High intensity physical activities increase troponin I level but does not affect changing in glutathione peroxidase in mice

Las actividades físicas de alta intensidad aumentan los niveles de troponina I pero no afectan los cambios en la glutatión peroxidasa en ratones

Authors

Moh Ali Ridho [1]
Nining Widyah Kusnanik [2]
Muhammad Ilham Mauluddin [3]
Bambang Purwanto [4]

[1] Universitas Airlangga (Indonesia)
[2,] Universitas Negeri Surabaya (indonesia)
[3] Universitas Airlangga (Indonesia)
[4] Airlangga University (Indonesia)

Corresponding author:
Bambang Purwanto
bambang-purwanto@fk.unair.ac.id

How to cite in APA

Ridho, M. A., Kusnanik, N. W., Mauluddin, M. I., & Purwanto, B. (2025). High intensity physical activities increase troponin I level but does not affect changing in glutathione peroxidase in mice. *Retos*, *65*, 437–444. https://doi.org/10.47197/retos.v65.110561

Abstract

Introduction: Muscle damage can be triggered by other free radicals formed during intense physical activites and the low response of antioxidants. The antioxidants that plays a role in responding to free radicals during physical activites is glutathione peroxidase.
Objective: The main purpose of this study was to analyze the effects of high-intensity and moderate-intensity physical activities on muscle glutathione peroxidase (GPx) and serum troponin I levels as markers of damage.
Methodology: The study was used a quantitative true experiment with a post-test only group design. This study was conducted at a total of 18 healthy mice with age of 8 weeks old male Balb/c mice, then divided into 3 groups: high-intensity physical activity (HI), moderate-intensity physical activity (MI), and control (CON) groups. The HI group performed running on a treadmill with a 90% of their maximum speed, while the MI group at 60%. Data was taken of 24 hours post-physical activity by taking blood samples to examine troponin I levels and skeletal muscle gastrocnemius homogenate to determine GPx activity.
Results: the study found that the MI group had a higher concentration of GPx in the skeletal muscle gastrocnemius compared to the other control group. However, the concentration of troponin I showed significant differences between the groups, HI group had significantly higher levels of troponin I compared to the other groups.
Conclusion: The high-intensity physical activity has the highest result in ncreasing Troponin I and the moderate intensity physical activity has the highest concentration of glutathione peroxidase compare to other groups.

Keywords

High intensity; moderate intensity; glutathione peroxidase; troponin I.

Resumen

Introduction: El daño muscular puede ser desencadenado por otros radicales libres formados durante actividades físicas intensas y la baja respuesta de los antioxidantes. El antioxidante que juega un papel en la respuesta a los radicales libres durante las actividades físicas es la glutatión peroxidasa.
Objective: El propósito principal de este estudio fue analizar los efectos de las actividades físicas de alta y moderada intensidad sobre la glutatión peroxidasa (GPx) muscular y los niveles de troponina I en suero como indicadores de daño.
Methodology: El estudio utilizó un experimento cuantitativo verdadero con un diseño de grupo con postprueba únicamente. Este estudio se llevó a cabo con un total de 18 ratones sanos machos Balb/c de 8 semanas de edad, que fueron divididos en 3 grupos: actividad física de alta intensidad (HI), actividad física de moderada intensidad (MI) y grupo de control (CON). El grupo HI realizó carreras en una caminadora a un 90% de su velocidad máxima, mientras que el grupo MI lo hizo al 60%. Los datos se tomaron 24 horas después de la actividad física, mediante la toma de muestras de sangre para examinar los niveles de troponina I y de homogeneizados del músculo esquelético gastrocnemio para determinar la actividad de la GPx.
Results: El estudio encontró que el grupo MI tenía una mayor concentración de GPx en el músculo esquelético gastrocnemio en comparación con el grupo de control. Sin embargo, la concentración de troponina I mostró diferencias significativas entre los grupos, siendo el grupo HI el que presentó niveles significativamente más altos de troponina I en comparación con los otros grupos.
Conclusion: La actividad física de alta intensidad mostró los resultados más altos en el aumento de la troponina I, mientras que la actividad física de moderada intensidad presentó la concentración más alta de glutatión peroxidasa en comparación con los otros grupos.

