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Effect of antioxidant supplement on performance in rowers

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EFFECT OF ANTIOXIDANT SUPPLEMENT ON PERFORMANCE IN ROWERS

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CERTIFICATE OF APPROVAL

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MASTER OF SCIENCE THESIS

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This is to certify that the thesis of

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submitted in partial fulfillment of the requirements for the
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ABSTRACT

The effects of flavanoid-containing sport drinks on athletic performance, as reported in previous research, are equivocal. In short, these studies have not modeled the direct and indirect effects of flavanoid-containing drinks on specific athletic performance; thus they have not explored their effects on improving and maintaining specific athletic performance. The purpose of this study was to explore the effects of a flavanoid (i.e., quercetin) containing sport drink on rowing performance during the off-season. This study had two parts. Part one measured the chronic effects of FRS (free radical scavenger) on rowing performance, whereas part two measured the acute effects. Fifty male and female collegiate rowers between the ages of 18 to 25 years were recruited and pair-matched on 2000 m rowing and then placed into either a supplement or placebo group for the first part of the study. Baseline data included 500 m and 2000 m rowing ergometer tests, 1RM bench press test, and vertical jump test. Subjects consumed a supplement or placebo drink twice a day for three weeks. After three weeks, subjects again reported to the lab for measurement of the aforementioned tests. In the second part of the study, subjects of the placebo group were pair-matched on 2000 m rowing ergometer performance and placed into the supplement or placebo group. This time, the supplement or placebo was given twice in the 150 min before testing 500m and 2000m rowing performances. Data were analyzed using 2×2 repeated measures ANOVA. Results from this study do not show any evidence for improvement in performances with the quercetin supplement. It can be concluded that acute and/or chronic supplementation with FRS does not have any effects on performance in rowers as tested.

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TABLE OF CONTENTS

LIST OF TABLES

Chapter 1

Introduction

For an athlete, it is very important to optimize performance and avoid fatigue throughout competition. Athletes, seeking to boost performance, rely on practicing proper technique, increasing conditioning, and improving nutritional intake. There are many putative performance enhancing products available and use is often based on magazine advertisements, peer advice, or coach recommendation rather than objective information. A growing awareness of the synergy between diet and performance has fueled expanding interest in the role that micronutrients might play in improving physical performance. Mitigating fatigue, muscle soreness, free radical production, and homeostatic imbalance are all mechanisms that micronutrients might impact to affect performance. Amongst these, free radical production and muscle fatigue might be altered by an antioxidant (e.g., quercetin), now available in a sport drink. FRS (Free Radical Scavenger) is a sport drink containing quercetin, several other antioxidants, and caffeine. Claims for FRS are that it can improve athletic performance by maintaining availability of catecholamines during exercise by inhibiting catechol-O-methyltransferase (COMT), sparing vitamin C, and performing antioxidant functions, which may reduce oxidative damage and muscle fatigue.

Metabolic stresses during high intensity endurance exercise and mechanical stresses during repeated lengthening (eccentric) muscle contractions result in generation of highly reactive oxygen species (ROS) that contain unpaired electrons (Kendall and Eston, 2002; McBride and Kraemer, 1999). ROS can accumulate in the working muscle inhibiting force production and contributing to acute muscle fatigue (Reid, 2001). ROS may reduce exercise performance by causing oxidative damage to adenosine triphosphate (ATPase) pumps, by significantly reducing calcium uptake by the sarcoplasmic retinaculum and by interfering with muscle excitation-contraction coupling and thereby reducing muscle contractility (Kai, Zweier, and Becker, 1997; Lawler, Hu, and Barnes, 1998; Sen, 1995). Furthermore, muscle contractile proteins (fast and slow myosin heavy chains) and mitochondrial enzymes required for energy provision (e.g.,

succinate dehydrogenase, cytochrome oxidase) appear to be susceptible to oxidative damage (Haycock, Peter, Harris, and Mantale, 1996).

Oxidative stress that occurs as a result of increased ROS production can be avoided if ROS are quickly scavenged by either endogenous or exogenous antioxidants. Quercetin is an exogenous antioxidant contained in the FRS sport drink that may help to attenuate oxidative damage caused by ROS during exercise and recovery days. Studies show that antioxidant supplements improve exercise performance (Antoni et al., 2007). However, quercetin has not been studied in this regard. Other studies reported quercetin may enhance selected types of exercise performance (Holden and Mefferd, 2006). However, these findings cannot be generalized to other types of exercises. The present study will examine the potential of FRS (with quercetin) to attenuate fatigue and to improve exercise performance in rowers.

Statement of Purpose

The purpose of the study was to determine if chronic and/or acute administration of FRS improves rowing performance in college-aged members of Division-III varsity crew team.

Hypotheses

1) Chronic FRS supplementation will improve performance as measured by 500 and 2000 m rowing time trials and tests of power and strength (i.e., vertical jump and 1 RM bench press).

2) Acute FRS supplementation will improve performance as measured by 500 and 2000 m rowing time trials and tests of power and strength (i.e., vertical jump and 1 RM bench press).

Assumptions

The basic assumptions of the study include the following:

1) The exercise protocol will be sufficient to cause generation of ROS and cause ROS-related mechanisms of muscle fatigue to be activated.

2) FRS will have antioxidant effects in working muscles that potentially affect performance.

3) Three weeks supplementation of FRS will load enough antioxidant, allowing the scavenging of ROS produced during high intensity exercise.

4) Participants will put forth a full effort during all testing procedures.

Delimitations

The delimitations of the study include:

1) College-aged, well trained, male and female rowers were used as subjects.

2) Rowing performance was measured using 500 m and 2000 m rowing ergometer tests.

3) Vertical jump and 1 RM bench press were used as measures of power and strength.

4) FRS (340ml) containing quercetin (325mg) was consumed twice daily for three weeks with no other dietary restrictions imposed.

5) Chronic effects of FRS on rowing performance were examined on two testing sessions.

6) Acute effects of FRS on rowing performance were examined on one testing session.

Limitations

The limitations of the study include:

1) The result may only be generalizable to well-trained, college-aged athletes but may not be applicable to sedentary individuals or those who are older or younger.

2) These results may only be generalizable to middle distance indoor rowing performance and may not apply to on water or other types of exercise performance.

3) The effects of individual diet, whether antioxidant rich or poor, was not controlled and may affect the results.

Definition of Terms

Free radicals: Free radicals are atomic or molecular species with unpaired electrons on an otherwise open shell configuration. Free radicals are produced when the body processes oxygen in the mitochondria, generating reactive oxygen species (ROS) (Fielding and Meydani, 1997).

Antioxidant (free radical scavengers): Is a molecule capable of slowing or preventing the oxidation of other molecules. Oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent. Oxidation reactions can produce free radicals, which start chain reactions that damage cells. Antioxidants terminate these chain reactions by removing free radical

intermediates, and inhibit other oxidation reactions by being oxidized themselves (Maxwell, 1995).

Catecholamine: Catecholamines are chemical compounds, derived from the amino acid tyrosine containing catechol and amine groups. Some of them are biogenic amines. Catecholamines are water-soluble and are 50% bound to plasma proteins, so they circulate in the bloodstream. Catecholamines are well-known to play a major role in these adaptive processes. Indeed, in response to acute physical stress, adrenaline (A) and noradrenalin (NA) increase cardiac output, stimulate ventilation, and contribute to substrate mobilization and utilization (Galbo, Kjaer, and Secher, 1987). Plasma concentrations of A and NA are known to increase markedly during exercise, especially when the intensity is really high, such as during sprint exercise (Zouhal et al., 1998).

Fatigue: Impairment in performance that leads to both the inability to maintain a certain level of force and an increased perception of task difficulty (Enoka and Stuart, 1992).

One-repetition maximum (1 RM): The maximum force one can apply throughout a single contraction. This experiment will use 1 RM bench press to measure maximum voluntary contraction (MVC) force (Swensen, Mancuso, and Howley, 1993).

Chapter 2

Review of Literature

When checking the effects of dietary supplements containing antioxidants on exercise performance and muscle fatigue, it is important to review previous related research. A number of studies have examined the ability of dietary supplement to combat oxidative muscle damage, but few studies have examined if they improve exercise performance. Fewer studies have examined if flavanoids can improve exercise performance, and generalization of these studies to all athletic populations is not clearly evidenced. Therefore, this chapter reviews, 1) Reactive Oxygen Species (ROS), 2) Generation of ROS and Exercise, 3) ROS and Muscle Fatigue, 4) Overview of Antioxidants, Vitamin C, and Vitamin E, 5) Flavanoids-quercetin, and 6) Antioxidants and Exercise Performance.

