Zeitschrift für
Ernährungswissenschaft Z Ernährungswiss 30:89–97 (1991)

Originalarbeiten

Function of vitamin E in physical exercise: a review

H. Gerster

Department of Human Nutrition and Health, F. Hoffmann-La Roche & Co Ltd, Basel, Switzerland

Summary: Even though vitamin E may not improve physical achievements in sports competitions, as shown in several swimming experiments, it is important for the health of skeletal muscle: in its role as the major lipid-soluble chainbreaking antioxidant in lipid cell membranes, vitamin E protects muscle tissue in aerobic exercise, in which oxygen metabolism and, consequently, free radical production are greatly accelerated. Animal studies in several laboratories have shown that endurance exercise results in the same type of oxidative muscle damage as does vitamin E deficiency: there is an increase in the peroxidation products pentane and malondialdehyde and in enzymes leaked from muscles to plasma. Oxidative tissue damage in vitamin-Edeficient animals is exacerbated by endurance training and, conversely, it is reduced by high-dose vitamin E supplementation; also, preliminary studies in humans have demonstrated antioxidant protection by high-dose vitamin E supplementation. After endurance exercise leakage of enzymes into the plasma and output of pentane in the breath were significantly reduced. During a high-altitude expedition in the Himalayas, protection was shown to be significantly better in the supplemented group than in the placebo group, as determined by anaerobic threshold and pentane exhalation.

Zusammenfassung: Ältere Studien haben gezeigt, daß Vitamin E die sportliche Leistungsfähigkeit bei Schwimmern nicht zu steigern vermag. Trotzdem ist das Vitamin für die Skelettmuskulatur der Sportler wichtig: Als das bedeutendste lipidlösliche Antioxidans in Zellmembranen schützt Vitamin E das Muskelgewebe bei aeroben physischen Belastungen, bei denen der Energiestoffwechsel stark beschleunigt ist, vor übermäßiger Peroxidation von ungesättigten Fettsäuren in Zellmembranen. In verschiedenen Labors führte Ausdauertraining von Versuchstieren zu denselben oxidativen Muskelschädigungen wie Vitamin-E-Mangel: Die Peroxidationsprodukte Pentan und Malondialdehyd waren erhöht, und Muskelenzyme waren vermehrt ins Plasma gelangt. Bei Tieren mit Vitamin-E-Mangel werden die Muskelschäden durch Ausdauertraining verstärkt, während sie durch Vitamin-E-Supplementierung vermindert werden. Präliminäre Studien am Menschen weisen ebenfalls auf eine antioxidative Schutzwirkung von Vitamin E gegen Muskelschädigung hin. Das zeigt sich in reduzierter Bildung von Pentan und in vermindertem Übertritt von Muskelenzymen ins Plasma nach Ausdauertraining. Bei einer Expedition von Bergsteigern ins Himalajagebiet stellten die Autoren ebenfalls eine signifikante Schutzwirkung von Vitamin-E-Supplementierung fest. Diese äußerte sich in einer erhöhten anaeroben Schwelle und in reduzierter Pentankonzentration im Atem bei Belastung.

Key words: Vitamin E; antioxidant; free radical muscle damage; exercise

Schlüsselwörter: Vitamin E, Antioxidans, Muskelschädigung, Ausdauertraining

Introduction

New biochemical and physical methods have identified vitamin E as the major lipid-soluble, free radical scavenging antioxidant in biological membranes. Its function is to protect the polyunsaturated fatty acids (PUFAs) (essential constituents of biomembranes) against oxidative damage mediated by oxygen free radicals (molecules containing a single unpaired electron). By scavenging oxygen free radicals, vitamin E interrupts the chain reaction of free radical production. It thus maintains the structural and functional integrity of cell membranes. Other antioxidants, with their own specialized functions, include vitamin C and carotenoids as well as antioxidant enzymes, among them glutathione peroxidase and reductase, superoxide dismutase and catalase. These antioxidants constitute an intricate defense network against uncontrolled oxidation. When the amount of free radicals generated exceeds the capacity of the antioxidants, tissue injury occurs (1–3).

