Georgia State University
ScholarWorks @ Georgia State University

Nutrition Theses

Department of Nutrition

Fall 12-15-2010

The Relationship between Omega-3 Fatty Acid Intake and Muscle Soreness in Collegiate Rowers

Danica R. Carswell

AUTHOR'S STATEMENT

In presenting this thesis as a partial fulfillment of the requirements for the advanced degree from Georgia State University, I agree that the library of Georgia State University shall make it available for inspection and circulation in accordance with its regulations governing materials of this type. I agree that permission to quote, to copy from, or to publish this thesis may be granted by the professor under whose direction it was written, by the College of Health and Human Sciences director of graduate studies and research, or by me. Such quoting, copying, or publishing must be solely for scholarly purposes and will not involve potential financial gain. It is understood that any copying from or publication of this thesis which involves potential financial gain will not be allowed without my written permission.

Signature of Author

NOTICE TO BORROWERS

All theses deposited in the Georgia State University library must be used in accordance with the stipulations prescribed by the author in the preceding statement. The author of this thesis is:

Danica Carswell
1195 Wyckfield Place
Lawrenceville, GA 30044

The director of this thesis is:

Dan Benardot, Ph.D, RD, LD, FACSM
Division of Nutrition
College of Health and Human Sciences
Georgia State University
Atlanta, Georgia 30303-3083

VITA

Danica R. Carswell

ADDRESS: 1195 Wyckfield Place
Lawrenceville, GA 30044

EDUCATION:

M.S.	2010	Georgia State University Health Sciences
B.S.	2009	Georgia State University Nutrition

PROFESSIONAL EXPERIENCE:

May 2009 – Dec. 2010 Dietetic Intern
Children's Healthcare of Atlanta, Grady Memorial Hospital, Georgia State University Counseling and Recreation centers, Davita Dialysis Campus West, WIC Campus Lawrenceville, Saint Joseph's Hospital, New Horizons Campuses North and West, GA

Aug. 2009 – June 2010 Cooking/Nutrition Afterschool Instructor
Drew Charter School, Atlanta, GA

May 2008 – Sept. 2008 Receptionist
NutriFit, Sport, Therapy Inc., Atlanta, GA

May 2008 – Sept. 2008 Cashier/Server
Robeks Fruit Smoothies and Healthy Eats, Atlanta, GA

Mar. 2006 – Aug. 2007 Server
Monterrey Mexican Restaurant, Lawrenceville, GA

PROFESSIONAL SOCIETIES AND ORGANIZATIONS:

2010-present Greater Atlanta Dietetic Association
2008-present American Dietetic Association

PRESENTATIONS AND PUBLICATIONS:

2010 Morningside Elementary School Business Plan for Schoolyard Sprouts

2010 Georgia Dietetic Association Poster Presentation: Independent Senior Meals Satisfaction Surveying for Atlanta Regional Commission

ABSTRACT

THE RELATIONSHIP BETWEEN OMEGA-3 FATTY ACID INTAKE AND MUSCLE SORENESS IN COLLEGIATE ROWERS

Background: Intense exercise may result in delayed-onset muscle soreness (DOMS), which is worsened with unaccustomed eccentric exercise, and is considered a major contributing factor to poor athletic performance. DOMS-related muscular inflammation appears to be especially prevalent in college-aged athletes. There is beginning evidence suggesting that omega-3 fatty acid (O3FA) intake may aid in alleviating muscle soreness, possibly because O3FA contribute to the production of anti-inflammatory eicosanoids. **Objective:** The primary objective of this study was to determine if the intake of omega-3 fatty acids was associated with a reduction in muscle soreness in collegiate rowers. **Methods:** Several groups of collegiate rowers were identified as potential subjects and, using an IRB-approved protocol, 61 volunteers from this subject pool agreed to participate in the study. At the end of the first week of fall practice, subjects were asked to provide information on age, weight, height, ethnicity, rowing level, sun exposure, exercise regimen, pre- and peak-soreness and exertion levels, hydration status, and food/supplement intake. A Talag scale was used to determine the severity of muscle soreness, and a modified Borg scale was used to determine the exertion intensity level. A food frequency questionnaire using a 10-point Likert scale was used to assess food and supplement intake, with a focus on omega-3 fatty acids. **Results:** Subjects with a higher intake of EPA/DHA rich bottled supplemental fish oil, taken by spoon, had significantly higher soreness levels than those with lower intakes ($p = 0.028$). Higher soreness was also significantly associated with higher intake of EPA/DHA rich fish oil capsules ($p < 0.026$). Lower soreness was significantly associated with higher fruit intake ($p = 0.030$). **Conclusions:** These findings suggest that high intakes of omega-3 fatty acids from fish oils, regardless of liquid or capsule form, may be contraindicated for collegiate rowers wishing to reduce muscle soreness. Recommending fruit intake may be warranted in rowers wishing to reduce muscle soreness.

