Chapter 9  Acute and Chronic Effects of Antioxidant Supplementation on Exercise Performance

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9.1. INTRODUCTION

Reactive oxygen and nitrogen species (RONS), also known as free radicals, are continually produced within the body as part of normal oxidative metabolism (Finaud et al. 2006; Powers et al. 2011). These molecules act as intracellular messengers and are necessary for proper physiological function (Dröge 2002; Niess 2005). However, high concentrations of RONS can be toxic and cause significant oxidative damage to the cellular structure of lipids, protein and DNA (Halliwell and Gutteridge 1999; Powers et al. 2004). The concentration of RONS within the body is controlled by an extensive antioxidant system, which works to scavenge free radicals (Halliwell and Gutteridge 1999; Powers et al. 2004). Antioxidants (AOX) are present in both the intra- and extra-cellular matrix forming a complex defence system to protect cells and tissue against oxidative damage (Powers and Lennon 1999). The antioxidant defence system is commonly divided into enzymatic (endogenous) and non-enzymatic (exogenous) AOX.

Seifried et al. (2007) define AOX as a group of compounds characterised by their ability to be oxidised in place of other compounds present. The main enzymatic AOX include superoxide dismutase, catalase and glutathione peroxidase. Vitamin A, vitamin C, vitamin E, thiols, flavonoids and ubiquinones represent the main non-enzymatic AOX, which can be obtained in a conventional diet and with supplementation. This has led to the suggestion that AOX supplementation may result in an acute improvement in skeletal muscle contractile performance (MacRae and Mefford 2006; Oh et al. 2010). This hypothesis is based on the finding that the rapid elevation of oxidant concentration during exercise may be a contributory factor to muscle fatigue (Reid et al. 1994; Gomez-Cabrera et al. 2008). However, other reports have stated that AOX supplementation in combination with exercise training may blunt exercise-induced biochemical adaptations to exercise (Reid 2001; Watson et al. 2005). This chapter provides a comprehensive overview of the acute exercise responses and longer term adaptations to the common antioxidant supplements.

Prolonged exercise training induces marked changes in physiological function and skeletal muscle contractile performance (Kubukeli et al. 2002; Laursen and Jenkins 2002). The oxidant–antioxidant balance during exercise has been shown to greatly influence muscular contraction (Clarkson and Thompson 2000) and adaptation to physical training (Palazzetti et al. 2003). It has been proposed that optimising skeletal muscle oxidant concentration by consuming antioxidant substances (acutely) results in a greater force production and power output during prolonged high-intensity endurance exercise (Poulsen et al. 1996; Alessio et al. 2000). However, studies have also shown that physiological adaptations to exercise may be blunted when oxidant production is suppressed by AOX (Palazzetti et al. 2003; Watson et al. 2005). Therefore, consideration should be made of the research concerning both acute supplementation with AOX and exercise performance and also more chronic consumption and the possible consequences. The type of AOX substance/content is also important when evaluating the potential use and physiological significance of the different AOX supplements.

9.2. REACTIVE OXYGEN SPECIES, EXERCISE PERFORMANCE AND FATIGUE

An elevated concentration of oxidants within the skeletal muscle may cause oxidative damage to the mitochondria and muscle contractile proteins, interfering with the excitation–contraction coupling process (Vollaard et al. 2005; Powers et al. 2011). However, in vitro studies have also shown that myofibril contraction is inhibited when RONS production is suppressed, prompting the suggestion that optimal force production requires the maintenance of a moderate level of oxidants within the muscle during exercise (Reid 2001; Gomez-Cabrera et al. 2008). Based on these
findings, an inverted-U shaped model of cellular redox balance and contractile function has been proposed as shown in Figure 9.1 (Reid 2001).

It is well established that RONS production is increased following most exercise types, durations and intensities in a dose-dependent manner (Ashton et al. 1998; Alessio et al. 2000; Aguiló et al. 2005; Watson et al. 2005; Finaud et al. 2006). In order to prevent the occurrence of severe oxidative damage during exercise, the body must continually re-enforce its antioxidant protection. Physiological adaptation to oxidative stress relies on the process of redox signalling (Dröge 2002). When an increased production of RONS occurs, redox signalling is used to induce protective mechanisms, predominately the up-regulation of antioxidant responses, to restore the redox homeostatic balance (Dröge 2002). During exercise, the redox balance is disturbed due to the exercise-induced increase in free radical production (Ashton et al. 1998). This initiates a redox signalling cascade, which leads to an increase in antioxidant enzyme expression and facilitates the mobilisation of exogenous AOX (Atalay et al. 1996; Miyazaki et al. 2001; Aguiló et al. 2003; Groussard et al. 2003). Allowing the body to adapt to the oxidative stress through improving the antioxidant defence system enables the re-establishment of redox homeostasis.

