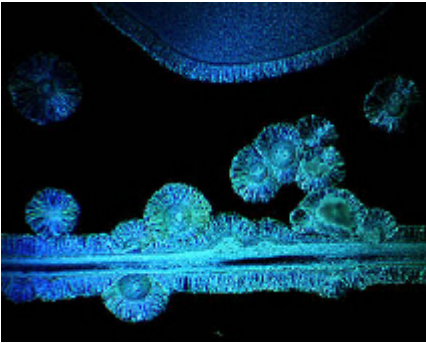


Role of Antioxidant Supplementation in Response to Exercise Induced Oxidative Stress



Researched and Composed by Adam "Old School" Knowlden

Abstract

A manifold of chemical reactions occur within the body as a result of intense physical training. During both aerobic and anaerobic training, one such reaction is the occurrence of excess and adverse free radical production. Furthermore, concentric and eccentric contractions, which are crucial to exploiting hypertrophy/hyperplasia, appear to enhance this reaction, known as Exercise Induced Oxidative Stress (EIOS).

Apposite antioxidant supplementation has been revealed through an abundant amount of studies to aid in counteracting such negative responses to training. In addition, the post-workout "window of opportunity" has shown itself to be an exceptional timeframe in which to administer anti-oxidant supplementation in direct combat of EIOS.

The focus of this journal entry is to demonstrate, using current scientific research, the most recent recommended daily allowances of crucial vitamins to counteract EIOS and post-exercise inflammation, and more specifically, application of these vitamins during the post-training scenario.

Recommend reading:

[The Anatomy of A Muscle](#)

[Take Fat Burning To A Whole New Level!!](#)

[The Window of Opportunity](#)

[Dextrose, Maltodextrin, and Sodium an In Depth Analysis](#)

[Endocrine Insanity Part III](#)[Metabolic Primer Part I](#)

Photo Explanation: Vitamin C crystals, dark field with homemade patch stop and blue filter, courtesy Ian Walker, UK.

Introductory Statements

Skeletal muscle is seldom considered a primary target of oxidative stress. Moreover, these tissues have proven to be distinctively *designed* to withstand stresses of countless sorts. During severe hypertrophy-inducing exercise, they are exposed to levels of mechanical and metabolic insult that would fatally injure or kill most other cell varieties.

Oxygen (O₂) is a universal electron acceptor that allows aerobic organisms to use energy stored in foodstuffs, such as carbohydrates, fats, and protein (24).

The sweeping incremental changes in O₂ metabolism that occur in the body during intense exercise are nothing short of mind-boggling.

Like a high-precision motor, O₂ flux through the mitochondria can increase 100 times when going from rest to maximum exercise in highly trained oxidative muscle fibers (40).

Free Radicals

A *Free Radical* is defined as any chemical species that possesses an unpaired electron or odd number of electrons.

Free radicals survive in a state of thermodynamic volatility. They are highly reactive and search to combine with another molecule to pair off its solitary electron. These radicals can be formed by several mechanisms (8):

- Electron transfer,
- Heterolytic fission,
- and Homolytic fission.

There are 3 important steps in free radical reactions (25).

The initial step is chain initiation, in which a free radical is formed, usually by homolytic fission. The second step, chain propagation, occupies a reaction where a free radical is consumed but a new free radical is produced to continue the chain. As the reaction proceeds, many radicals are present at once. The final step is when a chain termination reaction occurs when two free radicals combine, thus pairing off each other's lone electron.

Mechanisms for Free Radical Production include (8):

1. Mitochondria: Most free radical generation within the cell occurs by electron transfer reactions.
2. Inflammation. Not all free radical formation in biological systems is accidental. Catalysis caused by some enzymes is the result of their use of a free radical at the active site in response to inflammation (25).
3. Ischemia Reperfusion. All the previously mentioned mechanisms for free radical production can be related back to ischemia-reperfusion injury. Ischemia, from whatever cause, results in a decrease in oxygen and substrate availability. The lack of adenosine triphosphate (ATP), due to the inability of anaerobic means to maintain pace with energy demands, results in damaging effects (25).

Methods in which Free Radicals Instigate Tissue Damage

The reaction of free radicals with cell membranes is one of the actions that lead to tissue damage.

