

**Cuyo  
Biology Society**

**XXXVII  
Scientific Meeting  
5-6 dec 2019 - San Luis**

**Subjects**



**Education**

**Environment**

<https://sbccuyo.org.ar>

organs. Wistar rats (N=20) were administered via oral 75 µg/kg/day of seleno-methionine (Se-Met) during 7, 14, and 21 days. After sacrificed, the liver, heart, and kidney samples were collected at different *postmortem* intervals PMI (0-1-3-6-12 h). Total selenium concentration in organs was determined by Inductively Coupled Plasma Mass Spectrometry (ICP-MS). The administration of Se-Met produced a significant increase of selenium concentration in the liver (65%,  $P<0.001$ ), heart (40%,  $P<0.01$ ), and kidney (45%,  $P<0.05$ ) respect to control. At 21 days, selenium showed significantly higher levels in kidney than control animals ( $3.75\pm 0.24$  vs.  $2.02\pm 0.37$  mg/kg,  $P<0.01$ ). Oxidative stress was measured by malondialdehyde (MDA) liquid chromatography. The MDA decreased significantly in the heart ( $0.21\pm 0.04$  vs.  $0.12\pm 0.02$  mmol/g) and the kidney ( $0.41\pm 0.02$  vs.  $0.24\pm 0.03$  mmol/g) at one-hour PMI ( $P<0.01$ ). Organs removed at different PMI intervals showed lower production of MDA compared to the control group ( $P<0.05$ ). Se-Met decreased significantly oxidative stress in transplant organs from 1 to 12 h PMI. The results suggest that selenium is a protective agent that improves transplant organs' survival and the outcome of transplant surgeries. Future studies will be focused on the specific activity of GPx and selenium in transplant organs.

#### A46

### LONG-TERM EFFECTS OF NEONATAL HYPOXIA ON ANXIETY-RELATED BEHAVIORS AND HORMONAL RESPONSE TO ACUTE STRESS

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Neonatal lesions in the brain have less severe effects than in adults due to the neuronal plasticity of the developing nervous system, although they can cause cognitive and behavioral sequelae. Previously, we found that neonatal hypoxia (NH) transiently affected the expression of proteins associated with synaptogenesis in certain brain areas. The intermingled neural circuits controlling both stress and anxiety suggest a strong relationship between stress experiences and anxiety in both healthy and pathological conditions. We evaluated the long-term effects of NH on anxiety parameters and in stress-induced hormone release in adult female (estrous day of rat cycle) and male rats. Sprague Dawley rats at 4 Post-Natal Day (PND) were exposed to an atmosphere of low oxygen level (6.5% O<sub>2</sub> and 93.5% N<sub>2</sub>) for 70 min. 4PND control pups were exposed to normal oxygen levels (Co) for 70 min. The humidity and temperature conditions were controlled. Pups were then returned with their mother until weaned, and then they were allowed to grow. At 3 months of age, both groups of rats were subjected to two tests, Elevated Plus Maze (EPM) to measure anxiety parameters and a stressful challenge to determine hormone response to acute stress (exposure to ether vapors for 2 min). EPM reflected an unconditional aversion to heights and open spaces, an anxiogenic behavior. The hormonal response to stress included the release of pituitary prolactin (PRL), adrenal progesterone (P<sub>4</sub>), and adrenal corticosterone (CORT). Blood samples were collected before and after 5 min of stress exposure for serum hormone determinations by RIA. In the EPM test, both female and male hypoxic rats increased the number of entries to the open arms (OA) and the time spent in the OA compared to Co ( $P<0.05$ ). The results obtained indicated an anxiolytic-related behavior induced by NH, that was higher in female than in male hypoxic rats. Basal levels (unstressed) of PRL and P<sub>4</sub> in NH rats remained similar to Co ones in both sexes. Only in female rats, NH increased the basal levels of CORT compared Co rats ( $P<0.05$ ). In female and male rats, the hormonal release of PRL, P<sub>4</sub>, and CORT induced by stress, were differentially affected by NH. Hypoxia attenuated the stress-induced PRL secretion in female rats ( $P<0.05$ ) while this response was blocked in males. The release of CORT by stress was blunted in both sexes by NH. The release of P<sub>4</sub> by stress was inhibited in NH female but it was preserved in male rats. In conclusion, the long-term effects of NH were influenced by sex. NH altered the anxiety levels and the hormonal response to stress in adulthood. The alterations caused by NH at the brain level could be influencing the appropriate response to situations of stress and anxiety in adulthood.

#### A47

### AMPHETAMINE PRENATAL EXPOSURE INFLUENCES SEX-DEPENDENT PITUITARY RECEPTORS EXPRESSION IN RESPONSE TO STRESS IN ADULT RATS

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Sex steroids modulate brain dopaminergic systems and influence neuroadaptive changes induced by prenatal exposure to amphetamine (PEA). Pituitary dopamine D2 receptor (D2R) and prolactin receptor (PRLR) are involved in the modulation of prolactin synthesis and secretion, and the endocrine milieu regulates prolactin in basal and stress conditions. Our objective was to evaluate the sex differences in pituitary D2R and long PRLR isoform expression following stress in adult PEA and control rats. Female Wistar rats were treated daily with amphetamine 2.5 mg/kg i.p./saline during days 15 to 21 of pregnancy. Their offspring were sexed, and the females were OVX at day 60 and 15 days later treated with estrogen/oil (E2; 2 x 5 µg/rat/24 h). At 75 days, male, OVX, and OVX+E2 rats were exposed to immobilization stress for 30 min. Blood and tissue samples were collected for corticosterone by RIA, and the pituitaries were obtained to determine D2R, PRLR (long) expression, and PRL content by real-time PCR and Western blots (WB). The data were analyzed using a two-way ANOVA. Our results showed that, in basal male control rats, corticosterone levels were lower than OVX+E2, and PEA blunted this response ( $*P<0.05$ ). No sex differences were observed in