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Cell-permeable succinate prodrugs bypass mitochondrial complex I deficiency

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Respiration in platelets, PBMCs and human heart muscle fibres

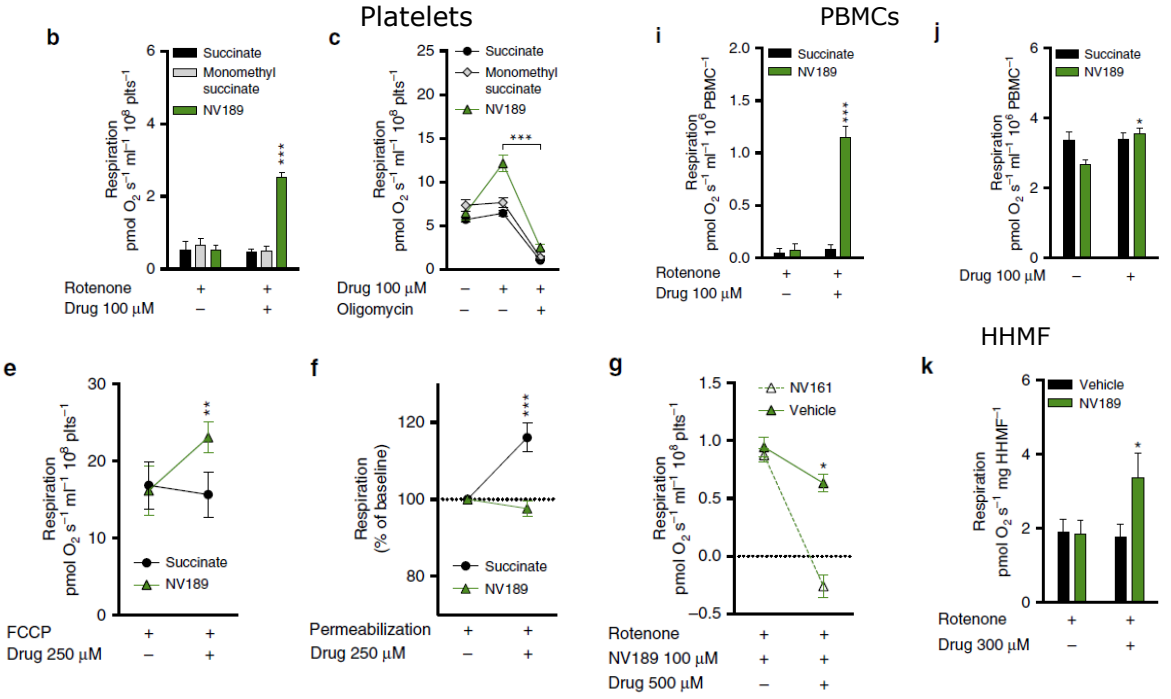

Figure 1. (b) Rotenone-induced mitochondrial complex I (CI) inhibition. **(c)** ATP-generating respiration. **(e)** FCCP-induced uncoupling. **(f)** Digitonin-permeabilized platelets. **(g)** Effect on respiration in platelets with addition of the cell-permeable complex II inhibitor NV161. **(i)** Respiration in peripheral blood mononuclear cells (PBMCs) with rotenone-induced CI inhibition. **(j)** Convergent respiration in PBMCs **(k)** Respiration in human heart muscle fibres (HHMFs).

Prodrug-delivered succinate can alleviate metabolic decompensation due to CI-related mitochondrial dysfunction

Reference: Ehinger JK, Piel S, Ford R, Karlsson M, Sjövall F, Frostner EÅ, Morota S, Taylor RW, Turnbull DM, Cornell C, Moss SJ, Metzsch C, Hansson MJ, Fliri H, Elmér E (2016) Cell-permeable succinate prodrugs bypass mitochondrial complex I deficiency. Nat Commun 7:12317

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