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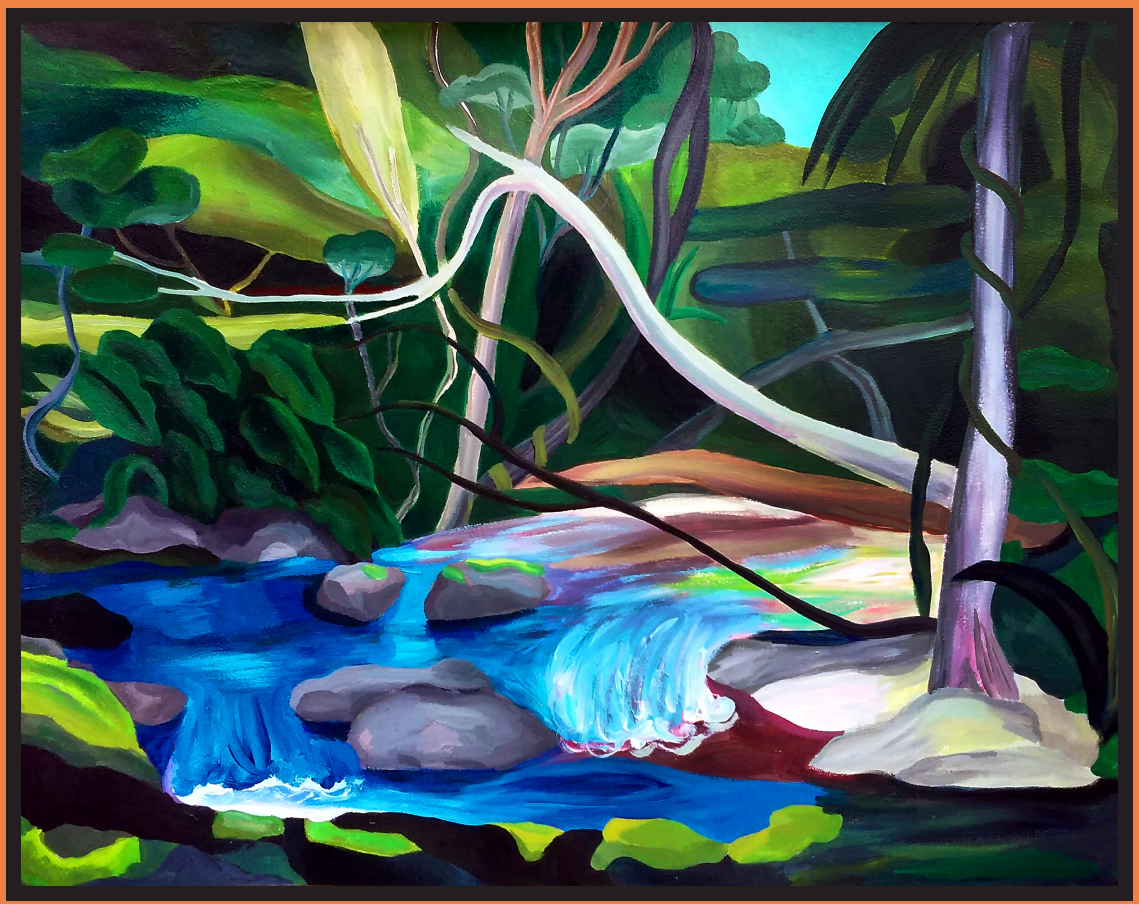

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TOR (MR) ANTAGONIST EPLERENONE IN EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS (EAE)

Guido Álvarez Quintero[1], Analía Lima[1], Paulina Roig[1], María Meyer[1], Alejandro F. De Nicola[1,2] and Laura I. Garay[1,2]
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There is growing evidence indicating that MR expression influences a wide variety of functions. In the context of the immune response, MR stimulation promotes proinflammatory responses and fibrosis. The present study explored if antagonism of the mineralocorticoid receptor reduces neuroinflammation in the spinal cord of mice with experimental autoimmune encephalomyelitis (EAE). Eplerenone (EPLE) (100mg/kg dissolved in 30% 2-hidroxypropyl-β-cyclodextrin) was administered i.p. daily from EAE induction (day 0) until sacrificed on day 17 post-induction. The mineralocorticoid receptor blocker (a) significantly decreased the inflammatory parameters TLR4, MYD88, IL-1β and iNOS mRNAs ;(b) attenuated HMGB1, NLRP3, TGF-B mRNAs, microglia and acquaporin4 immunoreaction reaction without modifying GFAP. Serum IL-1β was also decreased in the EAE+EPLE group. Moreover, EPLE treatment prevented demyelination and improved clinical signs. Interestingly, MR was decreased and GR remain unchanged in EAE mice while EPLE treatment restored MR expression, suggesting that a dysbalanced MR/GR associated with neuroinflammation. Thus, MR blockage with EPLE downregulated inflammation-related spinal cord pathology in the EAE mouse model of multiple sclerosis.

