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Daniela Kantor. Médanos, 2018

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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.

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LN229 cells, respectively.
These results highlight PIN1 implication in tumor progression and telomere maintenance in glioblastoma, suggesting this mechanism as a new potential therapeutic approach for the treatment of this disease.

494. (426) **SYNERGISTIC APOPTOTIC EFFECT OF 2´NITROFLAVONE COMBINED WITH SAFINGOL IN MURINE MAMMARY TUMOR CELLS**

Juan Manuel Anselmi Relats, Leonor Roguin, Mariel Marder, Julieta Marino, Viviana Blank

In a previous work, we have demonstrated that the synthetic flavonoid 2´-nitroflavone (2NF) is a potent and selective antitumor agent *in vitro* and *in vivo* in a murine LM3 breast cancer model. Sphingosine kinase 1 (SphK1), a lipid kinase overexpressed in some mammary tumor cells, regulates the balance between proapoptotic ceramides and prosurvival sphingosine-1-phosphate (S1P). Furthermore, safingol, a competitive SphK1 inhibitor, prevents catabolism of ceramides, contributing to tumor cell death. It has been reported that certain flavonoids exert antitumor activity through an increment in ceramide levels. Thus, we explored the antiproliferative effect of the simultaneous incubation of 2´NF and safingol in LM3 cells and found that they synergistically inhibited cell proliferation. Based on these results, we studied the apoptotic effect of 2NF in combination with safingol. When we evaluated the expression of bax proapoptotic protein, results obtained by Western blot showed that each compound alone did not modify bax expression, but a fivefold increase was observed after cell incubation with both 2NF and safingol for 24 h ($p<0.001$). Similarly, results obtained by flow cytometry showed a higher increment in the percentage of hypodiploid cells after simultaneous exposure to both compounds ($p<0.001$, 24h and $p<0.05$, 48 h). As recent reports demonstrated that some flavonoids inhibit SphK1 activity interacting directly with the kinase, we performed molecular docking analysis with the docking web server SwissDock. Results obtained showed that safingol and 2NF would bind to different molecular regions of the SphK1.
Based on the synergistic antiproliferative and apoptotic effects of 2NF in combination with safingol, we propose that the interaction of both molecules with different sites of SphK1 would favor the generation of apoptotic ceramides by inhibiting the formation of S1P. Further experimental approaches should be performed to confirm a direct interaction of 2NF and SphK1.

495. (453) **GADD45A AND CDKN1A, AMONG OTHER GENES, ARE EPIGENETICALLY REGULATED IN PROSTATE TUMORS FROM PATIENTS**

Lara Castagnola, Karen Graña, Rocío Belén Duca, Paula Lucía Farré, Georgina Daniela Scalise, Cintia Massillo, Adriana De Siervi.
Laboratorio de Oncología Molecular y Nuevos Blancos Terapéuticos, IBYME- CONICET.

DNA methylation, histone modifications and miRNAs regulation are important epigenetic mechanisms of gene regulation that can be detected before prostate cancer (PCa) becomes invasive, suggesting they are pivotal events in tumor initiation and progression. Metabolic syndrome (MeS) increases PCa's risk and aggressiveness. Previously, using MeS NSG male mice injected *s.c.* with PC3 PCa cells, we demonstrated that *DNMT1* and *SUV39H1* expression levels were repressed, while *RIZ1* and *GADD45A* were increased in prostate tumors from MeS mice compared with control. Also, mRNA levels of *CDH1* and *ZEB1*, two DNMT1 targets, were repressed and induced, respectively, in these tumors. Our hypothesis is that aberrant epigenetic changes in PCa favor tumor development and progression. Here we investigated expression and methylation levels of several epigenetic genes in PCa patients using Xena database: *DNMT1*, *SUV39H1*, *EZH2*, *DNMT3A*, *DNMT3B*, *TET2*, *TET3*, *RIZ1*, *EP300*, *HDAC2, SIRT1*, *HBO1*, *CDH1*, *ZEB1*, *GADD45A* and *CDKN1A.* We found that
GADD45A and *CDKN1A* mRNA levels were significantly diminished in prostate tumors compared to normal adjacent tissue (NAT). Accordingly, methylation levels of *GADD45A* and *CDKN1A* were significantly increased in comparison with NAT and between Gleason Scores. Moreover, expression and methylation levels of other genes involved in epigenetic changes were significantly altered in prostate tumors vs NAT and between Gleason Scores. Additionally, to further investigate epigenetic changes mediated by miRNAs, we assessed the expression levels of a panel of miRNAs involved in PCa development. Altogether these data showed that aberrant expression and methylation of epigenetic genes, particularly the correlation between the increased methylation and decreased expression levels of *GADD45A* and *CDKN1A* in prostate tumors from patients, might be consider as promising mechanisms to further investigate in PCa aggressiveness and even identifying novel biomarkers for patient prognosis.