THE RELATIONSHIP BETWEEN OMEGA-3 FATTY ACID INTAKE AND MUSCLE SORENESS IN COLLEGIATE ROWERS

By
Danica Rose Carswell

A Thesis

Presented in Partial Fulfillment of Requirements for the
Degree of Master of Science in Health Sciences
Division of Nutrition
College of Health and Human Sciences
Georgia State University

Atlanta, Georgia
2010

ACKNOWLEDGMENTS

I would like to thank my thesis committee – Dr. Dan Benardot, Dr. Anita Nucci, and Dr. Walter Thompson – for their invested time, assistance, and guidance through this incredible process – I value everything I have learned from it. Mary Robeck is also well-deserving of recognition for her invaluable editing support in the early stages of my paper. Big thanks to Dr. Barbara Hopkins for helping me find a validated urine color chart! I would also like to thank all previously unmentioned faculty from the Division of Nutrition who have taught and advised me during both my undergraduate and graduate degree programs at Georgia State – Dr. Dea Baxter, Dr. Mildred Cody, Dr. Vijay Gangi, Dr. Jana Kicklighter, Professor Cathy McCarroll, Dr. Murugi Ndirangu, Dr. Meera Penumetcha, and Professor Susan Roman. The Division of Nutrition at Georgia State has been my home for many years, and in those years, I have gotten to learn a great deal both from and about the wonderful people who teach and advise for the division - they are an awesome group of people! I would especially like to thank my mom, Carrie, my significant other, Jason Barry, my roommate, Alex Kennedy, and all of my classmates for their continued support, understanding, and encouragement throughout my Master's degree program and thesis preparation. Lastly, thanks to the rowers for their participation in this study and to their coaches, who took time from their practices to allow for study participation.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

ALA	Alpha-linolenic acid
ATP	Adenosine triphosphate
BCAA	Branched-chain amino acids
COX	Cyclooxygenase
DHA	Docosahexaenoic acid
DOMS	Delayed-onset muscle soreness
EAR	Estimated Average Requirement
EE	Eccentric exercise
EIMD	Exercise-induced muscle soreness
EPA	Eicospentaenoic acid
HEPE	Hydroxyeicosapentaenoic acid
IL	Interleukin
LOX	Lipooxygenase
NADPH	Nicotinamide adenine dinucleotide phosphate
NSAIDS	Non-steroidal anti-inflammatory drugs
O3FA	Omega-3 fatty acid
O6FA	Omega-6 fatty acid
PGG_2	Prostaglandin G_2
PGH	Prostaglandin endoperoxide
PGH_2	Hydroxyendoperoxide
PI	Primary investigator

PUFA	Polyunsaturated fatty acid
TNF-alpha	Tumor necrosis factor-alpha
ROM	Range of motion
ROS	Reactive oxygen species

CHAPTER ONE

INTRODUCTION

THE PROBLEM

Muscle soreness is commonly experienced by athletes and is among several factors that contribute to poor athletic performance. In recent literature, muscle soreness has been shown to have negative effects on athletic performance, because of its association with extreme reductions in range of motion and muscle strength (Cleary et al., 2005; Parr et al., 2009). Muscle soreness referred to as “delayed-onset muscle soreness” (DOMS) is that which peaks between 24 and 48 hours post-exercise and continues 2 to 4 days (Cleary et al., 2005; Frey-Law et al., 2008; Mayer et al., 2006; Rahnama et al., 2005; Tartibian et al., 2009).

DOMS is defined as a grade I muscle strain injury which represents a stretching and/or minor tearing of the muscle, but not partial (grade II) or complete (grade III) muscle tearing (Cheung et al., 2003; Ivins, 2006). It is thought to originate from inflammation secondary to eccentric exercise (EE): Tartibian et al. (2009) found increases in limb circumference accompanied by DOMS after EE intervention. EE is defined as a form of exercise that lengthens (or stretches) the muscle with contraction (Prasartwuth et al., 2005). Lengthened contractions (versus shortened ones) have been shown to increase muscle damage, which is triggers the inflammatory response.

The inflammatory response is initiated by the calcium-mediated production of eicosanoids (prostaglandins, thromboxanes, and leukotrienes) from polyunsaturated fatty acids (PUFA), and their production is upregulated by the production of cytokines [tumor necrosis factor-alpha (TNF-alpha), interleukin-1 (IL-1), and interleukin-6 (IL-6)] in response to damage or already present inflammation (Mahan & Escott-Stump, 2008). These eicosanoids control growth factor release, protease release, and attraction of phagocytic cells in response to cell injury or damage (Mahan & Escott-Stump, 2008). This increased movement of inflammatory particles to an area of damage and edema associated with inflammation are thought to increase intramuscular pressure, stimulating the group III and IV afferent sensory receptors that terminate in the myofibrils (Smith, 1991; Armstrong, 1990). Cheung et al. (2003) showed evidence of this, finding an association between decreased DOMS-linked inflammation and lower intramuscular pressure.

