

FULL PAPER

Antioxidant effect of purple sweet potato (*Ipomoea batatas* var. *Antin 3*) for the prevention of oxidative stress after high-intensity physical exercise in rat

Mahendra Wahyu Dewangga*  | Dimiyati  | Djoko Pekik Irianto 

Department of Sport Science, Faculty of Sport Science, Universitas Negeri Yogyakarta, Yogyakarta, Indonesia

High-intensity exercise produces free radicals in the body, resulting in oxidative stress. The presence of oxidative stress can be determined by measuring superoxide dismutase and malondialdehyde levels. Consuming foods in a high range in antioxidants is one way to prevent oxidative stress in the body. Purple Sweet Potato (*Ipomoea batatas* var. *Antin 3*) has anthocyanins and vitamin C, which can fight free radicals and keep the body healthy. To find out how *Ipomoea batatas* var. *Antin 3* can help prevent oxidative stress after a lot of exercise in rat. The experimental research with a post-test only control group design. The number of rats is 24, then they were divided into four groups. Group I was given the standard feed, while Group II was given both exercise and the standard feed. Group III was given exercise, standard feed, and purple sweet potato 2.6 mg/day. Group IV was given exercise, standard feed, and purple sweet potato 5.2 mg/day. The physical exercise was given every day for 28 days. Observation of SOD and MDA levels was done by spectrophotometry. Analysis using one-way ANOVA and post hoc LSD revealed that administration of purple sweet potato at a dose of 5.2 mg/day was the most effective dose for decreasing MDA levels ($p < 0.05$) and increasing SOD activity ($p < 0.05$), as well. Giving purple sweet potatoes prevented the accumulation of oxidative stress, but administration of purple sweet potatoes at a dose of 5.2 mg/day was more effective in preventing the buildup of free radicals which cause oxidative stress in the body.

***Corresponding Author:**

Mahendra Wahyu Dewangga

Email:mahendrawahyu.2020@student.uny.ac.id

Tel.: +6281227204161

KEYWORDS

Superoxide dismutase; malondialdehyde; oxidative stress, high-intensity physical exercise; purple sweet potato; *Ipomoea batatas* var. *Antin 3*.

Introduction

Today, many people are starting to diligently exercise to maintain health and improve the body's functional abilities. Of course, doing physical exercise makes the body to be fit [1].

However, it should be noted that exercise is a form of physical stressor which hurts the body if it does not pay attention to the basic principles of exercise [2].

Exercise with high intensity and not by the rules will increase oxidative stress in the body.

The oxidative stress is a condition of an imbalance in the production of Reactive Oxygen Species (ROS) or can further be called the oxidants with antioxidants in the body that triggers tissue damage, inflammation, decreased immunity, and even aging [3]. This is because high-intensity exercise requires more oxygen consumption than the measured exercise. In the physiological process, precisely during the oxidative phosphorylation, oxygen will be converted into Reactive Oxygen Species (ROS) as much as 4-5% [4]. This will increase many times in proportion to the intensity of the increasingly strenuous exercise. The increased formation of Reactive Oxygen Species (ROS) in the body begins 12-24 hours after physical activity, then increases after 48-72 hours, and will return to be normal after 72 hours according to the intensity and the duration of exercise [5].

One way to measure the accumulation of oxidative stress in the body is to look at high malondialdehyde (MDA) levels. Malondialdehyde (MDA) is a toxic compound as the end product of lipid peroxidation. Lipid peroxidation breaks fatty chains in the cell membrane, which results in cell damage [6]. If this situation lasts permanently, the body will experience oxidative stress, which impacts various pathological conditions such as cell damage in organs such as the kidneys, heart, spleen, and other organs. This damage can accelerate cell death and degenerative diseases such as immune system disorders, cancer, and premature aging [7]. Therefore, the effects of ROS should be addressed as quickly as possible, one of which is the consumption of antioxidant-rich foods [8].

Purple sweet potatoes are one of the tubers well known by the public. Purple sweet potato variant Antin 3 is the latest variant developed by the Indonesian Legumes and Tuber Crops Research Institute. This sweet potato has been able to grow in various parts of Indonesia. Purple sweet potatoes variant Antin 3 has a relatively high antioxidant content, namely

vitamin C at 20mg/100g and anthocyanin, which is relatively high at 150mg/100 [9]. Several studies have indicated that purple sweet potato can reduce oxidative stress in the body. One of the studies conducted by Sutirta Yasa concluded that purple sweet potato could reduce malondialdehyde (MDA) levels in the livers of mice due to the chronic alcohol administration [10]. This study aimed to determine the antioxidant effect of purple sweet potato (*Ipomoea batatas* var Antin 3) to prevent oxidative stress after high-intensity exercise in rat.

