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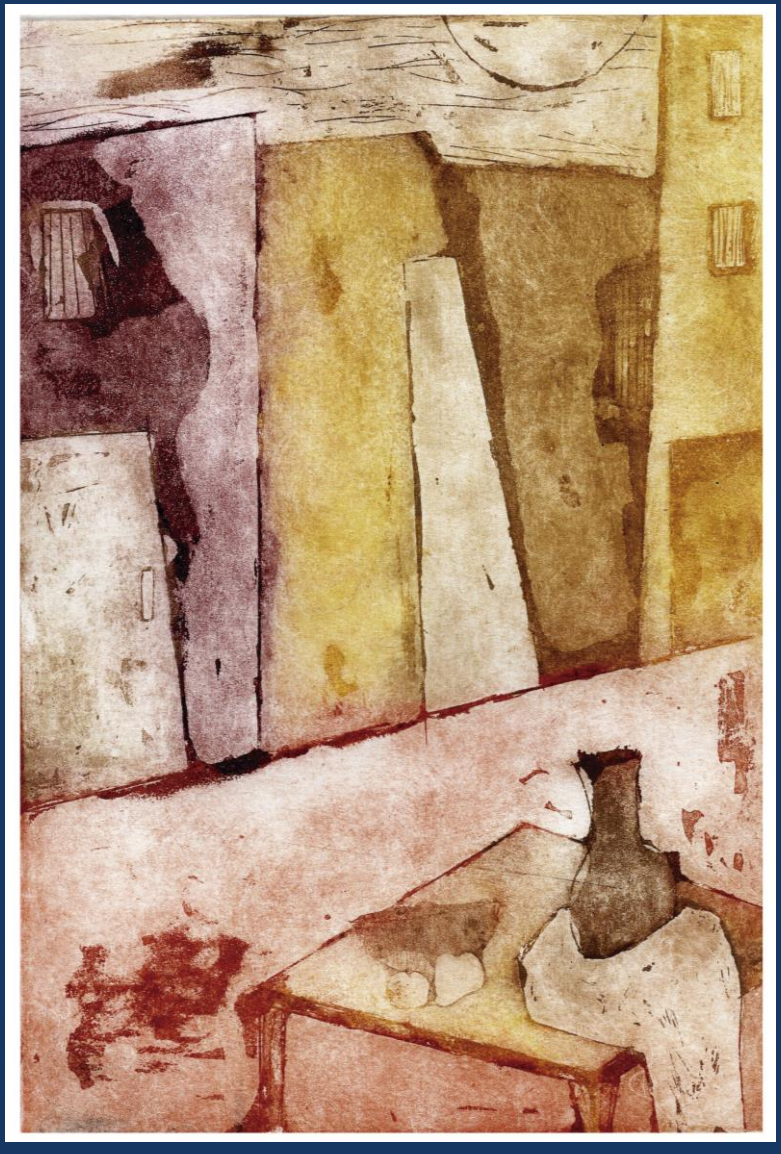

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La Tapa (Ver pág. 4)
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REUNIÓN ANUAL DE SOCIEDADES DE BIOCIENCIA 2019

LXIV Reunión Anual de la
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LI Reunión Anual de la
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XXI Reunión Anual de la
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XXXI Reunión Anual de la
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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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ANNUAL MEETING OF BIOSCIENCE SOCIETIES 2019

LXIV Annual Meeting of
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IX Annual Meeting of
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

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
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to elucidate if such forms of HO-1 impacts on head and neck cancer cells behavior. We established the FL-HO1 and t-HO1 overexpressing HN13 cells. We evaluated cell viability by crystal violet method, cell cycle progression by propidium iodide staining and flow cytometry, and cell migration by wound healing assay. In addition to our previous results using hemin, we found that 80 µM hemin increased cell number in S- ($p<0.001$) and G2/M ($p<0.001$) phases and diminished cell number in Go/G1 phase ($p<0.001$) at 72h. We also found that hemin delayed cell migration ($p<0.01$) respect to control. On the contrary, at same conditions, hemin failed to increase cell viability ($p>0.05$) neither alters cell cycle progression ($p>0.05$) in the normal keratinocyte cell line, HaCaT. By a genetic approach, we found that FL-HO1 HN13 cells have a higher growth rate ($p<0.001$) than its control and cell cycle progression is as similar as ($p<0,001$ vs control) it was observed with hemin treatment. However, FL-HO1 failed to alter migratory capacity ($p>0.05$). We also found that t-HO1 expression impaired HN13 cell viability ($p<0.01$ vs. FL-HO1 HN13) and induces a Go/G1 arrest ($p<0.01$) and a diminished cell number in SubGo ($p<0.01$) and S- ($p<0.05$) phases. Also, we found that t-HO1 expression delayed cell migration ($p<0.001$) respect to FL-HO1 HN13. In conclusion, our results show that head and neck cancer cells survival, cell cycle progression and migration capacity depends on predominant HO-1 form.

0407 - NOVEL CALCITRIOL ANALOGUES EM1 AND UVB1 AGAINST AGGRESSIVE BREAST CANCER CELLS AS A MONOTHERAPY OR IN COMBINATION WITH PACLITAXEL.

