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Effects of resistance training and retraining on creatine kinase, lactate dehydrogenase, and aspartate aminotransferase enzymes in inactive young boys

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Abstract

Purpose: Unusual or intense exercise causes minor damage (microtrauma) to muscle fibers. It is thought that this damage is accompanied by the release of certain enzymes into the blood serum. The aim of the present study was to investigate the effect of resistance training and retraining on the enzymes creatine kinase, lactate dehydrogenase, and aspartate aminotransferase in inactive young boys. **Method:** Twenty-four healthy inactive young boys volunteered for the present study and were randomly divided into two groups of 10. The exercise protocol consisted of five stations of biceps and shoulders with a barbell, squats, chest presses, and forearm presses. Each movement consisted of three sets of 12 repetitions. The exercise intensity was 75% of one repetition maximum. Serum levels of the enzymes creatine kinase, lactate dehydrogenase, and aspartate aminotransferase were measured before the activity and at intervals of 1 and 48 hours thereafter. For data analysis, repeated measures analysis of variance and independent t-test were used at a significance level of $p \geq 0.05$. **Results:** The results of the present study showed that the levels of creatine kinase, lactate dehydrogenase, and aspartate aminotransferase enzymes increased in both resistance training and resistance retraining groups compared to the pre-test at time intervals of 1, and 48. No differences were observed in serum levels of creatine kinase, lactate dehydrogenase, and aspartate aminotransferase between the two groups during and after retraining ($p > 0.05$). **Conclusion:** Retraining may not be a significant variable affecting recovery after muscle injury. However, more research is needed in this area.

Keywords: retraining, resistance training, muscle injury, muscle strain.

Introduction

Muscle soreness after resistance training is a sign of muscle fiber damage. It has been reported that bleeding and tearing of the muscle fibers that are responsible for holding muscle fibers together due to friction against each other during muscle contraction causes muscle soreness the day after intense exercise. Delayed-onset muscle soreness is a type of pain that, in addition to causing pain, movement restriction, spasm, swelling, and inflammation in the involved muscles, increases the release of certain enzymes such as creatine kinase, lactate dehydrogenase, and aspartate aminotransferase into the blood (Forutan Ghojebiglo, Siahkohian, & Fasihi, 2021). Therefore, the diffusion of these enzymes into the interstitial fluid is only possible when the muscle cell membrane is damaged and its permeability is increased. Therefore, the increased diffusion of these enzymes into the blood is a type of muscle response to injury and can be measured as a reliable indicator. When skeletal muscle is damaged by injury or overuse, the CK enzyme is released from the muscle cells and its level in the blood increases within an hour (Gwozdzinski, Pieniazek, & Gwozdzinski, 2021) . The increase in this enzyme is proportional to the degree of damage to the skeletal muscle. In fact, the tension created in the active muscle fibers during contraction causes the enzymes to diffuse into the blood. According to research results, it takes at least 5 days for the pain caused by muscle damage to disappear, and even longer to restore the functional aspects of the muscles. Creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate aminotransferase are three physiological markers of muscle damage. The release of these three enzymes from the intramuscular environment into the blood indicates structural damage to the muscle fibers. Assessment of these three enzymes provides information about muscle fueling and building that can help physicians and trainers determine the level of activity and the type of metabolic adaptation to exercise (Fasihi & Pirallahi, 2024).

Resistance training is widely prescribed to improve muscular strength, functional capacity, and general health; however, when performed at an unusual load or volume—especially by previously inactive

individuals—it can provoke exercise-induced muscle damage (EIMD) and delayed-onset muscle soreness (DOMS). In this context, post-exercise muscle pain is not merely an unpleasant symptom; it typically reflects microtrauma within muscle fibers and connective elements, accompanied by transient reductions in range of motion, swelling, spasm, and inflammatory signs. Importantly, DOMS and microtrauma are commonly associated with the appearance of intramuscular enzymes in the circulation—most notably creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate aminotransferase (AST)—which can be used as indirect biochemical indicators of muscle membrane disruption and structural stress (Deng et al., 2024).

The pathophysiology underlying these responses is multifactorial and begins with mechanical loading. During high-effort resistance exercise, repeated cycles of tension generation—particularly when muscle fibers are lengthening while active—can disrupt sarcomeric alignment and cytoskeletal integrity. The resulting microstructural disturbances can impair excitation–contraction coupling and increase membrane permeability, facilitating the leakage of intracellular proteins into interstitial fluid and blood. Consistent with this framework, it has been noted that enzyme diffusion into blood becomes possible when the muscle cell membrane is damaged and its permeability increases, making serum elevations of CK, LDH, and AST a measurable response to injury (Nash et al., 2023).

