2021

medicina

BUENOS AIRES Vol. 81 Supl. III - 2021

MEDICINA

Volumen 81, Supl. III, Págs. 1- 250

medicina

BUENOS AIRES, VOL. 81 Supl. III - 2021

COMITÉ DE REDACCIÓN

MIEMBROS EMÉRITOS

La Tapa
Médanos
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:
Basilio A. Kotsias, Eduardo L. De Vito, Isabel Narvaiz Kantor, Isabel Lüthy

Secretaría de Redacción: Ethel Di Vita, Instituto de Investigaciones Médicas Alfredo Lanari, Combatientes de Malvinas 3150, 1427 Buenos Aires, Argentina
Tel. 5287-3827 Int. 73919 y 4523-6619
e-mail: revmedbuenosaires@gmail.com – http//: www.medicinabuenosaires.com

Vol. 81, Supl. III, Noviembre 2021

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

REUNIÓN DE SOCIEDADES DE BIOCIENCIAS 2021

LXVI REUNIÓN ANUAL DE LA
SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LXIX REUNIÓN ANUAL DE LA
SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

LIII REUNIÓN ANUAL DE LA
ASOCIACIÓN ARGENTINA DE FARMACOLOGÍA EXPERIMENTAL (AAFE)

XI REUNIÓN ANUAL DE LA
ASOCIACIÓN ARGENTINA DE NANOMEDICINAS
(NANOMED-AR)

17-20 de noviembre de 2021

EDITORES RESPONSABLES
Dr. Alejandro Curino
Dra. Mariana Maccioni
Dra. Paula Schaiquevich
Dra. Hebe Duran

ANNUAL MEETING OF BIOSCIENCE SOCIETIES 2021

LXVI ANNUAL MEETING OF
SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LXIX ANNUAL MEETING OF
SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

LIII ANNUAL MEETING OF
ASOCIACIÓN ARGENTINA DE FARMACOLOGÍA EXPERIMENTAL (AAFE)

XI ANNUAL MEETING OF
ASOCIACIÓN ARGENTINA DE NANOMEDICINAS
(NANOMED-AR)

November 17-20, 2021

RESPONSIBLE EDITORS
Dr. Alejandro Curino
Dra. Mariana Maccioni
Dra. Paula Schaiquevich
Dra. Hebe Duran

LA TAPA

Daniela Kantor. Médanos, 2018

Técnica: Acrílico sobre cartón entelado. Medidas: 20x28 cm

Daniela Kantor nació el 23 de marzo de 1970. Es diseñadora gráfica (FADU-UBA), pintora, dibujante, historietista e ilustradora. Autora de la novela gráfica Mujer Primeriza (Ed. Burlesque, 2014), Aprendiza (2019) y Naturalella (con guión de Arekasadaro, 2017) publicada en Dis-Tinta (Ed. Sudamericana, coordinado por Liniers y Martin Pérez). Con guión de Alejandro Farias dibujó Las moradas de Santa Teresa de Jesús en historietas (Ed. Loco rabia + CCEBA Centro Cultural de España en Buenos Aires) y Marilyn (Tren en movimiento, 2019). Es miembro de la revista de historietas “El Tripero” fundada en 1993 junto al grupo de alumnos de Alberto Breccia. En el ámbito de la enseñanza es Jefa de Trabajos Prácticos en la materia Ilustración inicial, y docente en Ilustración Editorial en la Facultad de Arquitectura, Diseño y Urbanismo FADU/ UBA. Dicta talleres sobre pintura e ilustración (C C Recoleta, 2019/ Quinta Trabucco, 2020/ taller particular junto a Daniel Roldan, 2019). Es maestra de niños y niñas en Dibujo e Historieta en Escuelas primarias, talleres (Filbita, Festival de literatura de Buenos Aires, 2018-9/ CCK, 2018/ taller propio desde 2014). Estudió Dibujo de Historieta con Alberto Breccia, Técnicas de Acuarela y Pastel con Carlos Nine, charlas sobre Historieta con José Muñoz, Curso de Color con Carlos Gorriarena, Clínica de Pintura con Mariano Sapia y Tulio de Sagastizábal, y Sumi- e en el Centro Okinawense. Trabaja para editoriales y revistas con ilustraciones e historietas (Ed. Troquel, Abran Cancha, Ed. Norma, Unicef, Barcelona, Crisis, Suplemento Ñ/ Clarín, Borges en la Biblioteca Nacional- Lectores de Borges). Fue invitada a la Feria del libro de los Universitarios de UNAM para presentar el libro “Palabra de ilustrador”, y en 2019 ganó la Beca UBA Internacional en el marco de un programa de intercambio docente con la Universidad Regiomontana, Monterrey, México.

