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| Proteolytic processing of VE-cadherin during thrombin treatment**Konstantina Tasioula** 1#, **Panagiotis Karras** 1, **Violetta Maltabe** 1,2 and **Panos Kouklis** 1,2 \*1 Laboratory of Biology, School of Medicine, University of Ioannina, Ioannina, Greece2 Department of Biomedical Research, Institute of Molecular Biology & Biotechnology, FORTH, University Campus, Ioannina, Greece# Presenting author: Konstantina Tasioula, email: k.tasioula@uoi.gr\* Corresponding author: Panos Kouklis, email: p.kouklis@uoi.gr |

abstract

The role of ectodomain shedding in transmembrane proteins has been studied in basic cellular processes such as adhesion, transmigration, proliferation, and differentiation. Biological factors, such as inflammatory mediators, influence the endothelial permeability and induce the activation of proteases that cause ectodomain shedding in membrane proteins. This phenomenon has been observed in VE-cadherin, the major protein of endothelial adherens junctions, pivotal to endothelial permeability and barrier function. Recent studies associate shedding of membrane proteins with development of diseases (Blaise et al. 2015).

Our aim is the detection and the quantitation of soluble VE-cadherin caused by ectodomain shedding after endothelial cells treatment with thrombin, a pro-inflammatory factor. We analyzed the effect of thrombin: a) in VE-cadherin shedding in endothelial cells and b) in VE-cadherin’s cytoplasmic domain integrity. Both processes might modulate endothelial cell-cell adhesion and endothelial barrier function (Minami, Sugiyama et al. 2004).

To study the role of VE- cadherin’s extracellular domain in cell adhesion, we produced monoclonal antibodies specifically to this region of human VE-cadherin. Using purified monoclonals we performed competition studies to identify epitopes involved in homophilic binding between VE-cadherin molecules. We identify functional monoclonals interfering with endothelial cell-cell adhesion.

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