Reactive Oxygen Species (ROS)

ROS are molecules that possess an unpaired electron. This characteristic gives them a short half-life as they are always searching to combine with another molecule to achieve a stable configuration. Superoxide, hydroxyl, alkoxyl, and peroxyl radical groups are key radicals in biological systems (Cooper, Vallard, Choueiri, and Wilson, 2002; McBride and Kraemer, 1999; Sen, 1995). Although hydrogen peroxide is not a ROS, it is generated by the superoxide radical. Hydrogen peroxide reacts with transition metals to form the hydroxyl radical. Hydroxyl is one of the most highly reactive and destructive radicals of the ROS family (McBride and Kraemer, 1999; Packer, 1997). It will react with a variety of molecules that can potentially damage lipids, proteins, or nucleic acids (Packer, 1997).

ROS generation is also associated with oxidation of lipoproteins (McKay and Blumberg, 2002). Of particular interest to this study are the effects of ROS production during exercise on muscle membranes, proteins, ATPase pumps, and the integrity of the muscle cell following exercise.

Generation of ROS and Exercise

Exercise is thought to generate ROS by three main pathways, which are reviewed below: ischemia-reperfusion, mitochondrial production, and the inflammatory response (McBride and Kraemer, 1999; Packer, 1997).

Ischemia-Reperfusion: During intense exercise, blood flow is diverted from many organs (e.g., kidney and splanchnic regions) and non-working muscles. After cessation of exercise, normal blood flow is restored (Cooper et al., 2002; McBride and Kraemer, 1999; Packer, 1997). During reoxygenation of these organs and non-working muscles, xanthine oxidase activity increases, producing superoxide radicals (Cooper et al., 2002; Packer, 1997).

Mitochondrial Production: Another proposed exercise-induced mechanism for generating ROS is mitochondrial metabolism (Cooper et al., 2002; McBride and Kraemer, 1999; Packer, 1997). In this mechanism, electrons are leaked due to uncoupling at complexes I and III in the electron transport chain (ETC) (Leeuwenburgh and Heinecke, 2001; McBride and Kraemer, 1999). The mitochondrial uncoupling proteins (i.e., UCP-2 and UCP-3) are also involved in mitochondrial flux, and their expression can be affected by certain diseases and disorders like diabetes, and ultimately responsible for mitochondrial ROS leakage. In healthy people under non-stressful (low intensity) physiological conditions, UCP-2 and UCP-3 inhibit the release of ROS from the mitochondria (Orezechowski, 2003). However, mitochondrial uncoupling increases with increasing exercise intensity.

Inflammatory Response: The inflammatory response has also been implicated in ROS production. This response is intentional and is part of the immune system response to injury (McBride and Kraemer, 1999). Neutrophils are inflammatory cells (macrophages and phagocytes) that migrate to the injured site, phagocytize damaged tissue or foreign material, and release (activate) proteolytic enzymes. However, they also release ROS via oxidative bursts. This release of ROS results in uncontrolled tissue damage (McBride and Kraemer, 1999). Oxidative bursts are likely secondary responses during recovery from intense anaerobic, moderate to intense aerobic

or eccentric exercise (MacIntyre, Sorichter, Mair, Berg and Mckenzie, 2001; McBride and Kraemer, 1999; Pyne, 1994). The inflammatory response has also been strongly linked to muscle soreness (Clarkson, 1997).

ROS and Muscle Fatigue

Muscle fatigue has been associated with disturbances in Na^{+}-K^{+} balance, changes in intracellular pH, accumulation of inorganic phosphate, impaired energy metabolism, accumulation of free radical species or ROS, and impaired intracellular Ca^{2+} handling and sensitivity (Fitts, 1994). Accumulation of these reactive oxygen species has been suggested to be one of the main causes of fatigue during exercise (Reid, 2001). ROS may reduce exercise performance by causing oxidative damage to ATPase pumps, which significantly reduces calcium uptake by the sarcoplasmic retinaculum, thereby interfering with the excitation-contraction coupling process (Kai, Zweier, and Becker, 1997). Furthermore, muscle contractile proteins (fast and slow myosin heavy chains), and mitochondrial enzymes required for energy provision (succinate dehydrogenase, cytochrome oxidase) appear to be susceptible to oxidative damage (Haycock, Peter, Harris, and Mantale, 1996). ROS may also affect the ability to develop the action potentials required for muscle contraction because damaged ATPase pumps can't return potassium back into the skeletal muscle cells, thereby decreasing excitability, which can lead to fatigue during exercise and ultimately impair performance (Lawler, Hu, and Barnes, 1998; Sen, 1995).

Overview of Antioxidants

Antioxidants are molecules capable of slowing or preventing the oxidation of other molecules. Oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent. Oxidation reactions can produce free radicals, which start chain reactions that damage cells. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions by being oxidized themselves (Maxwell, 1995).

Dietary antioxidants may be able to detoxify the free radicals produced during exercise, which could otherwise result in damaging radicals (Dekkers, Van Doornen, and Kemper, 1996). These antioxidants are not generated by the body, and therefore must be constantly replenished through diet. Other antioxidants that are manufactured by the body can be bolstered through dietary supplementation of core antioxidants (Vasankari, Kujala, Rusko, Sarna, and Ahotupa, 1997). These endogenous antioxidants include lipoic acid, glutathione, bilirubin, urate, albumin, ceruloplasmin, as well as the antioxidant enzymes superoxide dismutase, glutathione peroxidase, and catalase (Alessio and Blasi, 1997; Clarkson, 1995; Dekkers et al., 1996; Powers and Lennon 1999).

When exercising, the body processes up to 30 times more oxygen due to increased metabolism (Alessio and Blasi, 1997; Appel et al., 1997; Clarkson, 1995; Feilding and Meydani, 1997). Although exercise training increases the production of the principal antioxidant enzymes, exercise seems to pertube the fine balance of the body's defense system, rendering the tissue more susceptible to damage (Alesio, Goldfarb, and Cao, 1997; Clarkson, 1995; Dekkers et al., 1996; Hong and Johnson, 1995; Ortenblad, Madsen, and Djurhuus, 1997; Sen, 1995). Some authors believe it is logical to expect that athletes could benefit from obtaining more dietary antioxidants in the diet to scavenge free radicals produced during exercise (Clarkson, 1995; Hong and Johnson, 1995; Ji, 1998).

Vitamin C

Vitamin C is a water soluble vitamin. The role of vitamin C as an antioxidant is twofold; it directly scavenges superoxide, hydroxyl, and lipid hydroxide radicals, and it helps recycle vitamin E radicals by returning them to their reduced state (Packer, 1997). In the latter process, reduced vitamin C is converted to vitamin C radical. Recycling of vitamin C radical can be achieved by NADH semiascorbyl reductase or cellular thiols such as glutathione and dihydrolipoic acid (Sevanian, Davies, and Hochstein, 1985). Vitamin C is probably the most important antioxidant in extracellular fluid (Dekkers et al., and Peter, 1997). At the biochemical

level, the protective action of vitamin C may relate to its ability to neutralize ROS, which are produced by neutrophilic leukocytes during exercise induced phagocytosis (Peters, 1997). The concentration of vitamin C within neutrophils is approximately 50 times the extracellular concentrations indicating that large amounts are available to protect the neutrophil against the autotoxic activity of its own ROS (Peters, 1997). Thus vitamin C allows the neutrophil to do its primary role of phagocytosis, which theoretically should lead to a decreased incident of cellular damage. Inside cells, vitamin C reinforces the antioxidant effect of vitamin E by regenerating the active form of the fat-soluble vitamin after it has reacted with a free radical (Alesio et al., 1997; Dekkers et al., 1996; Rokitzki et al., 1994). In other words, vitamin C has a "sparing effect" on vitamin E, as well as on other endogenous antioxidants. It has been postulated that vitamin C supplementation, in combination with vitamin E supplementation, may be the most effective method for stopping the chain of free radicals produced in cells during exercise (Antoni et al., 2007; Goldfarb, 1993; Peters, 1997). As well, various forms of stress can rapidly deplete vitamin C, therefore, athletes with reduced vitamin C status might benefit from vitamin C supplement. Vitamin C reportedly did not decrease markers of oxidative damage or improve recovery from unaccustomed exercise unless administered for at least 2 weeks prior to exercise stress, which then resulted in modest improvements in muscle soreness (Thompson et al., 2001, Thompson, Maynard, Morales, and Scordilis, 2003). Other well controlled studies showed that vitamin C did not affect exercise performance (Ashton et al., 1999; Clarkson, 1995).

Vitamin E

Vitamin E is a fat soluble vitamin made up of tocopherols and is thought to be one of the most important antioxidants because of its association with cell membranes (Tiidus, Pushkarenko, and Houston, 1996). Among these α-tocopherol is the best known and possesses the most potent antioxidant activity (Burton and Ingold, 1989, Janero, 1991). Because of its high lipid solubility, vitamin E is associated with lipid-rich structures such as mitochondria, sarcoplasmic retinaculum, and the plasma membrane. It protects against oxidative damage by acting directly with a variety

of oxygen radicals including singlet oxygen, lipid peroxide products, and the superoxide radical, to form a relatively innocuous tocopherol radical (Bieri, 1990).

