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Daniela Kantor. Médanos, 2018

Técnica: Acrílico sobre cartón entelado. Medidas: 20x28 cm

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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pression of activation markers, increased circulating follicular T cells with a skew towards a Tfh1 profile and elevated double-negative T cells with normal FOXp3$^+$ and reduced Th17$^+$ CD4$^+$ T cells. Impaired B-cell subsets showed low post-switch memory cells with high frequencies of CD21^{low} B cells. Normal lymphoprolipheration assay. Therefore, he was diagnosed with Common Variable Immunodeficiency with dysregulation and started immunoglobulin replacement. Whole exome sequence revealed c.368C>A and c.365G>A variants in the same allele of *SOCS1* gene. Enhanced phospho-STAT1 kinetic assay confirmed the pathogenic role of these variants. **Conclusion:** In humans, mutation in SOCS1 impacts the STAT1 signaling pathway thus affecting multiple JAK/STAT signaling pathways. Only a few patients with SOCS1 mutation have been recently reported worldwide with a broad phenotypic spectrum overlapping other inborn errors of immunity.

278. (154) **INDUCED PLURIPOTENT STEM CELL-DERIVED MESENCHYMAL STEM CELL RESPONSE TO BACTERIAL LIPOPOLYSACCARIDE AND SHIGA TOXIN**

Daiana Martire-Greco[1], Alejandro La Greca[2], Luis Castillo Montañez[1], Celeste Biani[2], Antonella Lombardi[2], Federico Birnberg-Weiss[1], Alessandra Norris[2], Nahuel Rodrigues-Rodriguez[1], Jose Ramón Pittaluga[1], Veronica Furmento[2], Veronica Inés Landoni[1], Santiago Gabriel Miriuka[2], Carlos Luzzani[2], Gabriela Cristina Fernández [1].

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Mesenchymal Stem Cells can be activated and respond to different bacterial toxins. Lipopolysaccharides (LPS) and Shiga Toxin (Stx) are the two main bacterial toxins present in Hemolytic Uremic Syndrome (HUS) that cause endothelial damage. In this work we aimed to study the response of induced Pluripotent Stem Cells derived Mesenchymal Stem Cells (iPSC-MSC) to LPS and/or Stx and its effect on the restoration of injured endothelial cells. For this purpose, we stimulate iPSC-MSC with LPS and/or Stx for 24 h. using Polymyxin B in the Stx treatments in order to avoid LPS contamination. The results obtained showed that LPS induced a pro-inflammatory profile on iPSC-MSC with an increment of IL-8 and TNF-α, but not Stx when we measure with ELISA kit, (pg/ml IL-8 Control: 1988±299; LPS: 20876±1233*#, Stx: 1801±137; LPS+Stx: 17935±213* and pg/ml of TNF-α Control:880±32; LPS:3291±116*#; Stx:627± 8; LPS+Stx:2092 ±59*, *vs. Control, #vs Stx p<0,05). Moreover, LPS induced on iPSC-MSC an increment on the migratory capacity of these cells (percentage of migration Control: 44±10; LPS:69±11*; Stx: 42±10; LPS+Stx: 72±10, *vs. Control p<0,05) and adhesion to gelatin substrate (number of cells adhere to gelatin Control: 533±37; LPS: 769±114*; Stx:702±102*; LPS+Stx:976±142, *vs. Control p<0,05). Finally, the addition of conditioned media of iPSC-MSC treated with LPS+Stx to HMEC-1 (Human Microvascular Endothelial Cells-1), decreased the capacity to close a wound in an endothelial monolayer (percentage of wound closure Control: 38±4; LPS:32±4; Stx:24±8; LPS+Stx: 18±6*, *vs. Control p<0,05). In conclusion, these results suggest that iPSC-MSC activated by LPS acquired a pro-inflammatory profile that induces migration and adhesion to extracellular matrix proteins, but the combination of both toxin decreased the repair of endothelial damage.

