UDC 576.3(075.8)

THE EFFECT OF BROCCOLI SPROUTS ON OXIDATIVE STRESS MARKERS IN MICE FED WITH CAFETERIA DIET

V. P. DERKACHOV, M. V. IVANOCHKO, M. M. BAYLIAK

Vasyl Stefanyk Precarpathian National University Department of Biochemistry and Biotechnology Ivano-Frankivsk, Ukraine

E-mail: derkachovvitalii@gmail.com

Received 2023/02/28 Revised 2023/04/08 Accepted 2023/04/28

In recent years, growing interest has been shown in the health advantages of broccoli sprouts [5]. These juvenile plants, which are taken just a few days after germination, contain bioactive substances that have been proven to offer a variety of health benefits. Sulforaphane has attracted the most interest among these substances because of its powerful anti-cancer properties [2]. Studies indicate that in addition to possibly protection against cancer, cardiovascular disease, diabetes, and neurological diseases, broccoli sprouts may also have other health advantages [1]. The antiinflammatory effects of broccoli sprouts are thought to be mediated by several mechanisms. Sulforaphane has been shown to inhibit the activation of NF-kB, a key regulator of the inflammatory response, while glucoraphanin has been shown to enhance the production of anti-inflammatory compounds such as interleukin-10 [4]. Kaempferol, another bioactive compound found in broccoli sprouts, has been shown to inhibit the production of inflammatory cytokines [3]. Anti-inflammatory properties of broccoli sprouts may help to attend inflammation-related oxidative stress that may be beneficial for preventing and treating a range of diseases associated with development of both inflammation and oxidative stress, in particular diet-associated obesity [6]. Therefore, the aim of our study was to determine the ability of broccoli sprouts to influence the intensity of lipid peroxidation in mice fed a high-calorie cafeteria diet [7].

Materials and Methods. In this study, C57BL/6J mice were used. Mice were divided in 4 groups. The first group was the control group and was fed with a standard food. The second group was fed with a standard food with the addition of broccoli sprouts (broccoli group). Mice of the third group fed with a cafeteria diet, which consisted of 70% cafeteria food and 30% standard food (cafeteria diet group). The fourth group was fed with a cafeteria diet with the addition of broccoli sprouts (5% by weight). At the beginning of the experiment, the mice were 9 months old. The experiment lasted 4 months. During experiment body mass of mice was monitored.

In the end of the experiment, the mice were euthanased and organs were harvested to determine the intensity of lipid peroxidation. The frozen tissues were homogenised in 96% ethanol at a ratio of 1:10, centrifugired for 10 min at 10,000 rpm, and supernatants were collected. For determinatiom of lipid peroxides (LOOH), we used a method based on the ability of lipid peroxides to convert Fe^{2+} to Fe^{3+} . Then Fe^{3+} forms a complex with xylenol orange that absorbs light at 580 nm at low pH. The reaction mixture contained coumene hydroperoxide (1 mM), FeSO₄*7H₂O(1 M), xylenol (4 mM), water and supernatant. Differences between groups were analyzed by Duncan's test for multiple comparision.

Results and Discussion. During the experiment, we monitored the changes in body mass of mice fed with different diets and results are presented in Fig. 1.

Next, we measured levels of LOOH in cortex, hypothalamus and muscles for understanding whether inflammatory and oxidative processes have begun. As we can see in Fig. 2, *A*, there was no differences in LOOH levels in cortexes of mice from all experimental groups, but there was a tendency to the lower content of LOOH in the brain of mice fed with cafeteria diet and broccoli.

Then, we decided to measure LOOH in hypothalamus (*B*) as an organ vulnerable for inflammation. No statistical differences in levels of LOOH were found between groups, but LOOH levels tended to the highest in the groups fed with broccoli sprouts alone and cafeteria diet.



Fig. 1. Body mass of mice fed with different diets, n = 7-8



Fig. 2. Level of LOOH in cortex(A), hypothalamus (B), muscles (C) n = 7-8. Used Dunkan's test.

A significant difference was observed in the muscles (C) between the broccoli sprout group and the cafeteria diet + broccoli group. We also found a significant difference between the group fed with the cafeteria diet and the cafeteria diet + broccoli, which may indicate protective effects of broccoli on lipid peroxidation on cafeteria diet.

Conclusions

1. Mice fed with cafeteria diet and broccoli spouts had higher body mass than control mice fed with standard group.

2. Hypothalamus of mice fed with standard diet with broccoli spouts or with cafeteria diet showed a tendency to higher LOOH levels, whereas no effects of the diets were found on cortexes LOOH levels.

3. The cafeteria diet + broccoli group had the lowest muscle LOOH content compared to all other groups. Also, LOOH levels tended to be lower in the cortexes in the hypothalamus of mice fed with cafeteria diet + broccoli as compared with the cafeteria diet group. This suggests the potential protective effects of broccoli spouts.

Keywords: broccoli; lipid peroxides; inflammation.

Authors' contribution. V. P. Derkachov — Investigation, Data analysis; M. M. Bayliak — supervisor, M. V. Ivanochko helping with measurements; Volodymyr I. Lushchak: funding acquisition, design of the study.

Funding. This work was supported by a grant from Ministry of Education and Science of Ukraine for VIL (#0122U000894).

REFERENCES

- 1. Egner P. A., Chen J. G., Zarth A. T., Ng D. K., Wang J. B., et al. Rapid and sustainable detoxication of airborne pollutants by broccoli sprout beverage: results of a randomized clinical trial in China. Cancer prevention research (Philadelphia, Pa.). 2014, 7(8), 813-823. https://doi.org/10.1158/1940-6207.CAPR-14-0103
- 2. Heber D., Li Z., Garcia-Lloret M., Wong A. M., Lee T. Y., Thames G., Krak M., Zhang Y., Nel, A. Sulforaphane-rich broccoli sprout extract attenuates nasal allergic response to diesel exhaust particles. Food & function. 2014, 5(1), 35-41. https://doi.org/10.1039/c3fo60277j
- 3. Kim H, Kim E, Choe J. Sulforaphane inhibits the activation of inflammatory response in human primary synovial cells. Food Sci Biotechnol. 2021, 30(3). 419-427. https://doi.org/10.1007/s10068-020-00851-2
- 4. *Kim H, Kim E, Park S, Kim J, Kim H*. Sulforaphane regulates the expression of Th1/Th2 cytokines and chemokines via the Nrf2/ARE pathway in mouse bone marrow-derived macrophages. *Food Sci Biotechnol.* 2018, 27(6), 1703–1710. https://doi.org/10.1007/s10068-018-0441-9
- 5. Kong F, Zhang J, Li Y. Protective effect of sulforaphane against skeletal muscle injury induced by ischemia-reperfusion through regulation of mitochondrial fusion/fission. Food Funct. 2021, 12(1), 79–88. https://doi.org/10.1039/d0fo02255a
- 6. *Talalay P, Fahey JW*. Phytochemicals from cruciferous plants protect against cancer by modulating carcinogen metabolism. *J Nutr.* 2001, 131(11 Suppl), 3027S-3033S. https://doi.org/10.1093/jn/131.11.3027S
- 7. Xiao D, Liu C, Li J. Sulforaphane attenuates microglia-mediated neuroinflammation and enhances microglial autophagy via the Nrf2/ARE pathway. Aging (Albany NY). 2021, 13(1), 358-374. https://doi.org/10.18632/aging.202184