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LA TAPA

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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.

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

151. (202) **FENOFIBRATE INCREASES THE POPULATION OF NON-CLASSICAL MONOCYTES IN ASYMPTOMATIC CHAGAS DISEASE PATIENTS AND MODULATES INFLAMMATORY CYTOKINES IN PBMC**

Azul Pieralisi[1] Ágata Cevey[1], Federico Penas[1], Nilda Prado[3], Ana Mori[3], Mónica Gili[4], Gerardo Mirkin[2], Juan Gagliardi[3], Nora Goren[1].

1. CONICET-Universidad de Buenos Aires. Instituto de Investigaciones Biomédicas en Retrovirus y SIDA (INBIRS). Buenos Aires. Argentina. Buenos Aires. Argentina. 2. CONICET-Universidad de Buenos Aires. Instituto de Investigaciones en Microbiología y Parasitología Médica (IMPAM). Buenos Aires Argentina. 3. Hospital General de Agudos Dr. Cosme Argerich, Buenos Aires, Argentina. 4. Hospital Municipal de Rehabilitación Respiratoria María Ferrer, Buenos Aires, Argentina.

Chagas heart disease (CHD) is the most important clinical manifestation of *Trypanosoma cruzi* (Tc) infection. Peripheral blood mononuclear cells (PBMCs) infiltrate the tissue and differentiate into inflammatory macrophages. Advances in pathophysiology show that myeloid cell subpopulations contribute to cardiac homeostasis, emerging as possible therapeutic targets. In this work we investigated the spontaneous release of inflammatory cytokines and chemokines, changes in the frequencies of monocyte (Mo) subsets and the effects of fenofibrate (Fen) on PBMC of patients with different clinical forms of Chagas disease. PBMC isolated from CHD by Ficoll® display higher levels of IL-12, TGF-β, IL-6, MCP1 and CCR2 than cells from uninfected individuals (HI) or asymptomatic (Asy), as tested by RT-qPCR, (P<0,05). Fen reduces the levels of pro-inflammatory mediators and CCR2 in both Asy and CHD (P< 0,05). Also, CHD patients display a significantly higher percentage of classical Mo in comparison with Asy and HI (P<0,05). Besides, Asy have a significantly higher percentage of non-classical Mo than CHD or HI (P<0,05). However, no difference in the intermediate Mo subpopulation was found between groups. Moreover, Mo from Asy or CHD patients exhibit different responses upon stimulation *in vitro* with Tc lysates and Fen treatment. Tc stimulation significantly increased the percentage of classical Mo and decreased percentage of intermediate Mo in the Asy group. Also, there were no changes in their frequencies in CHD or HI. Notably, stimulation with Tc did not alter the frequency of non-classical Mo in any of the groups. Moreover, Fen treatment of Tc-stimulated PBMC increased even more the frequency of non-classical Mo in Asy patients. Summing up, our results stress a potential role for Fen as modulator of Mo towards an anti-inflammatory profile in different stages of chronic Chagas disease.

152. (209) **AQUAPORINS CAN BE INVOLVED IN THE SWELLING CAUSED BY SHIGA TOXIN TYPE 2 ON HGEC AND HK-2 CELLS**

Fernando D Gómez[1], Julieta Reppetti [2], Romina Romero[3], Flavia Sacerdoti[1], Cristina Ibarra[1], María Marta Amaral[1].

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Servicio de Pediatría, Hospital Nacional Profesor "Alejandro Posadas", Buenos Aires, Argentina

Hemolytic uremic syndrome related to Shiga toxin–producing *Escherichia coli* (STEC-HUS) is the principal etiology of acute kidney injury in children in Argentina.

Previously, we demonstrated that Shiga toxin type 2 (Stx2) damages human glomerular endothelial cells (HGEC) and HK-2 human proximal tubular epithelial cell line by inducing swelling and detachment.

In this work, we analyzed cell volume changes of HGEC and HK-2 exposed or not to Stx2 or a hypoosmotic (HYPO) medium, in the presence or not of aquaporins (AQPs) inhibitors (mercuric chloride: $HgCl_2$ and tetraethylammonium: TEA), or an inhibitor of Stx2 receptor (Gb3) synthesis, Eliglustat (EG). For controls, an isosmotic (ISO) medium was used.

Cells were grown on 12 well plates and pretreated for 30 minutes with $HgCl_2$ (10 μM) or TEA (100 μM) or pretreated during 24 h with EG (10 μM). Then, HGEC and HK-2 were incubated with Stx2 (50 μM) for an additional 40 minutes. Cell volume was analyzed by light microscopy and measuring cell area by using Image J software.

