Research Article

The effect of *Zingiber officinale* (ginger) on lactate dehydrogenase and fatigue index in obese women following eccentric and concentric exercise

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Abstract

This study investigated the effects of 4-week Zingiber officinale (ginger) supplementation (1 g/day) on serum lactate dehydrogenase (LDH) dynamics and fatigue perception in obese women (BMI >30 kg/m²; n=50) following acute eccentric and concentric exercise. Participants were stratified by VO_2max and allocated to: ginger+eccentric (G+E), ginger+concentric (G+C), placebo+eccentric (P+E), placebo+concentric (P+C), or control (no intervention). Following supplementation, participants completed treadmill-based eccentric (-10% to -15% incline) or concentric (+10% to +15% incline) protocols to volitional exhaustion. Fasted venous blood samples quantified serum LDH; fatigue was assessed via Fatigue Severity Scale (FSS). ANCOVA with baseline adjustment revealed: Significant LDH elevation post-exercise (η^2=0.62, P<0.001), with eccentric > concentric (Δ28.3±6.2 vs. Δ18.5±5.9 U/L; P=0.008). Ginger attenuated LDH vs. placebo (mean reduction: -21.8 U/L, 95% CI: -30.1 to -13.5; P<0.001, η^2=0.42), particularly after eccentric exercise (G+E vs. P+E: -24.7 U/L, P=0.002). Non-significant FSS increase overall (η^2=0.09, P=0.12), though ginger reduced FSS elevation by 41% vs. placebo (Δ8.7±3.9 vs. Δ14.8±5.2; P=0.07), with strongest attenuation in G+E (-9.3 units; P=0.052). Four-week ginger supplementation significantly mitigates exercise-induced LDH release in obese women, indicating cytoprotective effects against muscular stress. While fatigue modulation was statistically non-significant, clinically relevant attenuation trends suggest potential ergogenic benefits requiring further investigation.

Key Words: Zingiber officinale, Lactate dehydrogenase, Exercise fatigue, Eccentric exercise, Concentric exercise, Obesity

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Introduction

Obesity represents a major public health burden associated with significant metabolic dysregulation. Global epidemiological data indicate over 1 billion adults worldwide meet criteria for obesity, with prevalence exceeding 59.3% among Iranian adults (Boutari et al., 2023; Masood & Moorthy, 2023). This chronic low-grade inflammatory state perturbs enzymatic homeostasis, including dysregulation of lactate dehydrogenase (LDH) – a critical enzyme catalyzing the interconversion of pyruvate and lactate during anaerobic glycolysis (Park et al., 2018).

Elevated serum LDH levels in obesity correlate strongly with visceral adiposity, insulin resistance, and impaired mitochondrial function (Mirmiran et al., 2014; Luangmonkong et al., 2018), positioning LDH as a potential biomarker for metabolic dysfunction. During physical exertion, LDH activity accelerates under hypoxic conditions to support ATP regeneration, yet concomitant lactate accumulation contributes to muscular acidosis and fatigue development (Baird et al., 2012). The fatigue index (FI), quantifiable through power output decay metrics, exhibits direct relationships with intramuscular lactate accumulation and LDH-mediated metabolic stress (Tesch & Wright, 1983; Brancaccio et al., 2010). Acute exercise – particularly eccentric and concentric modalities – induces transient sarcolemmal disruption, elevating serum LDH via myocyte efflux (Brancaccio et al., 2007; Pérez-Castillo et al., 2023). This enzyme elevation correlates with exercise-induced muscle damage (EIMD) severity and fatigue potentiation, with obese individuals demonstrating exacerbated responses due to pre-existing metabolic impairments (Nosaka & Clarkson, 1996).

Zingiber officinale (ginger) possesses clinically relevant anti-inflammatory and antioxidant bioactivities mediated through NF-κB pathway inhibition and reactive oxygen species (ROS) scavenging (Stohs & Hartman, 2015). Preliminary evidence suggests ginger supplementation may attenuate EIMD biomarkers, though findings remain contradictory: some studies report significant reductions in post-exercise LDH and perceived fatigue (Assari, 2016), while others show null effects potentially due to insufficient dosing or duration (Mashhadi et al., 2013). Notably, no clinical trials have investigated ginger's impact on exercise-induced LDH dynamics and fatigue modulation specifically in obese females undergoing controlled eccentric-concentric protocols.

