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LXIII REUNIÓN ANUAL DE LA

SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LXVI REUNIÓN ANUAL DE LA

SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

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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

EDITORES RESPONSABLES

Claudia Pérez Leirós
Pablo Baldi
Alberto Crottogini

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JOINT MEETING SAIC SAI SAFIS 2018

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RESPONSIBLE EDITORS

Claudia Pérez Leirós
Pablo Baldi
Alberto Crottogini

(4.62±0.02) groups (p<0.05). When animals were challenged with FMDV, 100% of mice vaccinated with iFMDV-ISPA and commercial vaccine were protected, and only 40% in iFMDV group. A significative increase of Ab α-iFMDV IgG1, IgG2a, IgG2b and IgG3 types was found in iFMDV-ISPA group
At 21 dpv, splenocytes were stimulated in vitro with iFMDV; iFMDV-ISPA group showed greater proliferation than iFMDV, ISPA or PBS groups (p <0.05), and similar that induced by commercial vaccine. A slight increase was observed in the CD4 +/IFNγ+ population in iFMDV-ISPA and commercial vaccine groups compared with iFMDV.
Mice inoculated with ISPA (sc), at 18 hpv, showed a significant increase in granulocytes in axillary lymph nodes and a decrease of granulocytes, DCs, monocytes and LT-CD8+ in spleen.
Calves (n=4) were vaccinated with iFMDV or iFMDV-ISPA, at 30 dpv there was an increase in total α-iFMDV Ab (3.7±0.6) in iFMDV-ISPA group in comparation with iFMDV group (p<0.05). On the other hand, Seroneutralizing antibodies were 1.8± 0.3 (these SN levels are correlated with protected animals) and 1.02±0.02 respectively (p<0.05).
Bovine Dendritic Cells incubated with ISPA showed an increase in the expression of CD40 and IL6.
Inclusion of ISPA in an experimental FMD vaccine induced an increase in humoral and cellular immunity. In mice, we observed a greater protection against viral challenge. In cattle, the titers reached are linked to a Percentage Expectation of Protection higher than 80%.

582. (675) **LIVER FIBROSIS IN HIV/HCV CO-INFECTED INDIVIDUALS: NK REDUCED CYTOTOXICITY IS NOT ASSOCIATED TO A DYSFUNCTIONAL LT-CD4+ STIMULATION**

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Natural killer (NK) cells ameliorate liver fibrosis by killing activated hepatic stellate cells. We have demonstrated in HCV/HIV co-infected patients with advanced liver fibrosis that functionality of peripheral blood NK cells is significantly affected. Since LT-CD4+ modulate NK cell activity, we aimed to evaluate LT-CD4+ regulation of NK cell functionality in HCV/HIV coinfected patients with different stages of liver fibrosis.
LT-CD4+ cells were purified from cryopreserved PBMCs from 30 HCV/HIV+ patients with mild or advanced fibrosis (METAVIR F0/F1 and F4, respectively). Similarly, NK cells were purified from one healthy volunteer. LT-CD4+ were stimulated with anti-CD3/CD28 beads at three different bead-to-cell ratios (1:1 48h, 1:5 and 1:35 24h), and conditioned medium (CM) were collected. Healthy NK cells were pre-activated with CM or vehicle overnight, followed by co-culture with K562 cells. CD107a externalization, and intracellular IFN-γ and TNF-α were measure by FC. Data was analysed using non-parametric statistics.
Frequency of CD107a+ NK cells progressively increased as the intensity of LT-CD4+ stimulation was incremented (p<0.05). Nevertheless, NK cell degranulation was similarly induced whether CM was produced by LT-CD4+ from patients with minimal or advanced fibrosis. Median CD107a+ NK cell percentages following F0/F1 or F4 CM pre-stimulation were 30.5 and 26.8 (1:35), 42.1 vs. 40.0 (1:5) and 65.0 vs. 64.8 (1:1), respectively. Finally, we did not find significant differences regarding cytokine secretion depending on the activation state of LT-CD4+ or the fibrosis levels of the patients. Median IFN-γ+ and TNF-α+ NK cell percentages after F0/F1 or F4 CM pre-stimulation were 7.3 and 10.8 vs. 7.6 and 11.3, respectively.
Results suggest that reduced cytotoxicity of NK cells may not be related to a deficient LT-CD4+ modulation. Lower percentage of NK cells, and/or an impaired NK cell functionality could act as markers of advanced liver fibrosis in HCV/HIV+ patients.

