

medicina

BUENOS AIRES Vol. 81 Supl. III - 2021



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BUENOS AIRES, VOL. 81 Supl. III - 2021

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MEDICINA (Buenos Aires) - Revista bimestral – ISSN 1669-9106 (En línea)

Registro de la Propiedad Intelectual N° 02683675
Personería Jurídica N° C-7497

Publicación de la Fundación Revista Medicina (Buenos Aires) Propietario de la publicación: Fundación Revista Medicina
Queda hecho el depósito que establece la Ley 11723

Publicada con el apoyo del Ministerio de Ciencia, Tecnología e Innovación Productiva.
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Vol. 81, Supl. III, Noviembre 2021

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Cerebrovascular diseases are the second leading cause of death within non-communicable diseases, according to the World Health Organization. The complex system morphology of blood vessels could be studied by mathematical algorithms to obtain fractal parameters: Fractal Dimension (FD) and predictive determination coefficient (R^2) which would indicate the irregular distribution of the vessels in space and their adaptation ability and interaction with the environment, respectively. The aim of this work was to analyze the encephalic carotid arterial system anatomy behavior through Higuchi Algorithm (HA) in patients with neurovascular diseases for prognostic purposes. Observational, cross sectional and descriptive study. Middle Cerebral (MCA), Anterior Cerebral (ACA) and Internal Carotid (ICA) arteries were analyzed in 75 angio-CT images of patients with diagnosed neurovascular pathology, mean age 36.25 years old \pm 15.95, 67% women and 33% men, chosen randomly. COREL-Draw, Phillips DICOM Viewer 3.0, Frakout! and Excel spreadsheet were using for HA application. FD and R^2 were determined, values below 0.8 would indicate loss of fractal properties. The results were expressed as median (M) and standard deviation (\pm). Pearson's correlation coefficient (R) was obtained between FD and R^2 established by arterial vessels studied. Results: FD (MCA): $M=0.5\pm 0.33$, R^2 (MCA): $M=0.88\pm 0.18$; FD (ACA): $M=0.38\pm 0.23$, R^2 (ACA): $M=0.76\pm 0.28$; FD (ICA): $M=0.32\pm 0.25$, R^2 (ICA): $M=0.64\pm 0.29$. Correlation for MCA: $r=0.64$ ($p<0.0005$), ACA: $r=0.81$ ($p<0.0001$) and ACI: $r=0.75$ ($p<0.0001$). Conclusion: HA reveals structural changes and maladjustment in the morphology of arteries studied. HA could evaluate structural changes in patients with neurovascular disease, helping to predict new possible clinical events.

57. (602) DIFFERENTIAL EFFECTS OF THE ETHANOL ACUTE OR CHRONIC EXPOSURE ON THE EARLY HYPOXIC VENTILATORY RESPONSE (HVR) IN RAT NEONATES

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Early ethanol exposure disrupts neonatal respiratory patterns and it has been suggested as risk factor associated with the Sudden Infant Death Syndrome. Ambient hypoxia acts as an environmental stressor eliciting breathing adaptations that may be altered by the EtOH exposure. However, the specific effects induce by chronic, acute or the combination of them EtOH intoxication are not clearly understood. In an animal model equivalent to the 3rd human gestational trimester, breathing frequencies and apneas were recorded against an intermittent hypoxic experience as a function of EtOH pre-exposure and/or acute EtOH intoxication. Pups pre-exposed to 0.0 or 2.0g/kg of EtOH (DPs 3-5-7, ig) were evaluated at DP9 in sobriety-0.0g/kg- or under the state of EtOH intoxication-2.0g/kg-. Breathing rates and apneas were recorded through whole body plexismography during 35 minutes [5 min of initial normoxia, followed by 3 episodes of hypoxia (O₂ 8%) of 5 min, separated by periods of recovery-normoxia of the same duration].

