



Correction to: Molecular regulation of skeletal muscle mitochondrial biogenesis following blood flow-restricted aerobic exercise: a call to action

Nicholas Preobrazenski¹ · Hashim Islam² · Brendon J. Gurd³

Published online: 20 May 2021
© Springer-Verlag GmbH Germany, part of Springer Nature 2021

Correction to: European Journal of Applied Physiology
<https://doi.org/10.1007/s00421-021-04669-6>

The original version of this article unfortunately contained a mistake. There is an error in Fig. 1.

The correct version of Fig. 1 is given in the following page.

The original article has been corrected.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

The original article can be found online at <https://doi.org/10.1007/s00421-021-04669-6>.

✉ Brendon J. Gurd
gurdb@queensu.ca

¹ Faculty of Medicine, University of Ottawa, Ottawa, ON K1H 8M5, Canada

² School of Health and Exercise Sciences, University of British Columbia, Kelowna, BC V1V 1V7, Canada

³ School of Kinesiology and Health Studies, Queen's University, Kingston, ON K7L 3N6, Canada

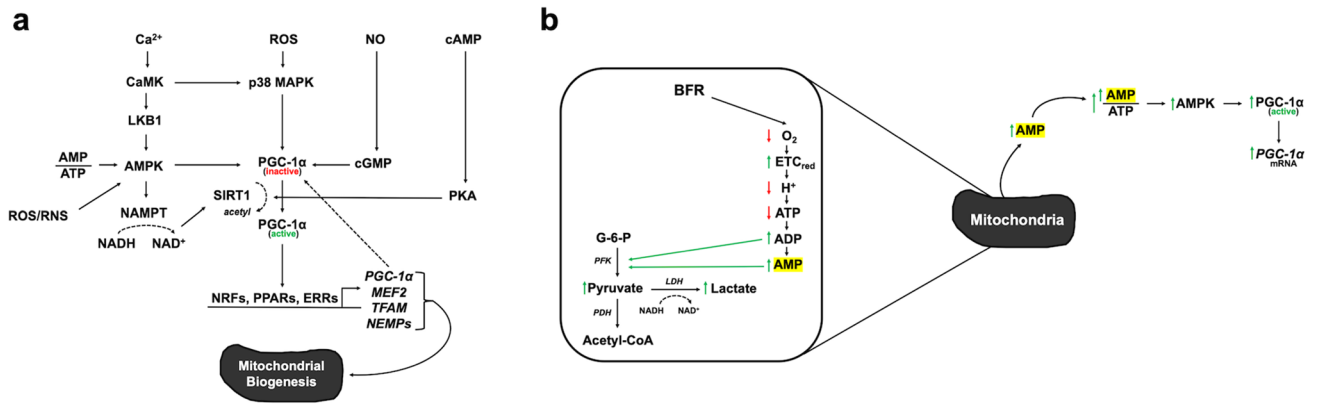


Fig. 1 a Regulation of peroxisome proliferator-activated gamma coactivator 1-alpha (PGC-1α). *Straight dashed arrow* on the right side of the figure represents the autoregulation of PGC-1α. **b** Speculated effects of BFR-induced ischemia on peroxisome proliferator-activated gamma coactivator 1-alpha (PGC-1α) expression via increased [AMP]:[ATP] ratio. Decreased local oxygen availability from reduced arterial blood flow leads to increased reduction of the electron transport chain (ETC). As a result, less H⁺ is pumped to the intermembrane space of the mitochondria, leading to a smaller electrochemical gradient across the ETC. ATP synthase thus phosphorylates less ADP, increasing [AMP] and [ADP]. Increased [ADP] and [AMP] stimulates the rate-limiting enzyme of glycolysis, phosphofructokinase-1 (PFK), and more pyruvate is produced. Given that the capacity for glycolytic flux is greater than the capacity for pyruvate dehydrogenase to oxidise pyruvate to acetyl-CoA (Spriet et al. 2000), the law of mass action dictates more pyruvate reducing to lactate.

Importantly, increased [AMP] also activates AMPK, which subsequently phosphorylates and activates PGC-1α. AMPK 5' AMP-activated protein kinase, ATP adenosine triphosphate, β -AR beta-2 adrenergic receptor, Ca²⁺ calcium ions, CaMKII calmodulin-dependent protein kinase II, cAMP cyclic adenosine monophosphate, cGMP cyclic guanosine monophosphate, Epi epinephrine, ERR oestrogen-related receptors, LDH lactate dehydrogenase, LKB1 liver kinase B1, MEF2 myocyte enhancer factor-2, NADH/NAD⁺ nicotinamide adenine dinucleotide, NAMPT nicotinamide phosphoribosyltransferase, NE norepinephrine, NEMP nuclear-encoded mitochondrial proteins, NO nitric oxide, NRF nuclear respiratory factors, p38 MAPK p38 mitogen-activated protein kinase, PDH pyruvate dehydrogenase, PFK phosphofructokinase, PKA protein kinase A, PPAR peroxisome proliferator-activated receptor, RNS reactive nitrogen species, ROS reactive oxygen species, SIRT1 silent mating-type information regulation 2 homolog 1, TFAM mitochondrial transcription factor A