Neuro-degenerative Diseases

Mechanisms, Clinical Strategies, and Promising Treatments of Neurodegenerative Diseases

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Abstracts

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MECHANISMS OF BETA-AMYLOID IMPAIRMENT OF ERYTHROCYTE FUNCTION: A ROLE FOR PKC ALFA AND CASPASE 3

C. Carelli-Alinovi[1], M. Girasole[2], S. Dinarelli[2], B. Sampaolese[3], F. Misiti[4]
[1]Biochemistry and Clinical Biochemistry Institute, Catholic University School of Medicine, Rome, Italy
[2]Istituto di Struttura della Materia (ISM), CNR, Rome, Italy
[3]Istituto di Chimica del Riconoscimento Molecolare (ICRM), CNR, Rome, Italy
[4]Human Social and Health Department, University of Cassino and SL, Cassino, Italy

Our attention is focused on the study of a new model based on the red blood cell (RBC) and on its interaction with Aß. RBCs are highly deformable to assist blood flow in the microcirculation, and in this context NO was proposed to be a regulatory factor of RBC mechanical properties since inhibitors of endogenous NO synthesis induces decreased erythrocyte deformability. For this reasons abnormalities in RBCs could contribute to AD by obstructing oxygen delivery to brain causing hypoxia. In our work, firstly we will focus on the morphology and nano-properties of RBC's membrane (i.e. roughness) by AFM (i.e. Atomic force microscopy), following to soluble Aß peptides exposure at different times, in order to characterize specific alterations induced by Aß. Secondly, considering that RBC membrane contains, among blood elements, higher acetylcholinesterase (AChE) levels, we can assume that there is a mechanism similar to the one which occurs at the neuronal level leading to an increase of Aß toxicity mediated by the binding with erythrocytic AChE. Since mechanical properties of RBC membrane are regulated by a number of molecular components of signalling and/or regulatory pathways, of these, particular interest has been addressed toward protein band 3, protein kinase C isoenzymes (PKC), endothelial nitric oxide synthase (eNOS) and caspase 3, due to their possible roles in the modulation of erythrocyte morphology, deformability and metabolic functions.

References:
Carelli-Alinovi C, et al. Clin Hemorheol Microcirc. 2014;
Misiti F et al.. Cell Biochem Funct. 2012