

International Conference on
**ALZHEIMERS, DEMENTIA AND RELATED
NEURODEGENERATIVE DISEASES**
December 03-04, 2018 Madrid, Spain

Interactions of 17 β -hydroxysteroid dehydrogenase type 10 and amyloid β 1-42 in Alzheimer's disease

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Statement of the problem: It is suggested that interactions of multifunctional mitochondrial protein 17 β -hydroxysteroid dehydrogenase type 10 (17 β -HSD10) and amyloid β (A β) peptides are involved in the pathogenesis of Alzheimer's Disease (AD). In our previous research, we evaluated total levels of 17 β -HSD10 and levels of 17 β -HSD10 – A β complexes as prospective biomarkers for AD. In this study, we tried to evaluate the levels of 17 β -HSD10 – A β 1-42 with the aim to find the better biomarker for AD than those available as yet.

Methodology: We detected complexes in cerebrospinal fluid of totally 212 subjects (patients with AD, mild cognitive impairment due to AD, mild cognitive impairment - others, fronto-temporal dementia, amyotrophic lateral sclerosis, vascular dementia, subjective memory complaints and controls by means of sandwich ELISA (Enzyme-Linked Immunosorbent Assay)).

Results: Significant decreases in the levels of complexes in MCI-AD and AD people were found when compared to healthy controls. A comparison with levels of A β 1-42, tau and phospho-tau revealed links especially to A β 1-42. However, the specificity and sensitivity were not here very high.

Conclusions: 17 β -HSD10 – A β 1-42 complexes in CSF (Cerebrospinal fluid) do not seem to be the better biomarker of AD than A β 1-42 itself, because the virtues and disadvantages are quite similar (high sensitivity but relatively low specificity towards AD differential diagnosis).

Acknowledgements: The study was supported by GA CR P-304-12G069

Biography

Jan Ricny is currently working in Department of Neurobiology at National Institute of Mental Health, Czech Republic. His research interest includes biochemistry of nervous system and its pathological conditions, cholinergic neurochemistry.

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