2k brief Cancer
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High-resolution respirometry: Cancer

Oncogenic *HSP60* regulates mitochondrial oxidative phosphorylation to support Erk1/2 activation during pancreatic cancer cell growth

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Knockdown of *HSP60* decreases mitochondrial function in pancreatic ductal adenocarcinoma (PDAC) cells

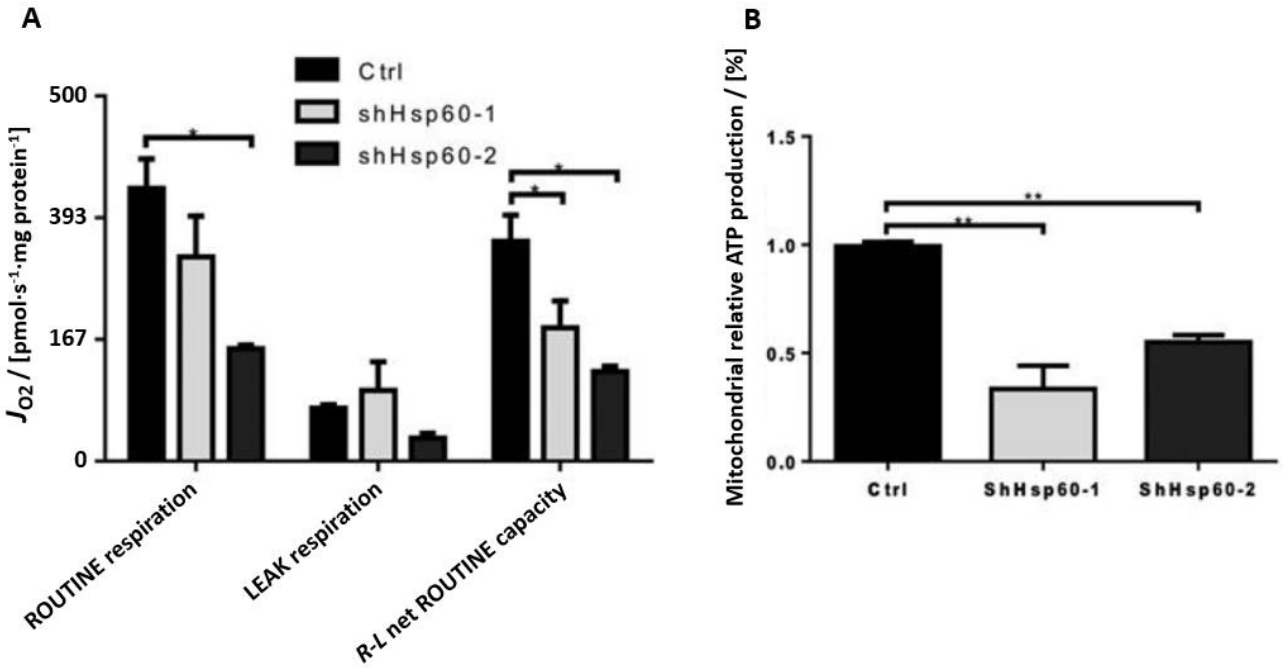

Figure 1. (A) Analysis of the oxygen flux (J_{O2}) per mass of various cell lines comparing shHSP60 Panc-1 cells with control (Ctrl) cells. Oxygen consumption was first measured for each cell line in ROUTINE followed by sequential addition of oligomycin (100 µg·mL^{-1}) to to assess LEAK respiration. *R-L* net ROUTINE capacity was calculated by subtracting LEAK from ROUTINE respiration. **(B)** Measurements of mitochondrial ATP levels. Cells were incubated with 10 mM glucose or 5 mM 2-DG plus 5 mM pyruvate to determine mitochondrial ATP generation. Relative average ATP levels in mitochondria per cell line are shown. All data are presented as mean ± SEM ($N \geq 3$). *$P < 0.05$, and **$P < 0.01$.

