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Author Correction: Enhanced mitochondrial oxidative metabolism in peripheral blood mononuclear cells is associated with fatty liver in obese young adults

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Correction to: *Scientific Reports* <https://doi.org/10.1038/s41598-023-32549-w>, published online 30 March 2023

The original version of this Article contained errors in Figure 1 and its accompanying legend, where the complex II and its surroundings were incorrect. The original Figure 1 and accompanying legend appear below.

The original Article has been corrected.

Published online: 21 March 2024

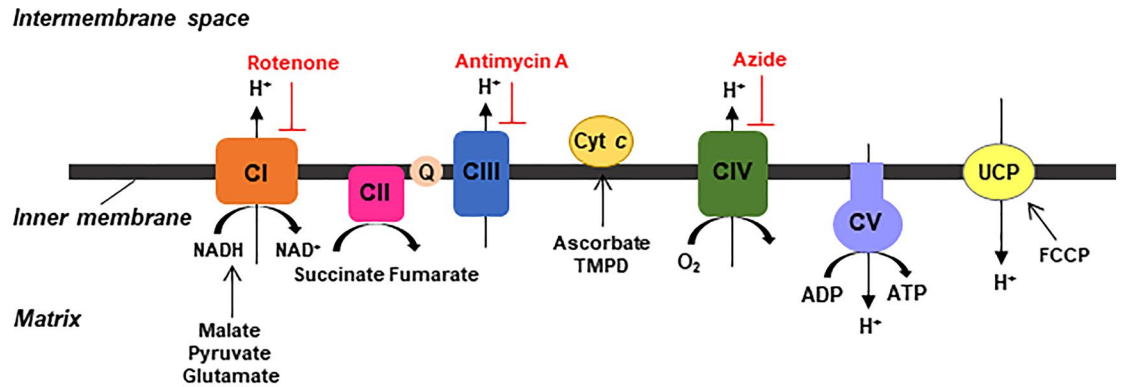
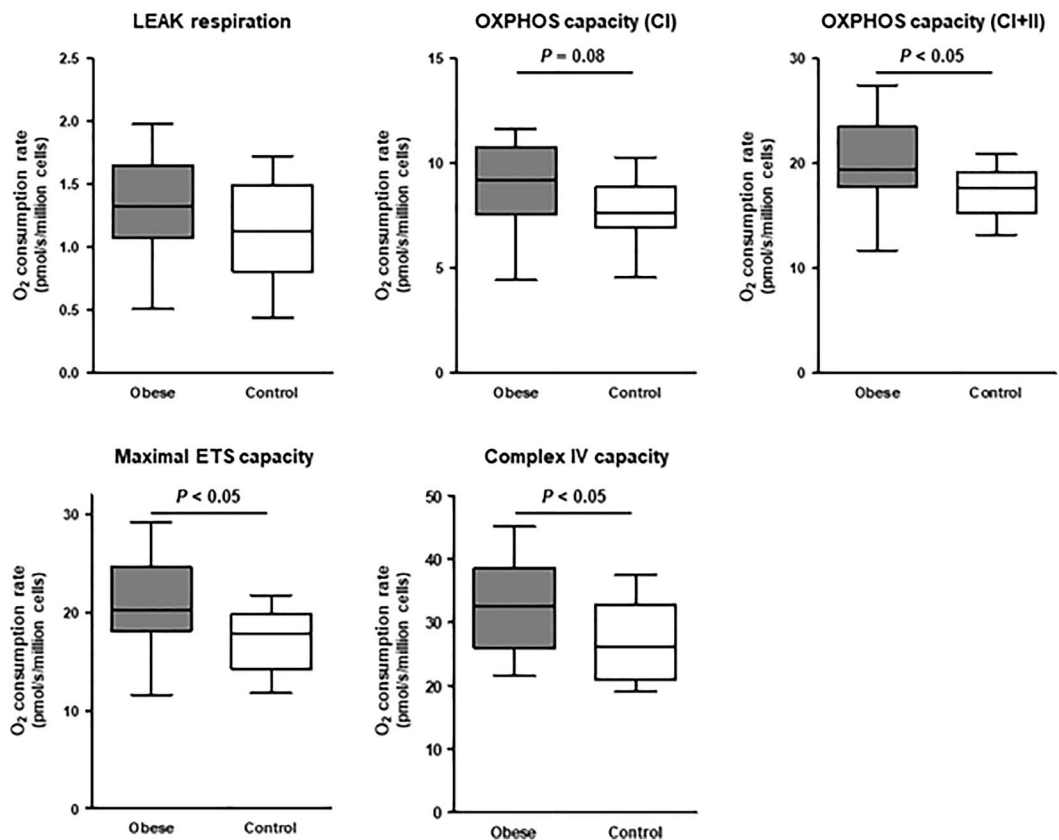
A**B**

Figure 1. Mitochondrial respiratory capacity in PBMCs. **(A)** Scheme of the mitochondrial electron transfer system (ETS) with the SUIT (substrate–uncoupler–inhibitor–titration) protocol that we used for evaluation of the mitochondrial respiratory capacity in the present study. **(B)** Summarized data of the O₂ consumption rate during each respiratory state with different respiratory substrates in the permeabilized PBMCs of obese ($n = 14$) and healthy control subjects ($n = 15$). The box bounds the interquartile range (IQR) divided by the median, and Tukey-style whiskers extend to a maximum of $1.5 \times$ IQR beyond the box. The LEAK respiration indicates non-ADP stimulated respiration (i.e., state 2 respiration) with CI-linked substrates. The oxidative phosphorylation (OXPHOS) capacity, an ADP-stimulated respiration (i.e., state 3 respiration), was measured in the presence of CI- or CI+II-linked substrates. The maximal ETS capacity was measured after addition of FCCP, an uncoupler, in the presence of CI+II-linked substrates. The capacity of complex IV was measured after addition of TMPD, an electron donor to cytochrome *c* (cyt *c*), in the presence of ascorbate. CI complex I, CII complex II, CIII complex III, CIV complex IV, CV complex V, UCP uncoupling protein.



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