During resistance exercise, ischemia reperfusion (the restoration of blood flow to an organ or tissue that has had its blood supply cut off) occurs within the active muscles, possibly even to a higher degree than within other organs. Muscles undertaking strong concentric and eccentric actions are open to experience brief hypoxic conditions. Intense muscle actions temporarily decrease blood flow and thus oxygen availability, whereas with muscle relaxation there is oxygen reperfusion. Furthermore, membrane disruption has also occurred, identified by a leakage of intra-muscular enzymes into the blood, such as creatine kinase.



It is also likely that the trauma to muscle cells during high-intensity exercise results in the activation of inflammatory intermediaries. These mediators act through phagocytic- and endothelial-mast cell pathways of free radical creation (48).

These mechanisms indicate that anaerobic training could result in free radical production beyond what has been measured with aerobic exercise. From the abovementioned mechanisms, the active muscle site in hypertrophy training may result in a significant increase in the production of free radicals either during or after exercise. Consequently, it is possible that a resistance exercise protocol will result in

quantifiable increases in lipid peroxidation. A previously proposed mechanism of free radical production during exercise, particularly resistance exercise, is an ischemia-reperfusion environment at the muscle site (38).

One study looked specifically at this concept using repetitive static muscle contractions. A knee extension exercise was used with a 10-second exertion phase and a 10-second resting phase protocol at 30% of maximal voluntary contraction force (39).

The current scientific evidence advocates that free radical production within the body depends on exercise intensity, whether one is referring to aerobic or anaerobic exercise. A body building protocol must provide a considerable disturbance to the physiological state of the body. This includes significant ischemia-reperfusion conditions and muscle damage (38).

Free Radicals and Muscle Injury

It has been theorized that the membrane disruption that transpires with high-intensity resistance exercise is largely due to the mechanical loads placed on the muscle. This would result in disarray in the muscle's structural integrity.

However, a fascinating study by Kraemer et al. (26) indicated that some mechanism is continuing to cause damage even after the actual exercise bout is completed.

The study compared a group performing a resistance training protocol using 5-repetition-maximum sets (5-RM set using resistance in which only 5 repetitions can be completed) with 1 minute of rest between sets (5/1) and a group performing the same exercises except with 10-RM sets and a 1-minute rest period between sets (10/1).

This study reported that the 10/1 group had a significantly higher creatine kinase response! This group was subjected to lighter loads but had greater muscle membrane disruption despite the fact that total work load was equivalent.

Corresponding studies also have shown that creatine kinase responses often peak between two to four days after eccentric exercise protocols (34, 15, 9).

Mechanisms outside motorized force may be accountable for muscle membrane disruption after high-intensity exercise. The number of circulating neutrophils has been shown to continually increase for several hours after exercise (7).

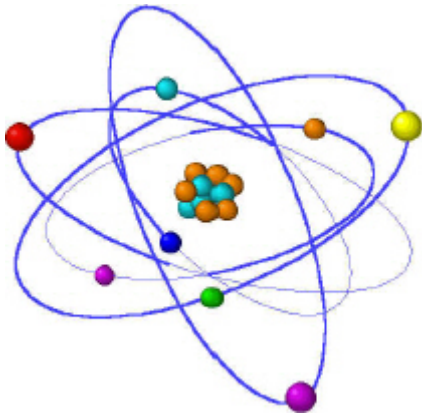
("Neutrophils, which are also known as polymorphonuclear leukocytes (PMN), represent 50 to 60% of the total circulating leukocytes and constitute the "first line of defense" against infectious agents or "nonself" substances that penetrate the body's physical barriers. Once an inflammatory response is initiated, neutrophils are the first cells to be recruited to sites of infection or injury. Their targets include bacteria, fungi, protozoa, viruses, virally infected cells and tumor cells.

Two types of free radicals are produced by neutrophils, macrophages, endothelial and other cells. The first type is represented by reactive oxygen intermediates which are formed in neutrophils by the activity of NADPH oxidase, the enzyme of the

respiratory burst. The second type includes reactive nitrogen intermediates, the first member of them, nitric oxide being produced by nitric oxide synthase." Academic Electronic Press. 1995.)