Josefina Alejandra GUEVARA (1) | Giuliana PAOLILLO(1) | Agustina IBARRA(1) | Eliana Noelia ALONSO(1) | Enrique Javier ARENAS LAHUERTA(2) | Mercedes NADAL SERRANO(2) | Joaquín ARRIBAS(2) | Cristina BERNADÓ MORALES(2) | Yagamare FALL(3) | Evangelina MASCARÓ(4) | Cristian VITALE(4) | Alejandro Carlos CURINO(1) | María Marta FACCHINETTI(1) | María Julia FERRONATO(1)

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Abstract/Resumen: Despite chemotherapy remaining as a primary therapeutic option for aggressive breast cancer (BC), its effectiveness is limited by intrinsic or acquired resistance and associated adverse effects. Therefore, new therapeutic strategies are needed. Previously, we demonstrated that the calcitriol analogue EM1 decreases the viability, migration and invasion of the 4T1 triple-negative BC (TNBC) cells. Additionally, we reported that UVB1, another calcitriol analogue synthetized by our group, reduces the viability of cells derived from the TNBC - Patient-Derived Xenografts (PDX). Hence, the aim of the present study was to continue evaluating the antitumoral effects of the calcitriol analogues EM1 and UVB1 on aggressive BC cells, alone or in combination with low concentrations of Paclitaxel (PTX). We found a synergistic effect by combining EM1 or UVB1 with non-effective PTX concentrations on viability of 4T1 cells. The resulting Combination Index values of Chou & Talalay method were 0.80059 and 0.13491 for EM1-PTX and UVB1-PTX combinations, respectively. In addition of our previous result on 4T1 cell migration, EM1 displayed antimigratory effects on MDA-MB-231 TNBC cell line ($p<0.001$). In contrast, UVB1 had no effect on these cells. However, interestingly, the combination of the analogues with non-effective concentrations of PTX over 4T1 cell migration displayed a better effect than drugs alone (EM1-PTX: $p<0.05$; UVB1-PTX: $p<0.001$). Finally, a pilot in vivo assay was conducted to test the sensitivity of the TNBC-PDX410 to UVB1. A reduction in in vivo tumor volume was detected after 18 days of UVB1 treatment at 40 µg/kg of body weight administrated three times a week ($p<0.05$). Altogether, these results suggest the potential use of these vitamin D analogues as antitumor agents, alone or as a complement to conventional chemotherapy.

0408 - ANTITUMORAL EFFECTS OF PLEUROTUS OSTREATUS I-FRACTION IN BREAST CANCER

Rocío RAMBURGER (1) | María Julia FERRONATO(1) | Josefina Alejandra GUEVARA(1) | Juan Manuel CUESTAS(2) | Pablo Daniel POSTEMSKY(2) | Alejandro Carlos CURINO(1) | María Marta FACCHINETTI(1) | Eliana Noelia ALONSO(1)

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Abstract/Resumen: Mushrooms are considered as "small pharmaceutical factories" producing hundreds of bioactive compounds, many of which have shown to exert antitumoral activity in different types of cancer. Argentina has a high mushrooms diversity and important scientific-technological development applied to its cultivation. However, the antitumoral phytotherapeutic potential of edible mushrooms cultivated in our country has not yet been considered. In this context, the purpose of the current study is to determine the antitumoral activity in breast cancer of Pleurotus ostreatus I-Fraction, an extract of water-soluble polysaccharides obtained from fruiting body, initially evaluating its potential immuno-independent antitumoral activity. To achieve the proposed objective, we employed a murine mammary adenocarcinoma 4T1 cells line and performed cell viability assays by colorimetric assay with crystal violet, cell cycle analysis by flow cytometry, and wound healing assay. We found that P. ostreatus I-Fraction at concentration from 2.5 mg/mL and ranging from 1.0 to 2.5 mg/mL decreased the viability of 4T1 cells in a concentration-dependent manner, at 24 hours and 48 hours respectively ($p<0.001$). These results also demonstrate a time-dependent effect of I-Fraction on 4T1 cells viability. In addition, P. ostreatus I-Fraction (2.5 mg/mL, 48 h) increased the number of 4T1 cells in the subG0/G1 phase (I-Fraction= 9.05 vs. vehicle= 2.3 %, $p<0.001$) and decreased those in the G0/G1 phase, compared to vehicle (I-Fraction= 42.3 vs. vehicle= 48.77 %, $p<0.001$). These results suggest that I-Fraction decreases 4T1 cell viability through an induction in cell death, without affecting cell cycle progression. By another hand, we found that I-Fraction decreased migratory capability of 4T1 cells at 13 h of treatment, compared to vehicle ($p<0.01$). In conclusion, these results demonstrate the antitumor activity of Pleurotus ostreatus I-Fraction on breast cancer cells.

0409 - P300 INVOLVEMENT IN METASTATIC PROCESS OF TRIPLE NEGATIVE BREAST CANCER

Guillermina Ana GALLARDO | Valentina CLEMENTE | Marilina MASCARÓ | María Marta FACCHINETTI | Alejandro Carlos CURINO | María Eugenia FERMENTO

LABORATORIO DE BIOLOGÍA DEL CÁNCER - INIBIBB - UNS-CONICET. DPTO. DE BIOLOGÍA BIOQUÍMICA Y FARMACIA.

Abstract/Resumen: Triple negative breast cancer (TNBC) are a heterogeneous group of tumors which lack specific molecular targets. Therefore, it is necessary to investigate potential tumor markers for this subtype of BC. Recent studies indicate that p300 has a pro-metastatic role in BC and we have previously shown that inhibition of p300 decreases cellular migration and invasion in a TNBC cell line. Therefore, in this work we aimed to analyze the expression and localization of p300 and its association with markers of tumor progression and clinic-pathological parameters in human TNBC. Also, we investigated the molecular mechanisms through which p300 inhibition impaired the processes previously mentioned. In TNBC biopsies (n= 45), we found that higher levels of cytoplasmic p300 correlates with lower tumor stages and a better overall patient survival (IHC, $p<0.05$). In TNBC (MDA-MB-231) and hormone-independent BC (LM3) cell lines, the genetic silencing of p300 induced an increase in the levels of