EFFECT OF EXERCISE AND NUTRITIONAL SUPPLEMENTATION ON ANTIOXIDANT STATUS AND HEALTH

Associate Prof. Dr. Chee Keong Chen,
Exercise and Sports Science Programme,
School of Health Sciences,
Universiti Sains Malaysia,
16150 Kubang Kerian, Kelantan,
Malaysia.
E-mail: ckchen@usm.my

Free radicals refer to reactive oxygen species and reactive nitrogen species which are highly reactive because of an unpaired valence electron. Oxidative stress occurs when the rate of production of free radicals exceed the body's antioxidant capacity to detoxify them^{1,2}. Oxidative stress has been postulated to contribute to a variety of diseases including atherosclerosis, cataracts, certain cancers, inflammatory diseases, ischemic diseases and neurological disorders^{3, 4,5,6}.

Antioxidants, on the other hand, are substances that scavenge free radicals and thus offer protection from the damaging effects of free radicals. However, several longitudinal studies which were funded primarily by National Institutes of Health, USA have generally reported that antioxidant supplementation have no beneficial effects on health. For example, the Physicians Health Study II found that neither vitamin E nor vitamin C supplements reduced the risk of major cardiovascular events or cancer⁷. The Selenium and Vitamin E Cancer Prevention Trial found that selenium and vitamin E taken alone or together did not prevent prostate cancer⁸. The Women's Health Study found that overall vitamin E did not reduce the risk of death and cardiovascular events ⁹. Similarly, the Nurses' Health Study did not show that dietary intake and additional antioxidant supplementation can reduce risk for endometrial cancer¹⁰.

Gifkins et al. (2012) however, has shown that the intake of total phenolic from foods indeed decreased the risk for endometrial cancer, but the supplementation of individual phenolic compound did not decrease the risk for this cancer¹¹. Another study indicated that intakes of vitamin C, carotene, proanthocyanidins, and manganese from fruits, vegetables, and other foods may reduce the risk for non-Hodgkin lymphoma but intakes of artificial vitamins C and E, selenium, zinc, copper, and manganese did not decrease the risk for this disease¹². It has also been reported that each portion of fruit consumed per day reduces the risk of oral cancer by 49%, whereas regular vegetable consumption decreases the overall risk

of oral cancer by 50 % ^{13, 14, 15}. In a recent systematic review of literature, it was reported that inorganic nitrate and beetroot juice reduces blood pressure in adults ¹⁶.

With these equivocal data, positive effect on overall health status with antioxidant intake is still unclear. Nevertheless, some of the previous studies mentioned above did demonstrate that antioxidants derived from natural food and vegetables seem to confer some beneficial effects on health and reduces the risk of certain diseases.

Several studies have also indicated that regular exercise training positively alters the oxidative homeostasis of cells and tissues by reducing the levels of oxidative damage and thus increases resistance to oxidative stress^{17, 18, 19}. Chronic exercise causes adaptations in the antioxidant capacity and thereby protects cells against the detrimental effects of oxidative stress²⁰. It has also been shown that there was an enhancement of endogenous antioxidant potential and a reduction in lipid peroxidation following habitual physical activity in rats ²¹. Other investigators have also consistently shown that exercise and physical activity upregulate antioxidant defenses, such as superoxide dismutase in the cardiovascular systems and increased level of total antioxidant capacity ^{22, 23, 24, 25}.

Recent studies from my research team in Universiti Sains Malaysia have also demonstrated that exercise training alone or in combination with antioxidant supplementation increases antioxidant status among the participants. For example, we found that 8 weeks of aerobic dance exercise (3 times/week) and when combined with supplementation of chocolate malt drink increased glutathione peroxidase and superoxide dismutase 26 . In another study on sedentary male university students, we found that jogging exercise (3 times/week) alone and when combined with oat bran supplementation (3.6 g of β -glucan daily) for 8 weeks resulted in an increase in predicted VO_{2max} and a reduction in total cholesterol and superoxide dismutase activity 27 .