The current research suggests free radical formation, by the above described pathways, plays a role in continuing the amount of muscle membrane disruption after exercise.

Additionally, malfunctioning mitochondria, due to intra-muscular increases in calcium, could resume free radical production after exercise ceases (38).



In Summary:

- Free radicals are very unstable,
- React quickly with other compounds, doing cell and body damage,
- Once produced they multiply unless neutralized by anti-oxidants (or other free radical scavengers).

Anti-Oxidants

An antioxidant has been defined as "*any substance that, when present at low concentrations compared to those of an oxidizable substrate (e.g., proteins, lipids, carbohydrates and nucleic acids), significantly delays or prevents oxidation of that substrate*". (22)

The definition proposed by the Panel on Dietary Antioxidants and Related Compounds of the Food and Nutrition Board is that "*a dietary antioxidant is a substance in foods that significantly decreases the adverse effects of reactive oxygen*

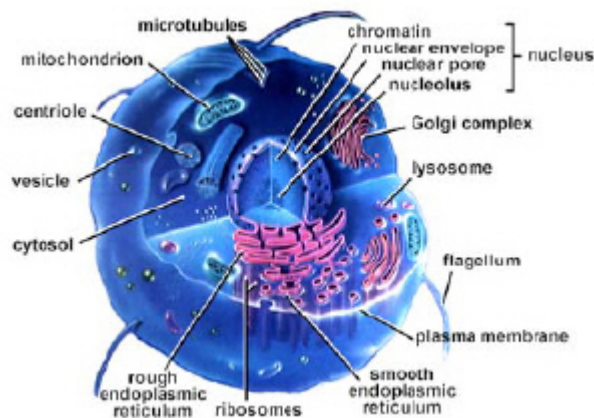
species, reactive nitrogen species, or both on normal physiological function in humans". (38)

The Core Anti-oxidants

Vitamin E

There are several non-enzymatic anti-oxidant materials in the body that can be easily supplemented. The most focused on and significant is vitamin E (alpha-tocopherol). It has been shown that this lipid-soluble vitamin is an effective antioxidant within the cell membrane. (5)

The capacity of vitamin E to prevent oxidation of unsaturated fatty acids is understood to be its primary function in the body. The absence of vitamin E results in the abnormal structure and function of cellular organelles and the cell membrane itself. (23)



Vitamin E status in rats has been highly correlated with the susceptibility of that animal to damage from muscle contractions (27). In addition, studies have shown the protective effect of oral vitamin E supplementation (47).

Experiments looking at the effects of vitamin E supplementation on muscle damage have involved muscle contraction (14). Vitamin E wields its foremost effect by the oxidation of free radicals. The mechanisms of ischemia-reperfusion injury have been the basis for which the damaging effects of free radical formation may be seen. Studies have shown vitamin E supplementation as an efficient means of reducing exercise-induced muscle damage due to free radical formation (35).

Vitamin A

Often there is confusion over how to identify vitamin A because of the varying forms that exist in nature.

Vitamin A is a retinol and is related to but different from retinoids and carotenoids. Beta carotene, which is commonly mistaken as a vitamin A equivalent, is actually two retinols with the alcohol groups removed. It is classified as a carotenoid (6). Beta carotene has been identified as a possible antioxidant because of its ability to scavenge singlet oxygen. On demand beta carotene can be broken down into two retinol equivalents (RE) if other sources of vitamin A are not available. This mechanism is how beta carotene has been identified as a vitamin A precursor (32). Much less work has been done with vitamin A compared with vitamin E and C as a protective antioxidant in relation to exercise (38).

Vitamin C

Vitamin C or L-ascorbic acid has been implicated as an important water-soluble antioxidant in biological fluids.

The meticulous role of vitamin C literally reaches to every cell of the body. This vitamin plays many vital roles including immune system functioning, connective tissue repair, and is a vital ingredient of collagen (16). For the purposes of this journal, focus will be upon its protection from free radical damage.

Vitamin C readily scavenges reactive oxygen and nitrogen species, such as superoxide and hydroperoxyl radicals, aqueous peroxy radicals, singlet oxygen, ozone, peroxy nitrite, nitrogen dioxide, nitroxide radicals, and hypochlorous acid, thus effectively protecting other substrates from oxidative damage (19).

