SAIB

54th Annual Meeting Agentine Society for Biochemistry and Molecular Biology

LIV Reunion Anual Sociedad Argentina de Investigación en Bioquímica y Biología Molecular

Paraná, Entre Ríos, Argentina

5 al 8 de Noviembre de 2018

CONICET

FONCYT

FONDO PARA LA INVESTIGACION CIENTIFICA Y TECNOLOGICA

AGENCIA

NACIONAL DE PROMOCION CIENTIFICA Y TECNOLOGICA

ISSN 1667-5746 (online version)

MEMBERS OF THE SAIB BOARD

Silvia Moreno
President
IQUIBICEN CONICET
Facultad de Cs Exactas y Naturales Universidad de Buenos Aires

Maria Isabel Colombo
Vicepresident
IHEM CONICET
Facultad de Ciencias Médicas
Universidad Nacional de Cuyo – Mendoza

Silvia Moreno
President
IQUIBICEN CONICET
Facultad de Cs Exactas y Naturales Universidad de Buenos Aires

Maria Isabel Colombo
Vicepresident
IHEM CONICET
Facultad de Ciencias Médicas
Universidad Nacional de Cuyo – Mendoza

José Luis Bocco
PastPresident
CIBICI CONICET
Facultad de Ciencias Químicas
Universidad Nacional de Córdoba

Silvia Rossi
Secretary
IQUIBICEN CONICET
Facultad de Cs Exactas y Naturales
Universidad de Buenos Aires

Sandra Ruzal
Treasurer
IQUIBICEN CONICET
Facultad de Cs Exactas y Naturales Universidad de Buenos Aires

Gabriela Salvador
Prosecretary
INIBIBB CONICET
Universidad Nacional del Sur

ISSN 1667-5746 (online version)

Eleonora Garcia Véscovi
Protreasurer
IBR CONICET
Facultad de Ciencias Bioquímicas y Farmacéuticas
Universidad Nacional de Rosario

Silvia Belmonte
Auditor
IHEM CONICET.
Facultad de Ciencias Médicas
Universidad Nacional de Cuyo - Mendoza

Romina Uranga
Auditor
INIBIBB CONICET.
Universidad Nacional del Sur

DELEGATES OF SCIENTIFIC SESSIONS

CellBiology

Gustavo Chiabrando
CIBICI CONICET
Facultad de Ciencias Químicas
Universidad Nacional de Córdoba

Lipids
Hugo Gramajo
Facultad de CienciasBioquimicas y Farmaceuticas
Universidad Nacional Rosario
IBR-CONICET

Plants
Paula Casati
Facultad de Ciencias Bioquimicas y Farmaceuticas
Universidad Nacional Rosario
CEFOBI-CONICET

Microbiology
Monica Delgado
Instituto Superior de Investigaciones Biológicas - Instituto de Química Biológica “Dr. Bernabé Bloj”
Universidad Nacional de Tucumán

Signal Transduction
Mario Rossi
IBIOBA-CONICET

FORMER PRESIDENTS SAIB

2016-2017	JOSE LUIS BOCCO
2014-2015	CARLOS ANDREO
2012-2013	LUIS MAYORGA
2010-2011	ALBERTO KORNBLIHTT
2008-2009	BEATRIZ CAPUTTO
2006-2007	NESTOR CARRILLO
2004-2005	ERNESTO PODESTA
2002-2003	NORMA STERIN DE SPEZIALE
2000-2001	RICARDO WOLOSIUK
1998-1999	DIEGO DE MENDOZA
1996-1997	RICARDO BOLAND
1994-1995	MIRTHA FLAWIA
1992-1993	ARMANDO J. PARODI
1990-1991	JUAN J. CAZZULO
1988-1989	HUGO MACCIONI
1986-1987	ISRAEL D. ALGRANATI
1984-1985	RICARDO FARIAS
1982-1983	JOSÉ SANTOME
1981-1981	HECTOR TORRES
1980-1980	JUAN DELLACHA
1979-1979	MARCELO DANKERT
1978-1978	FEDERICO CUMAR
1977-1977	ANTONIO BLANCO
1976-1976	HÉCTOR BARRA
1975-1975	RAÚL TRUCCO
1973-1974	ALEJANDRO PALADINI
1972-1972	HORACIO PONTIS
1971-1971	ANDRES STOPPANI
1970-1970	RODOLFO BRENNER
1969-1969	RANWEL CAPUTTO
1965-1968	LUIS F. LELOIR

MI-P06
BIOFILM FORMING PATHOGENIC *Klebsiella pneumoniae* AND *Escherichia coli* STRAINS: SIDEROPHORE PROFILE

Juarez GE; Feehan KT; Galván EM
Centro Estudios Biomédicos, Biotec., Ambient. y Diagnóstico (CEBBAD), Universidad Maimónides, CABA.
E-mail: juarez.guillermo@maimonides.edu

