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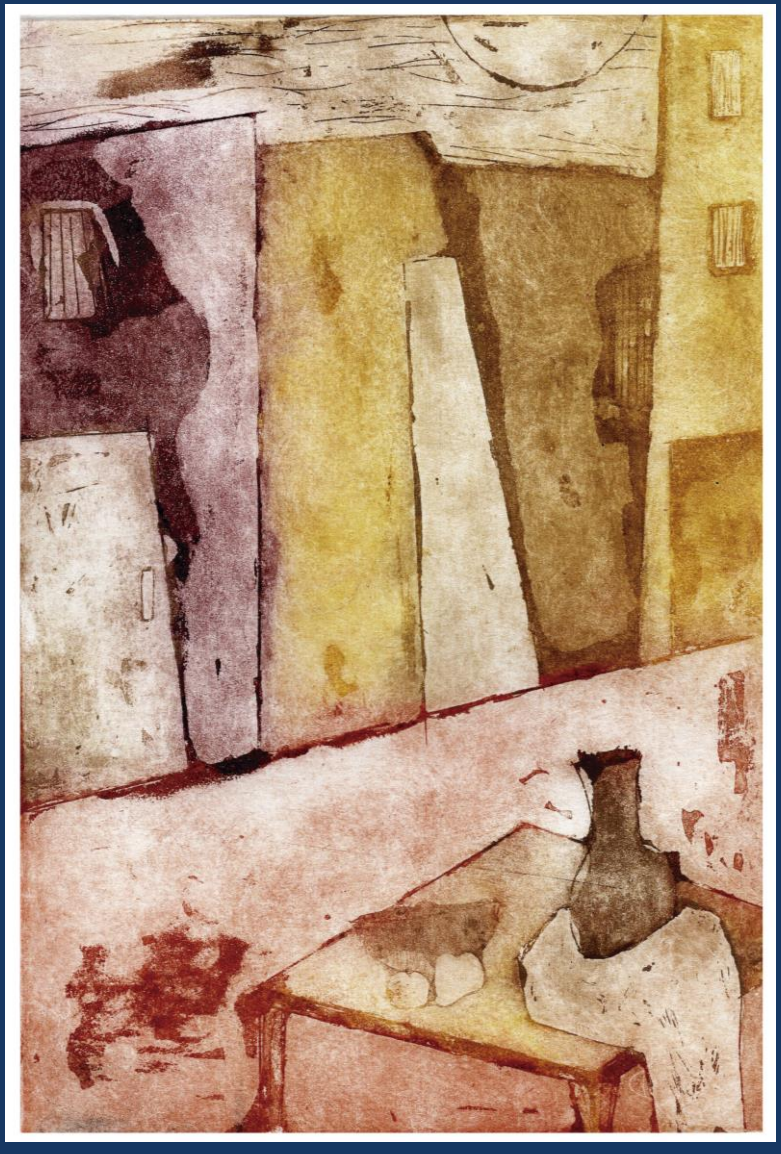

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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REUNIÓN ANUAL DE SOCIEDADES DE BIOCIENCIA 2019

LXIV Reunión Anual de la
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LI Reunión Anual de la
Asociación Argentina de Farmacología Experimental (SAFE)

XXI Reunión Anual de la
Sociedad Argentina de Biología (SAB)

XXXI Reunión Anual de la
Sociedad Argentina de Protozoología (SAP)

IX Reunión Anual de la
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

VI Reunión Científica Regional de la Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

con la participación de
The Histochemical Society

13 - 16 de noviembre de 2019
Hotel 13 de Julio - Mar del Plata

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VI Regional Scientific Meeting of Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

with the participation of
The Histochemical Society

November 13th – 16th, 2019
Hotel 13 de Julio - Mar del Plata

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INSTITUTO DE INVESTIGACIONES EN MEDICINA TRASLACIONAL, CONICET-UNIVERSIDAD AUSTRAL

