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THE EFFECT OF DISUSE ON MITOCHONDRIAL RESPIRATION RATE IN HUMAN M. SOLEUS

Abstract

One week of disuse has a negative effect on the functional characteristics of human skeletal muscles, including oxidative enzymes activity, mitochondrial density and function, though, the dynamics of these changes is unclear. Dry Immersion (DI) and head-down tilt bed rest (HDBR) are the disuse models that most accurately reproduce the main physiological effects of weightlessness on human body due to profound decrease in motor activity and support unloading.

The aim of the present study was to compare changes in the maximum mitochondrial respiration rate in m. soleus after 3- and 6-day DI, as well as after 20-day HDBR.

Six healthy women (25-39 years old) and ten healthy men (25-38 years old) were exposed to 3- and 6-day DI, respectively. Six healthy men (24-40 years old) spent 20 days in head-down tilt bed rest. Before and after DI and HDBR exposure, tissue samples were taken from m. soleus to assess the maximum mitochondrial respiration rate in permeabilized muscle fibers using high resolution respirometry (polarograph Oxygraph, Hansatech).

Three-day DI decreased maximal ADP-stimulated respiration rate via complex I (by 30%, $P < 0.05$) and complex I+II (by 25%, $P < 0.05$) and the proton leak (by 25%, $P < 0.05$). An increase in exposure duration – a 6-day DI – led to a comparable decrease in these indexes, as well as to impairment of the electron transport chain capacity induced by FCCP uncoupler (by 30%, $P < 0.01$). In comparison with a 6-day DI, 20-day HDBR induced a slightly greater decrease in maximal ADP-stimulated respiration rate via complex I and complex I+II, and the proton leak ($\sim 40\%$, $P < 0.05$), and a similar decrease in the electron transport chain capacity (by 30%, $P < 0.05$).

In conclusion, a rapid decrease in mitochondrial functions (respiration rate) was observed even after three days of DI in m. soleus. An increase in exposure duration up to six days does not exacerbate the reduction of mitochondrial functions, thus, their loss is slowing down. This was confirmed by data showing that 20-day HDBR exposure induces only a slight additional decrease in mitochondrial functions.

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