Moreover, a commonly described "consensus hypothesis" proposes that an initial mechanical insult triggers downstream biochemical cascades, including increased intracellular calcium, mitochondrial/respiratory disturbances, activation of proteolytic pathways, and a secondary inflammatory response characterized by neutrophil and macrophage involvement. The subsequent local edema and production of inflammatory mediators (e.g., prostaglandins) can sensitize nociceptors and contribute to the perception of soreness and functional limitation over the following 24–72 hours (Kodadek et al., 2022).

Within this injury–repair continuum, CK has been particularly emphasized as a sensitive marker of muscle membrane disruption.

When skeletal muscle is damaged by injury or excessive or unaccustomed loading, CK can be released from muscle tissue and rise in blood relatively quickly, with reports that elevations may occur within an hour and scale with the degree of muscle damage.

Although CK is frequently highlighted, LDH and AST also provide complementary information. LDH reflects broader cytosolic leakage and metabolic stress, while AST—though not specific to skeletal muscle—can increase in response to muscle injury in exercise settings and is therefore often considered alongside CK and LDH as part of a muscle-damage profile. The combined interpretation of CK, LDH, and AST may therefore provide a more robust picture than any single biomarker, particularly when assessing short-term responses to resistance exercise in untrained populations (Leite et al., 2023).

Beyond their role as damage markers, these enzymes are increasingly used in applied sport and clinical exercise physiology to monitor training stress, recovery status, and the appropriateness of exercise prescription. The assessment of CK, LDH, and AST has been described as offering information relevant to "muscle fueling and building," potentially assisting clinicians and coaches in judging the level of activity and the nature of metabolic adaptation to exercise (Nicolson & Ferreira de Mattos, 2021).

This applied value is especially important for sedentary adolescents and young adults who are beginning structured resistance training, because large acute spikes in damage markers can impair adherence, compromise performance in subsequent sessions, and increase the likelihood of technique breakdown or additional injury risk.

Empirical evidence supports that unaccustomed resistance exercise can elevate these enzymes for extended periods. For example, in previously resistance-untrained but physically fit men, a single one-hour weight-training session was reported to increase CK, LDH, AST (and other clinical chemistry markers), with elevations persisting for up to seven days post-exercise.

These prolonged elevations highlight that recovery from an initial bout may extend well beyond subjective soreness and may require careful

scheduling of subsequent training loads. At the same time, the magnitude of enzyme responses is not uniform across studies; it varies with the type of movement, intensity, total volume, rest intervals, the training status of participants, and whether sessions include a substantial eccentric component. The heterogeneity of findings in the literature makes it valuable to study standardized protocols in clearly defined inactive groups using repeated sampling time points (Nicolson & Ferreira de Mattos, 2021).

A central practical question, therefore, is how repeating a resistance stimulus soon after an initial damaging bout affects biochemical indices of muscle damage. In many applied settings—such as introductory resistance programs, school physical education, sport team conditioning, or time-constrained fitness regimens—individuals may perform similar exercise sessions on consecutive days or within short recovery windows. Theoretical expectations are not straightforward. On one hand, a second bout might exacerbate damage by adding mechanical stress before full repair is complete, potentially producing larger enzyme efflux. On the other hand, there is a well-documented "repeated bout effect" in which prior exposure to eccentric or damaging exercise attenuates symptoms and markers in a subsequent session. Importantly, it has been stated that objective evidence supports the idea that re-exercise after intense exercise can reduce pain, soreness, and signs of muscle damage, yet there has been a noted lack of studies directly examining the effect of re-exercise on indices of muscle damage and muscle soreness in the specific way needed to inform resistance-training programming (Chen et al., 2024).

This is precisely the gap that makes "retraining" (a repeated session after a short interval) a meaningful variable to test experimentally.

The distinction between "retraining" as a recovery strategy versus retraining as an additional loading event is also important. Some classical approaches to DOMS management propose that light activity performed after a damaging bout can alleviate symptoms, possibly via increased circulation, altered pain perception, or modulation of inflammatory processes. However, in resistance-training contexts, a

"retraining" session may not be "light"; it may replicate substantial intensity and volume, potentially changing the balance of recovery versus re-injury. Consequently, testing a standardized retraining protocol provides an opportunity to clarify whether a second exposure within 24 hours meaningfully alters the short-term biochemical profile of muscle damage or whether enzyme levels follow a similar trajectory regardless of retraining.