The balance between oxidant production and antioxidant removal is vital to the regulation of cellular functions (Banerjee et al. 2003). Low concentrations of free radicals are necessary for proper regulation of cellular function, but higher concentrations can lead to cellular damage and oxidative stress (Halliwell and Gutteridge 1999; Lachance et al. 2001; Reid 2001).

If the antioxidant response is insufficient or free radical production is chronically increased, the body may not be able to return to the previous level of redox homeostasis (Dröge 2002). Instead, the oxidant/antioxidant balance will find a new point of equilibrium, which will have a substantially increased concentration of free radicals. The new higher equilibrium point may affect the redox signalling pathways, resulting in altered patterns of gene expression and an inability to adapt to further oxidative stress (Dröge 2002).

A pro-oxidant shift in redox homeostasis has been observed in several pathological diseases such as diabetes, cancer, rheumatoid arthritis and ageing (Clarkson and Thompson 2000; Gomez-Cabrera et al. 2008). It is likely that intensified periods of physical training with minimal recovery may also perturb redox homeostasis, inducing a state of chronic oxidative stress (Itoh et al. 2000; Santos-Silva et al. 2001; Palazzetti et al. 2003). Chronic oxidative stress may then inhibit physiological adaptations to exercise and contribute to negative exercise physiological states including the development of overreaching and, in severe cases, overtraining (Palazzetti et al. 2003). It is therefore important to control the production of free radicals within the body in order to maintain redox homeostasis and normal physiological function.

Under conditions of physiological stress such as during intense exercise, free radical production increases dramatically, altering the redox state of the muscle and possibly inhibiting muscle contractile function (Reid et al. 1994; Powers et al. 2011). The resulting consequence would be an increased onset of muscle fatigue and reduction in exercise performance. However, it has been postulated that the consumption of AOX during exercise may assist in the maintenance of an optimal level of RONS within the skeletal muscle. Antioxidant substances may help neutralise free radicals and thereby prolong skeletal muscle integrity and prevent a decline in performance (Morillas-Ruiz et al. 2005; Oh et al. 2010).

9.3. ANTIOXIDANT SUPPLEMENTATION AND EXERCISE ADAPTATION

9.3.1. CHRONIC ADAPTATION TO EXERCISE TRAINING AND ANTIOXIDANT SUPPLEMENTATION

The theory of training highlights the importance of imposing a certain amount of stress upon the body in order to stimulate physiological adaptations. Exercise is a significant stressor to the body, which initiates adaptive processes in biological systems to accommodate the increase in physical work demand. The outcome of regular exercise training is largely known including physiological adaptations such as improved cardiovascular function and skeletal muscle respiratory capacity as well as improved functional performance (Bigard et al. 1996; Billat et al. 1999).
It has become increasingly evident that RONS act as intra-cellular messengers to stimulate changes in cell function and regulate gene expression (Pattwell and Jackson 2004). Nuclear factor kappa-B (NF-κB) is one of the most commonly investigated redox sensitive transcription factors. Some authors believe that activation of NF-κB may be an important regulator of adaptation to exercise training (Ji et al. 2004; Ho et al. 2005; Cuevas et al. 2005). This hypothesis is based on several factors including the NF-κB induced up-regulation of antioxidant enzymes to maintain red-ox homeostasis (Zhou et al. 2001), the inflammatory response associated with NF-κB on skeletal muscle (Ortega et al. 1999) and the reduced binding capacity of NF-κB during fatiguing exercise (Durham et al. 2004).

Exercise-induced increases in RONS production are likely to stimulate the red-ox sensitive NF-κB, resulting in the up-regulation of antioxidant gene expression (Hollander et al. 2001; Ho et al. 2005). An example of this has been shown by Hollander et al. (2001) where a significant increase in the binding capacity of NF-κB and subsequent up-regulation of the antioxidant enzyme superoxide dismutase was observed in response to a single bout of exercise in rat skeletal muscle. Moreover, in a study by Gomez-Cabrera et al. (2005), inhibition of xanthine oxidase-mediated RONS production by the antioxidant allopurinol significantly blunted the exercise-induced increase in NF-κB and prevented increases in antioxidant enzymes from occurring.