Palabras clave

Alta intensidad; intensidad moderada; glutatión peroxidasa; troponina I.

Introduction

There are two types of physical activities recommended for maintaining health: high-intensity and moderate-intensity. High-intensity physical activity can increase VO2max, quality of life, and metabolic health, but it also carries a greater risk of muscle injury compared to moderate-intensity activity. Moderate-intensity physical activity, on the other hand, is safer and still provides significant health benefits, including enhancing endogenous antioxidant activity (Kramps, Katle, & Cordova, Lane, 2021). High physical intensity is a method of physical activity that can enhance anaerobic capacity and athletes can apply high intensity cardio activity methods to develop muscle volume (Kusnanik., 2023). Studies have demonstrated that moderate-intensity physical activity boosts endogenous antioxidant activity, shields troponin I from free radicals, and lowers the risk of muscle injury. Endogenous antioxidant capacity doubles in individuals who regularly engage in moderate-intensity physical activity. High-intensity eccentric muscle contractions can cause exercise-induced muscle damage (EIMD) (Lazaro et al., 2020). Moderate-intensity physical activity also increases vagus nerve activity, reduces oxidative stress, and enhances learning (Kai et al., 2016; Kaidah et al., 2018).

Superoxide and other free radicals formed during intense physical activity can trigger muscle damage. Pain from physical activity-related muscle injuries commonly occurs and can result in an inability to resume training or competition (Delos et al., 2013). Physical activity performed beyond the body's adaptation threshold can induce stress with negative impacts (Kruk et al., 2019). These negative impacts include potential injuries leading to skeletal muscle damage (Taherkhani et al., 2021).

Troponin I serves as a marker of skeletal muscle damage due to physical stress during activity. Conduct testing for troponin I using appropriate instruments, methods, and samples to ensure accurate and rapid results. Free radicals can oxidize cysteine 133 residues on troponin I in skeletal muscle (Mollica et al., 2012). Increased free radicals lead to the massive release of skeletal muscle troponin I (sTnI) into the bloodstream (Purwanto et al., 2016). Troponin is a regulatory protein on thin filaments of muscle, comprising troponin T, I, and C. The release of sTnI into the extracellular space disrupts muscle contraction regulation, causing sustained intense contractions and muscle injury with severe pain.

Glutathione peroxidase (GPx) is a selenoenzyme that eliminates hydroperoxides within cells, protecting them from damage. The liver's cytosol predominantly contains this enzyme, which catalyzes the reduction reaction of H2O2 into ROOH. Glutathione is ubiquitous, found in animals, plants, and microorganisms, and exists in the forms of GSH (reduced) and GSSG (oxidized). GPx protects lipids from peroxidation, maintaining cell structure and function, particularly in the cytosol. Phospholipid glutathione peroxidase (PLGSH-Px), which reduces hydroperoxides from membrane phospholipids to alcohols, also contains selenium Se atoms. During muscle metabolism, mitochondria release superoxide as part of ATP production, triggering oxidative stress within contracting muscles. Superoxide and other free radicals oxidize troponin I's serine 133 residues. This stops actin and myosin from interacting, which causes the muscle to contract very strongly. Physical activity increases the production of endogenous antioxidants that prevent oxidative stress and cell damage. Elevated levels of endogenous antioxidants can prevent cell damage during physical activity. These antioxidants increase during physical activity and help protect cells from oxidative damage (Putri, 2018).

Extensive studies have not compared the effects of high-intensity and moderate-intensity physical activity on endogenous antioxidant glutathione and troponin I damage. Therefore, we need to investigate the effects of high-intensity and moderate-intensity physical activity on the activity of endogenous antioxidants and markers of muscle damage like troponin I. We expect this research to serve as a reference for implementing appropriate physical activity methods to maintain health and prevent free radical-induced muscle damage during physical activities.

Method

Study Design

This study is quantitative, true experimental research with a post-test-only group design. We randomly divided the mice in this study into three groups: a high-intensity activity group (HI), a moderate-intensity activity group (MI), and a control group without treatment. This research has received approval from the Research Ethics Committee of the Faculty of Medicine, Airlangga University, with approval number 153/EC/KEPK/FKUA/2023.

