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Mitochondrial respiratory control and early defects of oxidative phosphorylation in the failing human heart

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Human heart: OXPHOS analysis by high-resolution respirometry

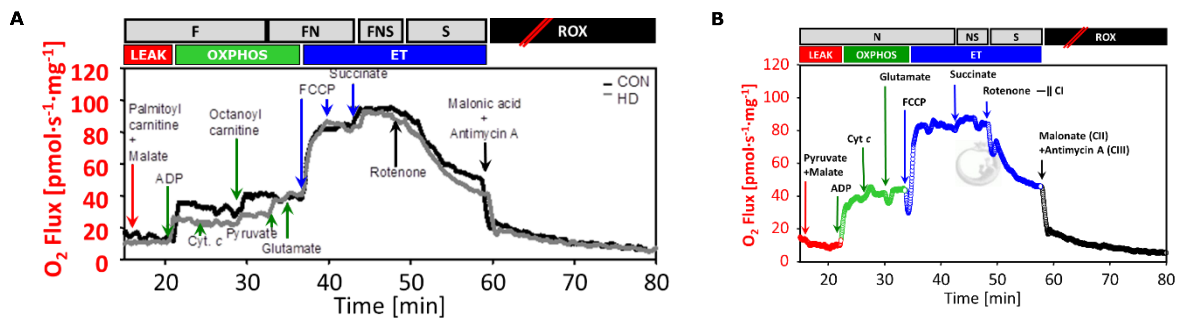


Figure 1. SUIIT protocols for evaluation of mitochondrial respiration capacities and OXPHOS control in permeabilized myocardial fibers. A. Fatty acid SUIIT protocol for testing the flavoprotein and Complex I pathway. **B.** Carbohydrates SUIIT protocol to test the additivity at the Q-junction. Both graphs are representative traces obtained by high-resolution respirometry.

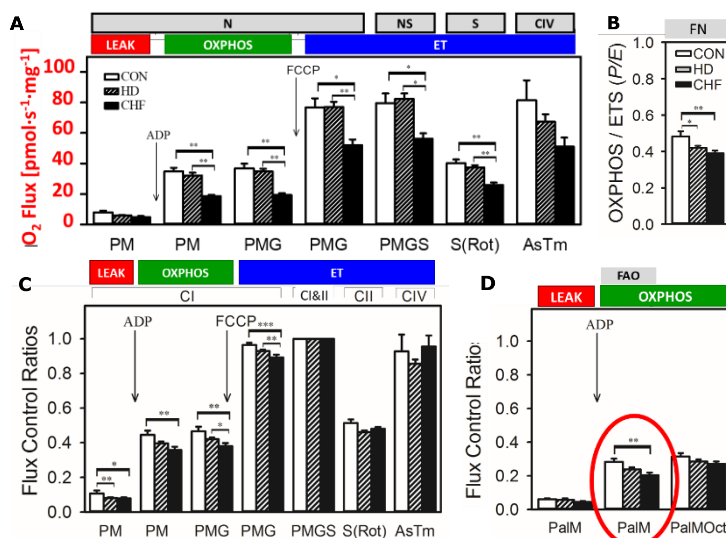


Figure 2. OXPHOS capacity, phosphorylation system, and OXPHOS and ET capacities for N- or FN-pathway. A. Carbohydrates SUIIT protocol to test the additivity at the Q-junction. **B.** Coupling Control Ratios with substrates for the FN-pathway. **C.** Flux Control ratios in protocols with carbohydrates. **D.** or fatty acids. $N=30-40$ depending on the experimental group. Data are means \pm SE.

Heart disease and chronic heart failure leads towards a general loss of OXPHOS capacity (mt-density), defects on the phosphorylation system, decrease in the FAO capacity and a reduction in the NADH-OXPHOS and ET capacities

Reference: Lemieux H, Semsroth S, Antretter H, Höfer D, Gnaiger E (2011) Mitochondrial respiratory control and early defects of oxidative phosphorylation in the failing human heart. *Int J Biochem Cell Biol* 43:1729–38.

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