



Phase-Dependent Interaction Between Oxidative Stress and Muscle Damage During Acute Recovery Following Eccentric Exercise

Roy Januardi Irawan^{1ABCDE}, Ratna Candra Dewi^{1BDE}, Ananda Perwira Bakti^{1BDE},
Nanda Rimawati^{1BCD}, Abdul Rohim Tualeka^{2ACD} and Putri Ayuni Allayannur^{2BDE}

¹Universitas Negeri Surabaya

²Airlangga University

Authors' Contribution: A – Study design; B – Data collection; C – Statistical analysis; D – Manuscript Preparation; E – Funds Collection

Corresponding Author: Roy Januardi Irawan, e-mail: royjanuardi@unesa.ac.id

Accepted for Publication: May 10, 2026

Published: May 30, 2026

DOI: 10.17309/tmfv.2026.3.20

Abstract

Background. Eccentric exercise induces oxidative stress, muscle damage, and decreased neuromuscular performance. However, the temporal relationship between these responses is not fully understood, particularly in recreationally active individuals.

Objectives. This study aimed to investigate the time-course changes and phase-dependent relationships between oxidative stress, muscle damage, and neuromuscular performance following an athletics-based eccentric exercise protocol.

Materials and Methods. A single-group repeated-measures design was employed involving 46 recreationally active male students (age: 19.15 ± 0.76 years). Participants performed an eccentric exercise protocol consisting of jump-landing, bounding, sprint deceleration, and slow-tempo squats. Malondialdehyde (MDA), Creatine Kinase (CK), and Countermovement Jump (CMJ) performance were measured at baseline and at 24, 48, and 72 hours post-exercise. Data were analyzed using repeated-measures ANOVA with Bonferroni post hoc tests and Pearson correlation analysis.

Results. Significant time effects were observed for all variables ($p < 0.001$). MDA and CK levels increased at 24 h, subsequently declining at 48 h and 72 h, yet remaining elevated compared to baseline. CMJ performance decreased at 24 h and showed partial recovery, but did not return to baseline at 72 h. No significant correlation was found between MDA and CK at 24 h; however, moderate positive correlations were observed at 48 h ($r = 0.557$, $p < 0.001$) and 72 h ($r = 0.496$, $p < 0.001$). No significant relationships were found between biochemical markers and CMJ.

Conclusions. Eccentric exercise induces oxidative stress and muscle damage alongside decreased neuromuscular performance. Biochemical markers peak at 24 h and decline thereafter, while neuromuscular recovery remains incomplete at 72 h. The relationship between oxidative stress and muscle damage is time-dependent, emerging during the recovery phase rather than the acute phase. These findings indicate distinct temporal patterns, suggesting that neuromuscular performance is not fully explained by biochemical markers.

Keywords: eccentric exercise, oxidative stress, creatine kinase, malondialdehyde, neuromuscular performance, recovery.

Introduction

Exercise-induced muscle damage (EIMD) is commonly observed following eccentric exercise and is typically characterized by muscle soreness, reduced strength, and

decreased neuromuscular performance (Leite et al., 2023; Li et al., 2024). Eccentric training is widely implemented in sport and physical education settings to enhance muscle strength, functional performance, and rehabilitation outcomes (Burgos-Jara et al., 2023; Cvečka et al., 2023; Maia et al., 2026). Eccentric muscle actions involve active muscle lengthening under tension, commonly occurring during deceleration phases of movement such as jumping and sprinting, and are integral to most sport-specific and

© Irawan, R., Dewi, R. C., Bakti, A. P., Rimawati, N., Tualeka, A. R., & Alayannur, P. A., 2026.

resistance training activities (Tecchio et al., 2024; Tomalka, 2023). Notably, eccentric contractions can generate greater force than concentric actions (approximately 20–50% higher) (Schoenfeld et al., 2017; Tecchio et al., 2024), making them effective stimuli for physiological responses associated with training.

However, this superior force-generating capacity is also associated with increased mechanical stress on muscle tissue, leading to exercise-induced muscle damage, characterized by structural disruption of muscle fibers, elevated oxidative stress, and temporary reductions in performance (Bontemps et al., 2020; Lепley et al., 2023; J. Peake et al., 2005). A conceptual contradiction can be identified within eccentric exercise: the same physiological processes that are associated with muscle damage and oxidative stress are also discussed in relation to recovery-related processes. This creates a challenge in determining whether attenuating these responses improves or limits functional outcomes.

In practice, attempts to maximize the benefits of eccentric training often involve increasing mechanical load, which may further exacerbate muscle damage and prolong recovery time (Chen et al., 2023; Rosvoglou et al., 2023; Zhang et al., 2026). Therefore, achieving an appropriate balance between induced muscle damage and recovery remains an important consideration in training design. A clearer understanding of acute physiological and functional responses to eccentric loading may help inform this balance.

This issue is particularly relevant in physical education contexts, where recreationally active students are frequently exposed to eccentric loading during practical sessions (Križaj et al., 2022; Maroto-Izquierdo et al., 2023), often without individualized recovery strategies (Lewis et al., 2012; Rosvoglou et al., 2023). One of the primary physiological responses to eccentric exercise is increased production of reactive oxygen species (ROS), contributing to oxidative stress, commonly assessed using biomarkers such as malondialdehyde (MDA) (Canals-Garzón et al., 2022; Wadley et al., 2019). Concurrently, muscle fiber disruption results in the release of intracellular enzymes into circulation, with creatine kinase (CK) widely recognized as a marker of muscle damage (Stożer et al., 2020). These physiological changes are often accompanied by reductions in neuromuscular performance, as assessed by countermovement jump (CMJ), a practical indicator reflecting muscle function and movement efficiency (Bishop et al., 2023; Yoshida et al., 2024). Collectively, these variables provide complementary indicators for evaluating responses to eccentric loading.

Previous literature suggests that oxidative stress and muscle damage are not solely negative responses. Moderate changes have been associated with physiological processes related to muscle repair (Di Meo et al., 2019; Wadley et al., 2019). However, excessive responses may delay recovery and impair subsequent performance (Şenışık et al., 2021). Thus, understanding the balance between these responses remains an important issue.

Despite this, these variables are often examined separately rather than in relation to each other. As a result, the temporal relationship between oxidative stress, muscle damage, and functional performance remains unclear, particularly following a single bout of eccentric exercise.

Therefore, this study aimed to investigate the temporal relationship between oxidative stress and muscle damage, in relation to changes in neuromuscular performance following eccentric exercise, using MDA, CK, and CMJ as respective indicators. It was hypothesized that increases in oxidative stress and muscle damage would be associated with reductions in performance, while their relationships may vary across the recovery period.

Materials and Methods

Study Design

This study employed a single-group experimental repeated-measures design to examine the temporal relationship between oxidative stress, muscle damage, and neuromuscular performance following an athletics-based eccentric exercise protocol. Measurements were conducted at baseline (pre-exercise), and at 24, 48, and 72 hours post-exercise to capture the temporal dynamics of physiological and neuromuscular performance.

