ubiquitin proteasome pathway Rather chemical or genetic activation of chaperone mediated autophagy CMA caused REDD1 localization to lysosomes and degradation of the protein Moreover exposure to hyperglycemic conditions prevented the suppressive effect of CMA activation on REDD1 expression Overall the findings support a model wherein diabetes promotes retinal REDD1 content by preventing the protein from being degraded by CMA

Visual Dysfunction and Impaired Defense Against Oxidative Stress in Diabetic Retinopathy Zeinab Nassrallah, 2016 Loss of visual function progressive neurodegeneration and oxidative stress are pathological characteristics

of diabetic retinopathy The mechanisms underlying the increase in oxidative stress and apoptosis in diabetic retinopathy are poorly understood but cytoprotective antioxidant transcription factor Nrf2 may be a target of the diabetic retinopathy pathology Existing reports on the effect of diabetes on retinal Nrf2 are conflicting but dysregulation of Nrf2 protein content and DNA binding may be a major contributing factor to the elevated oxidative stress that is evident in diabetic retinopathy In this study the effect of various stressors on Nrf2 protein content and DNA binding in the rat retina is determined including the effects of prolonged dark adaptation and diabetes In addition to an interest in Nrf2 recent attempts to restore oxidative balance by dietary supplementation with antioxidants have proven to be potentially therapeutic in humans and animal models of retinal degenerative disease Of particular interest is zeaxanthin a retinal protective macular pigment which is reduced in retina of individuals with diabetes In this study the streptozotocin diabetic rat model was used to determine the therapeutic potential of daily zeaxanthin treatment on retinal apoptosis synaptic protein synaptophysin content visual function morphology and the oxidative stress response proteins Nrf2 and NAD P H dehydrogenase quinone 1 NQO1 Phosphorylated Nrf2 and NQO1 protein as measured by immunoblot were significantly reduced in retinas of STZ diabetic rats compared to controls suggesting an impaired defense against oxidative stress Inner retinal thinning outer retinal thickening and photoreceptor outer segment thinning determined by OCT combined with loss of synaptophysin protein provided structural and molecular changes that may contribute to the loss of visual function observed in STZ diabetic rats The therapeutic effect of zeaxanthin treatment was limited to preventing apoptosis in STZ diabetic rats compared to control groups

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