After Stx2 and HYPO medium treatments, a significant increase in the cell volume of HGEC (Stx2: 42%; HYPO: 36%, n= 3, p<0.05) and HK-2 (Stx2: 70%; HYPO: 55%, n= 3, p<0.05) was detected respect to ISO medium. However, when HGEC and HK-2 were pretreated with $HgCl_2$ or TEA a significant swelling prevention was obtained for HGEC (Stx2+$HgCl_2$:100%; Stx2+TEA:86%; HYPO+$HgCl_2$: 42.5%; HYPO+TEA: 83%, n=3, p<0.05) and HK-2 (Stx2+$HgCl_2$: 90%; Stx-2+TEA: 85%; HYPO+$HgCl_2$: 55%; HYPO+TEA: 75%, n=3, p<0.05). In addition, EG also was able to prevent HK-2 swelling in 87 % (n=1) with respect to Stx2 treatment.

Results show that AQPs may be involved in the water movement inside HGEC and HK-2 induced by Stx2, since $HgCl_2$ and TEA avoided this effect. Furthermore, binding of Stx2 to Gb3 could be the initial step for the development of cellular mechanisms that possibly trigger the entry of solutes into the cells and the consequent osmotic gradient responsible for the hypotonic effect.

153. (210) **FABF8:STX2 RECOMBINANT MONOCLONAL ANTIBODIES AVOID THE DELETERIOUS EFFECTS OF SHIGA TOXIN TYPE 2 ON HUMAN MICROVASCULAR ENDOTHELIAL CELLS**

Gómez Fernando D[1], Luz D[2], Piazza, RMF[2], Presta A[1], Ibarra C[1], Sacerdoti F[1] Amaral MM[1].

1. Laboratorio de Fisiopatogenia, IFIBIO-HOUSSAY-UBA-CONICET, Departamento de Fisiología, Facultad de Medicina, Universidad De Buenos Aires, Buenos Aires, Argentina.

2. Laboratorio Bacteriología, Instituto Butantan, São Paulo, SP, Brasil.

Hemolytic Uremic Syndrome (HUS) associated with Shiga-toxigenic *Escherichia coli* (STEC) infections is the principal cause of acute renal injury in pediatric age groups in Argentina. Neither a licensed vaccine nor effective therapy for HUS is available for humans. Previously, we demonstrated the *in vitro* cytotoxic effects of Shiga toxin type 2 (Stx2) on human glomerular endothelial cells (HGEC). Recently, recombinant antibodies against Stx2, produced in bacteria, were developed, and characterized. In this work, we studied the ability of anti-Stx2 FabF8:Stx2 antibody to neutralize the Stx2 activity on primary cultures of HGEC. Cells were plated in 96-well plates and grown to confluence. Then, cells were treated in growth-arrested conditions for 72 h with different pre-incubations (1 h at 37ºC) or co-incubations of FabF8 with Stx2. Antibodies were used from 10 μg/mL to 0.001 μg/mL and Stx2 at the dilution required to kill 50% of cells (0.5 ng/mL). Finally, cell viability was assessed by neutral red uptake. In addition, cells were seeded on gelatine coated glass coverslips and then treated, as it was previously mentioned, with 1 μg/mL FabF8 and 0.5 ng/mL Stx2, during 72 h. Percentage of necrotic and apoptotic cells were established by fluorescence microscopy after staining with acridine orange/ethidium bromide. Under both conditions evaluated, FabF8:Stx2 significantly neutralized, in a dose-dependent manner, the cytotoxic effects caused by 0.5 ng/mL Stx2 in HGEC (p <0.05, n = 3). HGEC viability was protected by 10 μg/mL FabF8 in about 67.5% at the co-incubation condition, and about 83% at the pre-incubation condition. Additionally, FabF8:Stx2 significantly prevented HGEC necrosis (pre: 60%; co: 92.5%) and apoptosis (pre: 93% and co:75%) (p <0.05, n = 3). The results demonstrate the high efficiency of FabF8:Stx2 to avoid the cytotoxic effects of Stx2 on HGEC, therefore, they could be used as a therapeutic strategy to prevent the renal damage described in patients with HUS.

154. (240) ***B. PERTUSSIS*** **COMPROMISES THE EPITHELIAL BARRIER AND SURVIVES IN NON-DEGRADATIVE INTRACELLULAR COMPARTMENTS**

Baroli C[1], Gorgojo JP[1], Blancá B[1], Debandi M[1], Rodríguez