The design was structured to examine changes in oxidative stress, muscle damage, and neuromuscular performance across time. This approach allows for the description of temporal patterns in physiological and performance responses following eccentric exercise.

By integrating biochemical (malondialdehyde and creatine kinase) and neuromuscular performance (countermovement jump) indicators across multiple time points, the study aimed to describe the temporal relationship between biochemical responses and neuromuscular performance following eccentric exercise in recreationally active individuals.

Participants

Participants were recruited from male undergraduate students ($n = 128$) enrolled in an athletics course at the Department of Sports Science, Universitas Negeri Surabaya. This group represented the accessible population, consisting of recreationally active individuals regularly exposed to athletics-related activities as part of their academic curriculum.

A total of 46 participants were selected using purposive sampling based on predefined inclusion and exclusion criteria. An a priori power analysis conducted using G*Power (version 3.1) indicated that a minimum sample size of 30–35 participants was required to detect a moderate effect size ($f = 0.25$) with a significance level of $\alpha = 0.05$ and statistical power $(1 - \beta) = 0.80$. Therefore, the final sample size was considered sufficient to detect statistically significant changes across time points within the repeated-measures design.

The inclusion criteria were as follows: (1) male students aged 18–22 years; (2) engagement in regular physical activity at least three times per week for the previous two months; (3) enrollment in an athletics course; and (4) no history of musculoskeletal injury within the past six months.

Exclusion criteria included: (1) failure to complete the exercise protocol; (2) absence from any follow-up measurement session; (3) use of medications or supplements that could influence oxidative stress or muscle damage

responses; and (4) any medical condition that could interfere with study procedures or compromise participant safety.

All participants provided written informed consent prior to participation. Recruitment was conducted during scheduled course sessions. All participants completed the study, and no dropouts were recorded.

Ethical approval was obtained from the Health Research Ethics Committee of Airlangga University (No. 104/EA/KEPK/2023) in accordance with the Declaration of Helsinki.

Anthropometric Measurements

Anthropometric measurements were obtained prior to the experimental protocol. Body height was measured using a stadiometer to the nearest 0.1 cm, and body mass was recorded using a digital scale to the nearest 0.1 kg. Body mass index (BMI) was subsequently calculated as body mass divided by the square of height (kg/m²).

Eccentric Exercise Protocol

Participants performed an athletics-based eccentric exercise protocol consisting of jump-landing drills, bounding, sprint deceleration tasks, and slow-tempo squats. Prior to the protocol, participants completed a standardized warm-up consisting of 5 minutes of light jogging followed by dynamic stretching exercises targeting the lower limbs. All exercises were performed at a self-selected maximal effort while maintaining proper technique.

The protocol was specifically designed to be applicable to recreationally active university students enrolled in an athletics course, emphasizing movements commonly practiced in physical education settings. The exercises were structured to induce substantial eccentric loading on the lower limb musculature, particularly during phases of landing, ground contact, and rapid deceleration.

The exercises were performed in the following sequence: jump-landing, bounding, sprint deceleration, and eccentric squats. The jump-landing component consisted of 5 sets of 10 repetitions, where participants were instructed to achieve maximal jump height and perform controlled landings by flexing the hips, knees, and ankles over approximately 2–3 seconds to absorb impact forces safely.

Bounding was performed over a distance of 20 meters for 4 sets, emphasizing horizontal propulsion followed by controlled single-leg landings to increase eccentric demand during each ground contact.

Sprint deceleration tasks consisted of 6 repetitions of 20-meter sprints followed by an abrupt stop within a 3-meter deceleration zone, requiring rapid reduction of forward momentum and generating high eccentric loading in the quadriceps and hamstring muscle groups.

To further enhance eccentric stimulus, participants performed slow-tempo bodyweight squats for 4 sets of 12 repetitions, with a controlled eccentric phase lasting approximately 4 seconds per repetition.

A fixed rest interval of 90 seconds was provided between sets to maintain high mechanical stress while minimizing full recovery. The total duration of the protocol was approximately 45–50 minutes. The protocol was specifically designed to induce exercise-induced muscle damage and oxidative stress responses for subsequent analysis. A summary of the exercise protocol is presented in Table 1.

The progression of participants throughout the study, covering recruitment, group allocation, follow-up, and data analysis, is illustrated in Figure 1.

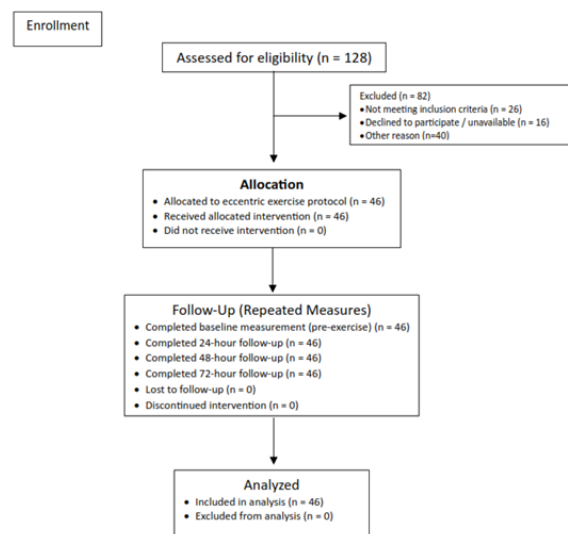


Fig. 1. CONSORT Flow Diagram

Table 1. Summary of the Eccentric Exercise Protocol

Component	Exercise Structure	Intensity / Execution	Volume	Rest Interval	Key Characteristics
Warm-up	Light jogging + dynamic stretching (lower limbs)	Low to moderate intensity	5 minutes	—	Prepare muscles and reduce injury risk
Jump-Landing	Repeated vertical jumps with controlled landing	Maximal effort; 2–3 s eccentric landing phase	5 sets × 10 reps	90 s	Emphasizes impact absorption and eccentric control
Bounding	Horizontal bounding over 20 m	Maximal horizontal propulsion with controlled single-leg landing	4 sets	90 s	Increases eccentric load during ground contact
Sprint Deceleration	20 m sprint + sudden stop within 3 m	Maximal sprint followed by rapid braking	6 reps	90 s	High eccentric demand on quadriceps and hamstrings
Eccentric Squat	Bodyweight squat	Controlled 4 s eccentric phase	4 sets × 12 reps	90 s	Prolonged time under tension for eccentric loading

Outcome Measures

The primary outcome measures included oxidative stress, muscle damage, and neuromuscular performance, assessed using Malondialdehyde (MDA), Creatine Kinase (CK), Countermovement Jump (CMJ), respectively. All measurements were conducted at baseline (pre-exercise), and at 24, 48, and 72 hours following the eccentric exercise protocol to describe changes in biochemical and neuromuscular performance variables over time.

Oxidative stress was assessed by measuring plasma Malondialdehyde (MDA) levels as an indicator of lipid peroxidation. Venous blood samples were collected under standardized conditions at each time point. Blood samples were centrifuged to obtain plasma, which was subsequently analyzed using a commercially available enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's instructions. MDA concentrations were expressed in nmol/mL.

