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Free Radic Biol Med. 2015 Sep;86:37-46. doi: 10.1016/j.freeradbiomed.2015.04.006. Epub 2015 Apr 15.

# Redox modulation of mitochondriogenesis in exercise. Does antioxidant supplementation blunt the benefits of exercise training?

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## Abstract

Physical exercise increases the cellular production of reactive oxygen species (ROS) in muscle, liver, and other organs. This is unlikely due to increased mitochondrial production but rather to extramitochondrial sources such as NADPH oxidase or xanthine oxidase. We have reported a xanthine oxidase-mediated increase in ROS production in many experimental models from isolated cells to humans. Originally, ROS were considered as detrimental and thus as a likely cause of cell damage associated with exhaustion. In the past decade, evidence showing that ROS act as signals has been gathered and thus the idea that antioxidant supplementation in exercise is always recommendable has proved incorrect. In fact, we proposed that exercise itself can be considered as an antioxidant because training increases the expression of classical antioxidant enzymes such as superoxide dismutase and glutathione peroxidase and, in general, lowering the endogenous antioxidant enzymes by administration of antioxidant supplements may not be a good strategy when training. Antioxidant enzymes are not the only ones to be activated by training. Mitochondriogenesis is an important process activated in exercise. Many redox-sensitive enzymes are involved in this process. Important signaling molecules like MAP kinases, NF-κB, PGC-1α, p53, heat shock factor, and others modulate muscle adaptation to exercise. Interventions aimed at modifying the production of ROS in exercise must be performed with care as they may be detrimental in that they may lower useful adaptations to exercise.

**KEYWORDS:** PGC-1α; Redox signaling; Skeletal muscle; Vitamins

PMID: 25889822 DOI: 10.1016/j.freeradbiomed.2015.04.006

[Indexed for MEDLINE]

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