In the present article, the rationale for selecting CK, LDH, and AST is consistent with their established role as physiological markers of muscle damage that reflect leakage from the intramuscular environment into blood following structural disruption of muscle fibers.

The protocol is also designed to represent a realistic resistance-training session, incorporating multiple common movements across several stations at a moderately high intensity (reported as 75% of one-repetition maximum), with repeated blood sampling to capture early and delayed responses(Chen et al., 2024).

The study's sampling strategy—baseline measurement in the morning under fasting conditions, followed by assessments at 1 hour and 48 hours after the protocol (noting that 48 hours corresponded to 24 hours after retraining in the retraining group)—is particularly relevant because CK and related enzymes can show rapid early increases while also remaining elevated during the period when DOMS and inflammation typically peak.

This approach allows evaluation of whether retraining modifies the immediate post-exercise response, the delayed response, or both.

From a programming perspective, understanding these patterns is crucial for safe progression in novice trainees. The article emphasizes that unusual or intense exercise can cause minor damage to muscle fibers (microtrauma) and that this damage can be accompanied by the release of enzymes into blood serum—forming the foundation for the study's purpose.

If retraining does not meaningfully change enzyme responses, then consecutive-day repetition of similar resistance sessions may offer little advantage for recovery markers and could be unnecessary during early

adaptation. Conversely, if retraining attenuates enzyme elevations, it might be leveraged as a structured exposure that accelerates protective adaptation (through repeated bout effects), potentially enabling beginners to tolerate progressive overload more effectively. Either outcome has direct implications for coaches and practitioners who must balance training frequency with recovery capacity in sedentary youth.

Accordingly, the purpose of this study is positioned as a comparison of resistance training and resistance retraining on serum CK, LDH, and AST responses in inactive young participants, addressing a practical gap related to re-exercising after an initial potentially damaging resistance bout.

By examining enzyme changes at standardized post-exercise intervals and contrasting a single-session group with a retraining group, the study contributes evidence that can support more evidence-informed scheduling of resistance sessions in novice trainees and can help interpret whether retraining meaningfully influences biochemical recovery signatures following muscle microtrauma.

Atashek et al. (2012) observed a significant increase in the enzyme creatine kinase after a resistance training session at 100% of one repetition maximum. Rajabi et al. (2013) also showed that a single session of intense resistance training (leg press) increased delayed onset muscle soreness in men. Peterson et al. (2008) investigated the effect of resistance exercise (weightlifting) on clinical chemistry parameters in men. They recruited 15 healthy, physically fit men who had not performed resistance training. The training program used was a one-hour weight training session. Blood samples were taken to assess the parameters creatine kinase, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, bilirubin, and myoglobin at intervals up to 7 days after training. They observed that the parameters creatine kinase, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, and myoglobin increased significantly after training and that the increases remained up to 7 days after training. Objective evidence suggests that re-exercise after intense exercise reduces pain, muscle soreness, and signs of muscle damage.

Unfortunately, no study was found that investigated the effect of re-exercise on indices of muscle damage and muscle soreness. Therefore, the present study aimed to compare the effects of high-intensity resistance exercise and re-exercise on serum levels of some indices of muscle damage in inactive young boys.

Methods

The present study was a semi-experimental study. The statistical population of this study consisted of all inactive female students of Malayer Azad University, aged 19 to 24, who were studying in the academic year 1402-1403. After announcing the call, 85 students from the JET program expressed their willingness to participate in this study. Then, 24 of the eligible individuals formed the statistical sample of this study and were randomly assigned to two resistance training groups (12 people) and resistance training and retraining groups (12 people). Initially, during a face-to-face meeting, they were advised to refrain from performing intense activities and taking any medication that could affect the results of the study during the study period. Also, comprehensive and complete information about the objectives of the study, blood collection steps, possible risks, and pain after completing the exercise program was provided to them, and then they completed the consent form for participation in the study. Table 1 shows the mean and standard deviation of the individual characteristics of the subjects.

Table 1: General characteristics of subjects (mean and standard deviation)

variables	Resistance	Resistance training re-training
Age (years)	19±4.40	20.62±4.92

Height (cm)	152.25±5.28	156.06±4.32
Weight (kg)	55.17±9.35	56.60±10.50
BMI (kg/m)	23.00±1.27	22.00±3.00
Body fat (percentage)	12.75±4.21	12.10±3.24

