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Resúmenes de Comunicaciones Orales y Posters

Abstracts of Oral Communications & Posters

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increase similar to that of *COX2* mRNA. NF-κB IF revealed an increase in its nuclear localization. Regarding MAPKs, the only treatment that blocked COX2 protein expression was ERK1/2 selective inhibitor. SIRT1 specific inhibition did not change COX2 expression. These results show that RSV pretreatment caused harmful effects in hyperosmolar cultures of MDCK cells, affecting cell number and cell cycle and impeding cells to reach differentiated phenotype; in contrast with our hypothesis, RSV did not decrease COX2 expression but significantly upregulated the protein which was not active. Herein we report a possible nephrotoxic effect of Rsv.

LI-06

FPR2/ALX signaling and their lipid mediator pathways: pleiotropic roles in neurotoxicity.

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Neurotoxicity generated by several environmental factors has been widely associated with Parkinson's disease (PD). Human prolonged exposure to the pesticide Maneb (MB) has been reported as a triggering insult for dopaminergic neurodegeneration and the onset of PD. Even though this effect has been well documented in numerous epidemiological and research studies, little is yet known about the mechanisms underlying MB neurotoxicity in neuron-glia crosstalk. Based on our previous reports about the involvement of cyclooxygenases (COX) and lipoxygenases (LOX) in the neuronal response to MB toxicity (SAIB2021), our aim was to elucidate the role of these lipid mediators' pathways in neuron-glia communication. To study the possible alterations in neuron-glia crosstalk caused by MB exposure, dopaminergic N27 cells were exposed to astrocyte (C6 cell line) secretome and vice versa. Astrocytes' secretome showed to be neuroprotective against MB, whereas neurons secreted glial proliferative factors after pesticide exposure. Neither COX-2 nor CYP450 pharmacological inhibition were able to revert the effect of secretomes on their respective acceptor cells. In contrast, the inhibition of LOX-15, enzyme responsible for the generation of anti-inflammatory lipid mediators, abolished the glial proliferative effect of neuronal secretome during MB toxicity. In addition, the neuroprotective effect of astrocyte-derived secretome was blocked.

Next, we evaluated the role of FPR2/ALX receptor, whose main ligands are lipid mediators associated with resolution. The antagonist of FPR2/ALX, Quin-C7, blocked the effect of the astrocytic secretome on

neuronal survival upon MB challenge. In agreement, FPR2/ALX activation by a specific agonist enhanced the neuroprotective effect of the astrocytic secretome.

To determine the role of FPR2/ALX downstream signaling, cells were incubated with PI3K and ERK1/2 pharmacological inhibitors, After MB exposure, neuronal and astrocytic viability was nondependent of ERK1/2 activation. On the contrary, the blockage of PI3K showed to increase pesticide-induced cell death. Moreover, ERK1/2 phosphorylation was diminished by MB in both cell types. Interestingly, we found that the astrocyte proliferation caused by the secretome derived from MB-exposed neurons was mediated by ERK1/2 activation. Our results suggest that FPR2/ALX signaling and their lipid ligands are involved in the neuronal-glial crosstalk during MB exposure. These findings pay the way for interventions aimed at enhancing the resolution response during pesticide-induced neurotoxicity.

Signal Transduction

ST-01

2-arachydonoyl glycerol (2-AG) drives TRPV-dependent sensory signals that increase the intracellular cholesterol trafficking in Caenorhabditis elegans

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Cholesterol is an essential lipid constituent of eukaryotic cell membranes. Furthermore, its derivated metabolites have important biological roles as signalling molecules. Because of its relevance,