Compounding Evidence of Research

Numerous studies have shown the effectiveness of antioxidant, in particular Vitamin C, supplementation for the athlete in combating Free Radical Damage, and also enhancing the hormonal effects of the post-exercise scenario.

Alessio HM, Goldfarb AH, and Cao G (1) concluded:

“Vitamin C (ascorbic acid) was supplemented (1 g/day) for 1 day and 2 weeks in the same subjects...It was concluded that exercise-induced oxidative stress was highest when subjects did not supplement with vitamin C compared to either 1 day or 2 weeks of vitamin C supplementation.”

In 2000, Evans WJ (18) found:

Vitamin C and, especially, vitamin E are shown to decrease the exercise-induced increase in the rate of lipid peroxidation. No ergogenic effects of either vitamin C or E have been shown. Vitamin E was shown to significantly increase circulating neutrophils in older, but not younger, subjects performing eccentric exercise that causes an increase in skeletal muscle damage. In addition to its effect in augmenting the neutrophil response to eccentric exercise, vitamin E causes a greater increase in circulating creatine kinase activity, perhaps indicating increased skeletal muscle repair. Increased vitamin E intake has been associated with enhanced glucose tolerance and insulin action as well as improved lipoprotein status.

Ghosh MK, Chattopadhyay DJ, and Chatterjee IB (19) have shown the powerful effect of vitamin C to a healthy diet. This can be directly correlated to the sport of body building:

The observations substantiate the previous in vitro findings that ascorbate specifically prevents oxidative degradation of microsomal membranes. The results indicate that vitamin C may exert a powerful protection against degenerative diseases associated with oxidative damage and play a critical role in wellness and health maintenance.

The observations made by Maxwell SR, Jakeman P, Thomason H, Leguen C, Thorpe GH (37) have shown the clear benefit of supplementing with anti-oxidants for body building specific training styles.

It is concluded that plasma antioxidant capacity rises in response to one hour of eccentric exercise and that the contribution of individual antioxidants to this change can be influenced by vitamin supplementation.

In a 2001 study (43) Peters EM, Anderson R, Nieman DC, Fickl H, and Jogessar V. revealed the effects Vitamin C can have on stress hormones.

The study demonstrates an attenuation, albeit transient, of both the adrenal stress hormone and anti-inflammatory polypeptide response to prolonged exercise in runners who supplemented with 1500 mg vitamin C per day when compared to $< \text{ or } = 500 \text{ mg per day}$.

Sastre J, Asensi M, Gasco E, Pallardo FV, Ferrero JA, Furukawa T, and Vina J showed the preventive power of antioxidant administration in a 1999 experiment (49).

Thus, both in rats and humans, exhaustive physical exercise causes a change in glutathione redox status in blood. We have also found that antioxidant administration, i.e., oral vitamin C, N-acetyl-L-cysteine, or glutathione, is effective in preventing oxidation of the blood glutathione pool after physical exercise in rats.

Tauler P, Aguiló A, Fuentespina E, Tur JA, and Pons A. demonstrated that "diet supplementation with vitamin E, vitamin C and beta-carotene cocktail enhances basal neutrophil antioxidant enzymes in athletes, (51)" and thus concluded the connotation of antioxidant supplementation:

Exercise increases oxygen consumption and causes a disturbance of intracellular pro-oxidant-antioxidant homeostasis... Plasma vitamin E, beta-carotene and vitamin C concentrations in the antioxidant-supplemented group were approximately 1.6, 10, and 1.2 times higher respectively than those of the placebo group. The antioxidant-supplemented group presented a significantly higher glutathione versus glutathione disulfide ratio in neutrophils (about 20%) than the placebo one. Antioxidant supplementation enhances the antioxidant enzyme activity of superoxide dismutase and catalase in neutrophils.

An exciting 2001 analysis (44) presented by Peters EM, Anderson R, Theron AJ. uncovered:

These observations provide evidence that supplementation with vitamin C may blunt the adaptive mobilization of this vitamin from the adrenals during exercise-induced oxidative stress and may be associated with an enhancement of the acute phase protein response and attenuation of the exercise-induced increase in serum cortisol.