Early studies in human volunteers indicated that dietary supplements of wheat germ oil containing vitamin E led to improved performance in bicycle riding and treadmill running beyond the effect of training (4), which aroused interest in vitamin E as a potential means of enhancing athletic performance. This hypothesis has been supported by findings in animal experiments showing that necrotizing myopathy is one of the most common manifestations of vitamin E deficiency. Muscular weakness of deficiency affects virtually all skeletal muscles in a symmetrical way. Ultrastructural damage is seen in mitochondria, the microcirculation, and fibroblasts (5). However, several controlled studies in trained and untrained swimmers have failed to show an improvement in endurance capacity, muscular strength, and other criteria of physical performance by vitamin E supplementation (6–9).

Oxidative muscle damage induced by exercise

On a biochemical level, vitamin E could play a protective role in aerobic exercise because oxygen consumption and metabolism in skeletal muscle are greatly accelerated, and increased mitochondrial respiration results in bursts of reactive oxygen species which interact with membrane lipids, eventually leading to membrane disruption if antioxidant defense systems are overwhelmed (10). The nature of the muscle damage is not yet known in detail, but may be associated with loss of cellular calcium homeostasis leading to an inability of mitochondria to retain calcium. Moreover, free radicals are known to cause considerable damage to muscle proteins and may release lysosomal enzymes by disrupting lysosomal membranes (11).

It has been proposed that damage from free radicals can occur, not only in hyperoxia with enhanced oxygen metabolism, but also in hypoxia when oxygen supply is reduced. Hypoxia, which is accompanied by reduced ATP synthesis, may alter ion transport systems thus disturbing calcium homeostasis. The reductive stress of hypoxia may be caused by an imbalance between electron acceptors and reducing equivalents. In some studies, increased production of superoxide and malondialdehyde, a reaction product of peroxidation, were noted during hypoxia (12). In isolated

guinea pig colon, vitamin E had a dose-dependent protective effect against hypoxia (13). Vitamin E has been shown to reduce damage induced in rabbit heart muscle by hypoxia as well as damage by the following reperfusion with oxygen-rich blood (14). Theoretically, milder forms of ischemia-reperfusion injury could arise upon the rapid descent from a hypoxic environment at high altitude to a normoxic environment near sea level.

Assessment of muscle damage

Muscular damage due to exercise or to vitamin E deficiency involves leakage of enzymes from muscles to plasma and can be assessed by measuring enzyme activity in muscle or plasma. The enzymes include creatine phosphokinase (CPK), lactic dehydrogenase (LDH), and glutamic oxaloacetic transaminase (GOT). A study in vitamin E depleted rats demonstrated reduced activity of these enzymes in heart and skeletal muscle which correlated with the degree of histopathological alterations. There was a concomitant increase in enzyme activity in the blood, suggesting leakage of enzymes from damaged tissue into blood (15). Another indicator is the glutathione system which serves to remove peroxides from tissues, but may be oxidized during exercise stress (10).

Analysis of free radical production is more complex, because radicals are highly reactive and short-lived chemical species which are technically difficult to measure directly. The most common assays, therefore, assess breakdown products of free radical attack on lipid membranes. One of these products is malondialdehyde (MDA) which is determined indirectly as thiobarbituric acid reactive substance. In isolated skeletal and cardiac muscle from vitamin E deficient mice the rate of MDA formation was increased by almost 50 %. Vitamin E deficiency also reduced the activity of the selenium-dependent enzyme glutathione peroxidase in skeletal muscle (16). The analysis of pentane in breath, a breakdown product of n-6 fatty acid, has been standardized to reflect lipid peroxidation (17). Pentane exhalation is inversely proportional to plasma vitamin E levels and increases with increasing intake of PUFAs. In vitamin E depleted rats, supplementation of the corn oil diet with 40 IU vitamin E/kg diet reduced pentane exhalation to one-sixth (18). Recently, electron-spin resonance spectrometry has been adapted for use in biological material. The technique can directly detect the signals of free radicals in frozen tissue samples (19).

