Absence of LPA1 receptor results in altered pattern of limbic activation after tail suspension test

Moreno-Fernández, Román D¹; Gómez-Salas, Francisco J¹; Pérez-Martín Margarita²; Rosell-Valle, Cristina¹; Castilla-Ortega, Estela³; García-Fernández, María I⁴; Chun, Jerold⁵; Rodríguez de Fonseca, Fernando³; Estivill-Torrús, Guillermo⁶; Santín, Luis J¹; Pedraza, Carmen¹

*Corresponding author: mdpedraza@uma.es

Stress serves as an adaptive mechanism and helps organisms to cope with life-threatening situations. However, individual vulnerability to stress and dysregulation of this system may precipitate stress-related disorders such as depression. The neurobiological circuitry in charge of dealing with stressors has been widely studied in animal models. Recently our group has demonstrated a role for lysophosphatidic acid (LPA) through the LPA1 receptor in vulnerability to stress, in particular the lack of this receptor relates to robust decrease of adult hippocampal neurogenesis and induction of anxious and depressive states. Nevertheless, the specific abnormalities in the limbic circuit in reaction to stress remains unclear. The aim of this study is to examine the differences in the brain activation pattern in the presence or absence of LPA1 receptor after acute stress.

For this purpose, we have studied the response of maLPA1-null male mice and normal wild type mice to an intense stressor: Tail Suspension Test. Activation induced by behaviour of brain regions involved in mood regulation was analysed by stereological quantification of c-Fos immunoreactive positive cells. We also conducted multidimensional scaling analysis in order to unravel coativation between structures.

Our results revealed hyperactivity of stress-related structures such as amygdala and paraventricular nucleus of the hypothalamus in the knockout model and different patterns of coactivation in both genotypes using a multidimensional map.

This data provides further evidence to the engagement of the LPA1 receptors in stress regulation and sheds light on different neural pathways under normal and vulnerability conditions that can lead to mood disorders.

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¹Departamento de Psicobiología y Metodología de las Ciencias del Comportamiento, Instituto de Investigación Biomédica de Málaga (IBIMA), Universidad de Málaga, Malaga, Spain

²Departamento de Biología Celular Genética y Fisiología, IBIMA, Universidad de Málaga, Malaga, Spain

³ Unidad de Gestión Clínica de Salud Mental, IBIMA, Hospital Regional Universitario de Málaga, Malaga, Spain

⁴ Departamento de Fisiología y Medicina Deportiva, IBIMA, Universidad de Málaga, Malaga, Spain

⁵ Department of Molecular and Cellular Neuroscience, Dorris Neuroscience Center, The Scripps Research Institute, La Jolla, USA

⁶ Unidad de Gestión Clínica Intercentros de Neurociencias, IBIMA, Hospitales Universitarios Regional de Málaga y Virgen de la Victoria, Málaga, Spain