583. (676) **EFFECT OF L. CASEI CRL 431 AND ITS CELLULAR WALL ON THE THYMUS AT DIFFERENT AGES IN A MICE MODEL**

María Florencia Balcells, Ivanna Novotny Nuñez, Gabriela Perdigón, Carolina Maldonado Galdeano
CERELA, CONICET

Thymus is responsible of ontogeny and maturation of T lymphocytes, however, it begins to regress with time.Aim: to study the effect of *L. casei 431* and/or its cell wall in the thymus at different ages. BALB/c mice were divided into groups of age: 21, 28, 45, 90 and 180 days(d), which were subdivided according to the supplement received: Normal control(NC) received the conventional balanced diet and water *ad libitum*; *Lactobacillus casei*(Lc) group received the conventional balanced diet and oral suspension of probiotic bacteria. Mice were sacrificed at the corresponding age. Initial and final weight of the animal, weight of the thymus was taken. Samples were: large intestine for microbiota analysis, serum(S), small intestinal fluid(IF) and thymus for cytokine analysis (IFNγ, IL-6, IL-10, IL-12 p70, TNFα, IL-3). Cytokines determination in culture supernatant of thymus stimulated with the bacteria (B) or its cell wall (W). Lymphocyte population in thymus and in mesenteric lymph nodes (MLN) were analyzed by flow cytometry.Results: probiotic supplementation increased body weight in all groups of age. Body weight/thymus ratio decreased at 28, 45 and 90d in Lc. Supplementation showed decreased population of enterobacteria and increased population of lactobacilli, without significant differences between NC and Lc in the total anaerobic population.Cytokines in cell culture supernatant showed a decrease in IFNγ and TNFα, increased values of IL-10, IL-12 and IL-3, IL-6 values were near to NC in TB and Lc. Decreases in the CD4+ population in thymus showed in Lc group at 45d and significantly increased at 90d respect to NC. In NC CD4+/CD8+ ratio a significant increase at 90d (p <0.05) respect to Lc (NC = 80.72 ± 0.32, Lc = 0.45 ± 0.06).Lc supplementation and in vitro stimulation with bacteria or its W induce effect in thymus at different periods of time in mice.

584. (690) **CLOSTRIDIUM DIFFICILE INFECTION: MURINE MODEL DEVELOPMENT AND IMMUNOLOGICAL CHARACTERIZATION.**

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Centro de Investigaciones y Transferencias del Noroeste de la Provincia de Buenos Aires (CIT NOBA, UNNOBA-CONICET). Centro de Investigaciones Básicas y Aplicadas (CIBA)

Clostridium difficile-associated disease is caused by a gram-positive, anaerobic, spore-forming bacterium *Clostridium difficile* (*C. difficile*) and is the most common cause of antibiotic associated diarrhea. BI/NAP1/027, an emerging strain of *C. difficile*, has dramatically changed the epidemiology of *C. difficile* infection (CDI) with outbreaks of severe disease in North America and Europe. Antibiotics treatment is the standard method of therapy but also the major cause of susceptibility to CDI. Therefore, identification of key effector molecules of host immune response against CDI may provide novel immunotherapies.
After induction of dysbiosis, mice were infected with spores from the hypervirulent BI/NAP1/027 strain. Post-infected mice evidenced typical CDI symptoms such as diarrhea, hunched posture, and weight loss (p<0.01). Mice were euthanized 8 days post-infection and colon was removed to isolated lamina propria mononuclear cells (LPMC). First, we evaluated the expression of pro-inflammatory cytokines on LPMC by flow cytometry. No differences were observed in $CD3^+IFN\text{-}\gamma^+$ cells after CDI, but we detected an increase of $CD3^+CD4^+IFN\text{-}\gamma^+$ cells (p<0.05) and a decrease of $CD3^+CD4^-IFN\text{-}\gamma^+$ cells (p<0.05) in infected mice. We also observed a positive modulation of $CD3^+CD4^+IL\text{-}17A^+$ cells (p<0.05) and in $CD3^+CD4^-IL\text{-}17A^+$ cells (p<0.05) from *C.difficile*-infected mice. Next, we determined the levels of SLAM and ICOS, two costimulatory molecules that regulates the innate and adaptive immune response. LPMC from *C. difficile*-infected mice showed a downmodulation of $CD3^+SLAM^+$ (p<0.01) and $CD3^+ICOS^+$ (p<0.01) cells compared to control mice.
Our results suggest that *C. difficile* infection regulates IFN-γ and IL-17A production by T cells, while inducing a downmodulation of the costimulatory molecules SLAM and ICOS on $CD3^+$ cells. Understanding cytokine-mediated responses and molecular drivers impli-