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High-resolution respirometry: Exercise

Acute RyR1 Ca^{2+} leak enhances NADH-linked mitochondrial respiratory capacity

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Effect of simulated sprint interval training on mitochondrial respiratory capacity compared to simulated moderate intensity continuous training

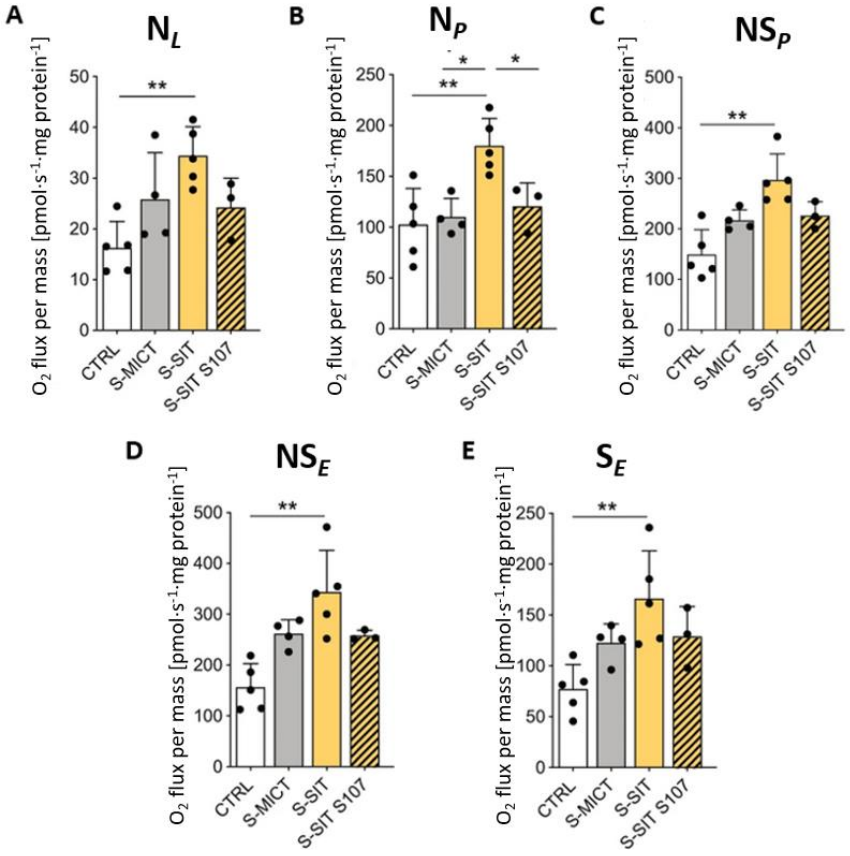

Figure 1. Simulated sprint interval training (S-SIT) in C2C12 myotubes induces higher mitochondrial respiratory capacity as compared to simulated moderate intensity continuous training (S-MICT), which are blunted by S107-induced ryanodine receptor protein stabilization (S-SIT S107). O_2 flux per mass (pmol·s^{-1}·mg protein^{-1}) 72 h after stimulation and S107 treatment. **(A)** N_L: NADH-linked LEAK respiration with malate and pyruvate. **(B)** N_P: NADH-linked OXPHOS capacity stimulated with ADP. **(C)** NS_P: N- and S-OXPHOS capacity. **(D)** NS_E: electron transfer ET capacity stimulated by the uncoupler FCCP, noncoupled and **(E)** S_E, Succinate-ET capacity; $n = 5$ (CTRL, S-SIT), 4 (S-MICT) and 3 (S-SIT S107) independent biological experiments.

S-SIT myotubes showed greater mitochondrial respiration than control and S-MICT myotubes. The RyR1-stablizer S107 specifically blunted NADH-linked respiration in S-SIT myotubes suggesting that the positive mitochondrial adaptations towards a more aerobic phenotype in response to S-SIT are driven, at least in part, by acute sarcoplasmic reticulum Ca^{2+} leak through RyR1/calstabin1 dissociation.

Reference: Zanou N, Dridi H, Reiken S, Imamura de Lima T, Donnelly C, De Marchi U, Ferrini M, Vidal J, Sittenfeld L, Feige JN, Garcia-Roves PM, Lopez-Mejia IC, Marks AR, Auwerx J, Kayser B, Place N (2021) Acute RyR1 Ca2+ leak enhances NADH-linked mitochondrial respiratory capacity. Nat Commun 12:7219.

Text slightly modified based on the recommendations of the COST Action MitoEAGLE CA15203. doi:10.26124/bec:2020-0001.v1

O2k-brief communicated by T Komlodi and L Tindle-Solomon
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