Methods

Research design

Using a post-test-only control group design method, the true experimental research was conducted. This research took place at the Integrated Biomedical Laboratory (IBL) Faculty of Medicine, Sultan Agung Islamic University, Semarang, Indonesia, and the Center for Food and Nutrition Studies, Gadjah Mada University, Indonesia. The medical ethics committee has approved this research of Sultan Agung Islamic University, Semarang. The ethical clearance number for this research is No.358/X/2021/Bioethics Commission. This research was conducted for four weeks conducted, in November-December 2021.

Animal

The male Wistar rats aged 8-10 weeks and weighing 150-200 grams in good health were used as test animals in this research. The number of samples in this study was 24 rats. These animals were obtained from the Experimental Animal Laboratory, Faculty of Medicine, Sultan Agung Islamic University, Semarang.

Purple sweet potato intake

Purple sweet potato (*Ipomoea batatas* var. Antin 3) was obtained from The Indonesian

Legumes and Tuber Crops Research Institut, Ministry of Agriculture, located in Malang, East Java, Indonesia. Then, the purple sweet potato is processed by peeling the skin, and after that steaming for 10 minutes. Next, the purple sweet potato is mashed. This preparation was further tested for anthocyanin content at the Food & Nutrition Laboratory, Faculty of Agriculture, Sebelas Maret University in Surakarta. Anthocyanin analysis was carried out using the Giusti & Worlstad method.

Purple sweet potatoes are processed by steaming for 15 minutes, then put into a blender machine until smooth. This processed purple sweet potato contains anthocyanins of 286.6 mg/100 gram (Figures 1 and 2). This processed result will be given to the sample. Then, the dose is adjusted according to the formula: $143.3 \text{ mg} \times 0.018$ to get 2.6 mg/day/head for group 3 and $286.6 \text{ mg} \times 0.018$ to get 5.2 mg/day/head for group 4. The administration of purple sweet potato was carried out 2 hours after the rats were given high-intensity physical exercise.

Treatment of test animals

This study consisted of 4 groups. Determination of the minimum number of subjects is determined based on Federer's formula, namely $(n-1) \times (t-1) \leq 15$, in which n is the sample size for each group, while t is the number of groups, so that $n \geq 6$ is obtained [11]. The procedure of this study begins with the acclimation for seven days with the standard feed and distilled water. Before exercising, the rats were initially checked for weight.

This study consisted of 4 groups. Determination of the number of samples using the Federer formula and each group obtained a total of 6 samples as well as a sample size of 24 individuals. The control group (G I) rats were neither given the high-intensity exercise, nor purple sweet potato, and the rats were only given the standard feed. Group 2 (G II) rats were given standard feed, physical

exercise swimming without being given purple sweet potato consumption. Group 3 (G III) rats were given the standard feed, swimming exercises every day for four weeks, and the consumption of purple sweet potato at a dose of 2.6mg/200gram body weight of rats. Group 4 (G IV) rats were given the standard feed, swimming exercises every day for four weeks, and consumption of purple sweet potato at a dose of 5.2mg/200gram body weight. We differentiated dosing in Group 3 (G III) and Group 4 (G IV) in order to determine the most effective dose to prevent oxidative stress.

Furthermore, in groups 2, 3, and 4, the high-intensity exercise was given using rats swimming in the aquarium for 20-30 minutes until they looked like they had almost drowned [12]. Purple sweet potato was given 2 hours after the sample did physical exercise orally.



FIGURE 1 The intake of purple sweet potato

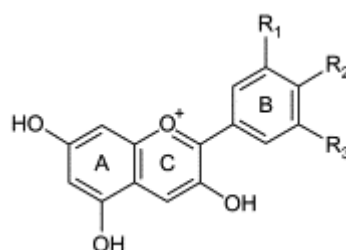


FIGURE 2 The basic structure of anthocyanin [9]

Procedure for checking superoxide dismutase (SOD) levels

After four weeks, or on day 29, blood serum was taken from mouse samples through the retro-orbital plexus. Subsequently, the levels of MDA and SOD were examined using spectrophotometry.