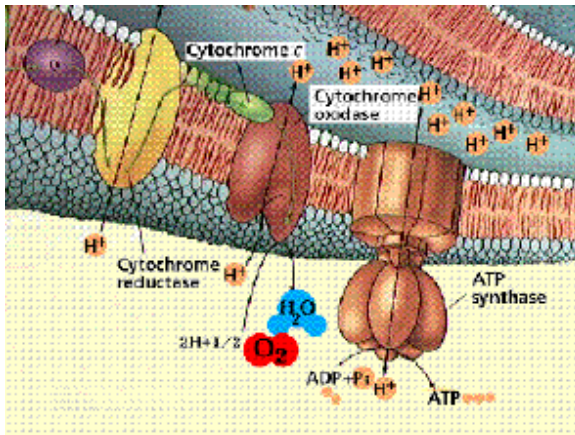
The above report reveals that Vitamin C can actually weaken the exercise-induced increase in cortisol, while increasing the sensitive post-workout protein response!

Course of Exercise Induced Oxidative Stress (EIOS)

In General, molecules contain pairs of electrons that orbit their nucleus. Conversely, an electron is occasionally "lost," which alters the molecule into a *free radical* (44).

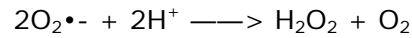
In the standard process of metabolism, this often happens to oxygen. Most of the oxygen consumed is condensed to water in the mitochondria. However, a small fraction of oxygen intermediates (i.e., $O_2^{\bullet-}$ and H_2O_2) are produced and escape the electron-transport-chain (the electron transport system is a chain of electron acceptors embedded in the inner membrane of the mitochondrion) (10).

The presence of free radicals is damaging to the cell, predominantly to cell membranes.



What emerges as an irony is that exercise increases the production of free radicals by virtue of an increase in oxygen exploitation. Overall, oxygen radicals and the reactive species that they spawn harm other species with which they come in contact. For instance, oxygen-centered radicals have been implicated with cancer as they are believed to aid in damaging DNA strands. Cell membranes possess polyunsaturated fatty acids that are highly susceptible to radical assault. This process is known as lipid peroxidation and increases permeability. As a result (44) this causes an influx of Ca^{2+} , a deficit of intracellular enzymes, and an advent of lysosomal (destructive) enzymes. There are numerous antioxidant defenses (44, 38)

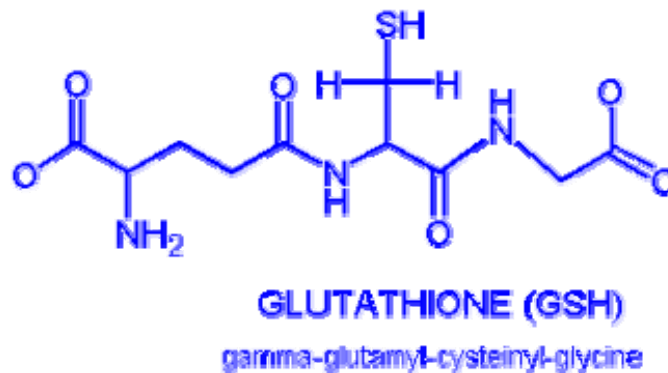
which comprise enzymes and non-enzymatic antioxidants that restrain or react with radicals and radical intermediates. Superoxide dismutase is the principal security in the cell and catalyzes the subsequent reaction:



Although H_2O_2 itself not a radical, it has been shown to injure nucleic acids. Other antioxidant enzymes such as catalase and glutathione peroxidase, catalyze the decrease of H_2O_2 . Glutathione is a foremost non-enzymatic antioxidant and has several important functions including elimination of H_2O_2 and recycling of vitamin E (18).

The body has other antioxidant defense schemes, including antioxidant vitamins. Embedded within the various membranes (sarcoplasm and inner mitochondrial membrane) is the lipid-soluble vitamin E (2).

Vitamin E is a chain-breaking antioxidant which reacts rapidly with fatty acid radicals, but must be reduced after each reaction by glutathione or vitamin C (ascorbate).



As well, glutathione catalyzes the renewal of ascorbate. The principal role of β -carotene (a precursor to vitamin A) is to quench singlet oxygen ($\text{O}_2^{\bullet-}$) in addition to slow down lipid peroxidation (44).