Subject

This study used 18 male Balb/c mice divided into 3 groups (6 HI, 6 MI, and 6 CON). Each group consisted of six mice acclimatized for 7 days with a reversed light-dark cycle. The inclusion criteria for this study included 8-week-old male Balb/c mice in good health

Intervention Procedures

Each group consisted of six Balb/c mice acclimatized for 7 days under a reversed light-dark cycle. The mice were then adapted to the treadmill for 7 days. In the research protocol, all groups underwent a 5-minute warm-up on the treadmill. Subsequently, the HI group engaged in high-intensity activity by running on the treadmill at 90% of the maximum treadmill speed. Meanwhile, the MI group received a moderate-intensity activity intervention by running on the treadmill at 60% of the maximum treadmill speed until the mice were fatigued. The control group, on the other hand, only underwent the warm-up and did not receive any physical activity treatment. This study conducted the physical activity treatment acutely, as a one-time trial.

Analysis

The Laboratory of Experimental Animals, Faculty of Veterinary Medicine, Airlangga University conducted examinations to determine the levels of troponin I and glutathione peroxidase (GPx) 24 hours after the intervention. We assessed the Troponin I levels by collecting mouse serum and then analyzed it using ELISA. In the meantime, we conducted a GPx examination on homogenized left gastrocnemius skeletal muscle tissue of the mice, utilizing the ELISA method.

Statistic Analysis

We used SPSS 25 software to analyze the data, which included descriptive analysis to determine the mean and standard deviation (SD), normality testing using the Shapiro-Wilk test, and inferential analysis. We used inferential analysis in this study to examine the differences in troponin I and GPx levels among the three groups. We assessed differences among groups using a one-way ANOVA test for normally distributed data. For data that did not follow a normal distribution, non-parametric analysis was conducted using the Kruskal-Wallis test with a significance level set at $p < 0.05$.

Results

Table 1 illustrates the effects of high-intensity (HI) and moderate-intensity (MI) physical activity on glutathione peroxidase (GPx) in mice. The results show that mice engaged in HI activity had a glutathione peroxidase activity of 71.308 ± 33.744 nmol/ml. Based on the Kruskal-Wallis test, there was no significant difference in glutathione peroxidase activity between three treatment groups ($p > 0.05$).

Table 1. Analysis of Glutathion Peroxidase (GPx)

Group	Glutathion peroxidase	Shapiro-Wilk	Kruskal Wallis
	Mean ± SD (nmol/ml)		
Control	32,552 ± 9,795	0,876	
HI	71,308 ± 33,744	0,020	0,106
MI	34,219 ± 13,218	0,065	

Table 2 presents the results of the analysis of troponin I for high-intensity (HI) and moderate-intensity (MI) physical activity in mice. The analysis showed that the HI activity group had a higher concentration serum of troponin I compared with control and the MI group. The HI activity group had a troponin I level of 535.133 ± 50.035 nmol/ml. Level concentration was higher compared to the MI group, which had a concentration of 367.575 ± 12.218 nmol/ml, and it also higher compare with control, which had a concentration of 338.911 ± 16.221 nmol/ml. Based on the one-way ANOVA test, there was a significant difference in serum troponin I level between three groups (p-value = 0.000).

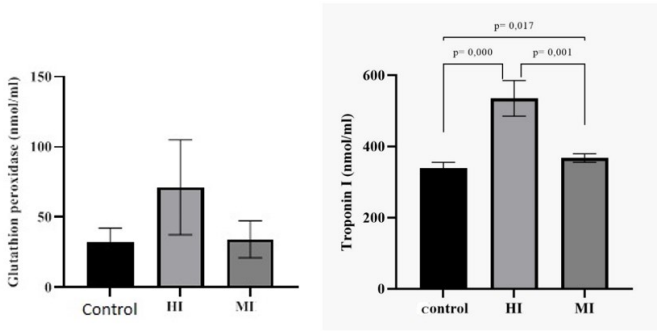
Table 2. Comparison of Serum Troponin I Levels

Group	Troponin I Mean ± SD (nmol/ml)	Shapiro-Wilk	ANOVA
Control	338,911 ± 16,221	0,871	
HI	535,133 ± 50,035	0,101	0,000*
MI	367,575 ± 12,218	0,057	

Information: SD: standar deviasi, HI: high intensity, MI: Moderate intensity, * significantly different (p<0,05)

Additionally, the post hoc analysis revealed significant differences in troponin concentrations between groups, with p-values of 0.000 for control vs. HI, 0.017 for control vs. MI, and 0.001 for HI vs. MI (Table 5.4). Figure 2 presents the differences in troponin I and GPx concentrations.

