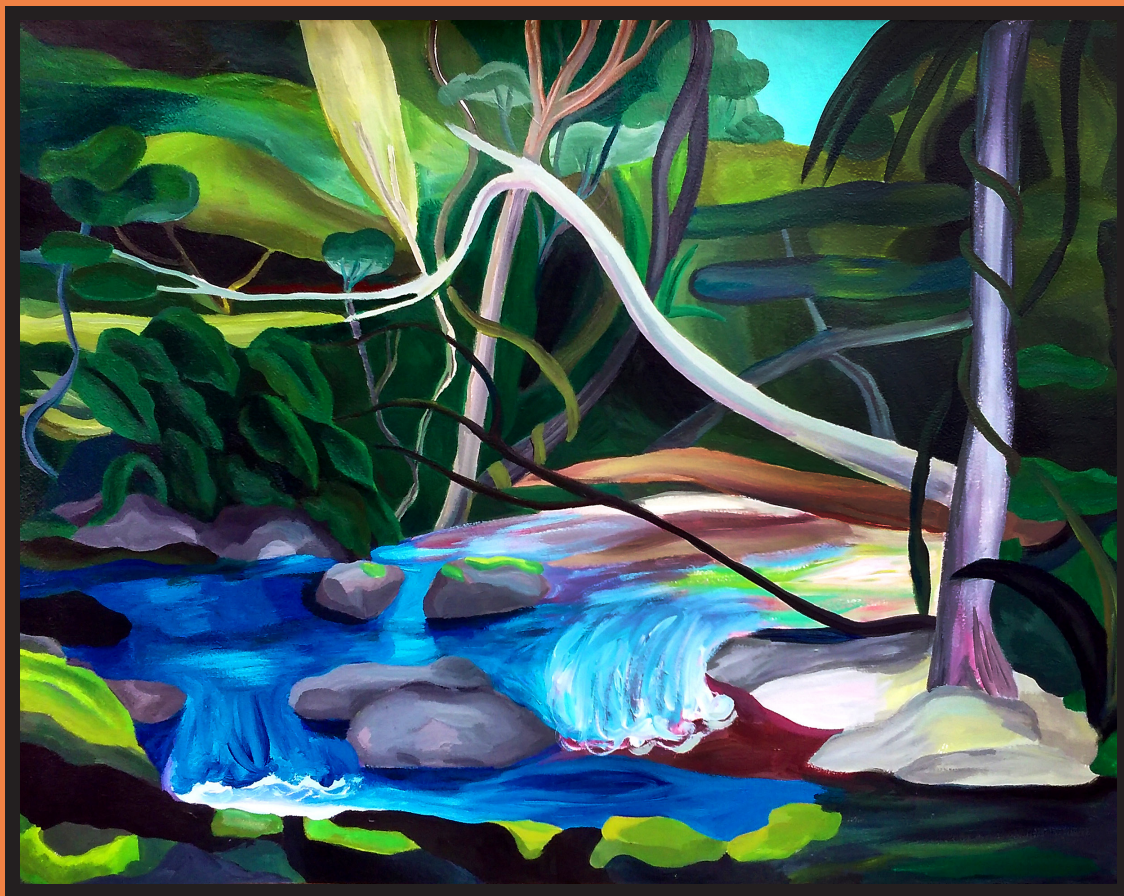


# medicina

BUENOS AIRES, VOL. 83 Supl. V - 2023



# medicina

BUENOS AIRES, VOL. 83 Supl. V - 2023

## COMITÉ DE REDACCIÓN

**Sebastián F. Ameriso**  
*FLENI, Buenos Aires, Argentina*

**Pablo J. Azurmendi**  
*Instituto de Investigaciones Médicas A. Lanari, UBA, Argentina*

**Damasia Becú Villalobos**  
*Instituto de Biología y Medicina Experimental-CONICET,  
Buenos Aires, Argentina*

**Gabriela V. Carro**  
*Hospital Nacional Prof. A. Posadas  
Buenos Aires, Argentina*

**José H. Casabé**  
*Instituto de Cardiología y Cirugía Cardiovascular,  
Hospital Universitario Fundación Favaloro, Buenos Aires, Argentina*

