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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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Around 2 billon children (almost 90% of the world's child population) are exposed to air pollution levels over 10 $\mu g/m^3$. Furthermore, child malnutrition is recognized as a major problem with devastating effects on children's health. Due to their physical and physiological characteristics, children conform a subpopulation highly susceptible to the adverse effect of environmental pollutants (gases and particulate matter -PM) and vulnerable to malnutrition. PM exposure increases the risk for cardiovascular disease progression, through vascular oxidative stress and inflammation, two main processes involved in the initiation of endothelial dysfunction.
Therefore, we investigate the effects of ROFA (Residual Oil Fly Ash) exposure on the vasculature, in a nutritional retardation (NGR) rat model. In order to achieve NGR animals, male weanling rats were fed a diet restricted 20% compared to *ad libitum* intake (control-C) for 4 weeks. NGR and C rats were intranasally instilled with either 1mg/kg BW of ROFA or its vehicle. 24h after exposure, the thoracic aorta was isolated and biochemical parameters were evaluated: CYP1A1, eNOS and Calcium channel type L by RT-PCR, TGFβ1 by Westen blot and cytokines (IL-6, IL-10) by ELISA.
Our data showed that ROFA exposure induced IL-6 and IL-10 augmentation vs. C rats (35% and 30%, $p<0.01$). No changes were observed in NGR cytokines levels.
Likewise, ROFA increased CYP1A1 levels vs. C (183%, $p<0.001$) and TGF-β1 levels vs. C (216%, $p<0.001$) and vs. NGR (133%, $p<0.01$). On the other hand, exposure to ROFA decreased eNOs levels vs. C (75%, $p<0.001$) and vs. NGR (38%, $p<0.01$), and calcium channels vs C (34%, $p<0.01$) and vs. NGR (20%, $p<0.05$).
These results showed that NGR animals failed to activate inflammatory and detoxifying response to ROFA. In relation to the contractile capacity of the vascular system, a frank decrease is observed, as well as a phenotypic change towards a more undifferentiated cell type.

568. (182) **THE UV FILTER BP-3 INDUCES INTRAUTERINE GROWTH RESTRICTION BY AFFECTING THE REMODELING OF SPIRAL ARTERIES**

María Laura Zenclussen[1,2], Valentina Galliani[1], Julián Abud[1,2], Horacio Adolfo Rodríguez[1,2]
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We are continuously exposed to personal care products containing chemicals that may affect our health. In previous works we have shown that dermal exposure to a commonly used UV-filter, benzophenone-3 (BP3) affected fetal growth of the progeny in mice. The aim of the present study was to evaluate the underlying mechanism of the effects of BP3 on fetal weight. For that purpose, C57BL6 mice were dermally exposed either to BP3 (50 mg/kg/day) or to vehicle (olive oil) on a daily basis, from gestational day 0 (gd0) until their sacrifice on gd10 or gd14. In the groups of animals sacrificed on gd10, there was a significant difference in the size of the whole implantation sites (WIS), being the WIS of the BP-3 treated animals smaller than those of the control group ($p<0,01$). When analyzing the wall-to-lumen diameter of the spiral arteries of paraffin-embedded WIS samples, those WIS of smaller size in the BP-3 group presented a bigger wall-to-lumen ratio whereas WIS of normal size from the BP-3 group ($p<0,01$) or from the control group ($p<0,001$) presented normal wall-to-lumen ratio, showing that only WIS of abnormal size had impaired spiral artery remodeling. When analyzing the animals at gd14, fetuses from the BP-3 treated animals were significantly smaller than fetuses from the control group ($p<0,05$), confirming the observations from gd10. Serum samples from all females were analyzed for their BP-3 content by HPLC. In BP3 treated animals, BP3 was found in 4 of 5 serum samples from gd10 and in 3 of 6 serum samples from gd14. BP3 was also found in amniotic fluid at gd14. These results indicate that BP3 is incorporated to the systemic circulation once applied dermally and it is able to reach the fetus. Taken all together, these results confirm that during pregnancy, BP3 is able to reach the maternal-fetal interface and affect the remodeling of spiral arteries, leading to an intrauterine growth restriction phenotype.

569. (189) **GLYPHOSATE BASED HERBICIDE EXPOSURE MAY EXACERBATE ENDOMETRIAL HYPERPLASIA INDUCED BY CAFETERIA DIET IN ADULT RATS**

