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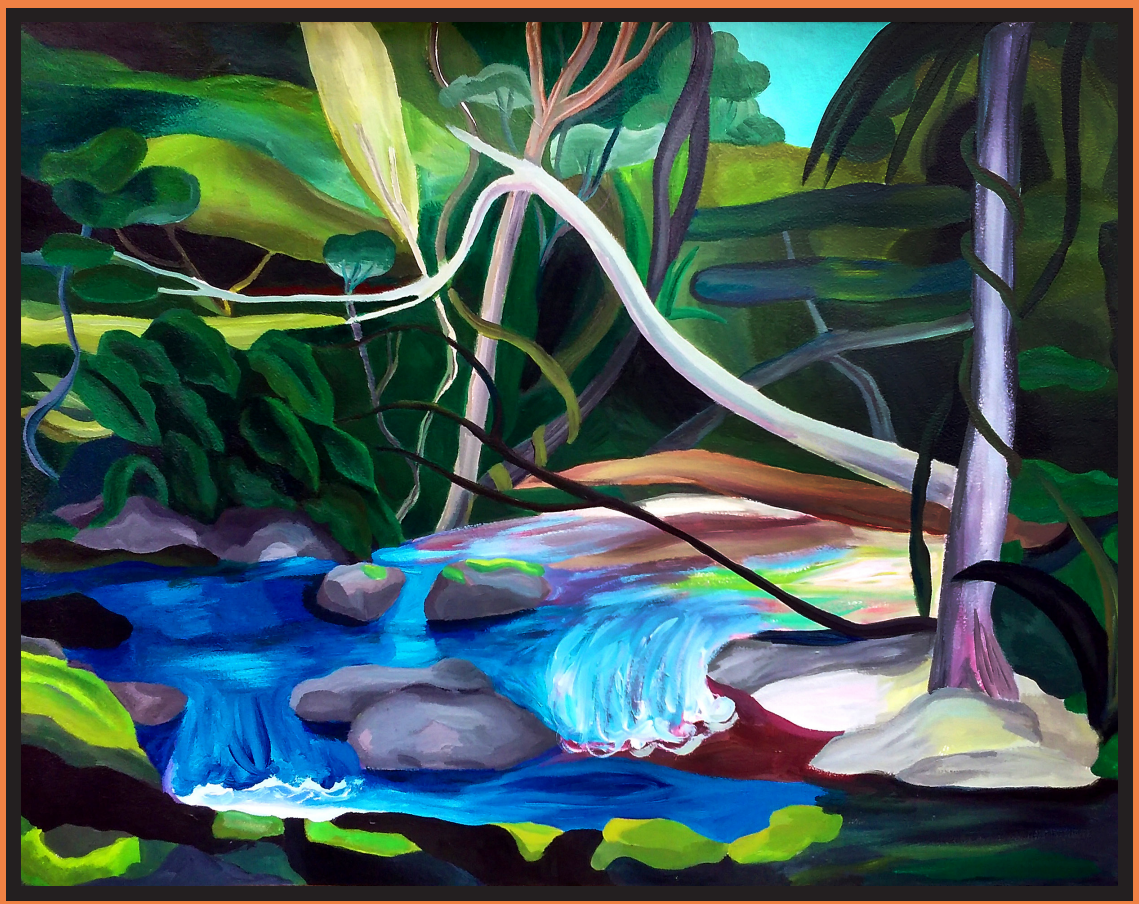

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mmHg n= 3 all values are at age of 18 weeks old). L94C showed potential in preventing ventricular mass increase and hypertrophy since LVMI and DPW values did not differ significantly from the normotensive group. Also, systolic function indicators MV% and FS improved with L94C. These results postulate L94C and L96B as promising GRK2 inhibitors with plausible application in clinical management of cardiovascular diseases.