**Hugo N. Catalano**  
*Hospital Alemán, Buenos Aires, Argentina*

**Eduardo L. De Vito**  
*Instituto de Investigaciones Médicas A. Lanari, UBA, Argentina*

**Elisa Estenssoro**  
*Hospital Interzonal de Agudos General San Martín de La Plata,  
Buenos Aires, Argentina*

**Laura I. Jufe**  
*Hospital General de Agudos J. M. Ramos Mejía,*

**Isabel Narvaiz Kantor**  
*Organización Panamericana de la Salud (OPS/OMS), Argentina*

**Basilio A. Kotsias**  
*Instituto de Investigaciones Médicas A. Lanari, UBA, Argentina*

**Gustavo Kusminsky**  
*Hospital Universitario Austral, Buenos Aires, Argentina*

**Oscar M. O. Laudanno**  
*Instituto de Investigaciones Médicas A. Lanari, UBA, Argentina*

**Isabel A. Lüthy**  
*Instituto de Biología y Medicina Experimental (IBYME),  
Buenos Aires, Argentina*

**Domingo J. Palmero**  
*Hospital de Infecciosas Dr. Francisco J. Muñiz  
Instituto de Tisiopneumología Prof. Dr. Raúl Vacarezza,  
Facultad de Medicina, UBA, Argentina*

**Guillermo B. Semeniuk**  
*Instituto de Investigaciones Médicas A. Lanari, UBA, Argentina*

**Oswaldo J. Stringa**  
*Hospital de Clínicas José de San Martín, UBA, Argentina*

**Carlos D. Tajer**  
*Hospital de Alta Complejidad El Cruce Néstor Kirchner,  
Buenos Aires, Argentina*

## MIEMBROS EMÉRITOS

**Héctor O. Alonso**  
*Instituto Cardiovascular Rosario, Santa Fe, Argentina*

**María Marta de Elizalde de Bracco**  
*IMEX-CONICET-Academia Nacional de Medicina, Buenos Aires, Argentina*

**Guillermo Jaim Etcheverry**  
*Facultad de Medicina, UBA, Argentina*

**Daniel A. Manigot**  
*Hospital San Juan de Dios, Buenos Aires, Argentina*

**Rodolfo S. Martin**  
*Facultad de Ciencias Biomédicas,  
Hospital Universitario Austral, Buenos Aires, Argentina*

La Tapa  
Todo, 2016  
Daniela Kantor

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.  
MEDICINA no tiene propósitos comerciales. El objeto de su creación ha sido propender al adelanto de la medicina argentina.  
Los beneficios que pudieran obtenerse serán aplicados exclusivamente a este fin.  
Aparece en MEDLINE (PubMed), ISI-THOMSON REUTERS (Journal Citation Report, Current Contents, Biological Abstracts, Biosis, Life Sciences), CABI (Global Health), ELSEVIER (Scopus, Embase, Excerpta Medica), SciELO, LATINDEX, BVS (Biblioteca Virtual en Salud), DOAJ, Google Scholar y Google Books.  
Incluida en el Núcleo Básico de Revistas Científicas Argentinas del CONICET.