496. (456) **COUMARIN 4-METHYLUMBELLIFERONE (4MU) REDUCES THE RESISTANCE TO CONVENTIONAL CHEMOTHERAPY AND THE TUMORIGENIC CAPABILITY OF CD133+ LUNG CANCER CELLS**

Flavia Piccioni[1], Mariel Fusco[1], Marco Aurelio Díaz Gutierrez[1], Fernando Gayet Preiss[2], Paula Rosello[1], Manglio Rizzo[1,2], Mariana Malvicini[1].
[1]Laboratorio de Inmunobiología del Cáncer Instituto de Investigaciones en Medicina Traslacional (Universidad Austral-CONICET), [2] Servicio de Oncología del Hospital Universitario Austral

Patients with non-small cell lung cancer (NSCLC) will ultimately progress or relapse after treatment with conventional chemotherapy (Qx) with platinum-taxanes. In the tumor microenvironment (TME) cancer stem cells (CSC), which express CD133, CD44 and CD47 among other markers form residual cell niches involved in the recurrence after treatment. Hyaluronan (HA), a component of the TME, regulates, at least in part, the function of CSCs. We previously demonstrated that tissue sections from murine Lewis Lung Carcinoma (LLC) tumors present high levels of HA and it is higher on isolated LLC CSCs (CD133+) compared with the non-CSCs population (CD133-; by FACS). Our analysis of TCGA data from patients with NSLCS showed that HA Synthase (HAS) 3 expression strongly correlates with levels of transcription factors involved in CSC phenotype. We modulated HA with the coumarin 4Mu, detecting an increase in the sensibility of LLC cells to paclitaxel (Pa). We aimed to cross-validate the TCGA findings on whole LLC and isolated CD133+ cells after exposure to Pa or cisplatin (Cis), alone or in combination with 4Mu. We analyzed the expression of HAS and CSCs genes by qPCR. The expression of HA and their clonogenic and tumor-forming capability was also evaluated. Pa increases mRNA levels of HAS3 ($p<0.05$) and HAS2 ($p<0.01$) while LLC treated with Pa+ 0.25 mM 4Mu showed reduced HAS3 levels($p<0.05$). 4Mu also reduced HA levels produced by LLC. CD47 and SOX2 gene expression were enhanced by Pa while they decreased with Pa+4Mu ($p<0.05$). About 8.53 ± 0.35% of LLC are CD133+, and express more HA compared to CD133- ($p <0.05$). Viability of CD133+ cells decreased when treated with 4Mu+ Qx (Pa: $p<0.01$, Cis: $p<0.05$). Remarkably, 4Mu reduced the clonogenic ($p<0.05$) as well as tumor-forming ability of LLC CD133+ treated with Pa and Cis ($p<0.05$). We suggest that 4Mu improves the efficacy of Qx to inhibit tumor progression and could be implicated in preventing tumor recurrence.

