Confirmation of Registration

February 13 , 2024

Dear Klytaimnistra Giannaki,

We hereby confirm your registration for the ESN-HSN Meeting 2025. See you soon in Naxos, Greece!

Should you have any questions or need further assistance, please do not hesitate to contact us:

Secretariat, ESN-HSN Naxos 2025 Meeting
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ANALYSIS OF DIFFERENTIAL EXPRESSION OF GENES IN CELLULAR MODELS FOR THE STUDY OF ALZHEIMER'S DISEASE

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Alzheimer's disease (AD) is a neurodegenerative disorder, with both familial and sporadic incidence. Mutations in the genes encoding for the Amyloid Precursor Protein (APP), and Presenilin 1 (PSEN1), which is part of the γ-secretase complex that cleaves APP and releases the toxic Aβ peptide, cause early onset AD. The physiological role of these proteins is however not completely elucidated.

In order to determine the functions and processes of these proteins, we established cells lines, knockout (KO) for Amyloid Precursor Protein (APPKO) and Presenilin 1 (PSEN1KO), and alpha-synuclein (SNCAKO) as a specificity control for AD related changes. The KOs were obtained using the CRISPR Cas9 system with 2 single-guide RNAs, and validated by PCR and Western Blot.

Based on our hypothesis that mutations in the APP and PSEN1 genes lead to a disturbance in neurotrophic processes and in the response to neurotoxic insults, we examined the response of these cells to H_2O_2 (oxidative stress) and $CoCl_2$ (chemical hypoxia) using the MTT assay. Our results show that our cells were resistant to these treatments.

KEGG and GO analysis after RNA sequencing showed that the genes that have changed their expression in the APPKO and PSEN1KO cell lines but not in the SNCAKO cell line are involved mainly in brain development processes. We therefore hypothesise that early on in development the mutations may trigger the disease processes and we propose that some of the differentially expressed genes may be potential biomarkers.