These studies show the importance of NF-κB activation to up-regulate antioxidant protection following exercise (Ji et al. 2004; Fernández et al. 2005). Individuals who regularly participate in physical activity display an augmented endogenous antioxidant enzyme capacity (Brites et al. 1999) and an increased tolerance to exercise-induced oxidative stress (Brites et al. 1999; Pittaluga et al. 2006). Without a dynamic and adaptable antioxidant defence system, the body would not be able to cope with increasing amounts of oxidant production and a state of chronic oxidative stress may occur (Dröge 2002). Consequently, it may be detrimental to the endogenous antioxidant defence system if the exercise-induced redox signalling processes are blunted by exogenous AOX supplementation. It is hypothesised that chronic antioxidant supplementation may blunt training adaptations and be harmful to exercise performance (Reid 2001; Watson et al. 2005). This is supported by a study where triathletes who regularly used antioxidant supplements incurred a greater amount of oxidative stress following an Ironman race than those who did not use antioxidant supplements (Knez et al. 2007). However, not all investigations have shown that AOX supplementation impedes exercise-induced activation of redox sensitive signalling pathways (Petersen et al. 2012). It has also been demonstrated that quercetin supplementation (1000 mg/day) promotes skeletal muscle mRNA expression of genes involved in mitochondrial biogenesis in 26 previously untrained males during a 2-week physical training period (Nieman et al. 2010). These studies highlight the difference in AOX supplementation strategies that have been used in combination with exercise training and also that it may be more beneficial to only increase antioxidant consumption during periods of elevated training stress. It is also likely that a dose-response effect could be evident, where the amount of AOX required to optimise oxidant content in the skeletal muscle is relative to the type and amount of exercise undertaken. This is yet to be fully investigated and should be a focus of future antioxidant research.

### 9.3.2. Acute Exercise Responses to Antioxidant Supplementation

The majority of studies have focused on the use of AOX to minimise exercise-induced oxidative stress, muscle damage and inflammation (Tauler et al. 2006; Gomez-Cabrera et al. 2005; Mastaloudis et al. 2004; Thompson et al. 2004; Morillas-Ruiz et al. 2005). While these data are relevant for the potential efficiency of AOX supplementation as a strategy for optimising adaptations to exercise training, there is less research available on the potential (acute) ergogenic effect of AOX on muscle contractile performance during exercise.

During exercise/muscle contraction antioxidant stores may become depleted, leaving the body susceptible to oxidative damage (Powers et al. 2004; Morillas-Ruiz et al. 2005). Numerous studies have investigated the effects of antioxidant supplementation on exercise-induced oxidative stress with equivocal results. This is most likely due to the wide variety of exercise/supplementation protocols including the length of supplementation period and type of AOX and the exercise task used by each research group. Many investigations have tested one particular AOX substance, yet it appears that a combination of different AOX could be more effective at combating RONS than a single substance (Tauler et al. 2006; MacRae and Mefford 2006; Bloomer et al. 2006). This reflects the complex nature of the antioxidant defence system, whereby each antioxidant is most efficient at quenching a certain type of radical species.
Medved et al. (2004) reported a 26.3% increase in cycling time to fatigue at 90% \( \text{VO}_2 \) peak when N-acetylcysteine (NAC) was intravenously infused in eight endurance trained men. Kingsley et al. (2005) observed that supplementation with 750 mg.day\(^{-1} \) phosphatidylserine for 10 days had a tendency to improve time to exhaustion in a shuttle running test. These studies support the idea that a single AOX supplement can improve exercise performance. An investigation by MacRae and Mefford (2006) reported that the addition of the flavonoid quercetin to a liquid antioxidant supplement (green tea extract, vitamin C, vitamin E, caffeine, niacin, taurine and vitamin B groups) significantly enhanced the antioxidant effect of the supplement and resulted in a 3.1% performance improvement during a 30 km cycle time trial. Hence, it is possible that a combination of AOX compounds may induce larger effects on exercise performance. However, in contrast, other investigations have not shown any improvement in exercise performance after a period of AOX supplementation. For instance, no increase in time to exhaustion in moderately fit men was found by Romano-Ely et al. (2006) when the subjects consumed a carbohydrate–protein–antioxidant drink containing vitamins E and C.

