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REUNIÓN CONJUNTA DE SOCIEDADES DE BIOCIENCIAS

LXII REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LIII REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE INVESTIGACIÓN BIOQUÍMICA Y BIOLOGÍA MOLECULAR (SAIB)

LXV REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

REUNIÓN DE LA SOCIEDAD ARGENTINA DE ANDROLOGÍA (SAA)

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REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE FISIOLOGÍA (SAFIS)

REUNIÓN DE LA SOCIEDAD ARGENTINA DE HEMATOLOGÍA (SAH)

XXIX REUNIÓN ANUAL DE LA SOCIEDAD ARGENTINA DE PROTOZOOLOGÍA (SAP)

13-17 de noviembre de 2017
Palais Rouge– Buenos Aires

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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)

MEETING OF ARGENTINE SOCIETY OF ANDROLOGY (SAA)

XLVI ANNUAL MEETING OF ARGENTINE SOCIETY OF BIOPHYSICS (SAB)

XIX ANNUAL MEETING OF ARGENTINE SOCIETY OF BIOLOGY (SAB)

XLIX ANNUAL MEETING OF ARGENTINE SOCIETY OF EXPERIMENTAL PHARMACOLOGY (SAFE)

ANNUAL MEETING OF ARGENTINE SOCIETY OF PHYSIOLOGY (SAFIS)

MEETING OF ARGENTINE SOCIETY OF HEMATOLOGY (SAH)

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apoptosis ($p<0.001$). To capitalize on this information for therapeutic purposes, we studied physicochemical properties that hinder translation of this immunoregulatory lectin to clinical settings. We found that Gal1-induced apoptosis of T cells is impaired by both acidic ($p<0.05$) and oxidative conditions ($p<0.01$), typical hallmarks of inflammatory settings. Thus, we genetically engineered Gal1 protein to generate stable mutants capable of circumventing these limitations. These mutants showed enhanced capacity to induced apoptosis of activated T cells ($p<0.05$), IL-10 secretion by T cells ($p<0.001$) and IL-27 secretion by DCs ($p<0.001$). Finally, these mutants showed enhanced therapeutic potential in mouse models of MS (EAE) ($p<0.001$), arthritis (CIA) ($p<0.01$) and colitis (TNBS-IBD) ($p<0.05$). Our findings provide novel therapeutic strategies for treating a broad range of autoimmune diseases.

SAI – SATZ AWARD

BIOINFORMATICALLY GUIDED DISCOVERY OF NOVEL Trypanosoma cruzi EPITOPES RECOGNISED BY CD4⁺ AND CD8⁺ T CELLS FROM CHRONIC CHAGAS DISEASE PATIENTS

GONZALO RAÚL ACEVEDO (1), LUCAS PÉREZ PERRI (1), MAGALÍ CELESTE GIRARD (1), MARISA FERNÁNDEZ (2), YOLANDA HERNÁNDEZ (2), RAÚL CHADI (3), MORTEN NIELSEN (4), KARINA ANDREA GÓMEZ (1).

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T cell-mediated response has been proven to play a major role in controlling *T. cruzi* infection and parasite burden. It also seems to be involved in the progression from asymptomatic Chagas disease (ACD) to chronic Chagas cardiopathy (CCC) in chronically infected patients. However, the complexity of the parasite-host interactions hampers the identification and characterization of T cell activating epitopes. We approached this issue by combining *in silico* and *in vitro* methods to interrogate patients' T cells specificity.

The sequences of the 53 *T. cruzi* proteins annotated on the Immune Epitope Database (IEDB) were split in all possible 15-aminoacid long peptides, and 50 candidate peptides were selected using a bioinformatic pipeline (MHCpan, MHCIIpan, PopCover), based on conservation between annotated parasite strains, non-identity with *H. sapiens* sequences, class I and II MHC molecules binding affinity and HLA polymorphic variants coverage. Candidate peptides were randomised in 5 pools of 10 peptides each, which were used to challenge peripheral blood mononuclear cells (PBMC) from chronic Chagas disease patients, in IFN-γ ELISPOT assays. Positive pool-patient pairs were re-assayed in a single-peptide fashion to identify individual active peptides. A total of 7 peptides induced IFN-γ secretion in at least one patient's PBMC, 4 of which do not contain any previously described epitope. In combination, response to these peptides covered 33% of the patients cohort in this study (n=51), 40% of the ACD group (n=25) and 27% of the CCC group (n=26). IFN-γ ELISPOT with CD8- or CD4-depleted PBMC showed that 6 of the peptides contain MHC class II epitopes, while 1 contains an MHC class I epitope. The fact that most of these subjects responded to 1 or 0 peptides is a strong sign of HLA restricted epitopes. In summary, we predicted, validated and characterized 7 T cell-activating peptides from *T. cruzi* antigens, 4 of which contain epitopes first described in this work.

THE BTK INHIBITOR IBRUTINIB IMPAIRS THE INNATE IMMUNE RESPONSE AGAINST Mycobacterium tuberculosis MEDIATED BY MACROPHAGES AND γδ T CELLS.

COLADO, ANA (1); GENOULA, MELANIE (2); KVIATCOVSKY, DENISE (2); PODAZA, ENRIQUE (1); RISNIK, DENISE (1); ELÍAS, ESTEBAN (1); MARÍN FRANCO, JOSÉ LUIS (2); COUGOULE, CÉLINE (3); MARIDONNEAU-PARINI, ISABELLE (3); JANCIC, CAROLINA (4); GIORDANO, MIRTA (1,5); SASIAIN, MARÍA DEL CARMEN (2); GAMBERALE, ROMINA (1,5); BALBOA, LUCIANA (2) Y BORGE, MERCEDES (1,5).

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The Bruton's Tyrosine Kinase (Btk) inhibitor, ibrutinib (ibru), was recently approved for the treatment of Chronic Lymphocytic Leukemia (CLL) patients. Previously we found that ibru affected macrophage polarization and