importance of the GTPase domain and behavior during apoptosis. Using immunofluorescence, we found that endogenous Mfn2 is enhanced in specific regions on the mitochondria, particularly at tips of mitochondria. Overexpressed mitofusin also forms distinct foci and we have found using FRAP (Fluorescence Recovery After Photobleaching) that mutants of mitofusins devoid of GTP binding display markedly different mobilities on the mitochondrial membrane reflecting changes in complex formation. During apoptosis the mitochondria fragment and fusion is inhibited. Interestingly, during apoptosis a mutant of Mfn2 that typically is incapable of forming foci moves into foci that coalesce with Bax, the pro-apoptotic Bcl-2 family member. Curiously, we have found that upon Bax translocation the mitofusins, along with Fis1 and OMP-25, lose mobility, indicating that the mitochondria outer membrane is undergoing additional changes not previously described.

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S9.9 MiD51 and MiD49: New mediators of mammalian mitochondrial distribution

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Mitochondria are organized into networks that are important for proper cellular function. These networks are regulated by fission and fusion events, as well as transport along cytoskeletal elements. The aim of this study was to characterize two novel mitochondrial proteins, *Mi*tochondrial *D*istribution protein 51 kDa (MiD51) and 49 kDa (MiD49), that appear to be involved in the regulation of mitochondrial morphology and distribution. Ectopic expression of both MiD51 and MiD49 in COS-7 cells results in two aberrant mitochondrial phenotypes — long extended tubules and peri-nuclear collapsed mitochondria. Live cell confocal microscopy was used to highlight the dynamic nature and connectivity of the mutant mitochondria by co-expression with the photoswitchable fluorescent protein mitochondrial-Dendra2. While mitochondrial movement in mammalian cells is principally driven by a connection with the microtubule network, these MiD proteins instead appear to induce an interaction between mitochondria and actin filaments. Only simultaneous knockdown of both endogenous proteins via RNAi, results in atypical mitochondrial distribution in both COS-7 and HeLa cells, as well as reduced HeLa cell viability. We therefore propose that MiD51 and MiD49 share functional redundancy in the distribution of mitochondria in mammalian cells, perhaps in regulation of actin/mitochondrial interactions.

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S9.10 Cell type specificity of mitochondrial dynamics: Striking differences between adult rat cardiomyocytes, HL-1 cells and human pancreatic cells

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The aim of this study was to analyze mitochondrial dynamics in adult rat cardiomyocytes, HL-1 cells and human pancreatic cells. Mitochondrial imaging was performed by real time confocal microscopy using mitochondria-specific fluorescent probes. The results revealed remarkable differences in mitochondrial dynamics, as well as in spatial arrangement of mitochondria in these cells, probably due to cell specific cytoskeleton organization. In adult rat cardiomyocytes, in which mitochondria are arranged regularly (crystal-like), no displacement of mitochondria was observed with only very small amplitude rapid vibration. In contrast, in primary human pancreatic and HL-1 cells we documented complex dynamic behaviour of mitochondria. The common types of mitochondrial dynamics observed were: 1) fission, fusion and small oscillatory movements of mitochondria; 2) larger movements including filament extension, retraction, and 3) fast oscillating branching in the mitochondrial network and fast long-distance intracellular translocation of single mitochondria or mitochondrial filaments. In summary, we show that mitochondrial dynamics may be very different in different cell types. These variations could be related to a significant role of cell specific integrations of mitochondria with other intracellular systems like cytoskeleton and ER.

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S9.11 Novel mechanism of elimination of malfunctioning mitochondria (mitoptosis): Formation of mitoptotic bodies and extrusion of mitochondrial material from the cell

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Energy catastrophe when mitochondria hydrolyze glycolytic ATP instead of producing it has been modeled. In highly glycolyzing HeLa cells 30–50% of the population survived after inhibition of respiration and uncoupling of oxidative phosphorylation for 2–4 days. The survival was accompanied by selective elimination of mitochondria. This program of mitoptosis includes (i) fission of mitochondrial filaments, (ii) clusterization of mitochondria in perinuclear area, (iii) occlusion of mitochondrial clusters by a membrane (formation of a "mitoptotic body"), (iv) decomposition of mitochondria to small vesicles, (v) protrusion of the body from the cell and (vi) disruption of the body boundary membrane. Autophagy was not involved in mitoptosis. Increased production of reactive oxygen species (ROS) was necessary for execution of the program, since antioxidants prevent mitoptosis and kill the cells treated with the mitochondrial poisons. Mitoptosis served for protection of the cells under the conditions of severe damage of mitochondria. It is suggested that exocytosis of mitoptotic bodies may be involved in maturation of reticulocytes and precursors of lens fiber cells.

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(S10) Mitochondria and reactive oxygen containing species symposium lecture abstracts

S10/1 Interactions of nitric oxide with cytochrome *c* and cytochrome *c* oxidase

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At low nM concentration nitric oxide (NO) is an intercellular messenger, interacting with the heme protein guanylate cyclase.