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.

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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.

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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.

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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.

These observations add to the list of AP-induced phenomena in plants, thus emphasizing the significance of AP as a multifunctional signal. We discuss the transient changes in pH-banding, effective quantum yield and non-photochemical quenching in relation to alterations in intracellular Ca^{2+} and H^+ concentrations during and after AP.

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S8.13 Effect of α-overexpression of F_1F_o-ATP synthase on iron-overloaded heart

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The aim of our study was to examine the effect of overexpression of α subunit of F_1F_o-ATP synthase on iron uptake of mitochondria and cardiac myocytes under iron overload. We did a transfection of α subunit gene on the primary cardiac myocytes of iron-overloaded rats. In α-overexpressed cells, mitochondrial ROS was reduced (4.14± 0.23 vs. 1.77±0.03 μM/mg; non- vs. overexpressed) and free iron as well, which was reduced to 40% (0.98±0.04 vs. 0.39±0.05 μmol/mg). The mitochondrial ATPase activity increased 2-fold (0.27±0.07 vs. 0.54± 0.05 mM/mg/min) and mitochondrial ATP was lowered to 29% (1.27± 0.10 vs. 0.37±0.03 $^{E-07}$ mol/mg) in α-overexpressed cells compared to control. In α-overexpressed cells of iron-overloaded heart, mitochondrial LIP was increased (2.65±0.42 vs. 3.67± 0.45 μmol/mg). Mitochondrial ATP was slightly reduced (0.35±0.10 vs. 0.25±0.03 $^{E-07}$ mol/mg) although mitochondrial ROS (7.39±0.23 vs. 16.69 ± 0.54) and ATPase activity (0.49±0.10 vs. 1.06±0.01) were expanded over 2-fold. Mitochondrial membrane potential was significantly augmented (100±15 vs. 270±39% by rhodamine 123 staining). We found declined cellular LIP level (13.7±0.36 vs. 7.43± 0.82 μmol/mg) but ugmented ATP amount over 2.4-fold (0.30±0.50 vs. 0.70±0.03 $^{E-07}$ mol/mg). Importantly, we observed that iron-overloaded cells have higher viability with α-overexpression than without. And, signals of apoptotic cell death were considerably declined in the presence of iron. Based on these data, we suggest that overexpressed α subunit contributes to the viability in iron-overloaded heart.

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S8.14 Low power long wavelength laser irradiation effects on human mononuclear cell mitochondrial membrane potential

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The objective of this study was to demonstrate therapeutic soft laser light effects on the mitochondrial membrane electrical potential in human peripheral blood mononuclear cell subpopulations in various culture and irradiation conditions. Furthermore we observed microenvironment dependent cross-talk between separately irradiated adherent and non-adherent mononuclear cells grown in co-culture. Quantitative analysis of JC-1 red/green fluorescence signals, gathered on surface antigen labeled single cells by flow cytometry, allowed us to disclose changes in both the relative sizes of adherent/non-adherent cell subpopulations with preponderently highly/weakly polarized mitochondrial membranes, and in the average mitochondrial membrane potentials of these subpopulations. The changes induced in the mitochondrial membrane state by the 680 nm far-red and 830 nm infrared laser lights were single and total dose, wavelength, irradiation regime, and cell-state dependent. Metabolic modulation of laser effects was evident. As a rule energy/nutrient restricted cells with altered mitochondrial membranes were more sensitive to soft laser irradiation than the non-injured controls. Irradiation of adherent cells caused more substantial changes in the mitochondrial membrane state. Cross-talk between irradiated and non-irradiated cells in co-culture was evident in the presence of growth factors.

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S8.15 Liver metabolic fluxes in response to high fat diet

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Aim: Determining adaptations of metabolic fluxes in liver with high fat diet (HF).
Methods: Glucose, lactate and pyruvate production by perifused hepatocytes isolated from rats fed either a standard or HF diet.
Results: HF increased gluconeogenesis from glycerol in the presence of octanoate (+30%) but decreased it in the absence of fatty acid. This effect was associated with an increase of glycerol metabolism without effect on glycolysis. In both conditions, cytosol was more oxidized whereas mitochondrial compartment was more reduced. Cellular and mitochondrial oxidative capacities were reduced by HF (−40%). Glycerol metabolism requires a stoichiometric utilization of ATP and NAD^+. Therefore depending on the redox condition, control of the pathway is either on the dehydrogenase step, (at low rate of glycerol metabolism), or on the phosphorylation step (high rate of metabolism). Hence, the lower glycerol metabolism observed with HF at high flux is due to diminished oxidative phosphorylation capacity and to an inability to maintain ATP. By contrast, when flux through the pathway is reduced by high redox pressure of fatty acid metabolism, the ability of HF animals to maintain an oxidized cytosolic compartment allows then to metabolize more glycerol. This feature probably results from a higher rate of NADH oxidation via the mitochondrial glycerophosphate dehydrogenase which could be an adaptation to HF for compensating the decrease in oxidative phosphorylation capacity.

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S8.16 Mitochondrial adjustment to energy demand when cell growth slows down

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During cellular proliferation on non-fermentable carbon source, mitochondrial activity must meet energy demand. Previous work in