Reactive Oxygen Species (ROS) are molecules that are unstable and take an electron or hydrogen from another molecule in order to achieve stability. The oxidative activity of these ROS is a part of normal function, but can impair the normal functioning of other molecules and systems if present in high amounts (McBride, 1999). The body has natural antioxidant systems that help to keep ROS levels normal; however, these antioxidant systems can be overwhelmed allowing an acute accumulation of ROS, a condition known as oxidative stress. The production of ROS beyond the normal capacity of the body’s own antioxidant system has been implicated in exercise fatigue, loss of function with aging, and several disease processes (McBride, 1999; Alessio, 2000b; Clarkson & Thompson, 2000; Sen et al., 2000; Lambert & Yang, 2003). Exercise can be a potent producer of ROS. During high intensity aerobic exercise some electrons leak out from the electron transport chain at specific complexes leading to the formation of superoxide radicals (Cooper et al., 2002). If tissues become ischemic or have insufficient blood flow during exercise, upon cessation of exercise, the reperfusion of the tissue with blood and oxygen will lead to the formation of a variety of ROS (McBride, 1999). Production of ROS is also part of the inflammatory process that accompanies muscle damage, such as that due to eccentric exercise (Clarkson & Thompson, 2000). There is also evidence that high intensity exercise well above the lactate threshold, that is more anaerobic, leads to greater oxidative stress than moderate intensity aerobic exercise (Quindry et al., 2003; Michailidis et al., 2007). The term antioxidant is difficult to define.
precisely because it is used to describe compounds with a wide variety of properties. However, a broad definition describes an antioxidant as any substance that when present at low concentrations compared with those of a substrate significantly delays or prevents oxidation of that substrate (Halliwell & Gutteridge, 1993). There are two general sources of the many different species of antioxidant molecules: exogenous and endogenous. Endogenous antioxidants stand for the body’s natural antioxidant system (i.e. Superoxide dismutase, Catalase, Glutathione peroxidase) which can inhibit the ROS activity in the cell membranes, deoxyribonucleic acid (DNA), proteins, and other cellular macromolecules. Both enzymatic and non-enzymatic antioxidants exist in intracellular and extracellular environments working with complex synergy to detoxify various ROS in animal tissue. Exogenous antioxidants (non-enzyme) are present in many kinds of foods, especially brightly-colored fruits and vegetables, as well as various vitamins and nutritional supplements. Due to the potential role of ROS in contributing to muscle homeostasis disturbance, it is logical that skeletal muscle cells contain defense mechanisms to minimize oxidative damage. Because exercise has been linked with increased ROS production, a clear reasoning exists for antioxidant adaptation to chronic exercise training (Ji et al., 1999). More than a decade ago, research demonstrated that increased exposure to ROS triggers an adaptation in antioxidant enzyme activity in mouse and rat cardiac and skeletal muscle tissues (Poulsen et al., 1999). In addition, there is evidence that aerobic endurance training can cause in increase in antioxidant capacity (AOC) compared to untrained individuals (Mena et al., 1991). Chronic moderate intensity exercise causes physiologic adaptations that enhance antioxidant defenses and could minimize oxidative stress both at rest and following exercise (Ortenblad et al., 1997; Selamoglu et al., 2000). Radicals are
molecules or fragments of molecules that possess an unpaired electron in their outer orbital. Because of this molecular instability, radicals are highly reactive and can promote damaging oxidation reactions with cellular proteins, lipids or DNA, leading to oxidative stress and impaired cellular function. Although regular physical exercise has many beneficial effects, it is now clear that muscular exercise results in increased production of radicals and other reactive oxygen species (Davies et al., 1982; Reid et al., 1992b; Borzone et al., 1994; O’Neill et al., 1996; Halliwell & Gutteridge, 1999). Furthermore, strong evidence indicates that reactive oxygen species are the primary cause of exercise induced disturbances in muscle oxidation–reduction status (i.e. redox balance). Severe disturbances in cellular redox balance have been shown to contribute to oxidative injury and muscle fatigue (Ji et al., 1988; Shindoh et al., 1990; Reid et al., 1992a; Nashawati et al., 1993; O’Neill et al., 1996). Given the potential role of reactive oxygen species in contributing to oxidative stress and muscle fatigue, it is not surprising that skeletal muscle fibres contain defence mechanisms to reduce the risk of oxidative damage. Two major classes of endogenous protective mechanisms, the enzymatic and non-enzymatic antioxidants, work to reduce the harmful effects of reactive oxygen species in cells. Furthermore, dietary antioxidants interact with endogenous antioxidants to form a cooperative antioxidant network.

