Role of the FPR2 receptor in uveitis

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Uveitis is an inflammation of the uveal tract including the iris, ciliary body, and choroid. This disease can be idiopathic or associated with infectious and systemic disordersand can be classified anatomically into either anterior, intermediate and posterior or panuveitis, and as acute or chronic disease, depending on whether it lasts more or less than 3 months in duration [1]. The inflammation may cause a permanent damage in various ocular tissues with visual impairment for macular edema, optic nerve dysfunction, vitreous opacificationand cataract formation [2]. Although, the exact pathogenesis of uveitis is not clearly described, it is well known that the mediators of immune-inflammatory responses are responsible of it [3]. Recently, it has been demonstrated by Rossi et al., (2012) a possibility to quench the immune-inflammatory response of uveitis through the stimulation of the formyl peptide receptor type 2 (ALX/FPR2). This receptor, ubiquitously present within the eye structure, mediates the resolution of the eye inflammation if conveniently stimulated. Indeed, FPR2 receptor stimulated with intravitreal administration of the resolvin D1 (RvD1) ameliorates the immuno-inflammatory profile of the eye by modulating the local levels of: (i) myeloperoxidase activity (MPO) in aqueous humor (AqH); (ii) neutrophils activation; and iii) the cytokines release. An effect accompanied by changes in four important determinants of the immune-inflammatory response within the eye: i) the B and T lymphocytes; ii) the miRNAs pattern; iii) the ubiquitin-proteasome system (UPS); and iv) the M1/M2 macrophage phenotype (Rossi S. et al., 2015). Resolvin D1 (RvD1), is a potent mediator that promotes the resolution of the inflammatory response back to a non inflamed state (Thrimawithana et al., 2011) and plays anti-inflammatory role in endotoxin-induced uveitis through action on ALX/FPR2 receptor.

References

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