Vitamin E is important in energy production as it ensures that glucose is fully oxidized and adenosine triphosphate (ATP) is generated most efficiently (Rokitzki et al., 1994). Because vitamin E is a fat soluble vitamin, it has been suggested that athletes on a low fat diet may benefit from the use of supplements (Loft and Poulsen, 1997). Endurance training, in addition to increasing the oxidative capacity of muscles, improves the body's enzymatic antioxidant defense (Alesio and Goldfarb, 1997; Brites et al., 1999, Dekkers et al., 1996; Jacob and Burri, 1996; Margaritis, Tessier, Richard, and Marconnet, 1997). However, muscle and other tissue consume vitamin E during increased physical activity (Appel et al., 1997; Avellini, Chiaradia, and Gaiti, 1999). Therefore, many researchers suggest that vitamin E supplementation may be warranted to prevent free-radical damage induced by exercise (Dekkers et al., 1996; Oostenbrug et al., 1997; Rokitzki, 1994). Antoni et al. (2007) demonstrated that long term antioxidant diet supplements (e.g.,Vitamin E, C, and β-carotene) often alternate the blood lactate concentration and production of ROS during exhaustive exercise, while improving the efficiency of aerobic metabolism.

Similarly exercise-induced oxidative stress has been demonstrated in resistant-training athletes. McBride et al. (1998) divided 12 recreationally weight-trained men into two groups: one consumed 1200 IU vitamin E each day for two week, whereas the other group consumed cellulose-based placebo pills for two weeks. Vitamin E supplementation elicited a significant decrease in creatine kinase (CK) activity at 24 and 48 hrs post-exercise compared with the placebo group. These activities suggested that vitamin E supplementation may decrease muscle membrane disruption in addition to decreasing the level of ROS that occurs during exercise. Hence, it may be beneficial for both endurance-trained and strength-trained athletes to supplement their diets with vitamin E to prevent excess free-radical oxidative stress.

Flavonoids-Quercetin

Flavonoids are a large family of diphenylpropanes (over 4000 members have been identified) that are commonly found in plants consumed by humans. Family members include, but are not limited to; flavones, isoflavones, flavanones, anthocynins, and catechins (Das, 1994). Flavonoids have been reported to possess a wide variety of biological activities ranging from inhibition of inflammatory enzymes (e.g., lipoxygenase, cyclooxygenase, xanthine oxidase, NADH-oxidase, phospholipase, anti-tumoral, anti-viral, anti-mutagen, anti-inflammatory, anti-ischemic, and anti-allergic activities (Cao, Sofic and Prior, 1997). Many of these biological effects are thought to be a result of the antioxidant capacity of flavanoids (Bors, Michel and Saran, 1994; Saija et al., 1995; Scalbert, Morand, Manach, and Remesy, 2002). Radical scavenging activities of flavonoids appear to vary greatly among family members, but include quenching of peroxyl, hydroxyl and superoxide radicals, as well as hydrogen peroxide and a variety of chemically generated radicals not naturally found in the body (Cao et al., 1997). Among the many flavonoids under investigation for their potential role in protecting against radical-mediated processes, is the polyphenolic flavonoid family, which includes quercetin. Quercetin is found in significant concentration in apple, tea, onion, nuts, berries, cauliflower and cabbage.

Quercetin has many health promoting effects, including improving cardiovascular health and reducing risk for cancer and inflammatory effects cancers (Ferrandina et al., 1993; Mayo Clinic News 2001; Sabitha and Shyamaladevi, 1999; Scambia et al., 1991; Stefani et al., 1999). All these effects are thought to be caused by the strong antioxidant action of quercetin, which helps to combat free radicals molecules that can damage cells. The anti-inflammatory action of quercetin is caused by the inhibition of enzymes, such as lipoxygenase, and the inhibition of inflammatory mediators (Sato, Miyazaki, Kambe, Maeda, and Seo, 1997). Quercetin also inhibits the release of histamine from basophils and mast cells, which limits congestion (Pearce, Befus, and Bienenstock, 1984). Studies have shown that quercetin reduces the risk of prostate, ovary,

oral, and upper digestive tract cancers (Mayo Clinic News 2001; Ferrandina et al., 1993; Scambia et al., 1991; Sabitha and Shyamaladevi, 1999; Stefani et al., 1999). Quercetin also seems to reduce the production of uric acid by inhibiting the xanthine oxidase, thereby easing gout symptom (Sato et al., 1997). Studies have shown an improved lung function and lower risk of certain respiratory diseases (asthma and bronchitis) for people with high apple (rich in quercetin) intake (Tabak, Arts, Smit, Heederik, and Kromhout, 2001; Shaheen et al., 2001). Quercetin can also potentially improve athletic performance by free radical scavenging, maintaining availability of catecholamines during exercise, sparing vitamin C, and performing antioxidant functions that can help to avoid oxidative damage and muscle fatigue. Holden and Mefferd (2006) shared that a quercetin containing dietary drink improved high-intensity cycling time-trial performance by increasing power output.

Antioxidants 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 muscle damage by ROS and hence impaired exercise performance. Beek et al. (1990) have shown that a vitamin C deficiency impairs exercise performance in marginally deficient humans. It is very well understood that strenuous exercise may generate ROS to a level to overwhelm tissue antioxidant defense systems (Sen, 2001). Preventing muscle tissue damage during exercise training may help to optimize the training effect and eventual competitive sports performance. Results from studies examining the effects of vitamins and other antioxidants on oxidative stress have been conflicting (Goldfarb, Bloomer, and McKenzie, 2005; Mastaloudis, Traber, Carstensen, and Widrick, 2006; Packer, 1997; Rokitzki et al., 1994) supplemented endurance trained runners with 300 mg of *RRR*-α-tocopherol acetate and 1000 mg of ascorbic acid, or a placebo, twice daily for six weeks. After the six weeks, the runners participated in an ultra-marathon run. Subjects performed a maximum voluntary contraction (MVC) prior to supplementation, one day pre-race, two hours post-race, and six days post-race.

Blood samples were also taken at 12 different times (prior to supplementation, one day pre-race, one hour pre-race, mid-race, immediately post-race, two hours post-race, and six days post-race). Plasma α-tocopherol was increased in the supplemented group and remained unchanged in the control group. Ascorbic acid levels followed a similar trend. Increases in CK were significant from baseline, but not between the treatments. Post-race maximum voluntary contraction results were lower compared to baseline and no differences among treatments were found. In this case, supplementation with vitamins C and E did not attenuate the loss of muscle force or the increase in CK associated with ultra-marathon running. The authors suggested that a larger dosage of vitamin E may be needed to prevent the muscle damage found in this protocol.

Nieman et al. (2001) supplemented experienced ultra-marathon runners with either a placebo or 1500 mg of vitamin C for seven days prior to an 80 kilometer (km) run. During the race, runners were given coded bottles of carbohydrate beverages (150 $mg \cdot l^{-1}$ with or without vitamin C). Saliva and blood samples were taken pre race, mid-race (32 km), and five minutes post-race. Isoprostanes (IsoP) were measured via mass spectrometry and lipid hydroperoxides were measured spectrophotometrically. Neutrophils and monocytes were determined using Enzyme-linked immunosorbent assay kits. They rose in both groups, but failed to reach significance. The level of serum vitamin C was significantly higher in the supplemented group. These data indicate that 1500 mg of vitamin C over seven days does not protect endurance athletes from oxidative stress. This lack of statistical significance could also be due to the timing of post-race sampling or perhaps vitamins C and E working synergistically. The study only asked participants to avoid foods with large amounts of vitamin C and to follow a high carbohydrate diet. Participants were also allowed to continue vitamin supplements as long as they did not provide more than 100% of the recommended daily values. Plasma vitamin E levels were not measured (Nieman et al., 2001).

In contrast to the previous studies, some data show decrease in oxidative damage but there is no effect on exercise performance. Dekkers et al. (1996) concluded that dietary

supplementation with antioxidant vitamins has favorable effects on lipid peroxidation and exercise-induced muscle damage. They recommended vitamin supplementation to individuals performing regular heavy exercise. Evans (2000) noted that several antioxidants, including vitamin C and especially vitamin E decreased exercise-induced oxidative damage, which could help prevent muscle tissue damage. Takanami (2000) found that vitamin E contributes to preventing exercise-induced lipid peroxidation and possible muscle tissue damage. They recommended that athletes supplement with 100-200 milligrams of vitamin E daily to help prevent exercise induced oxidative damage. Ji (1999) indicated that the delicate balance between pro-oxidants and antioxidants suggests that supplementation of antioxidants may be desirable for physically active individuals under certain physiological conditions by providing a larger protective margin. In particular, he noted that the aging process lessens the exercise training-induced improvement in natural antioxidant enzymes and suggests exercise training in older athletes might be assisted with antioxidant supplementation in attempts to optimize antioxidant defense. Sacheck and Blumberg (2001) concluded that the use of dietary antioxidants like vitamin E to reduce exercise-induced muscle injury have met with mixed success, which seems to be the prevailing viewpoint. All reviewers indicated more research is needed to address this issue and to provide guidelines for athletes.