The inflammatory nature of eicosanoids is determined by the fatty acids from which they are derived (Figure 1). Omega-6 fatty acids become pro-inflammatory eicosanoids, but omega-3 fatty acids (O3FA) become anti-inflammatory 3-series prostaglandins and thromboxanes via the cyclooxygenase-2 (COX-2) pathway and 5-series leukotrienes via the lipooxygenase (LOX) pathway (Shils et al., 2006). Tartibian et al. (2009) have found O3FA supplementation to significantly decrease muscle soreness

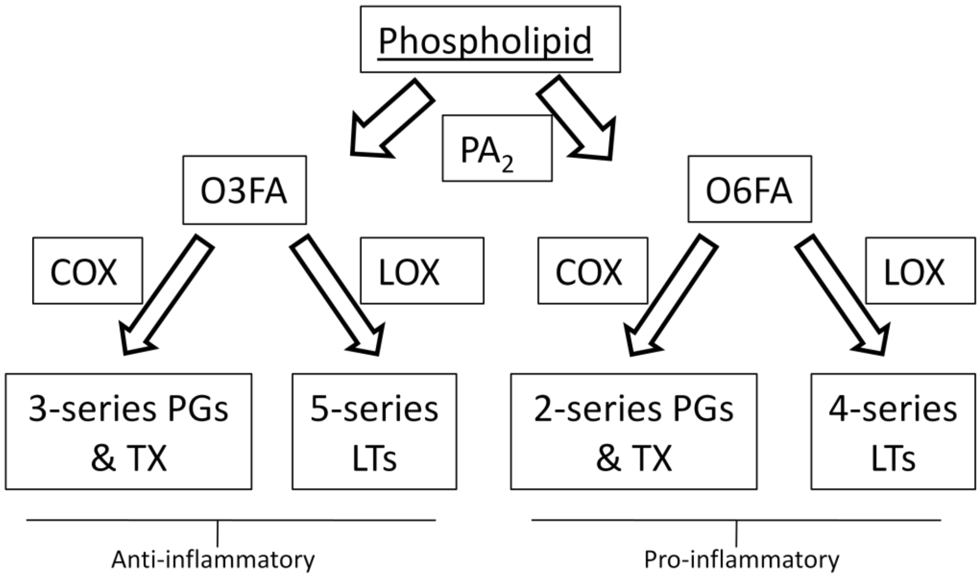

Figure 1 – Eicosanoid production (source: Champe et al.; 2008: 215).

Because O3FA have been shown to reduce muscle soreness through anti-inflammatory action, it is important to investigate their intake. Intake of O3FA in collegiate athletes is of particular interest, because they have a higher risk for developing muscle soreness than other age groups, which may reduce athletic performance (Chapman et al., 2008; Marginson et al., 2005). They have also been shown to have low intakes of O3FA-rich foods like fish, nuts, and seeds (Brunt et al., 2008). In addition, there are aspects of the rowing technique that involve inflammatory-inducing eccentric contractions vastus medialis action prevents further forward motion by stopping knee flexion at the catch of the stroke, and rectus femoris action prevents further backward motion by slowing hip extension at the stroke finish (Figure 2) (Soper & Hume, 2004).

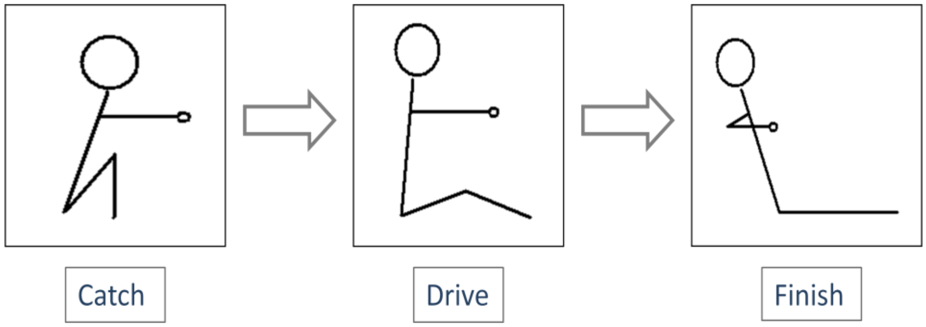

Figure 2 – The rowing technique. The catch represents the beginning of the stroke, where the oar blade is dropped into the water. The drive represents the oar being pulled through the water. The finish represents the end of the stroke, where the oar blade is lifted out of the water.

PURPOSE & HYPOTHESIS

Only two studies by Lenn et al. (2002) and Tartibian et al. (2009) have recently examined the effects of O3FA intake on muscle soreness. Results of these studies were conflicting, and neither study dealt with athletes or, specifically, rowers. The major purpose of this study is to assess whether different levels of O3FA consumption from both foods and supplements was associated with muscle soreness in collegiate rowers. Ultimately, this information may help determine whether higher levels of O3FA intake would be useful to collegiate rowers who currently experience high levels of muscle soreness.

Hypothesis: Higher omega-3 fatty acid intake is associated with reduced muscle soreness in collegiate rowers.

Null Hypothesis: Higher omega-3 fatty acid intake is not associated with reduced muscle soreness in collegiate rowers.

