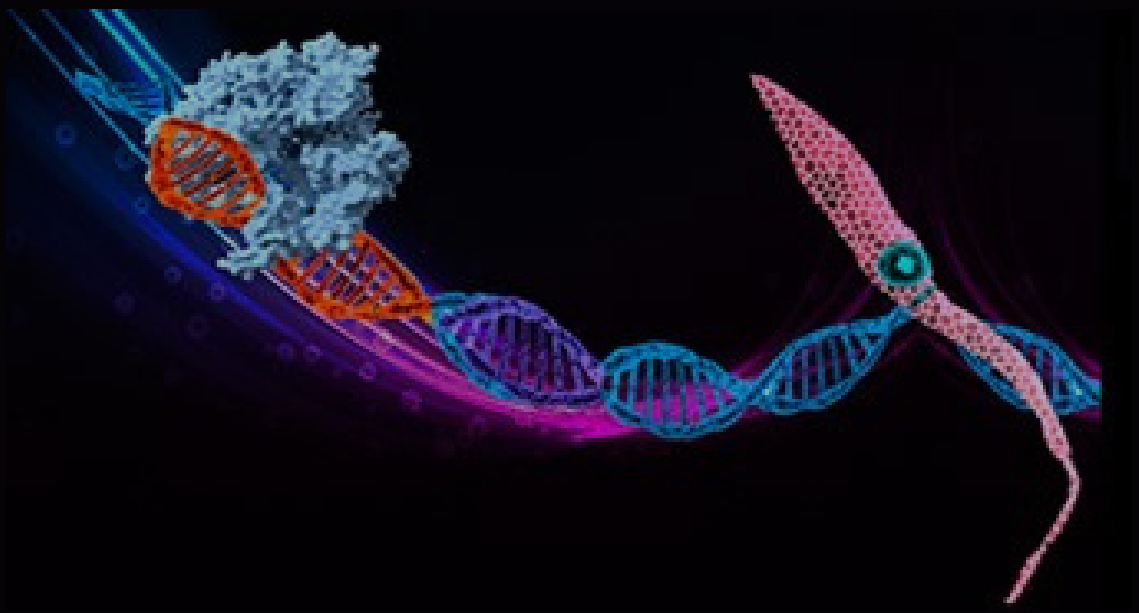
XXXVI Annual Meeting of the Brazilian Society of Protozoology

47th Annual Meeting of Fundamental in Chaga´s Disease

AUGUST 30th
SEPTEMBER 1st, 2021

ONLINE



SBPz

PROCEEDINGS

XXXVI Meeting of the Brazilian Society of Protozoology
XLVII Annual Meeting on Basic Research in Chagas' Disease

August 30th – September 1st , 2021
ONLINE

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Dear Participants,

On behalf of the Brazilian Society of Protozoology (SBPz) and the Organizing Committee, it is a great pleasure to welcome you all to the **XXXVI Meeting of the Brazilian Society of Protozoology** and the **XLVII Annual Meeting of Basic Research in Chagas´ Disease**, the so-called "**2021 Caxambu Meeting**".

Last year the Covid-19 pandemic surprised us and ended up preventing us from holding our traditional Annual Meeting. This year, still in a pandemic situation, we are conducting the Annual Meeting in a virtual format but honouring the spirit and the tradition of the meeting, we invited few keynote speakers and researchers from different countries and from Brazil to compose our scientific programme. In previous activities, that took place in April, May, and June, we organized three mini-courses and e Round Tables to discuss Immunology, Phylogenetic and In vivo cell culture of protozoa.

Despite the virtual format, we are keeping the students and young researcher's participation in virtual poster presentation that will culminate in discussion sections that we hope can be very full of challenges but resulting in productive participation of the audience. This way, we are guaranteeing the Zigman Brener Awards to be given to the best poster of different categories.

This year's Samuel Pessoa Award will be granted to Professor Santuza Maria Ribeiro Teixeira in recognition to her outstanding contribution to the field of protozoology.

Together with our sister society, British Society for Parasitology, we are also encouraging young researchers to discuss their problems in relation to the development of their careers and search for permanent positions in a Satellite Meeting that will allow them to create a favorable scenario for their work as scientists in research and teaching institutions.

We would like to acknowledge the invited speakers, SBPz members, colleagues, and students for supporting and attending this meeting. We are particularly indebted to the SBPz Secretariat, Mrs Ana Paula Lopes Vidal and Mrs Vilma de Araújo Andrade, whose commitment and dedication over the years along distinct Board of Directors have assured the persistence of the SBPz, even in times like the actual pandemic scenario, guaranteeing the quality of the meeting and contributed to its international recognition. We would also like to acknowledge the financial support received THE COMPANY OF BIOLOGISTS (UK) and FAPESP.

So, we are very glad to welcome you all to this virtual "Caxambu Meeting", hoping that the scientific program covers expectations and provides high-level discussions as they have always occurred in face-to-face meetings.

São Paulo, August 30th, 2021

Lucile M Floeter Winter

President of SBPz

HP-039 - **HYPERACTIVITY IN A MURINE MODEL OF LONG-LASTING CHRONIC Toxoplasma gondii IS REFRACTARY TO SULFADIAZINE AND PYRIMETHAMINE THERAPY**

BARRIOS, L.M.C.[1]; PINHEIRO, A.P.[2]; GIBALDI, D.[2]; DA SILVA, A.A.[3]; SILVA, P.M.R.E.[1]; MINEO, J.[4]; SILVA, N.[4]; LANNES-VIEIRA, J.[1].

