ISSN 0327- 9545
ISSN 1667-5746 (online version)

Abstracts from the

IV JOINT MEETING OF THE BIOLOGY SOCIETIES OF ARGENTINA

(Cuarta Reunión Conjunta de Sociedades de Biología de la República Argentina)

XXXVII Annual Scientific Meeting of the Tucumán Biology Association
XXIII Annual Scientific Meeting of the Córdoba Biology Society
XXXVIII Annual Scientific Meeting of the Cuyo Biology Society
Argentine Biology Society
Rosario Biology Society
Chilean Society of Reproduction and Development

September 9–15, 2020
Online edition

The abstracts have been revised and evaluated by a Scientific Committee prior to publication

A256
LONG-TERM EFFECTS ON HIPPOCAMPAL PROBDNF FOLLOWING SUCROSE CONSUMPTION IN JUVENILE VERSUS ADULT RATS

Kruse MS, Rey M, Coirini H.
Instituto de Biología y Medicina Experimental-CONICET. E-mail: sol.kruse@conicet.gov.ar

Excessive consumption of sucrose in early stages of development has deleterious neurobiological and behavioral effects in adulthood. We previously reported difficulties in memory retrieval. Here, we examined the proBDNF expression in the ventral hippocampus (vHIP) and media prefrontal cortex (mPFC) of animals exposed to sucrose during youth (SY) or adulthood (SA) by Western blot. Two-way ANOVA showed significant differences among groups in the vHIP ($Fi(1,16) = 13.456$, $P = 0.003$). Animals SY showed a decrease pBDNF levels (Fisher's LSD post hoc test, $P = 0.035$) while animals SA showed a raise of these values (Fisher's LSD post hoc test, $P = 0.013$).When all animals were considered, the proBDNF levels correlated positively with the exploration ratio in two memory tasks T3 and T4 (one-way ANOVA, $FT3(1,19) = 5.470$, $P = 0.0334$, $r^2 = 0.268$; and $FT4(1,19) = 14.617$, $P = 0.0034$, $r^2 = 0.3076$) indicating that higher levels of proBDNF corresponds to better memory response. No differences in proBDNF levels were found in the mPFC (two-way ANOVA, $Fi(1,16) = 0.539$, $P = 0.4743$). Taken together, these results show that sucrose affects long-term BDNF expression in vHIP and these abnormalities are different depending on the age of exposure. In addition, it also demonstrates that animals exposed to unlimited consumption of sucrose during youth require higher levels of proBDNF to achieve the same memory response as a control animal in adulthood.

A257
CONSEQUENCES OF PIOGLITAZONE-RETINOIC ACID ADMINISTRATION ON DAILY RHYTHMS OF TNFα, IN AN EXPERIMENTAL MODEL OF ALZHEIMER'S DISEASE

Ledezma C[1], Coria Lucero C[1], Anzulovich A[1], Delgado S[1], Navigatore Fonzo L[1]
Laboratorio de Cronobiología, IMIBIO-SL, CONICET-UNSL, San Luis. E-mail: lnavigatore@unsl.edu.ar

Alzheimer's disease (AD) is an age-related neurodegenerative disorder. The neuronal dysfunction and cell death mechanisms that are commonly found in this disease are due to the production of high levels of cytokines, TNFα among them, the formation of amyloid plaques and the alteration of the circadian rhythms. Due to the etiology of AD, multi-target therapies could be more effective. Both PPARγ agonist and retinoids are good candidates for this approach, since they regulate a large number of keys genes and proteins in various pathways, including neurotransmission, Aβ, inflammation, neurogenesis and circadian synchronization, among others. Previously, we found that an intracerebroventricular injection of Aβ (1-42) modified the daily rhythms of TNFα and clock proteins in the rat prefrontal cortex. Taking into account those observations, the objective of this study was, to evaluate the effect of the PPARγ agonist, pioglitazone, along to the RXR ligand, retinoic acid, on the 24-h rhythms of Aβ, ApoE, and clock protein. Four-month-old males Holtzman rats were used in this study. Groups were defined as: (1) control, (2) Aβ-injected, (3) Aβ-injected treated with Pioglitazone-Retinoic Acid (Pio-RA). Rats were maintained under 12 h light:12 h dark conditions with food *ad libitum*. Aβ, ApoE, BMAL1, and RORα proteins levels were analyzed by immunoblotting in prefrontal cortex samples isolated every 6 h during a 24-h period. We found that the treatment of Pio-RA reestablished rhythmicity of clock and TNFα proteins and decreased Aβ levels in the rat prefrontal cortex. These findings could indicate that PPARg-RXR heterodimer might be a potential target for restoration of circadian rhythmicity in neurodegenerative disorders.

