ISSN: 2586-7342 © 2020 KODISA & KJFHC
KJFHC website: http://www.kjfhc.or.kr
doi: http://dx.doi.org/10.13106/kjfhc.2021.vol7.no4.23.

Changes in Antioxidant Enzymes, According to Recovery Methods During Repeated Apnea Diving*

Hwang-woon MOON[1], Youn-sang SONG[2], Jeong-Weon KIM[3]

1. First Author Department of Sports & Outdoor, Eulji University, Seong Nam, Korea, E-mail: mhwgo21@eulji.ac.kr

2. Co- Author Department of Sports & Outdoor, Eulji University, Seong Nam, Korea, E-mail: sys3809@naver.com

3. Corresponding Author Graduate Schools of Professional Therapy, Gachon University, Seong Nam, Korea, E-mail: zeezone@gachon.ac.kr

Received: August 12, 2021. Revised: August 12, 2021. Accepted: September 03, 2021.

Abstract

This study analyzes the physiological changes that occur after free diving and studies peroxidants and antioxidants according to the recovery method. Accordingly, the purpose of this study is to provide proper rest methods after free diving and provide basic data for free diving research. Eight male college students recovered for 30 minutes after 20 free injections at a 5-meter diving site, 20 free injections after a week's car wash, and recovered for 30 minutes after collecting blood during recovery to analyze the effects of peroxidants and antioxidants. Comparison of changes in peroxidants and antioxidants in recovery methods after free-diving iterations showed that SOD tended to decrease immediately after free-diving, increasing MDA to 10 minutes after recovery, but no significant difference was found. The purpose of this study is to observe physiological changes according to the recovery method after free diving and to propose an appropriate recovery method after free diving. However, there was no significant difference in all the restoration methods, and 20 freediving was not high kinetic intensity for the subjects, which is believed to have resulted in the following results. Therefore, it was discussed that the repetitive diving strength should be higher to confirm a significant difference in the recovery method after free diving.

Keywords: free diving, SOD, MDA

Major classifications: diving physiology

---

* This study was supported by the research grant of the KODISA Scholarship Foundation in 2021.

1. Introduction

Humans have been collecting underwater for a long time to lead their lives, and it is now called 'FREE DIVING' that these activities have developed into sports. This has evolved into a sport that challenges and competes in the depth of human beings' ability to go through apnea. Free diving has thousands of recreational and elite divers, the most widely known of which are Korean and Japanese divers, called "AMA" (Linér, 1994). Women divers apnea a dive in South Korea and Japan became famous around the world, and they can hold its breath for up two minutes, down a depth of 25meters (Hong & Rahn, 1967; Schagatay, 2014).

Interested in free diving is increasing, and the depth of the water is rapidly deeper than before. Thus, research on the diving adaption mechanism of free divers is actively progressing. In particular, research has been conducted on respiratory and cardiovascular changes during free diving, MDR(mammal diving reflex) acting as an increase in bradycardia, peripheral vascular contraction and blood pressure during face flooding, these phenomenon suggested by research that the diving reactions of free divers are the same as those in diving mammals (Gooden, 1994; Ferretti & Costa 2003; Alboni, Alboni, & Gianfranchi, 2011). The physiological can changes that occur during free diving make it possible to dive deeper and longer.

The diving adaptation mechanism reduces acidity after apnea diving and reduces sensitivity to oxidative stress including respiratory and cardiovascular systems. Studies of trained free divers have reported that repeated hypoxiemia during long-term diving can induce metabolic adaptation, reducing blood acidity and oxidative stress. As a result, it is reported that the weakening of blood acidity and the generation of free diving activities lead to repeated adaptive mechanisms for free diving (Qvist, Hurford, Park, Radermacher, Falke, Ahn, & Weber,1993; Joulia, Steinberg, Wolff, Gavarry, & Jammes, 2002). Continuing free diving changes blood gas levels, in which changes in oxygen pressure exacerbate oxidative stress and cause the production of ROS (reactive oxygen species) that affect body function (Mrakic-Sposta, Vezzoli, Rizzato, Della Noce, Malacrida, Montorsi, Paganini, Cancellara, & Bosco, 2019).

Oxygen is essential for free diving, but active oxygen species, which are harmful oxygen due to metabolic activity using oxygen, are produced. This occurs oxidative tissue damage, and oxidative damage to various cells, including lipid peroxide, causes various dysfunctions and aging and diseases. As a result, our bodies will be able to defend it and improve antioxidant capacity to prevent tissue damage (Lee, Eun, & Im, 1997; Ji, Jeon, & Lee, 2006).