The majority of studies examining the effects of AOX supplementation have investigated the effects on endurance exercise performance. Resistance or weight training is known to induce considerable increases in RONS (Bloomer 2007). There are very few studies that have examined the effects of AOX supplementation (short or long term) on resistance exercise performance. In one study, Lafay et al. (2009), using a double-blind crossover design, investigated the influence of 4 week supplementation (400 mg/day) of a grape extract rich in polyphenols or a placebo on jumping force capacity. After 4 weeks of polyphenol supplementation, there was a 19% improvement in work capacity during 45 s of bodyweight squat jumps (Lafay et al. 2009). Similarly, 30 days of supplementing with a vitamin C and E combination helped attenuate a decline in maximal force production, peak concentric torque and total work performed during 300 consecutive isometric contractions (Shafat et al. 2004). Despite the equivocal findings, these results suggest that the acute consumption of AOX during exercise may assist in the maintenance of an optimal level of RONS within the skeletal muscle and lead to improvements in both endurance and resistance training performance.

### 9.4. SPECIFIC TYPES OF ANTIOXIDANT COMPOUNDS AND EXERCISE

#### 9.4.1. Vitamins E, C and Carotenoids

The vitamin E family consists of at least eight structural isomers, with \( \alpha \)-tocopherol displaying the most potent antioxidant properties (Burton et al. 1982). Vitamin E is a phenolic compound and is capable of hydrogen donation, which can convert superoxide and hydroxyl radicals to more stable forms (Burton et al. 1982; Powers et al. 2004). Vitamin E is lipid soluble and is found in lipid-rich structures such as the sarcoplasmic reticulum and cellular membrane where it is capable of scavenging free radicals produced from the mitochondria (Ji 1995).

Vitamin C (ascorbic acid) is water soluble and is present in the cytoplasm of the cell. Vitamin C can scavenge for superoxide and hydroxyl radicals and also help to recycle vitamin E back to its reduced state. Vitamin C also functions as a pro-oxidant by reducing ferric iron (Fe\(^{3+} \)) to ferrous iron (Fe\(^{2+} \)) (Aust et al. 1985; Powers et al. 2004; Yu 1994). Carotenoids are similar to vitamin C in that they are capable of antioxidant and pro-oxidant functioning. Both types are also lipid-soluble AOX mostly located in cellular membranes and are effective in preventing lipid oxidation by quenching free radicals such as singlet oxygen (Yu 1994; Powers et al. 2004).

The majority of studies have attempted to examine the longer term effects of vitamin E or C supplementation on exercise performance and antioxidant capacity (Kanter et al. 1993; Mastaloudis et al. 2004). Vitamin C and E supplementation for 6 weeks has been shown to decrease lipid peroxidation (F-2-isoprostanes) after endurance exercise (Mastaloudis et al. 2004). Similarly, 600 mg of vitamin E supplementation for 6 weeks also decreased lipid peroxidation (MDA) after 30 min of exercise (Kanter et al. 1993). There is evidence to suggest that the ergogenic effect of vitamins A, C and E during exercise is minimal as 500 mg vitamin C supplementation per day for 1 month did not improve maximal aerobic capacity (\( \text{VO}_2\text{max} \)) in healthy young males. Furthermore, vitamin E supplementation (900 IU/day) for 6 months did not improve swimming performance over the study period (Bell et al. 2005; Lawrence et al. 1975). An antioxidant cocktail mixture including vitamins A, C and E and orally consumed for 6 weeks had no ergogenic properties during a 30 km cycling time trial (TT) in well-trained male cyclists (MacRae and Mefford 2006).
These results suggest that elevated ingestion of these vitamins did not optimise oxidant content within the skeletal muscle or have any effect on muscle contractile function and therefore did not improve exercise performance.

### 9.4.2. **Glutathione and n-Acetylcysteine Supplementation**

Glutathione (GSH) is the most abundant low-molecular weight thiol present in muscle cells and the intracellular levels of GSH are normally around 0.5 mM (Yu 1994). Glutathione can react with free radicals, such as the hydroxyl radical and carbon radicals, by donating a hydrogen atom and is needed by the body to remove both hydrogen and organic peroxides such as lipid peroxide (Powers et al. 2004).

n-Acetylcysteine is a reduced thiol donor with antioxidant properties that support glutathione synthesis. Importantly, it is the acetylated derivative of cysteine which is the rate limiting step in glutathione synthesis (Sen 2001; Ferreira and Reid 2008).