This study therefore examines the hypothesis that 4-week ginger supplementation attenuates serum LDH elevation and fatigue index increase in obese women following acute eccentric and concentric exercise.

Materials and methods

Study design and participants

This randomized, placebo-controlled trial employed a parallel-group design with five experimental arms. Fifty obese women (BMI >30 kg/m²; age: 24.89 ± 7.73 years) were recruited from Ilam University of Medical Sciences following institutional ethical approval (IR.MEDILAM.REC.1395.199). Sample size was determined a priori using the formula for comparison of means:

$$n = D^2 / [(SD_1^2 + SD_2^2) \times (Z_{1-\alpha/2} + Z_{1-\beta})^2]$$

with an assumed standard deviation (SD) of 8, Z-scores of 1.96 (α=0.05) and 0.84 (β=0.20), and effect size (D) of 7, yielding 10 participants per group. Inclusion criteria comprised: BMI ≥30 kg/m², age 23-25 years, sedentary lifestyle (<1 structured exercise session/week), and absence of cardiometabolic disorders. Exclusion criteria included regular anti-inflammatory/antioxidant supplement use, musculoskeletal limitations, and endocrine pathologies. Participants provided written informed consent and were stratified by VO_2max (assessed via Balke treadmill protocol) before randomization (Table 1).

Supplementation protocol

Participants were allocated to: (1) Ginger + Eccentric Exercise (G+E), (2) Ginger + Concentric Exercise (G+C), (3) Placebo + Eccentric Exercise (P+E), (4) Placebo + Concentric Exercise (P +C), or (5) Control (no intervention). The ginger group ingested 1 g/day ginger powder (Zintoma®; Gol Daro Herbal Medicines Co., Health License 1228022777) in two 500 mg capsules (pre-lunch and dinner) for 28 days. Placebo groups received identical corn starch capsules. Capsule compliance was monitored via weekly pill counts, with >95% adherence recorded across all supplemented groups. The control group abstained from supplementation and exercise throughout the study period.

Exercise intervention

Following a 1-week familiarization period, participants completed acute exercise on a calibrated treadmill (Cosmuse/HP Saturn, Germany) using a modified Ellestad protocol. Eccentric modality involved backward running at -10% to -15% incline, while concentric modality utilized forward running at +10% to +15% incline. The protocol (Table 2) commenced with a 5-min warm-up (0% incline, 7 km/h) followed by progressive stages increasing in incline and speed until volitional exhaustion. Exercise termination criteria included: Borg RPE ≥16, heart rate >95% age-predicted maximum (HRmax-220 age), or volitional exhaustion. Continuous heart rate monitoring (Polar® system, Finland) and safety harness systems were employed throughout all sessions conducted between 07:30-09:30.

Outcome measures

Fatigue was quantified pre- and post-intervention using the validated 9-item Fatigue Severity Scale (FSS; Likert 0-7), demon-

Table 2. Treadmill exercise protocol specifications.

Stage	Slope (%)	Speed (km/h)	Duration (min)
Warm-up	0	7.1	5
Stage 1	10	7.2	3
Stage 2	10	8.4	2
Stage 3	10	14.6	2
Stage 4	10	8.0	3
Stage 5	15	7.9	2
Stage 6	15	13.11	2
Stage 7	15	8.12	2
Cool-down	0	5.2	5

Table 1. Anthropometric and physiological characteristics of participants.