**Overview of antioxidants**

Oxidative stress occurs due to an imbalance between oxidant production and the antioxidant capacity of the cell. Cells are protected against oxidant injury by a complex network of antioxidants. Specifically, enzymatic and non-enzymatic antioxidants exist in both the intracellular and extracellular environments and work as complex units to remove
different reactive oxygen species. To provide maximum intracellular protection, these scavengers are strategically compartmentalized throughout the cell. Several strategies are applied by both endogenous and exogenous antioxidants to protect against reactive oxygen species-mediated injury. These include conversion of reactive oxygen species into less active molecules (i.e. scavenging) and prevention of the transformation of the least reactive oxygen species into more damaging forms (i.e. conversion of hydrogen peroxide to the hydroxyl radical). In the following sections, we discuss important dietary antioxidants and define their role in maintaining muscle redox balance. Important non-enzymatic defences include, glutathione, vitamin E, vitamin C, lipoic acid, carotenoids, uric acid, bilirubin and ubiquinone.

**Glutathione**

Glutathione is the most abundant non-protein thiol source in muscle cells (Meister & Anderson, 1983). Glutathione is primarily synthesized in the liver and transported to tissues via the circulation. Because of the peptide structure of glutathione, it is degraded in the small intestine when ingested; hence, cellular concentrations of glutathione are not directly influenced by diet. Glutathione concentration in the cell is typically in the millimolar range, but there is wide variability in glutathione content across organs depending on their basal levels of radical production. For example, the two highest concentrations of glutathione in the body are found in the lens of the eye (10 mmol) and the liver (5–7 mmol) (Halliwell & Gutteridge, 1999). Other key organs such as the lung, kidney and heart contain about 2–3 mmol of glutathione (Ji, 1995a). Skeletal muscle glutathione concentration varies depending on muscle fibre type and animal species. In rats, (slow) type I fibres contain 600% more glutathione (3 mmol) than (fast) type IIB
fibres (0.5 mmol) (Ji, 1995a,b; Ji et al., 1992). Glutathione serves several roles in the cellular antioxidant defence system. First, glutathione directly scavenges a variety of radicals, including hydroxyl and carbon centered radicals, by donating a hydrogen atom (Yu, 1994). A second key antioxidant function of glutathione is to remove both hydrogen and organic peroxides (e.g. lipid peroxide) during a reaction catalyzed by the enzyme glutathione peroxidase. During this reaction, glutathione donates a pair of hydrogen atoms and two glutathione are oxidized to form glutathione disulphide. Glutathione has also been shown to be involved in reducing or ‘recycling’ a variety of antioxidants in the cell. For example, glutathione has been postulated to reduce vitamin E radicals that are formed in the chain-breaking reactions with alkoxy or lipid peroxy radicals (Packer, 1991). Furthermore, glutathione can also reduce the semi-dehydroascorbate radical (vitamin C radical) derived in the recycling of vitamin E and to reduce alpha-lipoic acid to dihydrolipoate. This reaction has recently been hypothesized to play an important role in the recycling of ascorbic acid (Packer, 1991). More will be said about these functions of glutathione in later sections.

Vitamin E

The generic term vitamin E refers to at least eight structural isomers of tocopherols or tocotrienols. Among these, a-tocopherol is the best known and possesses the most potent antioxidant activity (Burton & Ingold, 1989; Janero, 1991). From an antioxidant perspective, vitamin E is the primary chain-breaking antioxidant in cell membranes (Burton & Ingold, 1989; Janero, 1991). Because of its high lipid solubility, vitamin E is associated with lipid-rich structures such as mitochondria, sarcoplasmic reticulum and the plasma membrane. Under most dietary conditions, the concentration of vitamin E in tissues
is relatively low. For example, the ratio of vitamin E to lipids in the membrane may range from 1:1000 in red blood cells to 1:3000 in other tissues and organelles (Janero, 1991; Packer, 1991). Note, however, that vitamin E concentrations in tissues and organelles can be elevated with dietary supplementation (Janero, 1991). As an antioxidant, vitamin E is particularly important because of its ability to convert superoxide, hydroxyl and lipid peroxyl radicals to less reactive forms. Vitamin E can also break lipid peroxidation chain reactions that occur during free radical reactions in biological membranes (Burton & Traber, 1990). Although vitamin E is an efficient radical scavenger, the interaction of vitamin E with a radical results in a decrease in functional vitamin E and the formation of a vitamin E radical. Indeed, oxidative stress has been shown to significantly decrease tissue vitamin E concentrations (Burton & Traber, 1990; Janero, 1991; Packer, 1991).

