Chapter – 2
Review of Literature

In support of present study following reviews were collected:-

Exercise has been associated with an acute increase in the production of reactive oxygen species (ROS) above resting values (McBride, 1999). However, it is not known how chronic exercise training affects resting and exercise ROS levels or if there are differences between varying types of training (i.e. aerobic vs. anaerobic).

Three basic mechanisms of exercise induced ROS production are commonly describe; mitochondrial leakage, ischemia-reperfusion injury, and inflammation and immune response.

1. Under physiological conditions, mitochondrial leakage is the major source of exercise induced ROS production among these three basic mechanisms. Oxygen is absorbed by the blood circulation which is transported via the cardiovascular system to the various tissues in the body where it is eventually delivered to the mitochondria. During exercise, the dramatic increase in electron flux across components of the electron transport chain causes excessive leakage (uncoupling) at specific sites. An increase in the number of electrons present in the mitochondria that readily dissociate without a concurrent increase in molecular oxygen to bind to results in more electrons than oxygen, leading to ROS production (Ji, 1995; Clanton, 2007). Specifically, the inner mitochondrial membrane has been implicated as a major site of oxygen free radical production, because ROS are formed during the reduction of oxygen that takes place as part of the electron transport chain.
2. The second mechanism of exercise induced ROS is ischemia-reperfusion injury caused by an over abundance of oxygen following high intensity exercise (via excess post exercise oxygen consumption). When blood flow is decreased to tissue (ischemia) through arterial blockage or blood vessel occlusion (often seen with resistance exercise), xanthine oxidase (XO) activity is increased. When normal blood flow is restored (reperfusion) and oxygen levels are increased within the tissue the increased XO activity leads to ROS production.

3. Lastly, exercise that is characterized by excessive eccentric activity or intense resistance has been shown to cause muscle damage. The damaged tissue becomes necrotic and evokes an oxidative burst by a primary immune response in which ROS are produced by macrophages and leukocytes. Secondary damage to previously healthy cells has been hypothesized to occur due to the ROS production that accompanies this immune activity (Quindry et al., 2003; Magalhaes et al., 2004; Hagobian et al., 2006). There is a somewhat close relationship between ROS production and exercise intensity. Previously, many studies have used graded exercise test (GXT) protocols to produce ROS because high intensity aerobic exercise, particularly maximal effort, is associated with high levels of anaerobic metabolism and hypoxia. These factors are related to oxidative stress by creating a build-up of reducing agents in the mitochondria which stresses the electron transport chain (Clanton, 2007), or through ischemia-reperfusion injury caused by an over abundance of oxygen following high intensity aerobic exercise.

Athletes trained by long endurance-type exercise may have a high antioxidant capacity. In addition, acute aerobic exercise may increase oxidative stress, chronic aerobic
exercise may attenuate it due to increased endogenous production of antioxidants and/or a more tightly coupled electron transport system allowing fewer electrons to escape and form superoxide radicals (Jenkins & Goldfarb, 1993; Ji, 1995; Kim et al., 1996). There are some studies that show running a marathon may cause acute oxidative DNA damage and lipid peroxidation even in trained distance runners (Kant, et al., 1988; Maughan & Godt, 1989; Atalay et al., 1996; Niess, 1996; Tsai et al., 2001; Gomez et al., 2006). Knez et al., (2007) showed that Ironman athletes have a lower resting oxidative stress level than matched sedentary subject. In addition, Niess (1996) showed that trained long distance athletes have lower resting MDA levels than untrained subjects. In contrast, Marzatico et al. (1997) showed that marathon runners have higher resting oxidative stress than sedentary controls. There are some studies that show that resistance exercise can cause oxidative stress (Alessio et al., 2000; Bloomer & Goldfarb, 2006; Lee et al., 2002). Repeated isometric resistance exercise can elevate oxidative stress levels. Lee et al. (2002) suggested that a single bout of 60 eccentric contractions at 150% MVC may cause an increase in blood protein carbonyls. In addition, Zembron- Lacny et al. (2008) reported that TBARS levels were increased from 107.17±23.41 to 135.91± 23.10 nmol/g haemoglobin following multi- or single joint isokinetic resistance exercise. Sprinters are highly anaerobically trained individuals. This sprint type of exercise causes a high inflammatory response and this could lead to oxidative stress in the skeletal muscle. There is a study which conducted sprint training whereby rats performed sessions of 30-s bouts of intense sprinting. They were sacrificed at 48 h after their last sprint session and showed increased selected ROS in fast twitch and mixed skeletal muscle (Atalay et al., 1996). In addition, strenuous sprint training shares a very similar metabolic pathway with resistance training. Furthermore,
Marzatico et al. (1997) reported that repeated sprint exercise increased MDA and CD levels in a sprint trained group. However, some studies suggest that sprint training does not affect the production of ROS in highly trained individuals. Groussard et al. (2003) reported that sub-maximal sprinting exercise did not affect post exercise MDA levels. Following a review of the current literature, it is very hard to say that sprint exercise can produce high amounts of oxidative stress in trained athletes. In addition, some previous studies recruited highly trained subjects (Marzatico et al., 1997) and other studies recruited recreationally trained individuals. Baker et al. (2004) and Thompson, et al., (2001b) claimed that sedentary or recreationally trained individuals can be affected by sprint exercise in production of ROS.

