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ADAM10 gene expression does not differ in Alzheimer's disease

Patricia Regina Manzine¹, Elena Marcelo², Monica DiLuca² and Marcia Regina Cominetti¹

¹Federal University of Santa Catarina (UFSCar), Brazil

²University of Milan (UNIMI), Italy

Dementia from Alzheimer's disease (AD) is the most common type of dementia, accounting for approximately 60% of the cases. Regarding the immense financial, physical and emotional burden of AD, there is a pursuit to find sensitive and specific biomarkers for this disease. Since the brain histological hallmarks of the disease are amyloid- β 1-42 (A β 1-42), total Tau (T-Tau) and Tau phosphorylated (P-Tau), these molecules have been extensively studied. A β peptide is proteolytic fragment of APP released by sequential cleavages via β and γ -secretases. In healthy subjects the predominant route of APP processing consists of successive cleavages by α and γ -secretase. ADAM10 is the most important α -secretase involved in cleavage of APP. Previous studies have demonstrated that protein levels of platelet ADAM10 are reduced in AD patients. In this context, the aim of this study was to verify the total blood and platelet ADAM10 gene expression in elderly patients with AD dementia and to compare with mild cognitive impairment (MCI) and healthy elderly subjects. Blood from AD patients, MCI and normal controls was collected and analyzed by RT-qPCR techniques, using standardized primers for ADAM10 and to endogenous controls. One-way analysis of variance followed by Bonferroni as a post hoc comparison and Mann-Whitney U followed by multiple comparison (Kruskal-Wallis and Dunn's Multiple Comparison Tests) tests were performed. It was observed no differences between total blood and platelets ADAM10 gene expression in AD patients compared with MCI or controls patients. Samples for total blood were divided according to CDR and the results were similar, with no difference in ADAM10 gene expression between patients (CDR1, CDR2 or CDR3) and controls (CDR0). In this way, the decrease of ADAM10 protein levels in platelets from AD patients is not caused by a reduction in platelets or total blood ADAM10 mRNA levels. Further studies must be performed in order to investigate the influence of other molecules in this pathway, as micro RNAs, for instance. This would contribute to understand the pathophysiological mechanisms of AD.

mcominetti@ufscar.br