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Non-coding RNAs- novel therapeutic targets for diabetic retinopathy

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In the post genomic era, we have now the capacity to develop novel therapies, targeting epigenetic mechanisms. Such targets in the context of diabetic retinopathy include histone acetylation, histone methylation, microRNAs and long non-coding RNAs. We focused on microRNAs and long non-coding RNAs as potential treatment targets of diabetic retinopathy. In diabetic retinopathy, glucose-induced damage leads to changes in the cellular transcription. Endothelial cells (ECs), being exposed to hyperglycemia changes their synthetic and secretory phenotype. Subsequently, other cell in the retina is affected. We have demonstrated that diabetes activates transcription factors (eg: NFκB, regulated by transcription co-activators, p300). We have also demonstrated that some long noncoding RNAs (lncRNAs) such as ANRIL and MALAT1 as well as microRNAs (eg. mir2146a, miR200b) regulate glucose induced increased production of angiogenic factors, extracellular matrix (ECM) proteins and inflammatory cytokines. Our laboratory has carried out large number of experiments in the ECs, in animals with type 1 and type 2 diabetes and in the retina and vitreous from diabetic patients. Data generated from these studies shows novel pathogenetic mechanisms and drug development targets for diabetic retinopathy.

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