However, the vitamin E radical can be ‘recycled’ back to its native state by a variety of other antioxidants (Packer et al., 1979; Burton & Traber, 1990). Therefore, it is postulated that the ability of vitamin E to serve as an antioxidant is synergistically connected to other antioxidants, such as glutathione, vitamin C and a-lipoic acid, which are capable of recycling vitamin E during periods of oxidative stress. This point is discussed in more detail in the next sections on vitamin C and a-lipoic acid.

**Vitamin C**

In contrast to vitamin E, vitamin C (ascorbic acid) is hydrophilic and functions better in aqueous environments than vitamin E. Because the pKa of ascorbic acid is 4.25, the ascorbate anion is the predominant form that exists at physiological pH (Yu, 1994). Ascorbate is widely distributed in mammalian tissues, but is present in relatively high amounts in the adrenal and pituitary glands (Yu, 1994). The role of vitamin C as an
antioxidant is two-fold. Vitamin C can directly scavenge superoxide, hydroxyl and lipid hydroperoxide radicals. Additionally, vitamin C plays an important role in recycling the vitamin E radical back to its reduced state (Packer et al., 1979). In the process of recycling vitamin E, reduced vitamin C is converted to a vitamin C (semi ascorbyl) radical (Packer et al., 1979). Recycling of the vitamin C radical can be achieved by NADH semi ascorbyl reductase, or cellular thiols such as glutathione and dihydrolipoic acid (Sevanian et al., 1985). In light of the role of vitamin C in the recycling of vitamin E, increased cellular concentrations of vitamin C should provide protection against radical-mediated injury (Yu, 1994).

However, in high concentrations (i.e. 1 mmol) vitamin C can exert pro-oxidant effects in the presence of transition metals such as Fe3+ or Cu2+. The pro-oxidant action of vitamin C stems from its ability to reduce ferric iron (Fe3+) to the ferrous (Fe2+) state. Ferrous iron is known to be a potent catalyst in the production of free radicals. Therefore, the wisdom of mega-dose vitamin C supplementation has been questioned by some investigators due to its pro-oxidant potential (Yu, 1994).

**α-Lipoic acid**

α-Lipoic acid is an endogenous thiol that serves as a cofactor for α-dehydrogenase complexes and participates in S–O transfer reactions (Packer, 1994). Normally, lipoic acid is present in very small quantities (5–25 nmol) in animal tissues and is generally bound to an enzyme complex that renders α-lipoic acid unavailable as an antioxidant (Packer, 1994). However, unbound α-lipoic acid may be effective as an antioxidant and in recycling vitamin C (Kagan et al., 1992; Packer, 1994). α-lipoic acid can be consumed in the diet and has no known toxic side-effects (Packer, 1994). Following dietary supplementation, α-
Lipoic acid is reduced to dihydrolipoic acid (DHLA), which is a potent antioxidant, against all major oxyradical species (Packer, 1994). Furthermore, DHLA is an important agent in recycling vitamin C during periods of oxidative stress and can be an effective glutathione substitute (Kagan et al., 1992; Packer, 1994).

**Figure No. 1:** The interaction between α-lipoic acid (αLA), glutathione and vitamin C (VC) in the recycling of vitamin E (VE). VC. = ascorbate radical; VE. = Vitamin E; DHLA= Dihydrolipoic acid; GSSG = oxidized glutathione (adapted from Ji, 1995a).

Vitamin C and DHLA recycle vitamin E during periods of oxidative stress. After the recycling of vitamin E, the vitamin C radical can be reduced back to vitamin C by DHLA. Dihydrolipoic acid is then converted back to α-lipoic acid in this process and can be reconverted to DHLA by cellular enzymatic mechanisms (Packer, 1994).