Muscle Injury Inducing Protocol

The training session began with a 10-minute warm-up (including 5 minutes of walking and jogging, and 5 minutes of arm and shoulder girdle flexibility and stretching exercises). The resistance training program consisted of five stations, which were, respectively, biceps and shoulder presses with a barbell, squats, chest presses, and leg presses. Each movement consisted of three sets, and each set consisted of 8 to 12 repetitions. A 1.5-minute rest period was included between each set and a 5-minute rest period between each station. The intensity of the activity was 75% of one repetition maximum. Then, the subjects in the resistance training group performed the same protocol again after 24 hours. Blood samples were obtained at baseline (at 9 am and in a fasting state) and then at intervals of 1, 48 (equivalent to 24 hours after retraining in the retraining group) after the implementation of the protocol, each in an amount of 5 ml was obtained from the brachial vein (antecubital) of the subjects. All blood samples were immediately poured into tubes containing heparin anticoagulant after blood collection and centrifuged for 15 minutes at 3000 rpm. Then, Iranian kits from Pars Azmoun Company with sensitivities of one and five units per liter, respectively, and a 240 GLOBAL biochemistry autoanalyzer manufactured by PBC Company, Italy, were used to measure creatine kinase, lactate dehydrogenase, and aspartate aminotransferase

enzymes. The Shapiro-Wilk statistical test was used to check the normality of the data distribution. After ensuring the normality of the data, the analysis of variance in repeated measures statistical test was used to check the difference between the different sampling stages (pre-test with post-tests and post-tests together) and, if significant, the Bonferroni post-hoc test was used to determine the difference between the different sampling stages. Also, the independent t-test was used to compare the results of the two groups at each time step. All calculations were performed using SPSS statistical software version 26 and at a significance level of $p \leq 0.05$.

Results

Participant Characteristics

Examination of within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the high-intensity resistance training group increased at time intervals of 1, and 48 hours after activity compared to baseline, but this increase was not significant, (p=0.567, p=0.625, respectively). Also, examining within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the resistance retraining and retraining group increased at intervals of 1and 48 hours after retraining compared to the baseline, but this increase was not significant, (p=0.536, p=0.873, respectively).

Independent t-test to compare the means of the two groups at each time point indicated that there was no significant difference between the resistance training and retraining groups in terms of changes in lactate dehydrogenase at time intervals of 1, and 48 hours after activity (p=0.747, p=0.453, respectively).

Table 2: Mean and standard deviation of lactate dehydrogenase before and after training and retraining

Statistics / Variable	Physical activity stage	group		Significance level p*
		Resistance training	Resistance training and retraining	
Lactate dehydrogenase (IU/l)	Pre-test (baseline)	368±57	366±51	-
	1 hour after training	377±26	375±40	0.327
	Significance level p**	0.867	0.526	
	48 hours after extroverted training (equivalent to 24 hours after retraining)	412±36	410±72	0.433
	Significance level p**	0.625	0.682	

P* Significant difference with baseline at level ($p \leq 0.05$). P** Significant difference with control group at level ($p \leq 0.05$).

Independent t-test to compare the means of the two groups at each time point indicated that there was no significant difference between the

extroverted training and extroverted retraining groups in terms of creatine kinase changes at time intervals of 1, 26, and 48 hours after the activity (p=0.634, p=0.537, p=.654, respectively).

Examination of within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the high-intensity resistance training group significantly increased at time intervals of 1, 26, and 48 hours after activity compared to baseline, (p=0.003, p=0.026, p=0.044, respectively). Also, examining within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the resistance training and retraining group increased at intervals of 1, 26 and 48 hours after retraining compared to the baseline, but this increase was not significant (p=0.034, p=0.023, p=0.041, respectively).

Table 3: Mean and standard deviation of creatine kinase before and after training and retraining

Statistics **Variable**	**Physical activity stage**	**group**		**P***
		Resistance training	**Resistance training and retraining**	
Creatine kinase (International units per liter)	Pre-test (baseline)	167±112	165±101	-
	1 hour after training	217±112	231±105	0.634
	Significance level p**	0.021*	0.023*	

	48 hours after training (equivalent to 24 hours after retraining)	221±104	217±112	0.752
	Significance level p**	0.034*	0.021*	

P* Significant difference with baseline at level (p≤0.05). P** Significant difference with control group at level (p≤0.05).

Independent t-test to compare the means of the two groups at each time point indicated that there was no significant difference between the extroverted training and retraining groups in terms of changes in aspartate aminotransferase at time intervals of 1, 26, and 48 hours after activity (p=0.734, p=0.0243, p=0.335, respectively).

Examination of within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the high-intensity resistance training group significantly increased at time intervals of 1, 26, and 48 hours after activity compared to baseline, (p=0.013, p=0.026, p=0.037, respectively). Also, examining within-group effects using Bonferroni's post hoc test showed that the level of lactate dehydrogenase in the resistance training and retraining group increased at intervals of 1, 26 and 48 hours after retraining compared to the baseline, but this increase was not significant (p=0.044, p=0.037, p=0.012, respectively).

