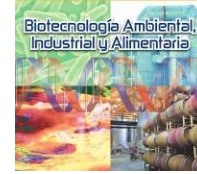


Poster



Evaluation of the effects of a hydrogen sulfide donor on neural plasticity

Conesa-Bakkali, Ryan (1), Vega-Blanco, Ángela (1), González-Morán, Daniel (1), El Kharoubi-Zamudio, Naym (1), Sola-García, Alejandro (1), Cáliz-Molina, María Ángeles (1), López-Fernández, Raúl (1), Martín-Montalvo, Alejandro (1), Espadas, Isabel (1)

(1) Department of Integrative Pathophysiology and Therapies, Andalusian Center for Molecular Biology and Regenerative Medicine (CABIMER)-CSIC-US-UPO, Seville, Spain

Tutor académico: Juan Rigoberto Tejedo Huamán. Department of Molecular Biology and Biochemical Engineering, University Pablo de Olavide, Seville, Spain.

Keywords: neural plasticity, hydrogen sulfide

ABSTRACT

The aging brain can exhibit significant modifications related with a progressive atrophy. Previous studies have shown that this atrophy may result from a combination of dendritic regression and neuronal death (1). Age-related memory and cognitive decline have been shown to coincide frequently with morphological changes which affect the neural plasticity and number of dendritic spines in the brains of both humans and animals (2). Furthermore, many neuropathologic conditions and neurodegenerative diseases exhibit abnormalities in dendritic tree structure. Animal studies have shown that even mild prolonged stress has been observed to induce the shrinkage of dendritic fields and the loss of dendritic spines (3).

Recent evidence suggest that H₂S is a gasotransmitter with neuroprotective properties. In addition, a few sulfur donors have shown beneficial therapeutic effects in experimental models of neurodegenerative diseases (4). Moreover, previous research in our lab suggests that a pharmacological treatment aimed at increasing intracellular H₂S improves physical and metabolic health in mice. Nonetheless, the specific properties of these compounds maintaining neuron homeostasis and plasticity remain unknown.

Here we aim to investigate whether modulation of intracellular H₂S by a pharmacological intervention can improve neuronal plasticity in terms of morphological changes at the level of dendritic arborization and dendritic spine density. To this purpose, we will perform analyses in murine primary neuron cultures that will be treated with increasing concentrations of drug “δ”. Experimental conditions will be: untreated (0, vehicle solution), 10 μM, 50 μM, and 100 μM. Cells will be maintained for 12-14 days in culture, and will be treated with compound “δ” for 48 hours. Then cells will be fixed and MAP2 immunocytochemistry analyses will be performed. Photos will be taken under a fluorescence microscope and analyzed using software ImageJ to determine the percentage of arborized area and the dendritic spine density. The results will provide us with an insight into the potential of drug “δ” as a neuroprotective agent to prevent age-related loss of neuroplasticity.

REFERENCES

- (1) Mattson, M. P., & Arumugam, T. V. (2018). Hallmarks of Brain Aging: Adaptive and Pathological Modification by Metabolic States. *Cell metabolism*, 27(6), 1176–1199. <https://doi.org/10.1016/j.cmet.2018.05.011>
- (2) Sikora, E., Bielak-Zmijewska, A., Dudkowska, M., Krzystyniak, A., Mosieniak, G., Wesierska, M., & Włodarczyk, J. (2021). Cellular Senescence in Brain Aging. *Frontiers in aging neuroscience*, 13, 646924. <https://doi.org/10.3389/fnagi.2021.646924>
- (3) Urbanska, M., Blazejczyk, M., & Jaworski, J. (2008). Molecular basis of dendritic arborization. *Acta neurobiologiae experimentalis*, 68(2), 264–288.
- (4) Nagpure, B. V., & Bian, J. S. (2015). Brain, Learning, and Memory: Role of H₂S in Neurodegenerative Diseases. *Handbook of experimental pharmacology*, 230, 193–215. https://doi.org/10.1007/978-3-319-18144-8_10