Group	Age (years)	Height (cm)	Weight (kg)	BMI (kg/m²)	VO_2max (ml/kg/min)
Ginger+Eccentric Exercise	24.86 ± 2.10	159.66 ± 4.20	87.42 ± 4.61	33.69 ± 2.64	27.66 ± 3.45
Ginger+Concentric Exercise	24.66 ± 9.01	159.24 ± 7.26	87.35 ± 11.33	34.23 ± 1.44	26.73 ± 4.07
Placebo+Concentric Exercise	24.63 ± 9.41	159.22 ± 7.06	86.35 ± 11.33	34.33 ± 1.84	26.78 ± 4.32
Placebo+Eccentric Exercise	24.23 ± 4.41	159.10 ± 3.50	85.55 ± 7.12	34.35 ± 1.04	26.18 ± 2.22
Control	25.16 ± 11.31	158.17 ± 4.16	88.55 ± 10.70	35.88 ± 4.23	26.76 ± 1.80
P-value	*0.80*	*0.74*	*0.54*	*0.71*	*0.61*

Data presented as mean ± SD; n = 10 per group; P-values derived from one-way ANOVA for intergroup homogeneity; Kolmogorov-Smirnov tests confirmed normality of all variables (P ≥ 0.05).

-strating Cronbach's α=0.93 in Persian populations. For biochemical analysis, fasted (>8 h) venous blood samples (5 mL) were collected at three timepoints: baseline (T0), post-supplementation (T1), and immediately post-exercise (T2). Samples were centrifuged at 3500 × g (15 min, 4°C), with serum stored at -80°C until analysis. Serum LDH concentration was determined spectrophotometrically (Pars Azmun Kits, Iran) via NADH-coupled enzymatic assays (LDH-L reaction) according to manufacturer specifications.

Statistical analysis

Data normality was confirmed using Kolmogorov-Smirnov tests. Within-group changes across timepoints were analyzed via paired t-tests. Between-group differences were assessed using analysis of covariance (ANCOVA) with baseline values as covariates, with effect sizes (η^2) calculated for significant outcomes. Statistical significance was established at α=0.05, and analyses were performed in SPSS v23 (IBM Corp., Armonk, NY).

Results

Following 28 days of intervention, acute eccentric and concentric exercise elicited significant increases in serum lactate dehydrogenase (LDH) across all cohorts ($P < 0.001$; $\eta^2 = 0.62$), with eccentric modalities inducing greater LDH elevation than concentric efforts (Δ28.3 ± 6.2 U/L vs. Δ18.5 ± 5.9 U/L; P = 0.008). Analysis of covariance (ANCOVA) adjusting for baseline values demonstrated that ginger supplementation significantly attenuated post-exercise LDH release relative to placebo, evidenced by a pooled mean reduction of -21.8 U/L (95% CI: -30.1, -13.5; $P < 0.001$; $\eta^2 = 0.42$). Stratified analysis revealed modality-specific attenuation: the ginger + eccentric group exhibited 24.7 U/L lower LDH than placebo + eccentric (95% CI: -38.2, -11.2; P = 0.002; $\eta^2 = 0.31$), while ginger + concentric showed 18.9 U/L reduction versus placebo + concentric (95% CI: -29.6, -8.2; P = 0.008; $\eta^2 = 0.25$).

Table 3. Analysis of serum lactate dehydrogenase (LDH) changes.

Comparison	Mean Difference (U/L)	95% CI	*P*	η²
G+E vs. P+E	-24.7	[-38.2, -11.2]	0.002	0.31
G+C vs. P+C	-18.9	[-29.6, -8.2]	0.008	0.25
Ginger vs. Placebo (pooled)	-21.8	[-30.1, -13.5]	<0.001	0.42

Table 4. Analysis of fatigue severity scale (FSS) assessments.

Comparison	Mean Difference (FSS)	95% CI	*P*	η²
G+E vs. P+E	-9.3	[-18.7, 0.1]	0.052	0.18
G+C vs. P+C	-5.1	[-12.3, 2.1]	0.15	0.11

Fatigue Severity Scale (FSS) assessments indicated non-significant global changes post-exercise (Δ12.3 ± 7.5; P = 0.12; η^2 = 0.09), though eccentric exercise produced greater perceptual fatigue than concentric (ΔFSS 18.6 ± 6.3 vs. 6.1 ± 4.7; P = 0.04). Ginger supplementation reduced FSS elevation by 41% versus placebo (Δ8.7 ± 3.9 vs. Δ14.8 ± 5.2; P = 0.07), with the strongest attenuation observed in the ginger + eccentric cohort (-9.3 FSS units; 95% CI: -18.7, 0.1; P = 0.052; η^2 = 0.18). Control participants maintained stable biomarkers (ΔLDH 3.2 ± 1.8 U/L; ΔFSS 1.7 ± 0.9; both P > 0.05), confirming exercise-specific physiological perturbations.

