Impaired mitochondrial activity explains platelet dysfunction in thrombocytopenic cancer patients undergoing chemotherapy

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OXPHOS respiration decreased for the NADH- and succinate pathway control states in platelets from thrombocytopenic cancer patients undergoing chemotherapy

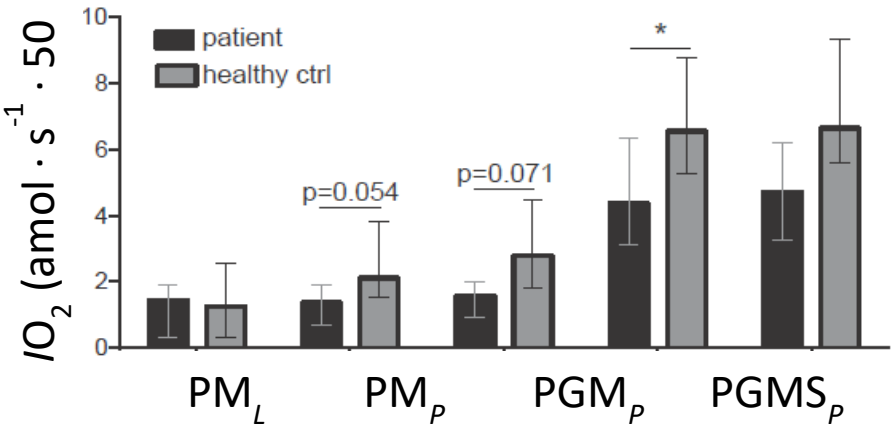

Figure 1. Mitochondrial respiration in platelets by high-resolution respirometry. A simplified version of the SUIT-008 protocol was used to assess the mitochondrial function of permeabilized platelets using pyruvate and malate (LEAK, PM_L), saturating concentrations of ADP (OXPHOS, PM_P), glutamate (OXPHOS, PGM_P), succinate (OXPHOS, $PGMS_P$) and testing for the integrity of the mitochondrial outer membrane with cytochrome *c* (OXPHOS, $PGMSc_P$). Medians with IQR are shown for samples obtained from patients (*N*=7) and controls (*N*=9).

Platelets from patients undergoing chemotherapy shows a significant reduction of the mitochondrial membrane potential and the oxidative phosphorylation capacity

Reference: Aragonés J, Schneider M, Van Geyte K, Fraisl P, Dresselaers T, Mazzone M, Dirkx R, Zacchigna S, Lemieux H, Jeoung NH, Lambrechts D, Bishop T, Lafuste P, Diez-Juan A, K Harten S, Van Noten P, De Bock K, Willam C, Tjwa M, Grosfeld A, Navet R, Moons L, Vandendriessche T, Deroose C, Wijeyekoon B, Nuyts J, Jordan B, Silasi-Mansat R, Lupu F, Dewerchin M, Pugh C, Salmon P, Mortelmans L, Gallez B, Gorus F, Buyse J, Sluse F, Harris RA, Gnaiger E, Hespel P, Van Hecke P, Schuit F, Van Veldhoven P, Ratcliffe P, Baes M, Maxwell P, Carmeliet P (2008) Deficiency or inhibition of oxygen sensor Phd1 induces hypoxia tolerance by reprogramming basal metabolism. Nat Genet 40:170-80.

Figures and texts slightly modified based on the recommendations of the COST Action MitoEAGLE CA15203. Doi:10.26124/mitofit:190001.v3