A258
BUILDING AN EXPERIMENTAL NUTRITIONAL MODEL OF OBESITY. EFFECTS OF HIGH FAT DIETS ON LIPID PROFILE AND SERUM ENZYMES ACTIVITY

Lopez M, Alfonso J, Navigatore Fonzo L, Anzulovich A
Laboratorio de Cronobiología, IMIBIO-SL, CONICET-UNSL. E-mail: anzulova@gmail.com

Obesity is the most common nutritional disorder and is associated with a cluster of chronic metabolic disorders such as dyslipidemia, atherosclerosis, and type 2 diabetes. As a part of an institutional project that studies Obesity as a base disease for the development of chronic age-associated diseases and the search for early biomarkers with predictive potential, one of our first main objectives is to establish a nutritional model of obesity in rat. Particularly, the objective of this work was to investigate the effects of high saturated fat diets on anthropometrical parameters, lipid profile, serum enzymatic activity of alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT), and glucose levels, in rats. For that, male Wistar rats weaned at 21 days of age were randomly separated and fed with a normocaloric (NC) diet containing 366 kcal from lipids/kg diet (control group) or one of two high saturated fat diets, one containing 1570 kcal from margarine/kg diet (HFM group) and other with 1698 kcal from pork fat/kg diet (HFP group), for 12 weeks. Rats were maintained under 12 h light:12 h dark and 22–24°C conditions, with food and water *ad libitum*, during the whole treatment period. Food consumption was recorded daily while animals' weight and body mass index (BMI) were registered weekly. After 12 weeks animals were euthanized, and blood samples were collected. Serum ALAT and ASAT enzymatic activity were determined by kinetic assays while glucose (G), triglycerides (TG), total cholesterol (TC), HDLc and [LDLc+VLDLc] were determined by colorimetric assays. Statistical differences between groups and throughout the treatment period were analyzed by two- or one-way ANOVA, depending on data, followed by Bonferroni *post-hoc* test, with $P < 0.05$ to confirm significant differences between groups and weeks. We observed HFM and HFP diets did not modify anthropometrical parameters nor serum glucose levels, during the whole treatment period, in comparison to the control group. However, interestingly, HFM and HFP significantly increased TG ($P < 0.01$ and $P < 0.05$, respectively), TC ($P < 0.001$ in both cases) and [LDLc + VLDLc] levels ($P < 0.001$ and $P < 0.01$, respectively) as well as ASAT activity ($P < 0.05$), in the rat serum. Our results also show decreased circulating HDLc levels in the HFP group in comparison to the NC group ($P < 0.05$). Thus, we can conclude that feeding rats with HF diets (~400–450% higher

fat's kcal in comparison to NC) during 12 weeks from weaning, induces early metabolic alterations; though, the treatment length, or the animals age, was not enough to generate a nutritional model of obesity.

A259
NAV1.8 RELATION WITH CHRONIC INFLAMMATORY PAIN ON AGING

Messina DN, Fóscolo MR, Acosta CG
Laboratorio de Neurobiología del Dolor, Instituto de Histología y Embriología de Cuyo. E-mail: diego.n.messina@gmail.com

Treatment of pathological pain (resulting from damage and inflammation of peripheral nerves and tissues innervated by them) is less effective in older adults. There is little research about the pain mechanisms involved, although there is evidence in rodents that nociceptors excitability differs between young and old ones. Nociception is mediated by primary nociceptive dorsal root ganglion (DRG) neurons, and pathological chronic pain is believed to arise from increased excitability in these nociceptors. This hyper excitability may be attributable to changes in the expression and regulation of voltage gated sodium channels (especially Nav1.7, 1.8 and 1.9). The aim of this work was to determine the expression pattern of Nav1.8 in primary sensory neurons of the DRG in young adult rats (aged 3 to 6 months) and compare it with aged rats (12 to 18 months) and correlate the expression pattern of this ion channel with the behavioral changes observed in a model of chronic pathological inflammatory pain. We quantitatively evaluated the expression of Nav1.8 by ABC/DAB immunohistochemistry in 7-µm serial cryostat sections of L4 and L5 DRGs from Wistar rats aged 3, 6, 12, and 18 months. We induced inflammation with a single intradermal injection of Complete Freund's Adjuvant solution (CFA) in 8 3-month-old and 12 14-month-old rats. We evaluated and followed during 120 days after CFA two types of pain: spontaneous pain, using the spontaneous foot lifting test (SFL) and evoked pain, which results in hypersensitivity to mechanical stimuli (mechanical hyperalgesia) using the von Frey test. Nav1.8 staining intensity in small neurons (area <400 µm^2) was lower in 3-month-old rats compared to 6-month-old rats (36.5 ± 0.9% *vs*. 49.9 ± 1.3%, $P <$ 0.0001), and it was similar when comparing 12 against 18 months (55.3 ± 1.4% *vs*. 55.1 ± 1.4%). There were no differences in staining intensity in medium neurons, while in large neurons, it was lower at 12 months compared to 18 months. On the other hand, the proportion of Nav1.8 positive neurons (intensity ≥40%) tended to increase with age, from 35% at 3 months to 69% at 18 months (P = 0.0368). We observed that aged rats showed a faster reversal of the SFL phenotype compared to young ones (21 *vs*. 28 days for young rats), although its intensity was higher to begin with. On the other hand, the reversal of mechanical hyperalgesia was slower in aged rats (49 *vs*. 21 days in young rats). In both groups, hypoesthesia manifested after 77 days. Based on these findings, lower Nav1.8 expression in young rats associates with lower intensity of SFL events along with faster reversal of mechanical hyperalgesia. We propose that a higher expression of Nav1.8 would be related to the persistence and intensity of pain in aged individuals.