**Carotenoids**

Carotenoids (e.g. β-carotene) are lipid-soluble antioxidants located primarily in biological membranes. The antioxidant properties of carotenoids come from their structural arrangement consisting of long chains of conjugated double bonds; this arrangement permits the scavenging of several reactive oxygen species, including superoxide radicals
and peroxyl radicals (Yu, 1994). Indeed, carotenoids display an efficient biological antioxidant activity, as evidenced by their ability to reduce the rate of lipid peroxidation induced by radical generating systems (Krinsky & Deneke, 1982). Similar to vitamin C, β-carotene can function both as an antioxidant and a pro-oxidant. Under physiological oxygen partial pressures (i.e. 5100 mmHg), β-carotene exhibits radical scavenging activity. However, exposure to hyperoxic partial pressures (i.e. 4150 mmHg) results in β-carotene exerting pro-oxidant properties with a concomitant loss of its antioxidant capacity (Burton & Ingold, 1989; Palozza et al., 1997).

**Ubiquinone**

Ubiquinones are lipid-soluble quinone derivatives that contain an isoprene or farnesyl tail. Ubiquinone homologues containing 1–12 isoprene units occur in nature. Reduced forms of ubiquinones are better antioxidants by several orders of magnitude (Mellors & Tappel, 1966). The predominant form of ubiquinone in humans and many mammals is ubiquinone-10 (often called coenzyme Q) (Karlsson, 1997). The major sources of ubiquinone-10 in the diet are soybean oil, meats, fish, nuts, wheat germ and vegetables (beans, garlic, spinach, cabbage) (Kamei et al., 1986). The concentration of ubiquinone-10 in human plasma varies between 0.4 and 1.0 mmol _l^−1_; approximately 80% is present in the reduced (ubiquinol) state (Stocker et al., 1987; Aberg et al., 1992). In human tissue, ubiquinone-10 is found in relatively high concentrations (60–110 mg) in heart, liver and kidney, of which 70–100% is in the reduced state (Aberg et al., 1992).

**Trace minerals associated with antioxidant defenses**

Several trace minerals play important but indirect roles in providing antioxidant protection in cells. Trace minerals involved in antioxidant-related functions include copper
(Cu), zinc (Zn), iron (Fe), selenium (Se) and manganese (Mn). These trace minerals contribute to the body’s antioxidant defense system by acting as co-factors for antioxidant enzymes. In the following paragraphs, we provide a brief overview of the antioxidant function of each of these trace minerals. Supplementation of trace minerals is not generally necessary in well-nourished populations, but possible deficiencies of these minerals are addressed in the following discussion. Copper contributes to cellular antioxidant protection as a co-factor for the antioxidant enzyme, Copper Zinc superoxide dismutase (CuZnSOD). This enzyme, located in the cytosol of cells, is responsible for eliminating superoxide radicals. Although copper deficiencies are uncommon in Western society, a copper deficiency would result in reduced levels of functioning CuZnSOD and an impaired cellular antioxidant defense system. Signs and symptoms of copper deficiency include anaemia, a reduction in circulating neutrophils, bone loss and heart disease (Wardlaw & Insel, 1996; Halliwell & Gutteridge, 1999). Zinc has been recognized as an essential nutrient since the early 1900s and is a co-factor for over 300 different enzymes (Wardlaw & Insel, 1996). In terms of the role of zinc as an antioxidant, it is an essential cofactor for CuZnSOD. As mentioned above, this enzyme is responsible for the removal of superoxide radicals from the cytosol of cells. It follows that a zinc deficiency would result in diminished CuZnSOD activity that would contribute to an impaired antioxidant capacity.

**Iron**

Iron is the most abundant transition metal in the body and plays a key role in the function of both haemoglobin and myoglobin (Halliwell & Gutteridge, 1999). Furthermore, iron is an essential co-factor in the antioxidant enzyme catalase. Catalase is located in both the cytosol and the mitochondria and is responsible for removing hydrogen
peroxide from cells. An iron deficiency would not only impair oxygen transport in the body, but would also compromise the body’s antioxidant capacity by lowering catalase activity in cells (Halliwell & Gutteridge, 1999).

**Selenium**

Selenium plays a critical role in antioxidant defence as a co-factor for the antioxidant enzyme glutathione peroxidase. Glutathione peroxidase is located in both the cytosol and mitochondria of cells and is responsible for removing hydrogen peroxide and other organic hydroperoxides from the cell (Halliwell & Gutteridge, 1999). Selenium deficiency in humans has been reported in some areas of Europe and China and is associated with muscle pain, muscle wasting and cardiomyopathy (Wardlaw & Insel, 1996).