Abstract/Resumen: Toxicity from and resistance to ionizing radiation therapy constitutes a major obstacle to curative treatments for non-small cell lung cancer (NSCLC). Regimens for radiation therapy are often limited by toxicity to normal tissues and the development of resistance. Thus, strategies to reduce the total amount of ionizing radiation (IR) used are required. IR results in a wide variety of chromosomal DNA damage including DSB. Epigenetics refers to a set of mechanisms that regulate chromatin accessibility and therefore DNA-Based process such as DNA repair. Particularly, Jumonji (JmjC) histone lysine demethylases (KDM) play roles in DNA repair pathways. Our aim is to study if pharmacological inhibiton of JmjC could be used as a targeted therapy to radiosensitize NSCLC. Liquid colony formation assay was performed to determine IC50 of JIB-04, a JmjC pan-inhibitor, and radioresponse curves in Human NSCLCs cell lines (H1299, A549, HCC95 and HCC1719) and immortalized non-cancerous human bronchial epithelial cells (HBEC3KT and HBEC30KT). For in vivo experiments NSCLC cells were injected subcutaneously (H1299 and A549) into the right posterior leg of female athymic nude mice. Mice were treated for a total of 12 doses EOD with JIB-04 (50 mg/kg/day) by oral gavage or with vehicle; radiation was administered 4 hours after treatment. Tumor growth delay, survival and the dose enhancement factor (DEF) were then determined. Pharmacological inhibition of JmjC KDM using JIB-04 resulted in a strong sensitization of radio-resistant NSCLC (H1299, A549, HCC95) ($p<0.001$) but not radio-sensitive NSCLC (HCC1719) to radiation. In addition, we found that JIB-04 does not radiosensitize normal cells (HBEC3KT and HBEC30KT). In vivo, treatment with JIB-04 plus IR inhibit tumor growth compared with control mice and either treatment alone ($p<0.001$, DEF>6). Even more, mice treated with JIB-04 and IR survived significantly longer than mice treated with either agent alone or with vehicle even after the end of treatment. In conclusion, our study suggests that the epigenetic inhibitor JIB-04 could help to overcome radioresistance both in vitro and in vivo.

0855 - EVALUATION OF CIRCULATING LYMPHOCYTES SUBPOPULATIONS DURING THE GROWTH OF M-406 TRIPLE NEGATIVE MURINE MAMMARY TUMOR

Antonela DEL GIÚDICE | María Celeste CAPITANI | Matías Ezequiel FUSINI | Leandro E MAINETTI | Olga Graciela SCHAROVSKY | María Jose RICO | Viviana Rosa ROZADOS

INSTITUTO DE GENÉTICA EXPERIMENTAL. FACULTAD DE CIENCIAS MÉDICAS. UNIVERSIDAD NACIONAL DE ROSARIO

Abstract/Resumen: Inbred mice models provide an interesting tool for identifying factors that control susceptibility to breast cancer. M-406 mammary adenocarcinoma appeared in an inbred CBi mouse. CBi- mice were artificially selected from CBi. Cells of the immune system play an important role in tumor development. In order to determine their participation on tumor growth in genetically different hosts, CBi, CBi- and F1 reciprocal hybrids (F1A: CBi x CBi- and F1B: CBi- x CBi) were s.c. challenged with M-406, tumors were measured, and blood samples were taken on days 0, 7 and 14 in CBi and F1 and on days 0, 5, 8 and 12 in CBi- mice. Circulating CD4+, CD8+, Treg and Th17 cells were quantified (flow cytometry). Tumors grew exponentially in 100% of CBi (susceptible) and F1 female and male mice. However, in CBi- (resistant) after a short period of growth, reaching the maximum size on day 8 (female) and 12 (male), 100 % of tumors were rejected. CBi, F1A and F1B mice, did not differ in tumor volume doubling time (TVDT) for both sexes, while in CBi-, TVDT in males was higher than in females ($p<0.05$). We determined the ratio CD8+/Treg in CBi males: day 0 > day 14; ($P<0.05$); CBi females: day 0 day 12 ($p<0.01$) without differences in CBi- females; F1A males and females: day 7 > day 14 ($p<0.0001$; $p<0.001$, respectively); F1B: without differences between days or sexes. Conclusions: 1) The susceptible phenotype is dominant over the resistant. 2) CD4+ and Th17 lymphocytes could not explain tumor growth/rejection behavior in genetically different hosts. 3) CBi males and females utilize different antitumor immune mechanisms leading to tumor escape and growth, without modifying tumor growth rate. 4) The decrease in CD8+/Tregs ratio in CBi- males could be partly responsible for the observed delay in tumor growth. 5) The similar values in CD8+/Tregs ratios for F1A and F1B (males and females) could explain, in part, the absence of differences in tumor growth rate.

