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JOINT MEETING OF BIOSCIENCE SOCIETIES

LXII ANNUAL MEETING OF ARGENTINE SOCIETY OF CLINICAL INVESTIGATION (SAIC)

LIII ANNUAL MEETING OF ARGENTINE SOCIETY OF BIOCHEMISTRY AND MOLECULAR BIOLOGY (SAIB)

LXV ANNUAL MEETING OF ARGENTINE SOCIETY OF IMMUNOLOGY (SAI)

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Keywords: Mucosal immunology – Biological Clock - Tolerance -

(1289) **DISSECTING THE ROLE OF GALECTIN-3, IFN-γ AND MICROBIOTA IN GERMINAL CENTER B CELL FATE DECISIONS.**

Cristian Gabriel Beccaria (1), Facundo Fiocca Vernengo (1), María Carolina Amezcua Vesely (1), Laura Almada (1), Juan Mucci (2), María Cecilia Ramello (1), Jimena Tosello Boari (1), Oscar Campetella (2), Carolina Lucia Montes (1), Eva Virginia Acosta Rodriguez (1), Adriana Gruppi (1)
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Germinal Centers (GC) are unique structures where high affinity antibodies and memory B cells are generated. With the aim of ensure fast selection of rapidly evolving clones in GCs, follicular dendritic cells, GC B cells and T cells engage in multiple interactions. So far, only co-stimulatory or inhibitory membrane immune receptor/ligand pairs and cytokines have been investigated and are known to be involved in this process. However, there is a pressing need to understand other factors that regulate GCs in order to optimize and control humoral responses following vaccination, antibody-mediated autoimmune diseases, and GC B cell-derived malignancies. Here, we report a novel effector function by which Galectin-3 (Gal-3), a β-galactoside binding protein, is critically involved in the development of GCs. Gal-3 KO mice exhibit high frequency of spontaneous GC (sGC) B cells and T follicular helper (Tfh) cells that correlated with hypergammaglobulinemia, high amounts of IFN-γ in serum and lupus-like autoimmune manifestations. We found that the main source of IFN-γ in Gal-3 KO mice were CD4+ T cells (including Tfh), since there are no significant differences between IFN-γ-producing CD8 T, NK, NKT and DCs cells between groups. IFN-γ blockade in Gal-3 KO mice prevents the autoimmune condition, demonstrating that IFN-γ overproduction sustain lupus-like disease. We were also wondering whether specific microbiota in Gal-3 mice could influence the induction of sGCs. Microbiota modulation by antibiotic treatment could not prevent sGCs induction in Gal-3 KO mice and, interestingly, these mice did not modify the generation of GCs in Peyer's patches after treatment, unlike the WT mice which showed a deep reduction of them. Finally, using chimeric mice with B cell-specific deficiency of Gal-3, we reveal that intrinsic Gal-3 signaling in B cells control sGC formation. Together, our data provide the first evidence that Gal-3 acts directly on B cells regulating GCs responses via IFN-γ.

Keywords: Germinal Centers; Galectin-3; Autoimmunity; IFN-γ; B cell Immunology.

(1598) **EFFECT OF *Trypanosoma cruzi* CYTOSOLIC TRIPAREDOXIN PEROXIDASE ON PERIPHERAL BLOOD MONONUCLEAR CELLS FROM PATIENTS WITH CHRONIC CHAGAS DISEASE**