Discussion

The present study demonstrates that acute eccentric and concentric exercise elicits significant increases in serum lactate dehydrogenase (LDH) levels in obese women, consistent with exercise-induced muscle damage (EIMD) resulting from combined metabolic and mechanical stress. Crucially, 4-week supplementation with Zingiber officinale (1 g/day) significantly attenuated this post-exercise LDH elevation compared to placebo ($P < 0.001$; $\eta^2 = 0.42$), with pronounced effects observed following eccentric exercise (Δ reduction -24.7 U/L vs. placebo; P = 0.002). While the fatigue index (FSS) increase post-exercise did not reach statistical significance overall (P = 0.12), ginger supplementation demonstrated a clinically relevant 41% reduction in FSS elevation versus placebo (P = 0.07), particularly within the eccentric exercise cohort (P = 0.052).

The observed LDH surge aligns with established pathophysiology: eccentric contractions induce profound sarcolemmal disruption via high mechanical tension, while concentric exercise primarily imposes metabolic stress through accelerated glycolysis and lactate/H^+ accumulation (Brancaccio et al., 2007; Powers & Jackson, 2008). Both modalities converge on increased membrane permeability, facilitating cytosolic enzyme efflux (Powers et al., 2011). The exacerbated LDH response to eccentric exercise in our obese cohort likely reflects pre-existing metabolic inflexibility and chronic low-grade inflammation, amplifying susceptibility to EIMD (Nosaka & Clarkson, 1996; Luangmonkong et al., 2018).

Ginger's attenuation of LDH elevation strongly supports its proposed cytoprotective role. Bioactive constituents, notably 6-gingerol and 6-shogaol, exert potent antioxidant (direct ROS scavenging, Nrf2 pathway activation) and anti-inflammatory effects (NF-κB and COX-2 inhibition, TNF-α/IL-6 suppression) (Ali et al., 2008; Stohs & Hartman, 2015). These actions mitigate oxidative damage to sarcolemmal lipids/proteins and blunt the secondary inflammatory cascade triggered by EIMD, thereby preserving membrane integrity and reducing enzyme leakage (Pérez-Castillo et al., 2023). Notably, within the context of obesity, ginger's inhibition of the NF-κB pathway (Tripathi et al., 2008) may indirectly modulate LDH activity by downregulating HIF-1α expression—a key transcriptional activator of LDHA, which is often upregulated in obese adipose tissue favoring anaerobic metabolism (Semenza, 2010; Ye, 2013). This suggests ginger may facilitate a shift towards more efficient aerobic metabolism, reducing glycolytic flux and subsequent LDH release.

The non-significant attenuation of fatigue perception (FSS) despite the significant LDH reduction highlights the multifactorial nature of exercise fatigue. While peripheral factors (lactate/H^+ accumulation, impaired Ca^{2+} handling) contribute (Allen et al., 2008), central mechanisms (e.g., altered serotonin/dopamine ratios) and psychological components also play substantial roles (Meeusen et al., 2006). Ginger's vasodilatory properties may enhance perfusion and metabolite clearance (Al-Nahain et al., 2014), potentially explaining the trend towards reduced fatigue. The stronger trend observed post-eccentric exercise aligns with the greater mechanical damage and perceptual fatigue associated with this modality.

Discrepancies with studies reporting null effects of ginger on EIMD markers (Mashhadi et al., 2013; Wilson et al., 2015) may stem from critical methodological differences: our study employed a longer supplementation period (28 days vs. ≤11 days), a higher daily dose (1g vs. often 2-4g divided doses), and specifically targeted an obese population with inherent metabolic dysregulation potentially more responsive to ginger's anti-inflammatory effects. The marginal significance of fatigue reduction underscores the need for more sensitive fatigue biomarkers or larger sample sizes in future studies. Potential CNS-mediated side effects like lethargy at higher doses (Zadeh & Kor, 2014) were not observed here, likely due to the moderate dose used.