Muscle damage was evaluated by measuring serum Creatine Kinase (CK) activity. Blood samples were analyzed using an automated enzymatic colorimetric method, and CK activity was expressed in U/L.

Neuromuscular performance was evaluated using the Countermovement Jump (CMJ) test. Participants performed the test with their hands placed on the hips to minimize arm swing, starting from an upright standing position followed by a rapid downward movement and an immediate maximal vertical jump. Jump height was recorded using a Jump MD device. Each participant performed three trials at each time point, and the highest value was used for analysis. A standardized rest interval of 60 seconds was provided between trials to minimize fatigue.

Statistical analysis

All data were analyzed using appropriate statistical software. Descriptive statistics are presented as mean \pm standard deviation (SD). The normality of data distribution was assessed using the Shapiro–Wilk test.

To examine changes in oxidative stress (MDA), muscle damage (CK), and neuromuscular performance (CMJ) across time (baseline, 24, 48, and 72 hours post-exercise), a one-way repeated-measures analysis of variance (ANOVA) was performed. When a significant main effect of time was detected, pairwise comparisons were conducted using Bonferroni post hoc tests.

In addition, Pearson correlation analysis was performed to evaluate the relationships between MDA, CK, and CMJ at each time point, to assess the relationships between physiological responses and neuromuscular performance.

Effect sizes for ANOVA were calculated using partial eta squared (η^2p) and interpreted as small (0.01), medium

(0.06), and large (0.14). Statistical significance was set at $p < 0.05$.

Results

Participant Characteristics

A total of 46 participants met all eligibility criteria and completed all measurement sessions (baseline, 24 h, 48 h, and 72 h post-exercise), with no loss to follow-up. Therefore, all analyses were conducted using a complete-case approach.

Participant characteristics are presented in Table 2. The participants were recreationally active male university students with a mean age of 19.15 ± 0.76 years, height of 167.97 ± 5.20 cm, and body mass of 59.10 ± 6.00 kg. The mean body mass index (BMI) was 20.91 ± 1.40 kg/m², indicating a normal weight range.

Table 2. Participant Characteristics

Variable	Mean (\pm SD)
Age (years)	19.15 \pm 0.76
Height (cm)	167.97 \pm 5.20
Weight (kg)	59.10 \pm 6.00
BMI (kg/m ²)	20.91 \pm 1.40

Descriptive and Time-Course Changes

Descriptive statistics for MDA, CK, and CMJ across all time points are presented in Table 3.

MDA levels increased from baseline to 24 h post-exercise, followed by a gradual decline at 48 h and 72 h, although values remained elevated relative to baseline. A similar pattern was observed for CK, which peaked at 24 h and progressively decreased thereafter, but did not fully return to baseline levels.

In contrast, CMJ performance showed a different pattern, with a reduction at 24 h, followed by a gradual recovery at 48 h and 72 h. However, CMJ values remained slightly lower than baseline even at 72 h.

These temporal patterns are illustrated in Figure 2.

These results show different temporal patterns between biochemical markers and neuromuscular performance, with peak MDA and CK values observed at 24 h alongside the lowest CMJ performance.

Repeated-Measures ANOVA

A repeated measures ANOVA revealed a significant main effect of time for all variables.

MDA levels were significantly affected by time ($F(1.203, 54.137) = 1166.676, p < 0.001, \eta^2p = 0.963$), with a large effect size.

Table 3. Changes in MDA, CK, and CMJ from baseline to 72 h post-exercise

Variable	Baseline	24 h	48 h	72 h
MDA (nmol/mL)	3.36 \pm 0.26 ^a	5.29 \pm 0.15 ^b	4.87 \pm 0.29 ^c	4.36 \pm 0.20 ^d
CK (U/L)	110.37 \pm 18.03 ^a	259.36 \pm 19.36 ^b	181.54 \pm 34.73 ^c	158.55 \pm 25.63 ^d
CMJ (cm)	40.48 \pm 2.96 ^a	35.34 \pm 2.59 ^b	36.85 \pm 2.47 ^c	39.32 \pm 2.68 ^d

Notes: Values are presented as mean \pm SD. Different superscripts (a–d) indicate significant differences between time points ($p < 0.05$, Bonferroni-adjusted).

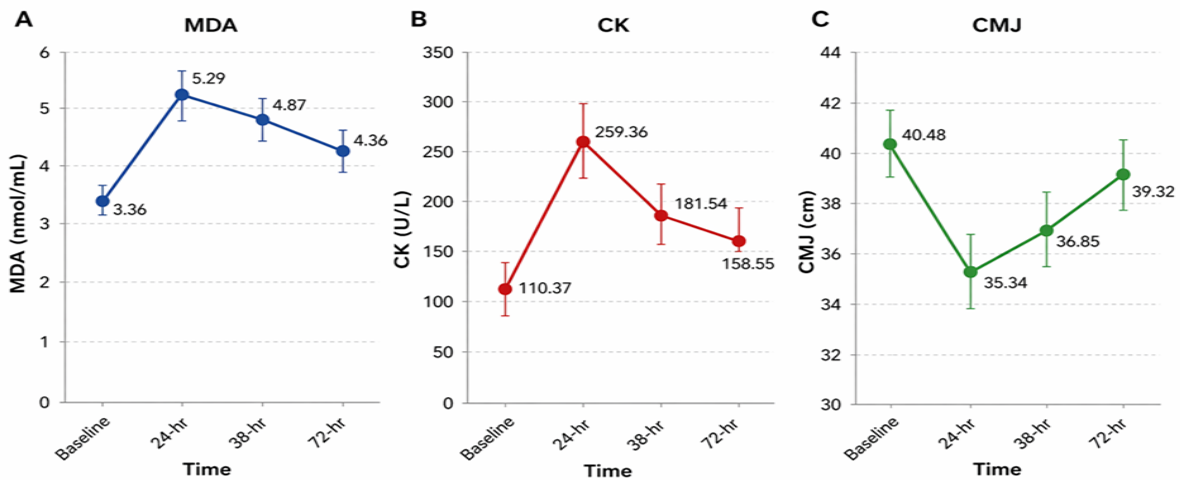


Fig. 2. Time-course changes in oxidative stress (MDA), muscle damage (CK), and neuromuscular performance (CMJ) following exercise. Notes: A: Malondialdehyde (MDA); B: Creatine Kinase (CK); C: Countermovement Jump (CMJ). Values are presented as mean \pm SD. Values are presented as mean \pm standard deviation

Table 4. Pairwise comparisons of MDA, CK, and CMJ across time points (Bonferroni-adjusted)

