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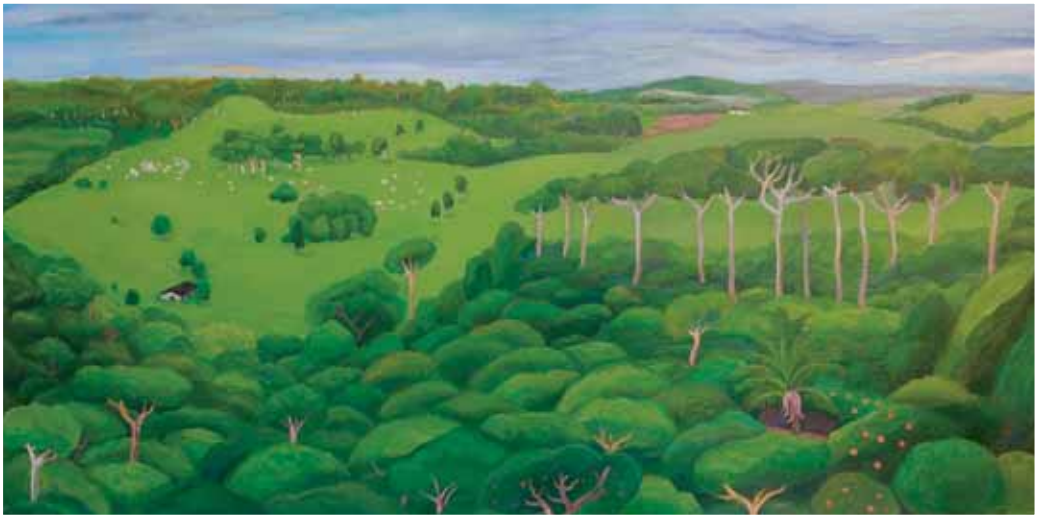

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LXVI REUNIÓN ANUAL DE LA

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CON LA PARTICIPACIÓN DE

SOCIEDAD ARGENTINA DE VIROLOGÍA (SAV)

ASOCIACIÓN ARGENTINA DE NANOMEDICINAS (NANOMED-ar)

14-17 de noviembre de 2018
Hotel 13 de Julio – Mar del Plata

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Claudia Pérez Leirós
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Alberto Crottogini

DHR test. P1 and P2 had T and B cells reconstitution 12 months post HSCT and discontinued IVIG. Conclusion: Most of our P are cured from CGD, but are still having complications related to HSCT. It is important to highlight that half of our P had graft failure.

96. (158) **EXACERBATION OF EXPERIMENTAL PSORIASIS SYMPTOMS IN DESMOGLEIN-4 DEFICIENT RATS IS MEDIATED BY INCREASED INFLAMMATION**

María Tamara Moreno Sosa, María Belén Sánchez, Flavia Judith Neira, Elisa Olivia Pietrobon, Graciela Alma Jahn, Juan Pablo Mackern Oberti
IMBECU-CONICET

Psoriasis is a chronic inflammatory skin disease, characterized by keratinocyte hyperproliferation, vasculature growth and leukocyte infiltration into the dermis and epidermis. Although it is known that desmogleins are proteins involved in cell adhesion mechanisms, their role in psoriasis has not been addressed. The aim of our work was to assess the impact of desmoglein-4 deficiency in the immunological response of the skin. To this end, OFA hr/hr rats, which are mutant for the desmoglein-4 gene and Sprague-Dawley (SD) wild type rats were used. Imiquimod (IMQ), which is an immune response modifier that acts via toll-like receptor 7 pathway, was administered to both rat strains in shaved skin for four days to generate psoriasis-like lesions. Skin biopsies from treated and untreated OFA and SD rats were weighed, minced, stained with PE-Cy5-anti CD45 and FITC-anti CD3 monoclonal antibodies and analyzed by flow cytometry. We observed that in both strains, CD45+, CD3+ cells in increased in IMQ-treated groups, but the rise was higher in OFA rats ($p<0.05$). Similarly, qPCR analysis of skin mRNA showed that pro-inflammatory genes such as IL-1β, IL-8, and CCR1 were increased in both IMQ-treated groups, SD and OFA, compared to untreated groups but the increase was also higher in OFA rats ($p<0.05$). Furthermore, TNF-α, CCR2, CCR3, CCR5 and CXCR5 mRNA expression rose only in the OFA IMQ treated group ($p<0.05$). When anti-inflammatory genes were evaluated, we found that both IMQ treated groups increased TGF-β expression similarly but OFA IMQ showed higher levels of IL-10 than SD IMQ and untreated groups ($p<0.05$). These results suggest that desmoglein-4 deficiency contributes to experimental psoriasis progress, promoting expansion of leukocyte population and increasing different pro-inflammatory genes mRNA expression in skin. Although further research is needed, these results could have a potential impact of desmoglein-4 on the diagnosis and prognosis of psoriasis.

