energy by uncoupling of respiration from ATP synthesis. The physiological function of UCP2 is still unknown. Despite its high homology with UCP1, UCP2 does not seem to have a role in adaptive thermogenesis while mounting evidence implicates UCP2 in the protection against oxidative stress. UCP2 is expressed in several mammalian tissues including white adipose tissue. Leptin, a hormone involved in the control of energy balance, increases lipolysis and fatty acid oxidation in white adipose tissue and simultaneously up-regulates UCP2. The aim of the present study is to investigate whether in white adipose tissue UCP2 serves as an energy dissipatory mechanism which facilitates fatty acid oxidation or prevents oxidative damage. Metformin (dimethylbiguanidine) is a drug widely used to treat type 2 non-insulin dependent diabetes mellitus. We show that metformin raises the UCP2 levels in white adipose tissue of mice and in 3T3-L1 adipocytes. Up-regulation of UCP2 correlates with a higher superoxide dismutase activity, lower aconitase and high levels of reactive oxygen species while lipolysis is not yet induced. These evidence strongly suggest that UCP2 is not directly involved in fatty acid metabolism and reinforces their role in the defence against oxidative stress.

doi:10.1016/j.bbabio.2008.05.197

S8.10 Brain mitochondrial bioenergetics is differentially affected by anesthetics fentanil and remifentanil

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Mitochondria have been proposed to be implicated in cellular effects of anesthetics. The purpose of our study was to investigate effects of fentanil and remifentanil on the bioenergetics of isolated rat brain mitochondria. Mitochondria were isolated and respiratory rates, respiratory control ratio (RCR), ADP/O ratio, mitochondrial membrane potential ($\Delta\Psi_m$) and mitochondrial respiratory complex activities (II, III, IV and V) were determined. Increased concentrations of fentanil and remifentanil resulted in compromised respiratory mitochondrial function in a dose-dependent decrease in RCR and uncoupling of oxidative phosphorylation evidenced by the decreasing ADP/O values. The $\Delta\Psi$ generated by respiration and mitochondrial complexes II, III, IV and V activities significantly decreased with concentration of fentanil ranging from 4 to 20 μg/mL. Similar effects were observed at higher concentrations of remifentanil (10 and/or 20 μg/mL). In conclusion our study demonstrated that fentanil and remifentanil exerted significant and differential effects on the mitochondrial bioenergetics: remifentanil slightly affects the mitochondrial bioenergetic functions in contrast with the deleterious effects of fentanil.

doi:10.1016/j.bbabio.2008.05.198

S8.11 Photobiomodulation of flavonoid effects on human T cells mitochondrial network state

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The goal of studies was to reveal flavonoid induced changes in human acute T leukemic Jurkat cells mitochondrial reticulum size and state, as well as soft laser irradiation influence thereon. Apoptosis induction and cell cycle progression of flavonoid treated, irradiated and/or non-irradiated T leukemia lymphoblasts, were additionally monitored. Analysis of JC-1 red/green fluorescence intensity ratios, PI, and of MitoTracker Green signals, collected by flow cytometry and/or by confocal microscopy allowed us to determine mitochondrial membrane potentials, mitochondrial membrane depolarization/hyperpolarization related apoptosis induction/cell cycle blockade/progression, and mitochondrial network weight. Natural flavonoids epigallocatechine gallate and quercetin induced changes in sizes and polarization degrees of cell subpopulations of high and low mitochondrial membrane polarization, and in the mitochondrial reticulum volume, in a dose and exposure-time dependent manner. Low concentrations of flavonoids caused no or mild effects, reversible in time, while alterations of mitochondrial reticulum size and state, induced by high concentrations of flavonoids, were correlated with apoptosis induction and cell cycle blockade. 680 nm far-red and 830 nm infrared laser lights promoted/reversed the flavonoid induced changes in a dose, wavelength and irradiation regime dependent manner.

Partial financial support of the Romanian Ministry of Education and Research (grants CNCSIS 924/2006 and CEEX 74/2006) is gratefully acknowledged.

doi:10.1016/j.bbabio.2008.05.199

S8.12 Opposite effects of action potential on spatial patterns of photosynthesis and extracellular pH in a plant cell

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Characean cells exposed to illumination arrange their plasmamembrane H^+ fluxes and photosynthesis in coordinated spatial patterns that facilitate the acquisition of inorganic carbon from the aquatic environment. The limited availability of CO_2 in alkaline bands accounts for lower effective quantum yield of photosystem II ($\Delta F/Fm'$) in chloroplasts of these bands compared to acidic zones. In this work we studied the effect of electrically triggered action potential (AP) on the spatial distribution of photosynthetic parameters ($\Delta F/Fm'$ and non-photochemical quenching, NPQ) and extracellular pH with fluorescence imaging and pH microsensors. In the resting cell at a range of light intensities, the periodic profile of extracellular pH is parallel to the profile of NPQ and antiparallel to that of $\Delta F/Fm'$. The principal discovery of this study is that, after triggering AP, the pH banding disappeared temporarily, whereas the effective quantum yield and NPQ patterns became more contrast.