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Modulation of the LC3B and p62 expression in conjunctival fibroblasts by pro-inflammatory macrophages

Elena Mattiuzzo[1], Elena Tarricone[1], Andrea Leonardi[2], Antonino Di Stefano[3] and Paola Brun[1]

[1] University of Padova, Department of Molecular Medicine, Padova, Italia
[2] University of Padova, Department of Neuroscience, Ophthalmology Unit, Padova, Italia
[3] Fondazione S. Maugeri, IRCCS, Istituto Scientifico di Veruno, Veruno, Italia

Autophagy is a key regulatory process involved in many biological aspects, including cell survival, tissue regeneration and homeostasis. Recent studies demonstrated that de-regulated autophagy is implicated in some inflammatory diseases. Previous data obtained in our laboratory demonstrated that some autophagic markers such as LC3B, cathpesin D, Beclin-1 are over-expressed in a severe inflammatory disease such as vernal keratoconjunctivitis (VKC).

In the present study VKC conjunctival cell cultures were exposed to the inflammatory media of activated U937 monocytes to explore the role of inflammatory factors in the induction of autophagy.

Macrophage differentiation of U937 was induced by LPS and PMA and then incubated with fresh DMEM and 10%FBS to produce the conditioned medium. qPCR analysis of the activated cells revealed that IL1beta, TNFalfa were overexpressed. Primary conjunctival cell cultures were then treated with the inflammatory medium conditioned by activated U937and analysed for expression of some autophagic markers at 4, 10 and 24 hours after exposure. qPCR results demonstrated that LC3B, Beclin-1, LAMP1 and p62 increased from 4 to 24 hours. Western blotting analysis revealed cleavage and lipidation of LC3B quantified as an increased LC3BII/LC3BI ratio.

In conclusion, our data demonstrated that the environment created by inflammatory macrophages enahances autophagy in VKC conjunctival fibroblasts.

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