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the imbalance between MMPs and TIMPs activities and changes in cell phenotype that result from hyperglycemic environment.

CB-P57
NOVEL STRATEGY TO STUDY ARGININE DEIMINASE PARTNERS IN THE PARASITE *Giardia lamblia*

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Giardia lamblia is a ubiquitous unicellular parasite of humans and other vertebrates that commonly causes diarrhea and gastrointestinal upset. During the last years, we have been studying the multiple roles played by the enzyme arginine deiminase (ADI) during growth and differentiation of this parasite. An interesting feature is the capacity of ADI to interact with different partners from surface protein to histones depending on its localization during the cell cycle of *Giardia*. In this study, we develop a novel strategy to further analyzed ADI partners, by generating monoclonal antibodies (mAb) against in vivo immunoprecipitated *Giardia* ADI. By using ADI-HA as immunogen, obtained from immunoprecipitation using anti-HA mAb, we obtained not only specific mAbs against ADI but also to other proteins that co-immunoprecipitated with it. It is important to highlight the reproducibility of this method using different protein baits, which, in combination with MS-MS, allowed to obtain specific mAbs and also to disclose the architecture of particular protein networks.

CB-P58
HIPPOCAMPAL NEURONAL RESPONSE TO AMYLOID β PEPTIDE OLIGOMERS. BIOLOGICAL AND BIOPHYSICAL INSIGHTS

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We have previously demonstrated that oligomeric amyloid β peptide (oAβ), known as the most harmful species of Aβ, concomitant with iron overload led to synaptic injury and local activation of several signaling cascades. In this work, we characterized hippocampal neuronal response to oAβ exposure both in the presence and absence of iron. HT22 neurons exposed to iron overload displayed increased lipid peroxidation, slight loss of mitochondrial function, and activation of ERK and Akt pathways. oAβ neither induced an increase in lipid peroxidation nor altered mitochondrial function. However, oAβ alone triggered the activation of ERK and Akt, and the coincubation with oAβ/iron restored pAkt and pERK to the control levels. In addition, we also studied the effect of iron, oAβ and both conditions together, on the biophysical state of the plasma membrane by measuring the generalized polarization of the fluorescence probe Laurdan and the fluorescence anisotropy of DPH and TMA-DPH. Both studies showed that the presence of iron (even at the highest concentration tested), oAβ, or both conditions together, did not perturb the lipid order of the membrane. We conclude that oAβ activates signaling pathways in the absence of oxidative stress or membrane disturbances in hippocampal neurons.

CB-P59
ANTITUMORAL EFFECTS OF BIOENERGETIC MODULATION IN FELINE MAMMARY CARCINOMA CELLS

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Feline mammary carcinoma (FMC) is a highly aggressive pathology that has been proposed as an interesting model of breast cancer disease. Most tumor cells metabolism rely in an increase of glycolysis that enhances the malignant phenotype. The aim of the present work was to investigate the effects and mechanisms of metformin (MET, antidiabetic drug), 2-deoxyglucose (2DG, hexokinase inhibitor) and dichloroacetic acid (DCA, piruvate deshydrogenase kinase inhibitor) in ALRB, an established FMC cell line. The antitumor effects of glucose metabolism modulation were evaluated by the acidic phosphatase assay (APH), 5 days after treatments. While all treatments significantly diminished cell viability ($p<0.05$), the combination MET+2DG displayed a potentiated effect that was significantly higher than the addition of the single treatments ($p<0.01$). In addition, MET+2DG caused an increase in both intracellular oxidants and G0/G1 subpopulation (as determined by DCFH-DA and propidium iodide flow cytometry). The drugs here evaluated displayed an increase of autophagic vacuoles (revealed by expression of lipofected RFP-LC3 plasmid). Finally, glucose consumption and lactate concentration were increased only by MET