Fuentes: https://www.instagram.com/daniela.kantor.9/; www.kantorconk.blogspot.com

CONSEJOS DIRECTIVOS

SAIC

Presidente
Alejandro Curino

Vicepresidente
Daniel Alonso

Secretario
Alejandro Urtreger

Tesorera
Laura Todaro

Prosecretaria
Stella Ranuncolo

Vocales
Evangelina Capobianco
María del Rocío Castilla Lozano
Pablo Gravina
Adriana Casas
Julieta Maymo
María Marta Amaral
Ricardo Cabrera
Sandra Ferreira
Marcela Bolontrade
Adriana Burgueño
Julia Halperin
Luis Di Ciano
María Laura Ruiz

Revisores de cuentas
Gabriela Lombardi
Mariela Pérez

SAI

Presidenta
Mariana Maccioni

Vicepresidente
Emilio Malchiodi

Secretaria
Silvia Correa

Tesorera
Mercedes Fuertes

Prosecretaria
Mariana Salatino

Protesorera
Marisa Castro

Vocales
Mercedes Borge
Karina Canziani
Esteban Grasso
Carolina Maldonado Galdeano
Gerardo Mirkin
Verónica Natoli
María Silvia Ventimiglia
Silvina Villar

AAFE

Presidenta
Paula Schaiquevich

Vicepresidente
Ventura Simonovich

Secretaria
Myriam Laconi

Tesorera
Susana Gorzalczany

Prosecretaria
Daniela Quinteros

Vocales
Carlos Reyes Toso
Fatima Nader
Santiago Palma

Revisores de cuentas
Héctor Alejandro Serra
María Victoria Aguirre

Revisores de cuentas
(suplentes)
Andrea Errasti
Ariel Perelsztein

NANOMED-ar

Presidenta
Hebe Durán

Vicepresidenta
Romina Glisoni

Secretaria
Leticia Higa

Tesorera
Julia Altube

Vocales
Dr. Eder Romero
Dra. Mariela Agotegaray

Vocal (suplente)
Dra. Priscila Schilrreff

Revisora de cuentas
Dra. Marisa Taverna Porro

Revisora de cuentas
(suplente)
María José Morilla

LAS SOCIEDADES QUE ORGANIZAN ESTA REUNIÓN CONJUNTA AGRADECEN EL APOYO DE

INSTITUCIONES OFICIALES

CONSEJO NACIONAL DE INVESTIGACIONES CIENTÍFICAS Y TÉCNICAS
MINISTERIO DE CIENCIA, TECNOLOGÍA E INNOVACIÓN PRODUCTIVA
AGENCIA NACIONAL DE PROMOCIÓN DE LA INVESTIGACIÓN, EL DESARROLLO TECNOLÓGICO Y LA INNOVACIÓN

OTRAS INSTITUCIONES Y AUSPICIANTES

FUNDACIÓN CHERNY
FUNDACIÓN HONORIO BIGAND
LABORATORIO GADOR S.A.
ETC INTERNACIONAL S.A.
LABORATORIO DE HEMODERIVADOS-UNC
ARCOR S.A.
FUNDACIÓN JOSÉ A. BALSEIRO
FUNDACIÓN ARGENTINA DE NANOTECNOLOGÍA
ATOM-PROTECT®