Summary

Literature in the study of antioxidants and exercise is inconsistent. Several possibilities, mainly related to methodology, exist for these inconsistencies. Studies evaluating vitamins E and C used different amounts, preparations and supplementation periods lasted anywhere from seven days to five months. The modes of exercise were running, cycling, or resistance exercise while some studies used a control group and some did not. Many of the variables recorded were measured with different assays and at different time periods. Finally, subjects ranged from non-resistance trained females to elite male cyclists.

The results from Tsai et al. (2005) and other previously mentioned studies, suggested that oxidative damage due to ROS may be attenuated by antioxidant supplementation. ROS produced during exercise can accumulate in the working muscle, inhibiting force production and contributing to acute muscle fatigue (Reid, 2001). ROS may reduce exercise performance by causing oxidative damage to adenosine triphosphate (ATPase) pumps, by significantly reducing calcium uptake by the sarcoplasmic retinaculum, by interfering with muscle excitation-contraction coupling and reducing muscle contraction (Kai et al., 1997). Quercetin is a flavonoid that may be a more effective and practical dietary antioxidant than vitamin C and E for enhancing exercise performance.

Chapter 3

Methods

This chapter describes in detail the methodology of the study. Three major sections are included: (1) Subject Selection and Characteristics, (2) Study Design and Procedures, and (3) Statistical Analysis.

Subject Selection and Characteristics

A total of 46 male and female collegiate rowers between the ages of 18 to 25 years served as subjects. Participants were recruited from the Ithaca College rowing team, through distribution of flyers (Appendix A) and a recruitment statement (Appendix B). At a preliminary group meeting, the project was explained to the subjects and their questions were answered. Those who wished to participate signed an informed consent (Appendix C). The Ithaca College's Human Subject Review Board approved the project. After informed consent was obtained, subjects filled out an exercise history (Appendix D) and health history questionnaire (Appendix E).

Study Design and Procedures

The primary purpose of this study was to test the effects of FRS on exercise performance measures in well-trained rowers. The study had two parts and total of five testing sessions. Part 1 measured the effect of chronic use of FRS on rowing performance, whereas Part 2 measured the acute effect of FRS on rowing performance. Subjects were instructed to maintain their usual training, racing, and dietary program throughout the course of the project and also were asked to refrain from any intense exercise 24 h prior to testing. A brief description of their previous day's training and dietary habits were recorded before each testing session to ensure subject compliance to pretest instructions.

Part 1: Chronic Effects of FRS

Testing session 1(Pre supplement): Day 1: All 60 subjects reported to the laboratory in the morning. Upon entry to the laboratory, anthropometric data such as height, weight, and body fat were measured. Each subject's body fat was estimated with a skinfold caliper according to the

methods of Pollock, Schmidt and Jackson (1980). In short, seven sites were measured twice. If measures at any site were 5% apart, a third measure was made. The mean of the two values in best agreement at the site was recorded as a site's skin-fold thickness. The means from each site were then summed and used to calculate body density, which was subsequently placed in Siri's equation for percent fat determination (Siri, 1961). All subjects completed a 10 to 15 min warm-up designed by the head coach of the Ithaca College crew team. Warm-up included 5-7 min of rowing on a Concept II rowing ergometer (Digital Rowing Inc., Boston, USA) at a self-selected intensity, followed by five bursts of 10 repetitions of rowing strokes. After the warm-up, each subject completed 500 m rowing ergometer test on the ergometer followed by 2-3 min of active recovery at a low intensity. Approximately 30 min later, subjects completed the 2000 m rowing ergometer test, which was preceded by 5 min of warm-up at a self-selected pace and completed with a 2-3 min active recovery period. The following data were recorded during and at the completion of each ergometer test: HRmax, peak watts, average watts, and time.

Testing session 2 (Pre supplement): Day 2: Twenty-four hours after the first testing session, subjects again reported to the lab so that their maximal vertical jump power and 1 RM for the bench press could be measured. Following 10-15 min of warm up, which included jogging on a treadmill, and arm ergometer cycling, the subjects then completed the vertical jump test. This test included three countermovement jumps with arm swing (Harman, Rosenstein, Frykman, and Rosenstein, 1990). Each jump was recorded to 0.1 cm and was separated by a rest period of 10 s. Jump height was measured with a portable hand-held computer unit connected to a contact timing mat (Newtest Oy, Finland). This technique has a validity and reliability similar to a force plate (Cronin, McNair, and Marshall, 2001). The height jump was recorded and this score was subsequently transformed into measures of peak and average power (W) using Harman's formula (Harman, Rosenstein, Frykman, Rosenstein, and Kramer, 1991). Fifteen minutes after the vertical jump test, the subjects then completed the 1 RM bench press test on a Cybex resistance training machine (Cybex international Inc., MA, USA), in accordance with the procedures of Swensen, Mancuso, and Howley (1993). In short, the test began with five repetitions with a light load

(~ 50% of estimated 1 RM). Approximately 2 min later, single repetitions were executed, starting with an initial load that was estimated as the subject's 1 RM as based on previous training. If the lift was completed, load was increased by 2.5 kg until the 1 RM was obtained. Each lift was separated by 2 min of rest. These 1 RM procedures have been shown to have test-retest reliability between 0.93 and 0.97 (Berger, 1970; O'Byrant, Byrd, and Stone, 1988).

After completion of both day one and two testing sessions, subjects were pair-matched into the treatment or placebo groups based on their 2000 m ergometer time recorded in testing session 1. Participants were required to consume the liquid FRS or placebo supplement, twice a day with meals for three weeks, with one drink in the morning and one in the evening. FRS and placebo drinks were the same in color and flavor and not distinguishable.

Testing session 3 (Post supplement): Day 1: After three weeks of intervention, subjects reported back to the laboratory where weight and body fat were measured as previously described. Subjects then completed 500 m rowing ergometer and 2000 m rowing ergometer test as described previously.

Testing session 4 (Post supplement): Day 2: After 24 hours, the subjects reported to the laboratory for the measurement of vertical jump test and 1 RM bench press test. Tests were completed and data were recorded as previously described.

Part 2: Acute Effects of FRS

Seventy-two hours after completion of test session four, the subjects in the placebo group were matched, based on 2000 m rowing ergometer performance, and divided into two groups, Group A and Group B. Both groups consumed their respective drinks twice on the day of testing with the first drink 150 min before and second drink just before the testing.

Testing session 5: The subjects reported to the laboratory for the measurement of 500 m and 2000 m ergometer tests. All the subjects went through the same testing protocols as previously described in testing sessions one and two.

Statistical Analysis

A double-blind, matched-pair protocol was used to assess the chronic effect of FRS on rowing performance. Consequently, the following dependent variables were analyzed with a 2 × 2 repeated measures ANOVA: 500 m rowing time, 2000 m rowing time, vertical jump test and 1 RM bench press. A double-blind, matched-pair protocol was also used to assess the acute effect of FRS on rowing performance. In addition to a 2 x 2 repeated measures ANOVA, post-hoc independent and dependent sample t-tests were employed to analyze 500 m and 2000 m rowing times. All the analyses were completed with SPSS v. 16.0; alpha (α) was set at level of 0.05.

Chapter 4

Results

The primary purpose of this study was to determine the effects of acute and/or chronic supplementation of a quercetin containing drink (FRS) on performance in rowers. Fifty subjects (26 males and 24 females) from a collegiate varsity rowing team volunteered for the study and were analyzed on these performance outcomes: 500 m row, 2000 m row, 1 RM bench press, and vertical jump test. Baseline data (Appendix F) were obtained for these variables and subjects were pair-matched on 2000 m rowing and then placed into supplement or placebo group. Following data collection, the results were analyzed and presented here in the following sections: 1) Chronic Supplementation of FRS, 2) Acute Supplementation of FRS and 3) Summary.

Chronic Supplementation of FRS

Three weeks of chronic loading was done for the supplement and placebo groups. The supplement group was provided with "real" FRS supplementation, whereas the placebo group was provided "sham" FRS supplementation for a period of three weeks. After three weeks: 1) 500 m row, 2) 2000 m row, 3) 1 RM bench press, and 4) Vertical jump test variables were re-measured.

