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Mitochondrial Genome Acquisition Restores Respiratory Function and Tumorigenic Potential of Cancer Cells without Mitochondrial DNA

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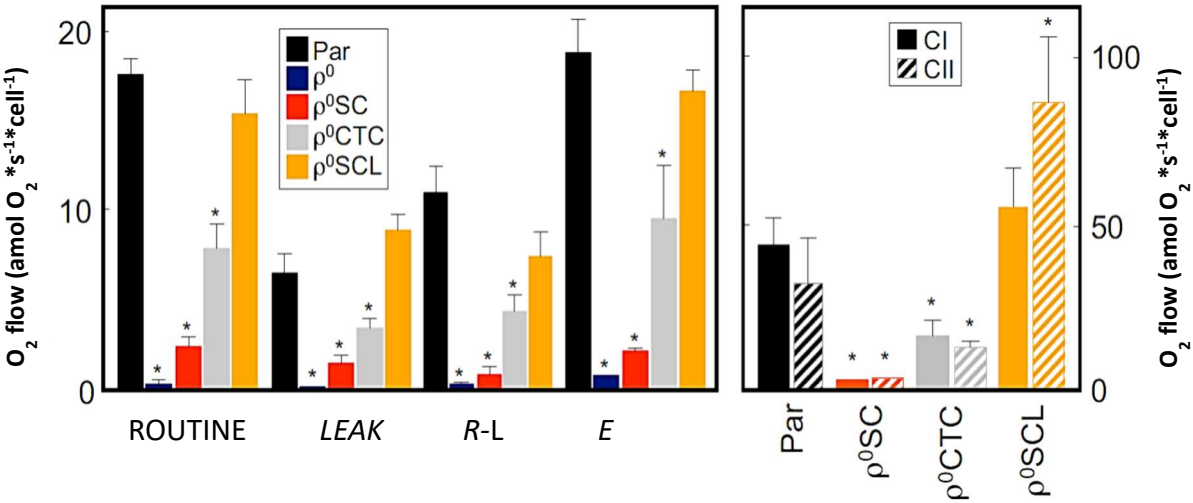
Mitochondrial respiration is recovered in models of grafted carcinoma cells with depleted mtDNA after mitochondrial transfer from the host



Bioenergetic properties of 4T1ρ⁰ sublines. (Left) 4T1 cells and derived cell lines (4T1 ρ⁰ carcinoma cells with sever mitochondrial genome damage, 4T1 ρ⁰SC primary tumours, 4T1 ρ⁰CTC circulating tumour cells, and 4T1 ρ⁰SCL lung metastases) were assessed for ROUTINE (*R*), LEAK (*L*), ROUTINE coupling efficiency (*R*–*L*), and noncoupled electron transfer (*E*) respiration using an Oroboros O2k-Respirometer. **(Right)** Cells were permeabilized with digitonin and evaluated for respiration at the presence of substrates specific for the NADH electron transfer-pathway state (CI) and the succinate pathway control state (CII). Data are from three independent experiments (mean ± SD and * $p<0.05$).

Reference: Tan AS, Baty JW, Dong L, Bezawork-Geleta A, Endaya B, Goodwin J, Bajzikova M, Kovarova J, Peterka M, Yan B, Pesdar EA, Sobol M, Filimonenko A, Stuart S, Vondrusova M, Kluckova K, Sachaphibulkij K, Rohlena J, Hozak P, Truksa J, Eccles D, Haupt LM, Griffiths LR, Neuzil J, Berridge MV (2015) Mitochondrial genome acquisition restores respiratory function and tumorigenic potential of cancer cells without mitochondrial DNA. Cell Metab 21:81-94.

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