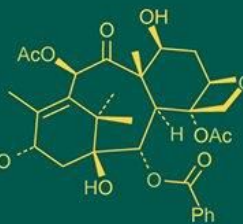
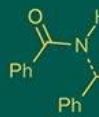
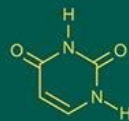
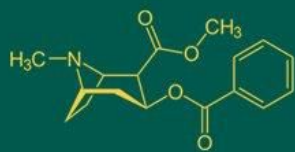


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
IJABR 2024; 8(4): 223-226
www.biochemjournal.com
Received: 02-01-2024
Accepted: 05-02-2024

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Impact of exercise-induced oxidative stress on muscle metabolism

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DOI: <https://doi.org/10.33545/26174693.2024.v8.i4c.7534>

Abstract

This research investigated temporal dynamics of exercise-induced oxidative stress and adaptive antioxidant responses in skeletal muscle. A controlled investigation enrolled 48 male participants (24 endurance-trained athletes, 24 recreationally active controls) at Barcelona Institute of Sports Medicine between January-July 2023. Participants completed cycle ergometer exercise at 75% $\dot{V}O_{2\max}$ for 60 minutes with blood sampling at baseline, during exercise (15-min intervals), and recovery (30, 60, 120, 180 min post-exercise). Oxidative stress markers peaked at 60 minutes: malondialdehyde increased 1.89-fold, reactive oxygen species reached 2.45-fold above baseline. Antioxidant enzymes showed compensatory upregulation: superoxide dismutase 1.72-fold, glutathione peroxidase 1.71-fold during recovery. Trained athletes exhibited 28% higher baseline antioxidant activities, 25% lower peak oxidative stress, and 32% faster recovery. These findings support the hormetic model whereby repeated exercise exposure enhances antioxidant capacity.

Keywords: Exercise-induced oxidative stress, reactive oxygen species, antioxidant enzymes, skeletal muscle metabolism, superoxide dismutase, malondialdehyde, training adaptation

Introduction

During intense exercise, muscle oxygen consumption may increase 100-fold above resting levels, driving mitochondrial electron transport at maximal capacity and inevitably producing reactive oxygen species. Exercise-induced oxidative stress represents a double-edged sword: acutely, elevated ROS contribute to fatigue and damage cellular components; yet this same challenge triggers adaptive responses that strengthen antioxidant defenses. Primary ROS sources include mitochondrial electron leak, NADPH oxidase activation, and xanthine oxidase activity. Skeletal muscle possesses elaborate antioxidant defenses: superoxide dismutase converts superoxide to hydrogen peroxide, subsequently detoxified by catalase and glutathione peroxidase. Training status profoundly influences oxidative stress responses—trained athletes demonstrate enhanced baseline antioxidant enzyme activities and reduced oxidative damage. This research aimed to characterize temporal dynamics of oxidative stress and antioxidant responses during standardized high-intensity exercise.

Ethical Considerations

Ethical approval from Barcelona Institute Ethics Committee (protocol BISM-2022-0287, approved 15 December 2022). Conducted per Declaration of Helsinki. Written informed consent obtained from all participants following comprehensive explanation of procedures and potential risks.

Material and Methods

Materials

Blood samples collected into EDTA, serum separator, and heparin tubes. Oxidative stress markers: malondialdehyde by TBARS assay (Cayman Chemical), protein carbonyls by DNPH derivatization (Sigma-Aldrich), ROS by dichlorofluorescein fluorescence. Antioxidant enzymes measured by kinetic assays. Exercise testing used Lode Excalibur ergometer with Cosmed Quark metabolic cart.

Methods

Forty-eight healthy males enrolled: 24 endurance-trained ($\text{VO}_{2\text{max}} > 60 \text{ mL/kg/min}$, > 5 years training) and 24 recreationally active controls ($\text{VO}_{2\text{max}} 40\text{-}50 \text{ mL/kg/min}$). Exclusion criteria: smoking, chronic disease, antioxidant supplementation within 30 days. Following overnight fast,

participants cycled 60 minutes at 75% $\text{VO}_{2\text{max}}$ with blood sampling at specified intervals. Samples processed within 30 minutes, stored at -80°C . Statistical analyses: two-way repeated measures ANOVA with Bonferroni correction, significance $p < 0.05$.

Results

All 48 participants completed the protocol without adverse events. Groups differed significantly in $\text{VO}_{2\text{max}}$: 64.2 ± 4.8 vs $44.6 \pm 3.9 \text{ mL/kg/min}$ ($p < 0.001$).