The exercises practiced excessively by athletes can promote ROS explosion which can exceed the protection effect of any adaptable response achieved during tanning. Other factors that can overcome the exercise inducing the oxidative damage, for instance, is the anti oxidative nutritional deficiency. During the execution of the low intensity and duration exercise protocols, antioxidant defenses seem to be enough against the ROS production, but in high intensity and duration levels of exercise, these defenses are no longer enough, resulting in oxidative damage to the tissue (Longo et al., 2008).

It is known that free radicals can attack every main class of bio-molecule, being the lipids the most susceptible. The oxidative destruction of the polyunsaturated fatty acids, known as the lipid peroxidation, is considerably harmful for being a reaction of self-propagation in the membrane (Halliwell & Gutteridge, 1990) . The disputes about the effects of exercise over the lipid peroxidation are innumerous, probably, due to differences in intensity and duration of the exercise protocols (Alessio & Goldfarb, 1988).
Published literature indicates that there are 8 primary assays used by researchers to measure oxidative stress in the body; lipid hydroperoxides (LH), reduced glutathione (GHS), conjugated dienes (CD), malondialdehyde (MDA), protein carbonyls (PC), isoprostanes, and 8-hydroxydeoxyguanosine (8-OHdG). Of these assays, malondialdehyde (MDA) and GSH appear to be somewhat equivocal, returning a mix of significant and non-significant differences in blood levels, even when similar groups are used (Lovlin et al., 1987; Sen et al., 1994; Ashton et al., 1998; Surmen-Gur et al., 1999). The lack of consistency in the research is due largely to major differences in methodology, including exercise protocol, type of oxidative stress marker assayed and timing of blood samples. However, most of the oxidative stress studies have used TBARS, GHS, and MDA for the oxidative stress markers, and lipid peroxidation (indicated by TBARS and MDA) is the most common response of OS. In addition, TBARS is not only an indicator of lipid peroxidation but is also a common oxidative stress marker.

Although the healthcare field is increasingly aware of the importance of free radicals and oxidative stress, screening and monitoring has not yet become a routine test. The method herein presented allows reliable, rapid, and noninvasive measurement of the instantaneous concentration of ROS directly in human peripheral blood (Simona et al., 2012).