Antioxidants that can defend against ROS in the human body include Superoxide desmutase (SOD), catalse (CAT), and glutation pre-oxidase (GPX). Among them, SOD is most identified after exercise, and the first enzyme that activate an antioxidant reaction (Min, & Kim, 2019). SOD changes the cell's ROS into oxygen and hydrogen peroxide to remove peroxides anions (Bast & Goris, 1989). It is reported that it defends nucleic acid, protein and biomass damage caused by ROS (Bae, 2001; Urso & Clarkson, 2003). However, antioxidant enzyme's defense role is limited, so if the antioxidant's defense role is influenced by more active oxygen, it forms additional ROS in the cell membrane, combined with lipid components of the cell membrane in the body, which leads to changes in the membrane and protein, and increases the MDA (Malondialdehyde) which is the last product of peroxide (Kappus, 1985; Urso & Clarkson, 2003). MDA is known as an enzyme that restrict motor performance, and production of ROS and lipid peroxidation are reported to cause skeletal muscle damage (Lee, & Kim, 1999; Davies, Quintanilha, Brooks, & Packer, 1982). Therefore, the increase of MDA is known as an indicator of the oxidized lipid peroxidization index caused by ROS by acting like lactate acid (Jung & Chung, 1999).

Therefore, many studies have been conducted in terms of minimizing the harmfulness of ROS by increasing their antioxidant capacity with additional antioxidant supplements to prevent the made a lot of ROS in the body (Packer, 1991; Urso & Clarkson, 2003; Ha, 2006; Kim & Hong, 2012; Patlar, Baltaci, & Mogulkoc, 2016).

Various studies have been conducted due to increased interest in free diving, but most of them are based on response and adaptation studies to improve oxidative stress. In addition, research on post freediving recovery is insufficient, and most studies on post exercise recovery have been conducted, but none of the studies on post Freediving recovery underwater have been conducted (Park, Ku, & Lee, 2004; Hur, 2009; Hong, 2013). Therefore, it is time to study physiological changes according to the method of recovery after free diving, and this study showed to observe changes in lipid peroxidant and antioxidant enzymes according to the method of recovery after repeated free-diving and provide basic data on apnea diving.

2. Method

2.1. Subjects

This experiment was conducted on eight-male university students from E University, located in Gyeonggi Province.

Subjects had more than five diving experiences and had no resistance to water and diving. All subjects provided written informed consent and participated voluntarily. After confirming that those who have cardiopulmonary or cardiovascular diseases that could affect the free-diving process were excluded. The subject demographics of the study are shown in Table 1.

Table 1: physical characteristics of subjects

Variable	**Total (n=8)**
Sex	male
Age(years)	22.25±0.9
Height(cm)	177.81±2.1
Weight(kg)	83.07±4.8
Muscle mass(kg)	36.35±3.4
Body fat(%)	22.78±2.7
Visceral fat(%)	7.5±4.2
BMI(kg/m2)	27.5±4.4

M±SD

2.2. Research procedures

This study was conducted at a 5 meters deep A-submarine pool located in S-si, Gyeonggi-do, and pre-test was conducted to adapt to free diving before the experiment. For the accuracy of the experiments, a washout period was applied for a total of three measurements, including full recovery, activity recovery, and pre-test. The Subjects arrived at the diving pool 60 minutes before the experiment to provide sufficient stability, warm-up and stretching.

2.3. Experimental procedures

Subjects performed diving three times in total, with water temperatures of 28 degrees, at the same time and environment. Before free diving, a 3mm wetsuit was worn to protect the subject's temperature and wireless heart rate monitor. Subjects were stabilized for 30 minutes before the experiment. An apnea dive was performed at a depth of 5M for 30 seconds, and an apnea dive was performed at a total 20 times after 30 seconds of rest was calculated as a one-time dive. The method of 20 times apnea diving was designed by referring to the repeated apnea diving method of Kim et al. (2012). After diving, the full recovery group took a full rest underwater for 30 minutes, and the activity recovery group performed a 30-minute activity recovery method with an intensity of HRmax 60%, which is based on 40% of maximum oxygen intake (Kim, et al 2015; ACSM, 1991). Considering the safety of the subjects during the experiment, a professional diver equipped with scuba diving equipment went down to the water about 5 meters and waited to supervise the safety of the subjects.