Superoxide dismutase levels were determined biochemically using the Ran-SOD-BioVision Colorimetric Assay Kit at the Center for Food and Nutrition Studies, Gadjah Mada University, Indonesia. The reagents in this kit consist of a mixed substrate containing xanthine, phosphate buffer to dilute (the standard or sample), xanthine oxidase, and the standard solution to create the standard curves. A total of 25 μL of plasma was used to measure blood SOD levels. Initially, 25 μL of the sample was put into the cuvette and 850 μL of the mixed substrate was added and mixed well. To inhibit SOD, 5 μL of 5 mM sodium cyanide was added to the mixture until properly mixed. After that, 125 μL of xanthine oxidase was added. The absorption was read at a wavelength of 505 nm with a spectrophotometer (Genesis). Superoxide dismutase (SOD) levels were determined using the equation obtained from the standard curve.

Procedure for checking Malondialdehyde (MDA) levels

The MDA examination was conducted using Malondialdehyde (MDA) Colorimetric Assay Kit (TBA Method) Elabscience. The measurement of the concentration of the experimental sample was carried out in the same way as the standard solution, namely 1.0 mL of blood plasma was reacted with 1.0 mL of 20% TCA and 1.0 mL of 1% TBA in 50% glacial acetic acid, and then incubated for 45 minutes at 95 °C. The solution was centrifuged for 15 minutes at 1000 rpm. The supernatant was separated, and then measured its absorbance using a UV-Vis spectrophotometer

at a wavelength of 532.2 nm. The sample concentration was obtained by plotting the absorbance data of the sample into a standard curve.

Statistical analysis

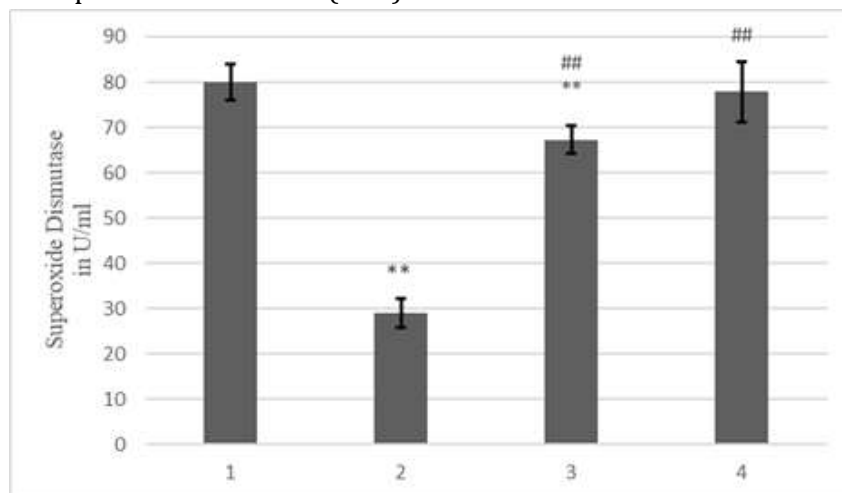
The obtained superoxide dismutase (SOD) and malondialdehyde (MDA) data were initially analyzed statistically with normality test and homogeneity test. Then, the data were analyzed by One Way Anova to find out whether there were significant differences between groups. Next, the process was continued with Post Hoc LSD to find out which intervention is more influential in reducing oxidative stress levels.

Results

Superoxide dismutase (SOD) levels

In this study, the observation of the average superoxide dismutase (SOD) level in group 1 had an average superoxide dismutase level of 81.1 U/ml. In group 2, post high-intensity exercise for 28 days had an average superoxide dismutase level of 29.4 U/ml. Group 3 after high-intensity exercise and given purple sweet potato 2.6mg/day had an average superoxide dismutase level of 63.3 U/ml. In the fourth group after high-intensity exercise and given purple sweet potato, the superoxide dismutase level was 75.2 U/ml. Based on the analyzed data, it can be concluded that administration of purple sweet potato at a dose of 2.6 mg/day and 5.2 mg/day can increase superoxide dismutase levels after high-intensity exercise ($p < 0.05$). These results are presented in Graph 1.