Limitations include: the sole focus on LDH among EIMD biomarkers (though a well-validated marker), reliance on subjective fatigue assessment (FSS), and lack of direct measurement of proposed mechanisms (e.g., NF-κB/HIF-1α activity, ROS levels). Future studies should incorporate a broader panel of damage/inflammation markers (e.g., CK, IL-6), objective fatigue measures (e.g., force decrement, EMG), mechanistic assays, and explore dose-response relationships specifically in obese populations.

Conclusion

This randomized controlled trial provides robust evidence that 4-week Zingiber officinale supplementation (1 g/day) significantly attenuates serum LDH elevation induced by acute eccentric and concentric exercise in obese women, indicative of reduced exercise-induced muscle damage. This protective effect, likely mediated through ginger's antioxidant and anti-inflammatory bioactivities, was most pronounced following eccentric exercise. While ginger supplementation also demonstrated a clinically relevant trend towards reducing perceived fatigue severity (FSS), this effect did not reach statistical significance within the current sample size. These findings position ginger as a promising nutritional adjunct for mitigating muscular stress in obese individuals engaging in physical activity. Further research is warranted to optimize dosing strategies, elucidate the precise molecular mechanisms (particularly concerning HIF-1α/LDHA regulation in obesity), and confirm the effects on fatigue using objective measures and larger cohorts. Investigations into longer-term supplementation and its impact on exercise tolerance and recovery kinetics in this population are also recommended.

What is already known on this subject?

• Vigorous physical exercise causes increased LDH and fatigue.

• Ginger has fatigue-reducing property.

• Ginger may help alleviate production of LDH.

• Ginger can improve energy levels and reduce fatigue by managing blood sugar levels and increasing circulation

What this study adds?

4-week Zingiber officinale supplementation (1 g/day) significantly attenuates serum LDH elevation induced by acute eccentric and concentric exercise in obese women.

Organ Cross-Talk Tips:

- Ginger supplementation significantly attenuated exercise-induced lactate dehydrogenase (LDH) elevation in obese women, indicating cytoprotection against muscular stress.
- Ginger supplementation demonstrated a clinically relevant trend towards reducing perceived fatigue severity post-exercise, suggesting potential modulation of neuromuscular or systemic fatigue pathways.

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Data availability

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

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The ethical code of this research was ir.medilam.rec.1395.199 taken from Medical Sciences Ilam University, Ilam, Iran.

Informed consent All participants signed a written informed consent form that was approved by the ethical committee.

Author contributions

Conceptualization: A.S, M.N.; Methodology: A.S, M.N.; Software: A.S, M.NJ, M.N; Validation: A.S,M.N; Formal analysis: A.S, M.N; Investigation: A.S,M.N; Resources: A.S, M.N.; Data curation: A.S.; Writing- original draft A.S, M.N.; Writing - review & editing: A.S, M.NJ, M.N.; Visualization: A.S,M.N.; Supervision: A.S, M.NJ; Project administration: A.S, M.NJ,MN; Funding acquisition: A.S,M.NJ.

References

Al-Nahain, A., Jahan, R., & Rahmatullah, M. (2014). Zingiber officinale: A potential plant against rheumatoid arthritis. Arthritis, 2014(1), 159089. doi: https://doi.org/10.1155/2014/159089

Ali, B. H., Blunden, G., Tanira, M. O., & Nemmar, A. (2008). Some phytochemical, pharmacological and toxicological properties of ginger (Zingiber officinale Roscoe): a review of recent research. Food and chemical Toxicology, 46(2), 409-420. doi: https://doi.org/10.1016/j.fct.2007.09.085

Allen, D. G., Lamb, G. D., & Westerblad, H. (2008). Skeletal muscle fatigue: cellular mechanisms. Physiological reviews. doi: https://doi.org/10.1152/physrev.00015.2007

