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+1-346-348-1205

TITLE: Modeling the microbiota-gut-brain axis role in age-related neurological disorders with a galectin-4-deficient mouse strain

Name: María Elvira Brocca[1], Arancha Mora-Rubio[1], Elena Alonso-Calviño[2], Elena Fernández-López[2], Juan Aguilar[2], Alonso Higuero[1] & José Abad-Rodríguez[1]
Affiliation: [1]Membrane Biology and Axonal Repair Laboratory and [2]Experimental Neurophysiology Laboratory, Hospital Nacional de Parapléjicos (SESCAM), Finca La Peraleda s/n, 45071, Toledo.
Country: Spain
Email ID: amorarubio@externas.sescam.jccm.es

ABSTRACT (upto300 words)

The role of the microbiota-gut-brain axis (MGBa) in neuropsychiatric age-related disorders has gained relevance during recent years. The microbiota regulates the immune and metabolic response of the host and its imbalance (dysbiosis), associated to aging, has been linked to the development of these pathologies. However, the molecular mechanisms underlying such a link remain obscure. Most animal models currently used for these studies do not reflect the clinical onset and development of these disorders, as they are based on the acute and almost complete ablation of the microbiota (germ-free mice or massive antibiotic treatment), harming the animal's general health status and favoring the proliferation of opportunistic microorganisms. In contrast, age-associated dysbiosis and its effects are chronic processes caused by different factors (diet, stress, genetics or aging) that induce a gradual evolution of neurological disorders.

Here we propose the Lgals4-KO mice strain as a suited model for the study of MGBa role in age-related neuropsychiatric disorders. These mice are deficient for Galectin-4 (Gal-4), an intestinal carbohydrate-binding protein that specifically recognizes and eliminates pathogenic bacteria. They have an altered composition of their microbiota and gut gene expression already young age. Neurologically, they show a reduced spine density on both apical and basal dendrites of CA1 hippocampal neurons, and a diminished phosphorylation of the GluA1 subunit of the AMPA receptor upon chemically-induced long term potentiation (LTP) *in vitro*. In good agreement, Lgals4-KO mice display an impaired LTP establishment after high frequency stimulation in CA3-CA1 synapses *in vivo*, consistent with a deficit in working memory formation detected in Y-maze forced alternation tests. We conclude that Lgals4-KO mice best mimic the chronic aspect of neurological pathologies derived from age-related gut dysbiosis, and can be key to unravel the molecular mechanisms linking altered MGBa with these disorders.

BIOGRAPHY (upto200words)

Arancha Mora-Rubio finished a Biochemistry degree in 2017 at University of Castilla-La Mancha (UCLM) and a Master's degree in Translational Biomedicine in 2018 at University of Córdoba (UCO), Spain. Currently, she is a pre-doctoral fellow in the UCLM Neuroscience program at the Membrane Biology and Axonal Repair Lab (Hospital Nacional de Parapléjicos) under the direction of Dr. José Abad-Rodríguez.

Presenter Name: Arancha Mora-Rubio.
Mode of Presentation: Poster.
Contact number: +34 925 39 68 32

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