An update on connexin gap junction and hemichannels in diabetic retinopathy

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Abstract
Diabetic retinopathy (DR) is one of the main causes of vision loss in the working age population. It is characterized by a progressive deterioration of the retinal microvasculature, caused by long-term metabolic alterations inherent to diabetes, leading to a progressive loss of retinal integrity and function. The mammalian retina presents an orderly layered structure that executes initial but complex visual processing and analysis. Gap junction channels (GJC) forming electrical synapses are present in each retinal layer and contribute to the communication between different cell types. In addition, connexin hemichannels (HCs) have emerged as relevant players that influence diverse physiological and pathological processes in the retina. This article highlights the impact of diabetic conditions on GJC and HCs physiology and their involvement in DR pathogenesis. Microvascular damage and concomitant loss of endothelial cells and pericytes are related to alterations in gap junction intercellular communication (GJIC) and decreased connexin 43 (Cx43) expression. On the other hand, it has been shown that the expression and activity of HCs are upregulated in DR, becoming a key element in the establishment of proinflammatory conditions that emerge during hyperglycemia. Hence, novel connexin HCs blockers or drugs to enhance GJIC are promising tools for the development of pharmacological interventions for diabetic retinopathy, and initial in vitro and in vivo studies have shown favorable results in this regard.

Author keywords
Connexin
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