269. 147. **LONG-LASTING AMPHETAMINE EFFECTS OVER CENTRAL ANGIOTENSIN II RESPONSES INVOLVE AT_1-R**

María Josefina Piermarini[1], Osvaldo Martín Basmadjian[1], Victoria Belén Occhieppo[1], Gustavo Baiardi[2], Claudia Bregonzio[1].
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Angiotensin II (Ang II), a pleiotropic neuropeptide, plays a critical role in regulating the sympathetic and neuroendocrine systems through the activation of AT_1 receptors (AT_1-R). Studies on mammals have shown that administering Ang II through the intracerebroventricular (i.c.v) route increases water and sodium intake, as well as renal sodium excretion. Our group's previous findings have shown that AT_1-R are involved in behavioural and neurochemical sensitization induced by amphetamine (Amph) administration. Our current aim is to assess the functional and neurochemical response to Ang II, via the AT_1-R activation, in animals that have been previously exposed to Amph. Male Wistar rats (250-320g) were injected intraperitoneally with Amph (2.5mg/kg/day) or saline for 5 days, and implanted with i.c.v. cannulae. Twenty-one days after the last Amph administration, the animals received Ang II (400pmol) i.c.v. First group: the animals were tested in a free choice paradigm for sodium (2% NaCl) and water intake, and sacrificed for Fos immunoreactivity determinations. Second group: urine and plasma samples were collected for electrolytes and plasma renin activity determination. Third group, the animals were tested in the plus maze or the holeboard for anxiety and memory work evaluation, respectively. Previous Amph exposure altered the physiological and behavioral responses described for central Ang II administration. Remarkably, the AT_1-R blockade prevented most of these alterations but anxiety. Our results highlight that repeated Amph exposure attenuates the AT_1-R functionality in a long-lasting manner, modifying the brain Ang II-induced responses.

270. 183. **ACTION OF *Tessaria absinthioides* ON SPATIAL MEMORY AND OXIDATIVE STRESS IN RATS**

Héctor Coirini[1], María Sol Kruse[1], Alejandro Tapia[2], Gabriela Egly Feresin[2], Mariana Rey[1]
[1]Lab. Neurobiología, IBYME-CONICET; [2]Instituto de Biotecnología, Fac. de Ing.-UNSJ.

Tessaria absinthioides Gillies (Hook. & Arn.) DC. (Asteraceae; TA) is popularly used to improve health. We previously described the effects of TA on the glucose and lipid metabolism of rats subjected to a sucrose overconsumption during the juvenile stage. The aim of this work was to evaluate whether TA improves spatial memory and produces central antioxidant effects in the same animal model. Male rats (SD) received water (CON), 10% W/V sucrose (SAC) or a TA decoction10% W/V with sucrose (TESSAC) from PND21-PND61. The novel object location (NOL) test was performed in the PND 68. In the test, the animal explores two identical objects placed in a quadrant for 5 minutes. After 2 hours, the animal explores for 5 minutes the device where one of the objects was relocated. The exploration rate (ER) is determined as the time spent exploring the relocated object over the time spent exploring both objects. Malondialdehyde (MDA) concentration was quantified by TBARs in hole homogenates of hippocampus (HC), hypothalamus (HT) and cerebral cortex (CC) obtained at PND69. SAC showed lower ER than CON (38.15%; $p<0.01$), whereas TESSAC had higher ER than SAC (31.98%; $p<0.01$) and similar to CON. In HT, the levels of MDA were higher in SAC than CON (72.37%; $p<0.05$). However, TESSAC had lower levels of MDA than SAC (34.07%; $p<0.05$) and similar to CON. In HC, no differences were found between SAC and CON, but TESSAC had lower levels of MDA than SAC and CON (86.02% and 88.72% respectively; $p<0.01$). In CC, no differences were observed between the groups. These results with those previously reported indicate that TA co-administration is capable of preventing alterations produced by sucrose overconsumption during critical stages of development. The properties of TA could be attributable to its high content of flavonoids, diterpenes and polyphenols. More complementary studies are necessary to consider TA for new therapeutic opportunities.

271. 353. **MITOCHONDRIAL COMPLEX I: STABILIZATION AND ACTIVITY IN A RAT MODEL OF CEREBRAL AMYLOIDOSIS**

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Bioenergetic dysfunction has been suggested as an early event and a cause of synaptic and cognitive deficiency in Alzheimer's Disease (AD). Previous reports showed impairments in mitochondrial Complex I (CI) activity associated to brain amyloidosis, however the molecular basis underlying this disturbance was not deeply established. Here we evaluated in McGill-R-Thy1-APP transgenic (Tg) rat, a model of AD-like cerebral amyloidosis, if there is a time-course disorganization of CI (a multimeric protein) in Supercomplexes (SCs) and if this process affects CI activity. Mitochondria were isolated from hippocampus of young (3 month) and old (9-12 month) animals (n=3/group) and the organization and abundance of SCs analyzed by electrophoretic runs on native gels. CI functionality was assessed by in-gel activity. T-test was applied for statistical analysis. We found by Western-blot (WB) similar amounts of the individual mitochondrial complexes (CI, CII, CIII, CIV and CV) and no differences in the assembly of SC1 ($I+III_2$), SC2 ($I+III_2+IV$) and SC3 (I_2+III_2) between genotypes. However, a lower CI activity was detected in the aged Tg as compared to young animals. To address if aged-associated impaired CI activity was linked to decrements of its relevant sub-units we assessed the expression of NDUFB8 (hydrophobic arm), NDUFA9 (hinge) and NDUFS2 (catalytic core). Similar levels of NDUFB8 and NDUFS2, and reduced amounts of NDUFA9 were detected in aged Tg vs. WT rats. NDUFA9 is encoded by a nuclear gene and translocates from cytoplasm to mitochondria, therefore we assessed its cytoplasmic levels in both genotypes. We detected higher cytoplasmic amounts of preprotein-NDUFA9 in aged Tg. NDUFA9 is imported to mitochondria after conversion of preprotein to mature one. We speculate that in a setting of high amyloid β levels, the processing of NDUFA9 may be impaired precluding its translocation and