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Yersinia enterocolitica (Ye) evades the immune response by injecting outer proteins (Yops) into the cytosol of host cells, but the interaction between Yops and host proteins is not completely understood. We have previously described higher expression of Galectin-1(Gal1) in the spleen and Peyers patches (PPs) of mice infected with Ye. Notably, this effect was prevented when mice were infected with a YopP-deficient mutant strain (Ye Δ*yopP*). Considering that Gal1 has an important immunoregulatory role, and has been shown to interact with certain bacterial glycoproteins, we decided to evaluate the role of Gal1 in Ye infection and its potential interaction with YopP. We observed that Gal1 is able to bind Ye and moreover, one of Gal1 ligands in these cells is YopP, as a Ye Δ*yopP* mutant showed decreased Gal1 binding ($p< 0.05$) by flow cytometry when compared to Ye wt. As early control of Ye infection involves activated macrophages (MΦ), we also evaluated Gal1-YopP interactions and their role in the modulation of macrophage function. Quantification of nitrites and TNF production in supernatants of *Lgals1*$^{-/-}$and WT MΦ infected either with Ye wt or Ye *ΔyopP* showed that even though Gal1 and YopP did not influence TNF levels, coordinately inhibited nitric oxide (NO) production ($p< 0.05$). Administration of exogenous recombinant Gal1 (rGal1) was not able to counterbalance the increase in NO levels observed in *Lgals1*$^{-/-}$ MΦ infected with Ye Δ*yopP*. Moreover, *in vivo* experiments showed that *Lgals1*$^{-/-}$ mice orally infected with the Ye Δ*yopP* presented lower bacterial load in PPs when compared to *Lgals1*$^{-/-}$ mice infected with the Ye wt ($p< 0.01$). Exogenous administration of rGal1 was not able to restrain the improved anti-Ye responses generated in absence of Gal1 and YopP. Our data reveal a role for YopP and endogenous Gal1 on macrophage NO production during Ye infection. This could be a promising area to explore the reinforcement of antibacterial responses by targeting Ye-Gal1 interactions.

411. (401) **CHRONIC HEPATITIS B AND CHRONIC HEPATITIS C IMMUNOPATHOGENESIS: SIMILAR BUT NOT THE SAME**

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HBV and HCV are hepatotropic viruses which differ in the way they induce chronic disease. We aimed to compare the hepatic immune response in Chronic Hepatitis B (CHB) and C (CHC) infections and assess their role in liver damage.

Immunostaining was done in 68 formalin-fixed and paraffin-embedded liver biopsies from 26 CHB and 42 CHC treatment-naive patients to characterize liver infiltrate: [Th (CD4+), Th1 (Tbet+), Th17 (IL-17A+), Treg (Foxp3+), and CTL (CD8+)]. Quantification: portal (P)= +/total lymphocytes or lobular= + lymphocytes in 10 fields; (400x). Hepatitis severity and fibrosis were assessed by the modified Knodell (HAI) and METAVIR.

Comparing CHB and CHC lymphocyte prevalence was alike in P areas (Th>CTL>Treg>Th17>Th1). However, CHC patients showed higher frequencies of Treg, Th17 and Th1 cells ($p=0.001$, $p=0.005$ and $p=0.003$, respectively, U-test). In contrast, cell distribution was different in the lobular area (CHB: CTL> Treg>Th17=Th1>Th vs CHC: CTL>Th1>Treg>Th=Th17) with higher frequencies of Th, Th17 and Th1 cells in CHC ($p=0.04$, $p=0.001$ and $p=0.001$, respectively, U-test) compared with CHB. Regarding liver damage, patients with analogous disease stage showed similar cell frequencies but only in CHC P Th17 were associated with advanced fibrosis ($p=0.03$, U-test) and just in CHB P Th ($p=0.04$, U-test) and lobular CTLs and Th17 cells ($p=0.02$ and $p=0.01$, respectively; U-test) were increased in severe hepatitis cases.

Even when all studied populations were identified in CHB and CHC, common and particular features related to liver damage were detected. Lobular CTLs prevalence in both infections implies their contribution in hepatitis pathogenesis. As for CHB, despite the presence of a regulatory microenvironment, CTLs and Th17 cells promote hepatitis severity, suggesting a Treg failure in limiting liver damage but favouring viral persistence. By contrast, CHC showed a highly inflammatory context with CTL and Th1 majority and Th17 cells enhancing liver fibrosis.

412. (402) **GERMINAL CENTER REACTION IN T*RYPANOSOMA CRUZI* INFECTION: CHARACTERIZATION OF FOLLICULAR CYTOTOXIC CD8+T CELLS**

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Germinal Centers (GCs) are specialized structures generated within the B cell follicles in response to T cell-dependent antigens in which B cells differentiate into antibody-secreting cells and memory B cells. Follicular helper T cells (Tfh) are crucial for GCs formation and antibody-affinity maturation. Other GCs-protagonists are follicular cytotoxic CD8+T cells (Tfc), who share gene signatures with Tfh cells but their function is not well established. In some models, Tfc contribute to eliminate infected cells inside the follicle.

Our aim was to study different protagonists of GC reaction and plasmablasts(PB) response along T. cruzi infection. For that, C57BL/6 mice were intraperitoneally infected with 5.000 trypomastigotes of T. cruzi (Tulahuén strain) and the frequency and number of Tfh, Tfc and PB were evaluated by flow cytometry at different days post infection(dpi) in the spleen. Mice injected with PBS were used as controls.

We observed that the peak of the Tfh(CD4+CXCR5+PD-1+ICOS+), Tfc(CD8+CXCR5+PD-1+ICOS+) and PB(B220loCD138+) response was at 18dpi.These responses preceded GC-B cell response (B220+FAS+GL-7+Bcl-6+)which peaked at 28 dpi. Tfc had a higher expression of Bcl-6 and Tcf-1 than non-Tfc CD8+ T cells($p<0,05$). Near 25% of Tfc, but only 3% of non-Tfc, were specific for the immunodominant T. cruzi TSKB20 peptide. Tfc were CD107a+ and IFN-γ, TNF-α, Granzyme B and Perforin-producing cells. By immunofluorescence of spleen sections, at 15 dpi, we detected CD8+T cells inside and around B cells follicles and spatially opposed to follicular dendritic cells; at 23dpi and 28dpi, all CD8+T cells were outside the follicles and some of them in close contact with PB.

To sum up, we observed an activated CD8+T cells subset whose peak of the response was prior to CG, expressed Tfh-related molecules and were observed in close contact with B cells subsets. Tfc could be influencing humoral response, controlling infection more efficiently, or regulating some population of CG.

413. (423) **EFFECT OF PGE2 ON THE FUNCTIONS OF NEUTROPHILS DURING HUMAN TUBERCULOSIS**

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Neutrophils have been associated with tuberculosis (TB) protection but also with excessive inflammatory burden. Previously we showed