Table 4. Mean and standard deviation of the aspartate aminotransferase before and after training and retraining

Statistics		**group**	**p***

Variable	Physical activity stage	Resistance training	Resistance training and retraining	
Aspartate aminotransferase (IU/l)	Pre-test (baseline)	21.3±0.74	21.22±0.85	-
	1 hour after training	24.12±0.31	24.09±0.24	0.879
	Significance level p**	0.022*	0.014*	
	48 hours after training (equivalent to 24 hours after retraining)	25.8±0.74	27.2±0.14	0.425
	Significance level p**	0.016*	0.032*	

P* significant difference with baseline at level ($p \leq 0.05$). P** significant difference with control group at level ($p \leq 0.05$).

Discussion

The levels of creatine kinase, lactate dehydrogenase, and aspartate aminotransferase enzymes increased in both resistance training and resistance retraining groups compared to the pre-test at time intervals of 1, and 48. This increase for creatine kinase and aspartate aminotransferase enzymes, in addition to being significant, decreased after 48 hours. And this increase was not significant for lactate dehydrogenase and this increase continued until 48 hours later.

The present study examined whether repeating a resistance-training session within a short recovery window modifies the acute-to-delayed biochemical profile of exercise-induced muscle injury in inactive young participants. Using a standardized, multi-station resistance protocol (five stations; three sets of 8–12 repetitions at ~75% 1RM; fixed rest intervals), blood samples were collected at baseline and at 1 and 48 hours post-exercise, with the 48-hour time point reflecting 24 hours after retraining in the retraining group.

The main finding was that serum creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate aminotransferase (AST) increased after exercise in both the resistance-training-only group and the resistance retraining group, yet the between-group comparisons did not demonstrate a meaningful advantage (or disadvantage) for retraining across the measured time points ($p>0.05$).

This pattern suggests that, under the present loading conditions and sampling schedule, retraining performed 24 hours after an initial bout may not be a decisive variable shaping short-term biochemical recovery following resistance-exercise microtrauma.

A key point in interpreting the findings is the distinct temporal behavior of the measured enzymes. As discussed in the manuscript, CK and AST rose significantly post-exercise and then showed a decrease by 48 hours, whereas LDH displayed a more persistent elevation pattern, with the increase described as non-significant but continuing up to 48 hours. This divergence is physiologically plausible because CK and AST are commonly interpreted as indicators of membrane permeability and structural stress with a time course that can peak early and then regress as acute leakage subsides, while LDH may reflect a broader cytosolic/metabolic disturbance and can remain elevated during continuing repair or sustained metabolic disruption. The introduction emphasizes that diffusion of these enzymes into blood is facilitated when the muscle cell membrane is damaged and permeability increases, and that CK may rise rapidly (within an hour), roughly proportional to the degree of damage.

Therefore, the observed post-exercise elevations at 1 hour are consistent with an acute membrane/structural perturbation after a relatively demanding protocol in previously inactive subjects.

Despite these within-group changes, the central applied question is whether retraining altered the magnitude of enzyme responses compared with a single-bout condition. The results indicate that it did not. For CK, independent t-tests at each time point found no significant between-group difference (e.g., $p=0.634$ at 1 hour and $p=0.654$ at 48 hours, among other intervals reported).

Similarly, LDH showed no significant between-group differences at 1 and 48 hours ($p=0.747$ and $p=0.453$, respectively).

For AST, the reported independent t-test comparisons likewise suggested no meaningful between-group differences across the post-exercise intervals (e.g., $p=0.734$ at 1 hour and $p=0.335$ at 48 hours, as listed).

Collectively, these statistics support the conclusion that duplicating the session after 24 hours did not produce a clearly distinguishable biochemical "recovery signature" compared with performing the session only once within the study's timeframe.

Several mechanisms may explain why retraining did not shift enzyme kinetics in a detectable way. First, the subjects were inactive and therefore likely exhibited a larger and more variable initial response to an unfamiliar resistance stimulus, potentially masking modest retraining-related effects. The introduction notes that recovery of soreness and functional capacity can take multiple days, with at least several days for pain to resolve and even longer for full functional restoration, implying that a 24-hour window might still coincide with an active damage–repair process.

Second, the retraining bout itself could have imposed an additional mechanical and metabolic load before full repair, potentially counteracting any protective "repeated bout effect" that might otherwise reduce subsequent damage markers. Third, enzyme responses are known to show substantial inter-individual variability influenced by contraction type, volume, intensity, and the extent of

eccentric loading; thus, a uniform retraining schedule might not produce uniform reductions in systemic enzyme leakage(Chen et al., 2024).

