Free radicals, exercise and antioxidant supplementation

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Proposed mechanisms of exercise-induced free radical generation

As a result of studies conducted by researchers such as Dillard *et al.* (1976, 1978) and Tappel (1973) in the 1970s, and Davies *et al.* (1982) and Jenkins *et al.* (1982) in the early 1980s, a link between physical exercise, elevated O_2 consumption, and free-radical production has been established. It has been estimated that for every twenty-five O_2 molecules reduced by normal respiration, one free radical is produced (McCord, 1979). Coupled with the fact that the rate of whole-body O_2 consumption during exercise may increase 10- to 15-fold, and that O_2 flux in an active muscle may increase 100-fold (Sen, 1995), it is easy to understand how an increased metabolism can greatly increase free-radical production. This relationship between an apparently healthy act (exercise) and a series of reactions that can, among other things, damage various organ systems has been troublesome to many researchers. The so-called 'oxygen paradox' evoked by exercise prompted Jenkins (1993) to state that: 'elemental and gaseous oxygen presents a conundrum in that it is simultaneously essential for and potentially destructive to human life'.

It should be noted, however, that an elevation in exercise-induced O_2 consumption is not the only mechanism that has been linked to the production of free radicals in physically-active individuals. For example, transient tissue hypoxia, which may occur with heavy-weight lifting or high-intensity anaerobic work can lead to an increase in hydrogen ions which can, in turn, react with superoxide anions to produce additional reactive oxygen species (Jenkins, 1993). Tissue hypoxia can also lead to the freeing of transition metals such as Fe and Cu from their normal transporters. These free metals can further catalyse free-radical reactions.

It has also been suggested that reperfusion of hypoxic muscle after a period of particularly stressful activity can result in post-ischaemic injury, including oedema formation, microvascular dysfunction, and muscle necrosis (Rubin *et al.* 1996). The widely held hypothesis regarding ischaemia–reperfusion injury suggests that xanthine is formed after ATP and other high-energy phosphates are depleted via anaerobic metabolism. Simultaneously, Ca influx and protease (*EC* 3.4.21.39) activation in damaged tissues stimulates the conversion of xanthine dehydrogenase (*EC* 1.1.1.204) to xanthine oxidase (*EC* 1.1.3.22). With reperfusion and the re-introduction of molecular oxygen into the microenvironment, xanthine oxidase catalyses xanthine to uric acid (Hamvas *et al.* 1992). Toxic reactive oxygen metabolites are produced as metabolic byproducts.

Tissue trauma or inflammation, which result in the activation of phagocytic cells such as neutrophils, have also been implicated in free-radical production via activity of enzymes such as NADPH oxidase (Ward *et al.* 1990).

Other mechanisms purported to induce oxidant stress during exercise include substrate depletion (purportedly via a diminution in pentose shunt-generated NADPH activity and, ultimately, a decline in tissue glutathione reductase (*EC* 1.6.4.2) activity; Dernbach *et al.* 1993) and mechanical trauma (via joint compression of synovial fluid; Symons, 1988). Further, a study by Salo *et al.* (1991) suggested that exercise-induced hyperthermia could trigger oxidative stress by promoting mitochondrial uncoupling, loss of respiratory control, and ultimately, free-radical production.

Despite the large number of potential mechanisms that apparently promote free-radical generation under exercising conditions, it should be pointed out that the true biological implications of these effects have yet to be determined. In discussing the role of oxidants in human diseases, Halliwell (1987) questioned whether free radicals are a major cause of tissue injury or are merely produced during injury. He attributed the relative lack of understanding of this issue to inadequate experimental techniques, as well as an overemphasis on lipid peroxidation as a mechanism of oxidant injury. Although Halliwell (1987) was discussing the oxidant–disease relationship, a review of the exercise literature would suggest that the points he raised are pertinent to the exercise literature as well.

Measurement of free-radical production in exercise studies

Very few studies have actually measured exercise-induced free-radical production. Davies *et al.* (1982) were one of the only groups to directly measure free radicals in animal tissue via electron paramagnetic resonance. Although their study was published about 15 years ago, their results have never been replicated, and their ability to have accurately

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quantified free-radical generation under biological conditions has been questioned.

Most of the studies that have looked at exercise-induced oxidant stress have actually measured indirect markers of lipid peroxidation, and not free-radical generation. The majority of these markers, including tissue malondialdehyde production (via GC or HPLC), conjugated diene formation, chemiluminescence, and expired breath pentane and ethane, represent minor reaction pathways in highly-complex systems (Halliwell & Chirico, 1993). Most have been criticized for their lack of specificity, sensitivity and reproducibility (Halliwell & Gutteridge, 1985; Wong *et al.* 1987). Nevertheless, much of our understanding and beliefs regarding exercise-induced free radicals are based on the results of studies that have employed combinations of these methodologies. Coupled with the fact that these studies have involved various modes of exercise and numerous population groups, it is understandable that our current knowledge of the exercise–free radical relationship is tenuous. New, promising methods, such as the use of spin traps, monoclonal antibodies and flow cytometry, should shed greater light in the future on the role of free radicals in exercise-induced tissue damage. A few recent studies have looked at the effects of exercise on the susceptibility of the LDL subfraction to oxidation; results of these studies suggest that strenuous exercise promotes oxidative changes to LDL (Sanchez-Quesada *et al.* 1995). This methodology represents a novel approach to studying exercise-induced oxidative stress, and preliminary results imply that exercise does, in fact, promote free-radical generation.