Although an abundance of studies (1, 18, 19, 43, 44, 49, 51) have shown that antioxidant supplements can be beneficial, there are data that demonstrate no effect of supplementation. As with all studies, controls can dictate their relation to the conclusion, particularly with exercise. An element of these conclusions is that many of these reviews have studied only a single vitamin when effectiveness of supplementation may depend on the presence of all the antioxidants.

Post-Workout Inflammation

Muscle contraction and shortening produces a concentric action; in contrast, when skeletal muscle lengthens as it produces force, the result is an eccentric muscle action. For example, lifting a weight is a concentric action, and lowering the weight is an eccentric action. At the same power output, the oxygen cost is lower for eccentric than for concentric exercise, but even so, eccentric exercise is a potent cause of muscle damage, delayed-onset muscle soreness, and increased circulating creatine kinase activity (18).

In 1999, Goldfarb (20) concluded that nutritional antioxidants are therapeutic in exercise-induced muscle damage.

Several mechanisms have been forwarded to explain the etiology of exercise-induced muscle damage. Free-radical mediated processes appear to be an important component of the inflammatory mediated response.

A growing quantity of evidence indicates that free radicals play an important role as mediators of skeletal muscle damage and inflammation after strenuous exercise (38, 44).

The literature suggests that dietary antioxidants are able to detoxify the peroxides produced during exercise, which could otherwise result in lipid peroxidation, and that they are capable of scavenging peroxy radicals, and therefore may prevent muscle damage (38).

Endogenous antioxidant enzymes also play a protective role in the process of lipid peroxidation.

Multiple studies (52, 10) using both rodent and human subjects have shown significant increases of malondialdehyde (a product of lipid peroxidation) after exercise to exhaustion, and also favorable changes in plasma antioxidant levels and in antioxidant enzyme activity.

In trained individuals and trained rats, the antioxidant enzyme activity amplifies markedly. In this way, the increased oxidative stress induced by exercise is compromised by increased antioxidant activity, preventing lipid peroxidation (22).

Human analyses have shown that dietary supplementation with antioxidant vitamins has note-worthy effects on lipid peroxidation post-exercise.

Although several points of debate still exist, the question whether antioxidant vitamins and antioxidant enzymes engage a protective role in exercise-induced muscle damage can be answered affirmatively (3, 6, 38).

The human studies reviewed by Dekkers JC, van Doornen LJP, and Kemper HCG (13) designate that antioxidant vitamin supplementation can be recommended to individuals performing recurring heavy exercise.

Goldfarb AH (21) studied the role of antioxidant supplementation to prevent exercise-induced oxidative stress.

Exercise of a sufficient intensity and duration has been shown to increase indicators of oxidative stress. Oxidative stress has been indicated in skeletal muscle, liver, blood, and in expired air samples as indicated by the by-products of lipid peroxidation. Antioxidants are known to reduce oxidative-radical-induced reactions.

Jakeman P and Maxwell S (28) studied the "Effect of antioxidant vitamin supplementation on muscle function after eccentric exercise.", and found:

This study investigated the effects of antioxidant vitamin supplementation upon muscle contractile function following eccentric exercise and was performed double blind...These data suggest that prior vitamin C supplementation may exert a protective effect against eccentric exercise-induced muscle damage.

A frequent retort of performing unacquainted or high-intensity exercise is the main occurrence for DOMS. Much like muscle damage, DOMS results primarily from eccentric exercise, however successive eccentric bouts will diminish the DOMS response (44).

However, soreness does not initially come about from damage to the muscle fiber. Peak soreness occurs within 24-48 hours post-workout, while peak muscle damage is seen three days post-training.

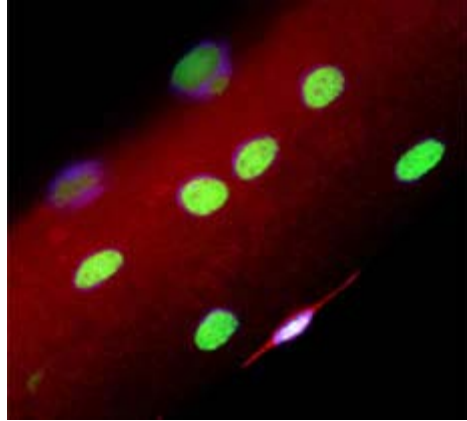
Although the eccentric range of motion is the general factor to both responses, DOMS and muscle damage are diverse physiological reactions and are caused by separate devices (3, 28).