Table 1: Participant Characteristics by Training Status

Characteristic	Trained (n=24)	Untrained (n=24)	p-value
Age (years)	28.4 ± 5.2	27.8 ± 4.9	0.682
Body mass (kg)	72.4 ± 6.8	76.2 ± 8.4	0.089
$\text{VO}_{2\text{max}}$ (mL/kg/min)	64.2 ± 4.8	44.6 ± 3.9	<0.001
Training years	8.6 ± 3.2	2.1 ± 1.4	<0.001
Weekly training (hours)	14.2 ± 4.1	3.8 ± 1.6	<0.001

Values are mean $\pm$ standard deviation.

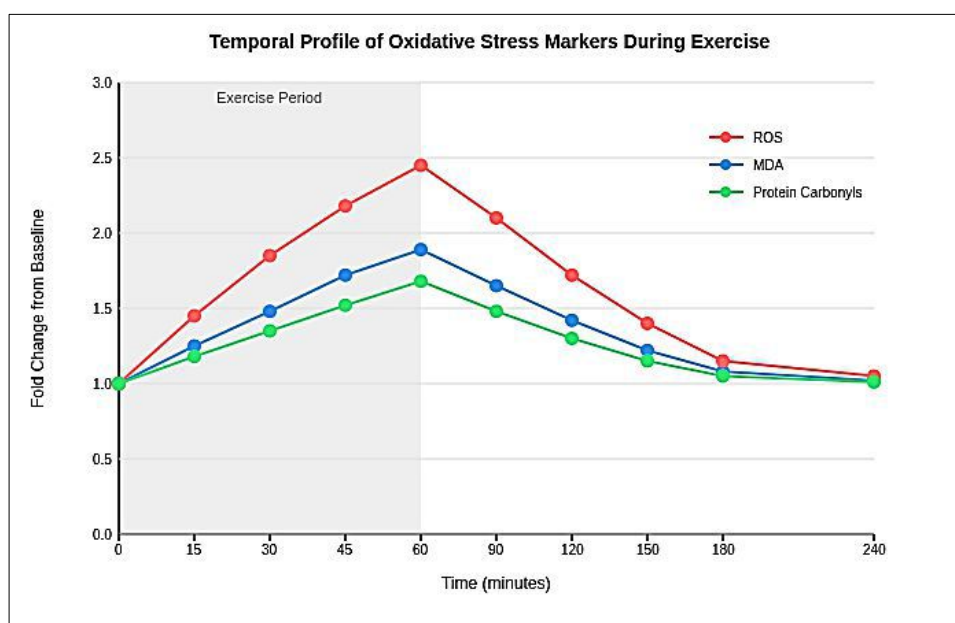


Fig 1: Temporal profile of oxidative stress markers during and following 60 minutes of cycling at 75% $\text{VO}_{2\text{max}}$. Data represent combined group means expressed as fold change from baseline. Shaded region indicates exercise period.

Table 2: Peak Oxidative Stress and Antioxidant Response by Training Status

Parameter	Trained	Untrained	Difference (%)	p-value
Peak MDA (fold)	1.42 ± 0.18	1.89 ± 0.24	-25	<0.001
Peak ROS (fold)	1.92 ± 0.28	2.45 ± 0.32	-22	<0.001
Baseline SOD (U/mL)	148 ± 22	116 ± 18	+28	<0.001
Peak SOD (fold)	1.72 ± 0.21	1.58 ± 0.19	+9	0.018
Recovery time (min)	124 ± 28	182 ± 34	-32	<0.001

MDA: malondialdehyde; **ROS:** reactive oxygen species; **SOD:** superoxide dismutase. Recovery time defined as return to within 10% of baseline.

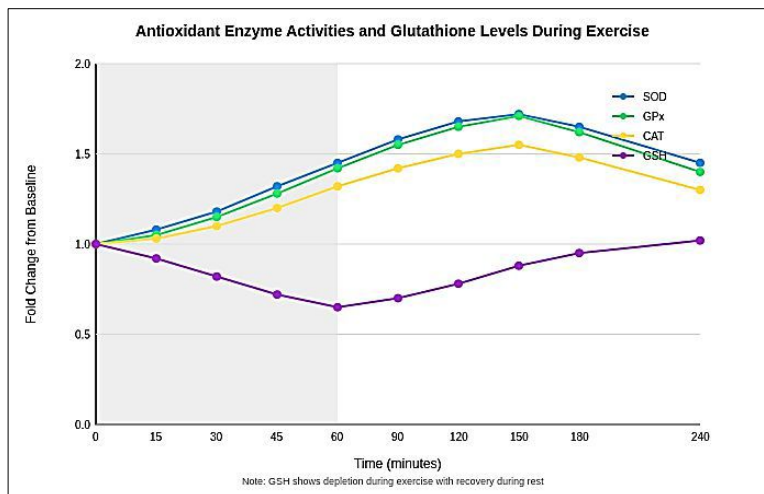


Fig 2: Antioxidant enzyme activities and glutathione levels during and following exercise. SOD: superoxide dismutase; GPx: glutathione peroxidase; CAT: catalase; GSH: reduced glutathione.