First acute EtOH intoxication diminished the hypoxic ventilatory response (HVR) during the test ($p=0.034$) relative to it expressed in pups never intoxicated. The prior experience with the drug significantly modified the HVR patterns, as follow: in sobriety, EtOH pre-exposed pups exhibited a depressed HVR relative to vehicle pre-exposed pups. On the contrary, under the state of intoxication, EtOH pre-exposed pups elicited an exacerbated HVR when were defied by hypoxia respect to vehicle pre-exposed pups. With regard to apneas, an increase in the number of apneas was triggered by both, the first acute EtOH intoxication or by the history with the drug in sober pups ($p=0.017$). In summary, specific HVR alterations and apneic episodes occurrence were observed in neonates depending on the type of EtOH exposure received (acute or chronic). These results emphasize the complexity of the disruptive EtOH effects upon breathing at this early and critical stage of development.

Funding: PICT 2016-0144/SECyT-UNC-CONSOLIDAR, Dra.

ABATE.

ENDOCRINOLOGÍA

58. (003) GENE EXPRESSION PROFILE DURING PITUITARY DEVELOPMENT IN A TWO-DIMENSIONAL MONOLAYER DIFFERENTIATION PROTOCOL FROM HUMAN INDUCED PLURIPOTENT STEM CELLS

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Multiple pituitary hormone deficiencies (MPHD) can be caused by mutations in several transcription factor genes in mouse and human, including *Prop1*, *Pou1f1*, *Hesx1*, *Sox2* and other genes involved in early pituitary embryogenesis. In order to study this process of disease development, we aimed to generate a human *in vitro* model of embryonic pituitary as a robust tool for functional testing of genetic variants found in MPHD patients. We hypothesized that combining different stimuli and differentiation protocols *in vitro* [1], [2], it is possible to induce fully differentiated hormone-pituitary cells from human induced pluripotent stem cells (iPSCs). We cultured the iPSCs in the presence of signaling factors involved in pituitary development including Bone Morphogenetic Protein 4 (BMP4), the Smoothened Agonist activator of Sonic Hedgehog pathway (SAG), and Fibroblast Growth Factor 2 (FGF2), for 15 days. During the entire protocol, cell morphology was observed and registered under the microscope, and cellular extracts corresponding to days 0, 4, 7, and 15 of the protocol were collected to assess gene expression by qRT-PCR. We observed an increase in the expression of representative markers of pituitary differentiation (*PITX2*, *SIX1*, *HESX1*, *OTX2*) and a decrease in the expression of pluripotency markers (*NANOG*, *OCT4*) over the days of treatment compared to control iPSCs (cultured with media only), suggesting that the protocols were effective in their cell specification. An interesting finding was the increase in mRNA levels of *FOXA2* at days 7 and 15 of the protocol, a novel gene in the etiology of MPHD, poorly characterized in human pituitary development and found mutated in one case of MPHD from our Argentinean cohort [3]. This study establishes a new approach to study protein's role in pituitary progenitor cell regulation and offers new candidate genes for MPHD that remain unexplained.

References: [1] B. Zimmer, J. Piao, K. Ramnarine, M. J. Tomishima, V. Tabar, and L. Studer, "Derivation of Diverse Hormone-Releasing Pituitary Cells from Human Pluripotent Stem Cells," *Stem Cell Reports*, vol. 6, no. 6, pp. 858–872, Jun. 2016.

[2] R. Matsumoto and Y. Takahashi, "Human pituitary development and application of iPSCs for pituitary disease," *Cell. Mol. Life Sci.*, vol. 78, no. 5, pp. 2069–2079, Mar. 2021.

[3] S. A. Vishnopska *et al.*, "Comprehensive Identification of Pathogenic Gene Variants in Patients With Neuroendocrine Disorders," *J. Clin. Endocrinol. Metab.*, vol. 106, no. 7, pp. 1956–1976, Jun. 2021.

59. (005) INFLUENCE OF PRENATAL AND NEONATAL EXPOSURE TO PHTHALATE ON THE SEXUAL BEHAVIOR AND ACTIVITY OF MALE RATS.

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The endocrine disruptor phthalate (DEHP) has estrogenic and / or anti-androgenic activity. We have previously demonstrated its antiandrogenic action in male rats dependent on the androgenic level.

Objective: to evaluate the effect of DEHP on sexual behavior parameters in adult male rats. **Materials and Methods:** Male rats were exposed to DEHP (n=14) during their intrauterine development and lactation at a dose of 30 mg/kg/d administered in drinking water to the mothers. Controls (C) only received the vehicle (n=13). Between 150 and 180 days of age, the sexual behavior tests were carried out