2.4. Blood analysis method

Blood collection was measured MDA and SOD to examine the lipid peroxidant and antioxidant enzymes of the recovery before and after diving. The measurement period was taken at stabilization, before and after diving, 10 minutes and 30 minutes after recovery and blood collection of SOD and MDA was collected directly from the forearm vein before and after apnea with the nurse. After collecting 3.5 ml of whole blood in 3.5 ml of SST, it was immediately mixed for 5-10 times. After 30 minutes at room temperature, it was used at 3,000 RPM for 10 minutes using centrifuges. Then, 1.0 ml of the separated supernatant was separately collected and transported to N company located in Y, Gyeonggi Province.

2.5. Data analysis method

The data used IBM SPSS 25.0 to calculate the average (M) and standard deviation (SD) of measurement variables. The interaction effect of the recovery method and the number of free diving was verified by Two-way ANOVA with repeated measurement, the main effect verification was performed on variables with no interaction effect, and post analysis was performed by LSD method on variable with significant differences. The differences between the two groups according to each test time were verified by utilizing the independent t-test, and the one-way ANOVA for the repetition time of the individual group. Post-analysis was used the LSD method on variables with significant differences and the statistical significance level was p<.05.

3. Results

3.1. MDA change by measurement period and difference between groups

At full recovery, MDA was 74.83pmol/mL before diving, 79.13pmol/mL after diving, 86.01pmol/mL after 10 minutes of recovery, and 80.14pmol/mL after 30 minutes of recovery. Therefore, it tends to increase to 10 minutes of recovery and decrease to 30 minutes of recovery. At activity recovery, MDA was 58.68pmol/mL before diving, 63.15pmol/mL after diving, 76.39pmol/mL after 10 minutes of recovery, and 67.58pmol/mL after 30 minutes of recovery. Therefore, it tends to increase to 10 minutes of recovery and decrease to 30 minutes of recovery. Thus, there was no significant difference between the two groups. The independent t-test was conducted to determine the difference between full recovery and activity recovery in each period. The analysis always showed no statistically significant differences, with t=1.050, p=0.311, t=0.387, p=0.385, recovery 10 minutes, p=0.707, recovery 30 minutes t=0.282, and p=0.782 before diving (table 2).

Table 2: Changes in blood MDA

Variable	Group	BD	AD	R10	R30	f-value	p
MDA	FR	74.83 ±35.05	79.13 ±38.71	86.01 ±50.65	80.14 ±34.45	0.065	0.463
	AR	58.68 ±25.76	63.15 ±32.32	76.49 ±48.56	67.58 ±30.83	2.324	0.089
t-test	t-value	1.050	0.897	0.384	0.282	-	-
	p	0.311	0.385	0.707	0.782		

M±SD
BD : Before Diving, AD : After Diving, R : Recovery
FR : Full Recovery, AR : Activity Recovery

3.2. SOD change by measurement period and difference between groups

At full recovery, SOD was 0.89 U/mL before diving, 0.94 U/mL after diving, 0.92 U/mL after 10 minutes of recovery, and 0.94 U/mL after 30 minutes of recovery. It increased immediately after diving but decreased to 10 minutes of recovery and then again increased to 10 to 30 minutes of recovery, but there was no significant difference. At activity recovery, SOD was 0.93 U/mL before diving, 0.95 U/mL after diving, 0.94 U/mL after 10 minutes of recovery, and 0.92 U/mL after 30 minutes of recovery. It tended to increase until immediately after diving and decrease to 30 minutes of recovery, with no significant difference between groups. The independent t-test was conducted to determine the difference between full recovery and activity recovery in each period. The analysis always showed no statistically significant differences, with t=-0.410 p=0.688, t=0.908, p=0.130, p=0.898, recovery 30 minutes t=0.143, and p=0.888, before diving (Table 3).

Table 3: Changes in blood SOD

Variable	Group	BD(A)	AD(B)	R10(C)	R30(D)	f-value	p
WBC	FR	5.40 ±1.47	6.11 ±1.51	5.29 ±1.46	5.28 ±1.39	0.085	0.869

	AR	5.82 ±1.18	5.85 ±1.37	5.71 ±1.17	5.47 ±1.22	4.643	0.906
t-test	t-value	-0.635	0.363	-0.631	-0.293	-	-
	p	0.535	0.722	0.538	0.774		

M±SD
BD : Before Diving, AD : After Diving, R : Recovery
FR : Full Recovery, AR : Activity Recovery