CHAPTER TWO

LITERATURE REVIEW

EXERCISE-INDUCED MUSCLE DAMAGE

Exercise-induced muscle damage (EIMD) is commonly experienced by athletes and is characterized by a decreased range of motion (ROM), delayed-onset muscle soreness (DOMS), and decreased muscle strength, all of which negatively impact athletic performance (Parr et al., 2009). It has been suggested that EIMD occurs with the disruption of muscle fibers with eccentric exercise (Allen et al., 2005). In a study by Cleary et al. (2005), 10 untrained male subjects (mean age of 21 years) reported a 10.5% decrease in muscle strength accompanied by significant DOMS ($p = 0.001$) at 24 hours post-eccentric exercise. After an eccentric exercise intervention in a separate study, 24 untrained subjects of mixed gender and college age reported 30-36% decreased strength ($p < 0.01$) and a decrease in ROM between 5 and 8 percent ($p < 0.01$), with significant muscle soreness ($p < 0.05$) (Parr et al., 2009). Theories on method(s) of causation ultimately discuss inflammation.

Mechanisms of Primary EIMD

The three major forms of resistance exercises are isometric, concentric, and eccentric (Prasartwuth et al., 2005, Mackey et al., 2008; Jubeau et al., 2007), all of which have been shown to cause muscle damage. Isometric exercise involves muscle contraction without a change in muscle length (Jubeau et al., 2007). Concentric exercise involves a shortening of the muscle with contraction (Mackey et al., 2008). Eccentric exercise (EE) is defined as a form of exercise that lengthens (or stretches) the muscle with contraction (Prasartwuth et al., 2005).

Lengthened contractions have been shown to increase muscle damage and significantly induce its characteristic symptoms (Philippou et al., 2003; Parr et al., 2009). It is this lengthening that has been proposed to cause primary muscle damage, where the sarcomeres of the muscle fiber are disrupted; the repeated lengthening causes overstretching of the myofilaments of the sarcomere, causing increased tension in others (Morgan & Proske, 2004). The change in structure may change the function, which would account for the decreased strength associated with EIMD.

The effects of eccentric exercise may apply to the sport of rowing, as there are aspects of the rowing technique that involve eccentric contractions: vastus medialis action stops knee flexion at the catch of the stroke, and rectus femoris action slows hip extension at the end of the stroke drive (Soper & Hume, 2004). Eccentric contractions are also involved in a number of cross-training activities: step-ups, squats, lunges, bicep curls, downhill running, low back extensions, and wrist extensions, among others (Prasartwuth et al., 2005; Dwyer et al., 2010; Cleary et al., 2005, Frey-Law et al., 2008)

Mechanisms of Secondary EIMD

There are several theories on methods of secondary muscle damage. One theory is that following primary muscle damage (where the sarcomeres are disrupted), muscle cell membranes become damaged and the intracellular and extracellular equilibrium changes, resulting in a large influx of calcium (Allen et al., 2005). Calcium influx activates phospholipase A_2, which frees fatty acids from the membrane (Gissel, 2005; Shils et al., 2006). One of these fatty acids is arachidonic acid, which can produce reactive oxygen species (ROS) via activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (Ayilavarapu et al., 2010; Gissel, 2005). These ROS may damage cell structures, which stimulates an inflammatory response (Mahan & Escott-Stump, 2008).

Another theory suggests that calcium influx leads to calcium overload in the mitochondria, causing the permeabilization transition pore in mitochondria to open (Duchen, 2004; Parekh & Putney, 2005). Opening of this pore leads to mitochondrial membrane depolarization and decreased adenosine triphosphate (ATP) production and use (Duchen, 2004). Decreased energy production and use by cells leads to apoptosis and necrosis, whereby the inflammatory response is stimulated (Duchen, 2004; Mahan & Escott-Stump, 2008).

