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**Uncoupling of mitochondria activates protein phosphatases and inactivates MBP protein kinases.**

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Dephosphorylation of PHF-tau was observed in carbonyl cyanide p-(trifluoromethoxy) phenylhydrazone (FCCP)-treated, but not in oligomycin-treated undifferentiated PC12 cells. FCCP depletes ATP levels by uncoupling oxidative phosphorylation and increases cytosolic calcium levels, while oligomycin inhibits the ATP synthase. We also observed inactivation of several myelin basic protein (MBP) kinases in FCCP-treated PC12 cells, using an in-gel kinase assay. In addition, several phosphotyrosine proteins were dephosphorylated following FCCP-treatment. These studies suggest that MBP kinases and tyrosine phosphatase may be regulated by mitochondrial activity and they may regulate the phosphorylation state of tau. Since mitochondrial dysfunction occurs in Alzheimer disease, such changes in protein phosphorylation may well be relevant to the disease.

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