Exercise training in animals

During exercise, oxygen consumption can be 20–40 times higher than during rest. As an adaptive response the number and size of mitochondria are greatly increased in certain muscles. A study in rats showed that animals which were endurance trained could run six times longer than their sedentary controls. Mitochondrial contents of skeletal muscles, determined by measuring the electron transmitter ubiquinone, had doubled, and free radical concentrations in muscle and liver had increased two- to threefold after exercise to exhaustion, mitochondria being the

major source of free radicals (20). In a controlled experiment pretreatment of mice with vitamin E significantly increased their endurance to swimming. Comparable results were obtained with spin-trapping compounds which inactivate free radicals by forming stable adducts (21). This indicates that the effect of vitamin E was equally due to inactivation of free radicals.

In rats undergoing endurance training, vitamin E was depleted from muscle and liver tissue more rapidly than in sedentary rats (22). Training of mice for several months showed that older animals were more susceptible to vitamin E depletion than younger ones (23). While causing increased vitamin E depletion, training also appears to afford a certain protection by leading to an increase in antioxidant enzymes in rat skeletal muscle and heart (24) and a decrease in MDA production (25). This may at least partly compensate for the loss of vitamin E.

Investigators at the University of California at Berkeley showed that 2-month endurance training of rats led to the same type of damage in hindleg muscles as dietary vitamin E deprivation. Muscle tissue levels of vitamin E were lower in endurance trained than in sedentary animals, whether they had been fed a deficient or a control diet (22). In another experiment control rats exercised to exhaustion developed the same degree of oxidative damage as vitamin E deficient sedentary rats. Vitamin E deficient untrained rats exhibited a 40–50 % reduction in endurance capacity compared with non-deficient untrained controls (20).

Oxidative tissue damage induced by vitamin E deficiency was potentiated in rats by endurance training (26). Similar findings were obtained in vitro upon incubation of mitochondria from vitamin E deficient and control rats when they were exposed to different types of oxidant stress (irradiation with visible light, various temperatures). The greater sensitivity to oxidant stress of mitochondria from deficient animals was more conspicuous in muscle than in liver mitochondria (27).

A study carried out in Great Britain confirmed that skeletal muscles of vitamin E deficient rats suffered greater damage from contractile activity than muscles from supplemented animals. Release of creatine phosphokinase into plasma served as a measure of muscle damage. In vivo, no increase in MDA was noted in depleted animals after free radical induction. However, muscles from deficient mice showed reduced antioxidant capacity in vitro, expressed as an increase in MDA (28).

A recent study demonstrated that in rats receiving a vitamin E sufficient diet the adaptive increase in mitochondrial content of muscle (measured as ubiquinone) was not accompanied by an increase in muscle vitamin E concentration. In deficient animals with very low vitamin E tissue stores, however, the elevated mitochondrial content was paralleled by elevated vitamin E concentrations, which may reflect redistribution or possibly regeneration of vitamin E in deficient animals. Detraining of the deficient rats markedly lowered these levels (29).

Biochemical effects of exercise in humans

Human volunteers expired significantly higher concentrations of pentane after exercise on a bicycle ergometer than at rest. Supplementation of

the subjects with 1200 IU vitamin E (dl-α-tocopheryl acetate) daily for 2 weeks was followed by a significant reduction of pentane exhalation after exercise while plasma vitamin E levels increased by about 240 % (30).

Endurance exercise on a bicycle ergometer in moderately trained male volunteers induced the formation of peroxides as suggested by increased concentrations of oxidized glutathione in the blood (31). Oxidation of glutathione can be prevented by 10-day supplementation with 1000 IU vitamin E daily, as shown by others (32).