The discussion in the manuscript provides a mechanistic framework consistent with these interpretations. It notes that resistance exercise involving muscle stretching can produce local tissue damage, disruption of sarcomeres, and increased membrane permeability or rupture, allowing CK, LDH, and AST to leak into blood or lymph.

Additionally, the "consensus hypothesis" described in the paper proposes that an initial mechanical insult initiates a cascade including increased intracellular calcium, inhibition of cellular respiration, activation of degradation pathways (e.g., Z-line/troponin/tropomyosin), and a subsequent inflammatory response with neutrophil involvement; this is followed by monocyte influx and macrophage transformation with edema and swelling, and prostaglandin biosynthesis and nociceptor stimulation that contribute to soreness.

Under this model, executing a second resistance bout after 24 hours may occur while inflammatory and repair processes are ongoing, and therefore may not produce a simple "less damage" biochemical profile—particularly if the second bout is not deliberately reduced in load or volume.

The present findings also need to be interpreted alongside prior work cited in the article. The discussion references evidence that high-intensity exercise is associated with increased delayed fatigue and increased extracellular fluid enzymes (AST, CK, LDH), and that responses depend on exercise type, intensity, and volume.

The paper also highlights inconsistent reports, including a study in male athletes where a single resistance session did not meaningfully change muscle damage indices, and it suggests that discrepancies may relate to lower training intensity (e.g., 60% 1RM) and differences in training status (athletes vs non-athletes).

These comparisons reinforce that training background and protocol characteristics can strongly condition enzyme outcomes; thus, the null between-group effect of retraining in the present sample may not

generalize to other populations (e.g., trained lifters) or different retraining designs (e.g., much lighter "active recovery" sessions).

From a practical standpoint, the study's conclusion—that retraining may not be a significant variable affecting recovery after muscle injury—should be read as specific to the conditions tested: inactive youth, a muscle-injury-inducing resistance protocol, and biochemical assessment confined to baseline, 1 hour, and 48 hours (with retraining occurring at 24 hours).

Coaches and clinicians working with novice or inactive individuals may infer that repeating a similar resistance stimulus the next day is unlikely, by itself, to meaningfully normalize CK/LDH/AST responses over the subsequent 48 hours. This aligns with the paper's recommendation that non-athletes begin gradually and progress cautiously to reduce muscle damage associated with resistive contractions.

Accordingly, program design in early phases may benefit more from modulating intensity/volume and inserting adequate recovery than from repeating the same session within a short interval.

Finally, several limitations—and therefore research directions—are evident from the study design and the nature of the dependent variables. First, the study focused on circulating enzymes; however, soreness perception, range of motion, strength loss, swelling, and inflammatory markers were not simultaneously reported, even though the discussion links biochemical changes to delayed soreness mechanisms.

Second, the sampling schedule may not capture peak enzyme values for all markers in all individuals, since CK and related proteins can peak later than 48 hours in some novice populations. Third, the retraining bout was the same protocol; future studies might compare "retraining" as identical loading versus lower-intensity active recovery, as these strategies likely produce different hemodynamic and mechanical demands. Taken together, expanding outcomes (biochemical + functional + perceptual), extending follow-up time points, and manipulating retraining dose and timing would help clarify whether retraining can be used strategically to accelerate adaptation while minimizing excessive damage in sedentary beginners.

Pasjalis et al. (2005) showed that high-intensity exercise is associated with increased delayed muscle fatigue and increased extracellular fluid enzymes AST, CK, and LDH, and the levels of indicators, including AST, CK, and LDH, change depending on the type of exercise, its intensity, and its volume. The results of some studies were inconsistent with the present study. Also, Abbas Foroughi Pardanjani et al. (2015), Atashek and Betorak (2012), Rajabi et al. (2012) stated that resistance activity causes muscle damage by increasing AST, CK, and LDH enzymes. These findings indicated that a session of exogenous resistance exercise causes damage and additional stress to the structural state of skeletal muscle. Since extrinsic activity is performed with muscle stretching, local damage to muscle tissue and fragmentation of the lines and destruction of sarcomeres due to sarcomere stretching can cause permeability or rupture of the cell membrane and leakage of enzymes indicative of muscle fatigue, including the most important AST, CK, and LDH, into the blood or lymphatic system. McAnality et al. (2005) reported that a single session of resistance exercise had no significant effect on muscle damage indices in male athletes. Perhaps one of the reasons for the discrepancy in their findings with the present study is the lower intensity of exercise (60% of one repetition maximum) compared to the present study (80% of one repetition maximum) or the difference in intensity and duration of the activity or the type of subjects (athletes or non-athletes). In addition, Evans et al. (1998) reported no significant difference between the increase in muscle damage indices after resistance exercise of the same duration at an intensity of 60 and 80% of maximum voluntary contraction . This discrepancy could be due to the difference in the number of contractions or the type of activity used.