GRAPH 1 Effects of purple sweet potato consumption after high intensity physical exercise on average levels of superoxide dismutase (SOD)



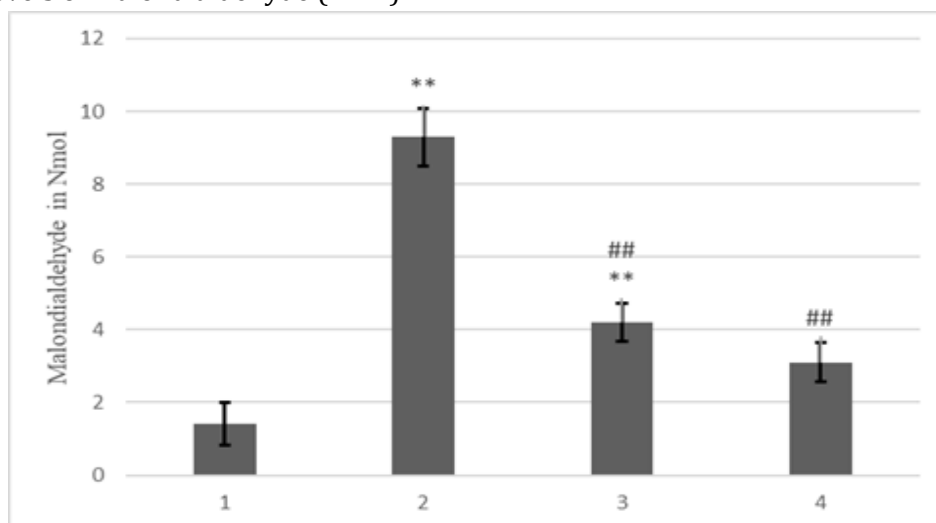
Values are represented as Mean \pm Standard error of mean (SEM) (n=6). ANOVA followed by Post Hoc Analysis by SPSS 26. **P < 0.05 has significant different SOD levels with control group. ##P < 0.05 has significant different SOD levels with only high-intensity physical exercise group.

Malondialdehyde (MDA) levels

In this study, the results of the observation of the mean level of Malondialdehyde (MDA) in group 1 had an average malondialdehyde level of 1.9 nmol/ml. In group 2, post high-intensity exercise for 28 days had an average superoxide dismutase level of 8.7 nmol/ml. Group 3 after high-intensity exercise and given purple sweet potato 2.6mg/day had an average superoxide dismutase level of 4.52

nmol/ml. In the fourth group after high-intensity exercise and given purple sweet potato, the superoxide dismutase level was 3.42 nmol/ml. Based on the analyzed data, it can be concluded that administration of purple sweet potato at a dose of 2.6 mg/day and 5.2 mg/day can increase superoxide dismutase levels after high-intensity exercise (p < 0.05). These results can be visible in Graph 2.

GRAPH 2 Effects of purple sweet potato consumption after high intensity physical exercise on average levels of malondialdehyde (MDA)



Values are represented as Mean \pm Standard error of mean (SEM) (n=6). ANOVA followed by Post Hoc Analysis by SPSS 26. **P < 0.05 has significant different MDA levels with control group. ##P < 0.05 has significant different MDA levels with only high intensity physical exercise group.

Discussion

Superoxide dismutase (SOD) levels

The physical exercise has an important impact on the human body. It improves immunity, strengthens physical fitness, and prevents various aging diseases [5]. Likewise, it is the movement of the limbs which causes the energy expenditure, which is essential for maintaining physical and mental health and maintaining the immune system in the body, as well [13]. When doing heavy physical training, an ischemia-reperfusion event occurs, increasing the production of free radicals, in which the oxygen supply is often unable to meet oxygen demand. This phenomenon is referred to as the ischemia phase [14].

Meanwhile, a high increase in oxygen supply will increase the formation of oxygen free radicals, which can even reach ten times. This phenomenon is called the reperfusion phase. Several studies have proven that high-intensity physical training can cause the oxidative stress in which the production of oxygen free radicals increases significantly [15].

The increased levels of oxidative stress after heavy intensity exercise are caused by an increase in the need for ATP. At the same time, the intracellular supply of ATP is minimal, resulting in the continuous formation of ATP through oxidative processes, the Krebs cycle, and electron transport. The process of ATP formation requires oxygen; the oxygen consumption in the respiratory chain in mitochondria affects the increase in the production of free radicals [16]. During the oxidative phosphorylation, the oxygen molecules can bind to single electrons which leak from electron carriers in the respiratory chain, and thus forms reactive oxygen species (ROS) [4]. In addition to the high-intensity physical exercise, blood which goes to the inactive organs such as the liver, kidneys, stomach, and intestines is diverted to the

active muscles (legs and heart). This causes an acute lack of oxygen (hypoxia) in these organs [17].