Protection from free-radical generation

Despite the uncertainty surrounding our understanding of the exercise–free radical relationship, the relatively consistent finding of an increase in antioxidant enzyme activity in various tissues of trained subjects (Jenkins *et al.* 1982; Higuchi *et al.* 1985; Kanter *et al.* 1985; Ohno *et al.* 1986; Alessio & Goldfarb, 1988; Ji *et al.* 1988; Girten *et al.* 1989; Hamaren *et al.* 1993) is highly suggestive of a protective adaptation to the habitual stress of exercise. It also suggests that a bout of exercise may outstrip the inherent capacity of the protective endogenous antioxidant enzyme system (Ohno *et al.* 1986; Pincemail *et al.* 1990), particularly in the non-habituated exerciser, necessitating greater protection. The findings of various research studies have suggested that this added protection could come in the form of an exogenous supplement (Gohil *et al.* 1986; Simon-Schnass & Pabst, 1988; Sumida *et al.* 1989; Kanter *et al.* 1993; Rokitzi *et al.* 1994; Hartmann *et al.* 1995).

Nutritional supplements

The most-well-researched nutritional antioxidants in exercising subjects have been the vitamins E and C. Vitamin E is considered to be extremely important because of its association with cell membranes (Bjorneboe *et al.* 1990). Vitamin C also serves as an important antioxidant and free-radical scavenger, it is extremely labile, and it is highly susceptible to a number of environmental factors, including temperature, smog, and cigarette smoke (Zapsilas & Anderle Beck, 1985).

There is little doubt that vitamin deficiencies in general can impair the ability of an individual to perform physical work. Animal studies by Gohil *et al.* (1986) and Davies *et al.* (1982) indicated that vitamin E deficiency impaired performance, and Salminen & Vihko (1983) demonstrated greater susceptibility to lipid peroxidation in rats fed on a vitamin E-deficient diet. However, vitamin deficiencies (particularly deficiencies of vitamins E and C) in healthy human subjects are rare, and findings from well-fed, exercising subjects who consume vitamin supplements have produced contradictory results.

Studies by Sumida *et al.* (1989), Cannon *et al.* (1990), and Rokitzi *et al.* (1994) reported post-exercise declines in serum enzymes indicative of muscle tissue damage in subjects who consumed 300–800 mg d-α-tocopherol acetate for 4–8 weeks (subjects of Rokitzi *et al.* (1994) consumed 200 mg vitamin C/d as well). Hartmann *et al.* (1995) demonstrated that short-term vitamin E supplementation (800 mg administered 12 and 2 h before exercise and 22 h post- exercise) reduced DNA damage in peripheral leucocytes following exhaustive exercise, and Kanter *et al.* (1997) recently reported a 35 % increase in T-lag time (indicative of a diminished LDL oxidation rate) in subjects who consumed 1000 mg d-α-tocopherol acetate daily for 1 week before exercise.

Various studies have also demonstrated beneficial physiological effects of vitamin C supplementation in physically-active people. Jakeman & Maxwell (1993) reported greater recovery of maximal voluntary contraction in subjects who consumed 400 mg vitamin C/d for 21 d. Kaminski & Boals (1992) reported less calf soreness in subjects who consumed 3 g vitamin C for 3 d before and 4 d after strenuous calf exercise. Peters *et al.* (1993) noted fewer cases of self-reported upper respiratory tract infection in runners who consumed 600 mg vitamin C/d for 3 weeks before a 42 km road race.

All the previously mentioned studies suggest tangible benefits of vitamin E and C supplementation in combating detrimental physiological processes that may be initiated by physical activity. Coupled with the results of various studies (Tappel, 1973; Dillard *et al.* 1978; Packer, 1984; Sumida *et al.* 1989; Kumar *et al.* 1992; Kanter *et al.* 1993) that have demonstrated post-exercise reductions in lipid-peroxidation markers in subjects who consumed supplements before exercise, it would appear that vitamin supplementation by physically-active people is warranted.

However, it should be noted that contradictory data do exist. Studies by Warren *et al.* (1992), Robertson *et al.* (1991), and Helgheim *et al.* (1979) have reported no effect of vitamin E supplementation on changes in post-exercise serum enzyme levels, and Nieman *et al.* (1997) found no beneficial effects of vitamin C supplementation on markers of immune function following a 2·5 h run. Results of studies that utilized antioxidant mixtures (Viguie *et al.* 1989; Kanter & Eddy, 1992; Kanter *et al.* 1993) have also produced contradictory findings.