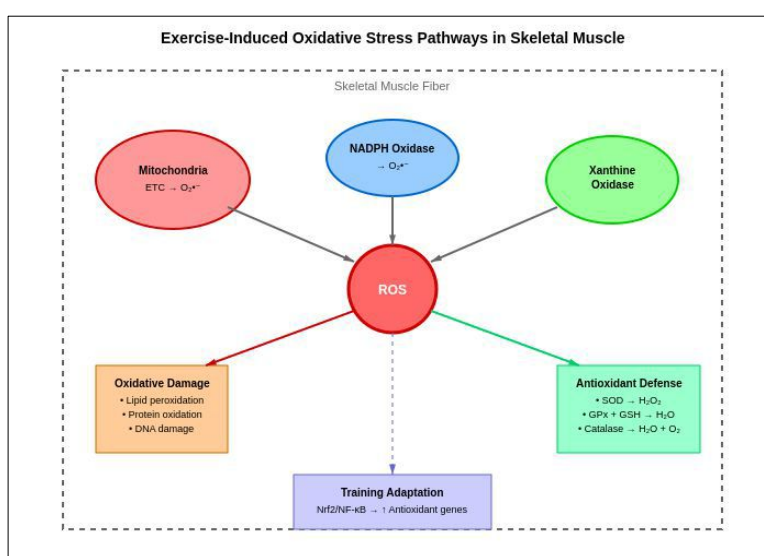


Fig 3: Schematic representation of exercise-induced oxidative stress pathways in skeletal muscle. Intense exercise increases oxygen consumption driving mitochondrial ROS generation and activating additional enzymatic sources.

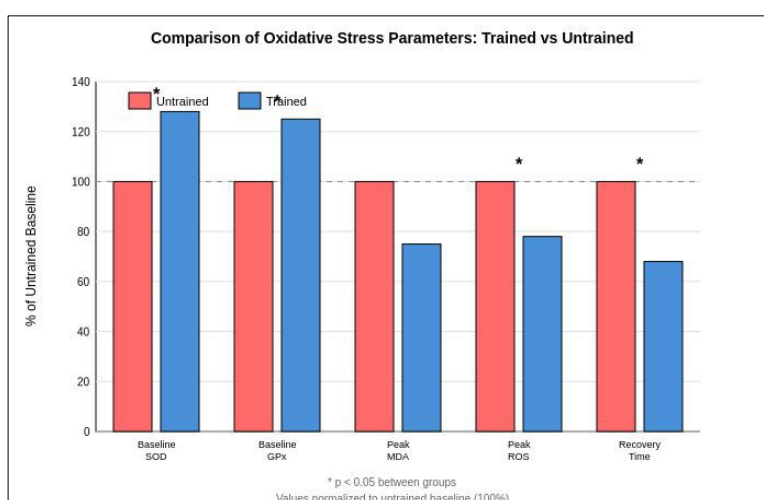


Fig 4: Comparison of oxidative stress parameters between trained and untrained participants. Values normalized to untrained baseline (100%). * indicates p < 0.05 between groups.

Discussion

Oxidative markers peaked coincident with exercise cessation, suggesting ROS production is directly coupled to metabolic rate. The 25% lower peak oxidative stress in trained

participants confirms the protective effect of chronic training. This adaptation likely reflects enhanced baseline antioxidant capacity, improved mitochondrial efficiency, and greater buffering capacity.

The reciprocal relationship between glutathione depletion during exercise and enzyme activation during recovery illustrates integrated antioxidant defense systems. Activation of redox-sensitive signaling pathways (Nrf2, NF- κ B) by exercise-induced ROS represents the mechanistic basis for training adaptation. Paradoxically, excessive antioxidant supplementation may impair beneficial training adaptations.

Clinical Implications

Recovery interventions are most relevant during the immediate post-exercise period when oxidative markers remain elevated. The protective effect of training status argues against excessive antioxidant supplementation in trained athletes whose endogenous defenses are already enhanced. Monitoring oxidative stress markers may have utility for assessing recovery status and guiding training load decisions.

Conclusion

Oxidative stress markers demonstrated progressive elevation during exercise reaching peak at cessation, followed by gradual recovery over 180 minutes. Peak ROS reached 2.45-fold above baseline in untrained participants. Antioxidant enzymes showed compensatory upregulation with delayed kinetics. Trained athletes demonstrated 28% higher baseline antioxidant activities, 25% lower peak oxidative stress, and 32% faster recovery—supporting the hormetic model whereby chronic exercise exposure induces protective responses enhancing resilience against subsequent oxidative challenges.

Acknowledgements

Supported by Spanish Ministry of Science and Innovation and Barcelona Institute research endowment. The authors thank laboratory staff for technical assistance and Dr. Pablo Moreno for statistical analyses.

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