1. FUNDAÇÃO OSWALDO CRUZ, RIO DE JANEIRO - RJ - RJ - BRA; 2. FUNDAÇÃO OSWALDO CRUZ, RIO DE JANEIRO - RJ - BRA; 3. UNIVERSIDADE FEDERAL FLUMINENSE, NITEROI - RJ - BRA; 4. UNIVERSIDADE FEDERAL DE UBERLÂNDIA, UBERLÂNDIA - GO - BRA

Toxoplasma gondii, an intracellular protozoan, is the etiologic agent of toxoplasmosis, an infectious disease of medical importance. The *T. gondii* protozoan is found worldwide and currently one third of the world population is seropositive. The parasite has tropism for the central nervous system (CNS), where it remains for long periods influencing the behavior of the host. In fact, infection by *T. gondii* has been related to mental illnesses such as schizophrenia, bipolar disorder, obsessive-compulsive disorder, and other behavioral abnormalities. The aim of the present study was to determine whether the presence of *T. gondii* cysts in the CNS leads to hyperactivity in chronically infected animals. For that, 4-6 weeks old C57BL/6 mice were infected with the cystic form of the ME-49 type II *T. gondii* strain and evaluated at 30-, 60- and 90-days post-infection (dpi). Later, we tested the role of parasite in the behavioral alterations. Thus, chronically infected mice were orally treated for 30 days (30 to 60 dpi) with a combined therapeutic strategy with sulfadiazine (S) and pyrimethamine (P). The open field (OF) and elevated plus maze (EPM) tests, widely used to assess hyperactive and impulsive components of attention deficit hyperactivity disorder (ADHD), respectively, were used. Subsequently, the number of cysts present in the brain was evaluated. Hyperactivity was detected in the early (30 dpi) and long-term (60 and 90 dpi) chronic *T. gondii* infection. For example, at 60 dpi infected mice showed increase in walking speed in the OF than non-infected (NI) mice, supporting the presence of hyperactivity. In addition, infected animals remained longer time exploring the open arms of the EPM, when compared to NI, indicative of impulsivity. The S+P treatment reduced the number of cysts in the CNS, when compared to vehicle. However, there was no effect on the hyperactivity observed in the OF, nor on impulsivity in the EPM. **Keywords:** Behavioral tests.ME-49 strain.Hyperactivity

HP-040 - **Expression and functionality of co-inhibitory receptors TIGIT, TIM-3 and LAG-3 in CD4+ T cells from patients with chronic Chagas disease.**

FERRAGUT, F.E.D.V.[1]; ALCARÁZ, P.[1]; CHADI, R.[2]; FERNANDEZ, M.[3]; HERNANDEZ, Y.[3]; GÓMEZ, K.[1]; ACEVEDO, G.[1].

1. INSTITUTO DE INVESTIGACIONES EN INGENIERÍA GENÉTICA Y BIOLOGÍA MOLECULAR “DR. HÉCTOR N.TORRES”, - ARG; 2. HOSPITAL GENERAL DE AGUDOS “DR. IGNACIO PIROVANO”, - ARG; 3. INSTITUTO NACIONAL DE PARASITOLOGÍA “DR. MARIO FATALA CHABÉN” (INP-ANLIS), - ARG

Chagas disease, caused by *Trypanosoma cruzi*, affects ~7 million people worldwide, mainly in Latin America where it is endemic. Unless treated early after infection, the disease progresses to a chronic form in which some patients develop cardiac or digestive alterations, while others stay asymptomatic. $CD4^+$ T cell response plays critical and diverse roles during infection, and becomes impaired over time with defective cytokine release. This process known as T cell exhaustion is also defined by the upregulation of inhibitory receptors, mainly PD-1 and CTLA-4. In this work, we aimed to explore the expression of the second line of inhibitory receptors TIGIT, TIM-3 and LAG-3 in parasite stimulated $CD4^+$ T cells from patients with different stages of chronic Chagas disease (CCD), and whether their blockade restores cell functionality. Antigen-specific $CD4^+$ T cells were identified by activation induced markers (AIM) assay, using the surface molecules Ox40 and CD25. CCD patients, independently of their clinical stage, showed an increased frequency of $CD4^+TIGIT^+$ T cells. $TIM\text{-}3^+$ $CD4^+$ T cells were more abundant in patients with cardiac manifestations, while $LAG\text{-}3^+$ cells were increased in asymptomatic CCD patients, with higher frequency within the non-activated cells subpopulation upon *T. cruzi* lysate stimulation. Preliminary data showed that TIM-3 blockade tended to increase $IFN\text{-}\gamma^+$ $CD4^+$ T cells in CCD patients with cardiomyopathy. Furthermore, in the same group of subjects, the incubation with an anti-TIGIT blocking antibody led to a greater frequency of $IL\text{-}10^+$ $CD4^+$ T cells compared to the isotype control antibody. Our results highlight the role of the inhibitory receptors TIGIT, TIM-3 and LAG-3 in the modulation of anti-*T. cruzi* $CD4^+$ T cell responses, in relation with the progression of chronic Chagas disease, and paves the way for the development of novel therapeutic strategies. **Supported by:**PIP 2015 **Keywords:** chronic Chagas disease.T-cell exhaustion.inhibitory receptors