Aside from the damage to the actual muscle fiber, eccentric training also upsets the connective tissue that surrounds the muscle. It is this impairment which is considered to be the chief source of soreness (34).

As fluid travels into the fiber the intra-muscular pressure is amplified, which is believed to stimulate pain sensors positioned in the connective tissue. Without a doubt, increases in intra-muscular stress correlate exceedingly with the incidence of DOMS. Moreover, chemicals released from the inflammation development are alleged to increase the sensitivity of the pain sensors (39, 44).

Hypertrophic/ Hyperplastic Regeneration

Subsequent to the degeneration process, regeneration commences with the muscle precursor cell, the myoblasts. In adults, these myoblasts are commonly referred to as satellite cells and are found between the basement membrane, or basal lamina, and the sarcolemma (41, 44).



(A satellite cell (blue nucleus) adherent to a muscle fiber surface by the muscle-specific adhesion molecule, M-cadherin. Terry Partridge, Muscle Cell Biology)

Typically they are inactive; however, when an injury transpires, the basal lamina of the damaged fibers releases a growth factor which kindles satellite cell proliferation within two days after the injury (32). Within three days post-injury, satellite cells can be observed to have crossed the sarcolemma and migrated to the site of damage. Once at the injury site, the cells recognize each other and fuse together into a myotube, or immature muscle fiber. Total regeneration occurs within five days to several weeks.

Furthermore, chronic hypertrophic/hyperplastic precise training results in a release of a growth factor which stimulates satellite cells proliferation.

Other postulations as to how exercise generates free radicals include (29, 30, 32, 50):

1. Additions in epinephrine and other catecholamines that can create oxygen radicals when they are metabolically inactive.
2. Manufacture of lactic acid that can translate a weakly damaging free radical (superoxide) into a powerfully damaging one (hydroxyl).
3. Inflammatory feedback to secondary muscle damage incurred with excess exertion.

The structure of the body (42) encloses a complex antioxidant defense grid that relies on dietary intake of antioxidant vitamins and minerals and the endogenous assembly of antioxidant compounds like glutathione.

Vitamins C, E, and beta-carotene are the primary vitamin antioxidants (10). Along with glutathione, there are copious amounts enzymes involved in the quenching or abstraction of free radicals (50).

Heavy physical exercise enhances free radical production in skeletal muscle and other tissues (4).

Chronic exercise also represents a form of oxidative stress (5) to the organisms and therefore can alter the balance between pro-oxidants and antioxidants.

Because acute strenuous exercise and chronic exercise training increase the consumption of various antioxidants, it is conceivable that dietary supplementation of specific antioxidants would be beneficial, both in daily supplementation and post-exercise (38).

Daily Anti-Oxidant Supplementation in Conjunction with Post-Workout Application



Although the intention of this journal entry is to focus discussion on anti-oxidant complementation for the anaerobic post-exercise window of opportunity, supplementation covering the route of an entire day must be planned out to confer ratios specifically for post-training consumption.

It has been suggested that doses over 1,000% of the recommended daily allowance (RDA) are not toxic for all 3 vitamins (31).

Regular physical activity in association with dietary habits that ensure adequate supply of a combination of appropriate antioxidants may be expected to yield desirable results.

The Following are FRDA (Freak Recommended Daily Allowances) based upon current research. These numbers are allowances covering an entire day's span.

JHR recommends these allowances be divided throughout the day for regular supplementation, aside from post-workout when increased anti-oxidant intake may be included to combat EIOS.

Vitamin E

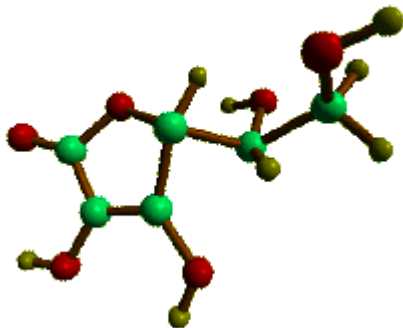


Five times the RDA for vitamin E may be necessary for prevention of free radical damage (12).