Figure 2. Differences in Troponin I and GPx Concentrations Among Groups



Discussion

The point of this study was to look at how high-intensity (HI) and moderate-intensity (MI) exercise affected the body's natural antioxidant glutathione peroxidase (GPx) and troponin I, which shows muscle damage in mice. The results indicate that the HI activity group had a higher concentration of GPx in the gastrocnemius muscle compared to both the MI activity group and the control group, although the difference was not statistically significant. In contrast, there was a significant difference in troponin I concentrations. Specifically, the MI activity group had higher troponin I concentrations compared to both the control group and the HI activity group 24 hours after physical activity.

It is known that GPx activity rises during physical activity, as seen in studies on both humans and animals (Leeuwenburgh & Heinecke, 2012). GPx protects against reactive oxygen species (ROS) that are created during intense physical activity. This finding is consistent with a study by Awang Daud, Ahmedy, Baharuddin, & Zakaria (2022), which indicates that high-intensity exercise carries a risk of tissue damage and contributes to excessive injury. Quindry et al. (2003) also found that high-intensity resistance training increases oxidative stress levels in the blood of untrained subjects. Additionally, not only the intensity and duration of exercise, but also the mode of exercise, influence the amount of oxidant production.

Another study, by Leeuwenburgh and Heinecke (2012), found that trained rats have 62% more glutathione peroxidase activity and 27% more superoxide dismutase activity than untrained rats. This is similar to the study by Revan et al. (2010), which found that short-term, exhausting running exercise did not significantly lower GPx levels. Additionally, Powers et al. (1993) found that the increase in muscle antioxidant enzymes caused by physical activity is muscle-specific. They also demonstrated that both

high-intensity and moderate-intensity exercise increase superoxide dismutase activity in the ventricular myocardium.

Two independent preclinical studies have found that high-intensity training (HIT) results in increased antioxidant enzyme activity in the skeletal muscles of rats. These studies indicate that HIT enhances total GPx activity in the skeletal muscles of trained rats subjected to HIT for 6 weeks (Scott K. Powers, Goldstein, Schrager, & Ji, 2023). To date, there are only a few human studies investigating the effects of HIT on skeletal muscle antioxidant enzyme activity. Results from these studies show that seven weeks of physical activity increased total GPx activity in skeletal muscle by 27%. This rise in GPx activity makes it easier for skeletal muscles to get rid of hydrogen peroxide and other organic hydroperoxides, especially when they are used for a long time (Hellsten et al., 1996).

Physical activity, particularly when involving unusual intensity and duration, can lead to increased production of free radicals and heightened oxidative stress, even in trained individuals. An imbalance between free radical production and antioxidant defenses can elevate oxidative stress levels. Physical activity enhances the absorption and flow of oxygen to the mitochondria, but beyond a certain intensity and/or duration, it can lead to oxidative stress. Several things might lead to more free radicals, such as: i) better blood flow; ii) faster phagocytic respiration; iii) xanthine oxidase; iv) catecholamines; v) inflammation after exercise; vi) lactate buildup; and vii) the type of exercise (Revan et al., 2010).

Perrone, Iellamo, Donatucci, Caminiti, & Lombardo (2020) also state that oxidative stress resulting from exercise depends on the capacity and adaptive ability of the body's antioxidant defenses. Although reactive oxygen species (ROS) generated by exercise are necessary for normal force production in skeletal muscles, high levels of ROS can contribute to contractile dysfunction. The Ca2+ release channels in the sarcoplasmic reticulum are highly sensitive to ROS, which can reduce the sensitivity of myofibrils to Ca2+ and subsequently affect muscle contraction. Additionally, the accumulation of ROS in the body depends on the mode of exercise, exercise intensity, and duration. Therefore, exercise-induced oxidative stress can play a significant role in the pathophysiological function of skeletal muscles (Wang et al., 2021).