279. (312) **AIRWAY INFLAMMATION IN A *SALSOLA KALI* POLLEN-INDUCED MURINE MODEL OF ALLERGY**

Marcelo Javier Galvez, Gisela Giorgi, Ileana Lencinas, Adriana Martínez, María Gabriela Murray, María Inés Prat

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Allergic rhinoconjunctivitis and asthma are diseases with an increasing worldwide prevalence. In our region, common weeds, e.g. *Salsola kali*, are one of the major causes of pollinosis. Murine models are useful for studying the mechanism of allergic disease. Regarding the model antigen, the majority of studies have been performed using ovalbumin. The aim of this work were to develop an experimental animal model of allergy based on relevant human aeroallergens, such as *S. kali* pollen, and to define the immunological and cellular airway features of the allergic response. BALB/c mice (n = 5/group) were administrated with PBS or *S. kali* pollen extract through i.p. route and later challenged by nasal instillation of PBS or *S. kali* pollen respectively for 3 consecutive days. *S. kali*-specific IgE were measured by ELISA. After sacrifice, the noses and lungs were fixed and paraffin embedded for histological analysis (H&E, toluidine blue and periodic acid-Schiff). After nasal challenge with *S. kali* pollen, sensitized mice manifested early-phase (sneezing) and late-phase (eosinophilic and basophilic accumulation) response compared with the control group. Frequency of sneezing in sensitized mice were higher than the control throughout the challenge phase ($p < 0,01$). The histology showed goblet cell hyperplasia and eosinophil infiltration in nasal lateral mucosa (135 ± 58 in sensitized mice vs. 8 ± 1 in control group) and septum (52 ± 36 vs. 1 ± 1 respectively). Also, sensitization induced moderate to severe inflammatory infiltration in lungs. *S. kali*-specific IgE value was not increased in all sensitized mice. Our results confirm upper airway inflammation correlated with lower airway inflammation in response to allergen exposure. The symptoms and histology observed encourages us to think about the establishment of an alternative murine model based on relevant human allergens that allow to understanding the disease and exploring therapeutic approaches.

280. (343) **PLATELETS MODULATES CD4$^+$ T CELL FUNCTION IN COVID-19 THROUGH A PD-L1 DEPENDENT MECHANISM**

Ana Paletta[1], Facundo Di Diego García[1], Augusto Varese[1], Fernando Erra Diaz[1], Julián García[2], Juan Carlos Cisneros[2], Guillermina Ludueña[3], Ignacio Mazzitelli[1], Andrea Pisarevsky[3], Gonzalo Cabrerizo[1], Álvaro López Malizia[1], Alejandra G. Rodriguez[2], Nicolás Lista[2], Yesica Longueira[1], Juan Sabatté[1], Jorge Geffner[1], Federico Remes Lenicov[1], Ana Ceballos[1].

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Severe COVID-19 is associated with a systemic inflammatory response and a progressive CD4$^+$ T cell lymphopenia and dysfunction. Here, we analyzed whether platelets might contribute to CD4$^+$T cell dysfunction in COVID19.

Blood samples were obtained from healthy donors (HD) n=30 or COVID19 patients, n=60. Patients were classified into mild, moderate and severe according to WHO criteria. Each participant provided written informed consent. Oncologic and vaccinated patients were excluded from the study. Proportion of CD4+T Cells–platelets aggregates was measured by flow cytometry (CD4+CD62p+ cells). CD4+T cells were isolated from HD and cultured with platelets from a single HD or a COVID19 patient (1:100 ratio). CD25 was evaluated by flow cytometry and cytokine production was measured by ELISA.

We observed a high frequency of CD4$^+$ T cell-platelet aggregates in COVID19 (n=30-60, p<0.0001) that inversely correlated with lymphocyte counts (n=60, p=0.0267). Platelets from COVID19 but not from HD inhibited the up-regulation of CD25 expression (n=7, p=0.002) and TNF-α production by CD4$^+$T cells (n=7-13, p=0.0236). IFN-γ production was increased by platelets from HD but not from COVID19 (n=19-33, p=0.0016). An available RNAseq from purified platelets showed that COVID19 patients presented higher expression of PD-L1 than HD (n=5-9, p=0.02), and the same was observed by flow cytometry (n=26-30, p<0.0001). The proportion of PD-L1+platetelets inversely correlated with IFN-γ production by activated CD4$^+$T cells cocultured with platelets (n=43, p=0.0009).