Ninj1: a novel regulator for microenvironment

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Ninjurin1 (Ninj1) mediates hyaloid vessels regression through increaseing the cell death of the vascular endothelial cells and also is involved in the multiple sclerosis by upregulating the macrophages activity. However, the role and underlying molecular mechanism of Ninj1 in retinal vessel network formation has not yet been known. Here we studied the biological relevance of Ninj1 in retianl vascular system *in vivo* and *in vitro*.

We found that Ninj1 was mainly expressed in macrophages during retinal development. Silencing of the Ninj1 decreased the regression of hyaloid vessels, the expression of Ephrin B2, and the number and length of filopodia in tip cells, resulting in the delayed retinal vessels. Also, miR-125a-5p was identified as a key regulator of Ninj1 expression and decreased the Ninj1 expression, in which abolished the Ninj1-mediated proinflammatory gene expressions and the adhesion of macrophages to the extracellular matrix. Finally, miR-125a-5p suppressed inhibited the entry of

Ninj1-expressing macrophages into the LPS-induced retinal inflammation and the streptozotocin -induced diabetic retinopathy.

Therefore, this study provides new insights of action of Ninj1 in the treatment of diabetic retinopathy and macular degeneration by investigating its biological functions and underlying molecular mechanisms which is involved in the formation of retinal blood vessel network.