**Manganese**

Manganese is a co-factor for several enzymes, including the important antioxidant enzyme, manganese- superoxide dismutase. This key antioxidant enzyme is located in the mitochondria and is responsible for eliminating superoxide radicals produced by oxidative phosphorylation (Halliwell & Gutteridge, 1999). Manganese deficiency in animals results in impaired brain development and reproduction. Nonetheless, manganese deficiency has not been reported in humans (Wardlaw & Insel, 1996).

**Exercise-induced oxidant production**

Davies et al. (1982) were the first to report that skeletal muscles produce radicals during contractile activity. Since then, many investigations have confirmed these observations and have explored the potential sources of exercise-induced radicals and reactive oxygen species (O’Neill et al., 1996; Jackson, 1998; Reid, 2001; Ji, 2002; Reid &
Determining the primary sites of reactive oxygen species production in skeletal muscle is a complex undertaking, as many pathways are capable of generating radicals in skeletal muscle. Nonetheless, current evidence indicates that the primary sources of radical production in skeletal muscle are the mitochondria, xanthine oxidase, NAD(P)H oxidase and the production of nitric oxide by nitric oxide synthase (Davies et al., 1982; Jackson et al., 1985; Reid et al., 1992a,b; Borzone et al., 1994; O’Neill et al., 1996; Jackson, 1998). Secondary sources for radical production during exercise include auto-oxidation of catecholamines, radical generation by phagocytic white cells and radical formation due to the disruption of iron-containing proteins (Jackson, 1998; Halliwell & Gutteridge, 1999). Most investigators have concluded that radical production in the mitochondria is the primary source of radical production in contracting skeletal muscles. Indeed, while 95–98% of the oxygen consumption of skeletal muscle results in the formation of adenosine triphosphate (ATP) and water, the remaining 2–5% of this oxygen undergoes one electron reduction to produce superoxide radicals (Jackson, 1998; Halliwell & Gutteridge, 1999). It follows that increased muscular activity results in an elevation in oxidative metabolism and a proportional increase in superoxide production. Hence, this increased production of radicals must be balanced by the antioxidant capacity of the muscle to prevent oxidant mediated damage to proteins, lipids and deoxyribonucleic acid (DNA).

**Antioxidants and exercise performance**

Antioxidant deficiencies and exercise performance Antioxidant nutrient deficiencies are not widely reported among athletes (Clarkson, 1995). However, it is conceivable that an antioxidant nutrient deficiency could result in an increased
susceptibility to exercise induced damage by reactive oxygen species and thus lead to impaired exercise performance. Indeed, studies utilizing animal models have documented that vitamin E deficiency results in skeletal muscle degeneration and impaired exercise performance in rats (Davies et al., 1982; Gohil et al., 1986; Coombes et al., 2002). Vitamin C deficiency in guinea pigs has also been shown to reduce times to exhaustion during treadmill running (Packer et al., 1986). Failure to reverse the effects of vitamin E deficiency by vitamin C supplementation highlights the synergistic nature of antioxidant action (Gohil et al., 1986). It is important to emphasize caution when extrapolating results of animal studies to human populations. Although it is well established that a vitamin C deficiency impairs exercise performance in humans, marginally deficient individuals have not demonstrated similar adverse effects (van der Beek et al., 1990). Additionally, in contrast to the findings of animal studies, vitamin E deficiency in humans does not appear to be associated with impaired exercise performance. Males who were made vitamin E deficient over a period of 13 months did not suffer from impaired performance or muscle weakness despite blood concentrations of vitamin E that were indicative of deficiency (Bunnell et al., 1975). While the potential for antioxidant nutrient deficiencies do exist in athletes, the low incidence of vitamin deficiencies among athletes indicates that antioxidant deficiencies are not common (Clarkson, 1995).