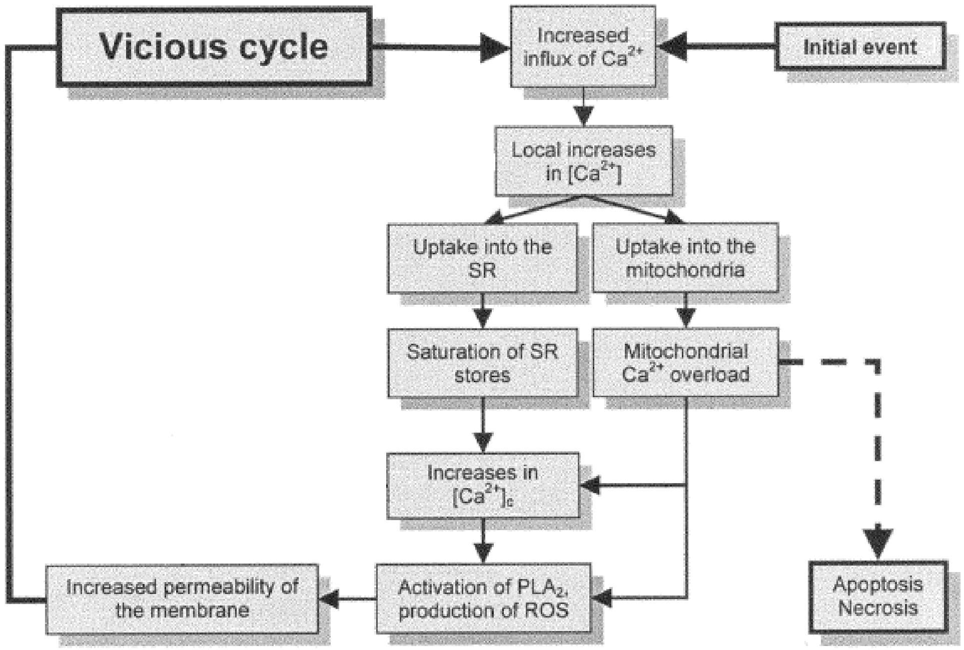

Figure 3 – Mechanisms of secondary muscle damage (source: Gissel, H; 2005: 172).

Inflammatory Mechanisms

In each of the inflammation theories mentioned above, muscle cell damage ultimately leads to an inflammatory response. This process is initiated by the production of eicosanoids (prostaglandins, thromboxanes, and leukotrienes) from polyunsaturated fatty acids (PUFA), and their production is upregulated by the production of cytokines [tumor necrosis factor-alpha (TNF-alpha), interleukin-1 (IL-1), and interleukin-6 (IL-6)] in response to damage or inflammation (Mahan & Escott-Stump, 2008). These eicosanoids control growth factor release, protease release, and attraction of phagocytic cells in response to cell injury or damage. This process is calcium-mediated (Shils et al., 2006), so calcium influx may have an influence on the size of the inflammatory response.

DELAYED-ONSET MUSCLE SORENESS

EIMD is characterized by several factors, including DOMS. DOMS is considered a grade I muscle strain injury which represents a stretching and/or minor tearing of the muscle, but not partial (grade II) or complete (grade III) muscle tearing (Cheung et al., 2003; Ivins, 2006). Older theories indicate that DOMS from EIMD results from increased movement of inflammatory particles to an area of damage and edema associated with inflammation, thought to increase intramuscular pressure and stimulate the group III and IV afferent sensory receptors that terminate in the myofibrils (Smith, 1991; Armstrong, 1990). More recent studies indicate that the mechanisms behind pain caused by DOMS are unclear (Close et al., 2005; Parr et al., 2009); however, Cheung et al. (2003) found an association between decreased DOMS-linked inflammation and lower intramuscular pressure, and Tartibian et al. (2009) found increases in limb circumference accompanied by DOMS, both suggesting the presence of edema and inflammatory particles.

How is DOMS Elicited?

All studies examining DOMS use EE to induce soreness, but another major component to induction is the use of subjects that are “unaccustomed” to exercise. Several studies using untrained subjects report greater soreness responses (Cleary et al., 2005; Lavender & Nosaka, 2008; Mackey et al., 2008; Rahnama et al., 2005; Tartibian et al., 2009); however, the time length of pre-study inactivity is not standardized amongst studies. For example, these time periods ranged from 3 weeks (Howatson et al., 2009) to 1 year (Prasartwuth et al., 2006), and some studies did not always a time length.

Using unconditioned subjects is important, as studies with trained participants may not induce high soreness scores. Lavender and Nosaka (2008) examined the effects of training on 18 males, aged 21.4 +/- 2.6 years. Participants were randomly divided into heavy-load-only exercise (40% of maximal isometric strength) and combined light and heavy load exercise (10% of maximal isometric strength followed by 40%) groups, with the combined exercise group separating light and heavy exercise by 2 days. In both cases the intervention consisted of 6 sets of 5 eccentric bicep curls. Based on a 50-unit visual analogue scale, perceived pain scores were significantly lower upon palpation ($p = 0.002$) and passive extension ($p = 0.02$) of the muscle in the group that performed light eccentric exercise 2 days before heavy eccentric exercise than in the heavy-load-only exercise group.

A study by Howatson et al. (2009) used a different intervention for the 16 male subjects; exercise bouts were separated by 2 weeks, and the intervention intensity did not increase. Training demonstrated a DOMS-reducing effect: participants reported significantly lower perceived pain scores after a *repeated* bout of 100 drop jumps (5 sets of 20 repetitions) ($p < 0.05$). Combined with those of the Lavender and Nosaka (2008), these findings suggest that a time period longer than 2 days may allow subjects to become unaccustomed to eccentric exercise. More research is needed to determine a more exact time period.

After soreness-inducing exercise, most studies did not use the same method to elicit a pain response post-exercise. For example, studies surveyed subjects at rest, during stretch, during passive extension, with palpation, and during contraction. (Cleary et al., 2005; Jubeau et al., 2008; Mackey et al., 2008; Mayer et al., 2006; Tartibian et al., 2009), several of which elicited DOMS, several of which did not elicit DOMS.

How is DOMS Measured?