**Directores Responsables:**  
Eduardo L. De Vito, Isabel Lüthy, Oscar M. O. Laudanno, Isabel Narvaiz Kantor

**Secretaría de Redacción:** Ethel Di Vita, Instituto de Investigaciones Médicas Alfredo Lanari, Combatientes de Malvinas 3150,  
1427 Buenos Aires, Argentina  
e-mail: revmedbuenosaires@gmail.com – http://: www.medicinabuenosaires.com

Vol. 83, Supl. V, Noviembre 2023

Diagramación y Diseño: Andrés Esteban Zapata - aez.sgi@gmail.com

# **REUNIÓN CONJUNTA SAIC SAB AAFE AACYTAL 2023**

**LXVIII REUNIÓN ANUAL DE LA  
SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA  
(SAIC)**

**XXV JORNADAS ANUALES DE LA SOCIEDAD  
ARGENTINA DE BIOLOGÍA  
(SAB)**

**LV REUNIÓN ANUAL DE LA ASOCIACIÓN  
ARGENTINA DE FARMACOLOGÍA EXPERIMENTAL  
(AAFE)**

**VIII REUNIÓN CIENTÍFICA REGIONAL DE LA  
ASOCIACIÓN ARGENTINA DE CIENCIA Y  
TECNOLOGÍA DE ANIMALES DE LABORATORIO  
(AACYTAL)**

15-17 de noviembre de 2023  
Hotel 13 de Julio – Mar del Plata

**EDITORES RESPONSABLES**

Dra. Isabel Luthy  
Dra. Silvina Pérez Martínez  
Dr. Ventura Simonovich  
Dr. Gabriel Pinto

# **JOINT MEETING SAIC SAB AAFE AACyTAL 2023**

**LXVIII ANNUAL MEETING OF  
SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA  
(SAIC)**

**XXV ANNUAL CONFERENCES OF SOCIEDAD  
ARGENTINA DE BIOLOGÍA  
(SAB)**

**LV ANNUAL MEETING OF ASOCIACIÓN ARGENTINA  
DE FARMACOLOGÍA EXPERIMENTAL  
(AAFE)**

**VIII REGIONAL SCIENTIFIC MEETING OF  
ASOCIACIÓN ARGENTINA DE CIENCIA Y  
TECNOLOGÍA DE ANIMALES DE LABORATORIO  
(AACyTAL)**

November 15-17, 2023  
13 de Julio Hotel – Mar del Plata

**RESPONSIBLE EDITORS**  
Dra. Isabel Luthy  
Dra. Silvina Pérez Martínez  
Dr. Ventura Simonovich  
Dr. Gabriel Pinto

healthy females (n=8). Paternal sperm was collected from cauda epididymis to count spermatozoa and measure motility. On day 21 of gestation, pregnant rats were euthanized, and the placenta and the fetuses were sexed and weighed. In paternal and fetal plasma, glycemia, triglyceridemia (TG), and cholesterolemia (Ch) were measured by colorimetric assays. The placenta was stored at -80°C for the evaluation of PPAR $\alpha$ , PPAR $\gamma$  and PGC1 $\alpha$  (by Western blot). Results: Glycemia, TG and Ch was higher in D than in C males (P<0.05). A significant reduction in motility and sperm count was found in D males when compared to C (54% and 46%, respectively, P<0.01). Fetal glycemia and Ch, and placental and fetal weight were similar in paternal C and D groups, but TG were increased in male fetuses from paternal D group (12%, P<0.05). In fetuses, a decrease in PPAR $\gamma$  (females 39%; males 77%, P<0.05) and an increase in PGC1 $\alpha$  (females 15%; males 84%, P<0.05) and PPAR $\alpha$  (males 45%, P<0.05) were found in the placenta of paternal D group. Conclusion: In this model of diabetes, we showed alterations in male reproductive functions and the paternal programming of sex-dependent alterations in PPARs, master genes that regulate the development and function of the placenta, an organ highly related to the programming of the diseases of the offspring.