500 m Row: A 2 x 2 (Group x Time) ANOVA with repeated measure on time was used to look for differences in 500 m row time-trial performance between groups. Table 1 shows no significant Group x Time interaction ($p = 0.464$). Both placebo and supplement groups increased 500 m rowing performance across time as evidenced by a significant time main effect ($p = 0.006$). This was likely the result of continued physical training by both groups. However, neither group had greater 500 m times as there was no difference between groups as demonstrated by non- significant group main effect ($p = 0.100$). These data shows that chronic FRS Loading did not improve 500 m time-trial performance. The descriptive data for the 500 m row time-trial can be seen in Table 2.

Table1. 500 m Row Time-Trial: ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	0.013	1	0.013	2.947	0.100
Error (Group)	0.098	22	0.004		
Time	0.004	1	0.004	9.071	0.006*
Error (Time)	0.011	22	0.000		
Group x Time	0.000	1	0.000	0.556	0.464
Error (Group x Time)	0.008	22	0.000		

Note: * $p < 0.05$

Table 2. 500 m Row Time-Trial Descriptive Data Table

	FRS		Placebo	
	Pre	Post	Pre	Post
Mean	1.387	1.380	1.376	1.367
SD	0.092	0.086	0.098	0.088

Note: Data are minutes.

2000 m Row: A 2 x 2 (Group x Time) ANOVA with repeated measures on time, was employed to inspect differences in 2000 m time-trial performance between supplement and placebo groups. Table 3 shows a significant Group x Time interaction ($p = 0.009$). Table 4 displays independent sample t-test comparing the groups' baseline 2000 m rowing times; there was no difference at baseline ($p = 0.932$). Similarly, there was no difference in 2000 m rowing time after supplementation ($p = 0.176$). Table 5 displays a significant time effect ($p = 0.035$) in 2000 m rowing time across time in the placebo group, but not for the supplemental group ($p = 0.079$). Thus, FRS supplementation does not improve 2000 m rowing performance across time. Tables 4 and 5 show the descriptive data for the 2000 m row.

1 RM Bench Press: A 2 x 2 (Group x Time) ANOVA with repeated measure on time was employed to inspect for differences in 1 RM bench press performance between the groups. Table 6 shows no significant Group x Time interaction ($p = 0.171$). There also was no significant time main effect found ($p = 0.990$); hence, the groups collectively did not improve across time. Neither group was different than the other as demonstrated by the non-significant group main effect ($p = 0.683$). Thus, chronic loading with FRS supplementation does not improve 1 RM bench press performance. Table 7 shows the descriptive data for 1 RM bench press.

Vertical Jump: A 2 x 2 (Group x Time) ANOVA with repeated measure on time was employed to inspect for differences in vertical jump performance between the groups. Table 8 shows no significant Group x Time interaction ($p = 0.689$). Both groups increased vertical jump performance significantly across time as evidenced by significant time main effect ($p = 0.013$), but here was no group main effect ($p = 0.799$). These data show that chronic FRS supplementation does not improve vertical jump performance. Table 9 shows the descriptive data for the vertical jump.

Table 3. 2000 m Row Time-Trial ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	0.235	1	0.235	4.158	0.054
Error (Group)	1.244	22	0.057		
Time	0.154	1	0.154	2.771	0.110
Error (Time)	1.227	22	0.056		
Group x Time	0.311	1	0.311	8.189	0.009*
Error (Group x Time)	0.008	22	0.000		

Note: * $p < 0.05$

Table 4. 2000 m Row Time-Trial Post-hoc Independent Sample T-tests Table

	Group	M	SD	T	DF	*p*
BL	T	7.353	0.603	-0.086	44	0.932
	P	7.368	0.591			
PT	T	7.387	0.577	1.375	44	0.176
	P	7.260	0.493			

Note: Data are minutes; BL: Baseline; PT: Post-treatment; T: Treatment Group; P: Placebo Group

Table 5. 2000 m Row Time-Trial Post-hoc Dependent Sample T-test Table

	Group	M	SD	T	DF	*p*
T	BL	7.353	0.602	-1.839	22	0.079
	PT	7.387	0.577			
P	BL	7.368	0.591	2.244	22	0.035*
	PT	7.260	0.493			

Note: *$p < 0.05$; Data are minutes; T: Treatment Group; P: Placebo Group; BL: Baseline; PT: Post-treatment

Table 6. 1 RM Bench Press ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	36.494	1	36.494	0.171	0.683
Error (Group)	4475.648	21	213.126		
Time	0.002	1	0.002	0.000	0.990
Error (Time)	302.381	21	14.399		
Group x Time	9.776	1	9.776	2.007	0.171
Error (Group x Time)	102.266	21	4.870		

Table 7. 1 RM Bench Press Descriptive Data Table

	FRS		Placebo	
	Pre	Post	Pre	Post
Mean	66.63	65.95	67.59	68.28
SD	16.65	18.55	16.82	18.19

Note: Data are kilograms

Table 8. Vertical Jump ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	7.102	1	7.102	0.081	0.799
Error (Group)	1849.148	21	88.055		
Time	51.011	1	51.011	7.376	0.013*
Error (Time)	145.239	21	6.916		
Group x Time	0.920	1	0.920	0.165	0.689
Error (Group x Time)	117.330	21	5.587		

Note: *P< 0.05

Table 9.Vertical Jump Descriptive Data Table

	FRS		Placebo	
	Pre	Post	Pre	Post
Mean	34.73	36.45	34.87	36.09
SD	8.30	7.45	10.45	9.71

Note: Data are centimeters

Acute Supplementation of FRS

This section presents analyses of dependent variables of rowing performance in supplement vs. placebo groups after acute FRS supplementation. Drinks were provided to both supplement and placebo groups as described in the chronic treatment but with supplement given twice on testing day. FRS was administered 150 min before testing and again immediately prior to testing. The performance variables measured in this acute FRS condition were: 1) 500 m Row and 2) 2000 m Row.

500 m Row: A 2 x 2 (Group x Time) ANOVA with repeated measure on time was used to inspect for differences between groups in 500 m time-trial performance. ANOVA summary (Table 10) shows a significant Group x Time interaction ($p = 0.045$). As shown in Table 11, an independent sample t-test at baseline did not reveal a significant difference between groups ($p = 0.955$) meaning the groups were similar before supplementation. The groups were also similar after supplementation ($p = 0.898$). Table 12 displays a dependent sample t-test comparison of baseline to post-supplementation 500 m row times for the placebo group and did not find a significant effect ($p = 0.138$). A similar comparison for the supplemental group also showed no significant improvement over time ($p = 0.468$). There is no evidence to conclude that acute loading with FRS supplementation improved 500 m time-trial performance.

2000 m Row: A 2 x 2 (Group x Time) ANOVA with repeated measure on time was employed to inspect for differences in 2000 m rowing ergometer performance between supplement and placebo groups. Table 13 shows no significant Group x Time interaction ($p = 0.154$). There was not any significant time main effect found ($p = 0.071$) meaning that the groups did not change from pre to post-supplementation. Finally, the acute groups were not different in 2000 m performance as no significant group main effect ($p = 0.953$) was obtained. There is no evidence to conclude that acute loading with FRS supplementation improved 2000 m time-trial performance. The descriptive data for 2000 m row time-trial can be seen in Table 14.

Summary

Three weeks of chronic FRS supplementation did not change the 500 m row, 2000 m row, 1 RM bench press, and vertical jump performances; the only significant change was seen in the placebo group for 2000 m row performance. Similarly, acute supplementation did not improve 500 m and 2000 m row performances. It can be concluded that acute and/or chronic supplementation of FRS does not have any effects on performance in rowers as tested.