**Antioxidant effects on muscle contraction and exercise performance**

It is well documented that exercise-related oxidant stress is associated with damage to lipids and protein in both muscle and blood cells (Alessio, 1993; Lawler et al., 1993; Jackson, 1998; Mastaloudis et al., 2001). In addition to imposing cellular damage, excessive reactive oxygen species has been shown to have an adverse effect on skeletal...
muscle contractile function and to exert a negative impact on performance (Reid & Durham, 2002). Pharmacologic antioxidant administration has been reported to decrease fatigue after electrically stimulated contractions of animal skeletal muscle (Barclay & Hansel, 1991; Reid et al., 1992a; Supinski et al., 1997). Additionally, infusion of N-acetylcysteine, a cysteine donor thought to increase the endogenous antioxidant glutathione, has been shown to attenuate muscle fatigue of the tibialis anterior and diaphragm muscles after low-frequency electrical stimulation in humans (Reid et al., 1994; Travaline et al., 1997). Collectively, these findings suggest that antioxidant supplementation may play a role in preserving skeletal muscle contractile function by scavenging exercise induced reactive oxygen species and reactive nitrogen species. The most convincing data suggesting ergogenic benefits from dietary antioxidant supplementation come from animal studies. Rodents with adequate nutritional status have demonstrated improved exercise performance after the administration of various forms of antioxidants (Novelli et al., 1990, 1991; Asha Devi et al., 2003). However, not all animal studies have demonstrated enhanced performance following antioxidant administration. For example, rats supplemented with vitamin E failed to improve treadmill endurance time to exhaustion (Mehlhorn et al., 1989; De oliveira et al., 2003). One of these reports, however, was based on a preliminary experiment involving only one animal (Mehlhorn et al., 1989). In contrast to studies conducted on animals, studies in humans generally have not demonstrated enhanced exercise performance after antioxidant supplementation. The vast majority of studies investigating vitamin E supplementation have not demonstrated improvements in exercise performance (Shephard et al., 1974; Lawrence et al., 1975; Sumida et al., 1989; Rokitzki et al., 1994a,b). The main exception is a study conducted at
high altitude in which the consumption of vitamin E was associated with a preservation of the anaerobic threshold (Simon-Schnass & Pabst, 1988). It was hypothesized that vitamin E supplementation at high altitude reduced red cell fragility and allowed for more efficient oxygen transport (Simon-Schnass & Pabst, 1988). Lack of a whole-body ergogenic effect for other antioxidants has also been reported in humans. In contrast to the findings of Reid et al. (1994), who found that administration of N-acetylcysteine resulted in decreased fatigue development in the tibialis anterior, infusion of N-acetylcysteine did not improve high intensity cycling performance in untrained males (Medved et al., 2003). Furthermore, other studies using antioxidant mixtures (Snider et al., 1992) or selenium (Tessier et al., 1995; Margaritis et al., 1997) have not demonstrated improved exercise performance. Investigations into the effects of vitamin C supplementation on exercise performance have demonstrated variable results. Vitamin C reportedly did not decrease markers of lipid peroxidation or improve recovery from unaccustomed exercise unless administered for 2 weeks prior to the exercise stress, which then resulted in modest improvements in muscle soreness (Thompson et al., 2001a, 2003). Other well-controlled studies with vitamin C have reported no beneficial effects on performance (Clarkson, 1995; Ashton et al., 1999). Ubiquinone-10 has been touted to possess ergogenic properties by increasing energy production via facilitating electron flux through the mitochondria and by functioning as an antioxidant. Nonetheless, among healthy individuals, only limited data illustrate the potential ergogenic properties of ubiquinone-10. In one study, a positive relationship between exercise capacity and the concentration of ubiquinone-10 in the vastus lateralis was reported in physically active males (Karlsson et al., 1996). However, most studies investigating the effects of ubiquinone-10 supplementation on exercise performance have
failed to authenticate these ergogenic claims. For example, supplementation of ubiquinone-10 alone (Braun et al., 1991; Weston et al., 1997; Bonetti et al., 2000) or in combination with other antioxidants (Snider et al., 1992) among groups of male athletes did not enhance performance. Additionally, male triathletes consuming ubiquinone-10 with ascorbic acid and vitamin E did not demonstrate altered energy metabolism or fatigue of the gastrocnemious muscle after plantar flexion exercise (Nielsen et al., 1999). Furthermore, some studies have actually demonstrated impaired performance following high-intensity (Malm et al., 1997) and endurance exercise tests (Laaksonen et al., 1995) among males supplemented with ubiquinone-10. Collectively, these studies do not support the use of ubiquinone-10 as a dietary supplement for the purpose of enhancing exercise performance.

**Exercise and antioxidant requirements**

As discussed earlier, current opinion holds that exercise-induced oxidative stress may be deleterious to exercise performance. This notion is based on cellular (Alessio, 1993; Lawler et al., 1993) and extracellular (Mastaloudis et al., 2001) indices of oxidant damage to lipids and proteins after exercise. Empirical data usually demonstrate that dietary antioxidant supplementation diminishes blood (Sumida et al., 1989; Ashton et al., 1999) and cellular markers (Goldfarb et al., 1994) of radical-mediated damage during exercise. Excessive exposure to environmental pollutants during training may further support the need for antioxidant supplementation (Papas, 1996). However, the current consensus on antioxidant supplementation for athletes remains equivocal because of a paucity of well-designed studies that clearly outline the need for dietary antioxidants in highly trained populations.
Exercise and oxidative stress: are athletes at increased risk?

