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ABSTRACT BOOK

ORAL COMUNICATIONS **ESSION 7**

THE LIMBIC BRAIN UNDER STRESS: A ROLE FOR THE LPA1 RECEPTOR

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Adverse events can impact brain structure and function and are considered primary sources of risk for depression, anxiety, and other psychiatric disorders. In this sense, the neurobiological circuitry in charge of dealing with stressors has been widely studied in animal models. Our group has demonstrated a role for lysophosphatidic acid (LPA) through the LPA1-receptor in controlling anxious and depressive states, owing to aggravation of the detrimental consequences of stress in the brain. Indeed, our group has recently proposed the variant maLPA1-null mice, i.e. mice lacking the LPA1 receptor, as an endophenotype for anxious depression. In addition, we have previously reported hyperactivation of key stress-related brain areas after stress, such as basolateral amygdala.

Here, we seek to further examine the engagement of the LPA1 receptor in the regulation of the limbic circuit following an acute stressor, tail suspension test, in wild-type and knockout animals. To that end, c-Fos expression was evaluated as a measure of functional activity in both basal and stress conditions, followed by interregional correlation matrices to establish the brain map of functional activation. Additionally, we observed whether one single dose of the antidepressant treatment with desipramine is able to normalize the functional brain map.

Results revealed that the absence of the LPA1 receptor induce an anomalous pattern of brain functional activity after TST, which was reverted by desipramine administration.

These results provide further insight to the involvement of the LPA1 receptor in stress regulation and shed light on divergent brain pathways under normal and vulnerability conditions that can be implicated in depressive symptoms. Finally, how this pattern might be reverted by antidepressant treatment can be useful for developing new pharmaceutical targets regarding the LPA1 receptor.

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