Studies using isolated muscle preparations have shown a reduction in fatigue in NAC-treated rabbit diaphragm muscles and NAC infusion has also been shown to improve the functioning of skeletal muscle (Reid et al. 1994). Similarly, in a double-blind crossover study, transcutaneous electrical stimulation was employed to stimulate the tibialis anterior muscle for 30 min in healthy young males with and without NAC infusions (150 mg/kg). The NAC infusion trial resulted in an improved fatigue resistance of, on average, 15% during the trial. However, as transcutaneous stimulation is not a normal physiological process, the practical application of these findings on exercise performance is limited (Ferreira and Reid 2008; Reid et al. 1994). More recent work involving endurance-trained participants has demonstrated that intravenous infusion of NAC at 125 mg·kg⁻¹·h⁻¹ prior to exercise (20 min) and then during the exercise protocol at a rate of 25 mg·kg⁻¹·h⁻¹ improved endurance cycling time to fatigue (Medved et al. 2004; McKenna et al. 2006). Participants cycled for 45 min at a workload that corresponded to 71% peak \(\dot{V}O_{2max}\); they then cycled to fatigue at a workload of 92% \(\dot{V}O_{2max}\). During the NAC trials, time to fatigue at 92% \(\dot{V}O_{2max}\) increased by 23.8% and 26.3%, respectively, during two separate studies (Medved et al. 2004; McKenna et al. 2006). While these results show a positive effect of NAC on exercise performance, the effect of NAC via methods other than infusion needs to be established. Oral NAC supplementation of 1800 mg per day for 4 days increased knee extensor endurance during sub-maximal knee extensions and an oral solution containing 150 mg kg⁻¹ of NAC improved the endurance of handgrip repetitions during sub-maximal handgrip contractions (Koehlin et al. 2004; Matuszczak et al. 2005). In a recent study, Slattery et al. (2014) have shown that an oral dose of NAC (1200 mg/day for 9 days) improved repeat sprint performance during a cycle ergometer race simulation. Not all studies have shown that NAC improves exercise performance. NAC infusion did not have any effect on total work production during three short intensive cycling bouts (45 s/bout) in healthy male subjects and it did not improve time to fatigue at 130% \(\dot{V}O_{2max}\) (Medved et al. 2003). The authors suggested that NAC supplementation did not improve performance because of the high force requirements of the exercise task. The available studies suggest that NAC delays fatigue in moderate intensity exercise, but NAC appears to have few ergogenic properties during severe high-intensity exercise tasks (Ferreira and Reid 2008).

### 9.4.3. **Flavonoids**

Flavonoids are commonly found in edible plants and are diphenylpropanes which include family members such as flavones, isoflavones and anthocyanins (Cao et al. 1997). Polyphenolic flavonoids are capable of scavenging superoxide, hydroxyl and peroxyl free radicals and are therefore potent AOX (Powers et al. 2004).

Quercetin is an example of one such polyphenolic flavonoid substance that has been shown to improve exercise performance (MacRae and Mefferd 2006; Davis et al. 2010). In a recent meta-analysis containing data from 11 studies on human participants, quercetin was reported to have a small but significant ergogenic effect on endurance performance (Kressler 2011). Indeed, Davis et al. (2010) utilised a placebo- controlled crossover design to investigate the effects of a 7-day quercetin supplementation protocol (500 mg twice per day) on time to fatigue at 75% \(\dot{V}O_{2max}\) during a cycle ergometer trial in healthy male and female participants. Following quercetin supplementation, time to fatigue increased by 13% (105 min compared to 93 min) when compared to the placebo trial (Davis et al. 2010). A
beneficial effect of quercetin on exercise was also observed by Nieman et al. (2010) using a double-blind crossover design in male participants supplemented with 1 g/day of quercetin for 2 weeks. The participants walked on a treadmill for 60 min at an intensity of 60% \( \dot{V}O_{2\text{max}} \) and then had 12 min to cover as much distance as possible. Quercetin supplementation significantly increased the distance covered in the 12 min distance trial by \(~3\%\) compared to the placebo trial. There was also a \(3\%\) improvement in 30 km time trial performance in trained male cyclists after 6 weeks of quercetin supplementation (MacRae and Mefford 2006). Nonetheless, these improvements in performance with quercetin supplementation have not been consistently reported (Ganio et al. 2010). Exercise performance has been shown to improve following supplementation with other flavonoid antioxidant substances. In a recent study, ekclonia cava polyphenol (ECP) was shown to improve time to fatigue during a running trial (Oh et al. 2010). Using a double-blind crossover design, healthy male subjects consumed either a liquid placebo solution or a liquid ECP solution containing 60 mg of ECP 30 min before exercise. The acute polyphenol supplementation with ECP significantly increased time to fatigue by around 2.5 min (Oh et al. 2010). These results may be either due to ECP’s ability to scavenge excessive RONS or to the ability of polyphenol flavonoids to exert a vasodilatory effect which may promote blood flow to the working muscles (Pietta 2000; Oh et al. 2010).