Exercise is known to be a strong stimulus for elevating troponin I levels in the systemic circulation. The absence of a significant increase in troponin I in the high-intensity physical activity (HIT) group compared to the moderate-intensity exercise (MIE) group suggests that the total volume of HIT or HIE performed did not exert sufficient stress on the heart. Heart rate (HR) quickly increased during both HIT and MIE. However, HR quickly decreased afterward, which suggests that the total workload and oxygen demand on the heart muscles were not as high as they were in previous studies that used endurance exercise that lasted for hours, days, or even weeks (Shave et al., 2010).

Shave, Ross, Low, George, & Gaze (2010) found that short-duration, high-intensity physical activity can release troponin I. The release of troponin I after short-duration, intense exercise displays a varied release pattern. Some individuals may not show a response; some may release only limited amounts of troponin I, while others might exhibit a significant release that could exceed the threshold. Across different studies, there is considerable variability in how each factor affects troponin concentrations following physical activity. Intensity and duration of physical activity have the greatest impact on post-exercise troponin concentrations, likely reflecting the overall cardiac workload. Additionally, factors influencing post-exercise troponin I concentrations include cardiovascular health status, exercise history, hydration status, blood pressure, sex, body composition, age, intensity, duration, and environmental conditions (Aengevaeren et al., 2021). Other research also indicates that active recovery leads to lower cardiac troponin release after physical activity, which may be associated with improved aerobic capacity (Li et al., 2023).

Marshall et al. (2020) found that moderate (lactate threshold 60%–70% for 4 hours) and high-intensity (lactate threshold 80%–90% for 60 minutes) exercise increased troponin I levels, but low-intensity exercise (lactate threshold 50%–60% for 60 minutes) did not. This study conducted the sampling strategy 24 hours after physical activity, in line with Sorichter et al.'s (1997) findings that exercise-induced muscle damage leads to elevated troponin I levels that peak at 24 hours and stay elevated for at least 1 to 2 days afterward, mirroring changes in creatine kinase (CK) levels.

During high-intensity exercise, oxygen flow through skeletal muscle cells increases significantly, while ATP usage exceeds ATP production. Metabolic stress in cells causes many biochemical changes that

make the production of reactive oxygen species (ROS) from semiquinone and xanthine oxidase much higher. Consequently, substantial attacks by free radicals on cell membranes can lead to cell viability loss and cell necrosis, potentially triggering skeletal muscle damage and inflammation (Baker & Tao, 2022). Furthermore, increased free radical formation triggers a massive release of skeletal muscle troponin I (sTnI) into the extracellular space, leading to elevated blood levels (Purwanto et al., 2016). Actin continuously exposes itself to myosin heads as a result of the release of sTnI into the extracellular space, disrupting its role as a regulator of muscle contraction. This causes sustained high-intensity muscle contractions (hypercontraction), during which the muscle undergoes injury characterized by severe pain accompanying extreme contractions. This finding fits with what Weippert et al. (2016) found: there is a link between the release of troponin I and the state of metabolism in skeletal muscles in response to the amount of exercise. The finding suggests that peripheral anaerobic metabolism, which can change the acid-base balance and/or cause more oxidative radicals to be made, may be one reason why cardiomyocytes break down.

Conclusions

High-intensity physical activity resulted more haigher greater damage in calf muscle with compared moderate-intensity activity, due to serum troponin I level in treatment. It also the reased activity activity also increased the endogenous antioxidant activity of glutathione peroxidase in muscles. Therefore, we recommend to chooseng an appropriate activity intensity to minimize muscle damage and reduce the risk of exercise-related injuries

Acknowledgements

The author would like to thank the Dean of the Faculty of Medicine, Airlangga University, Prof. Dr. Budi Santoso, dr., Sp.O.G, Subps, F.E.R for providing the opportunity to conduct research. A big thank you to the supervisors Prof. Dr. Bambang Purwanto, dr., M.Kes. and Prof. Dr. Nining Widyah Kusnanik. M.Appl. Sc who guided and directed me in conducting the research and preparing this manuscript.

