

Antioxidants Supplementation and Exercise Performance

補充抗氧化劑對運動能力的影響

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Abstract

Physical exercise is known to have many beneficial effects. However, intense physical training can concomitantly result in increased free radical production in human body, thereby inducing a greater oxidative-stress that may cause damage to the cells. In this article, the mechanism of free radicals formation, the assessment of oxidative stress in human, and the defense system in the body against the anti-oxidative damage will be discussed. Studies in the effect of antioxidant supplementation on exercise performance will also be reviewed. Research findings suggest that supplementation of antioxidants can reduce the oxidative damage to muscle and other tissues induced by vigorous exercise. However, the effect of such supplementation on exercise performance remains unclear.

摘要

雖然運動對人體所產生的益處已被廣泛肯定，可是高強度的運動訓練卻會增加體內自由基的濃度，從而損害身體的組織。本文綜述自由基形成的機制，身體對自由基的反應，與及如何量度自由基對身體的損害和補充抗氧化劑對運動能力的影響。研究證實補充抗氧化劑能有效減低身體受自由基的傷害，但能否增進運動能力則尚需探究。

Introduction

It is well-established that regular exercise can lead to greater muscular strength and endurance (Poehlman & Melby, 1998), improved cardiovascular functions (Laughlin, 1999), optimal body composition (Martinez, 2000; Votruba et al., 2000) and decreased risk of osteoporosis (Lane et al., 2000; Nichols et al., 2000). However, intensive physical training can concomitantly result in increased free radical production in human body, thereby inducing a greater oxidative-stress that may damage the muscles, liver, blood cells and other tissues (Evans, 2000; Ji et al., 1998; Packer, 1997).

Free radical is chemically defined as a molecule capable of independent existence that contains one or more unpaired

electrons in the valence shell (Halliwell & Chirico, 1993). Being highly reactive species, free radicals can interact with numerous biologic molecules such as proteins, lipids or DNA and set in motion a series of damaging reactions inside the body (Kanter, 1995).

Formation of Free Radicals During Exercise

There are several mechanisms by which free radicals are produced inside the human body. One is via an electron 'leak', probably at the ubiquinone-cytochrome b level of the mitochondrial electron transport chain, which produces superoxide radical (Sjodin et al., 1990). It has been estimated that for every 25 oxygen molecules reduced through the cytochrome chain during normal respiration, one free radical is produced (McCord, 1979). Mitochondrial

superoxide production is greatly increased during exercise as the rate of oxygen consumption of the whole body may increase 10- to 15- fold and the oxygen flux in an active muscle may increase 100- fold during exercise (Clarkson, 1995; Sen, 1995).

Another possible mechanism is called ischaemia-reperfusion. During exercise, blood flow is shunted away from many organs and tissues to the working muscles. Some regions of the body may experience hypoxia. At the cessation of exercise, these regions then undergo reoxygenation, and such reoxygenation may lead to the burst of reactive oxygen species production that occurs after ischaemia-reperfusion (Wolbarsht & Fridovich, 1989). Besides, free radicals can also be generated in the auto-oxidation of catecholamines, which levels are increased many-fold during exercise (Singh, 1990). The superoxide radicals produced through those mechanisms mentioned can react with each other in the presence of protons producing hydrogen peroxide. When the hydrogen peroxide reacting with the transition metals, hydroxyl radicals, which is one of the most reactive and destructive species known, are produced (Packer, 1997).

Antioxidant Nutrients

As the most well known antioxidant nutrients, vitamin C and vitamin E play a vital role in protecting us against tissues' or cells' damage by the free radicals (Kanter & Williams, 1995). Vitamin C is the major antioxidant vitamin in aqueous environments inside the body. It is water-soluble and can exist in two forms, i.e. ascorbic acid and dehydroascorbic acid. These two forms can interconvert and give the antioxidant capabilities (Moser & Bendich, 1991). In the process of an ascorbic acid molecule being converted to dehydroascorbic acid, the ascorbic acid molecule can scavenge one free radical molecule, and thus neutralizing the reactive oxygen species such as superoxide and hydrogen peroxide (Moser & Bendich, 1991). The dehydroascorbic acid can then be reduced back to ascorbic acid by reduced glutathione (Packer, 1997).

Vitamin E is also fat-soluble and comprised of a family of hydrocarbon compounds characterized by a chromanol ring with a phytol side chain referred to as tocopherols and tocotrienols. Tocopherols possess a saturated phytol side chain whereas the side chain of tocotrienols has three unsaturated residues (Wang and Quinn, 1999). Because of the lipophilic properties of the vitamin, it partitions into lipid storage organelles and cell membranes and acts as the major antioxidant vitamin in the lipid environments. It can scavenge free radicals, inhibit the initiation and chain propagation of lipid peroxidation and hence protect cellular structures against free radical damage (Serafini, 2000). When a lipid peroxy radical collides with vitamin E, the peroxy radical is converted to a relatively unreactive hydroperoxide, while the vitamin E

molecule is converted to the vitamin E radical. Vitamin E radical is relatively unreactive and is either reconverted back to vitamin E or undergoes further reaction to harmless by-products (Zhang & Omaye, 2000).