Pycnogenol (PYC) is a commercially available pine bark extract containing oligomeric proanthocyanidins (OPC), which has been shown to be associated with a variety of clinical therapeutic benefits (Slayback and Watson 2002; D'Andrea 2010). However, the effects of PYC supplementation on exercise performance have received little scientific investigation. In one study by Pavlovic (1999), PYC supplementation was evaluated using a double-blind crossover design incorporating athletic males who were supplemented with 200 mg of either a placebo or PYC per day for a month. The exercise test was a time to fatigue protocol at 85% \( \dot{V}O_{2\text{max}} \) and there was a significant 21% increase in this parameter after a month of PYC supplementation.

In a more recent investigation, Clifford et al. (2013) found that orally consuming 120 mg of PYC with 600 mg of bioflavonoids (PYC-B) had a positive influence on cycling performance. Using a double-blind crossover design, trained cyclists and triathletes supplemented with either a placebo or the PYC-B supplement for 3 days prior to a 20 km time trial. The PYC-B supplement enabled participants to significantly enhance their power output in the final 5 km of the trial and improve their time by 3.8 s in the final 1 km.

Pycnogenol is the active ingredient of a new commercially available supplement ‘cocktail’. The acute effects of this supplement have been investigated in both endurance and resistance exercise modes (Bentley et al. 2012; Mach et al. 2010). In a double-blind crossover design study, the effects of a single dose of this supplement on time to fatigue in an endurance cycle exercise in trained and untrained subjects were investigated. The subjects consumed 150 mL of liquid placebo or 150 mL of the supplement containing 360 mg of PYC prior to exercise. The subjects cycled at workloads of 50% and 70% peak power output for 4 min per stage; then time to fatigue was assessed at 95% peak power output, and PYC supplementation significantly increased time to fatigue in both trained and untrained cyclists by approximately 17% (Mach et al. 2010). This was subsequently confirmed by another separate investigation in trained cyclists (Bentley et al. 2012). These studies indicate that, depending upon the supplement type and dosing period, PYC has potential acute ergogenic effects, which are in contrast with those of other AOX compounds.

### 9.5. SUMMARY

In summary, RONS or free radicals are required at low concentrations for many important physiological functions. However, the dramatic increase in RONS during severe exercise can damage cell membranes and interfere with excitation contraction coupling, having deleterious effects on skeletal muscle performance. An increase in RONS disturbs redox balance within the muscle, prompting red-ox signalling to up-regulate the use of AOX, particularly exogenous AOX. It has therefore been suggested that antioxidant supplements may be able to assist exercise performance by reducing the excessive exercise-induced oxidative stress response. However, other important chronic physiological adaptations to exercise may be blunted if skeletal muscle oxidant concentration is too low. Nonetheless, as skeletal muscle requires a moderate oxidant concentration to optimise force production, it has been suggested that acute doses opposed to chronic consumption of AOX may be more beneficial to exercise performance. Numerous antioxidant supplements have been studied for their ergogenic potential including vitamins A, C and E, N-
acetylcysteine and various flavonoids. While not all studies have found positive results on exercise performance or adaptations, many investigations have demonstrated that antioxidant supplementation, particularly when combined as a ‘cocktail’, can have an ergogenic effect on both high intensity endurance and resistance exercise performance.

REFERENCES


**Figures**

**FIGURE 9.1**

Optimal redox state for skeletal muscle contraction. A is the basal level of ROS within the muscle, B is the optimal level of ROS for contractile function and C is the excessive amount of ROS inhibiting muscle function and inducing fatigue.