**514. 135. MITOCHONDRIAL DYNAMIC IS ALTERED IN THE OVARIES FROM RATS WITH POLYCYSTIC OVARY SYNDROME**

Mayra Bordaquievich<sup>1</sup>, Melanie Neira<sup>1</sup>, Candela Velazquez<sup>1</sup>, Yamila Herrero<sup>1</sup>, Rocío Marinoni<sup>1</sup>, Fernanda Parborelli<sup>1</sup> and Dalhia Abramovich<sup>1</sup>

<sup>1</sup> Instituto de Biología y Medicina Experimental (IByME). Vuelta de Obligado 2490, C1428. Buenos Aires, Argentina

Introduction: Polycystic ovary syndrome (PCOS) is the most common endocrine disorder, affecting 5-10% of women in reproductive age. In spite of being described eighty years ago, the pathological mechanisms of PCOS are not completely understood and therefore, studying the molecular mechanisms of this syndrome is essential to improve diagnosis and treatment of these women. Objective: To evaluate the involvement of mitochondria in the ovarian alterations in a rat model of PCOS. Methods: 21 days old Sprague Dawley rats were treated with dehydroepiandrosterone (DHEA) for 15 days (PCOS group). Control group received vehicle. At day 16, rats were sacrificed and the ovaries recovered to perform histology and western blot. Histological slides were stained with picosirius red to evaluate fibrosis or H&E. Results: Unlike the control group, we observed the presence of ovarian cysts and the ovarian anomalies previously described in the PCOS group. We found a significant decrease in TOMM-20 protein, in the mitochondria-shaping proteins MFN-2 and DRP-1 and in Sirtuin-1, with no changes in OPA-1. Ovarian fibrosis was increased in the PCOS group. Conclusions: Our results suggest a decreased in the number of mitochondria and a deregulation in mitochondrial dynamic that could be involved in ovarian dysfunction in this rat model of PCOS. Therapies that target mitochondria, such as metformin, are worth to study in this pathology to improve ovarian performance.

**515. 201. ELEVATED CHORIONIC GONADOTROPIC HORMONE IN TRANSGENIC MICE INDUCES PARTHENOGENETIC ACTIVATION AND OVARIAN TERATOMAS**

Susana B. Rulli<sup>1,2</sup>, Laura D. Ratner<sup>3</sup>, Matti Poutanen<sup>2</sup>, Ilpo Huhtaniemi<sup>2,4</sup>

<sup>1</sup> Centro de Estudios Biomédicos Básicos, Aplicados y Desarrollo (CEBBAD), Universidad Maimónides, Buenos Aires, Argentina. <sup>2</sup> Institute of Biomedicine, Research Centre for Integrative Physiology and Pharmacology, University of Turku, Kiinamylynkatu 10, FIN-20520 Turku, Finland. <sup>3</sup> Facultad de Agronomía, Departamento de Producción Animal, Laboratorio Biotecnología Animal (LabBA), Universidad de Buenos Aires, Buenos Aires, Argentina. <sup>4</sup> Department of Digestion, Metabolism and Reproduction, Institute of Reproductive and Developmental Biology, Hammersmith Campus, Imperial College London, London W12 0NN, UK.

Both male and female reproductive functions are impacted by al-

tered gonadotrophin secretion and action, which may also influence the development of endocrine tumors. Female transgenic (TG) mice overexpressing both the  $\alpha$ - and  $\beta$ - subunits of human chorionic gonadotropin (hCG), produced high levels of bioactive hCG and presented with precocious puberty, infertility, and enhanced gonadal steroidogenesis. The objective of this work is to ascertain if chronic hypersecretion of hCG contributes to the development of gonadal tumors. Ovaries from double TG female mice for hCG were analyzed at different ages. By the age of two months, ovarian tumors with characteristics of teratomas developed with 100% penetrance. Tissues such as keratinized epithelium, hair follicles, cartilage, sebaceous glands, neural tissue, and intestinal and respiratory-like epithelium were identified in the TG ovaries. Teratomas were also seen in wild-type ovaries orthotopically transplanted into TG mice, demonstrating an endocrine mechanism for the hCG-induced ovarian tumorigenesis. Both *in vitro* and *in vivo* experiments showed oocyte parthenogenetic activation in TG females, developing up to the blastocyst stage. In addition, ovaries showed reduced ovulatory gene expression, inhibited ERK1/2 phosphorylation, and impaired cumulus cell expansion (p< 0.01). In conclusion, persistently high endocrine hCG activity causes parthenogenetic activation and development of ovarian teratomas, along with altered follicle development and impaired ERK signaling, offering a novel mechanism associated with the molecular pathogenesis of ovarian teratomas.