When a group of 21 moderately trained or sedentary college students exercised to exhaustion on a bicycle ergometer there was a small but significant rise in the concentration of MDA in plasma. An increase in plasma enzymes suggesting peroxidation of cell membrane lipids was observed as well. Repetition of the exercise tests after 4 weeks' supplementation with 300 mg vitamin E daily showed that the production of MDA was significantly lower than at baseline without vitamin E. Leakage of enzymes into plasma was greatly reduced as well (33). The authors concluded that lipid peroxidation resulting from acute heavy exercise can be inhibited by vitamin E supplementation.

Other investigators demonstrated an exercise-induced increase in serum concentrations of CPK, LDH and aspartate aminotransferase. Exercise consisted of 10-min cycling sessions at 90 % work capacity or heavy short-term strength exercise. But supplementation of the 26 subjects with 300 mg d-α-tocopherol or placebo for six weeks showed no effect on enzyme leakage in either group (34). The reason for these discrepant results is unknown.

The role of high doses of vitamin E on adaptation to a hypoxic environment and aerobic work performance was investigated in a double-blind cross-over study. Twelve volunteers received either 1200 IU vitamin E daily for 6 weeks or a placebo preparation. This procedure was then reversed. Submaximal and maximal aerobic work performance was assessed at a height of 1524 m and at a simulated height of 4572 m. Blood lactate levels during recovery and O_2 debt were determined as well. Work capacity, which is limited by the maximal rate of oxygen delivery to, and oxygen utilization in, the working muscle, was improved by vitamin E intake at both altitudes, but more so at 4572 m (by 14.2 %) than at 1524 m (by 8.9 %). Total O_2 debt was reduced by 20.1 % at 4572 m and by 16.5 % at 1500 m. Thus, the overall positive effect of vitamin E on aerobic performance was more pronounced at the higher altitude (35).

An interesting experiment was carried out during a high mountain climbing expedition to the Himalayas where altitudes of 8049 m (Broad Peak) and 8611 m (K2) were reached. The authors' aim was to study the effect of vitamin E (400 mg dl-α-tocopheryl acetate) or matching placebos on the oxidative damage caused by prolonged hypoxia and physical exertion during the 10-week expedition. The participants included six highly trained mountain climbers both in the vitamin E and in the placebo groups. To exclude confounding effects of dietary inadequacies, the subjects additionally received a multivitamin and mineral preparation which lacked vitamin E. The two main tests performed were a) lactic acid analysis in serum after work on the bicycle ergometer as a measure of anaerobic threshold, and b) pentane exhalation as a measure of lipid

peroxidation. The lactate test was performed on day 0 at an altitude of 2500 m and subsequently on days 15, 30, and 43 at altitudes of about 5000 m; the pentane test was carried out on days 0 and 30.

The 14-day walk from an altitude of 2500 m to the base camp at 5000 m led to an improvement of the anaerobic threshold in both the vitamin E and placebo groups with a slightly, but not significantly better result in the vitamin E group. After another 2 weeks at 5000 m and various climbing expeditions, the anaerobic threshold was further increased in the vitamin E supplemented subjects while it declined in the placebo group, the difference being statistically significant. Pentane output in the breath on day 30 had doubled in the placebo group compared with day 0. In the vitamin E group pentane exhalation had remained stable, indicating that vitamin E had protected lipid membranes against peroxidation induced by hypoxia and/or exertion (36).