0865 - ADRENERGIC RECEPTORS IN BREAST CANCER: DIFFERENTIAL EFFECTS OF ALPHA 2A AND 2C-ADRENERGIC RECEPTOR EXPRESSION ON TAMOXIFEN SENSITIVITY IN STABLY TRANSFECTED LUMINAL MCF-7 CELLS.

Evangelina APARICIO (1) | Ezequiel RIVERO(2) | Carlos David BRUQUE(3) | M. Sol RODRIGUEZ(1) | Ariana BRUZZONE(4) | Cecilia PEREZ-PIÑERO(1) | Alicia Isabel LÜTHY(1)

IBYME-CONICET (1); CENTRE FOR GENOMIC REGULATION (2); ANLIS-MALBRÁN (3); INSTITUTO DE INVESTIGACIONES BIOQUÍMICAS BAHÍA BLANCA INIBIBB -CONICET (4)

Abstract/Resumen: Breast cancer is the most frequently diagnosed and leading cause of cancer death among women worldwide. Epinephrine and norepinephrine, released during stress, bind to 9 different adrenoceptors. Our group has already described (SAIC 2015, poster 660) by in silico analysis in a great database that patients with high expression of Alpha2A-adrenoceptors (A2A-AR) have better disease-free survival than those with lower expression, mainly in luminal tamoxifen-treated ones. Contrarily, a high expression of Alpha2C-AR was associated with worse outcome in luminal B but not in luminal A patients. The aim of the present work was to study the sensibility of tamoxifen on A2A or A2C-AR-overexpressing cells. The human luminal breast cancer MCF-7 cells were stably transfected with A2A or A2C-AR or the empty vector. The expression of A2-AR and Estrogen Receptor Alpha (ER) was measured by RT-qPCR, the sensitivity to tamoxifen by tritiated thymidine incorporation and ER, progesterone receptor and pERK relative to ERK, by Western Blot. They were all performed in the absence of adrenergic stimulation because catecholamines released during stress bind to all receptors and no specific ligand for individual A2-AR exists yet. We successfully over expressed alpha2A and alpha2C on MCF-7 cells: 65 (A2A) and 28 % (A2C) increase compared with empty vector (pCDNA, $p<0.05$ and $p<0.01$, respectively). When analyzing the sensitivity to tamoxifen treatment, the A2A cells exhibited an EC50 of $2.867e^{-}10$ vs. 4.250 e-10 of pCDNA, $p<0.01$; while A2C of $1.202e^{-}9$, $p<0.001$.This was accompanied by a decrease in both cases of ER levels measured by RT-qPCR, $p<0.05$ and WB. A2A cells also showed diminished cell proliferation ($p<0.01$) in the absence of any stimulation when compared with pCDNA and A2C. We suggest that the increase of tamoxifen sensitivity in A2A cells could be due to the combined effect of inhibiting ER expression and cell proliferation.

0871 - 4-METHYLUMBELLIFERONE INDUCES SENESCENCE, INHIBITS MIGRATION AND MODULATES CD44 AS WELL AS RHAMM IN HUMAN GLIOBLASTOMA CELL LINES

Daniela POODTS (1) | Matías PIBUEL(1) | Mariángeles DÍAZ(1) | Yamila MOLINARI(2) | Élida ÁLVAREZ(1) | Silvia HAJOS(1) | Paula FRANCO(2) | Silvina LOMPARDIA(1)

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