Despite conflicting results in the literature, one could argue that physically-active individuals might benefit from supplementation for a number of reasons. Recent data

indicate that the 'typical' North American diet provides about two-thirds of the recommended daily intake of vitamin E (Goldfarb & Sen, 1994). Furthermore, data from the ongoing National Health and Nutrition Examination Survey longitudinal study in the USA suggest that 90 % of the study participants failed to ingest the recommended daily intake of five servings of fruits and vegetables daily (Patterson *et al.* 1990). Of course, these dietary staples are excellent sources of numerous vitamins and minerals; failure to ingest sufficient quantities of fruits and vegetables would also suggest suboptimal intake of various antioxidant nutrients.

Potential of 'non-traditional' nutrients

The present paper has focused primarily on the potential of more 'traditional' nutrients (particularly vitamins E and C) for physically-active people. It should be noted, however, that a burgeoning area of research involves the study of less-well-researched nutrients that presently exist in many of the common foods that we eat (Niki *et al.* 1985; Namiki, 1990). These 'phytonutrients' have been studied more in relation to various disease conditions, and generally in *in vitro* systems or in animal models. Nevertheless, some of these compounds do warrant mention in a discussion of the antioxidant needs of physically-active individuals; their efficacy may or may not be demonstrated in future studies.

Examples of these 'non-traditional' compounds include various phenolics and isoflavones, such as genestein in soyabean, caffeic and ferulic acids in oats, and lycopene in tomatoes. Preliminary research suggests, for example, that caffeic and ferulic acids can inhibit rat liver xanthine oxidase activity (Talla *et al.* 1996), and that genestein from soyabean can inhibit *in vitro* lipoprotein oxidation in serum (Hodgson *et al.* 1996). Furthermore, green-tea catechins have exhibited anti-mutagenic effects in cultured Chinese hamster cells (Kuroda, 1996). Certainly, most of this research is in its preliminary stages. Much more research will need to be done to further establish the properties, efficacy and safety of these compounds before they are used as food additives and supplements. Nevertheless, preliminary findings have demonstrated that these compounds possess antioxidant potential. They also underscore the importance of eating whole foods, as opposed to relying heavily on supplements as a means of boosting antioxidant intake. As stated previously, the majority of these compounds exist in common foods. Failure to eat a diet that contains a variety of foods diminishes the ability of the individual to consume many of these natural antioxidant compounds.

Summary

Although conflicting data exist, the preponderance of available information suggests that physical exercise promotes an increase in free-radical generation. However, few studies have actually measured exercise-induced free radicals directly, primarily because of a lack of sophisticated methodologies to measure this phenomenon. Instead, researchers have relied heavily on the measurement of lipid peroxidation as the principal indicator of exercise-induced free radicals. It should be noted that free radicals can also alter and inactivate enzyme complexes, damage DNA and RNA, and promote mutations and cancer, among other activities. However, there have been few reported studies dealing with exercise and oxidant stress which have measured these outcomes. It should also be noted that free-radical species are continuously produced in the human body, and that some have beneficial effects (Arouma, 1994), notably as a part of the body's natural immune system. It is not presently known if long-term ingestion of antioxidant compounds will affect these positive aspects of free-radical generation.

The preponderance of available evidence suggests that antioxidant supplementation, particularly with the vitamins C and E, has favourable effects on markers of lipid peroxidation following exercise. Although the physiological implications of these effects remain to be elucidated, the prudent use of an antioxidant supplement can provide insurance against a suboptimal diet and/or the elevated demands of physical activity. Future research may uncover additional nutritional antioxidants that can benefit the physically-active individual.

Numerous additional questions regarding the antioxidant needs for physical activity remain to be answered. Little is known about the needs of physically-active women, particularly those who habitually consume an energy-restricted diet, or the effects of monthly menstrual blood loss (coupled with exercise) on antioxidant requirements. The needs of the 'weekend warrior' (an individual who participates in strenuous activity sporadically) *v.* those of the habitual exerciser have not been addressed adequately. The needs of the ageing athlete are largely unknown; a study by Meydani *et al.* (1993) suggested that elderly, physically-active people can benefit from antioxidant supplementation more than their younger counterparts, but follow-up studies have not been reported to-date.

Dietary issues also need to be addressed. How much supplementation is too much, and can chronically-elevated antioxidant intakes have an adverse impact on the positive effects of free radicals in living systems? Does a high-carbohydrate diet raise antioxidant needs, particularly of the fat-soluble vitamin E? Does a high polyunsaturated fat intake, or increased Fe intake affect needs? Obviously, there are a number of issues regarding the antioxidant needs of the physically-active individual that need to be elucidated. Future research utilizing newer, more sophisticated methodologies should provide answers to many of these questions.

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