Dynamic regulation of mitochondrial network and oxidative functions during 3T3-L1 fat cell differentiation

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Résumé en anglais	Mitochondria have been shown to be impaired in insulin resistance-related diseases but have not been extensively studied during the first steps of adipose cell development. This study was designed to determine the sequence of changes of the mitochondrial network and function during the first days of adipogenesis. 3T3-L1 preadipocytes were differentiated into adipocytes without using glitazone compounds. At days 0, 3, 6, 9, and 12, mitochondrial network imaging, mitochondrial oxygen consumption, membrane potential, and oxidative phosphorylation efficiency were assessed in permeabilized cells. Gene and protein expressions related to fatty acid metabolism and mitochondrial network were also determined. Compared to preadipocytes (day 0), new adipocytes (days 6 and 9) displayed profound changes of their mitochondrial network that underwent fragmentation and redistribution around lipid droplets. Drp1 and mitofusin 2 displayed a progressive increase in their gene expression and protein content during the first 9 days of differentiation. In parallel with the mitochondrial network redistribution, mitochondria switched to uncoupled respiration with a tendency towards decreased membrane potential, with no variation of mtTFA and NRF1 gene expression. The expression of PGC1α and NRF2 genes and genes involved in lipid oxidation (UCP2, CD36, and CPT1) was increased. Reactive oxygen species (ROS) production displayed a nadir at day 6 with a concomitant increase in antioxidant enzyme gene expression. This 3T3-L1-based in vitro model of adipogenesis showed that mitochondria adapted to the increased number of lipid droplets by network redistribution and uncoupling respiration. The timing and regulation of lipid oxidation-associated ROS production appeared to play an important role in these changes.
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