5. Discussion

After high intense exercise, the body requires a lot of oxygen, which increases cardiac output, increases the metabolism in the mitochondria in skeletal muscle cells, increasing the activity of oxidase. As a result, physical activity such as exercise causes an imbalance between the ROS and antioxidants through various reasons (Power & Jackson, 2008). ROS is also known to contribute to various diseases, accelerating aging, causing muscle injury due to inflammation and hypoxia and oxygen saturation, with repeated apnea diving reported to increase gradually in body hypoxia, high carbonation over a short period of time (Paulev, 1965). Rapid changes in oxygen levels in diving mammals are reported to cause many changes in metabolism to prevent ROS production due to antioxidant depletion, oxidative stress, and ischemic-reductive processes (Elsne, Øyas, Saugstad, & Blix, 1995; Halliwell, 1999; Kjeld, Møller, Fogh, Hansen, Arendrup, Isbrand, Zerahn, Højberg, Ostenfeld, Thomsen, Gormsen, & Carlsson, 2021).

Ji & Lardy (1988) reported that if exercise is continued, the processing power of the peroxidizing lipids produced by the promotion of the enzyme SOD, CAT, and GPX were enhanced. Reduction of this oxidation stress, the production of antioxidants increases, and SOD is the first antioxidant to be expressed, which is reported to remove peroxide anions, which increase the expression of SOD to minimize the production of ROS (Fielding & Meydani, 1997). Previous studies on the action of SOD show that Adkinson (1987) is exposed to toxic substances by generating endless free radicals in the body's metabolism, which increases ROS production by increasing oxygen intake during high-intensity exercise, resulting in negative effects, such as the destruction of tissue membrane. Increasing of ROS resulted in an increase in peroxidized lipid, MDA (Kappus, 1985), and Lovlin, Cottle, Pyke, Kavanagh, and Belcastro (1987), which reported a decrease in lipid peroxidation at 40% and 70% of the maximum oxygen intake after exercise. Thus, the level of oxidative stress during exercise suggests that it is related to intensity of exercise.

Previous studies have shown that trained divers result in an increase in SOD (Joulia et al. 2002). Other studies compared trained and untrained divers to see antioxidant defenses appearing at peak diving, and although trained divers saw an increase in SOD, MDA showed no difference between groups (Bulmer, Coombes, Sharman, & Stewart, 2008).

In this study, changes in blood MDA tended to decrease from 10 minutes of recovery after only increasing to 10 minutes of recovery before diving, but there was no significant numerical difference. This shows that previous study by Lovlin et al. (1987) Show an increase in lipids preceding from 100% of the maximum oxygen intake, while a decrease of 40% and 70% of the maximum oxygen intake. Thus, these results are thought to have shown no change in MDA levels due to the low intensity of exercise of the experiment. According to Davies et al. (1982), The SOD, an antioxidant enzyme, should also be increased as MDA is produced by activating oxygen after exercise to provide the body's defense role. However, our study showed that a little change in the level of SOD, an antioxidant, and MDA also showed no difference. Prior study show the MDA levels increase immediately after low-intensity exercise in female college students, and MDA levels decrease to pre-exercise conditions 30 minutes after recovery, keeping them stable (Lee et al, 2008). The MDA showed the same results as this experiment, but SOD showed a tendency for the number of full recovery groups to increase again in the 10 minutes of recovery. In addition, another study analyzed SOD and MDA changes resulting from full recovery and activity recovery in high school students and found that the activity recovery group had high SOD levels and tended to increase again from 30 minutes of recovery (Kim & Kim, 2005). However, con our study showed that the level of SOD increases again in the full recovery group for10 minutes, this study showed the opposite of that of prior study (Kim & Kim, 2005). This data that the full recovery group had a higher level than the activity recovery group, and that three diving experiences, including pre-test, have performed two repetitive dives prior to the activity recovery experiment, and have adapted to the psychological stability of the experiment. The data of MDA are higher in the full recovery group, and three diving experiences, including Pre-test, were previously repeated during the activity recovery experiment, and 20 diving experiences were adapted during the experiment, thus reducing exercise fatigue.

As a result, the changes in SOD and MDA enzymes were influenced by exercise intensity, but there was no significance change during 20 repetitive dives. And in the recovery period, only in activity recovery tended to decrease, but there was no significant difference between full and activity recovery.

6. Conclusion

This study analyzes the physiological changes that occur according to the recovery method after freediving and studies the peroxidizer and antioxidant according to the recovery method.