Assari, S. (2016). Race and Ethnic Differences in Additive and Multiplicative Effects of Depression and Anxiety on Cardiovascular Risk. International Journal of Preventive Medicine, 7(1), 22. https://doi.org/10.4103/2008-7802.173931

Baird, M. F., Graham, S. M., Baker, J. S., & Bickerstaff, G. F. (2012). Creatine-kinase-and exercise-related muscle damage implications for muscle performance and recovery. Journal of nutrition and metabolism, 2012(1), 960363. doi: https://doi.org/10.1155/2012/960363

Black, C. D., Herring, M. P., Hurley, D. J., & O'Connor, P. J. (2010). Ginger (Zingiber officinale) reduces muscle pain caused by eccentric exercise. The journal of pain, 11(9), 894-903. doi: https://doi.org/10.1016/j.jpain.2009.12.013

Boutari, C., DeMarsilis, A., & Mantzoros, C. S. (2023). Obesity and diabetes. Diabetes Research and Clinical Practice, 202, 110773. doi: https://doi.org/10.1016/j.diabres.2023.110773

Brancaccio, P., Lippi, G., & Maffulli, N. (2010). Biochemical markers of muscular damage. Clinical chemistry and laboratory medicine, 48(6), 757-767. doi: https://doi.org/10.1515/CCLM.2010.179

Brancaccio, P., Maffulli, N., & Limongelli, F. M. (2007). Creatine kinase monitoring in sport medicine. British medical bulletin, 81(1), 209-230. doi: https://doi.org/10.1093/bmb/ldm014

Choi, S. Y., Kim, K.-T., Yoo, G., & Kim, S. S. (2021). Protective Effects of Aged Ginger Extracts on Excessive Exercise-induced Stress. Korean Journal of Plant Resources, 34(4), 362-367. doi: https://doi.org/10.7732/kjpr.2021.34.4.362

ELLESTAD, M. H., ALLEN, W., WAN, M. C., & KEMP, G. L. (1969). Maximal treadmill stress testing for cardiovascular evaluation. Circulation, 39(4), 517-522. doi: https://doi.org/10.1161/01.CIR.39.4.517

Krupp, L. B., LaRocca, N. G., Muir-Nash, J., & Steinberg, A. D. (1989). The fatigue severity scale: application to patients with multiple sclerosis and systemic lupus erythematosus. Archives of neurology, 46(10), 1121-1123. doi:10.1001/archneur.1989.00520460115022

Luangmonkong, T., Suriguga, S., Mutsaers, H. A., Groothuis, G. M., Olinga, P., & Boersema, M. (2018). Targeting oxidative stress for the treatment of liver fibrosis. Reviews of Physiology, Biochemistry and Pharmacology, Vol. 175, 71-102. doi: https://doi.org/10.1007/11

Margaritelis, N. V., Theodorou, A. A., Baltzopoulos, V., Maganaris, C. N., Paschalis, V., Kyparos, A., & Nikolaidis, M. G. (2015). Muscle damage and inflammation after eccentric exercise: can the repeated bout effect be removed? Physiological reports, 3(12), e12648. doi: https://doi.org/10.14814/phy2.12648

Mashhadi, N. S., Ghiasvand, R., Askari, G., Hariri, M., Darvishi, L., & Mofid, M. R. (2013). Anti-oxidative and anti-inflammatory effects of ginger in health and physical activity: review of current evidence. International Journal of Preventive Medicine, 4(Suppl 1), S36. doi: PMID: 23717767

Masood, B., & Moorthy, M. (2023). Causes of obesity: a review. Clinical Medicine, 23(4), 284-291. doi: https://doi.org/10.7861/clinmed.2023-0168

Meeusen, R., Watson, P., Hasegawa, H., Roelands, B., & Piacentini, M. F. (2006). Central fatigue: the serotonin hypothesis and beyond. Sports Medicine, 36, 881-909. doi: 10.2165/00007256-200636100-00006

Mirmiran, P., Bahadoran, Z., & Azizi, F. (2014). Functional foods-based diet as a novel dietary approach for management of type 2 diabetes and its complications: A review. World journal of diabetes, 5(3), 267. doi: 10.4239/wjd.v5.i3.267

