Mitochondrial effects of dexamethasone imply both membrane and cytosolic-initiated pathways in HepG2 cells

Titre	Mitochondrial effects of dexamethasone imply both membrane and cytosolic-initiated pathways in HepG2 cells
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Résumé en anglais	Glucocorticoid treatment is often linked to increased whole-body energy expenditure and hypermetabolism. Glucocorticoids affect mitochondrial energy production, notably in the liver, where they lead to mitochondrial uncoupling reducing the efficacy of oxidative phosphorylation. However, the signaling pathways involved in these phenomena are poorly understood. Here we treated HepG2 cells with dexamethasone for different times and, by using different combinations of inhibitors, we showed that dexamethasone treatment leads to recruitment of two main signaling pathways. The first one involves a G-protein coupled membrane glucocorticoid binding site and rapidly decreases complexes I and II activities while complex III activity is upregulated in a p38MAPK dependent mechanism. The second one implies the classical cytosolic glucocorticoid receptor and triggers long-term transcriptional increases of respiration rates and of complex IV activity and quantity. We concluded that mitochondria are the target of multiple dexamethasone-induced regulatory pathways that are set up gradually after the beginning of hormone exposure and that durably influence mitochondrial oxidative phosphorylation.
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