Conclusions

The hope entertained by many athletes that micronutrients such as vitamins and minerals will act as ergogenic aids must be abandoned. In the case of vitamin E, studies in swimmers have shown that supplementation with vitamin E does not improve swimming performance beyond the effect of training. Because genetic disposition and training to maximize the genetic potential are the major factors determining athletic achievement, and because psychological and other factors are responsible for considerable day-to-day variation in performance, any conceivable effect of a micronutrient may be too small to be measurable in competitive events. It has been estimated that – statistically – a sample size of 200 to 300 athletes would be needed in both treatment and placebo group to detect a difference of 10 s in a 400-m swim or a 1600-m run. Up to 5000 athletes per group would have to be included to demonstrate a 2-s difference in performance (37).

This does not imply that athletes can achieve their physical potential without an optimal dietary intake of vitamins and minerals, nor that for certain types of activity a particular nutrient may not be required in amounts that surpass the RDA value calculated for a moderately active person. Such a case can be made for vitamin E on the basis of preliminary findings in humans which are supported by results from animal experiments.

There exist interesting inverse correlations between the species-specific metabolic rate and life span, as recently discussed by Packer (10). Small animals like rats and mice with their comparatively accelerated metabolism have a much shorter life span than humans. It could be shown in insects that life span was closely related to their rate of oxygen consumption: if this was increased by raising the ambient temperature their life span was shortened accordingly. It therefore seemed theoretically possible that human physical exercise, which may increase oxygen consumption 20–40 times, could have some undesirable effect in the human organism.

Even though oxygen metabolism is indispensable for survival it can have toxic effects by producing free radicals, which may destroy cell membranes, proteins and genetic material in cells. Ordinarily, oxygen

turnover is compatible with life because it is counterbalanced by a protective network consisting of antioxidant enzymes and antioxidant nutrients. Cell damage occurs if prooxidant and antioxidant factors are unbalanced.

The increased metabolic turnover induced by exercise leads to a number of changes in muscle cells. As an adaptive response, there is an accelerated biosynthesis of mitochondria expressed by an increase in ubiquinone and cytochrome c, the electron transmitters in the respiratory chain. In animal experiments, moderate exercise was found to raise also the synthesis of antioxidant enzymes while muscle tissue levels of vitamin E decreased. At the same time, the production of free radicals can be greatly increased during exercise, the mitochondria being the major site of free radical production. Interestingly, vitamin E deficiency also results in increased production of free radicals and damage to skeletal muscle as shown by enzyme leakage. Myopathy is, in fact, one of the earliest manifestations of vitamin E deficiency in animals.

Muscular damage caused by vitamin E deficiency is exacerbated by endurance training, which indicates that the mechanism of action is the same. Animal studies in several laboratories have supported the relationship between free radical production leading to biochemical changes in muscle tissue and the protective action of vitamin E. The vitamin additionally interacts with other protective antioxidants as for instance with the selenium-dependent glutathione peroxidase. However, much is still unknown regarding the complex interaction between muscular exercise and the protective antioxidative network.

Preliminary studies in humans tend to confirm the observations in animals. Exercise was found to increase lipid peroxidation measured as pentane output with breath, plasma MDA or enzyme leakage. These effects were reversed by supplementation with vitamin E. The same findings could be made under hypoxic conditions on the occasion of a Himalayan expedition. In another context, an association between reduced antioxidant status and muscle damage was suggested by a study in alcoholic patients. Alcohol metabolism is associated with increased free radical production which is a possible factor in the muscle atrophy frequently noted with alcohol abuse. Those alcoholic patients showing muscle damage had significantly lower mean plasma levels of vitamin E, as well as of vitamin A and selenium than did alcoholic patients without muscle atrophy. The general nutritional status did not differ between the two groups (38). These interesting findings pointing to a potential connection between low antioxidant status and muscle damage need to be further investigated.

It is not yet possible to recognize a clear pattern showing which type of exercise may benefit from vitamin E supplementation, though endurance training and exercise at high altitude appear – at this point – to derive greater benefit from it than short-term strength exercise. This question will require systematic investigation. Moreover, the amount of vitamin E required for antioxidative muscle protection is unknown. Further studies will have to determine the appropriate dosage.