97. (181) **VACCINE CANDIDATE BASED ON OUTER MEMBRANE VESICLES FROM BORDETELLA PERTUSSIS TRIGGERS THE CANONICAL INFLAMMASOME ACTIVATION**

Maia Lina Elizagaray[1], Martín Rumbo[1], Daniela Hozbor[2], Griselda Moreno[1]
[1]*Instituto de Estudios Inmunológicos y Fisiopatológicos - IIFP, Facultad de Ciencias Exactas, Universidad Nacional de La Plata, Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), La Plata, Argentina.* , [2]*IBBM-Instituto de Biotecnología y Biología Molecular-UNLP-CONICET*

The resurgence of the respiratory disease named pertussis has urged the need to develop a new vaccine. Under this context we have already identified and characterized with very good results, a vaccine candidate based on outer membrane vesicles (OMVs). Recent advances in the field have shed light on some of the multifaceted roles of OMVs in host-pathogen interactions. In this study, we investigated the ability of OMVs derived from B.pertussis, to activate the inflammasome pathway. To this end we evaluated the secretion of IL-1beta and the ASC-speck formation after the activation of THP-1 cells with different quantities of OMVs (5ng/mL to 5ug/mL). The cytokine levels were measured in culture supernatant by ELISA and the specks formation from THP1-ASC-GFP cells were observed and quantified by fluorescence microscopy.
By these assays we detected that the release of IL-1b from THP1 cells stimulated with at least 100ng OMVs was significantly higher than non treated cells. Release of IL-1b was independent of previous priming of cells with TLR agonists. In particular when 200ng OMVs was used, the IL-1b level was 72,3±2,1 pg/mL vs. 4,10±0,02 pg/mL ($p\leq0,001$) detected in the non treated cells. The percentage of ASC+/total THP1-ASC-GFP cells was also significantly higher for cells stimulated with at least 200ng OMVs in comparison with non-stimulated cells (6,8±0,6 vs. non detected $p\leq0,001$).
These results show for the first time that our vaccine candidate based on the OMVs derived from B. pertussis activate the iflammasome in a human macrophage cell line. Furthermore, this data would strengthen the concept of inflammasome activation as one of the innate immune pathways with the ability to profile the protective adaptive response of our vaccine.

98. (189) **BRUCELLA ABORTUS-INFECTED PLATELETS ACTIVATE THE ENDOTHELIUM AND PROMOTE THE MIGRATION OF MONOCYTES TOWARDS THE SITE OF INFECTION**

Aldana Trotta, Maria Ayelen Milillo, Ana Maria Rodriguez, Luciana Balboa, M. Victoria Delpino, Guillermo Giambartolomei, Paula Barrionuevo
IMEX-CONICET, Academia Nacional de Medicina, INIGEM (UBA-CONICET)

Brucellosis is an infectious disease elicited by bacteria of the genus *Brucella*. Platelets have recently got involved in the modulation of innate and adaptive immune responses. We have previously reported that platelets act as carriers of bacteria, promoting the invasion of monocytes. Thus, the aim of this study was to further investigate the role of platelets in the immune response against *Brucella*. First, we wondered whether the presence of platelets modulates the time course of *B. abortus* infection. For this, THP-1 cells (human monocytic cell line) were infected with *B. abortus* in presence or absence of platelets. Then, extracellular bacteria were killed and cells were incubated for different times. Our results demonstrate that the presence of platelets significantly increased the percentage of *B. abortus*-infected THP-1 cells at early time-points ($p<0.001$). Nevertheless, the presence of platelets subsequently improved the contention of the infection. Taking into consideration that *B. abortus* localization within different tissues requires its extravasation across the endothelium, our next aim was to study the role of platelets in the modulation of monocytes extravasation in the context of *B. abortus*-mediated infection. We first studied the ability of platelets to recruit monocytes. Our results showed that supernatants collected from infected platelets promote the transmigration of monocytes ($p<0.01$). Moreover, the pre-treatment of monocytes with this supernatant enhance the responsiveness of monocytes towards other chemoattractant stimuli ($p<0.01$). Finally, we studied the ability of platelets to activate the endothelium. For this, HMEC cells (human endothelial cell line) were stimulated with supernatants collected from *B. abortus*-infected platelets. This supernatant stimulated the expression of ICAM-1 (CD54) ($p<0.01$). At the same time, it enhanced the secretion of both IL-8 and MCP-1 ($p<0.01$). These results showed that infected platelets are able to activate the endothelium and promote the migration of monocytes towards the site of the infection

99. (191) **SLPI IS TAKEN UP BY MONOCYTES AND IMPAIRS THEIR DIFFERENTIATION TO DENDRITIC CELLS**

Mariana Noelia Mardirosian[1], Fiorella Caro[1], Nella Ambrosi[1], Ximena Villalonga[1], Macarena Reiteri[1], Diego Guerrieri[1], Claudio Incardona[2], Domingo Casadei[3], Eduardo Chuluyan[1]
[1]*CEFYBO-CONICET-UBA* , [2]*GADOR SA* , [3]*Instituto de trasplante y alta complejidad (ITAC)*

Secretory leukocyte proteinase inhibitor (SLPI) is a serine protease inhibitor produced mainly by epithelial cells. It has anti-inflammatory and antimicrobial activity, and enhances wound healing. We have previously described that SLPI inhibits lymphocyte proliferation which depends on the presence of monocytes. The aim of the present study was to assess whether SLPI is captured by monocytes and its effect on monocytes differentiation to dendritic cells.
To study SLPI uptake, human myelomonocytic U937 cells and human peripheral blood mononuclear cells (PBMC) were treated or