Magalí Celeste Girard (1), Gonzalo R Acevedo (1), María D Piñeyro (2), Juan P Grosso (3) Micaela S Ossowski (1), Marisa Fernandez (4), Yolanda Hernandez Vasquez (4), Carlos Robello (2), Karina A Gómez (1)
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Trypanosoma cruzi, the etiological agent of Chagas disease (ChD), has a highly efficient detoxification system to deal with the oxidative burst imposed by its host. One of the antioxidant enzymes involved is the cytosolic tryparedoxin peroxidase (TcCPx) which catalyzes the reduction of hydrogen peroxide, small-chain organic hydroperoxides and $ONOO^-$. This enzyme is present in all parasite stages, and its over-expression renders parasites more resistant to oxidative defenses of macrophages, indicating an important role in parasite survival. However, TcCPx not only constitutes a relevant virulence factor in ChD, but also promotes inflammatory reactions in *T. cruzi*-infected mice. The aim of this study was to analyze the specific humoral and cellular immune response triggered by TcCPx in chronic asymptomatic (n=7) and cardiac patients (n=6), and non-infected individuals (n=5). Results showed that levels of IgG antibodies against TcCPx were low (titer<1/400) in sera from individuals across all groups. However, after five days of *in vitro* stimulation, TcCPx induced proliferation of peripheral blood mononuclear cells (PBMC) from asymptomatic patients (p<0.01). In addition, only interferon-γ, but not GM-CSF or IL-10, were detected in the supernatant of PBMC from asymptomatic patients (p=0.04) under the same culture conditions. No response of either type was observed in cardiac patients and non-infected individuals. NetMHCpan and NetMHCIIpan were used to predict potential binding peptides within TcCPx for HLA-class I and II, respectively. The algorithm highlighted four regions of 16-23 aminoacids long containing peptides with high binding probability to most prevalent alleles of α and β chains of HLA-II in central and Northern Argentine population, while potential HLA-I epitopes were mostly found in N-terminal region of the protein. We expect that our research can contribute to understand the role of TcCPx on the immune response of chronic Chagas disease patients.

Keywords: Cytosolic tryparedoxin peroxidase, *Trypanosoma cruzi*, chronic Chagas disease, epitope prediction.

(1810) **DESMOGLEIN-4 DEFICIENCY INCREASES RESIDENT CD3+ T CELL SUBSET IN A RAT PSORIASIS MODEL**

María Tamara Moreno Sosa (1), Elisa Pietrobon (1), Alicia Carolina Innocenti Badano (2), Marta Soaje (1), Susana Valdez (1), Belén Sánchez (1), Florencia Yúdica Sedano (3), Flavia Neira (1), Gisella Pennacchio (1), Graciela Alma Jahn (1), Juan Pablo Mackern Oberti (1)
(1) IMBECU. (2) Hospital Luis Lagomaggiore. (3) Universidad Juan Agustín Maza.

It is known that desmogleins are involved in cell adhesion mechanisms and are crucial in keeping structural integrity of different tissues, including skin. They also play important roles in differentiation, cell activation and migration. Keratinocytes (KC) produce several inflammatory factors that modulate leukocytes. Psoriasis is a chronic inflammatory skin disease, characterized by KC hyperproliferation, vasculature growth, and leukocyte infiltration into the dermis and epidermis. Imiquimod (IMQ) is an immunomodulator used in mice to induce lesions closely resembling human psoriasis. The aim of our work was to assess the impact of desmoglein-4 deficiency in the amount of skin leukocyte infiltration in an IMQ induced psoriasis model in rats. To this end, OFA hr/hr rats, which are mutant for the desmoglein-4 gene and Sprague-Dawley (SD) wild type rats were used. IMQ was administered to both strains in shaved skin for four days to generate psoriasis-like lesions. Skin biopsies from treated and untreated OFA and SD rats were weighed and minced to obtain cell suspensions that were stained with monoclonal antibodies against CD45 (panleukocytary lineage) and CD3 (T cell lineage) conjugated with fluorochromes and analyzed by flow cytometry. Interestingly, we found that IMQ treatment to both groups increased CD45+ CD3+ cells in OFA skin compared to controls, but this difference was much greater and significant in OFA rats (SD 0.79±0.24 vs OFA 4.12±0.75, p<0.05) that correlated with increased inflammatory area and histopathologic changes in OFA rats. These results suggest that desmoglein-4 absence contributes to psoriasis progress, promoting expansion of leukocyte population in skin. Although further research is needed, these results could have a potential impact on the design of clinical therapies for psoriasis progression.

Keywords: Desmoglein-4, psoriasis, imiquimod, immune response

(1894) **DENDRITIC CELLS ARE RESPONSIBLE FOR THE FAILURE IN CTLs GENERATION IN LSP1-DEFICIENT MICE**

Rachel Paola Acland Strack (1), Marine Gros (2), Mercedes Pascual (1), Belkys Maletto (1), María Cristina Pistoresi (1), Ana María Lennon-Dumenil (2), Sebastián Amigorena (2), Gabriel Morón (1)
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