María Victoria Zanardi[1,2], María Paula Gastiazoro[1], Virginia Lorenz[1], Marlise Guerrero Schimpf[1], Ailín Almirón[1], María Mercedes Milesi[1], Oliver Zierau[2], Jorgelina Varayoud[1], Milena Durando[1]
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Many lifestyle factors can predispose to the development of endometrial carcinoma (EC). Previously, in rats, we detected that: a) cafeteria diet (CAF) induced endometrial hyperplasia (EH), a precursor lesion of EC; b) exposure to glyphosate-based herbicide (GBH) altered uterine development causing EH and increased cell proliferation. In the present work we sought to evaluate whether the exposure to a low-dose of a GBH plus CAF diet might enhance the uterine morphological alterations induced by CAF diet alone. Female Wistar rats were fed from postnatal day 21 (PND21) until PND240 with: chow diet (CON) or CAF. At PND140 one group of rats feed with CAF also received GBH through food, yielding 3 experimental groups: CON; CAF; CAF+GBH. Rats were sacrificed at PND240 and serum, fat depots, and uterine samples were obtained. Data were analyzed by Kruskal–Wallis followed by Mann-Whitney post-test ($p<0.05$). CAF and CAF+GBH increased the fat depots relative to body weight compared to CON group. At morphological level, CAF and CAF+GBH increased glandular volume fraction and stromal nuclei density. At protein level, we detected higher expression of estrogen receptor alpha (ESR1) and cell proliferation in both treated-groups respect to CON. Moreover, both CAF and CAF+GBH treatments increased volume fraction of abnormal glands (glands with cellular anomalies plus glands with daughter glands). This alteration was more pronounced in CAF+GBH group, suggesting that GBH could enhance CAF effects. Also, only CAF-GBH reduced the expression of PTEN (a tumor suppressor gene) in the subepithelial stroma respect to CON. Progesterone serum levels were higher in CAF+GBH compared to CON. In conclusion, although CAF induces several changes associated with EH, the co-exposure with GBH increases the risk of developing EC. It is important to highlight that the study of lifestyle factors in combination could have a better contribution to understand a complex pathology like the EC.

570. (193) **IN VITRO CYTOTOXIC ASSESSMENTS OF *NECTANDRA ANGUSTIFOLIA* AND *CISSAMPELOS PAREIRA* EXTRACTS AGAINST HUMAN CANCER CELL LINES**

Ferrini Leandro[1,2], Ojeda Gonzalo[2], Melana Colavita Juan Pablo[1], Rodríguez Juan Pablo[1], Torres Ana[2], Aguirre María Victoria[1]
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Secondary metabolites represent an established source of bioactive compounds with different chemical structures. Among the polyphenols, the subgroup of flavonoids arises as promising anticarcinogenic drugs. *Nectandra angustifolia* (Schrad.) Nees & Mart. and *Cissampelos pareira* L. are native plants of the Northeast re-

gion of Argentina. Previous studies conducted by our research group showed the presence of flavonoids in the ethanolic extracts and their bioactivities in diverse models. Therefore, the objective of this work was to test the cytotoxic effect of both ethanolic extracts (NaE and CpE) against several human cancer cell lines. The extracts were tested on the following cell lines Caki-2 (kidney clear cell carcinoma), A549 (lung carcinoma), HT-29 (colon adenocarcinoma) and THP-1 (acute monocytic leukemia). Assays were extended to non-cancerous cells lines, the embryonic HEK-293 (kidney) and L-929 (fibroblast). Cells were seeded according to ATCC guidelines and incubated 24 h in humidified atmosphere with 5% CO_2 at 37ºC. Then were treated with NaE or CpE ranging from 10 to 200 mg/mL for 24 h and 48 h. Proliferative rates were assessed with XTT assay compared to untreated cells for determining the IC50. Values were analyzed following the cytotoxicity of the National Cancer Institute, IC50 ≤ 20 μg/mL: high, IC50 ranged from 21 to 200 μg/mL: moderate, IC50 from 201 to 500 μg/mL : weak and IC50 > 501 μg/mL : no cytotoxicity. Both extracts showed a similar bioactivity at 24 h, with a low dose increase in proliferation. However, when analyzed at 48 h, NaE exhibited an IC50 33.37 mg/mL ± 5.4 mg/mL for THP-1, and for non-cancer cell lines an IC50 of 90.10 ± 19.58 μg/mL for L929 fibroblast and 81.11 ± 15.45 μg/mL for HEK-293 embryo cell line. These preliminary results of cytotoxic activity in TPH-1 cell line by NaE encourage us to develop further studies in the identification of its bioactive compounds, as well as, on the underlying anticancer mechanism of action.

571. (269) **THE UV FILTER BENZOPHENONE 3 (BP3) ALTERS THE MIGRATION OF THE EXTRAVILLOUS TROPHOBLAST CELL LINE SWAN 71 VIA ANDROGEN RECEPTOR PATHWAY**

Abud Julián [1,2], Pagotto Romina [3], Zenclussen M. Laura [1,2], Galliani Valentina, Bollati-Fogolín, Mariela [3]; Rodríguez Horacio [1,2].