Table 10. 500 m Row ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	6.25E-005	1	6.25E-005	0.010	0.922
Error (Group)	0.056	9	0.006		
Time	0.000	1	0.000	0.648	0.442
Error (Time)	0.002	9	0.000		
Group x Time	0.000	1	0.000	5.413	0.045*
Error (Group x Time)	0.001	9	7.81E-005		

Note: *p < 0.05

Table 11. 500 m Row Post-hoc Independent Sample T-test Table

	Group	Mean	SD	T	DF	*p*
BL	T	1.617	0.154	-0.058	18	0.955
	P	1.621	0.157			
PT	T	1.627	0.153	0.130	18	0.898
	P	1.618	0.156			

Note: Data are minutes; BL: Baseline; PT: Post-treatment; T: Treatment Group; P: Placebo Group

Table 12. 500 m Row Post-hoc Dependent Sample T-test Table

	Group	Mean	SD	T	DF	*p*
T	BL	1.617	0.154	-1.627	9	0.138
	PT	1.627	0.153			
P	BL	1.621	0.157	0.758	9	0.468
	PT	1.618	0.156			

Note: Data are minutes; BL: Baseline; PT: Post-treatment; T: Treatment Group; P: Placebo Group

Table 13. 2000 m Rowing ANOVA Summary Table

	SS	DF	MS	F	*p*
Group	0.000	1	0.000	0.004	0.953
Error (Group)	0.878	9	0.098		
Time	0.023	1	0.023	4.201	0.071
Error (Time)	0.049	9	0.005		
Group x Time	0.009	1	0.009	2.425	0.154
Error (Group x Time)	0.033	9	0.004		

Table 14. 2000 m Row Descriptive Data Table

	FRS		Placebo	
	Pre	Post	Pre	Post
Mean	7.31	7.23	7.29	7.27
SD	0.66	0.62	0.56	0.52

Note: Data are minutes

Chapter 5

Discussion

The purposes of this study were to determine the effects of the chronic and acute FRS supplementation on performance in rowers during an off-season training period. Subjects were pair-matched, based on their 2000 m rowing performance, and were randomly placed in either a placebo or supplemental group to understand the effects of FRS on both rowing performance and muscular power. This chapter includes discussion on the performance effects of: a) Chronic Supplementation of FRS and, b) Acute Supplementation of FRS; it concludes with a summary.

Chronic Supplementation of FRS

This present study found that three weeks of FRS supplementation did not improve performance in off-season workouts for collegiate male and female rowers, which does not support the contention that chronic FRS supplementation improves performance. Holden et al. (2006) previously examined a quercetin- containing compound in athletes performing cycling time trials. Their results demonstrated modest but significant improvements in performance after quercetin supplementation. Holden and Mefferd (2006) used a supplement like the present study containing 150 mg vitamin C, 300 mg of green tea, B vitamins, and quercetin. In contrast to the present study, Holden and Mefferd (2006) used six weeks of supplementation to obtain significant improvement in time trials, which suggests that length of supplementation may influence its ergogenicity. Apart from difference in duration of supplementation, the present study did not eliminate effects of other antioxidants. Thus, further investigation on how quercetin may act to enhance exercise performance is needed.

The period of FRS supplementation may be critical to performance enhancing effects and, in the present study, FRS was taken for three weeks. Thompson et al. (2001, 2003) found that at least two weeks of antioxidant supplementation is necessary for modest loading and to combat against ROS produced during exercise. Holden and Mefferd (2006) used six weeks of loading with antioxidants to obtain performance enhancing effects by presumably avoiding

muscle fatigue caused by ROS produced during exercise. In the present study, after three weeks of supplementation, there was some trend for improvement in performance but it failed to reach statistical significance. It is possible that the reason for not reaching statistical significance might be the duration of supplementation or the selectivity of the exercise performance. Holden and Mefferd (2006) investigated a 30 km time-trial cycling performance as a measure of high intensity variable and found significant improvement in performance.

Exercise-induced muscle damage occurs as a result of resistance exercise. Dekkers et al. (1996) concluded that dietary supplementation with antioxidant vitamins has favorable effects on lipid peroxidation and exercise-induced muscle damage. They recommended vitamin supplementation to individuals performing regular heavy exercise. In the current study, variables for measuring power were included as rowing is not only considered an endurance event but also considered a power sport. Bench press (1 RM) and vertical jump tests were administered to obtain measures of power but neither improved with FRS supplementation. Eccentric resistance exercise has been shown to increase markers of oxidative stress (Goldfarb et al., 2005) or may have no effects on oxidative stress (Lee et al. 2002). McBride et al. (1998) found two weeks of antioxidant (vitamin E) supplement reduces ROS levels during high intensity exercise. On the contrary, Mastaloudis et al. (2006) found no improvement in MVC after antioxidant supplementation was given for six weeks. Like Mastaloudis et al. (2006), the present study supports the notion that there is no significant improvement in power performance after chronic antioxidant supplementation.

Some previous work showed a decrease in oxidative damage with no effect on exercise performance. Evans (2000) noted that several antioxidants, including vitamin C and especially vitamin E, decrease the exercise-induced oxidative damage, and may thereby prevent muscle tissue damage. Takanami et al. (2000) concluded that vitamin E contributed to preventing exercise-induced lipid peroxidation and muscle tissue damage, and they recommended that athletes supplement with 100-200 milligrams of vitamin E. Sacheck and Blumberg (2001),

however, concluded that evidence for the use of dietary antioxidants like vitamin E to reduce exercise-induced muscle injury is equivocal and this seems to be the prevailing viewpoint. More research is needed to address the issue of exercise-induced tissue damage and supplementation to provide guidelines for athletes.

In addition to potentially enhancing performance, quercetin and quercetin-containing drinks have also been shown to enhance immune function. Nieman et al. (2007) showed that quercetin supplementation reduced the incidence of upper respiratory tract infection in the weeks after three days of intensive cardiovascular exercise. Pearce (1984) reported quercetin can inhibit histamine release and may prevent congestion. In this regard, the correct dosage and timing of quercetin supplementation is not clearly defined. Whether the antioxidant and anti-inflammatory properties of quercetin are mechanistically linked, perhaps through redox perturbation, is currently unknown. In the current study, 320 mg of quercetin and other antioxidants (Vitamin A, C, E and catchins) supplementation were given twice daily. Athletes filled out a subjective cold questionnaire before and after the supplementation period. We did not find any improvement in cold symptoms with FRS supplementation. In fact, one subject in the FRS supplementation group dropped out of the study due to severe congestion. These cold-related data are mainly qualitative and no statistical significance is implied.

Acute Supplementation of FRS

The present study also found that acute supplementation of FRS did not improve 500 and 2000 m rowing time trial performance. It was speculated that acute supplementation may enhance performance in two ways: first, FRS contains 48 mg of caffeine, a well documented ergogenic aid. Second, as previously described, FRS contains the anti-oxidant quercetin. Oxidative stress is a well-defined outcome of acute physical activity (Powers et al., 1999). In the current study, acute FRS supplementation was given 2.5 h before, and immediately before exercise. The plasma half life of quercetin is 2 to 28 h and its action peaks about 2 h after consumption (Manach and Donavan, 2004). Acute FRS supplementation, as used in this study, did not improve performance

suggesting that either 48 mg of caffeine, the antioxidant load, or some combination of both variables were insufficient to act as ergogenic aids.

Proven ergogenic doses of caffeine range from 250 to 500 mg, so it is not surprising that 48 mg of caffeine, as found in FRS, did not improve performance. The acute effects of quercetin on exercise have not been studied. Indeed, the acute effects of antioxidants on exercise performance are not well studied. In contrast, the acute effects of antioxidants on various physiological variables following exercise are well studied. Hartmann et al. (1995) demonstrated that short-term vitamin E supplementation (800 mg administered 12 and 2 h before and 22 h post-exercise) reduced DNA damage in peripheral leucocytes following exhaustive exercise. Others have reported attenuated oxidative-stress when antioxidants are given before exercise (Goldfarb, 1999; Goldfarb et al., 2005). In contrast, several studies did not demonstrate a reduction in markers of oxidative stress when antioxidants are given (Davison and Gleeson, 2006; Davison, Gleeson, and Phillips, 2007).

Antioxidant supplementation, during or after exercise, may have little impact or actually enhance the oxidative-stress response (McAnulty et al., 2003; Thompson et al., 2003). High-dose vitamin C taken during an endurance run was reported to exacerbate the oxidative-stress response to lipid peroxidation (Nieman et al., 2002). Goldfarb et al. (2005) reported that the recovery of oxidative-stress markers to baseline might also be affected by exercise type, intensity, duration, training status, and diet. Antioxidant supplementation given for some time before exercise attenuated the interleuken-6 (IL-6) response (Fischer et al., 2004; Phillips, Childs, Dreon, Phinney, and Leeuwenburgh, 2003; Vassilakopoulos et al., 2003). In contrast, several studies showed that acute antioxidant treatment reduced oxidative stress but not the inflammatory response, suggesting these effects may be independent of each other (Davison and Gleeson, 2006). Future research should examine FRS effects on exercise-induced oxidative damage. The present study only made an indirect assessment of damage and found no effect of acute supplementation on exercise performance.

Summary

The purposes of this study were to determine the effects of the chronic and acute FRS supplementation on performance in rowers during an off-season training period. It was found that chronic FRS supplementation did not improve 500 and 2000 m rowing times, vertical jump or 1 RM bench press. Similarly, acute FRS supplementation did not improve 500 and 2000 m rowing time trial performance. It is concluded that under the experimental conditions used in this study chronic and acute FRS supplementation did not affect performance in collegiate rowers in their off-season.

Chapter 6

Summary, Conclusions, and Recommendations

This chapter provides an overview of the experiment and contains three sections: (1) Summary (2) Conclusions, and (3) Recommendations.