As a result of comparing the changes in oxidative stress and antioxidant enzymes according to the recovery method after diving, SOD showed a tendency to decrease immediately after diving and MDA increased up to 10 minutes after recovery. There was a trend, and there was no significant difference between groups in MDA.

This study, we tried to suggest an appropriate recovery method by observing the physiological changes according to the recovery method after repeated apnea diving, but there was no significant difference. confirmed that. In future research, it is thought that it will be a better study if the oxidation reaction is increased by increasing the number of repeated diving or by increasing the diving time.

References

Alboni, P., Alboni, M., & Gianfranchi, L. (2011). Diving bradycardia: a mechanism of defence against hypoxic damage. *Journal of cardiovascular medicine, 12*(6), 422-427.

ACSM. (1991). Guidelines for exercise testing and prescription. *4th ed. Philadelphia: Lea &Febiger,* 62-63.

Adkinson, D. (1986). Role of free radicals in ischemia-reperfusion injury to the liver. *Acta Physiol Scand*, 548, 101-107.

Bae, C. U. (2001). The Effect of Long - term Regular Running Aerobic Exercise on the Activation of Antioxidation Enzymes and Lipid Peroxidation*. korean Alliance for Health, Physical Education, Recreation, And Dance, 40*(4), 829-839.

Bast, A., & Goris, R. J. A. (1989). Oxidative stress. *Pharmaceutisch Weekblad, 11*(6), 199-206.

Bulmer, A. C., Coombes, J. S., Sharman, J. E., & Stewart, I. B. (2008). Effects of maximal static apnea on antioxidant defenses in trained free divers*. Medicine and science in sports and exercise, 40*(7), 1307-1313.

Davies, K. J., Quintanilha, A. T., Brooks, G. A., & Packer, L. (1982). Free radicals and tissue damage produced by exercise. *Biochemical and biophysical research communications, 107*(4), 1198-1205.

Elsner, R., Øyas, S., Saugstad, O. D., & Blix, A. S. (1995). Seal adaptations for long dives: recent studies of ischemia and oxygen radicals. Developments in marine biology. *Vol. 4. Elsevier Science, 1995*. 371-376.

Ferretti, G., & Costa, M. (2003). Diversity in and adaptation to breath-hold diving in humans. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology, 136*(1), 205-213.

Fielding, R. A., & Meydani, M. (1997). Exercise, free radical generation, and aging. *Aging Clinical and Experimental Research, 9*(1), 12-18.

Gooden, B. A. (1994). Mechanism of the human diving response. *Integrative physiological and behavioral science, 29*(1), 6-16.

Ha, C. S. (2006). The effect of vitamin C, E supplementation on the lipid peroxide and SOD activity, and blood fatigue factors after submaximal exercise. *The Korean Society of Sports Science, 15*(4), 805-815.

Halliwell, B. (1999). Antioxidant defence mechanisms: from the beginning to the end (of the beginning). *Free radical research, 31*(4), 261-272.

Hong, S. K., & Rahn, H. (1967). The diving women of Korea and Japan. *Scientific American, 216*(5), 34-43.

Hong, S. W. (2013). The Effect of Antioxidant Supplementation on Oxidative Stress Antioxidant Enzyme and Immune Function in Ballet Movement. (Doctoral Dissertation, Hanyang University).

Hur, S. (2009). The Effect of Vitamin E Supplement on Oxidative Stress, Antioxidant Capacity and Immune Response Factors by Exercise Types in Athletes. (Doctoral Dissertation, Kangwon University).

Ji, L. L., Stratman, F. W., & Lardy, H. A. (1988). Antioxidant enzyme systems in rat liver and skeletal muscle: influences of selenium deficiency, chronic training, and acute exercise. *Archives of biochemistry and biophysics, 263*(1), 150-160.

Ji, Y. S., Jeon, E. J., & Lee, S. J. (2006). Effects of all-out exercise on SOD, MDA, lipid profile and lactate levels between athletes and general persons. *Journal of Coaching Development, 8*(2), 307-315.

Joulia, F., Steinberg, J. G., Wolff, F., Gavarry, O., & Jammes, Y. (2002). Reduced oxidative stress and blood lactic acidosis in trained breath-hold human divers. *Respiratory physiology & neurobiology, 133*(1-2), 121-130.

Jung, D. J., & Chung, S. T. (1999). The Effect of antioxidants supplementation and exercise intensity on the lipid peroxidation. *Exercise Science, 8*(3), 423-436.