References

Daud, D. M. A., Ahmedy, F., Baharuddin, D. M. P., & Zakaria, Z. A. (2022). *Oxidative Stress and Antioxidant Enzymes Activity after Cycling at Different Intensity*.

Delos, D., Maak, T. G., & Rodeo, S. A. (2013). Muscle Injuries in Athletes. *Sports Health: A Multidisciplinary Approach*, *5*(4), 346–352. https://doi.org/10.1177/1941738113480934

Fernández-Lázaro, D., Mielgo-Ayuso, J., Seco Calvo, J., Córdova Martínez, A., Caballero García, A., & Fernandez-Lazaro, C. (2020). Modulation of Exercise-Induced Muscle Damage, Inflammation, and Oxidative Markers by Curcumin Supplementation in a Physically Active Population: A Systematic Review. *Nutrients*, *12*(2), 501. https://doi.org/10.3390/nu12020501

Hellsten, Y., Apple, F. S., & Sjödin, B. (1996). Effect of sprint cycle training on activities of antioxidant enzymes in human skeletal muscle. *Journal of Applied Physiology*, *81*(4), 1484–1487. https://doi.org/10.1152/jappl.1996.81.4.1484

Kai, S., Nagino, K., Ito, T., Oi, R., Nishimura, K., Morita, S., & Yaoi, R. (2016). Effectiveness of Moderate Intensity Interval Training as an Index of Autonomic Nervous Activity. *Rehabilitation Research and Practice*, *2016*, 1–4. https://doi.org/10.1155/2016/6209671

Kramps, K., & Lane-Cordova, A. (2021). High-intensity interval training in cardiac rehabilitation. *Sport Sciences for Health*, *17*(2), 269–278. https://doi.org/10.1007/s11332-021-00731-0

Kruk, J., Aboul-Enein, H. Y., Kładna, A., & Bowser, J. E. (2019). Oxidative stress in biological systems and its relation with pathophysiological functions: the effect of physical activity on cellular redox homeostasis. *Free Radical Research*, *53*(5), 497–521. https://doi.org/10.1080/10715762.2019.1612059

Kusnanik, N., Fajrin, F., Bird, S., Januarumi, F., Sudarso, S., Puteri, A., & Fenanlampir, A. (2021). The Effect of High Intensity Interval Training in Improving Aerobic Capacity. *Proceedings of the 5th International Conference on Sports, Health, and Physical Education, ISMINA 2021, 28-29 April 2021, Semarang, Central Java, Indonesia*. https://doi.org/10.4108/eai.28-4-2021.2312141

Leeuwenburgh, C., & Heinecke, J. (2001). Oxidative Stress and Antioxidants in Exercise. *Current Medicinal Chemistry*, *8*(7), 829–838. https://doi.org/10.2174/0929867013372896

Li, S., Shaharudin, S., Cirer-Sastre, R., Li, F., Abdul Manaf, F., & Mohd Shukri, M. F. (2023). Effects of high-intensity interval exercise on cardiac troponin elevation when comparing with moderate-intensity continuous exercise: a systematic review and meta-analysis. *PeerJ*, *11*, e14508. https://doi.org/10.7717/peerj.14508

Marshall, L., Lee, K. K., Stewart, S. D., Wild, A., Fujisawa, T., Ferry, A. V., Stables, C. L., Lithgow, H., Chapman, A. R., Anand, A., Shah, A. S. V., Dhaun, N., Strachan, F. E., Mills, N. L., & Ross, M. D. (2020). Effect of Exercise Intensity and Duration on Cardiac Troponin Release. *Circulation*, *141*(1), 83–85. https://doi.org/10.1161/CIRCULATIONAHA.119.041874

Mollica, J. P., Dutka, T. L., Merry, T. L., Lamboley, C. R., McConell, G. K., McKenna, M. J., Murphy, R. M., & Lamb, G. D. (2012). S-Glutathionylation Of Troponin I (Fast) Increases Contractile Apparatus Ca2+ Sensitivity In Fast-Twitch Muscle Fibres Of Rats And Humans. *The Journal of Physiology*, *590*(6), 1443–1463. https://doi.org/10.1113/jphysiol.2011.224535