Many years ago, research demonstrated that increased exposure to ROS triggers an adaptation in antioxidant enzyme activity in mouse cardiac and rat skeletal muscle tissues (Oberley et al., 1987; Jenkins et al., 1993). Because exercise has been linked with increased ROS production, a clear reasoning exists for antioxidant adaptation to chronic exercise training (Ji et al., 1999). Prolonged exercise increases ROS production in skeletal
muscle and this stimulates the antioxidant capacity (Powers & Lennon, 1999). Chronic training causes ROS production and this chronic exposure to ROS can increase antioxidant capacity via long-term antioxidant adaptation. Indeed, the endogenous antioxidant defense system is enhanced through intensive endurance exercise training (Ji et al., 1999). There are many endurance exercise induced oxidative stress studies. However, it is hard to find consistency in their post exercise antioxidant values. It has been suggested that high intensity endurance exercise is superior to low intensity training in the up-regulation of muscle glutathione peroxidase (GPX) activity (Powers et al., 1994). In addition, GPX has been shown to have high activity and expression in some studies which employed high intensity aerobic exercises in animal models (Sen et al., 1992; Powers et al., 1994; Powers & Lennon, 1999). Furthermore, GPX activity was not found in rat soleus muscle but red gastrocnemious muscles (Hollander et al., 1985; Leeuwenburgh et al., 1994; Venditti & Di Meo, 1996). As seen with GPX adaptation, exercise induction of SOD appears to be fiber type specific, with highly oxidative muscles being most responsive (Higuchi et al., 1985; Ji et al., 1991; Powers et al., 1994; Gore et al., 1998; Vincent et al., 1999). These studies also show that, similar to GPX adaptation, high intensity training generally appears to be superior to low intensity training in SOD up-regulation in muscle. However, human studies do not show this tendency because of different exercise protocols, blood sampling time, supplementation status, exercise status, and fitness levels.

Physical exercise is known to increase the generation of ROS in response to increased oxygen utilization causing a disturbance in the pro-oxidant/antioxidant balance in favor of the former which results in oxidative stress. On the other side of the same coin, antioxidant compounds may alter redox status too, by reducing ROS production (Poljsak, 2011).
In general, the body has adequate antioxidant reserves to cope with the production of ROS under physiological conditions and perhaps during low-moderate intensity exercise (Ji, 1995c), but when ROS production is excessive, such as during prolonged intensive physical efforts, an imbalance between oxidants and antioxidants in favor of the oxidants may occur, leading to a disruption of redox signaling (Powers et al., 2011).

Long-duration exercise training may augment the physiological antioxidant defences in several tissues; however, this enhanced protection may not be sufficient to completely protect highly fit individuals from exhaustive exercise-induced oxidative stress (Sen et al., 1994). Recently, several studies have tested the effect of a variety of endurance exercise training regimens on antioxidant capacity (Ji, 1995c; Sen et al., 1994). Subjects with high aerobic capacity (VO2max>60ml/kg/min) show significantly greater muscle SOD and CAT activity (Jenkins & Goldfarb, 1993). Only one study has investigated the resting ROS and antioxidant levels to both aerobic and anaerobic training status (Marzatico et al., 1997). This study said that marathoners have better antioxidant capacity than sprinters in lipid oxidation because aerobic type training improves erythrocyte antioxidant enzymatic activity at rest and during post-exercise recovery. This greater superoxide scavenging and hydrogen peroxide detoxification capacity of the marathoners in conjunction with accentuated muscular blood flow and tissue mitochondrial aldehyde dehydrogenase activity could be responsible for the significant decrease in the peroxidative indices (Halliwell & Gutteridge, 1993). Only one study investigated the effects of resistance exercise on AO activity, finding an increase in SOD and CAT activity in response to both multi- and single-joint weight lifting (Zembron-Lacny et al., 2008).
Powers et al. (1999) described that regular exercise training increases the expression of both enzymatic and non-enzymatic antioxidants in the active muscles to provide protection against exercise-induced oxidative stress. Therefore, compared to untrained individuals, well-trained athletes possess higher levels of endogenous antioxidants.

McArdle et al., (2002) suggests that muscular exercise induces radical production and that contracting skeletal muscles are a major source of these radicals. The magnitude of exercise-induced radical production is influenced by several factors, including the intensity and duration of exercise and the environmental conditions. Specifically, skeletal muscle radical production increases as a function of both the exercise intensity and duration. Moreover, contracting skeletal muscles produce more radicals during exercise in hot environments or at high altitude (i.e., >2,000 m) (Arbogast & Reid, 2004; Clanton, 2007; Radak et al., 1997). Therefore, the extent of exercise-induced radical production can range from relatively low to high levels, depending upon the exercise conditions.