TUMOR MICROENVIRONMENT IN BREAST CANCER: ANALYSIS AND CHARACTERIZATION OF ADIPOSE TISSUE

Priscila Ayelén Pagnotta[1,2], Tomás Gonzalez Garello[3], Rubén Dreszman[4], María Luján Crosbie[5], Natalia Santiso[5], Anabela Ursino[5], Celeste Frascarolli[5], Alicia Amato[5], Juan Carlos Calvo[1] and Judith Toneatto[1]

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Adipose tissue (AT) exerts influence on cancer progression. Our goal was to investigate the modulation of tumor-associated adipocytes (TAA), located within breast AT explants from cancer patients. These samples were gathered from two sites: immediate tumor adjacency (AC) and a 2 cm distance (BC), alongside normal controls. Human AT sections were paraffin-embedded and examined using immunofluorescence, quantified by automated intensity assessment. Tissue lysates underwent Western Blot analysis. Variance assessment utilized linear regression models with covariates, signifying $p<0.05$. In AC, perilipin 1, indicating lipid droplet interaction, surged notably in patients with: IIB over IIA/I, grade 1 over grades 2/3, and pre-menopause vs. post-menopause (obese patients). Lipases HSL and ATGL elevated within BC versus AC (lobular cancer), smaller tumors, and pre-menopause over post-menopause. HSL surged with grade 3 vs. grades 1/2, BC over AC (obese patients), and age advancement. Conversely, HSL dropped in obese vs. normal/overweight (invasive ductal cancer), AC region. ATGL surged in normal weight vs. overweight/obese, stage II vs. I, and grade 3 vs. grades 1/2.

UCP1, TBX1, mature adipocyte markers (FABP4, adiponectin, CAV-1) were constant; vimentin, CD44 rose in TAA. Glut4, LDH fell; MCT4, GAPDH hardly varied. MCT1 and browning markers (UCP1, TBX1) spiked solely in a specific subgroup. In sum, breast AT encounters metabolic shifts in cancer context, marked by lipid metabolic protein expression changes, adverse prognosis markers, and sporadic browning. Tumor traits and patient attributes drive these shifts, potentially fueling cancer advancement.

AN ORPHAN LIPID LIGAND ACTIVATES RESOLUTION PATHWAYS IN NEURON-GLIA CROSSTALK

Oriana Nicole Benzi Juncos[1,2], Natalia Paola Alza[1,3], José Cordero[4], Nelson Patricio Barrera[4], Gabriela Alejandra Salvador[1,2]

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Environmental neurotoxicants, such as Maneb (MB) and other dithiocarbamate pesticides, trigger chronic neuroinflammation probably due to defective resolution mechanisms, leading to neurodegeneration. The inflammation/resolution balance is governed by a plethora of specialized pro-resolving lipid mediators (SPM) that act as ligands of the GPCR receptor FPR2/ALX. SPM are mainly synthetized by lipoxygenases from arachidonic acid (AA) and docosahexaenoic acid (DHA). Thus, our aim was to study the resolution pathway modulated by FPR2/ALX in response to MB challenge in a context of neuro-glial communication. By metabolomics we detected significant changes in 11 metabolites in neurons and 27 metabolites in astrocytes as a response to MB treatment ($p<0.05$). In both cell types, phosphatidylcholine was reduced with a simultaneous increase in lysophosphatidylcholine. IPA software's Path Explorer, Connect and MAP functions revealed the upregulation of a secretory phospholipase A2, PLA2G2D. GC-MS fatty acid profile showed increased neuronal DHA content and decreased AA and DHA levels in astrocytes ($p<0.05$). In addition, increased phosphatidylcholine (DHA/16:0) content in neurons exposed to MB was confirmed by metabolomics. To evaluate resolution events under MB injury in neuron-glia crosstalk, cell-derived secretomes and their lipid extracts were used. Astrocyte secretome and its lipid extract were able to revert MB-induced neurotoxicity. This neuroprotective effect was abolished by blocking AA and DHA oxygenation as well as by the FPR2/ALX antagonist Quin-C7. Neurons secreted ERK1/2 -dependent glial proliferation signals, also inhibited by Quin-C7. The role of lipidome obtained from conditioned media in neuro-glia responses to MB injury confirmed the lipid nature of mediators involved in resolution.

Our results show that neurons and astrocytes secrete lipid ligands for FPR2/ALX -mediated resolution in response to MB toxicity.