Variable	Comparison	Mean Difference (I-J)	95% CI	p-value
MDA (nmol/mL)	Baseline vs 24 h	-1.928	-2.025 to -1.831	$p < 0.001$
	Baseline vs 48 h	-1.505	-1.652 to -1.359	$p < 0.001$
	Baseline vs 72 h	-0.995	-1.118 to -0.871	$p < 0.001$
	24 h vs 48 h	0.423	0.353 to 0.492	$p < 0.001$
	24 h vs 72 h	0.933	0.892 to 0.974	$p < 0.001$
	48 h vs 72 h	0.510	0.472 to 0.549	$p < 0.001$
CK (U/L)	Baseline vs 24 h	-148.994	-157.912 to -140.076	$p < 0.001$
	Baseline vs 48 h	-71.173	-86.761 to -55.584	$p < 0.001$
	Baseline vs 72 h	-48.184	-60.271 to -36.097	$p < 0.001$
	24 h vs 48 h	77.821	63.967 to 91.675	$p < 0.001$
	24 h vs 72 h	100.810	90.106 to 111.513	$p < 0.001$
	48 h vs 72 h	22.989	19.167 to 26.811	$p < 0.001$
CMJ (cm)	Baseline vs 24 h	5.137	3.648 to 6.627	$p < 0.001$
	Baseline vs 48 h	3.627	2.600 to 4.653	$p < 0.001$
	Baseline vs 72 h	1.155	0.294 to 2.016	$p = 0.003$
	24 h vs 48 h	-1.511	-2.746 to -0.276	$p = 0.009$
	24 h vs 72 h	-3.982	-5.029 to -2.936	$p < 0.001$
	48 h vs 72 h	-2.472	-3.357 to -1.586	$p < 0.001$

Notes: Values represent mean differences (I-J). Confidence intervals (CI) are presented at 95%. P-values were adjusted using the Bonferroni correction for multiple comparisons. Statistical significance was set at $p < 0.05$.

Similarly, CK levels showed a significant effect of time ($F_{(1.465, 65.904)} = 446.903$, $p < 0.001$, $\eta^2_p = 0.909$), with a large effect size.

CMJ performance also demonstrated a significant effect of time ($F_{(2.165, 97.431)} = 66.879$, $p < 0.001$, $\eta^2_p = 0.598$), with a moderate to large effect size.

Post Hoc Analysis

Bonferroni-adjusted pairwise comparisons are presented in Table 4.

MDA levels were significantly higher at 24 h, 48 h, and 72 h compared to baseline ($p < 0.001$ for all comparisons). Significant differences were also observed between all post-exercise time points (24 h vs 48 h, 24 h vs 72 h, and 48 h vs 72 h; $p < 0.001$).

CK levels were significantly higher at 24 h, 48 h, and 72 h compared to baseline ($p < 0.001$ for all comparisons). Significant differences were also observed between all post-exercise time points ($p < 0.001$).

CMJ performance was significantly lower at 24 h, 48 h, and 72 h compared to baseline ($p < 0.01$ for all comparisons).

Significant differences were also found between 24 h and 48 h ($p = 0.009$), 24 h and 72 h ($p < 0.001$), and 48 h and 72 h ($p < 0.001$).

Correlation Analysis

Pearson correlation coefficients are presented in Table 5.

No significant correlations were observed between variables at 24 h post-exercise, suggesting that the association between oxidative stress and muscle damage has not yet fully developed during the acute phase.

However, significant positive correlations between MDA and CK emerged at 48 h ($r = 0.557$, $p < 0.001$) and 72 h ($r = 0.496$, $p < 0.001$), indicating a stronger association between oxidative stress and muscle damage during the recovery phase.

No significant relationships were observed between CMJ and either MDA or CK at any time point.

Table 5. Pearson correlation coefficients between MDA, CK, and CMJ at 24 h, 48 h, and 72 h post-exercise

Time	Variable	MDA	CK	CMJ
24 h post-exercise	MDA	1	0.199	-0.131
	CK	0.199	1	0.111
	CMJ	-0.131	0.111	1
48 h post-exercise	MDA	1	0.557**	0.131
	CK	0.557**	1	-0.036
	CMJ	0.131	-0.036	1
72 h post-exercise	MDA	1	0.496**	-0.061
	CK	0.496**	1	0.024
	CMJ	-0.061	0.024	1

Notes: Values represent Pearson correlation coefficients (r). Statistical significance was set at $p < 0.05$ (2-tailed). ** $p < 0.01$ (2-tailed). MDA = Malondialdehyde; CK = Creatine Kinase; CMJ = Countermovement Jump.

Scatter Plot Analysis

The relationships between MDA and CK at 48 h and 72 h are illustrated in Figure 3.

At 48 h, a moderate positive linear relationship was observed ($R^2 = 0.31$), indicating a moderate shared variance between MDA and CK. A similar, though slightly weaker, relationship was observed at 72 h.

These findings visually reinforce the correlation analysis, highlighting that the association between oxidative stress and muscle damage becomes more pronounced during the recovery phase rather than the acute phase, suggesting a delayed temporal association between oxidative stress and muscle damage.

Discussion

Principal Findings

The present study shows that eccentric exercise is associated with increases in oxidative stress (MDA) and muscle damage (CK), along with a reduction in neuromuscular performance (CMJ). MDA and CK peaked at 24 h post-exercise and remained elevated up to 72 h, whereas CMJ showed a gradual recovery pattern but had not fully returned to baseline at 72 h.

Correlation analysis showed no significant association between MDA and CK at 24 h. Moderate positive correlations were observed at 48 h and 72 h. No significant relationships were found between biochemical markers (MDA and CK) and CMJ performance at any time point.

These results indicate that biochemical and neuromuscular responses may follow different temporal patterns during recovery. These findings reflect acute recovery responses following a single bout of eccentric exercise.

Time-Course of Oxidative Stress and Muscle Damage

The increase in MDA at 24 h reflects elevated lipid peroxidation, which is commonly used as an indirect marker of oxidative stress following eccentric exercise.

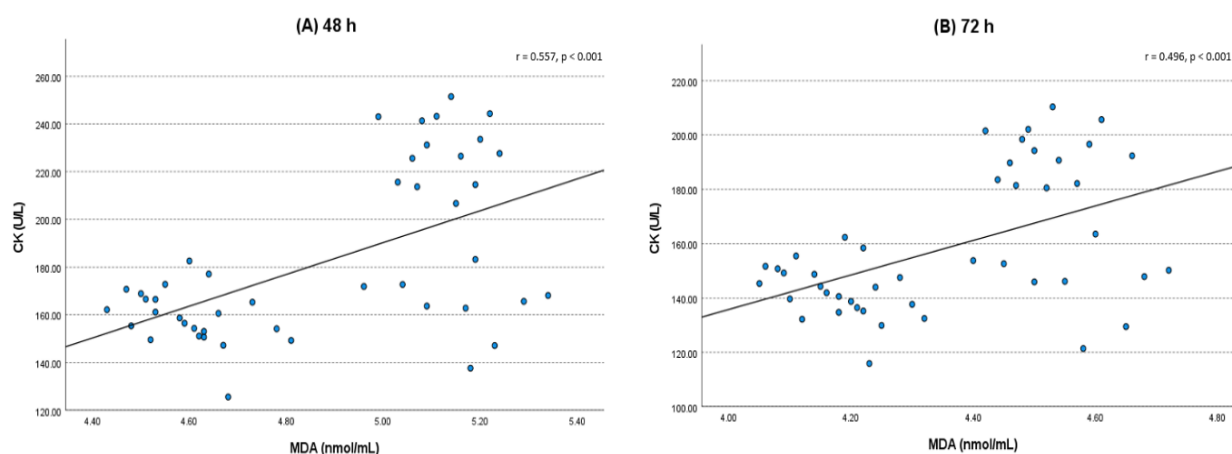


Fig. 3. Relationship between malondialdehyde (MDA) and creatine kinase (CK) at 48 h (A) and 72 h (B) post-exercise. Significant positive correlations were observed at both time points (48 h: $r = 0.557$, $p < 0.001$; 72 h: $r = 0.496$, $p < 0.001$)

Previous studies have suggested that eccentric loading may increase reactive oxygen species (ROS) production, which is associated with oxidative stress responses (Blazevich et al., 2025; Xu & Guo, 2025).