Powers et al. (2004), proposed that contracting skeletal muscles produce radicals, exercise bouts do not always result in oxidative damage to muscles. For example, exercise of low intensity and short duration does not generally promote oxidative stress in skeletal muscles. Nonetheless, prolonged exercise performed at moderate-to-high intensities often results in oxidative damage in skeletal muscles of untrained persons. However, highly trained endurance athletes have well-adapted endogenous antioxidant buffer systems in their skeletal muscles that resist exercise-induced oxidative stress (Powers et al., 1999). Therefore, whether an exercise bout results in oxidative stress depends not only on the intensity and duration of exercise but also on the exercise training status of the individual.
Hargreaves, (2005) defined muscle fatigue as a reduction in the ability of a muscle to generate force. Fatigue can occur during a wide variety of sporting events and during intense exercise training sessions. Muscle fatigue is a multifactorial process, and the specific causes of fatigue can vary due to the environmental conditions and the type of exercise performed. Growing evidence indicates that radical production in skeletal muscles contributes to fatigue during prolonged exercise (i.e., events lasting >30 min).

Reid, (2001 & 2008) carried out well-controlled animal studies indicate that scavenging or neutralizing radicals via antioxidants delays muscle fatigue during prolonged sub-maximal exercise. In contrast, antioxidant scavengers of radicals are not effective in delaying muscle fatigue in animals during high intensity exercise (Reid et al., 1992a; Matuszczak et al., 2005). Finally, studies examining the effects of antioxidants on muscle performance during recovery from fatiguing exercise are inconsistent; some reports indicate a faster recovery of force production (Diaz et al., 1998), whereas others failed to show a faster recovery time (Khawli & Reid, 1994; Reid et al., 1992a, 1992b).

Antioxidants are especially important during exercise because ROS production increases with intensity and duration modulated by the training status of the population in question (Donato et al., 2010). Thus, in the case of athletes consuming low antioxidant diets and completing a heavy training load, the accumulation of ROS and therefore increased oxidative stress may contribute to poor recovery, fatigue and muscle damage (Powers & Jackson, 2008).

Fatouros and his colleagues (2010), found that soccer induce a marked inflammatory response during recovery, but it also increases the concentration of antioxidant and oxidant molecules, such as total antioxidant status (TAS), glutathione
peroxidase (GPx), thiobarbituric acid reactive substances (TBARS), protein carbonyls (PC) and uric acid.

Uric acid, as the final product of purine metabolism, has been considered an important plasma antioxidant, and contributes between 35-65% of the antioxidant activity detected in TAC (Benzie & Strain, 1996).

A growing number of studies indicates through the use of a strong antioxidant, that fatigue in human muscle can be delayed during submaximal exercise (Matuszczak et al., 2005; McKenna et al., 2006; Medved et al., 2004a, 2004b; Reid et al., 1994; Travaline et al., 1997). In this model N-acetylcysteine (NAC) is administered as the free-radical scavenger and has been reported to delay muscular fatigue during a variety of submaximal exercise tasks including; (a) electrically stimulated contractions of human limb muscles (Reid et al., 1994); (b) breathing against an inspiratory load (Travaline et al., 1997); (c) cycling exercise (McKenna et al., 2006; Medved et al., 2004a, 2004b); and (d) repetitive handgrip exercise (Matuszczak et al., 2005). Compared to the effects of the placebo, fatigue during exercise in these studies is diminished by 15-62%. Importantly, and consistent with the aforementioned animal studies, NAC does not appear to retard human muscle fatigue during more intense exercise at near VO$_2$ max (Diaz et al., 1994; Matuszczak et al., 2005; Medved et al., 2003). In summary, based on the model using NAC, both animal and human experiments indicate that radical accumulation during submaximal exercise may promote muscular fatigue but the role of radicals during short duration, high intensity exercise remains in question.

