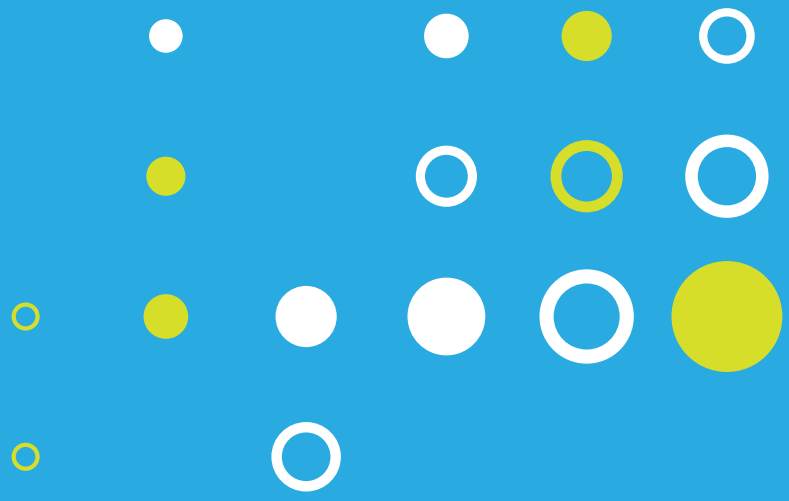
BIOCELL

nº 42

ISSN: 0327-9545 (print)
ISSN: 1667-5746 (online)

December 2018



SAIB
Sociedad Argentina de
Investigaciones en Bioquímica
y Biología Molecular

SAIB

54th Annual Meeting Agentine Society for Biochemistry and Molecular Biology

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

CONICET

FONCYT

AGENCIA

BIOCELL 42 (suppl. 4), 2018
ISSN 1667-5746 (online version)

MEMBERS OF THE SAIB BOARD

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

BIOCELL 42 (suppl. 4), 2018
ISSN 1667-5746 (online version)

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

BT-P16
MORPHOLOGY, NUTRITIONAL COMPOSITION AND ACCUMULATION OF ASTAXANTIN IN*Oedocladimcirratum*

Marsili SN[1], Rearte TA[2], Cerón - García, MC[3], Pitta-Alvarez S[1], Vélez CG[1]
[1]Lab. of Microalgae Experimental Cultivation, FCEN, UB, [2]Cat. of Analytical Chemistry, FA, UBA, [3]Dept. of Chemical Engineering, UAL, Spain
E-mail:santiagonicolasmarsili@gmail.com

The objective of the present work is to evaluate the morphological characteristics, nutritional composition and accumulation of carotenoids with special emphasis on astaxanthin, product of great commercial interest, due to the effect of nitrogen limitation on the algae *O. cirratum* (UTEX LB 1532) of terrestrial habit (humid soils). As an adaptation to face conditions of rapid desiccation of soils and high intensities of sunlight, the vegetative cells can develop thick cell walls and accumulate a high amount of carotenoids, differentiating into acinetas. Cultures for vegetative growth were performed in 1-liter bioreactors of standard mineralized medium, with aeration and continuous light at 23 ±1 °C and then this culture was subjected to nutritional stress conditions in standard mineralized medium without N source for production of acinetas rich in astaxanthin. This work presents the observations under growing and stress conditions and the nutritional composition of vegetative cells: total lipids. (5.27 ± 0.77%); proteins (44.6 ± 5.1%); carbohydrates (48.1 ± 4.4%); chlorophyll a and b (2.42 ±0.66 and 4.49 ± 0.39 mg / g) and acinetas: total lipids (23.7 ± 3.16%); proteins (28.2 ± 3.16%); carbohydrates (19.3 ±2.25%); chlorophyll a and b (1.61 ±0.43 and 1.49 ± 0.48 mg / g); total carotenoids (8.19 ± .46 mg / g). *O. cirratum* has a high potential for the production of astaxanthin since it is possible to induce the accumulation by modifying the culture conditions obtaining high levels compared, mainly, with *H. pluvialis*.

CELL BIOLOGY

BC-P01
A SINGLE NMT IS RELEVANT FOR *Toxoplasma gondii*LYTIC CYCLE

Alonso AM[1],Turowski VR[1], Ruiz DM[1],Orelo BD[2], Moresco JJ[2], Yates JR[2],Corvi MM[1]
[1]IIB-INTECH, Chascomus. [2]The Scripps Research Institute, La Jolla, California. E-mail: amalonso@intech.gov.ar

Toxoplasma gondii is the causative agent of toxoplasmosis. This disease affects almost one third of the world's population with devastating effects. Despite the significant progress that has been made in order to develop new compounds to treat toxoplasmosis, the current therapeutic agents frequently used have toxic side effects. As such, scientists are in real need of finding new targets of intervention.Protein myristoylation is a post- and co-translational modification that affects a variety of proteins in many cells including parasites. It is catalyzed by N-myristoyltranferase (NMT), a conserved enzyme that has been described to be essential in many protozoan pathogens. However, up to date, there is scarce information on NMT and the extent of this modification in *T. gondii*. In this work *T. gondii* NMT (TgNMT) was identified and characterized. Structural analyses suggest that there are differences between human and *T. gondii* NMTs, which could be of importance to design specific inhibitors. Furthermore, this protein presents NMT activity *in vitro*, is expressed in both intra- and extracellular parasites and interacts with predicted TgNMT substrates. Additionally, TgNMT activity seems to be important for the lytic cycle. An *in silico*myristoylome predicts 157 proteins to be targeted by this modification with some of them being critical for the life cycle of this parasite. This analysis suggests that myristoylation could be regulating calcium homeostasis which is critical for *T.gondii* pathogenesis. Together, these data indicate that TgNMT could be an interesting target of intervention for the treatment of toxoplasmosis**.**

BC-P02
ANALYSIS OF EPITHELIAL-MESENCHYMAL TRANSITION (EMT) PROCESS IN RENAL COLLECTING DUCTS OF AGING RATS

Brandán YR[1],Guaytima EV[1],Favale NO[2],Sterin-Speziale NB[2], Márquez MG[1]
[1]Instituto de Investigaciones en Ciencias de la Salud Humana-UNLaR. [2]IQUIFIB-CONICET. E-mail: brandanyamila@gmail.com

Renal function declines progressively with age. The EMT process has been suggested as a mechanism that drives fibrosis, with the consequent loss of tissue functions. In previous works, we demonstrated that inhibition of sphingomyelin synthase I (SMS1) activity induces collecting duct (CD) cells to lose their epithelial phenotype and to undergo an EMT process. Now we investigated the occurrence of EMT in renal papilla CD cells of aging rats (6 month). Taking advantage of the fact that CD in primary culture retains many characteristics of their behavior in intact tissue, primary cultures of CD cells from young (70 days) and aging rats were performed. We analyzed the expression of epithelial (α- and β-catenin) and mesenchymal (vimentin and α-smooth muscle actin, α-SMA) cell markers, cell proliferation, and the presence of primary cilia by immunocytochemistry. Contrary to what was observed in young rats, CD cells from aging rats exhibited impairment of cell-cell adhesion, a high expression of vimentin and α-SMA, and a significant increased number of isolated cells with fibroblastoid-like morphology expressing both proteins. We also observed greater proliferation and a decreased number of cells with primary cilium. These features are consistent with the alterations reported in tubular epithelial cells during renal fibrosis. The increased proliferation probably represents a mechanism to restore the integrity of the tubular epithelium. Our results suggest that as aging occurs, the balance between the EMT-MET processes in renal papilla is altered, but the link between these alterations and the impairment of SMS1 activity requires further studies.