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BP3 is one of the most commonly substances used in sunscreens and personal care products due to its UV blocking efficacy. Several *in vitro* and *in vivo* studies evidenced the ability of BP3 to act like an endocrine disrupting chemical. The present study focuses on the effect of BP3 on the migration capacity of human trophoblast cells (Swan 71 cell line) and the potential involvement of the androgen receptor (AR) pathway. We analyzed three different BP3 concentrations: a) BP3-2: the predicted no-effect concentration (2 µg/L), b) BP3-20: the concentration detected in the amniotic fluid (20 µg/L) in our previous studies and c) BP3-200: the plasma concentrations reported in humans (200 µg/L). We examined cell migration activity by scratch-wound healing assay, as well as mRNA relative expression levels of molecules of interest such as, AR, matrix metalloproteinase 2 (MMP2), inhibitor of MMP-2 (TIMP2) and laminin a4 (LAMA4). The three doses of BP3 reduced the area of wound closure after 24 h of exposure, evidencing reduced migration of Swan 71 cells when compared to the vehicle. Interestingly, BP3 induced an augmented expression of AR mRNA levels in all concentrations assayed, and of TIMP2 and LAMA4 only in BP3-2. MMP-2 did not show significant changes. In order to confirm whether BP3 acts via an AR-dependent pathway, we then analyzed BP3 effects with and without an AR inhibitor (Flutamide, 1 μM). When the cells were treated with BP3 in the presence of flutamide, the area of wound closure did not change after 24 h, clearly indicating that BP3 acts through a AR-dependent pathway. This was confirmed by the AR mRNA expression restoration in cells exposed to BP3 + flutamide. In conclusion, exposure to relevant doses of BP3 is enough to perturb the migration capability and the expression of AR mRNA levels of the trophoblast cell line Swan 71. These effects were reversed in the presence of an AR inhibitor indicating that BP3 could act via AR-dependent pathway.

572. (338) **AIRBORNE PARTICULATE MATTER EXPOSURE IMPAIRS LUNG REDOX METABOLISM INVOLVED IN TISSUE DAMAGE REPAIR MECHANISMS**

Sofía Reynoso[1], Timoteo Marchini[1], Mariana Garcés[1], Lourdes Cáceres[1], Agustina Freire[1], Valeria Calabró[1], Alejandro Berra[2], Pablo Evelson[1], Natalia Magnani[1]

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Is estimated that 91% of the world's population breathes polluted air leading to more than 7 million premature deaths per year. Airborne pollutants such as particulate matter (PM) are associated with enhanced health risk as they can trigger or aggravate several pulmonary diseases. Our aim was to assess if alterations in the lung oxidative metabolism initiated by toxicological mechanisms triggered after PM inhalation were associated with a delayed tissue injury repair. To characterize our model, BALB/c mice were exposed to filtered air (FA) or urban air (UA) from Buenos Aires City, in whole-body exposure chambers. Results showed that after 8 weeks of UA exposure, mice developed lung redox alterations and local inflammation without histological damage, therefore that was the time point selected to further evaluate the oxidative metabolism after a moderate lung injury induced by intratracheal instillation of 0.1 N hydrochloric acid (HCl). Pulmonary tissue was evaluated 5 days after HCl treatment. Tissue oxygen consumption was assessed as a whole lung metabolism marker, and the increase observed in mice breathing FA by HCl treatment, was not detected in HCl-mice exposed to UA (p<0.05). Interestingly, SOD activity showed the same trend (p<0.05), even though transcription factor Nrf2 expression was higher after the injury in the UA group (p<0.05). While no edema was observed in any group, local inflammation measured as total cell count in bronchoalveolar lavage (BAL), was significantly increased only after UA exposure and HCl instillation (p<0.05) compared to control values. Hence, mice breathing UA might not be able to modulate the redox metabolism involved in lung damage repair mechanisms. Our results highlight the importance of tissue healing mechanisms evaluation as valuable knowledge for developing adequate therapeutic approaches aiming to ensure restoration of normal alveolar architecture required for proper lung function.

573. (352) **CHRONIC EXPOSURE TO URBAN AIR POLLUTION IN BUENOS AIRES CITY INDUCES OXIDATIVE STRESS AND INFLAMMATION IN MICE OLFACTORY BULB**

Freire, Agustina[1]; Garcés, Mariana[1]; Cáceres, Lourdes[1]; Magnani, Natalia[1]; Marchini, Timoteo[1]; Martinefski, Manuela[2]; Tripodi, Valeria[2]; Berra, Alejandro[3]; Alvarez, Silvia[1]; Calabró, Valeria[1]; Evelson, Pablo[1].

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Previous reports indicate that central nervous system (CNS) is a target of air pollution, causing tissue damage and functional alterations. Oxidative stress and neuroinflammation have been pointed out as possible mechanisms mediating these effects. The aim of this work was to study the chronic effects of urban air pollution on mice olfactory bulb (OB), focusing on oxidative stress and inflammation markers. Male 8-week-old BALB/c mice were exposed to filtered air (FA, control) or urban air (UA) inside whole-body chambers, located in a highly polluted area of Buenos Aires city, for up to 4 weeks. Reduced glutathione levels (GSH) were decreased by 75% after 4 w of exposure to UA (p<0.05). In accordance with these results, an increase in glutathione reductase activity was found at the same time point (p<0.05 vs. FA). Total superoxide dismutase (SOD) activity, including a differential analysis of its cytosolic and the mitochondrial isoforms, Cu/Zn-SOD and Mn-SOD respectively, were determined. Cu/Zn-SOD activity showed an initial decrease after 1 w of UA exposure compared to FA, and a subsequent increase of 50% at week 4 (p<0.05), while no changes were observed for Mn-SOD activity.