Kappus, H.(1985). Lipid peroxidation: mechanisms, analysis, enzymology and biological relevance. *Oxidative stress, pp.* 273-310

Kim, B. J., Lee, D. T., & Lee, W. Y. (2012). Influence of Repeated Breath-hold Diving in Cold Water on Heart Rate, Lactate, and Blood Oxygen Saturation. *The Korean Society of Living Environmental System, 19*(1), 75-81.

Kim, J. H., Kim, D. Y., Lee J. A., & Ha, H. D. (2015). Effects of Aquatic Dynamic Recovery on Blood Lactate, Ammonia, LDH and CK after Performing Rowing Ergometer in Rowers. *Journal of Coaching Development, 17*(3), 141-149.

Kim, M. H., & Hong S. W. (2012). Effect of ShortTerm Vitamin Supplementation on MDA, SOD, GPx in Ballet. *The Korean Society Of Dance Science, (26),* 161-171.

Kim, Y. A., & Kim, H. (2005). The alternation of SOD, CAT & MDA on Physical Activity Recovery Different. *The Korean Society of Sports Science, 14*(2), 547-554.

Kjeld, T., Møller, J., Fogh, K., Hansen, E. G., Arendrup, H. C., Isbrand, A. B., Zerahn, B., Højberg, J., Ostenfeld, E., Thomsen, T., Gormsen, L. C., & Carlsson, M. (2021). Cardiac hypoxic resistance and decreasing lactate during maximum apnea in elite breath hold divers. *Scientific reports, 11*(1), 1-10.

Lee, G. P., Eun, H. G., & Im I. S. (1997). Potentially Harmful Effects and Effect of Antioxidant by Oxygen Free Radical during Maximal Exercise, *The Korean Journal of Physical Education, 36*(1), 243-255.

Lee, K. S., & Kim, J. U. (1999). The Effect of Exhaustive Sprint Training on Tissues Damage and Antioxidant Enzyme Activities of Skeletal and Heart Muscle in Mice. *Exercise Science, 8*(3), 461-471.

Lee, Y. M., Lee, S. E., Lee, J. M., & Choi, S. W. (2008). The Effects of Exercise Intensities on MDA and SOD. *Physical activity and nutrition, 12*(2), 83-88.

Linér, M. H. (1994). Cardiovascular and pulmonary responses to breath-hold diving in humans. *Acta Physiologica Scandinavica. Supplementum, 620*, 1-32.

Lovlin, R., Cottle, W., Pyke, I., Kavanagh, M., & Belcastro, A. N. (1987). Are indices of free radical damage related to exercise intensity. *European journal of applied physiology and occupational physiology, 56*(3), 313-316.

Min, J. U., & Kim, S. H. (2019). Effects of Phytoncide-rich Indoor Exercise on Cardiovascular Index, Stress and Antioxidant Capacity Changes. *Journal of Sport and Leisure* Studies*, 77*, 487-496.

Mrakic-Sposta, S., Vezzoli, A., Rizzato, A., Della Noce, C., Malacrida, S., Montorsi, M., Paganini, M., Cancellara, P., & Bosco, G. (2019). Oxidative stress assessment in breath-hold diving. European journal of applied physiology, *119*(11), 2449-2456.

Packer, L. (1991). Protective role of vitamin E in biological systems. *The American journal of clinical nutrition, 53*(4), 1050S-1055S.

Park, E. J., Ku, H. J., & Lee, J. G. (2004). Setting of Proper Recovery Exercise Intensity to Recover from Lactate Fatigue. *Journal of Korean Physical Education Association for Girls and Women, 18*(1), 67-75.

Patlar, S., Baltaci, A. K., & Mogulkoc, R. (2016). Effect of vitamin A administration on free radicals and lactate levels in individuals exercised to exhaustion. *Pak. J. Pharm. Sci, 29*(5), 1531-1534.

Paulev, P. (1965). Decompression sickness following repeated breath-hold dives. *Journal of Applied Physiology, 20*(5), 1028-1031.

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.

Qvist, J., Hurford, W. E., Park, Y. S., Radermacher, P., Falke, K. J., Ahn, D. W., ... & Weber, R. E. (1993). Arterial blood gas tensions during breath-hold diving in the Korean ama. *Journal of Applied Physiology, 75*(1), 285-293.

Schagatay, E. (2014). Human breath-hold diving ability and the underlying physiology. *Human evolution, 29*(1-3), 125-140.

Urso, M. L., & Clarkson, P. M. (2003). Oxidative stress, exercise, and antioxidant supplementation. *Toxicology, 189*(1-2), 41-54.