The consequences of adequate protection against exercise-induced muscle cell injury are difficult to foresee. Supplementation of athletes will have long-term effects rather than short-term ones. It may – with due

caution – be hypothesized that antioxidant protection could prevent, reduce or delay degenerative muscle damage.

References

1. Machlin LJ, Bendich A (1987) Free radical tissue damage: protective role of antioxidant nutrients. FASEB J 1:441–445
2. McCoy PB, King MM (1980) Vitamin E: its role as a biologic free radical scavenger and its relationship to the microsomal mixed function oxidase system. In: Machlin LJ (ed) Vitamin E. A Comprehensive Treatise. Marcel Dekker, New York Basel, pp 289–317
3. Burton GW, Joyce A, Ingold KU (1983) Is vitamin E the only lipid-soluble, chain-breaking antioxidant in human blood plasma and erythrocyte membranes? Arch Biochem Biophys 221:281–290
4. Cureton TK (1954) Effect of wheat germ oil and vitamin E on normal human subjects in physical training programs. Am J Physiol 179:628
5. Nelson JS (1980) Pathology of vitamin E deficiency. In: Machlin LJ (ed) Vitamin E. A Comprehensive Treatise. Marcel Dekker, New York Basel, pp 397–428
6. Sharman IM, Down MG, Sen RN (1971) The effects of vitamin E and training on physiological function and athletic performance in adolescent swimmers. Br J Nutr 26:265–276
7. Sharman IM, Down MG, Norgan NG (1976) The effects of vitamin E on physiological function and athletic performance of trained swimmers. J Sports Med 16:215–225
8. Shephard RJ, Campbell R, Pimm P, Stuart D, Wright GR (1974) Vitamin E, exercise, and the recovery from physical activity. Eur J Appl Physiol 33:119–126
9. Lawrence JD, Bower RC, Riehl WP, Smith JL (1975) Effects of α-tocopherol acetate on the swimming endurance of trained swimmers. Am J Clin Nutr 28:205–208
10. Packer L (1988) Oxygen radicals and antioxidants in endurance exercise. In: Benzi G, Packer L, Siliprandi N (eds) Biochemical Aspects of Physical Exercise. Elsevier, Amsterdam, pp 73–92
11. Jackson MJ (1990) Free radicals and skeletal mucle disorders. In: Das DK, Essman WB (eds) Oxygen Radicals: Systemic Events and Disease Processes. Karger, Basel, pp 149–171
12. Jones DP (1985) The role of oxygen concentration in oxidative stress: Hypoxic and hyperoxic models. In: Sies H (ed) Oxidative Stress, Academic Press, London, pp 151–195
13. Kelly MJ (1986) α-Tocopherol as agonist in hypoxia. J Pharm Pharmacol 38:66–68
14. Guarneri C, Ferrari R, Visioli O, Caldarera CM, Nayler WG (1978) Effect of α-tocopherol on hypoxic perfused and reoxygenated rabbit heart muscle. J Mol Cell Cardiol 10:893–906
15. Chen LH, Lin C-T (1980) Some enzymatic changes associated with pathological changes in rats with long-term vitamin E deficiency. Nutr Rep Int 21:387–395
16. Salminen A, Kainulainen H, Arstila AU, Vihko V (1984) Vitamin E deficiency and the susceptibility to lipid peroxidation of mouse cardiac and skeletal muscles. Acta Physiol Scand 122:565–570
17. Lemoyne M, Van Gossum A, Kurian R, Ostro M, Axler J, Jeejeebhoy KN (1987) Breath pentane analysis as an index of lipid peroxidation: a functional test of vitamin E status. Am J Clin Nutr 46:267–272
18. Dillard CJ, Litov RE, Tappel AL (1978) Effects of dietary vitamin E, selenium, and polyunsaturated fats on in vivo lipid peroxidation in the rat as measured by pentane production. Lipids 13:396–402
19. Davies MJ, Slater TF (1988) The use of electron-spin resonance techniques to detect free-radical formation and tissue damage. Proc Nutr Soc 47:397–405

