Supplementary Materials

VDAC1 Knockout Affects Mitochondrial Oxygen Consumption Triggering a Rearrangement of ETC by Impacting on Complex I Activity

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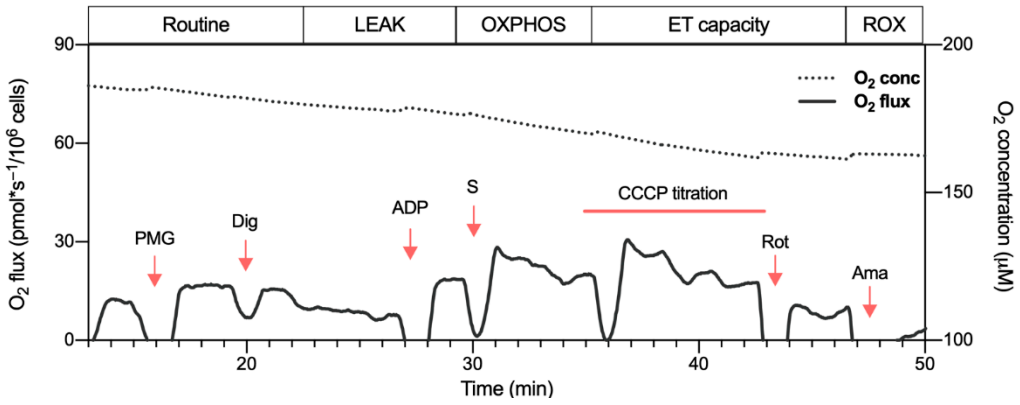

Figure S1. Oxygen consumption in HAP1 VDAC1 knock-out cells. A representative curve of mitochondrial respiratory profile of HAP1 ΔVDAC1 cells along with the SUIT protocol used in this work. P, pyruvate; M, malate; G, glutamate; Dig, digitonin; S, succinate; Rot, rotenone; Ama, antimycin.

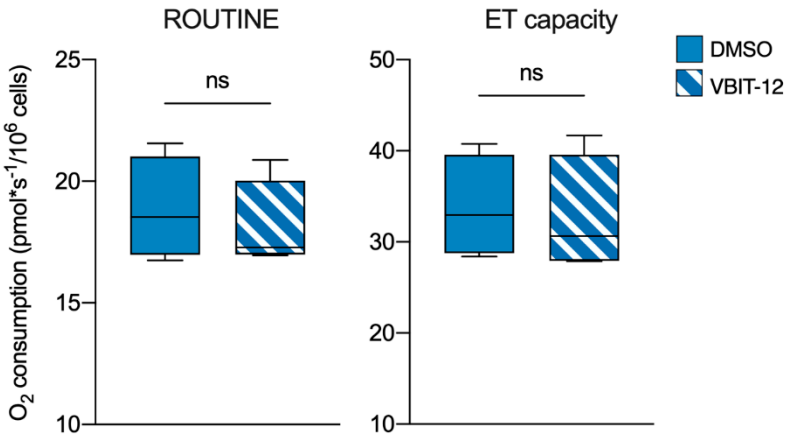

Figure S2. Analysis of VBIT-12 effect on respiration of ΔVDAC1 cells. Quantitative analysis of the oxygen consumption rates of ROUTINE and maximal ET capacity in HAP1 ΔVDAC1 cells previously treated with VBIT-12 or DMSO (control). No significant variation has been observed for both ROUTINE or ET capacity. Data are expressed as pmol/second per million cells and shown as median ± SEM of n=6 independent experiments.

Table S1. Raw data of the nicotinamide dinucleotides expressed as nmol/million cells. Data are relative to the quantification of each nicotinamide dinucleotide in HAP1 parental and VDAC1 knockout cells. Data are expressed as means ± SD of n=4 independent measurements.

	NAD^+	**NADH**	**$NADP^+$**	**NADPH**
Parental	1.48 ± 0.64	0.18 ± 0.04	0.42 ± 0.19	0.11 ± 0.04
ΔVDAC1	0.87 ± 0.44	0.09 ± 0.41	0.22 ± 0.06	0.14 ± 0.04