Though not fully supported experimentally, a plausible rationale for supplementation of antioxidants does exist. The increased oxidant load experienced by competitive athletes during training is thought to necessitate antioxidant supplementation (Van der Beek, 1985). Training-related oxidant stress is associated with adaptations that improve the ability of the muscle cells to quench reactive oxygen species. Well-characterized examples of these adaptations include increases in enzymatic antioxidants within active skeletal muscle (Powers et al., 1999; Powers & Shanely, 2002). Despite these protective adaptations of exercise training against cellular oxidative stress, sustained exercise imposes an acute cellular oxidative stress even in highly adapted skeletal muscle (Higuchi et al., 1985). The greater training loads of competitive athletes, compared with their recreationally fit counterparts, may further compound this oxidative stress (Tiidus, 1998). In support of this notion, the magnitude of blood oxidative stress markers appears to be dose dependent relative to increased exercise duration (Hessel et al., 2000) and intensity (Alessio et al., 2000; Quindry et al., 2003). If the need for dietary antioxidant supplements exists, however, one would expect a causal relationship between acute physical activity and decreased concentrations of key plasma antioxidants. A recent investigation reported a 75% increase in a plasma marker of lipid peroxidation that corresponded with an increased rate of plasma vitamin E turnover after an ultramarathon run (Mastaloudis et al., 2001). Similarly, Bergholm and colleagues reported that chronic endurance running resulted in decreases of 18, 20 and 15% of plasma concentrations of α-tocopherol, β-carotene and retinal, respectively (Bergholm et al., 1999). Nonetheless, others have found either no change or even an increase in blood concentrations of both vitamin E and ascorbate after
an acute bout of endurance exercise (Liu et al., 1999). Importantly, caution is recommended when interpreting altered plasma antioxidant concentrations in response to acute exercise. Indeed, changes in plasma vitamin E and vitamin C during exercise may represent a complex redistribution between tissue and plasma antioxidant stores (Ji, 1995a; Liu et al., 1999). One also cannot assume that plasma redox status is indicative of cellular (e.g. muscle) redox balance (Quindry et al., 2003). Extended exposure to environmental air pollutants, including ozone, sulphur dioxide and nitrogen dioxide, during endurance training may present an additional oxidant source (Van Klaveren & Nemery, 1999). Moreover, high amounts of environmental air pollutants can limit exercise performance (Pierson et al., 1986). Certainly, exposure to these and other environmental factors, including ultraviolet radiation, has led to the suggested need for antioxidant supplementation (Papas, 1996; Packer & Valacchi, 2002). However, logic would suggest that if exercise creates a clear need for dietary antioxidant supplementation, epidemiological and/or empirical data would directly link exercise and oxidative stress to some form of morbidity. On the contrary, epidemiological data generally indicate that fit individuals are healthier than their sedentary counterparts (Blair et al., 2001).

Table No. 1: Functions of Non-enzymatic Antioxidant Molecules

<table>
<thead>
<tr>
<th>S.No</th>
<th>Non Enzymatic Antioxidants</th>
<th>Mode of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Vitamin E</td>
<td>Prevent plasma membranes from oxidation</td>
</tr>
<tr>
<td>2</td>
<td>Vitamin C</td>
<td>Regenerates Vitamin E and act against radicals</td>
</tr>
<tr>
<td>3</td>
<td>Uric Acid</td>
<td>Removes Hydroxyl radicals</td>
</tr>
<tr>
<td>4</td>
<td>A- Lipoic Acid</td>
<td>Recycling of Vitamin C</td>
</tr>
<tr>
<td>5</td>
<td>Glutathione</td>
<td>Perform numerous functions as cellular antioxidant</td>
</tr>
<tr>
<td>6</td>
<td>Bilirubin</td>
<td>May be act as extra cellular antioxidant</td>
</tr>
<tr>
<td>7</td>
<td>B-Carotene</td>
<td>Protect Plasma membrane from oxidative damage</td>
</tr>
<tr>
<td>8</td>
<td>Ubiquinones</td>
<td>Act as antioxidant in its reduced form</td>
</tr>
</tbody>
</table>

# The data in the above tables was taken from Powers & Hamilton (1999)
Figure No. 2: Free radicals in Mitochondrion. Mitochondrial oxidant production. Reactive intermediates such as superoxide, hydrogen peroxide, nitric oxide, and peroxynitrite form in mitochondria, where they cause oxidative damage. MtNOS, Mitochondrial nitric oxide synthase. MnSOD, manganese superoxide dismutase.