In an animal study, our findings indicated that continuous 16 weeks of combined jumping (40 jumps/day for 5 days/week) and honey supplementation (1 g/kg body weight/rat/day) resulted in a significant increase in levels of total antioxidant status, blood superoxide dismutase and reduced glutathione ²⁸. We found similar effect of exercise and honey supplementation in humans. There was a significantly better maintenance of the beneficial effects induced by 8 weeks of combined aerobic dance exercise (3 times/week) and honey supplementation (20g.day⁻¹) on blood total antioxidant status and reduced glutathione

after 8 weeks of the cessation of exercise and honey supplementation as compared to exercise alone and honey supplementation alone²⁹.

In conclusion, there is adequate evidence that exercise alone or when combined with certain antioxidant supplements elicited increased antioxidant status in both animals and humans. However, the role of this enhanced antioxidant status either through regular physical activity or antioxidant supplementation or a combination of exercise plus supplementation in reducing the risk of chronic diseases is still unclear. Hence, more studies to identify the precise mechanism and the prophylactic property of this enhanced antioxidant status in ameliorating diseases in humans are warranted.

References:

- 1. Sjodin B, Hellsten-Westing Y, Apple FS. Sports Med 1990; 10(4):236-254.
- 2. Marzatico F, Pansarasa O, Bertorelli L, et al. J. Sports Med Phys Fitness 1997; 37(4):235-239.
- 3. Pham-Huy LA, He H, Pham-Huy C. Int J Biomed Sci 2008; 4(2):89-96.
- 4. Lobo V, Patil A, Phatak A, Chandra N. Pharmacognosy Rev 2010; (8):118-126.
- 5. Kunwar A, Priyadarsini KI. J Med & Allied Sci 2011; (2):53-60.
- 6. Halliwell B. Nutr Rev 2012; 70(5):257-65.
- 7. Sesso HD, Buring JE, Christen WG, et al. JAMA 2008; (18):2123-2133.
- 8. Lippman SM, Klein EA, Goodman PJ, et al. JAMA 2009; 301(1):39-51.
- 9. Lee IM, Cook NR, Gaziano JM, et al. JAMA 2005; 294(1):56-65.
- 10. Qin X, Cui Y, Shen L, et al. in Int J Cancer 2011; 133(5):1033-1041.
- 11. Gifkins D, Olson SH, Demissie K, et al. Cancer Causes Control 2012; 23(6): 887–895.
- 12. Thompson CA, Habermann TM, Wang AH, et al. Int J Cancer 2010; 126(4):992-1003.
- 13. Riboli & Norat. Am J Clin Nutr 2003; 78 (3):559S-569S.
- 14. Pavia M, Pileggi C, Nobile CG, et al. Am J Clin Nutr 2006; 83(5):1126-1134
- 15. Conway DI. Evid Based Dent 2007; 8(1):19-20
- 16. Siervo M, Lara J, Ogbonmwan I, et al. J Nutr 2013;143(6):818-26

- 17. Cooper CE, Vollaard NB, Choueiri T, et al. Biochem Soc Trans. 2002; 30(2):280–285
- 18. Urso ML, Clarkson PM. Toxicology 2003; 189 (1-2):41–54.
- 19. Nojima H, Watanabe H, Yamane K, et al. Metabolism 2008; 57(2):170–176.
- 20. Golbidi S, Badran M, Laher I. Experimental Diabetes Research 2012: Article ID 941868.
- 21. Radak Z, Sasvari M, Nyakas C, et al. Arch Biochem Biophy 2000: 383(1) 114–118.
- 22. Hollander J, Fiebig R, Gore M, et al. Am J of Physiol 1999; 277(3): R856–R862.
- 23. Ferraria GSL, Ferrari CKB. Frontiers in Life Science 2011; 5(3–4): 81–90.
- 24. Marciniak J, Brzeszczyńska K, Gwoździński, et al. Biol Sport 2009; 26(3): 197-213.
- 25. Berzosa C, Cebrian I, Fuentes-Broto, L et al. J Biomed Biotech 2011; Article ID 540458.
- 26. Atiqah WAN, Ooi FK, Chen CK, et al. Sport Exerc Med Open J. 2015; 1(3): 71-80.
- 27. Sahrir NA, Ooi FK, Chen CK, et al. JPES 2017; 17 (Suppl 1):48 59.
- 28. Tavafzadeh SS, Ooi FK, Chen CK, et al. J Exerc Sports Orthop 2015;2(2):1-8.
- 29. Tavafzadeh SS, Ooi FK, Chen CK, et al. (PhD thesis, Universiti Sains Malaysia)