Recent evidence demonstrated that vitamin C and vitamin E are tightly interlinked with each other (Hamilton et al., 2000). Chan et al. (1991) and Sies et al. (1995) have both illustrated that vitamin C is capable of regenerating intact vitamin E from its radical form. Hence the loss or consumption of vitamin E can be kept low. It was found that green vegetables, citrus fruits, tomatoes, berries and potatoes are rich in vitamin C, whereas fortified dairy products and some meat and fish have minor amount of vitamin C (Levine et al., 1995). Vitamin E can be found mostly in plant oils because only plants can synthesize it. Soybean, corn, safflower, and cottonseed oils are rich in vitamin E, while there are only moderate amounts of vitamin E in meat and dairy products (Murphy et al., 1990).

Assessment of Oxidative Damage

Direct or indirect measurement can be used to find out the evidence of increased free radical production during exercise. Electron spin resonance [ESR or also known as electron paramagnetic resonance (EPR)] spectroscopy is widely used in direct measurement of free radicals production by detecting free radical species, such as superoxide and hydroxyl free radical, in the blood sample or muscle sample (Alessio, 1993; Ashton et al., 1998; Guo & Packer, 1999).

Assessment of oxidative damaged biomolecules is the indirect method of free radicals measurement. It involves the detection of by-products from DNA oxidation, protein oxidation and lipid peroxidation (Duthie, 1999). 8-oxoGua, protein carbonyls and aldehydes are the by-products from DNA oxidation, protein oxidation and lipid peroxidation respectively (Han, 2000; Packer, 1997; Poulsen et al, 1996). High performance liquid chromatography (HPLC), colorimetric assay, enzymatic assay and immunological methods can also be used to measure the concentrations of the above by-products (Alessio, 1993; Han, 2000; Jackson, 1999; Levine et al., 1994).

Effects of Antioxidant Supplementation on Exercise

The effects of antioxidant supplementation on exercise-induced oxidative damage to the tissues and exercise performance have been investigated (Packer, 1997). Dykens (1994) reported that humans taking 600mg vitamin E three times daily for two weeks showing a decreased of lipid peroxidation products during exercise. Alessio et al. (1997) found that for the same group of athletes, their blood protein carbonyls concentrations after 30 min submaximal

exercise were significantly lower when they were supplemented with vitamin C (1g/day for two weeks). The authors concluded that exercise-induced oxidative damage is lower when subjects are supplemented with vitamin C. Sanchez-Quesada et al. (1998) also found that 1g ascorbic acid supplementation immediately before a 4-h athletic race can inhibit the exercise-induced lipid oxidation. Besides, Ashton et al. (1999) demonstrated that vitamin C supplementation to the healthy subjects after exercise can prevent the exercise-induced oxidative damage.

The effect of antioxidant supplementation on professional basketball players was examined in a recent study (Schroder et al., 2000). The subjects of this particular study were competing in the First Spanish League. The players were divided into two groups who received either mixture of 600mg alpha-tocopherol and 1000mg vitamin C pills or placebo over 32 days during a regular competition season. The results showed that the oxidative-stress was lower in the antioxidants supplement group as their plasma lipoperoxide concentration was significantly lower than the counterparts.

Whilst research suggests that antioxidant supplementation may be able to reduce the oxidative damage to muscle and other tissues, the majority of the well-controlled studies have reported no significant effect on physical performance from vitamin C and vitamin E supplementation (Clarkson & Thompson, 2000; Neiman, 1999; Takamami et al., 2000). The variables which are used to assess the effect of antioxidant supplement on exercise performance include maximal oxygen uptake, heart rate after exercise, blood lactic acid level and the time of onset of muscle fatigue (Gerster, 1989). Rokitzki et al. (1994) evaluated the effects of five months of vitamin E supplementation on aerobic exercise in 30 top-class cyclists. Heart rates and lactate concentrations measured in the vitamin E supplemented and placebo groups were not significantly different. Physical performance did not improve in the vitamin E supplemented group compared with the placebo group. Oostenbrug et al. (1997) also examined the effect of vitamin E supplementation on endurance cycling performance and red blood cell deformability. Twenty-four trained cyclists received three weeks of fish oil with vitamin E supplementation, the exercise performance and RBC deformability showed no difference compared with the trial without vitamin E supplementation.

Recently, Nielsen et al. (1999) evaluated the effect of antioxidant supplementation on maximal oxygen uptake and muscle fatigue during cycling. Seven male triathletes received daily oral antioxidant supplementation in capsule form including 600mg vitamin C and 270mg vitamin E or placebo over a six-week interval. Maximal oxygen uptake did not change between the antioxidant and placebo trials. Similarly no difference in the time of onset of muscle fatigue was found between the two trials.

Conclusion

Research findings suggest that supplementation of antioxidants can reduce the oxidative damage to muscle and other tissues induced by vigorous exercise. However, the effect of such supplementation on physical performance remains unclear, even though some physiological parameters may change after antioxidant supplementation. Further research is needed on this area of sport nutrition to draw a conclusion on the effect of antioxidant supplementation on physical performance.

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