Numerous dietary antioxidants can also contribute to cellular protection against radicals. Important dietary antioxidants include vitamin E, vitamin C, carotenoids, and
flavonoids. Vitamin E is one of the most widely distributed antioxidants in nature and protects cell membranes against radical-mediated damage (Janero, 1991; Packer, 1991). The generic term "vitamin E" refers to at least eight structural isomers of tocopherols (Janero, 1991; Schaffer et al., 2005). Among these, alpha-tocopherol is the best known and possesses the most antioxidant activity (Stocker, 2007). In addition to its direct antioxidant properties, growing evidence suggests that some of the beneficial effects of vitamin E in cells reside in its ability to regulate gene expression of proteins (Azzi et al., 2004; Han et al., 2004; Schulte et al., 2006; Traber et al., 2008).

Several studies have investigated the effects of acute and chronic exercise on vitamin E levels in skeletal muscles of rodents. Unfortunately, the results are not consistent; some studies report that exercise decreases muscle vitamin E concentrations (Bowles et al., 1991; Gohil et al., 1987), whereas others conclude that neither acute nor chronic muscular activity alters muscle vitamin E levels (Coombes et al., 2002; Salminen & Vihko, 1983; Starnes et al., 1989). Studies investigating the effect of regular exercise on vitamin E in human skeletal muscle suggest that exercise does not change vitamin E levels (Tiidus & Houston, 1995; Tiidus et al., 1996).

The maximum aerobic capacity (VO$_2$ max) is a determining factor in endurance events (Shephard et al, 1968) and as a result of endurance training, distance runners acquire a high relative VO$_2$ max (Saltin & Astrand, 1967). The relative VO$_2$max of Indian long distance runners reported by Ghosh et al (1988), is much lower than that of elite international counterparts (Bunc et al, 1987; Kenney & Hodgson, 1985). Since VO$_2$ max is a test for assessing the running endurance, the distance runners who acquire a high VO$_2$ max, will obviously be at an advantage. VO$_2$ max improves with training but reaches a
plateau at a certain time, whereas, continuous improvement of distance running performance has been observed (Londereee, 1986; Ekblom, 1969; Pollock, 1973; Daniels et al., 1978).

Khanna et al., (1994) studied the physiological profiles of Indian national and Cuban Olympic Boxers. They have reported that Cuban boxers were taller than Indian boxers, but there was no significant difference in maximum aerobic capacity between Indian and Cuban boxers. Jossellin et al., (1984) reported that in light and middle weight categories, the Cuban boxers had higher VO$_2$ max, as compared to French boxers.

Guidetti et al., (2002) studied physiological factors in middle weight boxing performance and found that there are two basic factors related to boxing performance: physical fitness as indicated by individual anaerobic threshold and maximal oxygen consumption and upper body muscular strength as indicated by hand grip strength. Kravitz et al., (2003) reported that the fitness programs in boxing are comparable to other exercise modalities in cardiovascular response and caloric expenditure. The average VO$_2$max of elite boxers was observed to be 54.5 ml.kg$^{-1}$.min$^{-1}$ in Indian boxers, 55.8 ml.kg$^{-1}$.min$^{-1}$ in Greek National boxers, 56.6 ml.kg$^{-1}$.min$^{-1}$ in Hungarian boxers and was 64.7 ml.kg$^{-1}$.min$^{-1}$ in French boxers (Ghosh et al., 1995).

Lipids have important beneficial biologic functions that include the use of triglycerides, for energy production or as stored fat in adipose tissue and use of cholesterol as a component, in conjunction with phospholipids of cellular membranes or in the synthesis of steroid hormones (Durstine & Haskell, 1994). Dose response relationships between exercise training, volume training and blood lipid changes, suggest that exercise can favorably alter blood lipids at low training volumes, although the effects may not be observable, until certain exercise thresholds are met (Durstine et al., 2001).
Physical activity is known to promote health and welfare. The exercise is also responsible for raising the production of reactive oxygen species (ROS) through the addition of mitochondrial oxygen consumption by the tissues. The imbalance between the ROS production and the oxidant defenses of the tissues can result in oxidative damages to proteins, lipids and DNA. The oxidative damage to lipoprotein, particularly, low density lipoprotein (LDL), is know to have an important role in a number of age related diseases such as cardiovascular diseases, cancer and dementia (Strobel et al., 2011).