20. Davies KJA, Quintanilha AT, Brooks GA, Packer L (1982) Free radicals and tissue damage produced by exercise. Biochem Biophys Res Comm 107:1198–1205
21. Novelli GP, Bracciotti, Falsini S (1990) Spin-trappers and vitamin E prolong endurance to muscle fatigue in mice. Free Rad Biol Med 8:9–13
22. Aikawa KM, Quintanilha AT, de Lumen BO, Brooks GA, Packer L (1984) Exercise endurance-training alters vitamin E tissue levels and red-blood-cell hemolysis in rodents. Biosci Rep 4:253–257
23. Elmadfa I, Schmidberger S, Müller H (1987) Physical exercise and vitamin E status in aged mice. Age 10:108
24. Quintanilha AT, Packer L (1983) Vitamin E, physical exercise and tissue oxidative damage. Ciba Foundation Symposium 101, Biology of Vitamin E. Pitman, London, pp 56–69
25. Alessio HM, Goldfarb AH (1988) Lipid peroxidation and scavenger enzymes during exercise: adaptive response to training. J Appl Physiol 64:1333–1336
26. Packer L (1984) Vitamin E, physical exercise and tissue damage in animals. Med Biol 62:105–109
27. Quintanilha AT, Packer L, Szyszlo-Davies JM, Racanelli TL, Davies KJA (1982) Membrane effects of vitamin E deficiency: bioenergetic and surface charge density studies of skeletal muscle and liver mitochondria. Ann NY Acad Sci 393:32–47
28. Jackson MJ, Jones DA, Edwards RHT (1983) Vitamin E and skeletal muscle. Ciba Foundation Symposium 101. Biology of Vitamin E. Pitman, London, pp 224–239
29. Packer L, Almada AL, Rothfuss LM, Wilson DS (1989) Modulation of tissue vitamin E levels by physical exercise. Ann NY Acad Sci 570:311–321
30. Dillard CJ, Litov RE, Savin WM, Dumelin EE, Tappel AL (1978) Effects of exercise, vitamin E, and ozone on pulmonary function and lipid peroxidation. J Appl Physiol 45:927–932
31. Gohil K, Viguie C, Stanley WC, Brooks GA, Packer L (1988) Blood glutathione oxidation during human exercise. J Appl Physiol 64:115–119
32. Costagliola C, Libondi T, Menzione M, Rinaldi E, Auricchio G (1985) Vitamin E and red blood cell glutathione. Metabolism 34:712–714
33. Sumida S, Tanaka K, Kitao H, Nakadomo (1989) Exercise-induced lipid peroxidation and leakage of enzymes before and after vitamin E supplementation. Int J Biochem 8:835–838
34. Helgheim I, Hetland O, Nilsson S, Ingjer F, Stromme SB (1979) The effect of vitamin E on serum enzyme levels following heavy exercise. Eur J Appl Physiol 40:283–289
35. Kobayashi Y (1974) Effect of vitamin E on aerobic work performance in man during acute exposure to hypoxic hypoxia. Dissertation. University of New Mexico, Albuquerque,
36. Simon-Schnass I, Pabst H (1988) Influence of vitamin E on physical performance. Int J Vit Nutr Res 58:49–54
37. Dwyer T, Brotherhood J (1981) Long-term dietary considerations in physical training. Proc Nutr Soc Aust 6:31–40
38. Ward RJ, Jutla J, Duane PD, Peters TJ (1988) Reduced antioxidant status in patients with chronic alcoholic myopathy. Biochem Soc Trans 16:581

Received October 11, 1990

Author's address:

Helga Gerster, F. Hoffmann-La Roche & Co. Ltd., Grenzacher Strasse 124, 4002 Basel, Switzerland