Intense exercise by athletes (46) may result in free radical production three times that of sedentary individuals.

Because of these findings, it has been stated at the Colgan Institute (11, 38) that 1,200–2,000 IU (equivalent to 800–1,350 mg of RRR-D-alpha-tocopherol or 800–1,350 TE) of vitamin E have been taken daily by athletes. This may be a necessary dosage to counter free radical formation during exercise.

Vitamin C



Always behind the times for athletes, the RDA for vitamin C is 60 mg. It has been suggested (11) that this is based on an inaccurate and antiquated method for calculating vitamin C requirements.

Dosages given to athletes have been reported to be 2–12 grams daily. A consensus on reviews has shown complete safety with dosages of vitamin C of 1–5 grams daily (6, 38).

For musculoskeletal healing, dosages of 500–1,000 mg, 2 to 4 times daily, have been taken (6) in the form of ascorbic acid.

Beta Carotene

From current research, the amount of beta carotene that would be necessary for it to be a significant contributor to anti-oxidation is unclear at this point. However, deductions can be made (38).

The RDA for vitamin A is approximately 800–1,200 R.E. per day.

(Toxicity has been reported in rare instances (6) at levels of 25,000 I.U., which is approximately 7,500 RE --7,500 g of retinol, 9,000 g of retinyl acetate, and 13,500 g of retinyl palmitate)

A safe dosage would fall somewhere between these 2 values (21). However, it is beta carotene and not vitamin A that acts as an antioxidant, but specific values for beta carotene are not clear.

Beta carotene has been shown to be safe at any dose (38). Adverse effects such as oily diarrhea have been reported, but only at absurdly high levels. The suggested dosage of vitamin A for effective injury repair assistance is 25,000 I.U.'s or 7,500 R.E.'s, which would be approximately 45 mg per day of beta carotene (11).

The following is a sample supplementation outline of a bodybuilder's regular anti-oxidant regime:

Meal One	Multi-Vitamin
Meal Two	Anti-Oxidant Supplement including Vitamin C, E, and A
Meal Three	500-1000mg Gram Vitamin C
Meal Four	500-1000mg Gram Vitamin C
Meal Five	Post Workout, 1-2 grams Vitamin C, or Anti-Oxidant Supp
	* Recommended to Consume 10-20 minutes into post-workout shake
Meal Six	500-1000mg Gram Vitamin C

Concluding Discussion



Exercise induced oxidative stress is a corporal reaction that needs to be counterbalanced during the body's peak time of receptiveness to nutrition (44).

This phase of the physiques response to extreme physical stress can best be remedied through appropriate post-workout supplementation.

A proper post-workout anabolic cocktail elaborating on the shuttling effects of insulin and rapid gastric emptying is the ideal atmosphere for anti-oxidant consumption in the effort to combat EIOS.

Job 22 states,

²² Accept instruction from his mouth
and lay up his words in your heart.

Psalm 19

¹⁴ May the words of my mouth and the meditation of my heart
be pleasing in your sight,
O Lord, my Rock and my Redeemer.

Psalm 119

¹¹ I have hidden your word in my heart
that I might not sin against you.

Ezekiel 3

¹⁰ And he said to me, "Son of man, listen carefully and take to heart all the words I speak to you.

Luke 8

¹⁵ But the seed on good soil stands for those with a noble and good heart, who hear the word, retain it, and by persevering produce a crop.

Revelation 1

³ Blessed is the one who reads the words of this prophecy, and blessed are those who hear it and take to heart what is written in it, because the time is near.

By constantly supplementing with God's Word, we can cast out the vain imaginations and pollution put into our bodies through our flesh.

In conclusion, the adverse effects of post-exercise free radical production can be remedied through proper supplementation of anti-oxidants, Vitamins E, C, and A.

While the adverse effects of the flesh can be banished through proper supplementation of God's Word.

Adam "Old School" Knowlden

Vice-President of Biomechanical Engineering

oldschoolabcbbing@gmail.com

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