Eccentric exercise has been reported to induce mechanical strain at the muscle fiber level, which may contribute to structural disturbances such as Z-disk disruption and altered calcium regulation (Dowling et al., 2022; Tomalka, 2023). These processes are often discussed in the literature as part of muscle damage-related responses following unaccustomed eccentric loading.

Similarly, CK increased significantly at 24 h, which is widely considered an indicator of muscle membrane disruption and leakage of intracellular enzymes into circulation (Blank et al., 2022; Ying et al., 2024).

Although both MDA and CK peaked at 24 h, the absence of a significant correlation at this time point suggests that these markers may reflect partially independent physiological responses during the early recovery phase. This interpretation aligns with previous literature suggesting that early post-exercise responses are influenced by multiple overlapping mechanisms (J. M. Peake et al., 2017; Proske & Morgan, 2001) intense exercise causes an “open window” of immunodepression during recovery after exercise is well accepted. Repeated exercise bouts or intensified training without sufficient recovery may increase the risk of illness. However, except for salivary IgA, clear and consistent markers of this immunodepression remain elusive. Exercise increases circulating neutrophil and monocyte counts and reduces circulating lymphocyte count during recovery. This lymphopenia results from preferential egress of lymphocyte subtypes with potent effector functions [e.g., natural killer (NK).

At later time points (48–72 h), a moderate association between MDA and CK was observed, suggesting a closer temporal alignment between oxidative stress and muscle damage during recovery. Nevertheless, this relationship remains correlational and does not imply causality or a direct mechanistic link. Accordingly, this interpretation should be approached with caution, particularly given the inherent limitations of biochemical markers in reflecting underlying physiological processes.

Neuromuscular Performance and Recovery Pattern

CMJ performance decreased at 24 h following eccentric exercise, indicating a temporary reduction in neuromuscular performance. This finding is consistent with previous reports showing reduced jump performance in the early recovery phase after eccentric loading (Hemmatinfar et al., 2023).

The decline in performance may be associated with multiple factors described in the literature, including altered excitation–contraction coupling, changes in calcium handling, and transient reductions in neural drive (Tabuchi et al., 2022). In addition, delayed onset muscle soreness (DOMS) and protective neural inhibition may also contribute to reduced performance capacity (Boyd et al., 2023; Sozlu et al., 2025).

Although CMJ showed gradual improvement at 48 h and 72 h, it had not fully returned to baseline within the observation period, suggesting that neuromuscular recovery may not be fully complete within 72 h under the present conditions.

Importantly, no significant correlations were found between CMJ and either MDA or CK. This suggests that neuromuscular performance recovery is likely influenced by multiple factors beyond biochemical markers alone.

Phase-Dependent Relationship Between Oxidative Stress and Muscle Damage

The present findings indicate that the relationship between MDA and CK may vary across time. No significant association was observed at 24 h, whereas moderate positive correlations appeared at 48 h and 72 h.

During the early phase (24 h), CK elevation likely reflects structural muscle perturbation, while oxidative stress markers may represent a parallel response that is not directly linked at this stage. This interpretation aligns with previous studies indicating that early responses to eccentric exercise involve multiple independent physiological processes (Qian et al., 2023).

In the later phase (48–72 h), previous studies have reported increased involvement of inflammatory and immune-related processes, including ROS production and tissue remodeling activity (J. M. Peake et al., 2017; Tidball & Villalta, 2010) intense exercise causes an “open window” of immunodepression during recovery after exercise is well accepted. Repeated exercise bouts or intensified training without sufficient recovery may increase the risk of illness. However, except for salivary IgA, clear and consistent markers of this immunodepression remain elusive. Exercise increases circulating neutrophil and monocyte counts and reduces circulating lymphocyte count during recovery. This lymphopenia results from preferential egress of lymphocyte subtypes with potent effector functions [e.g., natural killer (NK). These processes may contribute to the observed association between oxidative stress and muscle damage markers during recovery.

Overall, the time-dependent pattern observed in this study suggests a changing relationship between these variables across the recovery period. However, due to the observational nature of the data, causal interpretation should be made cautiously. This pattern may reflect differences between early and later phases of recovery, with changes in the relationship between variables over time.

Lack of Association Between Biochemical Markers and Performance

The absence of significant correlations between CMJ and biochemical markers suggests that neuromuscular performance is not directly explained by MDA or CK levels in this study.

This finding aligns with previous studies suggesting that recovery of jump performance is influenced by a combination of peripheral and central factors, including neuromuscular control, pain perception, and psychological responses. Therefore, biochemical markers alone may not fully represent neuromuscular recovery.

Practical Implications

The results indicate that eccentric exercise induces time-dependent changes in biochemical and neuromuscular

variables, with the highest levels of oxidative stress and muscle damage observed at 24 h, and gradual recovery observed thereafter.

These findings may be useful for informing general recovery considerations in recreationally active individuals exposed to eccentric loading, particularly in understanding the temporal pattern of recovery responses.

However, practical applications should be interpreted cautiously, as this study did not directly test training interventions or recovery strategies.

Final Synthesis

In summary, eccentric exercise induces distinct temporal responses in oxidative stress, muscle damage, and neuromuscular performance. The relationship between biochemical markers appears to vary across time, while neuromuscular recovery seems to involve additional factors beyond biochemical changes alone.

These findings contribute to a descriptive understanding of the time-course response following eccentric exercise. However, further studies with controlled designs and additional physiological measurements are required to clarify the underlying mechanisms.

Conclusion

Eccentric exercise is associated with increased oxidative stress and muscle damage, accompanied by a reduction in neuromuscular performance. Biochemical markers peaked at 24 h post-exercise and gradually declined, whereas neuromuscular recovery remained incomplete at 72 h.

The relationship between oxidative stress and muscle damage appeared to be time-dependent, with associations emerging during the recovery phase rather than the acute phase.

These findings indicate that biochemical and neuromuscular responses may follow different temporal patterns following eccentric exercise. Neuromuscular recovery does not appear to be fully explained by biochemical markers alone, suggesting the involvement of additional physiological and neuromuscular factors.

