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