Summary

The purpose of this study was to explore the effects of a flavanoid (i.e., quercetin) containing sport drink (i.e., FRS) on rowing performance. This study had two parts. Part 1 measured the chronic effects of FRS on rowing performance, whereas Part 2 measured the acute effects. Forty-six male and female collegiate rowers, between the ages of 18 to 25 years, were randomly selected and pair-matched on their performance on 2000 m rowing ergometer test. They were then placed into either a supplement or placebo group for the first part of the study (i.e., chronic supplementation).

Baseline data included 500 m and 2000 m rowing ergometer tests, 1RM bench press and vertical jump. Subjects were then advised to consume FRS sport drink (supplement or placebo) twice a day for three weeks.

After three weeks, subjects again reported to the lab for the measurement of the aforementioned tests.

In the second part of the study, subjects of the placebo group were pair-matched on 2000 m rowing ergometer test and placed into a supplement group or placebo group. But this time, FRS was given twice before testing (i.e., first drink 150 min prior and second drink just prior to testing). Both 500m and 2000m rowing were measured. Data were analyzed using 2×2 repeated measures ANOVA and dependent sample t-test.

Conclusions

1. Three weeks of chronic supplementation of FRS did not improve rowing or power performances. On the contrary, placebo group 2000 m row time improved significantly

($p < 0.05$). This result should be interpreted with caution as this study was conducted during the off-season and subjects were training by themselves.

2. Acute supplementation was also found ineffective, as both 2000 m and 500 m row tests failed to reach statistical significance. There was a significant time main effect, which means there was a trend for improvement across groups, but post-hoc independent and dependent sample analysis failed to reach statistical significance. It can be concluded that acute supplementation did not affect performance.

Recommendations

Upon completion of the study, the following recommendations for further investigations are deemed appropriate:

1. To further investigate the time of the training year in which the study is being conducted. The present study was conducted during an off-season without control of exercise routines.

2. To see if all the athletes followed proper or an identical training program throughout the study. In the present study subjects were not monitored for their training program.

3. In the present study, markers of oxidative stress were not included. Future studies should employ markers of oxidative stress to more conclusively examine the relationship between FRS supplementation and oxidative stress.

4. To compare effects of quercetin when administered exclusively versus combined with other flavanoids and caffeine.

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Appendices

Appendix A

Recruitment Flyer

Would you like to participate in a research project that is examining the effect of querectin containing free radical scavenger (FRS) sport drink on performance in rowers? The Company claims that this drink has performance enhancing effects if it is consumed for several weeks. Minimum of 1 year of competitive rowing experience is required to participate in this project. Subjects already consuming FRS sport drink and those who are having allergy to certain dyes, or having diabetes, taking antibiotics should not participate in this project. This experiment requires you to consume the FRS drink twice a day for three weeks. If you participate, there will be a maximum of five laboratory sessions although some of you might only do four sessions. In these sessions, 500 m and 2000 m rowing, vertical jump and 1 RM bench press tests will be done in that order. Adequate rest periods will be given between tests. Each lab session will take less than two hours. Generally you should not experience any discomforts from participating in this project other than you might get fatigued or might have some muscle soreness the next day. Participation is voluntary and you can withdraw from the study at any time without penalty. You must be at least 18 years or over to participate. Laboratory sessions will be scheduled according to your availability and coach's advice.

Do you have any question??? Would you like to read the informed consent form, which describes what will happen in greater detail?

IC CREW TEAM!!!

Appendix B

Subject Recruitment Statement

Would you like to participate in a research project that is examining the effect of querectin containing free radical scavenger (FRS) sport drink on performance in rowers? The Company claims that this drink has performance enhancing effect if it is consumed for several weeks. This experiment requires you to consume the FRS drink twice a day for three weeks. If you participate, there will be a maximum of five laboratory sessions although some of you might only do four sessions. In these sessions, 500 m and 2000 m rowing, vertical jump and 1 RM bench press tests will be done in that order. Adequate rest periods will be given between tests. Each lab session will take less than two hours. Generally you should not experience any discomforts from participating in this project other than you might get fatigued or might have some muscle soreness the next day. Participation is voluntary and you can withdraw from the study at any time without penalty. You must be at least 18 years or over to participate. Laboratory sessions will be scheduled according to coach's advice and your availability.

Do you have any question??? Would you like to read informed consent, which describes what will occur in greater detail?

Appendix C

Informed Consent Form for
Participation in Human Subjects Research
Ithaca College

Purpose of study:
The purpose of the study is to examine if chronic and acute administration of quercetin containing Free Radical Scavenger (FRS) sport drink improves athletic performance while reducing the incidence of muscle fatigue in college-aged members of a varsity crew team.

Benefits of the study:
Improving and maintaining sport performance is very important to all athletes. Muscle fatigue may occur due to oxidative damage through free-radicals produced during exercise. FRS is a sport drink formula, which contains several antioxidants including a flavanoid-quercetin. The manufacturer claims FRS improves athletic performance by scavenging those free radicals produced during the exercise. Large amounts of money are spent on various types of sport drink for an athlete to get an extra edge during the competition, but little scientific evidence exists to support the use of these drinks. The result of this project may give credence for chronic or acute FRS for enhancing rowing performance. Alternatively, we may find, use of this drink is not effective and not worth any expenses.

What you will be asked to do:
Testing will be done in five different sessions at the Exercise Physiology Laboratory and Ithaca College Boat House. Each visit will take approximately 1.5-2.0 hours. You will be asked to attend an orientation meeting to get full details of the study, be introduced to the testing procedures, and answer your questions. 500 m and 2000 m time trial rowing ergometer tests, vertical jump and maximum bench press tests will be measured as follow:1st part: Pre-supplemental day 1: This will be your baseline measurements. Upon entry height, weight and body fat will be measured. After this, you will do your routine warm-up for 10-15 min and will start with a 500 m rowing ergometer time-trial, followed by cool-down for 2-3 min. After completing 500 m rowing, you will be given a rest for 30 min. After that, you will undergo 5 min of warm-up followed by 2000 m rowing ergometer time-trial and a cool-down for 2-3 min.On another day, there will be 2nd laboratory session to measure vertical jump and maximum bench press. For the vertical jump test, you will be given 3 trials and the best one will be recorded. For bench press, your maximum bench press will be recorded. After this testing, you will consume FRS drink twice a day for three weeks. One drink in the morning with breakfast or lunch and one drink in the evening with dinner. During this period of three weeks you will otherwise be continuing your routine work out plan.

After three weeks, you will be scheduled for the 3rd and 4th laboratory session to assess the post-supplement changes in aforementioned tests on two separate days.
2nd Part: Some of you will be scheduled for laboratory session 5 and will be asked to consume FRS twice more in a 24 hour period. This will be the final laboratory session when we will again measure 500 m rowing, 2000 m rowing, vertical jump height, and maximum bench press tests.

Risks:
Your risks of participation are minimal and are no more than the risks faced during the each testing session. These include fatigue, soreness, and potential injuries, which always accompany heavy exercise. FRS is a commercially available beverage with no known adverse effects; however, adding anything new to your diet may cause some digestive problems. Although no

special medical arrangements have been made, in the event of injury, standard first aid will be available during each testing session. All emergency procedures will be followed if need arises. If outside care is warranted then 911 will be called. There is no compensation available for injury; you are responsible for all medical costs in such an event.

If You Would Like More Information About the Study
Please contact principal investigator, Ankit Trivedi, Dr. G. A. Sforzo to get more information about the study, or to get a copy of the results.

Email/phone Ankit Trivedi- atrived1@ithaca.edu/ 607-591-9041
Dr. G.A. Sforzo- sforzo@ithaca.edu / 607-274-3359

Withdrawal from the Study
You may stop participating or withdraw from this study at any time without any penalty.

Confidentiality of the Data
All collected data will be confidential and shared only in group form. The data files will be kept in the graduate office in the Center for Health Sciences at Ithaca College in a secure lockable cabinet. Computer files will be accessed only by the principal investigator with a password. Your name will not be used in a connection with this study.

I have read the above and I understand its contents. I agree to participate in this study. I acknowledge that I am 18 years of age or older. I have received a copy of this consent for my own records.

Name (Print):_____

Signed (Sign): _____ Date: _____

Appendix D

Exercise History Questionnaire

General Instructions: Please complete this form as much detail as possible.