497. (457) **A BRIEF REPORT OF COVID-19 CASES IN CANCER PATIENTS FROM AMBA: DESCRIPTION OF HOSPITALIZED POPULATION AND THEIR IMMUNITY AGAINST SARS-COV-2**

Marco Aurelio Díaz[1], Mariel Fusco[1], Carlos Rafael Picón[2], Ana Bezazián[1], Nicolás Marcolini[2], Laura Alvarez[3], Pablo Brenzoni[3], Esteban Fiore[4], Paula Roselló[4], Pablo Mandó[5], Alex Kostianovsky[5], Guillermo Bizantino[6], Marcelo Rodriguez[6], Matías Tisi Baña[7], Carlos Silva[2], Manglio Rizzo[1,2], Diana Hansen[8], Mariana Malvicini[1]
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BACKGROUND: Until today there are 229,414,751 registered cases and 4,707,872 deaths attributable to the disease caused by the new severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) called COVID-19, all over the world. COVID-19 can be asymptomatic or cause a respiratory distress syndrome and death. The risk of COVID-19 and mortality for COVID-19 have been associated with older age and comorbidities, such as cancer. The situation is a great challenge for these patients. Furthermore, in patients with active treatment, the decrease in cell populations involved in protection is very common and immunity against SARS-CoV-2 may be impaired. OBJECTIVES: Here we described the population of oncological (P On) and non-oncological (P NOn) patients with moderate or severe COVID-19 hospitalized at the HUA from December, 2020 to September, 2021. Peripheral blood samples were collected at different times to analyze the duration of the protective response (Elisa; flow cytometry). RESULTS: We incorporated 24 patients: 11 [45%] with oncological disease in treatment, 5 male median age 59 with lung (4) and scalp (1) cancer and 6 female median age 36 with ovarian (1), cervix (2), renal (1) rectum (1) and breast cancer (1) with moderate (9) or severe (2) COVID-19 and 13 patients without oncological disease [54%], 7 male median age 59 years, 6 female median age 40, with moderate (12) or severe (1) COVID-19. The POn group had a longer hospital stay (15.9 vs 8.8 days; p 0.016) and higher oxygen requirement (high flow 36.4% vs 7.7%; p 0.085). The mortality rate in POn was 36% and there were no deaths in PNon. The circulating immune response for SARS-CoV-2 was analyzed in 50 samples at different times from the 24 hospitalized patients. The protective response was significantly lower in the POn population ($p < 0.001$) with low detection of IgM and IgG. CONCLUSIONS: in POn the protective response is lower compared to PNon, with probable implications in morbidity and mortality.

498. (458) **ANALYSIS OF RUNX-CBFβ ACTIVITY ON BASAL BREAST CANCER PROGRESSION THROUGH DIRECT CONTROL OF RSPO3 EXPRESSION**

Carla M Felcher*[1], Micaela N Stedile*[1], Ana Ortiz[1], Camila D Arcuschin[1], Johanna M Tocci[1], Emilia S Bogni[1], Sabrina A Vallone [1], Ignacio E Schor [1], John H Bushweller [2], Lucio H Castilla [3], Edith C Kordon [1,4].

1 Instituto de Fisiología, Biología Molecular y Neurociencias-CONICET-UBA Argentina, 2 University of Virginia-USA, 3 University of Massachusetts-USA, 4 Departamento de Química Biológica-Universidad de Buenos Aires.

** Carla M. Felcher and Micaela N Stedile contributed equally to this study.*

We have determined that R-spondin3 (RSPO3), a secreted protein that potentiates Wnt signaling pathway, is a key modulator of tumor progression and stem cell behavior in basal breast cancer. Previous reports indicated the potential role of RUNX1-CBFβ axis on Rspo3 expression in mammary tumor cells. Besides, we found that treating basal breast cancer cells with small molecules that block the interaction between CBFβ and RUNX reduced Rspo3 mRNA and protein levels. These treatments also induced inhibition of cell migration, an ability that was recovered upon addition of recombinant RSPO3. Also, we have determined that those inhibitors enhanced the effects of the chemotherapeutic drug Doxorubicin on MDA-MB231 cell survival and migration. To determine whether there is a direct control of RUNX1-CBFβ on Rspo3 mRNA transcription, we performed an *in silico* analysis of publicly available data from two RUNX1 CHIP-seq reports and an ATAC-seq study from human breast cell lines. We aligned the emerging data with the occurrences of the RUNX1 DNA-recognition-motif in the Rspo3 locus. A few putative RUNX1 binding sites were revealed by this analysis. Among them, a region located on Rspo3 first intronic region that seems to be particularly active in triple negative breast cancer cells. Importantly, we have determined that RUNX1 actually binds to that site in MDA-MB231 cells by ChIP-qPCR assay (3-fold increase compared to control). Then, to further explore the mechanisms underlying the control exerted by RUNX-CBFβ on Rspo3 expression, we proceeded to delete that RUNX1 binding site by introducing, through electroporation, Cas9 protein together with specific sg-RNAs in MDA-MB231 cells. We are currently analyzing the phenotype of the obtained cells to verify the relevance of the discovered RUNX1 binding site. In summary, our results confirm that RUNX-CBFβ axis may contribute to basal breast cancer progression by directly inducing Rspo3 expression in mammary tumor cells.