A260
IN VITRO EFFECTS OF BPA, BP2 AND BP3 ON CELL PROLIFERATION IN A MATURE GNRH NEURONAL CELL LINE, GT1-7 CELLS

Riaño Gómez JM[1], Sorianello EM[1], Libertun C[1,2], Lux-Lantos VAR[1], Fernandez MO[1]
[1]Instituto de Biología y Medicina Experimental-CONICET. [2]Facultad de Medicina-UBA. E-mail: mfernandez@dna.uba.ar

Previously we showed that the in-vitro exposure to BPA, BP2 y BP3, endocrine disruptors, and E_2, (1×10^{-7} and 1×10^{-9} M, 24 h) increased cell proliferation in an immature GnRH cell line, GN11 cells (Susan Wray, USA). The aim of this study us to evaluate the effects of the in-vitro exposure of the aforementioned compounds on cell proliferation in GT1-7 cells (mature GnRH neurons, Pamela Mellon, UCSD, USA). Cell proliferation was evaluated using a Non-Radioactive Cell Proliferation Assay, MTS (Promega, WI, USA), after BPA, E_2, BP2 and BP3 exposure (1×10^{-7} and 1×10^{-9} M, 24 h). We also evaluated if the estrogen receptor antagonist ICI 182780, (1×10^{-6} M) was able to block the effects. Results were recorded as Abs490/Abs490 (Control), presented as mean ± SE and analyzed by repeated measures ANOVA with a Fisher post test (Statistica, StatSoft, OK, USA). Neither BPA nor E_2 modified cell proliferation (ANOVA ns, N = 5). BP2 did not modify cell proliferation either, but BP3 increased cell proliferation compared to control values [Control = 1 ± 0.03; $BP2^{-7}$= 0.92 ± 0.12; $BP2^{-9}$ = 0.94 ± 0.11; $BP3^{-7}$ = 1.29 ± 0.13; $BP3^{-9}$ = 1.29 ± 0.09; Repeated measures ANOVA $P < 0.05$, $BP3^{-7}$ and $BP3^{-9}$ different from Control $P < 0.05$, N = 5]. The estrogen antagonist ICI 182780 only blocked the effects of $BP3^{-9}$ (Repeated measures ANOVA $P < 0.05$, N = 5). The results obtained show that exposure to ED have different effects on mature and immature GnRH neurons. This reinforces the notion that effects of the exposure to ED depend on the developmental period, among other factors.
(Supported by CONICET, ANPCYT, International Society for Neurochemistry, Fund. Williams, Fund. R. Barón).

A261
PRENATAL D-AMPHETAMINE EXPOSURE ALTERS THE HYPOTHALAMUS PITUITARY AXIS RESPONSE THAT REGULATES PRL SECRETION IN ADULTHOOD. INVOLVEMENT OF STRESS AND SEXUAL STEROIDS

Santonja FE, Pietrobón E, Neira F, Sanchez MB, Moreno-Sosa T, Jahn GA, Bregonzio C, Valdez SR, Soaje M
Laboratorio de Reproducción y Lactancia-IMBECU. Mendoza; IFEC-CONICET; FCEN-UNCuyo; FCM-UNCuyo.
E-mail: msoaje@mendoza-conicet.gob.ar

Prenatal amphetamine exposure (PEA) induces long-lasting changes that are evident even in adulthood. D-amphetamine is a stimulant of CNS and acts on the dopaminergic and noradrenergic systems. Prolactin (PRL) synthesis and secretion is regulated by an inhibitory hypothalamic tone exerted by tuberoinfundibular dopaminergic neurons (TIDA). Dopamine (DA) is synthesized by tyrosine hydroxylase (TH) and released into portal blood to act on pituitary dopaminergic receptors (D2R) to inhibit PRL. Stress and sex steroids modulate PRL release, and PRL regulates its own secretion