**516. 296. EFFECT OF ESTRADIOL-17 $\beta$  INJECTIONS DURING THE LUTEAL PHASE ON THE PLASMA PROGESTERONE CONCENTRATION IN LLAMAS**

Carolina Bianchi<sup>1,2</sup>, Micaela Benavente<sup>1,2</sup>, Juan Manuel Herrera<sup>3</sup>, Marcelo Rodríguez<sup>4</sup>, Marcelo Aba<sup>1,2</sup>, María Florencia Gallelli<sup>5</sup>

<sup>1</sup>UNCPBA, FCV, FISFARVET, Tandil, Buenos Aires, Argentina. <sup>2</sup>CIVETAN, UNCPBA-CICPBA-CONICET, Tandil, Buenos Aires, Argentina. <sup>3</sup>UNCPBA, FCV, CIB, Tandil, Buenos Aires, Argentina. <sup>4</sup>UNCPBA, FCV, SAMP, Área de Estadística, Tandil, Buenos Aires, Argentina. <sup>5</sup>UBA, FCV, INITRA, Buenos Aires, Argentina.

The aim of this study was to evaluate the effect of estradiol-17 $\beta$  (E2) injections during the luteal phase on the plasma progesterone (P4) concentration in llamas. Twenty-one females were induced to ovulate by GnRH injection in presence of a follicle  $\geq$  8 mm and randomly assigned into one of three groups: control (n=7) without treatment; G7-8 (n=7) treated with 1.6 mg of E2 IM on Day 7 and 0.8 mg of E2 IM on Day 8; and G7-10 (n=7) treated with 1.6 mg of E2 IM on Day 7 and 0.8 mg of E2 IM from Day 8 to 10 post-GnRH. Blood samples were collected every other day from Day 0 to 7 and daily until Day 15 post-GnRH to determine plasma P4 concentration by RIA. The data were analyzed using an ANOVA test, and the effect of days within groups was compared by a Tukey Test with Bonferroni adjustment. Mean plasma P4 concentration increased from Day 0 to Day 8 post-GnRH in all females. In the control group, mean plasma P4 concentration decreased to below 1 ng/ml on Day 10, being statistically different from treated groups (P<0.01). In the G7-8, mean plasma P4 concentration decreased between Day 11 and 12 post-GnRH, being in those days significantly lower than in llamas from G7-10 (P<0.01). In conclusion, E2 injection from Day 7 post-induction of ovulation results in a luteotrophic effect in llamas. The E2 administration from Day 7 to Day 10 post-GnRH prolongs the corpus luteum function for three more days than in control animals, which gives further support to the hypothesis that E2 would be involved in the process of maternal recognition of pregnancy in this species.

**517. 327. DOES PRENATAL CANNABIS EXPOSURE INFLUENCE OFFSPRING DEVELOPMENT AND REPRODUCTIVE OUTCOMES?**

Ayelen Mirón Granese<sup>1</sup>, Carolina Marvaldi<sup>1</sup>, Julieta Aisemberg<sup>1</sup>, Fernando Correa<sup>1</sup>, Daniela Sedan<sup>2</sup>, Dario Andrinolo<sup>2</sup>, Ana María Franchi<sup>1</sup>, Manuel Luis Wolfson<sup>1</sup>.

<sup>1</sup>Laboratorio de Fisiopatología de la preñez y el parto. Centro de Estudios Farmacológicos y Botánicos (CEFYBO)-UBA/CONICET. Facultad de Medicina, Universidad de Buenos Aires. <sup>2</sup>Centro de investigaciones del Medio