Table No. 2: Functions of Enzymatic Antioxidants Molecules

<table>
<thead>
<tr>
<th>S.NO.</th>
<th>Enzymatic Antioxidants</th>
<th>Their Role</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Glutathione Peroxidase</td>
<td>Remove Organic hydroperoxides and hydrogen peroxides</td>
</tr>
<tr>
<td>2</td>
<td>Catalase</td>
<td>Remove hydrogen peroxide</td>
</tr>
<tr>
<td>3</td>
<td>Superoxide dismutase</td>
<td>Converts two superoxide radicals ($O_2^-$) into hydrogen peroxide and molecular oxygen</td>
</tr>
</tbody>
</table>

# The data in the above tables was taken from Powers & Hamilton (1999)
**Figure No. 3:** Antioxidant enzymes defence mechanism against free radicals.

*Intermittent exercise* is a phrase used to describe a variety of different physical training types. The terms "intermittent," which means to stop and start at intervals. Intermittent is a term used to describe exercise where the intensity alternates, as occurs during interval training or in a game of boxing. For example, boxers are required to alternate between various modes of activity such as footwork, standing, upper body movement and rest intervals. In contrast to an endurance athlete such as a marathon runner whose movement speed tends to be relatively constant, the alternating modes of activity performed by the boxers place unique physical and physiological requirements on the body. For example, it has been found that performing intermittent exercise is more energy demanding than continuous exercise at the same mean running speed (Bangsbo, 1994).
Aerobic exercise involves the use of oxygen to produce energy, whereas anaerobic exercise makes the body produce energy without oxygen. The literal meaning of aerobic is oxygen. Aerobic exercise includes any activity that uses the large muscles of the body such as the arms and legs. When these muscles move in repetitive motion for an extended period of time oxygen is carried to the cells and energy is released. Ideally, the activity should last at least 20 minutes include constant movement.

The literal meaning of **anaerobic** is in the absence or insufficient supply of oxygen. Anaerobic exercise includes activities that build agility as well as strengthen and tone muscles. Anaerobic activity has been shown to increase longevity. For example, resistance training increases bone mass, reduce muscle loss, and improve balance.

1.1 **Statement of the problem**

Oxidative stress and level of antioxidants among physically active and sedentary males.

1.2 **Aims and Objectives**

Following were the aims and objectives of this study

1. To investigate the status of lipid-peroxidation and antioxidant levels in several distinct physically active and sedentary groups.

2. To compare the level of lipid-peroxidation and antioxidants among distinct physically active and sedentary groups.

1.3 **Hypotheses**

It was hypothesized that:-

1. The oxidative stress of the physically active population will be more as compare to sedentary population.
2. The level of anti-oxidants will be more in physically active population as compared to sedentary population.

1.4 Limitations

1. The present study will be limited to various genetic factors.
2. The present study will be limited to different exercise/training programme as the subjects of the present study will be from different sports disciplines.

1.5 Delimitations

1. The study will be delimited to only those athletes, who would be available in NS NIS, Patiala during the period of study.
2. The study will be delimited to selected biochemical parameters in the serum/plasma for the oxidative stress and antioxidant status.
3. The study will be delimited to the male subjects of age group of 18 to 24 years only.

1.6 Inclusion Criteria

Only physically active subjects with a minimum of eight training sessions per week, more than 8 hours of training per week and with each training session lasting 1 hour were selected. Sedentary subjects with no regular physical activity for a minimum of 1 year were selected. Participation in the study was voluntary.

1.7 Exclusion Criteria

The exclusion criteria were:

1. Subjects with illness.
2. Known metabolic disease
3. Smoking and regular alcohol users
1.8 Significance

1. This study will help us to know the pattern of various oxidative and antioxidant markers among the different sports disciplines as well as in sedentary population.

2. This study will also help us to identify which type of training (Aerobic, anaerobic & intermittent) is beneficial to develop antioxidant system.

3. This study will also helpful to know that which type of activity is responsible to produce more oxidative stress.

4. The present study serves as guidelines for the nutritionist and sport’s doctors for prescribing/suggesting dietary antioxidant supplements to the athletes.