499. (493) **MECHANISMS ASSOCIATED WITH CYTOSINE DEAMINASE::URACIL PHOSPHORIBOSYL TRANSFERASE/5-FC SUICIDE GENE SYSTEM BYSTANDER EFFECT IN HUMAN MELANOMA**

Bernabei-Cornejo SG[1], Allende JB[1], Glikin GC[1], Finocchiaro LME[1]

1. Unidad de Transferencia Genética, Área Investigación, Instituto de Oncología "Ángel H. Roffo", Universidad de Buenos Aires.

We have previously shown the therapeutic potential of yeast cytosine deaminase::uracil phosphoribosyl transferase/5-fluorocytosine (CDU/5-FC) suicide gene (SG) system in 8 human melanoma cell lines. CDU catalyzes 5-FC conversion to 5-fluorouracil (5-FU), which interferes with RNA processing and DNA synthesis. The aim of the present work was to explore CDU lipofected-cells ability to release particulate factors and to analyze its contribution to the cytotoxic effect.

Methods: Five melanoma cell lines (A375, M8, hM1, hM4 and hM9) were used. After lipofection, lipoplex-rich medium was reserved and cells were washed thrice with medium, to remove remaining lipoplexes. After the last wash, cells were cultured in fresh medium. Conditioned media (CM) was obtained after 48 h of incubation from CDU- and HSVtk-lipofected cells with or without 5-FC and from unlipofected cells with or without 5-FU. The supernatant (SN) and pellet (P) fractions of the CM were obtained by centrifugation at 12000 × g for 60 min. These fractions were incubated on receptor cells with or without 5-FC, and cell viability was determined by the APH method after 5 days.

Results: As expected, the SN fraction of 5-FU CM was accountable for bystander cytotoxicity, while the P fraction was not. In most cell lines only the SN fraction of CDU/5-FC treated cells was responsible for cytotoxicity, presumably given by 5-FU presence on the CM. Interestingly, the addition of 5-FC on cells that received both fractions of CDU CM caused significant cytotoxicity ($p<0,05$), indicating transfer of the enzyme or its gene. While CDU lipoplex-rich medium was able to deliver enzyme or its gene ($p<0,05$), the last-wash medium was unable to do it, denoting that there were no remaining lipoplexes on CM.

Conclusion: The CDU/5-FC suicide gene therapy system bystander effect would be mainly attributable to 5-FU and to the CDU fusion enzyme or its coded information (plasmid DNA o mRNA) carried by particulate subcellular fractions.

500. (506) **THE ANDROGEN RECEPTOR REGULATES RUNX1, A NEW POTENTIAL WAY TO TARGET CHEMOTHERAPY RESISTANT TRIPLE NEGATIVE BREAST CANCER**

Natalia B Fernández1, Sofía M Sosa1, Justin T Roberts2, Jennifer K Richer3 and Natalia Rubinstein1

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2 University of Colorado; School of Medicine; Department of Biochemistry and Molecular Genetics.

3 University of Colorado Anschutz Medical Campus; Department of Pathology.

Triple negative breast cancer (TNBC) is an aggressive breast cancer (BC) subtype for which no effective targeted therapies are avail-