able. Growing evidence suggests that chemotherapy-resistant BC cells with stem-like properties (CSC) may repopulate the tumor. Therefore, therapies that target the CSC in combination with chemotherapy might prevent tumor recurrence. Androgen Receptor (AR) is expressed in at least half of all TNBC. AR inhibition decreases CSC *in vitro* and tumor initiation i*n vivo*. RUNX1 is regulated by AR in prostate cancer. In TNBC patients, RUNX1 protein levels correlate with poor prognosis. Our group has shown that RUNX1 promotes TNBC cell migration and regulates tumor gene expression, such as the oncogene *RSPO3*. Also, by RUNX1 ChIP assays, we found SOX4 as a potential target gene. We hypothesized that RUNX1 is regulated by AR and that both may work together in TNBC CSCs to promote persistence and disease recurrence following chemotherapy. Here we show that, in MDA-MB-453 cells, RUNX1 expression is upregulated by dihydrotestosterone, an AR agonist, and that this effect is blocked in the presence of Enzalutamide (AR antagonist). ChIP-seq experiments revealed AR binding to RUNX1 regulatory regions, suggesting direct regulation. RUNX1 expression is increased in a CSC-like experimental model and responds to AR activity. Inhibition of RUNX1 transcriptional activity by AI-10-104 (a synthetic drug) reduced the expression of the CSC marker SOX4. Interestingly, this inhibition drives a reduction of MDA-MB-453 and BT-549 cell proliferation and enhanced paclitaxel sensitivity. It was reported that AR inhibition combined with chemotherapy results in a more effective outcome than chemotherapy alone *in vitro* and *in vivo*. In sum, RUNX1 inhibition may also be an attractive target to potentiate the anti-tumor effect of AR inhibition, specifically in the slow growing CSC-like populations that resist chemotherapy and lead to metastatic disease.

501. (507) **VITAMIN D RECEPTOR AND PACLITAXEL IN TRIPLE NEGATIVE BREAST CANCER: IS THERE A LINK BETWEEN THEM?**

Josefina Alejandra Guevara[1], Agustina Ibarra[1], Alfredo Quevedo[2], Eliana Noelia Alonso[1], Georgina Pamela Coló[1], María Marta Facchinetti[1], María Julia Ferronato[1*], Alejandro Carlos Curino [1]

*jferronato@criba.edu.ar

[1] Laboratorio de Biología del Cáncer, Instituto de Investigaciones Bioquímicas Bahía Blanca (INIBIBB), Universidad Nacional del Sur (UNS)-CONICET, Departamento de Biología, Bioquímica y Farmacia (UNS), Bahía Blanca, Argentina.[2] Unidad de Investigación y Desarrollo en Tecnología Farmacéutica (UNITEFA), CONICET- Departamento de Ciencias Farmacéuticas, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba, Córdoba, Argentina.

Paclitaxel (PTX) is an antitumor agent employed in the treatment of Triple-Negative Breast Cancer (TNBC). TNBC expresses Vitamin D Receptor (VDR), a member of the nuclear receptor superfamily. The aim of this work was to investigate the involvement of VDR in the antitumor action of PTX in TNBC cells. To this end, viability assays by crystal violet staining were performed in murine 4T1 TNBC cells and in 4T1 stably expressing a shRNA against VDR (4T1 shVDR), treated with PTX (10 nM) or vehicle. Also, cell cycle was studied by flow cytometry. Cellular studies were complemented with in silico analyses including molecular docking and molecular dynamics (MD) simulations to describe the pharmacodynamic interaction between PTX and VDR. The results show that PTX reduced the viability of 4T1 wild type cells ($p<0.001$). These viability effects were lost in 4T1 shVDR cells which display approximately 53% of VDR levels with respect to control cells. Cell cycle analysis of 4T1 wild type and 4T1 shVDR cells treated with PTX showed that the chemotherapy causes an increase in the percentage of cells in sub G0/G1 phase compared to vehicle-treated cells. However, this PTX effect was significantly higher in wild type than in VDR-silenced cells (13.72 ± 2.37% vs 6.18 ± 1.07%, $p<0.001$). Docking and MD studies showed that PTX was not able to bind to the classical ligand-binding pocket of VDR. However, an exhaustive search of allosteric sites identified its stable binding to a cavity adjacent to the activating factor 2 (AF-2) region. MD studies verified a conformational restraint on AF-2, which triggers transcriptional and antitumor effects. Furthermore, a potential cooperativity in the interaction with VDR between PTX and the natural ligand of the receptor was observed. Altogether, these results suggest that PTX could interact with VDR to display its antitumor effects in TNBC by its binding in an alternative site to that of the classical VDR agonists.

