Redox Biology Of Exercise

By: Michalis G. Nikolaidis, Chad M. Kerksick, Manfred Lamprecht, and **Steven R. McAnulty**

Abstract

Redox biology is probably the most rapidly expanding field in biology. Indeed, the number of conferences, journals, and books devoted to redox biology is increasing and it is very often seen that major biology journals publish special issues on this area (e.g., [1–5]). This fact is probably due to the disclosure of the diverse roles reactive species have been found to serve, such as the control of the signaling of intracellular pathways [6], the mediation of enzyme activation [7], and the participation in antibiotic synthesis [8]. The significance of reactive species has been further underlined by the emerging links between cellular redox events and the etiology of many human diseases [9]. As a result of this progress in basic redox biology, the subfield of exercise redox biology has also markedly advanced.

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Editorial

Redox Biology of Exercise

Michalis G. Nikolaidis,[1] Chad M. Kerksick,[2] Manfred Lamprecht,[3] and Steven R. McAnulty[4]

[1] *Department of Physical Education and Sports Science at Serres, Aristotle University of Thessaloniki, 62110 Serres, Greece*
[2] *Department of Health, Exercise and Sport Sciences, The University of New Mexico, Albuquerque, NM 87131, USA*
[3] *Centre for Physiological Medicine, Medical University of Graz, Harrachgasse 21/II, 8010 Graz, Austria*
[4] *Department of Health, Leisure, and Exercise Science, Appalachian State University, Boone, NC 28608, USA*

Correspondence should be addressed to Michalis G. Nikolaidis, nikolaidis@auth.gr

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Redox biology is probably the most rapidly expanding field in biology. Indeed, the number of conferences, journals, and books devoted to redox biology is increasing and it is very often seen that major biology journals publish special issues on this area (e.g., [1–5]). This fact is probably due to the disclosure of the diverse roles reactive species have been found to serve, such as the control of the signaling of intracellular pathways [6], the mediation of enzyme activation [7], and the participation in antibiotic synthesis [8]. The significance of reactive species has been further underlined by the emerging links between cellular redox events and the etiology of many human diseases [9]. As a result of this progress in basic redox biology, the subfield of exercise redox biology has also markedly advanced.

Exercise is perhaps one of the most characteristic examples demonstrating that reactive species are not necessarily "harmful" entities, considering that the well-known benefits of regular exercise on muscle function and health are accompanied by repeated episodes of oxidative and nitrosative stress. In addition, an ongoing debate exists in the literature regarding the implications of antioxidant supplementation on physical performance and redox homeostasis. Considering that the redox biology of exercise is by nature multidisciplinary, this special issue is compiled of original and review articles combining chemical, analytical, biochemical, nutritional, physiological, and medical aspects relevant to reactive species biology. Reading through these papers the multiple facets of exercise redox biology are revealed.

The review article by E. C. Gomes et al. presents the current state of knowledge on the redox biology of exercise. It provides a comprehensive perspective on the contribution of various intracellular and extracellular sources and the identity of oxidants produced by exercising animals and humans. It also focuses on the possible role of these exercise-induced oxidants in important training adaptations such as angiogenesis, mitochondria biogenesis, and muscle hypertrophy. This article lays the groundwork for the other articles of the special issue that address oxidant effects on exercise performance and redox homeostasis and diseases. Specifically, H. Pan et al. indicated that electrical stimulation of skeletal muscle cells increased the production of reactive species as well as the mRNA and protein levels of interleukin-6. The authors hypothesized that reactive species generation induced by skeletal muscle contraction may be one of the factors regulating muscle-derived interleukin-6 production and release. Using a more physiological relevant methodology, S. Mrakic-Sposta et al. employed an electron paramagnetic resonance technique for the rapid and noninvasive measurement of reactive species concentration directly in fresh human peripheral blood. Using this innovative approach, they reported that short-term high-intensity exercise increased reactive species production whereas the resting levels of reactive species decreased following supplementation with the antioxidant cofactor α-lipoic acid.

Three papers investigated whether alterations in redox homeostasis can be monitored to assess the health and fitness of the intensively training athlete. T. K. Tong et al. evaluated the impact of professional training on serum oxidant and antioxidant status in adolescent endurance runners and cyclists and compared it with that of untrained individuals. The authors reported that the resting blood redox homeostasis was well maintained in the adolescent athletes apparently due to the increase of antioxidants as a result of adaptations to chronic exercise. Similarly, C. A. Williams and A. O. Burk demonstrated that a three-day training event increased markers of antioxidant status in horses as a potential response to increased generation of reactive species during exercise. Finally, R. L. P. Ferraresso et al. using an innovative rat model showed that overtraining was associated with increased antioxidant enzyme activities and increased lipid peroxidation in blood and muscle. These data imply that monitoring of redox homeostasis in elite athletes may serve as a tool for overtraining diagnosis.