O2k-brief communicated by C Cecatto and L Tindle-Solomon
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HSP60 associated with the phosphorylation of Erk1/2

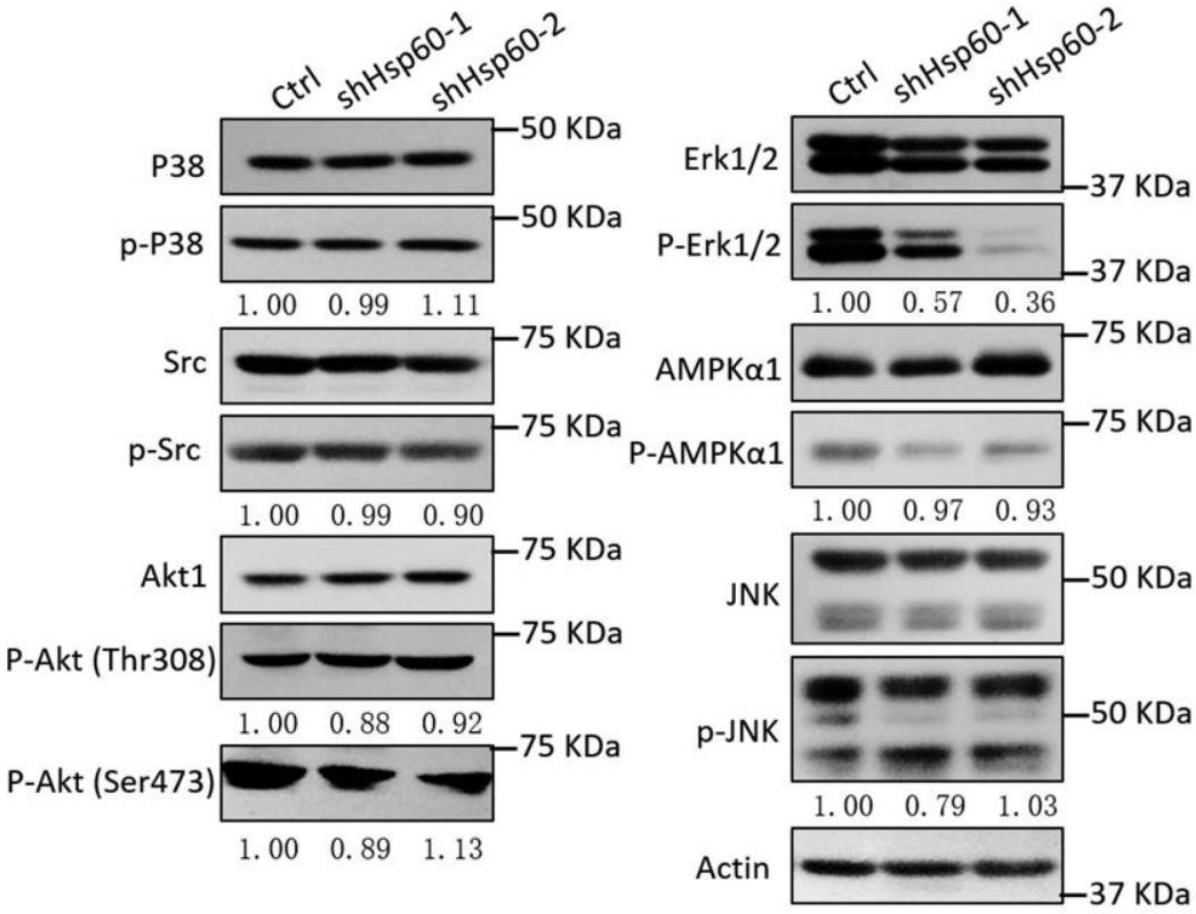

Figure 2. Analysis of mitochondrial-to-nucleus pathways in shHSP60 Panc-1 cells and control (Ctrl) cells. Cell lysates were analyzed by western blot with antibodies against P38, p-P38, Src, p-Src, Akt1, p-Akt (Thr308), p-Akt (Ser473), Erk1/2, p-Erk1/2, AMPKα1, p- AMPKα1, JNK, p-JNK, and actin.

Knockdown of *HSP60* decreased pancreatic ductal adenocarcinoma (PDAC) cell respiration, ATP production and Erk1/2 phosphorylation, leading to decreases in proliferation, invasion, and clonal formation of PDAC cells. These results highlight the role of mitochondria in pancreatic cancer cell growth.

Reference: Zhou C, Sun H, Zheng C, Gao J, Fu Q, Hu N, Shao X, Zhou Y, Xiong J, Nie K, Zhou H, Shen L, Fang H, Lyu J (2018) Oncogenic HSP60 regulates mitochondrial oxidative phosphorylation to support Erk1/2 activation during pancreatic cancer cell growth. Cell Death Dis 9:161.

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