Name (please print): _____

Age: _____ Sex: _____ Phone no: _____

Status (circle one): FR SO JR SR GRAD FAC/STAFF

Please rate your exercise level on a scale of 1 to 5 (5 indicating very strenuous):_____

Do you start exercise program but then find you self unable to adhere to them?
Yes ☐ No ☐

Do you currently exercise?
Yes ☐ No ☐
If yes, indicate the major Activities:
_____

Enter duration of exercise/day: _____ No. of days/week you exercise: _____

I have been doing the above mentioned/similar exercise for _____weeks/months/years now (Circle)

Rate your perceived exertion during your current exercise program (check the corresponding box):
☐Light ☐ Fairly Light ☐Somewhat Hard ☐ Hard

How long have you exercised on a consistent basis? _____ Months _____ Years

Please indicate the types of equipment you enjoy using at IC fitness center:
☐Exercise Bike ☐Elliptical trainer ☐ rower ☐ Treadmill
☐Step machine ☐Cybex (variable resistance machine) ☐ Free weights

Use the following scale to rank your fitness goals:

Extremely important				Somewhat important					Not at all important
1	2	3	4	5	6	7	8	9	10

_____ Improve cardiovascular fitness _____ Body-fat weight loss

_____ Reshape or tone my body _____Improve sport performance

_____ Improve mood and stress tolerance _____ Improve flexibility/Strength

Appendix E

Health History Questionnaire

Name _____ Date _____

Street Address _____ City _____

State _____ Zip _____

Phone (home) _____ (work) _____

Sex M F Age _____ Date of Birth _____/_____/_____ Class Level _____

Physician's Name _____ Phone _____

Address _____

*** Person to Contact In Case Of Emergency ***

Name _____ Relationship _____

Home Ph _____ Work Ph _____

Do you now, or have you had in the past: YES NO

1. History of heart problems, chest pains or stroke? _____ _____

2. Increased blood pressure? _____ _____

3. Any chronic illness or condition? _____ _____

4. Difficulty with physical exercise? _____ _____

5. Advice from physician not to exercise? _____ _____

6. Recent surgery (in the last 12 months)? _____ _____

7. Pregnancy (now or within the last 3 months)? _____ _____

8. History of breathing or lung problems? _____ _____

9. Muscle, joint or back disorder, or any previous injury still affecting you? _____ _____

10. Diabetes or thyroid condition? _____ _____

11. Cigarette smoking habit? _____ _____

12. Obesity (more than 20 percent over ideal body weight)? _____ _____

13. Increased blood cholesterol? _____ _____

14. History of heart problems in immediate family? _____ _____

15. Hernia, or any condition that may be aggravated by lifting weights? _____ _____

16. Are you taking any medication or drugs? _____ _____

If yes, please list: _____

17. Does your physician know you are participating in this exercise program? _____ _____

Sign:_____

Appendix F

Raw Data

Chronic Supplementation Raw Data (Supplementation Group)

	PRE	POST	PRE	POST
Gender	500 m	500 m (2)	2000 m	2000 m (2)
Male	1.38	1.42	6.63	6.4
Male	1.38	1.38	6.35	6.4
Male	1.48	1.45	6.78	6.67
Male	1.51	1.53	6.83	6.92
Male	1.5	1.5	6.63	6.63
Male	1.5	1.47	6.95	6.92
Male	1.5	1.5	6.8	6.8
Male	1.52	1.48	6.72	6.65
Male	1.52	1.52	6.97	6.87
Male	1.52	1.53	7.13	7.1
Male	1.55	1.57	7.95	6.9
Male	1.53	1.53	7.97	6.88
Male	1.6	1.58	7.62	7.57
Female	1.6	1.57	7.15	7.57
Female	1.77	1.75	7.63	7.62
Female	1.72	1.68	7.68	7.67
Female	1.8	1.78	7.72	7.72
Female	1.75	1.72	7.72	7.63
Female	1.77	1.75	7.8	7.75
Female	1.85	1.77	7.95	7.93
Female	1.93	1.85	7.98	7.93
Female	1.77	1.77	8.03	8.03
Female	1.92	1.88	8.48	8.35

Mean	1.62	1.61	7.37	7.26
SD	0.17	0.15	0.59	0.57

Appendix F continued

Chronic Supplementation Power Data (Supplementation Group)

	PRE	POST	PRE	POST
Gender	1 RM	1 RM (2)	V.J	V.J (2)
Male	97.7	99	40	40
Male	75.0	74.25	45	45
Male	93.2	94.5	39	41
Male	97.7	101.25	41	50
Male	79.6	74.25	44	41
Male	75.0	74.25	40	38
Male	81.8	83.25	45	46
Male	75.0	76.5	46	45
Male	75.0	78.75	34	38
Male	84.1	85.5	55	55
Male	75.0	92.25	47	42
Male	65.9	63	43	41
Male	65.9	65.25	39	41
Female	61.4	56.25	23	31
Female	43.2	45	23	28
Female	52.3	51.75	28	31
Female	52.3	49.5	25	25
Female	47.7	49.5	20	19
Female	54.6	56.25	21	23
Female	54.6	49.5	24	27
Female	50.0	49.5	30	31
Female	50.0	51.75	31	33
Female	47.7	49.5	19	19

Mean	67.59	68.28	34.87	36.09
SD	16.82	18.19	10.45	9.71

Appendix F continued

Chronic Supplementation Raw Data (Placebo Group)

	PRE	POST	PRE	POST
Gender	500 m	500 m (2)	2000 m	2000 m (2)
Male	1.43	1.43	6.43	6.5
Male	1.48	1.45	6.63	6.7
Male	1.52	1.5	6.65	6.7
Male	1.52	1.48	6.72	6.68
Male	1.52	1.52	6.75	6.83
Male	1.5	1.57	6.78	6.98
Male	1.42	1.45	6.8	6.93
Male	1.53	1.52	6.93	6.98
Male	1.53	1.55	7.08	7.12
Male	1.58	1.55	7.07	6.98
Male	1.62	1.58	7.3	7.27
Male	1.67	1.65	7.47	7.47
Female	1.65	1.65	7.35	7.32
Female	1.72	1.7	7.47	7.58
Female	1.62	1.63	7.48	7.6
Female	1.75	1.72	7.67	7.92
Female	1.65	1.63	7.7	7.68
Female	1.78	1.82	7.83	7.8
Female	1.8	1.8	7.93	7.9
Female	1.88	1.82	7.95	7.95
Female	1.9	1.87	8.23	8.3
Female	1.85	1.82	8.32	8.22
Female	1.92	1.88	8.58	8.5
Mean	1.65	1.63	7.35	7.39
SD	0.15	0.14	0.60	0.58

Appendix F continued

Chronic Supplementation Power Data (Placebo Group)

Gender	PRE 1 RM	POST 1 RM (2)	PRE V.J	POST V.J (2)
Male	70.5	74.3	44	47
Male	75.0	76.5	39	35
Male	77.3	76.5	44	48
Male	90.9	96.8	44	44
Male	77.3	65.3	49	51
Male	93.2	96.8	40	43
Male	106.8	112.5	42	35
Male	65.9	65.0	33	38
Male	65.9	63.0	36	38
Male	63.6	67.5	37	37
Male	79.6	78.8	37	34
Female	59.1	58.5	26	34
Female	52.3	47.3	21	25
Female	75.0	72.0	37	36
Female	52.3	47.3	35	35
Female	63.6	60.8	34	34
Female	54.6	54.0	26	33
Female	52.3	51.8	30	28
Female	47.7	47.3	20	26
Female	47.7	42.8	25	26
Female	54.6	51.8	23	29
Female	40.9	45.0	42	46
Mean	66.63	65.95	34.73	36.45
SD	16.65	18.55	8.30	7.45

Appendix F continued

Acute Supplementation Raw Data (Placebo Group)

Gender	PRE 500 m	POST 500 m (Acute)	PRE 2000 m	POST 2000 m (Acute)
Male	1.38	1.38	6.4	6.38
Male	1.53	1.52	6.92	6.88
Male	1.5	1.5	6.65	6.65
Male	1.5	1.5	6.8	6.75
Male	1.57	1.57	6.92	6.93
Female	1.78	1.77	7.72	7.7
Female	1.75	1.72	7.75	7.6
Female	1.78	1.8	7.93	7.88
Female	1.85	1.85	8.45	8.33
Female	1.57	1.57	7.57	7.23
Mean	1.62	1.62	7.31	7.23
SD	0.16	0.16	0.66	0.62

Appendix F continued

Acute Supplementation Raw Data (Supplementation Group)

Gender	PRE 500 m (Chronic)	POST 500 m (Acute)	PRE 2000 m (Chronic)	POST 2000 m (Acute)
Male	1.42	1.42	6.42	6.5
Male	1.45	1.47	6.67	6.65
Male	1.47	1.52	6.93	6.98
Male	1.53	1.53	6.87	6.9
Male	1.53	1.52	7.1	7.1
Female	1.75	1.75	7.62	7.39
Female	1.68	1.67	7.67	7.67
Female	1.72	1.75	7.63	7.62
Female	1.85	1.87	7.93	7.93
Female	1.77	1.77	8.03	7.95
Mean	1.62	1.63	7.29	7.27
SD	0.15	0.15	0.56	0.52