Perrone, M. A., Iellamo, F., Donatucci, B., Caminiti, G., & Lombardo, M. (2020). Oxidative stress, redox state and antioxidant supplementation in physical exercise and professional sports: A brief review. *Acta Medica Mediterranea*, *36*(2), 1245–1251. https://doi.org/10.19193/0393-6384_2020_2_195

Powers, S. K., Criswell, D., Lawler, J., Martin, D., Lieu, F. K., Ji, L. L., & Herb, R. A. (1993). Rigorous exercise training increases superoxide dismutase activity in ventricular myocardium. *American Journal of Physiology-Heart and Circulatory Physiology*, *265*(6), 634–636. https://doi.org/10.1152/ajpheart.1993.265.6.H2094

Powers, S. K., Goldstein, E., Schrager, M., & Ji, L. L. (2022). Exercise Training and Skeletal Muscle Antioxidant Enzymes: An Update. *Antioxidants*, *12*(1), 39. https://doi.org/10.3390/antiox12010039

Purwanto, B. (2014). *Mechanism of Curcumin in Preventing Skeletal Muscle Damage in Mice During Acute Eccentric Exercise* [Unpublised doctoral thesis]. Universitas Airlangga.

Purwanto, B., Harjanto, & Sudiana, I. K. (2016). Curcuminoid Prevents Protein Oxidation but not Lipid Peroxidation in Exercise Induced Muscle Damage Mouse. *Procedia Chemistry*, *18*, 190–193. https://doi.org/10.1016/j.proche.2016.01.029

Putri, M. A. (2018). Increased Endogenous Antioxidants Triggered by Physical Activity. *Jurnal Kedokteran Yarsi*, *26*(3), 163–172.

Quindry, J. C., Stone, W. L., King, J., & Broeder, C. E. (2003). The Effects of Acute Exercise on Neutrophils and Plasma Oxidative Stress. *Med. Sci. Sports Exerc*, *35*, 1139–1145.

Revan, S., Balci, Ş. S., Pepe, H., Kurtoğlu, F., Erol, A. E., & Akkuş, H. (2010). Short duration exhaustive running exercise does not modify lipid hydroperoxide, glutathione peroxidase and catalase. *Journal of Sports Medicine and Physical Fitness*, *50*(2), 235–240.

Shave, R., Ross, P., Low, D., George, K., & Gaze, D. (2010). Cardiac troponin I is released following high-intensity short-duration exercise in healthy humans. *International Journal of Cardiology*, *145*(2), 337–339. https://doi.org/10.1016/j.ijcard.2009.12.001

Sorichter, S., Mair, J., Koller, A., Gebert, W., Rama, D., Calzolari, C., Artner-Dworzak, E., & Puschendorf, B. (1997). Skeletal troponin I as a marker of exercise-induced muscle damage. *Journal of Applied Physiology*, *83*(4), 1076–1082. https://doi.org/10.1152/jappl.1997.83.4.1076

Taherkhani, S., Valaei, K., Arazi, H., & Suzuki, K. (2021). An Overview of Physical Exercise and Antioxidant Supplementation Influences on Skeletal Muscle Oxidative Stress. *Antioxidants*, *10*(10), 1–16. https://doi.org/10.3390/antiox10101528

Tao, D., & Baker, J. S. (2022). High intensity exercise performance and muscle damage. A role for free radicals. *Journal of Clinical Images and Medical Case Reports*, *3*(12), 2189. https://doi.org/10.52768/2766-7820/2189

Wang, F., Wang, X., Liu, Y., & Zhang, Z. (2021). Effects of Exercise-Induced ROS on the Pathophysiological Functions of Skeletal Muscle. *Oxidative Medicine and Cellular Longevity*, *2021*(1), 3846122. https://doi.org/10.1155/2021/3846122

Authors' and translators' details:

Moh Ali Ridho	moh.ali.ridho-2021@fk.unair.ac.id	Author
Nining Widyah Kusnanik	niningwidyah@unesa.ac.id	Author
Muhammad Ilham Mauluddin	Muhammad.ilham.mauluddin-2021@fk.unair.ac.id	Author
Bambang Purwanto	bambang-purwanto@fk.unair.ac.id	Author