Six papers dealt with the effect of antioxidant supplementation on redox homeostasis and performance employing in vitro, in situ, in vivo, and even a combination of in vitro and in vivo approaches. R. J. Bloomer et al. reported that supplementation with coenzyme Q10 (an electron carrier in the electron transport chain) for four weeks affected neither exercise performance nor blood redox homeostasis in humans. On the other hand, A. E. Wagner et al. showed that combined supplementation of skeletal muscle cells with α-lipoic acid plus coenzyme Q10 improved energy homeostasis, stress response, and antioxidant defense mechanisms. Unless the additional supplementation of α-lipoic acid was responsible for these effects, the apparent contradiction between the two studies indicates that the potential antioxidant function of coenzyme Q10 in vivo cannot be safely extrapolated from in vitro tests. This may be due to the metabolic transformations and interactions that clearly affect the bioavailability and biological action of coenzyme Q10. To this end, A. S. Veskoukis et al. examined whether a polyphenol-rich grape pomace extract possesses in vitro antioxidant properties and whether the in vitro properties of the extract translate to an in vivo model when the extract was administered before exhaustive exercise to rats. The authors found that the polyphenol-rich extract possessed in vitro antioxidant activity which was not translated to in vivo antioxidant activity either at rest or after exercise (in fact, even some prooxidant effects were noted in vivo). In the light of these findings, it was suggested that the term “antioxidant” may be system related. Along the two poles of the in vitro-in vivo continuum, the study by A. Kyparos et al. employed an in situ model to investigate whether vitamin E can attenuate eccentric exercise-induced skeletal muscle injury. The authors found that vitamin E protected the soleus muscle from injury as indicated by the decreased fatigability at low-frequency stimulation and the almost complete recovery of single-twitch force immediately after fatigue. In an in vivo study in horses, E. D. Lamprecht and C. A. Williams reported that oral superoxide dismutase supplementation (encapsulated in a gliadin biopolymer to protect the enzyme against gastric proteolysis) did not affect the exercise-induced disturbances in redox homeostasis. Based on these studies, it is evident that antioxidant supplementation has discrepant effects on performance and redox homeostasis. This was also the major conclusion of the review article by M. G. Nikolaidis et al. regarding the effect of vitamin C and/or E supplementation on training and redox adaptations. Indeed, the relevant studies provided conflicting outcomes regarding the efficacy of vitamin C and E supplementation, mostly due to methodological differences in assessing redox status and training adaptations.

Lastly, two review articles analyzed the evidence of whether regular exercise can be used as a tool to combat two common and related lifestyle disease states: Type II Diabetes Mellitus and Metabolic Syndrome. Based on detailed analysis, E. T. de Lemos et al. supported that there are pathophysiological pathways that are associated with oxidative stress and inflammation in the development of Type II Diabetes Mellitus. The authors also asserted that regular exercise may act as a natural antioxidant and anti-inflammatory agent to prevent the serious complications of Type II Diabetes Mellitus. The Metabolic Syndrome is a clustering of obesity, diabetes, hyperlipidemia, and hypertension that affects roughly 20% of the population in Western industrialized countries. S. Golbidi et al. reviewed the relevant data and concluded that oxidative stress and the consequent inflammation induce insulin resistance (as supported by E. T. de Lemos et al. as well), which likely links the various components of the Metabolic Syndrome.

We hope that this compilation of research and review articles will stimulate further efforts to understand the biological importance and mechanisms of redox processes during exercise. Redox biology is at the heart of life sciences. This is because electron flow may be one of the most universal and fundamental approaches to biology [10]. Consequently, we believe that the field of exercise redox biology will be one of the key topics that will drive the exercise science in the future.

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Michalis G. Nikolaidis
Chad M. Kerksick
Manfred Lamprecht
Steven R. McAnulty

References

[1] R. Banerjee and W. Smith, “Thematic minireview series on redox sensing and regulation,” *The Journal of Biological Chemistry*, vol. 287, no. 7, pp. 4395–4396, 2012.

[2] R. Bottinelli and H. Westerblad, “Reactive oxygen and nitrogen species in skeletal muscle: acute and long-term effects,” *Journal of Physiology*, vol. 589, no. 9, pp. 2117–2118, 2011.

[3] P. C. Dorrestein and K. S. Carroll, “’Omics’ of natural products and redox biology,” *Current Opinion in Chemical Biology*, vol. 15, no. 1, pp. 3–4, 2011.

[4] C. Horst Lillig and C. Berndt, "Preface to the special issue on redox control of cell function," *Biochimica et Biophysica Acta*, vol. 1780, no. 11, p. 1169, 2008.
[5] M. B. Reid, "Editorial," *Journal of Applied Physiology*, vol. 102, no. 4, pp. 1299–1300, 2007.
[6] H. J. Forman, M. Maiorino, and F. Ursini, "Signaling functions of reactive oxygen species," *Biochemistry*, vol. 49, no. 5, pp. 835–842, 2010.
[7] J. Stubbe and W. A. Van Der Donk, "Protein radicals in enzyme catalysis. [Chemical Reviews 1998, 98, 705–762]," *Chemical Reviews*, vol. 7, no. 2, pp. 2661–2662, 1998.
[8] W. Lesniak, V. L. Pecoraro, and J. Schacht, "Ternary complexes of gentamicin with iron and lipid catalyze formation of reactive oxygen species," *Chemical Research in Toxicology*, vol. 18, no. 2, pp. 357–364, 2005.
[9] M. Valko, D. Leibfritz, J. Moncol, M. T. D. Cronin, M. Mazur, and J. Telser, "Free radicals and antioxidants in normal physiological functions and human disease," *International Journal of Biochemistry and Cell Biology*, vol. 39, no. 1, pp. 44–84, 2007.
[10] J. Herrmann and D. Tobias, "Redox biology on the rise," *Biological Chemistry*, vol. 393, no. 9, pp. 999–1004, 2012.