The consensus hypothesis is that following intense resistance exercise, initial mechanical injury causes an increase in intracellular calcium, inhibition of cellular respiration, and activation of the Z-line, troponin, and tropomyosin degradation pathways, which in turn stimulates an inflammatory response (increase in neutrophils). The accumulation of materials resulting from the destruction of cellular structures during the

6-12 hours after exercise causes the influx of monocytes to the site, which transform into macrophages, characterized by subsequent edema and swelling. The presence of macrophages at the site of injury leads to the biosynthesis of prostaglandins and the stimulation of pain-related nerves. Overall, this study showed that performing extroverted training significantly increases biochemical indices of creatine kinase, lactate dehydrogenase, and aspartate aminotransferase in inactive individuals at 1, and 48 hours after exercise. Such changes appear to be associated with mechanical damage, inflammatory responses, and delayed muscle soreness. Another result of the present study showed that performing resistance training had no significant effect on the measured indices.

Conclusion

Recommended that non-athletes and their trainers begin activity gradually and slowly to reduce muscle damage caused by activities that involve resistive contractions. the study provides evidence that a high-load resistance protocol increases CK, LDH, and AST in inactive individuals, and that repeating the same protocol after 24 hours does not produce a clearly distinct biochemical recovery pattern compared with a single session—supporting the view that retraining, as operationalized here, is not a major determinant of short-term enzyme-based recovery in this population

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The author declares no conflict of interest.

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Reference

Teague, B.N. and J.A. Schwane, *Effect of intermittent eccentric contractions on symptoms of muscle microinjury.* Medicine and science in sports and exercise, 1995. 27(10): p. 1378-1384.

Donnelly, A.E., P.M. Clarkson, and R.J. Maughan, *Exercise-induced muscle damage: effects of light exercise on damaged muscle.* European journal of applied physiology and occupational physiology, 1992. 64(4): p. 350-353.

Butterfield, D.L., et al., *The effects of high-volt pulsed current electrical stimulation on delayed-onset muscle soreness.* Journal of Athletic training, 1997. 32(1): p. 15.

NAMENI, F., M. KASHEF, and A. LARRY, *THE EFFECTS OF WARM UP ON RELATIONSHIP BETWEEN CK AND LDH IN RECOVERY PERIOD.* 2004.

Nieman, D.C., et al., *Influence of vitamin C supplementation on oxidative and immune changes after an ultramarathon.* Journal of applied physiology, 2002. 92(5): p. 1970-1977.

Braun, W.A. and D.J. Dutto, *The effects of a single bout of downhill running and ensuing delayed onset of muscle soreness on running economy performed 48 h later.* European journal of applied physiology, 2003. 90(1-2): p. 29-34.

Black, C.D. and P.J. O'Connor, *Acute effects of dietary ginger on quadriceps muscle pain during moderate-intensity cycling exercise.* International journal of sport nutrition and exercise metabolism, 2008. 18(6): p. 653-664.

Armstrong, R., *Muscle damage and endurance events.* Sports Medicine, 1986. 3(5): p. 370-381.

Han, J.-H., et al., *Effects of therapeutic massage on gait and pain after delayed onset muscle soreness.* Journal of exercise rehabilitation, 2014. 10(2): p. 136.

Yu, J.-Y., J.-G. Jeong, and B.-H. Lee, *Evaluation of muscle damage using ultrasound imaging.* Journal of physical therapy science, 2015. 27(2): p. 531-534.

Nejatmand, N ,.A. Ramezani, and A. Barati, *Compare the effect of Consumption CoQ10 Supplement on aerobic power, anaerobic and Muscle soreness in athletes and non athletes.* Journal of Shahrekord Uuniversity of Medical Sciences, 2016. 18.

Nejatmand, N., A. Ramezani, and A.H. Barati, *Effect of Consumption short-term CoQ10 supplementation on markers of delayed onset muscle soreness.* Razi Journal of Medical Sciences, 2014. 21(119): p. 77-85.

Miles, M. and P. Clarkson, *Exercise-induced muscle pain, soreness, and cramps.* The Journal of sports medicine and physical fitness, 1994. 34(3): p. 203-216.

