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U.PORTO

Recently, we have shown the ability of the polyphenol curcumin to induce cellular antioxidant defenses through induction of a stress response in normal human skin fibroblasts, affording protection from a further oxidant challenge with tert-BOOH [1]. Curcumin incubation for 24h induced heme oxygenase-1 (HO-1) protein levels, GST activity, GSH levels and GSH/GSSG ratio. These effects were preceded by induction of oxidative stress as shown by increased levels of ROS and DNA damage, and impairment of the cells' GSH redox state. The induction of antioxidant defenses in human fibroblasts was shown to be redox and PI3K/Akt dependent [1]. In conclusion, these results support the view that phytochemical-induced hormetic stimulation of cellular antioxidant defenses can be a useful approach toward anti-aging intervention.

[1] Lima CF, Pereira-Wilson C, Rattan SIS (2011). Mol. Nutr. Food Res., 55: 430-42.

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P3 Cellular distribution and regulation of intestinal SGLT1

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Diabetes is achieving epidemic proportions in many countries. In addiction to high blood glucose it is associated with increased intestinal expression of the sodium-glucose cotransporter (SGLT1). This transporter is located in brush-border membrane (BBM) of the enterocytes and is responsible for transporting glucose and galactose from the intestinal lumen into the cytosol, using the inward Na^+ gradient maintained by the basolateral Na^+/K^+-ATPase. Our previous results show that the adaptive response to increase dietary carbohydrates also involves increased intestinal expression of SGLT1 at the BBM. This raise does not seem to reflect changes in mRNA suggesting an involvement of posttranscriptional mechanisms in SGLT1 BBM expression. In Caco-2 cells, the intracellular SGLT1 resides in endosomes and the abundance of the transporter at BBM seems to be affected by the cellular endocytic pathway. Currently, we are focusing our studies on the regulation by glucose, insulin and other dietary factors on the cellular distribution of SGLT1 and the mechanisms of its traffick to the plasma membrane in Caco-2 cells.

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P4 Autophagy triggered by ursolic acid synergistically enhances 5-fluorouracil induced cell death in HCT15 (MSI p53 mutant) colorectal cancer cells

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Colorectal carcinoma (CRC) is a common cause of cancer-related death and tumors with microsatellite instability (MSI) and p53 mutations have been shown to be resistant to chemotherapy with 5-fluorouracil (5-FU). Therefore, it is essential to find compounds that could contribute to treatment efficacy by increasing the sensitivity to 5-FU. HCT15, a MSI human CRC derived cell line that harbours a p53 mutation, was incubated with the triterpenoid ursolic acid (UA) at a concentration that induces approximately 50% cell death (measured by PI stainning) after 48h. A synergistic enhancement of apoptosis was observed when co-incubating 5-FU with UA (measured by TUNEL assay). UA induction of apoptosis was totally abolished by the JNK inhibitor SP600125 (SP), but not by the caspase inhibitor zVAD-fmk. Apoptosis did not account

for all the observed cell death induced by UA. Thus, we asked whether UA was also inducing autophagy. We observed that UA induced accumulation of autophagossomes (using fluorescent dyes) as well as of LC3-II (assessed by western blot), which was also significantly inhibited by SP. These results suggest that UA induction of apoptosis and autophagy is JNK dependent. A decrease in mutated p53 and phospho mTOR, which are associated with an induction of autophagy, were also observed. In conclusion, UA showed anticancer activity by inducing apoptosis and autophagy, which was JNK-dependent in HCT15 cells. In addition, in these resistant cells, UA synergistically cooperate with 5-FU to induce cell death

P5 DNA DAMAGE PREVENTION AND SIGNALING PATHWAY REGULATION BY SAGE IN A COLON CANCER MODEL

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Colorectal cancer (CRC) is a common malignancy and significant cause of mortality in Western societies. It develops through an accumulation of genetic and epigenetic alterations, transforming normal colon cells and giving them growth advantage. Many food plants are rich in bioactive compounds and have shown to posses anticancer properties.
We proposed to explore the effects of sage (*Salvia officinalis* (SO)) water extract (herbal tea) drinking on colon cancer prevention and modulation of epigenetic events. F344 rats were used to study the effects of sage tea drinking on pre-initiation (SO treatment before AOM exposure) and post-initiation (SO after AOM exposure) phases of carcinogenesis. We found a chemopreventive effect of SO in the pre-initiation group, but not in the post-initiation. We then investigated if SO affected AOM metabolism, searching for effects on CYP2E1 expression and activity. We found that AOM decreased CYP2E1 activity when compared with control, but SO treatment before AOM prevented this effect. The capacity of SO in vivo treatment to protect colonocytes from H_2O_2 damage induced in vitro was also investigated. SO decreased significantly the oxidative H_2O_2-induced DNA damage. We also are searching for alterations in expression of key proteins involved in signalling pathways important for cell proliferation or apoptosis and proteins involved in DNA repair.
Sage water extract seems to have the ability to prevent CRC and studies to further explore this potential are ongoing.

P6 PREPARATION OF *JATROPHA CURCAS* OIL AS FEEDSTOCK FOR BIODIESEL PRODUCTION

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Jatropha curcas plant can grow in arid soils and produce high non edible oil yields. *Jatropha curcas* oil is considered as one of the most important feedstock for biodiesel production. Preparation of this oil must meet the specification of feedstock that could ensure the highest quality of biodiesel. Adjustment of Free Fatty Acid (FFA) content is one of the main steps before the transesterification process using base as