When physical exercise is stopped, blood will quickly flow back to these organs, which is associated with the release of large amounts of oxidants from these organs which were previously deprived of oxygen. Free radicals are atoms or molecules (groups of atoms) with unpaired electrons in their outer orbits [18]. The presence of unpaired electrons causes the compound to be very reactive, looking for a partner by attacking or binding the electrons of the other molecules around it to form new free radicals [19]. Free radicals stand-alone for only a brief period because they will soon fuse with other atoms [20].

Naturally, the body can ward off free radicals by forming endogenous antioxidants produced by the body, whose levels can be measured through GPx (Glutathione Peroxidase), catalase, and SOD (Superoxide Dismutase) enzyme activity [21]. Superoxide Dismutase is an enzyme which catalyzes the dismutation of superoxide anions into hydrogen peroxide (H₂O₂) and O₂ [22]. Although superoxide radicals are not very toxic, they can attract electrons from cell membranes or other components and cause the radical chain reactions. Superoxide radicals can participate in the formation of hydroxyl radicals [23]. Hydrogen peroxide, once formed, should be further removed to prevent the formation of hydroxyl radicals. Catalase and glutathione peroxidase converts hydrogen peroxide into water [21]. Physical activity which is too high will result in the level of free radicals in the body also increasing so that endogenous antioxidants cannot neutralize free radicals. The exogenous antioxidants are needed in more significant quantities to neutralize the effects of free radicals [2].

The results revealed that the lowest levels of SOD were found in group 2, who were given high-intensity physical exercise without purple sweet potato, as presented in the tables

and graphs. The low SOD levels in the treatment indicated high oxidative stress when given high-intensity physical exercise. SOD activity can change due to changes in ROS levels [24]. The increased ROS in high-intensity physical exercise was caused by an increase in mRNA expression of the NADPH oxidase subunit, a complex of ROS-producing enzymes which inhibits the expression and regulation of enzymatic antioxidants. The increase in SOD levels is in line with the given dose [25].

The SOD levels in group 3 given purple sweet potato at a dose of 2.6mg/day after high-intensity physical exercise had significant enough results to prevent a decrease in superoxide dismutase, but the obtained results were not close to the control group. In group 4, the purple sweet potato dose of 5.2 mg/day had the closest mean results to the control group. This proves that the administration of purple sweet potato can prevent a decrease in superoxide dismutase levels after high-intensity exercise. The higher the purple sweet potato extract dose given, the tendency for the average lung SOD level to increase. This indicates that the antioxidant effect of purple sweet potato is getting stronger with increasing the given dose. The antioxidant effect of purple sweet potato extract was most substantial at a 5.2 mg/day dose. Anthocyanins in purple sweet potatoes act as antioxidants which neutralize free radicals from high-intensity physical exercise so that SOD levels in the blood increase [26]. Anthocyanin in purple sweet potato is bound to at least one caffeoyl group, which functions as a free radical scavenger and antimutagenic [27].

Malondialdehyde (MDA) levels

Free radicals are highly reactive and quickly lead to the uncontrolled reactions, resulting in cross-linking DNA, proteins, lipids, or the oxidative damage to the critical functional groups in their biomolecules. Lipid

peroxidation is the earliest known and the most studied mechanism of cell or tissue damage due to free radical attack [6]. Most lipid peroxidation occurs in cell membranes, primarily in unsaturated fatty acids, essential components. Cell membranes are rich sources of polyunsaturated fatty acids (PUFA), which oxidizing agents easily damage. The process is called fat peroxidation. This is very destructive because it is a continuous process [28].

Malondialdehyde (MDA) is one of the results of lipid peroxidation caused by free radicals during maximal physical exercise or high-intensity endurance exercise, so Malondialdehyde (MDA) is a general indicator which is used to determine the number of free radicals and indirectly assess the oxidant capacity of the body [29]. Cell membranes are essential for receptor and enzyme functions, resulting in lipid peroxidation of cell membranes by free radicals, resulting in total loss of cellular function [29]. The results revealed that the average total MDA levels in group 1 had an average malondialdehyde level of 1.9 nmol/ml. In group 2, post-high-intensity exercise for 28 days had an average superoxide dismutase level of 8.7 nmol/ml. Group 2 demonstrated that high-intensity physical exercise performed every day for four weeks resulted in an imbalance between the number of antioxidants and free radicals in the body. The number of antioxidants will be significantly influenced by the number of free radicals formed in the body during high physical activity [30].