Increased physical activity induces a number of positive changes in the metabolism of lipoproteins. Serum triglycerides are lowered by the increased lipolytic activity and the productions of native high density lipoprotein (HDL) particles are increased. Increase in aerobic energy metabolism required during prolonged physical activity is significant control parameters for lipid metabolic process that occur during regular physical exercises training (Berg et al., 1994). Aerobic exercise has been shown to reduce the risk of cardiovascular disease (CVD). This reduction is proportional to the intensity of the exercise. The reduction of CVD risk is at least partially mediated by changes in circulating lipoproteins, resulting from adaptive changes in enzymes, involved in their metabolism. Specially, aerobic exercise is associated with reduction in low density lipoprotein (LDL), total cholesterol, and triacylglycerol (TAG), and increase in HDL (Herzberg, 2004). Kohl, (2001) reported that there was an inverse relationship between cardiovascular disease incidence and mortality and physical activity intensity. Endurance trained individuals have consistently higher plasma HDL levels than less active individuals (Lakka & Salonen, 1992; Marti et al., 1991). The influence of resistance training (Strength training) on HDL is contradictory. Some authors reported that HDL level of endurance trained and strength
trained athletes is similar (Cuppers et al., 1980; Hurley et al., 1984; Wallace et al., 1991) but other observed, lower HDL levels within strength trained group (Berg et al., 1980b; Farell et al., 1982). As the elevation of HDL is closely related to the catabolism of triglycerides, the key enzyme lipoprotein lipase (LPL) in triglycerides hydrolysis has been focussed on to explain the increase in HDL levels during exercise. Children have been reported to have low triglycerides concentration, which differs during developmental periods. Active children tend to have lower triglycerides level than their less active counterparts but training induced changes have not always been found (Higuchi et al., 1990; Durstine & Huskel, 1994). Low density lipoprotein cholesterol (LDL-C) is directly associated with cholesterol. A highly significant correlation is observed between cholesterol and LDL-C (Khanna et al., 1999). It has been reported that LDL-C has the greatest correlation to severity of coronary atherosclerosis (Aro et al., 1986). Cholesterol is removed by HDL from the tissues and transported it to the liver for degradation. The ratio of total cholesterol to HDL-C is considered as a risk factor of coronary atherosclerosis diseases (CAD).

Haskell, (1984) reported lower triglycerides concentrations in endurance trained subjects, while total cholesterol was either not changed or only slightly different. However, it became evident that physical activity had an impact on the lipoprotein lipid distribution. Generally, regular participation in physical activity is associated with lower plasma triglyceride concentrations. Results from cross – sectional studies using endurance athletes (Marti et al., 1991; Martin et al., 1977 ; Wood & Stefanick,1990), cross country skiers (Lehtonen, 1978) and tennis players indicate, lower triglycerides concentrations in active persons than in inactive controls.
The primary function of HDL has become clearer in recent years. It serves as the cholesterol acceptor in the reverse transport and excretion of cholesterol. Reverse cholesterol transport involves the movement of cholesterol by the HDL molecule from peripheral tissues back to the liver, where it is catabolised and excreted into the small intestine as bile. The reverse transport of cholesterol may incorporate several different pathways for the removal of cholesterol from the circulation (Tall, 1990). The pathways have the same origin for HDL, but it seems that several different exit points for cholesterol removal may exist (Tall, 1990).