We have reported that the antagonism between uropathogenic strains of *K. pneumoniae* and *E. coli* in mixed biofilms in artificial urine medium (AUM) is due to nutrient competition with a greater ability of *K. pneumoniae* to utilize ferric iron. We aim to investigate the siderophore production profile of both strains and its role in inter-species competition. Genotypic characterization was performed by PCR using specific primers for siderophore genes related to its biosynthesis and its receptor. Bacteria were grown planktonically in modified M9 (MM9) for 2 d at 37°C and as biofilms in AUM for 5 d at 37°C. Culture supernatants were evaluated for both siderophore production by chrome azurol S (CAS) assay and chemical determination of catechol- and hydroxamate-containing molecules. Results showed that both strains contain enterobactin (cathechol) related genes, but only *E. coli* also has salmochelin (cathecol), aerobactin (hidroxamate), and yersiniabactin (mixed) related genes. CAS assays evidenced the production of siderophores by both species when growing in MM9. *E. coli* produce both catechol- and hydroxamate-containing compounds (89±33 and 111±50 pmol/10^6 cells), whereas *K. pneumoniae* only displayed catechols (47±17 pmol/10^6 cells). Biofilms showed similar levels of catechols in all single-species *K. pneumoniae* and *E. coli* biofilms and mixed biofilms. Single-species *E. coli* biofilm express higher amounts of hydroxamates, compared to mixed biofilms. Even when *E. coli* produce a higher amount and variety of siderophores than *K. pneumoniae*, in mixed biofilms, a differential siderophore utilization might take place, allowing *K. pneumoniae* to outcompete *E. coli*.

MI-P07
TRANSCRIPTOMIC ANALYSIS IN BLV-EXPERIMENTALLY INFECTED CATTLE LEADING TO HIGH OR LOW PROVIRAL LOAD

Juliarena MA[1]; Forletti A[2]; Lützelschwab CM[1]; Gutiérrez SE[1]
[1]Lab de Virología, Dpto SAMP, CIVETAN, Fac de Cs Veterinarias, UNCPBA-CIC-CONICET. [2]BIOTANDIL SRL
E-mail: mjuliare@vet.unicen.edu.ar

Bovine Leukemia Virus (BLV) is a highly prevalent pathogen causing a fatal lymphoproliferative disease in the bovine species. After experimental infection, proviral load peaks at 30 days post infection (dpi), then, cattle progress to two different phenotypes: one is characterized by high proviral load (HPL) in peripheral blood, while the other is identified by low proviral load (LPL) in peripheral blood. In LPL cattle a sharp decrease in proviral load is evidenced at 38 dpi. We studied the transcriptome in peripheral blood cells from 10 cattle (5 of each phenotype) infected with BLV at 38 dpi, to identify the host genes differentially expressed in those animals that progress to LPL, recognized as BLV-resistant. RNA seq experiments showed 499 genes differentially expressed ($p<0.05$) between both phenotypes: 281 upregulated and 218 downregulated in LPL compared to HPL cattle. Gene ontology analysis revealed that genes related to inflammatory response, humoral immune response and leukocyte migration were up regulated in cattle progressing to LPL compared to HPL ($p< 0.05$), while the regulation of homeostatic process, antigen receptor signaling pathway and phospholipid transport were downregulated in LPL compared to HPL cattle ($p< 0.05$). The huge difference in transcript expression found at 38 dpi, suggests that mechanisms used to control the proviral load are turned on early after the infection, although the phenotype of LPL or HPL is established only after 90 dpi.

MI-P08
DEPLETION OF TbRRM1 INDUCES RNA POL II TRANSCRIPTION-ELONGATION IMPAIRMENT AND R-LOOPS ACCUMULATION

Bañuelos CP; Levy GV; Níttolo AG; Saborit JI; Tekiel V; Sánchez DO
Instituto de Investigaciones Biotecnológicas, Universidad Nacional de San Martín (IIB-UNSAM)-CONICET. E-mail: caropau86@gmail.com

TbRRM1 is an essential SR-related RNA binding protein from *Trypanosoma brucei*, the causative agent of sleeping sickness. Previous studies from our lab indicate that TbRRM1 depletion leads to both decreased RNA Pol II transcription-elongation rate and compacted chromatin in a particular polycistronic transcription unit.In the present work we showed, by chromatin and RNA immunoprecipitation assays, that TbRRM1 is both recruited to chromatin and to specific RNAs. Given this association, we further characterized TbRRM1 binding properties in the presence of RNAse A, RNAse H and Actinomycin D. Interestingly, TbRRM1 recruitment to chromatin increased under these treatments, thus suggesting that RNA and chromatin compete for TbRRM1 binding. In addition, we showed by RTqPCR and chromatin fractionation, that the abundance of transcripts belonging to genes downregulated after TbRRM1 depletion increases in the chromatin-associated RNA fraction. Finally, as the RNAse H results suggested that TbRRM1 binds DNA-RNA hybrid, we studied whether *TbRRM1* knockdown induces the formation of R-loops. To this end, we performed indirect immunofluorescence assays with the S9.6 antibody. TbRRM1 depleted cells showed a significant increase in the number of positive intranuclear dots, thus suggesting that TbRRM1 prevents R-loops accumulation.Altogether, our results suggest that RNA Pol II transcription-elongation impairment, induced by TbRRM1 depletion, might be a consequence of RNA Pol II slowing down due to R-loops accumulation. Our hypothesis is that TbRRM1 helps to displace the nascent mRNAs from the site of transcription, which prevents the formation of R-loops.