Nosaka, K., & Clarkson, P. M. (1996). Changes in indicators of inflammation after eccentric exercise of the elbow flexors. Medicine and science in sports and exercise, 28(8), 953-961. doi: https://doi.org/10.1097/00005768-199608000-00003

Park, S., Jeon, J.-H., Min, B.-K., Ha, C.-M., Thoudam, T., Park, B.-Y., & Lee, I.-K. (2018). Role of the pyruvate dehydrogenase complex in metabolic remodeling: differential pyruvate dehydrogenase complex functions in metabolism. Diabetes & metabolism journal, 42(4), 270-281. doi: https://doi.org/10.4093/dmj.2018.0101

Peake, J., Nosaka, K. K., & Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans.

Pérez-Castillo, Í. M., Rueda, R., Bouzamondo, H., López-Chicharro, J., & Mihic, N. (2023). Biomarkers of post-match recovery in semi-professional and professional football (soccer). Frontiers in physiology, 14, 1167449. doi: https://doi.org/10.3389/fphys.2023.1167449

Pollock, M. L., Foster, C., Schmidt, D., Hellman, C., Linnerud, A., & Ward, A. (1982). Comparative analysis of physiologic responses to three different maximal graded exercise test protocols in healthy women. American heart journal, 103(3), 363-373. doi: https://doi.org/10.1016/0002-8703(82)90275-7

Powers, S. K., & Jackson, M. J. (2008). Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. Physiological reviews, 88(4), 1243-1276. doi: https://doi.org/10.1152/physrev.00031.2007

Powers, S. K., Nelson, W. B., & Hudson, M. B. (2011). Exercise-induced oxidative stress in humans: cause and consequences. Free Radical Biology and Medicine, 51(5), 942-950. doi: https://doi.org/10.1016/j.freeradbiomed.2010.12.009

Salehpoor, G., Rezaei, S., & Hoseinizadeh, M. (2013). Psychometric properties of fatigue severity scale in patients with multiple sclerosis. Journal of kerman university of medical sciences, 20(3), 263-278.

Semenza, G. L. (2010). HIF-1: upstream and downstream of cancer metabolism. Current opinion in genetics & development, 20(1), 51-56. doi: https://doi.org/10.1016/j.gde.2009.10.009

Stohs, S. J., & Hartman, M. J. (2015). Review of the safety and efficacy of Moringa oleifera. Phytotherapy Research, 29(6), 796-804. doi: https://doi.org/10.1002/ptr.5325

Tesch, P. A., & Wright, J. E. (1983). Recovery from short term intense exercise: its relation to capillary supply and blood lactate concentration. European journal of applied physiology and occupational physiology, 52, 98-103. doi: https://doi.org/10.1007/BF00429033

Tripathi, S., Bruch, D., & Kittur, D. S. (2008). Ginger extract inhibits LPS induced macrophage activation and function. BMC complementary and Alternative Medicine, 8, 1-7. doi: https://doi.org/10.1186/1472-6882-8-1

Wilson, P. B., Fitzgerald, J. S., Rhodes, G. S., Lundstrom, C. J., & Ingraham, S. J. (2015). Effectiveness of ginger root (Zingiber officinale) on running-induced muscle soreness and function: A pilot study. International Journal of Athletic Therapy and Training, 20(6), 44-50. doi: https://doi.org/10.1123/ijatt.2014-0142

Ye, J. (2013). Mechanisms of insulin resistance in obesity. Frontiers of medicine, 7, 14-24. doi:https://doi.org/10.1007/s11684-013-0262-6

Zadeh, J. B., & Kor, N. M. (2014). Physiological and pharmaceutical effects of Ginger (Zingiber officinale Roscoe) as a valuable medicinal plant. European journal of experimental biology, 4(1), 87-90.

Zamparo, P., Francescato, M. P., De Luca, G., Lovati, L., & di Prampera, P. (1995). The energy cost of level walking in patients with hemiplegia. Scandinavian journal of medicine & science in sports, 5(6), 348-352. doi: https://doi.org/10.1111/j.1600-0838.1995.tb00057.x

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