Overall, the present results provide a descriptive account of acute recovery dynamics following eccentric loading. Further research using controlled designs and additional physiological measures is required to better understand the underlying processes.

Limitations

This study has several limitations that should be considered when interpreting the findings. First, the sample consisted exclusively of young male participants, which limits the generalizability of the results to other populations, including females, older individuals, or different training backgrounds.

Second, only indirect biochemical markers of oxidative stress and muscle damage were assessed. The absence of direct measurements of inflammatory and cellular processes (e.g., cytokines or immune responses) restricts the ability to comprehensively characterize the underlying physiological responses.

Third, neuromuscular performance was evaluated using a single test (CMJ), which may not fully capture the multidimensional nature of neuromuscular recovery.

Fourth, the single-group design without a control or comparator group limits the ability to attribute the observed changes exclusively to the eccentric exercise protocol and does not account for potential influences such as repeated testing effects or natural variability over time.

Finally, dietary intake and physical activity outside the experimental protocol were not strictly controlled, which may have influenced individual recovery responses.

Taken together, these limitations indicate that the present findings should be interpreted as descriptive of acute responses under the specific study conditions, rather than as evidence of causal mechanisms or generalizable recovery patterns. Additionally, the observational nature of the study and the use of correlational analyses preclude any causal inference regarding the relationship between oxidative stress, muscle damage, and neuromuscular responses.

Ethics Approval

This study was approved by the Health Research Ethics Committee of Airlangga University (No. 104/EA/KEPK/2023) and conducted in accordance with the Declaration of Helsinki.

Informed Consent

Written informed consent was obtained from all participants prior to participation in the study.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

AI Transparency Statement

The authors did not use AI-assisted tools in the preparation of this manuscript.

Acknowledgements

The authors gratefully acknowledge the financial support provided by the Ministry of Education, Culture, Research, and Technology of Indonesia through the BIMA research grant program (Grant No. 195/DST/C3/DT.05.00/2026, 2026). The authors also thank Universitas Negeri Surabaya and Airlangga University for institutional and technical support.

Conflicts of Interest

The authors declare no conflicts of interest.

References

- Leite, C.D.F.C., Zovico, P.V.C., Rica, R.L., Barros, B.M., Machado, A.F., Evangelista, A.L., Leite, R.D., Barauna, V.G., Maia, A.F., & Bocalini, D.S. (2023). Exercise-

- Induced Muscle Damage after a High-Intensity Interval Exercise Session: Systematic Review. *International Journal of Environmental Research and Public Health*, 20(22), 7082. <https://doi.org/10.3390/ijerph20227082>
- Li, D.C.W., Rudloff, S., Langer, H.T., Norman, K., & Herpich, C. (2024). Age-Associated Differences in Recovery from Exercise-Induced Muscle Damage. *Cells*, 13(3), 255. <https://doi.org/10.3390/cells13030255>
- Burgos-Jara, C., Cerda-Kohler, H., Aedo-Muñoz, E., & Miarka, B. (2023). Eccentric Resistance Training: A Methodological Proposal of Eccentric Muscle Exercise Classification Based on Exercise Complexity, Training Objectives, Methods, and Intensity. *Applied Sciences*, 13(13), 7969. <https://doi.org/10.3390/app13137969>
- Cvečka, J., Vajda, M., Novotná, A., Löfler, S., Hamar, D., & Krčmár, M. (2023). Benefits of Eccentric Training with Emphasis on Demands of Daily Living Activities and Feasibility in Older Adults: A Literature Review. *International Journal of Environmental Research and Public Health*, 20(4), 3172. <https://doi.org/10.3390/ijerph20043172>
- Maia, C.O.P., Pérez, D.I.V., Teixeira, R.P.A., Brito, C.J., Aedo-Muñoz, E., & Miarka, B. (2026). Eccentric vs. Concentric Training: A Systematic Review and Meta-Analysis of Randomized Controlled Trials on Performance and Health Benefits Across Diverse Populations. *Sports*, 14(3), 119. <https://doi.org/10.3390/sports14030119>
- Tecchio, P., Raiteri, B.J., & Hahn, D. (2024). Eccentric exercise ≠ eccentric contraction. *Journal of Applied Physiology*, 136(4), 954-965. <https://doi.org/10.1152/jappphysiol.00845.2023>
- Tomalka, A. (2023). Eccentric muscle contractions: from single muscle fibre to whole muscle mechanics. *Pflügers Archiv - European Journal of Physiology*, 475(4), 421-435. <https://doi.org/10.1007/s00424-023-02794-z>
- Schoenfeld, B.J., Ogborn, D.I., Vigotsky, A.D., Franchi, M.V., & Krieger, J.W. (2017). Hypertrophic Effects of Concentric vs. Eccentric Muscle Actions: A Systematic Review and Meta-analysis. *Journal of Strength and Conditioning Research*, 31(9), 2599-2608. <https://doi.org/10.1519/JSC.0000000000001983>
- Bontemps, B., Vercruyssen, F., Gruet, M., & Louis, J. (2020). Downhill Running: What Are The Effects and How Can We Adapt? A Narrative Review. *Sports Medicine*, 50(12), 2083-2110. <https://doi.org/10.1007/s40279-020-01355-z>
- Lepley, L.K., Stoneback, L., Macpherson, P.C.D., & Butterfield, T.A. (2023). Eccentric Exercise as a Potent Prescription for Muscle Weakness After Joint Injury. *Exercise and Sport Sciences Reviews*, 51(3), 109-116. <https://doi.org/10.1249/JES.0000000000000319>
- Peake, J., Nosaka, K., & Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans. *In Exercise Immunology Review*, 11.
- Chen, T.C., Kang, H., Tseng, W., Lin, S., Chan, C., Chen, H., Chou, T., Wang, H., Lau, W.Y., & Nosaka, K. (2023). Muscle damage induced by maximal eccentric exercise of the elbow flexors after 3-week immobilization. *Scandinavian Journal of Medicine & Science in Sports*, 33(4), 382-392. <https://doi.org/10.1111/sms.14279>
- Rosvoglou, A., Fatouros, I.G., Poulos, A., Tsatalas, T., Papanikolaou, K., Karampina, E., Liakou, C.A., Tsimeas, P., Karanika, P., Tsoukas, D., Katrabasas, I., Chatzinikolaou, A., Deli, C.K., Giakas, G., Jamurtas, A.Z., & Draganidis, D. (2023). Recovery kinetics following eccentric exercise is volume-dependent. *Journal of Sports Sciences*, 41(13), 1326-1335. <https://doi.org/10.1080/02640414.2023.2272101>
- Zhang, X., Weakley, J., Li, H., Marcos-Frutos, D., & García-Ramos, A. (2026). Acute and Chronic Effects of Accentuated Eccentric Loading vs. Constant-Load Resistance Training: A Systematic Review and Meta-analysis. *Sports Medicine*. <https://doi.org/10.1007/s40279-026-02422-7>
- Križaj, L., Kozinc, Ž., Löfler, S., & Šarabon, N. (2022). The chronic effects of eccentric exercise interventions in different populations: an umbrella review. *European Journal of Translational Myology*. <https://doi.org/10.4081/ejtm.2022.10876>
- Maroto-Izquierdo, S., Martín-Rivera, F., Nosaka, K., Beato, M., González-Gallego, J., & de Paz, J.A. (2023). Effects of submaximal and supramaximal accentuated eccentric loading on mass and function. *Frontiers in Physiology*, 14. <https://doi.org/10.3389/fphys.2023.1176835>
- Lewis, P.B., Ruby, D., & Bush-Joseph, C.A. (2012). Muscle Soreness and Delayed-Onset Muscle Soreness. *In Clinics in Sports Medicine*, 31(2), 255-262. <https://doi.org/10.1016/j.csm.2011.09.009>
- Canals-Garzón, C., Guisado-Barrilao, R., Martínez-García, D., Chiroso-Ríos, I.J., Jerez-Mayorga, D., & Guisado-Requena, I.M. (2022). Effect of Antioxidant Supplementation on Markers of Oxidative Stress and Muscle Damage after Strength Exercise: A Systematic Review. *International Journal of Environmental Research and Public Health*, 19(3), 1803. <https://doi.org/10.3390/ijerph19031803>
- Wadley, A.J., Keane, G., Cullen, T., James, L., Vautrinot, J., Davies, M., Hussey, B., Hunter, D.J., Mastana, S., Holliday, A., Petersen, S.V., Bishop, N.C., Lindley, M.R., & Coles, S.J. (2019). Characterization of extracellular redox enzyme concentrations in response to exercise in humans. *Journal of Applied Physiology*, 127(3), 858-866. <https://doi.org/10.1152/jappphysiol.00340.2019>
- Stožer, A., Vodopivec, P., & Krizančič Bombek, L. (2020). Pathophysiology of exercise-induced muscle damage and its structural, functional, metabolic, and clinical consequences. *Physiological Research*, 565-598. <https://doi.org/10.33549/physiolres.934371>
- Bishop, C., Jordan, M., Torres-Ronda, L., Loturco, I., Harry, J., Virgile, A., Mundy, P., Turner, A., & Comfort, P. (2023). Selecting Metrics That Matter: Comparing the Use of the Countermovement Jump for Performance Profiling, Neuromuscular Fatigue Monitoring, and Injury Rehabilitation Testing. *Strength & Conditioning Journal*, 45(5), 545-553. <https://doi.org/10.1519/SSC.0000000000000772>
- Yoshida, N., Hornsby, W.G., Sole, C.J., Sato, K., & Stone, M.H. (2024). Effect of Neuromuscular Fatigue on the Countermovement Jump Characteristics: Basketball-Related High-Intensity Exercises. *Journal of Strength & Conditioning Research*, 38(1), 164-173. <https://doi.org/10.1519/JSC.00000000000004610>
- Di Meo, S., Napolitano, G., & Venditti, P. (2019). Mediators of Physical Activity Protection against ROS-Linked Skeletal Muscle Damage. *International Journal of Molecular Sciences*, 20(12), 3024. <https://doi.org/10.3390/ijms20123024>