502. (509) **NOVEL HISTAMINE H_3 RECEPTOR ANTAGONISTS WITH POTENT ANTINEOPLASTIC PROPERTIES AS TARGETED DRUG THERAPY FOR BREAST CANCER**

Mónica A. Táquez Delgado[1], Melisa B. Nicoud[1], Ignacio Ospital[1], Michelle F. Corrêa[2], Gustavo A. B. Fernandes[2], Diego Martinel Lamas[1], João P. S. Fernandes[2], Vanina A. Medina[1].

1 Laboratorio de Biología Tumoral e Inflamación. Instituto de Investigaciones Biomédicas (BIOMED), Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), Universidad Católica Argentina (UCA), Argentina

2 Departamento de Ciências Farmacêuticas, Universidade Federal de São Paulo (UNIFESP), Diadema-SP, Brazil

We have reported the expression of the histamine H_3 receptor (H_3R) in human benign and malignant lesions, and cell lines derived from human mammary glands. Its expression is highly correlated with proliferation in breast cancer specimens.

In this work, we aimed at investigating the potential antitumoral activity of 4 novel H_3R antagonists, 1-(2,3-dihydro-1-benzofuran-2-yl) methylpiperazines (LINS01 compounds), which showed excellent selectivity and high affinity for the human H_3R.[1,2] Cell viability and proliferation were assessed by cell titer blue assay and colony formation in human MDA-MB-231 and murine 4T1 triple negative breast cancer cells. Cell apoptosis was assessed by Annexin V staining and flow cytometry, while cell migration was evaluated by wound-healing assay and transwell system. The lipid accumulation was assayed by flow cytometry using Nile-red staining.

Results indicate that compounds LINS01022, LINS01023, LINS01009, LINS01010 (0.1-100 μM) produced a concentration-dependent inhibition on cell growth. The highest responses were observed for LINS01022 and LINS01023, showing an IC_{50} in the cell viability assay of 82.7 and 78.2 μM for MDA-MB-231 cells, and 87.0 and 59.2 μM for 4T1 cells. LINS01022 and LINS01023 (25-50 μM) induced cell apoptosis (4 to 7 fold-increase) and differentiation (2 to 3 fold-increase), while suppressed cell migration in both cell lines ($P<0.01$).

The allylpiperazines LINS01022 and LINS01023 exhibited better antiproliferative and proapoptotic effects together with a higher affinity constant for the H_3R than their corresponding methylpiperazine analogues LINS01009 and LINS1010, respectively.

These effects were not observed with the selective H_3R agonist, (*R*)-alpha-methylhistamine.

In conclusion, this study demonstrates that the H_3R is involved in the regulation of cell growth and progression, offering novel therapeutic potentials for H_3R antagonists.

[1]Correa et al. *Front Pharmacol* **2017**,*8*,825

[2]Correa et al. *Bioorg Med Chem* **2021**,*30*,115924

503. (510) **HIF-1α REGULATES TUMOR PROGRESSION IN A HUMAN EPITHELIAL OVARIAN CANCER MODEL**

España De Marco María José[1], Irusta Griselda[1], Tesone Marta[1], Pérez Piñero Cecilia[2].

1 Laboratorio de Fisiología y Biología Tumoral del Ovario - Instituto de Biología y Medicina Experimental (IBYME) - CABA, Argentina

2 Laboratorio de Hormonas y cáncer – Instituto de Biología y Medicina Experimental (IBYME) – CABA, Argentina

Ovarian cancer is the seventh most common cancer in women and the eighth cause of cancer death. The treatment of this disease has been the same for the past decades, and the development of new drugs is needed. Hypoxia is a common characteristic of solid tumors, usually associated with a more aggressive phenotype. The main transcriptional factor involved in this process is Hypoxia Inducible Factor 1 alpha (HIF-1α).