DOMS is measured immediately post-eccentric exercise intervention by the subject. Several scales have been used, including a 10-point Likert scale, visual analogue scale, and Talag scale, none appearing to have an advantage over the other, although the visual analogue scale has been used most often (Nieman et al., 2006; Lenn et al., 2002; Tartibian et al., 2009). The visual analogue scales used endpoints indicating "no pain" and "worst pain" and were typically accompanied by a 10-point numerical pain-rating scale (Frey-Law et al., 2008). Regardless of elicitation method or measuring scale, the post-EE perceived pain scores of many soreness-related studies follow the same trajectory over time, peaking between 24 and 48 hours and lasting about 2 to 4 days (Cleary et al., 2005; Frey-Law et al., 2008; Mayer et al., 2006; Rahnama et al., 2005; Tartibian et al., 2009).

What Populations are at Risk for Developing DOMS?

Those subjects who are of collegiate age have been shown to perceive higher soreness levels. It is still undecided as to whether gender has an effect on soreness levels.

Age and DOMS

Those near college age (18 to 24 years) have a higher risk of developing DOMS. Chapman et al. (2008) compared DOMS between young and old males untrained in exercises similar to that of the intervention. The younger population ($n = 10$) was comprised of university students with a mean age of 25 +/- 1.8 years, while the older population ($n = 10$) was comprised of local community members, aged 64 +/- 1.2 years. Between 24 and 72 hours post-eccentric exercise (5 sets of 6 bicep curls of fast velocity), muscle soreness scores were found to be significantly lower in the older subjects ($p < 0.05$). While the older population experienced significantly decreased muscle strength ($p = 0.016$), there were no significant work differences between the two groups after the intervention. In a similar study comparing an older population aged 20-29 years ($n = 10$) to a younger population aged 9-10 years ($n = 10$), post-exercise soreness scores were significantly higher in the older group ($p < 0.05$) through a 72-hour period (Marginson et al., 2005).

Gender and DOMS

Gender has not shown to be a factor in relieving DOMS, but it may be associated with performance. Tiidus (2003) indicates in his review that estrogen may help to stabilize skeletal muscle by acting as an antioxidant to reduce membrane phospholipid peroxidation and by having a cholesterol-like influence on membrane stability. These actions may help maintain calcium balance in the muscle, which may decrease inflammation after exercise by neutrophils (Tiidus, 2003). Tiidus (2003) indicates that, while these mechanisms are possible, no experimental evidence has demonstrated differential DOMS in males and females. A more recent study has shown evidence that college-aged females ($n = 55$) were significantly more likely than males to experience pain that interfered with daily activities in the third day post-eccentric exercise (bicep curls) intervention (p = 0.01), though the actual between-gender pain scores were not significantly different (Dannecker et al., 2008).

NUTRIENT INTAKE OF THE COLLEGIATE ATHLETE

It is possible that college-aged populations experience DOMS-related poor athletic performance due to inadequate intakes of either micro- or macronutrients (Burke et al., 2006; Driskell & Wolinsky, 2006). Burke et al. (2006) indicate that increased physical activity results in higher macronutrient needs, which arise from higher energy expenditure, and increased micronutrient requirements, which arise from energy-creating metabolic reactions. Several studies have shown significant associations between reduced DOMS and supplementation of O3FA, branched-chain amino acids (BCAA) and vitamins C, D, and E intakes and DOMS (Tartibian et al., 2009; Benson et al., 2006; Silva et al., 2010; Bryer & Goldfarb, 2006; Shimomura et al., 2006).

These findings appear to be based on actual deficiencies or areas of low intake. Brunt et al. (2008) found that college-aged subjects have low intakes of O3FA-rich fish, nuts, and seeds. In a study by the American College Health Association (2010), more than 50% of college students reported consuming less than 3 servings of fruits and vegetables, indicating that they may be deficient in some of the micronutrients found in the fruit and vegetable groups, micronutrients like vitamin C. A study using NCAA Division I athletes (n = 250) by Hinton et al. (2004) reported that 60% of the surveyed population did not meet the estimated average requirement (EAR) for vitamin E, and only 26% of collegiate athletes reported adequate protein intake (partially representing BCAA intake).

The same study using NCAA athletes reported vitamin D intake; the average female consumed 6.7 +/- 4.3 micrograms per day, and the average male consumed 6.9 +/- 5.4 micrograms per day. Mean vitamin D intake in this study was above the recommended intake of 5 micrograms, but large standard deviations indicate deficiencies in a portion of this population (distribution of subjects for each intake level was not reported).

Fatty Acid Supplementation and Effects on DOMS

Because O3FA specifically may indirectly enhance athletic performance via their association with reduced DOMS, it is important to investigate their intake in collegiate athletes and their effects on athletic performance. The most common dietary O3FA include eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), and alpha-linolenic acid (ALA). These are found in fish and shellfish, nuts and seeds, and plant oils (Pickova, 2009).