- Şenışık, S.Ç., Akova, B., Şekir, U., & Gür, H. (2021). Effects of Muscle Architecture on Eccentric Exercise Induced Muscle Damage Responses. *Journal of Sports Science and Medicine*, 655-664. <https://doi.org/10.52082/jssm.2021.655>
- Blazevich, A.J., Herzog, W., & Nunes, J.P. (2025). Triggering sarcomerogenesis: Examining key stimuli and the role attributed to eccentric training-Historical, systematic, and meta-analytic review. *Journal of Sport and Health Science*, 14, 101073. <https://doi.org/10.1016/j.jshs.2025.101073>
- Xu, B., & Guo, S. (2025). Daytime eccentric exercise and its impact on inflammatory markers and antioxidant defenses in physically active young men. *Frontiers in Immunology*, 16. <https://doi.org/10.3389/fimmu.2025.1655034>
- Dowling, P., Gargan, S., Swandulla, D., & Ohlendieck, K. (2022). Proteomic profiling of impaired excitation-contraction coupling and abnormal calcium handling in muscular dystrophy. *PROTEOMICS*, 22(23-24). <https://doi.org/10.1002/pmic.202200003>
- Blank, P.S., Golding, A.E., Benavides, I.M., Waters, H., Mekhedov, E., Bezrukov, L., Wachter, R.D., Mikhailenko, I., Brown, R.H., Bönnemann, C.G., Demidowich, A.P., Jain, M.S., Yanovski, J.A., & Zimmerberg, J. (2022). *Physical activity-correlated changes in plasma enzyme concentrations in fragile sarcolemmal muscular dystrophies*. <https://doi.org/10.1101/2022.04.01.22273213>
- Ying, W., Da-Wei, W., Jun, L., Xian-Jie, X., Zhi-Dan, G., Hong-Yan, L., Yong, Z., & Peng, L. (2024). Protective Effect of Dihydromyricetin Against Exercise-Induced Muscle Damage and Its Mechanism. *Chinese Medical Sciences Journal*, 39(1), 46-53. <https://doi.org/10.24920/004272>
- Peake, J.M., Neubauer, O., Walsh, N.P., & Simpson, R.J. (2017). Recovery of the immune system after exercise. *Journal of Applied Physiology*, 122(5), 1077-1087. <https://doi.org/10.1152/jappphysiol.00622.2016>
- Proske, U., & Morgan, D.L. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *The Journal of Physiology*, 537(2), 333-345. <https://doi.org/10.1111/j.1469-7793.2001.00333.x>
- Hemmatinafar, M., Zaremoayedi, L., Koushkie Jahromi, M., Alvarez-Alvarado, S., Wong, A., Niknam, A., Suzuki, K., Imanian, B., & Bagheri, R. (2023). Effect of Beetroot Juice Supplementation on Muscle Soreness and Performance Recovery after Exercise-Induced Muscle Damage in Female Volleyball Players. *Nutrients*, 15(17), 3763. <https://doi.org/10.3390/nu15173763>
- Tabuchi, A., Tanaka, Y., Takagi, R., Shirakawa, H., Shibaguchi, T., Sugiura, T., Poole, D.C., & Kano, Y. (2022). Ryanodine receptors mediate high intracellular Ca²⁺ and some myocyte damage following eccentric contractions in rat fast-twitch skeletal muscle. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 322(1), R14-R27. <https://doi.org/10.1152/ajpregu.00166.2021>
- Boyd, L., Deakin, G.B., Devantier-Thomas, B., Singh, U., & Doma, K. (2023). The Effects of Pre-conditioning on Exercise-Induced Muscle Damage: A Systematic Review and Meta-analysis. *Sports Medicine*, 53(8), 1537-1557. <https://doi.org/10.1007/s40279-023-01839-8>
- Sozlu, U., Basar, S., Semsi, R., Akaras, E., & Sepici Dincel, A. (2025). Preventive effect of the neurodynamic mobilization technique on delayed onset of muscle soreness: a randomized, single-blinded, placebo-controlled study. *BMC Musculoskeletal Disorders*, 26(1), 464. <https://doi.org/10.1186/s12891-025-08723-8>
- Qian, Z., Ping, L., & Xuelin, Z. (2023). Re-examining the mechanism of eccentric exercise-induced skeletal muscle damage from the role of the third filament, titin (Review). *Biomedical Reports*, 20(1), 14. <https://doi.org/10.3892/br.2023.1703>
- Tidball, J.G., & Villalta, S.A. (2010). Regulatory interactions between muscle and the immune system during muscle regeneration. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 298(5), R1173-R1187. <https://doi.org/10.1152/ajpregu.00735.2009>