The present work aimed to study the effect of Acriflavine (ACR), a specific HIF-1α inhibitor, on a human epithelial ovarian cancer model (SKOV3), both *in vivo* and *in vitro*. For the *in vitro* experiments, we

performed cell proliferation and wound healing assays to assess cell migration with different ACR concentrations. Cell proliferation was significantly diminished with ACR 1uM after 72 hours of treatment as compared with control cells (1 μM ACR: 22.69 % ± 0.66 vs Control 100,00% ± 10,40 , p<0.0001), and migration was reduced with ACR 1uM after 18 h of incubation (Control, 85,75% wound closure ± 5,12 vs ACR 1uM, 52,40 % wound closure ± 3,90, p<0.0001).
For the *in vivo* experiments, 5 x 106 cells were s.c. injected into the flank of immunosuppressed NSG mice. The treatment with ACR daily injections (5 mg/kg, 15 days) started when tumors reached 25 mm2. ACR-treated tumors were significantly smaller than control tumors (p<0.001), showed a lower proliferation index (*Ki67)* and a lower VEGF and GLUT1 expression through immunohistochemistry as compared with control tumor samples. VEGF and GLUT1 expression are used to evaluate the transcriptional activity of HIF-1α, as both proteins are HIF-1α downstream targets.
Our results show that HIF-1α plays an important role in the proliferation, migration, and tumor growth of the SKOV3 ovarian cancer model. We conclude that ACR could be a potential drug for the treatment of ovarian cancer, alone or in combination with standard therapy.

504. (514) **CLINICAL FEATURES IN PATIENTS WITH CANCER AND COVID-19 IN SANTA FE AND BUENOS AIRES**

Gastiazoro Maria Paula[1], Cardozo María Alejandra[1], Ramos Jorge Guillermo[1], Ballina Ariel[2], Maillo Martín[2], Bernal Maria Florencia[3], Bergero Miguel[3], Calafell Gabriela[3], Cayol Federico[4], Bossio Juan Carlos[5], Varayoud Jorgelina[1].
[1]Instituto de Salud y Ambiente del Litoral (ISAL) – Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET) – Facultad de Bioquímica y Ciencias Biológicas (FBCB) - Universidad Nacional del Litoral (UNL), Santa Fe, Argentina.
[2]Hospital Cullen, Santa Fe, Argentina.
[3]Sanatorio Privado San Gerónimo. Centro Oncológico J.B. Iturraspe, Santa Fe, Argentina.
[4]Hospital Italiano, Buenos Aires, Argentina.
[5] Cátedra de Epidemiología y Salud Pública, FBCB-UNL, Jefe del Departamento de Programas de Salud del Instituto Nacional de Enfermedades Respiratorias (INER), Santa Fe, Argentina.

Patients with cancer are high-risk population in the COVID-19 pandemic. We aimed to describe clinical characteristics of patients with cancer and COVID-19 in city of Santa Fe and Buenos Aires. We did a cross-sectional study of 80 patients with laboratory-confirmed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and with a pathological diagnosis of a malignant tumor from hospitals of city of Santa Fe and from Hospital Italiano (Buenos Aires). Clinical characteristics and cancer histories were investigated and analyzed.
During the study, 80 patients with cancer and laboratory-confirmed SARS-CoV-2 infection were included (median age 65 years). 45 (56,2%) patients were women and 34 (42,5%) were men. 72 patients had solid tumours and 3 patients had haematological malignancies. The most frequently symptoms were fever (29 [40,28%] patients) and cough (28 [38,89%] patients); then dyspnoea (18 [25%] patients) and fatigue (19 [26,36%] patients). The most common solid tumor types were breast (20 [26,7%] patients) and lung cancer (8 [10,7%] patients). 33 (41,25%) of 80 patients had comorbidities: 11 (20,37%) had coronary disease, 10 (18,52%) had diabetes, 7 (12,96%) had chronic kidney disease and 5 (9,26%) chronic obstructive pulmonary disease. The rate of mortility was of 18.75%.
In addition, if we analyze how many patients did not survive among the cases with severe pneumonia (41,86%) and compare them with non-severe pneumonia (16,28%), it gives an OR of 20.25 (2.32, 176.6, p = 0.0028), indicating that severe pneumonia could be a risk factor of mortality. Current statistics data from Argentina indicate that the rate of mortility of COVID-19 patients was 2% (114000 of 5240000). The data from our analysis indicates that cancer and COVID-19 patients have an 8 times higher risk of death than patients with only COVID-19.
Cancer patients have deteriorating conditions and together with a high frequency of comorbidities, these patients become a vulnerable population. It would be a priority that patients with cancer and COVID-19 infection receiving regular screening and preventives therapies.