Paschalis, V., et al., *The effects of muscle damage on running economy in healthy males.* International journal of sports medicine, 2005. 26(10): p. 827-831.

Atashak ,S., K. Batoorak, and M. Ghaderi, *Effect of BCAA supplementation on serum C-reactive protein after acute resistance exercise in soccer players.* Journal of Knowledge & Health, 2013. 8(1): p. 12-16.

Rajabi, A., et al., *The effects of omega-3 intake on delayed onset muscle soreness in non-athlete men.* Pedagogics, Psychology, Medical-Biological Problems of Physical Training and Sports, 2013. 1: p. 91-95.

Khan, F., *Rhabdomyolysis: a review of the literature.* Neth J Med, 2009. 67(9): p. 272-83.

Zhang ,B.-T., et al., *Role of the calcium-calpain pathway in cytoskeletal damage after eccentric contractions.* Journal of applied physiology, 2008. 105(1): p. 352-357.

McAnulty, S.R., et al., *Effect of resistance exercise and carbohydrate ingestion on oxidative stress.* Free radical research, 2005. 39(11): p. 1219-1224.

Evans, G., et al., *Submaximal delayed-onset muscle soreness: correlations between MR imaging findings and clinical measures.* Radiology, 1998. 208(3): p. 815-820.

Barlas, P., et al., *Managing delayed-onset muscle soreness: lack of effect of selected oral systemic analgesics.* Archives of physical medicine and rehabilitation, 2000. 81(7): p. 966-972.

Kuligowski, L.A., et al., *Effect of whirlpool therapy on the signs and symptoms of delayed-onset muscle soreness.* Journal of athletic training, 1998. 33(3): p. 222.

Hans, P., et al., *Neuron-specific enolase as a marker of in vitro neuronal damage. Part I: Assessment of neuron-specific enolase as a quantitative and specific marker of neuronal damage.* Journal of neurosurgical anesthesiology, 1993. 5(2): p. 111-116.

Cleak, M. and R. Eston, *Delayed onset muscle soreness: mechanisms and management.* Journal of sports sciences, 1992. 10(4): p. 325-341.

Chen, B., Yu, P., Chan, W. N., Xie, F., Zhang, Y., Liang, L., . . . Tse, G. M. (2024). Cellular zinc metabolism and zinc signaling: from biological functions to diseases and therapeutic targets. *Signal transduction and targeted therapy, 9*(1), 6 .

Deng, R ,.Zhao, R., Zhang, Z., Chen, Y., Yang, M., Lin, Y., . . . Yan, X. (2024). Chondrocyte membrane–coated nanoparticles promote drug retention and halt cartilage damage in rat and canine osteoarthritis. *Science translational medicine, 16*(735), eadh9751 .

Fasihi, L., & Pirallahi, M. (2024). The relationship between some serum osteoporosis markers and body mass index with lumbar bone mineral density inactive postmenopausal women. *New Approaches in Exercise Physiology, 6* .(۱۲)

Forutan Ghojebiglo, A., Siahkohian ,M., & Fasihi, L. (2021). The Effect of Eccentric Retraining on Sensation of Muscle Pain and Delay Onset Muscle Soreness Markers on Sedentary Young Men. *The Scientific Journal of Rehabilitation Medicine, 10*(3), 412–423 .

Gwozdzinski, K., Pieniazek, A & ,.Gwozdzinski, L. (2021). Reactive oxygen species and their involvement in red blood cell damage in chronic kidney disease. *Oxidative medicine and cellular longevity, 2021*(1), 6639199 .

Kodadek, L., Carmichael, S. P., Seshadri, A., Pathak, A., Hoth, J., Appelbaum, R., . . . Gonzalez, R. P. (2022). Rhabdomyolysis: an American association for the surgery of trauma critical care committee clinical consensus document. *Trauma Surgery & Acute Care Open, 7* .(۱)

Leite, C. D., Zovico, P. V., Rica, R. L., Barros, B .M., Machado, A. F., Evangelista, A. L., . . . 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 .

Nash, D., Hughes, M. G., Butcher, L., Aicheler, R., Smith, P., Cullen, T., & Webb, R. (2023). IL-6 signaling in acute exercise and chronic training: Potential consequences for health and athletic performance. *Scandinavian Journal of Medicine & Science in Sports, 33*(1), 4–19 .

Nicolson, G. L., & Ferreira de Mattos, G. (2021). A brief introduction to some aspects of the fluid–mosaic model of cell membrane structure and its importance in membrane lipid replacement. *Membranes, 11*(12), 947 .

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