Фазово-залежна взаємодія між оксидативним стресом і пошкодженням м'язів під час гострого відновлення після ексцентричних вправ

Рой Януарді Іраван^{1ABCDE}, Ратна Чандра Деві^{1BDE}, Ананда Первіра Бакті^{1BDE},
Нанда Рімаваті^{1BCD}, Абдул Рохім Туалека^{2ACD}, Путрі Аюні Аллаяннур^{2BDE}

¹Державний університет Сурабаї

²Університет Аїрланга

Авторський вклад: А – дизайн дослідження; В – збір даних; С – статаналіз; D – підготовка рукопису; E – збір коштів

Реферат. Стаття: 11 с., 5 табл., 3 рис., 37 джерел.

Обґрунтування. Ексцентричні вправи спричиняють оксидативний стрес, пошкодження м'язів та зниження нервово-м'язової працездатності. Проте часовий взаємозв'язок між цими реакціями залишається недостатньо зрозумілим, зокрема у фізично активних осіб, які займаються рекреаційним спортом.

Мета дослідження. Це дослідження мало на меті вивчити часову динаміку змін та фазово-залежні взаємозв'язки між оксидативним стресом, пошкодженням м'язів і нервово-м'язовою працездатністю після виконання протоколу ексцентричних вправ на основі легкоатлетичних елементів.

Матеріали і методи. Застосовано одногруповий дизайн дослідження з повторними вимірюваннями, до якого залучили 46 фізично активних студентів чоловічої статі, які займаються рекреаційним спортом (вік: $19,15 \pm 0,76$ року). Учасники виконували протокол ексцентричних вправ, що включав стрибки з приземленням, багатоскоки, уповільнення під час спринту та присідання в повільному темпі. Рівні малонового діальдегіду (МДА), креатинкінази (КК) та показники стрибка з контррухом (СКР) вимірювали на вихідному рівні, а також через 24, 48 та 72 години після фізичного навантаження. Аналіз даних проведено за допомогою дисперсійного аналізу з повторними вимірюваннями із застосуванням *post-hoc* критерію Бонферроні та кореляційного аналізу Пірсона.

Результати. Для всіх змінних виявлено значущі часові ефекти ($p < 0,001$). Рівні МДА та КК зросли через 24 години, після чого знизилися через 48 і 72 години, проте залишалися підвищеними порівняно з вихідним рівнем. Показники СКР знизилися через 24 години й продемонстрували часткове відновлення, але не повернулися до вихідного рівня через 72 години. Не було виявлено значущої кореляції між МДА та КК через 24 години; однак помірні позитивні кореляції спостерігалися через 48 годин ($r = 0,557$, $p < 0,001$) та 72 години ($r = 0,496$, $p < 0,001$). Значущих взаємозв'язків між біохімічними маркерами та показниками СКР не виявлено.

Висновки. Ексцентричні вправи індукують оксидативний стрес і пошкодження м'язів на тлі зниження нервово-м'язової працездатності. Біохімічні маркери досягають пікових значень через 24 години з подальшою тенденцією до зниження, тоді як нервово-м'язове відновлення залишається незавершеним навіть через 72 години. Взаємозв'язок між оксидативним стресом і пошкодженням м'язів є часозалежним, проявляючись під час фази відновлення, а не в гострій фазі. Отримані результати вказують на відмінності в часових закономірностях і свідчать про те, що оцінювати нервово-м'язову працездатність виключно за біохімічними маркерами є недостатнім.

Ключові слова: ексцентричні вправи, оксидативний стрес, креатинкіназа, малоновий діальдегід, нервово-м'язова працездатність, відновлення.

Information about the authors:

Irawan, Roy Januari: royjanuardi@unesa.ac.id; <https://orcid.org/0000-0002-0996-8718>; Sports Science Department, Sports Science and Health Faculty, Universitas Negeri Surabaya, Kampus FIKK-Unesa Jl. Lidah Wetan, Surabaya 60213, East Java, Indonesia.

Dewi, Ratna Candra: ratnadewi@unesa.ac.id; <https://orcid.org/0000-0003-0612-244X>; Sports Science Department, Sports Science and Health Faculty, Universitas Negeri Surabaya, Kampus FIKK-Unesa Jl. Lidah Wetan, Surabaya 60213, East Java, Indonesia.

Bakti, Ananda Perwira: anandabakti@unesa.ac.id; <https://orcid.org/0000-0002-8778-8114>; Sports Science Department, Sports Science and Health Faculty, Universitas Negeri Surabaya, Kampus FIKK-Unesa Jl. Lidah Wetan, Surabaya 60213, East Java, Indonesia.

Rimawati, Nanda: nandarimawati@unesa.ac.id; <https://orcid.org/0000-0001-8400-492X>; Sports Science Department, Sports Science and Health Faculty, Universitas Negeri Surabaya, Kampus FIKK-Unesa Jl. Lidah Wetan, Surabaya 60213, East Java, Indonesia.

Tualeka, Abdul Rohim: abdul-r-t@fkm.unair.ac.id; <https://orcid.org/0000-0002-8276-2441>; Department of Occupational Safety and Health, Faculty of Public Health, Universitas Airlangga, Fakultas Kesehatan Masyarakat Kampus C Unair, Mulyorejo, Kec. Mulyorejo, Surabaya 60115, East Java, Indonesia.

Alayyannur, Putri Ayuni: putri.a.a@fkm.unair.ac.id; <https://orcid.org/0000-0001-8701-6547>; Department of Occupational Safety and Health, Faculty of Public Health, Universitas Airlangga, Fakultas Kesehatan Masyarakat Kampus C Unair, Mulyorejo, Kec. Mulyorejo, Surabaya 60115, East Java, Indonesia.

Cite this article as: Irawan, R., Dewi, R. C., Bakti, A. P., Rimawati, N., Tualeka, A. R., & Alayyannur, P. A. (2026). Phase-Dependent Interaction Between Oxidative Stress and Muscle Damage During Acute Recovery Following Eccentric Exercise. *Physical Education Theory and Methodology*, 26(3), 590-600. <https://doi.org/10.17309/tmfv.2026.3.20>

Received: 20.04.2026. Accepted: 10.05.2026. Published: 30.05.2026

This work is licensed under a Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0>)