The results showed that after administration of purple sweet potato, the MDA levels in groups 3 and 4 that were given high-intensity physical exercise, had a lower mean value. In group 3, the results were 4.52 nmol/ml, while in group 4, the administration of purple sweet potato after high-intensity exercise had superoxide dismutase levels of 3.42 nmol/ml. This indicates that the administration of purple sweet potato can reduce MDA levels that increase due to

maximum physical exercise. The phytochemical screening on purple sweet potatoes conducted by the Indonesian Legumes dan Tuber Crops Research Institute illustrated that purple sweet potatoes contained flavonoids, glycosides, and anthraquinone compounds. Flavonoids are one of the ingredients which can reduce the destructive effects of free radicals by inhibiting lipid peroxidation through peroxidase activation. Flavonoids isolated from purple sweet potato can protect the FUPA phospholipid membrane by donating or donating one of its Hydrogen ions (H⁺) to the lipid peroxy radicals (LOO^{*}). LOO^{*} is the result of the HO^{*} reaction in the lipid peroxidation process of the HO^{*} attack on PUFA (Poly Unsaturated Fatty Acid). Giving H^{*} by an antioxidant can stop other radical reactions so that it makes less reactive radicals [20].

Conclusion

In this study, we can conclude that high-intensity physical exercise can accumulate oxidative stress levels. Therefore, the high consumption of antioxidants is required. Purple sweet potato (*Ipomoea batatas* var. Antin 3) is a food which has a high antioxidant content and can prevent the accumulation of oxidative stress after high-intensity physical exercise. In this study, we suggest that consuming a high enough dose of purple sweet potato after high-intensity physical exercise can prevent the accumulation of oxidative stress.

Acknowledgements

The laboratory personnel of the Faculty of Medicine, Sultan Agung Islamic University, Semarang, and the laboratory team of the Center for Food and Nutrition Studies, Gadjah Mada University, are gratefully acknowledged by the researcher.

Declaration of interest

There are no conflicts of interest reported by the authors. The paper's content and writing are solely the responsibility of the writers.

Orcid

Mahendra Wahyu Dewangga:

<https://www.orcid.org/0000-0002-2777-268X>

Dimiyati:

<https://www.orcid.org/0000-0002-9002-5513>

Djoko Pekik Irianto:

<https://www.orcid.org/0000-0002-8635-7830>

References

- [1] N.A. Dasso, *Nurs. Forum*, **2019**, *54*, 45–52. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [2] C. Simioni, G. Zauli, A.M. Martelli, M. Vitale, A. Gonelli, L.M. Neri, *Oncotarget*, **2018**, *9*, 17181–17198. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [3] J. Finsterer, *BMC Musculoskelet. Disord.*, **2012**, *69–74*. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [4] J.N. Bazil, D.A. Beard, K.C. Vinnakota, *Biophys. J.*, **2016**, *110*, 962–971, [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [5] R.J. Simpson, H. Kunz, N. Agha, R. Graff, *Prog. Mol. Biol. Transl. Sci.*, **2015**, *135*, 355–380. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [6] A. Ayala, M.F. Muñoz, S. Argüelles, *Oxid. Med. Cell. Longev.*, **2014**, *2014*, [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [7] I. Liguori, G. Russo, F. Curcio, G. Bulli, L. Aran, D. Della-Morte, G. Gargiulo, G. Testa, F. Cacciatore, D. Bonaduce, P. Abete, *Clin. Interv. Aging*, **2018**, *13*, 757–772. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [8] S. D'Angelo, *Curr. Sports Med. Rep.*, **2020**, *19*, 260–265. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [9] E. Ginting, R. Yulifianti, F.C. Indriani, *IOP Conf. Ser. Earth Environ. Sci.*, **2020**, *456*. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [10] I.W.P. Sutirta-Yasa, I.M. Jawi, I.B. Ngurah, A.A.N. Subawa, *Indones. J. Clin. Pathol. Med.*

