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2/15-LIPOXYGENASE CONTRIBUTES TO RETINAL NEURONAL INJURY IN DIABETIC RETINOPATHY

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12/15-Lipoxygenase Contributes to Retinal Neuronal Injury in Diabetic Retinopathy

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ABSTRACT

Diabetic retinopathy (DR) is a neurovascular complication of diabetes in which treatment is limited due to a lack of neuroprotection. The goal of the current study was to investigate the potential role of 12/15-lipoxygenase (12/15-LO) lipid metabolites 12 and 15-hydroxyeicosatetraenoic acids (12 and 15-HETE) in neurodegeneration associated with diabetic retinopathy. To characterize the role of 12/15-LO, we generated a unique model of diabetic mice that lacks 12/15-LO (Ins2Akita+/12/15-LO-/-). Electroretinogram (ERG) was used to evaluate the functional changes in diabetic (Ins2Akita/+) compared to control and diabetic mice that lack 12/15-LO. Retina from these mice were also stained with hematoxylin and eosin (H&E) and retinal thickness was measured via image-J. ERG assessment showed improvement in retinal function of the Ins2Akita+/12/15-LO-/- mice compared to the Ins2Akita/+ mice and restored the loss of RGC marker, Brn3. Interestingly, the Photopic B-Wave amplitude in Ins2Akita+/12/15-LO-/- mice improved significantly compared to diabetic mice. Deletion of 12/15-LO restored the total and inner retinal thickness in diabetic mice. Immunolocalization of the neuronal marker, NeuN showed marked reduction in diabetic mice and was restored in diabetic mice lacking the 12/15-LO. Our findings indicate that 12/15-LO contributes to neuroglial injury and its inhibition potentially protects retinal neurons in diabetes.

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