Title: **High-intensity exercise in hypoxia studies shows that training intensity promotes increase in mitochondrial content**

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Each side of this cross-talk [1,2] properly defended their views. Another way to shed light on the importance of training intensity or volume may come from studies on exercise in hypoxia through the interdependence of environmental oxygen availability and mitochondrial oxidative phosphorylation.

Hypoxic exercise can elicit specific transcriptional responses [through the upregulation of hypoxia-inducible factor 1] leading to skeletal muscle adaptations[3]. Interestingly, high-intensity training in hypoxia induces higher increase in total mitochondrial volume density and capillary length density than low-intensity training in hypoxia [4,5]. It has been then postulated that exercise intensity in hypoxia *per se* modulates muscle molecular mechanisms of oxygen homeostasis [6]. In partial agreement, performing maximal-intensity exercise training in hypoxia (*i.e.*, repeated-sprint training in hypoxia, RSH) *versus* normoxia upregulates genes involved in oxygen signaling, oxygen carrying and pH regulation but not in mitochondrial biogenesis in moderately-trained male cyclists [7]. In team-sport athletes, however, superimposing RSH to 14-d of hypoxic residence (200 h) elicits higher oxygen signaling and oxygen carrying as well as mitochondrial biogenesis and mitochondrial metabolism compared with similar normoxic intervention [8]. Altogether, these recent studies on high-intensity hypoxic exercise tend to support that *'training intensity is of primary importance for increasing human skeletal muscle mitochondrial content'*.

Competing interests

None declared.

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