Is there a systemic mitochondrial biogenic response to aerobic training?
- Mitochondrial respiration in platelets from athletes forced to rest following a knee injury

Eleonor Åsander Frostner[1], K Steding[2], H Arheden[2], MJ Hansson[1,2]

1. Mitochondrial Pathophysiology Unit, Lund University, Lund, Sweden; 2. Department of Clinical Physiology, Skåne University Hospital & Lund University, Lund, Sweden - eleonor.asander@med.lu.se

Endurance exercise on a regular basis induces skeletal and cardiac muscle performance adaptation, lower mean arterial blood pressure and metabolic adaptation in a number of organs [1,2]. The latter has been shown to involve mitochondrial biogenesis. Upon injury when training intensity decreases, as well as in aging, these events tend to reverse [2,3]. The aim of the present study was to investigate whether the level of aerobic performance affects mitochondrial respiration in platelets.

Six male and female athletes were subjected to magnetic resonance imaging (MRI) of the heart and blood sampling within three days following an anterior cruciate ligament (ACL) injury. An initial follow-up was performed at the start of rehabilitation training and a late follow up at eight months following injury. The latter exams also included a maximal incremental exercise test with gas analysis. Platelets were isolated by centrifugation and mitochondrial respiration was analyzed using a substrate-uncoupler-inhibitor-protocol.

The total heart volume (THV) was significantly lower following the period of reduced exercise intensity from the time of injury to initial follow-up (p = 0.042, n = 6). There was no significant difference in THV between initial and late follow-up. The maximal VO_2 uptake showed a trend toward increase from initial to late follow-up (p = 0.086, n = 4). There were, however, no significant differences or any discernable trends in respiratory parameters between the time points studied.

In conclusion, there was no difference in platelet mitochondrial respiration in response to alterations in exercise level in this small pilot study.

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