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we could barely increase them in vivo after intraperitoneal injections at 12, 15 and 18 dpi in mice. To identify signals that could counteract IL33 effect *in-vivo*, we cultured Tregs from non-INF Sp with IL33 plus IFNg/TNF/IL18, parasites or INF Sp conditioned media. In all cases, trTregs expanded normally. As IL33 could expand trTregs in non-INF mice, we treated INF mice at early dpi (0, 3 and 6) and evaluated disease evolution. This treatment resulted in increased trTreg in all tissues ($p<0.05$) lower levels of plasma LDH, CPK, GOT and Glucose and reduced body weight loss and tissue parasitism at 21 dpi. These was accompanied by an increase of Tc-specific CD8+ T cells and ILC2. We conclude that trTreg cell response is compromised during acute Tc infection likely because the inflammatory milieu is unfavorable for trTregs survival, preventing IL-33-mediated rescue. Yet, early IL-33 treatment improves the course of the acute infection. As trTregs and effector immune cells are increased by IL-33, further studies are required to establish the underlying mechanisms in the IL-33 protective effects.

211. (143) **INTERACTION OF HELJA LECTIN ISOLATED FROM *HELIANTHUS ANNUUS* WITH *CANDIDA ALBICANS* INHIBITS PHAGOCYTOSIS AND PROMOTES PHENOTYPIC MATURATION IN DENDRITIC CELLS.**

Maia Chop[12#], Melisa Radicioni[13#], Marianela Del Rio[13#], Christian Rodriguez Rodrigues[12&], Mariana Regente[13&].
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Background: Helja is a sunflower lectin that recognizes mannose motifs. Candidiasis is a fungal infection caused by *Candida spp*. that affects immunocompromised individuals. This opportunistic yeast can be recognized by dendritic cells (DCs). The aim of this work is to analyze if the interaction between Helja and *Candida albicans* (*Ca*) cell wall could inhibit recognition and phagocytosis by DCs and modulate cell maturation. Methods: Bone marrow dendritic cells (BMDCs) were cultured in RPMI 1640 medium supplemented with FLT3-L. Helja was purified from sunflower seeds with a D-mannose-agarose resin. Phagocytosis assay was evaluated by FACS and Confocal Microscopy. *C. albicans* (NGY152 strain) were stained with FITC and then incubated with Helja for 45 minutes prior to BMDC stimulation. LPS (100 ng/ml) and 4°C incubation were used as controls. BMDCs phenotype was analyzed by FACS. Results: Confocal images and quantification of labeled-yeast revealed that the preincubation of fungal cells with Helja inhibited recognition by BMDCs and induced a statistically significant reduction on phagocytosis compared to control (n=2, ****$p<0.0001$ Helja-*Ca*+LPS-treated BMDCs vs *Ca*+LPS-treated BMDCs). This observation was confirmed by measuring FITC+ dots in the BMDC gate by FACS analysis (Helja-*Ca* treated BMDCs -53,7%- vs *Ca*-treated BMDCs -60.4%). Finally, *Ca* preincubated with Helja improved the upregulation of MHCII compared to untreated cells or *Ca*-stimulated BMDCs (n=3 **$p<0.01$). The same pattern was observed in the co-stimulatory molecule CD86, Helja-*Ca* treated BMDCs induced a significant increase in the percentage of CD86+ cells (54.1% vs 42.0% observed in *Ca*-treated BMDCS or 24.0% observed in untreated cells, n=3). Conclusions: These data suggest that the mannose-binding lectin blocks epitopes on *Candida* cell wall important for DC recognition. However, Helja promotes DCs maturation, a process required to activate adaptive immunity, highlighting its potential biomedical application.

212. (145) **A MANNOSE-BINDING LECTIN FROM *HELIANTHUS ANNUUS* RECOGNIZES GP120 FROM HIV-1 AND PROMOTES PHENOTYPE MATURATION IN DENDRITIC CELLS**

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#& equal contribution

Background: Plant lectins have potential use in biomedicine as antimicrobial reagents_due to their binding specificity to glycoconjugates. Helja is a mannose-binding lectin isolated from sunflower seeds. Half of the molecular mass of the HIV-1 envelope glycoprotein (gp120) is constituted by high-mannose and complex N-glycans. The aim of this work is to analyze if Helja could recognize gp120 and induce dendritic cell maturation. Methods: Helja was purified from *Helianthus annuus* seeds on a D-mannose-agarose affinity chromatography. Potential molecular interaction between Helja and recombinant gp120 (M Group, B Subtype, SinoBiological, USA) was evaluated by dot blot and ligand blot approaches using anti-Helja antibodies. Bone marrow dendritic cells (BMDCs) were cultured in complete RPMI medium supplemented with FLT3-L. Cellular activation state was evaluated in Helja-treated BMDCs by measuring membrane proteins (CD86, CD40, MHCII, CD11c, CD80, Clec9a, Ly6G, SIPRα, CD103, SiglecH) by FACS. Results: The interaction of Helja with gp120 was initially detected in dot blot assays by immunodetection of lectin bound to viral protein immobilized on nitrocellulose membranes. This interaction was confirmed in ligand blot assays by detecting a signal whose molecular weight corresponds to gp120 (95 kDa). On the other hand, the presence of Helja (10 μg/ml) in BMDC culture induced an upregulation of MHCII, MHCI, CD80, CD40 and CD86 (n=3, **$p<0.01$ Helja-treated BMDCs vs controls, *$p<0.05$ Helja+LPS-treated BMDCs vs LPS control). No changes in Clec9a, Ly6G, SIRPα, CD103 and SiglecH were detected. **Conclusions:** These results suggest that Helja induces dendritic cell maturation, important to promote immune activation, and recognizes specific glycosidic residues and arrangements on the HIV-1 envelope protein which could have a potential biomedical use as a phyto-neutralizing agent in viral infection.

213. (216) **P2Y6 RECEPTOR ACTIVATION IS NECESSARY TO INDUCE PHAGOPTOSIS OF NEURONS BY B. ABORTUS-ACTIVATED MICROGLIA**

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B. abortus-activated microglia kill neurons through primary phagocytosis or phagoptosis. Phagocytosis is a finely regulated process that involves the interaction of different receptors and their ligands. It has been shown that the purinergic pathway is involved in the modulation of different functions of phagocytes. The objective of this work was to investigate whether this signaling pathway is involved in the phagoptosis of neurons mediated by B. abortus-activated microglia. Primary cultures of neurons and microglia from Balb/c mice were infected. Neuron survival was assessed at 48 h by fluorescence microscopy. Co-cultures were treated with apyrase (an enzyme that degrades di and tri nucleotides), Reactive Blue 2 (RB2) (a P2X/P2Y purinergic receptor inhibitor), BBG (a P2X7 specific inhibitor) and MRS2578 (a P2Y6 specific inhibitor). Treatment of B. abortus-infected co-cultures with apyrase inhibited neuronal death, when compared to untreated cultures ($p<0.05$). Treatment of B. abortus-infected co-cultures with RB2 also prevented neuronal death ($p>0.05$). By using the specific inhibitors of P2X7 and P2Y6, we were able to demonstrate that the P2Y6, but not P2X7 purinergic receptor, is involved in the modulation of phagoptosis ($p>0.05$). In all cases microglia activation was not affected since TNF-α secretion was not significant different between treatments ($p>0.05$). These results demonstrate that the P2Y6 purinergic receptor and the nucleotides that activate it would be necessary for neuronal death mediated by microglia activated by B. abortus, describing new molecular mechanisms involved in the pathogenesis of neurobrucellosis.