Although cholesterol is an important component of cell membranes and is needed for the synthesis of steroid hormones, and elevated plasma cholesterol concentrations have been implicated in the development of CAD (Goodman, 1988). Some observational studies have reported lower plasma cholesterol concentration for endurance trained male (Williams et al., 1986) and female runners (Wood et al., 1977), but most studies observe no difference in plasma cholesterol concentration for male runners (Adner & Castelli, 1980; Hurter et al., 1975; Marti et al., 1991 ; Martin et al., 1977), female runners (Durstine et al., 1987), cross country skiers (Enger et al., 1980), and other endurance trained athletes compared to inactive counterparts. Similar (Berg et al., 1980 a, b) or higher plasma cholesterol concentration (Farrel et al., 1982) have been observed for speed trained and power trained athletes, compared to inactive controls. Maximal aerobic capacity has been inversely related to cholesterol concentration (Cooper et al., 1976), but when adjusted for age and body weight, these relationships no longer exist (Eriksen et al., 1981; Montoye et al., 1978). Accordingly, factors such as body weight, percentage of body fat and different patterns of dietary intake are important considerations in evaluating the effects of physical activity on plasma cholesterol concentration.
Observational studies, comparing LDL-C concentrations in men and women athletes, from various sports with those of inactive subjects, have produced mixed results, with no differences (Durstine et al., 1987; Haskell et al., 1980; Marti et al., 1991) and differences (Martin et al., 1977; Williams et al., 1986), being reported. Athletes participating in power or speed related events, have LDL–C concentration either similar to (Berg et al., 1980a) or lower than those inactive controls. Some, but not all (Brownell et al., 1982; Despres et al., 1988), longitudinal endurance exercise training studies have reported lower LDL-C concentrations for men and women (Despres et al., 1990; Sopko et al., 1985; Wood et al., 1988) following endurance training. Thompson et al., (1984) reported that exercise cessation, however, was associated with 10% increase in LDL-C level after only 2 days of inactivity.

The exogenous antioxidant intakes from habitual diets and the type of exercise and its intensity and duration applied during training are all factors that would influence blood antioxidant status in athletes (Ji et al., 1999).

Athletes at greatest risk for poor antioxidant intakes are those following a low-fat diet, restricting energy intakes, or limiting dietary intakes of fruits, vegetables and whole grains (Dunford, 2006; Mastloudis and Traber, 2006).

The antioxidant nutrients, vitamins C, E, beta carotene and selenium, play important roles in protecting cell membranes from oxidative damage. Because exercise can increase oxygen consumption by 10- to 15-fold, it has been hypothesized that chronic exercise produces a constant “oxidative stress” on the muscles and other cells (Powers et al., 2004) leading to lipid peroxidation of membranes.

Vitamin C is hydrophilic and widely distributed in mammalian tissues. It can act as a radical scavenger and recycles vitamin E (Powers et al., 2004; Powers and Jackson,
Vitamin E is lipid soluble and the major chain-breaking antioxidant found in cell membranes (Powers et al., 2004; Powers and Jackson, 2008).

The major dietary sources of vitamin C were fruits and vegetables with a very minor contribution from animal organs and milk. Fruit and vegetables may contribute vitamin C, other antioxidants and phyto-nutrients that confer health benefits. While vitamin C is a good marker of fruit intake (Szeto et al., 2002).

Independent of its function as an antioxidant vitamin C has a number of known and very important functions in relation to exercise. The vitamin C has long been known to be necessary for normal collagen synthesis and the formation of the vitamin like compound carnitine, necessary for the transport of long-chain fatty acids into the mitochondria. The neurotransmitters, norepinephrine and epinepherine also require vitamin C for their synthesis. Ascorbic acid assists in the transport of non-heam iron and the reduction of folic acid intermediates. The vitamin exerts antioxidant functions in plasma and probably interfaces at the lipid membrane level with vitamin E to regenerate vitamin E from the vitamin E radical (Peake, 2003; Keith, 2006).

Vitamin E, as well as vitamin C, is widely known as an important endogenous antioxidant against free radicals (Slavin, 2006). Under conditions of oxidative stress, perhaps these vitamins protect cell membranes and lead to reduced cell breakdown. The antioxidant effect of these vitamins on exercise, however, is controversial. Vitamin E supplementation during exercise does not appear to decrease exercise-induced lipid peroxidation in humans (Bhaskaram, 2002). More recently, another study has demonstrated that vitamin C and E supplementation in soccer players may reduce lipid peroxidation and muscle damage during high intensity efforts (Viitala & Newhouse 2004), but it was not shown to enhance performance (Zoppi et al., 2006).