A study supporting the soreness-ameliorating effects of omega-3 fatty acids examined 27 male subjects (mean age of 33.4 +/- 4.2 years) for perceived pain after O3FA supplementation and an eccentric exercise intervention (Tartibian et al., 2009). Volunteers took a fish oil capsule, containing 324 mg EPA and 216 mg DHA, once per day for 30 days pre-exercise and for 2 days post-exercise. This study also used supplementation of 100 IU d-alpha-tocopherol acetate in addition to the omega-3 capsule, but frequency of administration was not indicated. Exercise consisted of 40 minutes of bench step-ups, alternating 5 minutes stepping and one minute rest. There was a significant difference between the control or placebo group’s and the supplement group’s pain scores seen 48 hours after exercise ($p = 0.001$), but not 24 hours after. Thigh circumference, used as an indicator of inflammation, was significantly lower in the supplemented group at 24 hours ($p < 0.05$) and 48 hours ($p < 0.02$).

The use of tocopherol supplementation may have had an antioxidant effect in these subjects, although Tartibian et al. (2009) wrote that those effects are “regarded as inconclusive.” Another limitation of the study was that, while a food frequency questionnaire was administered, participants were asked to report their intake during the pre-supplementation 12-month time period, so it is unknown whether the groups differed significantly in non-supplemental omega-3 intake *during* supplementation. Another issue the study faced was that the mean age of the participants was about 10 years above collegiate age, so it is possible the effects might have been more pronounced in a younger population.

A study by Lenn et al. (2002) reported a lack of soreness-ameliorating effects from omega-3 fatty acid supplementation. This study examined 10 untrained males (mean age: 22.7 +/- 3.92 years) and 6 untrained females (mean age: 24.5 +/- 5.47) and the effects of O3FA supplementation on muscle soreness. Subjects were divided into three supplement groups: a fish oil group ($n = 6$) receiving 1.8 grams of fish oil, an isoflavone group ($n = 5$) receiving a combination of 120 mg of soy isolate and a non-O3FA-rich fat blend, and a placebo group ($n =6$) receiving a combination of the non-O3FA-rich fat blend and wheat flour. All groups were supplemented 30 days prior to and in the week during the exercise protocol, and they also received 100 IU of vitamin E to reduce fat oxidation. After the 30-

day supplementation period, subjects performed 50 eccentric bicep curls using the KinCom Dynamometer to induce muscle soreness, which was measured 2, 4, and 7 days post-exercise. While a significant increase was seen in serum EPA and DHA ($p < 0.05$), there were no significant differences among soreness levels of the subject groups. Unlike the findings of Tartibian et al. (2009), the findings of this study indicate there is no relationship between O3FA and DOMS. More research is needed to form a conclusion on the relationship between O3FA and DOMS.

Mechanisms of O3FA

O3FA reduce perceived pain associated with inflammation by acting as both antioxidants (indirectly) and anti-inflammatory agents. O3FA help combat inflammation by competing with arachidonic acid – and O6FA – for eicosanoid generation. The more O3FA in the blood and cell membrane, the more likely it will be released for eicosanoid generation.

Arachidonic acid becomes pro-inflammatory 2-series prostaglandins (PG) and thromboxanes (TX) via the cyclooxygenase (COX) pathway and 4-series leukotrienes (LT) via the lipooxygenase (LOX) pathway (Shils et al., 2006). When arachidonic acid is replaced as a substrate, O3FAs become anti-inflammatory 3-series PGs and TXs via the cyclooxygenase (COX) pathway and 5-series LTs via the lipooxygenase (LOX) pathway (Shils et al., 2006). These paths are initiated with the physiological stimulation of normal cells, which signals the activation of phospholipase A_2 (PLA_2) by calcium mediation

(Shils et al., 2006). PLA_2 releases omega-3 PUFA from the cell membrane for eicosanoid generation.

Specifically, the COX-2 pathway (versus COX-1) mediates inflammation (Champe et al., 2008). Prostaglandin endoperoxide (PGH) synthase catalyzes the COX-2 isozyme to oxygenate fatty acids to become prostaglandin G_2 (PGG_2) and then catalyzes glutathione peroxidase, reducing PGG_2 to hydroxyendoperoxide (PGH_2) (Champe et al., 2008; Shils et al., 2006). This intermediate is converted into PGs and TXs, depending on the tissue. PGs and TXs promote platelet aggregation and control contraction of smooth muscle and vasoconstriction/dilation (Champe et al., 2008), but those derived from O3FA have lower biological activity, making them less inflammatory (Mickleborough & Rundell, 2005).

The LOX pathway also mediates inflammation (Champe et al., 2008). Via the 5-LOX pathway, fatty acids are converted into leukotrienes (Shils et al., 2006). LTs increase vascular permeability and control contraction of smooth muscle and broncho/vasoconstriction (Champe et al., 2008), but those derived from O3FA have lower biological activity, making them less inflammatory (Mickleborough & Rundell, 2005). In addition to the 5-LOX pathway, the 15-LOX pathway oxidizes EPA and DHA to 15-hydroxyeicosapentaenoic acid (15-HEPE) and 15-hydroxydocosahexaenoic acid, respectively, and both of these products inhibit the formation of pro-inflammatory 4-series leukotrienes from arachidonic acid (Shils et al., 2006).

