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The adipose tissue plays an essential role in metabolism and physiology which impacts on animal lifespan and disease susceptibility. Dicer-1, a conserved type III endoribonuclease involved in miRNA processing, has been shown to be crucial in this organ for the adaptation to nutrient deprivation. However, mechanisms underlying Dcr-1 regulation in response to nutrient and metabolic challenges and the precise role of Dcr-1 in modulating metabolism, stress responses and aging remain unknown. Here we provide evidence that Dcr-1 plays a key role in the Drosophila fat body, a functional analog of vertebrate adipose and hepatic tissues, in the regulation of metabolism, stress resistance and longevity. We showed that Dcr-1 expression is tightly regulated in the fat body under different stress types and physiological conditions including starvation, oxidative stress and aging. Fat body specific depletion of Dcr-1 resulted in altered lipid metabolism and increased resistance to oxidative and nutritional stress, while a substantial extension in lifespan was observed in Dcr-1 heterozygous mutants. We also provide mechanistic evidence showing that the transcription factor FOXO regulates Dcr-1 expression upon nutrient deprivation. Under these conditions, JNK-dependent activation of FOXO in the fat body is required for the repression of Dcr-1 expression and miRNA biogenesis. Chromatin immunoprecipitation (ChIP) assays revealed that FOXO binds to a conserved DNA binding site in Dcr-1 promoter, thus directly repressing its transcription under starvation. The mechanism described here coupling FOXO activation in the adipose tissue to the repression of Dcr-1 implicates a novel and previously uncharacterized function for JNK-FOXO axis integrating nutrient status with miRNA biogenesis and physiological responses at the organismal level.

## CB-03

## CHARACTERIZATION AND BIOLOGICAL ACTION OF POLYETHYLENE GLYCOL-COATED MAGNETITE NANOPARTICLES IN A CELLULAR MODEL OF VIRAL ONCOGENESIS

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Magnetic nanotheranostics represent promising alternatives to the traditional diagnostic and treatment procedures available for different pathologies. The goal of this work was to analyze the biological action of polyethylene glycol-coated iron oxide nanoparticles (MAG.PEG) to generate a non-toxic carrier to optimize the delivery of drugs for Kaposi's sarcoma treatment. The MAG.PEG were synthesized by the hydrothermal method displaying a hydrodynamic diameter of 204 nm and a zeta potential of -22.1 mV. Firstly, MAG.PEG effects on cytotoxicity and cell viability were evaluated on endothelial cells expressing vGPCR, which prompts Kaposi's sarcoma. By Trypan blue technique, we found that the incubation of these cells neither with MAG nor MAG.PEG (1-150 µg/ml) provoked significant changes in the number of living cells. In addition, cell viability analyzes (MTS and neutral red) revealed that a significant increase in metabolic and lysosomal activity was detected at higher concentrations of MAG or MAG.PEG (100-150 µg/ml) after 48 h of incubation. Secondly, the localization and accumulation of MAG.PEG (1-150 µg/ml) towards the cells was observed directly under a phase contrast microscope. In addition, MAG.PEG assembled within or nearby the cells and cell morphology remained unchanged regardless of the nanoparticles concentration. Furthermore, the presence of vesicles containing MAG.PEG inside vGPCR cells was confirmed by transmission electron microscopy. Thirdly, the iron content quantified by Prussian blue staining showed that the degree of accumulation of MAG.PEG depends on the concentration used. Finally, to steer the MAG.PEG to certain localization in the cell culture, a magnetic field generated by a moderated power magnet was used. The results indicated that the magnetic stimuli induced MAG.PEG accumulation in the zone where the magnet was placed. In conclusion, concentrations between 1 and 50 µg/ml of MAG.PEG would be suitable as drug carriers in this cellular model since no