LONGDOM PUBLISHING

Journal of diabetology

ISSN: 2329-888X

2020

Vol.4 No

Title: Mitochondrial activity of thapsigargin treated SH-SY5Y neuroblastoma cells.

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Abstract

Anew Mitochondrial dysfunction and endoplasmic reticulum stress (ERS) are often implicated in the development and progression of neurodegenerative diseases. Examination of mitochondrial respiration of the cellular models could represent a powerful tool for evaluation of mitochondrial functions in health and disease. In this study, we examined the effect of thapsigargin-induced ERS on relative cell viability and mitochondrial respiration of SH-SY5Y cells that are widely used as in vitro model for the study of neurodegeneration associated with Parkinson's disease.

The relative viability of the cells was assessed with MTT assay at 24, 48, or 72 h after the treatment with thapsigargin. The cells were further treated with nonlethal 800 nM thapsigargin for 6, 16, and 24 hours and mitochondrial respiration was determined by O2k-FluoRespirometer (Oroboros, AT) in triplicated experiments using 2 – 3 million cells per chamber. Coupling control protocol was used for a measurement of intact cells respiration in different states including ROUTINE, LEAK and ET-capacity. For data analysis Origin Pro 9 (One way ANOVA, Tukey test) and MS Excel were used.

Treatment of the SH-SY5Y cells with thapsigargin revealed a concentration- and time-dependent reduction of the relative cell viability. After 16 and 24 hours of treatment with thapsigargin we detected significantly decreased ROUTINE respiration but not significant changes of respiratory reserve. We also observed statistically significant changes ET-capacity of treated cells in coupled with NADH-linked respiration.

Our results indicate that ERS is associated with decreased mitochondrial respiration in time-dependent manner.

This work was supported APVV-16-0033, VEGA-1/0554/1

Speaker Biography:

Andrea Evinová has completed his PhD at the age of 28 years from Comenius University and up to now she is at postdoct position on the same University.

Abstract Citation:

Vascular Dementia Congress 2020, 16th world congress on vascular dementia and neurodegenerative disorders;- May 18-19,2020

16th world congress on vascular dementia and neurodegenerative disorders; - May 18, 2020.

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