- Lab., **2016**, *14*, 151–154. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [11] M.R. Wahyuningrum, E. Probosari, *J. Nutr. Coll.*, **2012**, *1*, 192–198. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [12] M.W. Dewangga, D.P. Irianto, Dimiyati, Sumaryanto, T. Nasihun, Y. Febrianta, Wahyuni, Wijianto, Agustiyawan, *J. Kerman Univ. Med. Sci.*, **2021**, *28*, 539–547. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [13] M.P. da Silveira, K.K. da S. Fagundes, M.R. Bizuti, É. Starck, R.C. Rossi, D.T. de R. e Silva, *Clin. Exp. Med.*, **2021**, *21*, 15–28. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [14] S. Brickson, J. Hollander, D.T. Corr, L.I. Ji, T.M. Best, *Med. Sci. Sports Exerc.*, **2001**, *12*, 2010–2015. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [15] F. Magherini, T. Fiaschi, R. Marzocchini, M. Mannelli, T. Gamberi, P.A. Modesti, A. Modesti, *Free Radic. Res.*, **2019**, *53*, 1155–1165. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [16] R.Z. Zhao, S. Jiang, L. Zhang, Z. Bin Yu, *Int. J. Mol. Med.*, **2019**, *44*, 3–15. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [17] T.L.V. Hoek, L.B. Becker, Z. Shao, C. Li, P.T. Schumacker, *J. Biol. Chem.*, **1998**, *273*, 18092–18098. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [18] S.T. Zulaikhah, *Sains Med.*, **2017**, *8*, 39–45. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [19] S. Di Meo, P. Venditti, *Oxid. Med. Cell. Longev.*, **2020**, *2020*. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [20] A. Phaniendra, D. B. Jestadi, and L. Periyasamy, *Indian J. Clin. Biochem.*, **2015**, *30*, 11–26. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [21] O.M. Ighodaro, O.A. Akinloye, *Alexandria J. Med.*, **2018**, *54*, 287–293. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [22] H. Wicaksono, M.A. Rahman, R. Irawan, I.K.A. Utamayasa, T. Ontoseno, T. Hidayat, *GSC Biol. Pharm. Sci.*, **2021**, *16*, 150–156. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [23] P. Poprac, K. Jomova, M. Simunkova, V. Kollar, C.J. Rhodes, M. Valko, *Trends Pharmacol. Sci.*, **2017**, *38*, 592–607. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [24] Z. Jahangiri, Z. Gholamnezhad, M. Hosseini, F. Beheshti, N. Kasraie, *J. Physiol. Sci.*, **2019**, *69*, 993–1004. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [25] N. Vargas-Mendoza, Á. Morales-González, E.O. Madrigal-Santillán, E. Madrigal-Bujaidar, I. Álvarez-González, L.F. García-Melo, L. Anguiano-Robledo, T. Fregoso-Aguilar, J.A. Morales-Gonzalez, *Antioxidants*, **2019**, *8*, 196. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [26] A. Elvana, H. Rusmarilin, R. Silaban, R.N. Sinaga, *Indones. J. Med.*, **2016**, *01*, 116–120. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [27] A. Li, R. Xiao, S. He, X. An, Y. He, C. Wang, S. Yin, B. Wang, X. Shi, J. He, *Molecules*, **2019**, *24*, 1–21. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [28] W.W. Christi, J.L. Harwoo, *Essays Biochem.*, **2020**, *64*, 401–421. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [29] L.J. Su, J.H. Zhang, H. Gomez, R. Murugan, X. Hong, D. Xu, F. Jiang, Z.Y. Peng, *Oxid. Med. Cell. Longev.*, **2019**, *2019*. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [30] M. Yimcharoen, S. Kittikunnathum, C. Suknikorn, W. Nak-On, P. Yeethong, T.G. Anthony, P. Bunpo, *J. Int. Soc. Sports Nutr.*, **2019**, *16*, 1–9. [[crossref](#)], [[Google Scholar](#)], [[Publisher](#)]

How to cite this article: Mahendra Wahyu Dewangga*, Dimiyati, Djoko Pekik Irianto. Antioxidant effect of purple sweet potato (Ipomoea batatas var. Antin 3) for the prevention of oxidative stress after high-intensity physical exercise in rat. *Eurasian Chemical Communications*, 2022, 4(9), 921–929. **Link:** http://www.echemcom.com/article_149619.html