505. (516) **DENDRITIC CELL PROFILE IN TUMORAL MICROENVIRONMENT OF HUMAN BREAST CARCINOMAS**

Clara Garcia Samartino1, Cintia Celina Vaquer1, Rodrigo Militello1,2, Diego Santoni1, Leandro Sarrio3, Ignacio Cebrián1,2, Emanuel Martin Campoy1,2.
1 Facultad de Ciencias Médicas, UNCUYO, 2 Instituto de Histologia y Embriologia Mendoza (IHEM)-CONICET, 3 Anatomía Patológica, Hospital Central de Mendoza.

Argentina has the second highest mortality rate for breast cancer (BC) in South America. The immune cells present in the tumor microenvironment (TME) performs dual functions, being able to eliminate or promote malignancy according to the signals present. We believe that the dendritic cells (DCs) found in the TME play a fundamental role in the development of the mammary tumor. Our hypothesis is that there are different subpopulations and maturation profiles of DCs in the TME, and these profiles are associated with tumor traits of mammary carcinomas. Initially, we propose as a general **objective** to study the different types of DCs present in the TME and to determine the profile of these different subtypes in human breast carcinomas. **Methodology**: Once the tumors were obtained by surgery, breast carcinoma-derived fractions were mechanically and enzymatically disaggregated. Tumoral (EpCAM+) and non-tumoral (EpCAM-) populations of each fraction were isolated using cell sorting flow cytometry. DC populations were characterized by flow cytometry using the HLA-DR, CD14, CD11c, CD1c cell surface markers to perform the gating strategy. **Results**: We obtained tumoral and non-tumoral populations derived from eight human breast carcinomas fractions. We defined four different DC subpopulations present in the TME: pDCs, inflammatory DCs, cCD1 and cCD2 DCs. Interestingly, we observed that each tumoral fraction has a unique DC profile, according to the high heterogeneity already described for this type of tumor. Based on the tumor cohort analysis, we evidenced a negative correlation between tumor cell and cDC2 DC populations (r= -0.76, p<0.027). **Conclusion**: Breast TME contains different DC profiles associated with tumoral cell proportions in human breast carcinoma fractions. Ongoing and future experiments will allow us to determine the maturation profiles of these DCs and analyze their relationship with genomic/epigenomic and clinicopathological tumor characteristics.

506. (525) **ENZALUTAMIDE AND NOTCH PATHWAY INHIBITION RESTRAIN PROSTATE CANCER CELL GROWTH AND MIGRATION**

Agustina Chimento[1], Nadia Bonadeo[1], Sofía Perrone[1], María Lucia Romano[1], Ana Laura Fontanazza[2], Kurt Villalba[3], Licina Tessone[4]; Fernanda Parenti[5], Carolina Cristina[1].
[1]CIBA – CITNOBA, UNNOBA – CONICET, J. Newbery 261, Junín, Provincia de Buenos Aires; [2]HIGA Abraham Félix Piñeyro, Junín, Provincia de Buenos Aires; [3]Clínica Centro, Junín, Provincia de Buenos Aires; [4]Laboratorio de Patología Dr Alberto Petraglia, Junín, Provincia de Buenos Aires; [5]Centro Medico Famyl, Junín, Provincia de Buenos Aires.

Prostate cancer (PCa) remains among the leading causes of cancer-related deaths in men. Standard therapies for castration resistant prostate cancer (CRPC) include second-generation anti-androgens, such as Enzalutamide (Enz), which prolong patient lifespan. Emerging evidence indicates a regulatory role of Notch signaling in prostate development and growth.
In this work, we aimed to study the Notch and AR pathway involvement and their interaction in prostate cancer.
We first determined the expression of Notch receptors (1/2/4) and the proliferation marker PCNA by IHQ in prostate tumor samples obtained by surgery and its association with Gleasson score.
In prostate cancer PC3 cells, we demonstrated AR expression by RT-qPRC. Instead, PSA expression was absent when evaluated culture cell supernantants by chemiluminescence. Moreover, under