Vitamin Supplementation and DOMS

The college-aged population may be at higher risk for developing vitamin and mineral deficiencies due to poor intake. In the literature, vitamins D, E, and C have been examined to determine their effects on muscle soreness.

Vitamin D Supplementation: Effects Similar to O3FA?

The EPA and DHA used to supplement subjects in the O3FA studies are marine-based, meaning they come from seafood sources. Fatty fish are not only good sources of O3FA, but they are also good sources of vitamin D. It is possible that vitamin D may be partly responsible for the soreness-reducing actions of O3FA. In a study of NCAA Division I athletes, Hinton et al. (2004) found that the average vitamin D intake in femalesBenson et al. (2006) examined a native Australian population that had chronic muscle pain and vitamin D deficiencies. The researchers chose eight age- and gender-matched pairs, with one person from each pair presenting with muscle pain and the other not presenting with muscle pain. After the subjects (of mixed age and gender) were selected, blood tests were run to determine presence of vitamin D deficiency. All subjects presenting with muscle pain were found to be vitamin D deficient, while seven of those eight who did not present with muscle pain were found to have normal vitamin D levels.

Vitamin D deficiency has also been linked to decreased muscle strength. Houston et al. (2007) examined muscle strength in 976 elderly subjects of mixed gender with either vitamin D deficiency (< 25 nmol/L), insufficiency (<50 nmol/L), or normal levels. Subjects underwent two sessions (per hand) with the handgrip dynamometer, and the average of the 2 scores was used in the analysis. The study found that vitamin D levels were significantly associated with decreased handgrip (p =< 0.01). The study also found that subjects with a vitamin D deficiency had significantly lower handgrip scores than those with an insufficiency and that those with an insufficiency had significantly lower handgrip scores than those with normal levels ($p < 0.05$). Parathyroid hormone levels – which increase when vitamin D levels are low to stimulate production – were found to be significantly and inversely associated with handgrip in this population ($p = 0.01$). Decreased muscle strength can accompany DOMS (Cleary et al., 2005), so it is possible that the pain described by Benson et al. (2006) is indicative of soreness, though eccentric exercise was not discussed in the study. This information suggests it may be wise to examine the diets of collegiate athletes for vitamin D-rich foods (i.e., fatty fish, fortified dairy products, eggs) to determine an association with DOMS.

Vitamin E Supplementation and DOMS

While studies on O3FA supplementation from both Lenn et al. (2002) and Tartibian et al. (2009) regard vitamin E's effects on muscle soreness as "inconclusive," vitamin E has been found to have a reducing effect on DOMS. Given that vitamin E acts as an antioxidant, it can reduce oxidative damage initiated by exercise-induced muscle damage. Silva et al. (2010) wanted to both examine the antioxidant effects of vitamin E and test for anti-inflammatory effects as well. The researchers recruited 21 male college students from Brazil and split them into placebo and supplement intervention groups. The vitamin E group was instructed to take 800 IU of D-alpha-tocopherol acetate once per day for 21 days – 14 days pre-exercise and 7 days post-exercise. Exercise testing involved eccentric bicep curls held for 6 to 8 seconds in 3 intervals of 2 minutes. Subjects in the supplement group reported significantly lower soreness scores at 2, 4, and 7 days post-exercise than those of the placebo group ($p < 0.05$). To measure oxidative damage reduction, researchers looked at each individual intervention group's effects on lipid peroxidation and protein carbonylation. In both cases, the vitamin E group had significantly lower levels on days 4 and 7 than those seen in the placebo group ($p < 0.05$). To measure anti-inflammatory effects, the researchers examined presence of tumor-necrosis factor-alpha (TNF-alpha) and interleukin-10 (IL-10), both of which were not significantly different between groups. The evidence from this study showed that vitamin E has an antioxidant effect, but no anti-inflammatory effect, concerning DOMS. One weakness with this study was that there was no dietary recall involved, so it is unknown whether the groups significantly differed in non-supplemental vitamin E intake. This information suggests it may be wise to examine the diets of collegiate athletes for vitamin E-rich foods (i.e., plant oils, nuts, seeds) to determine an association with DOMS.

Vitamin C Supplementation and DOMS

Evidence on vitamin C's supplemental effects on DOMS is mixed, although its mechanisms are similar to those of vitamin E. One study, conducted by Bryer and Goldfarb (2006), examined vitamin C supplementation in 18 untrained males (mean age near 23 years). Subjects were split into placebo and vitamin C groups, with the latter group receiving 3 grams of vitamin C per day. Supplementation lasted 2 weeks prior to and 4 days after eccentric exercise intervention (70 bicep curls). Researchers found that the pain scores of the supplemented group were significantly lower at 24 hours post-eccentric exercise than those of the placebo group ($p < 0.05$). Unlike the other supplement-related studies reviewed for this paper, this study by Bryer & Goldfarb

(2006) used dietary recall to ensure that their groups did not have significantly different vitamin C and vitamin E intakes. This information suggests it may be wise to examine the diets of collegiate athletes for vitamin C-rich foods (i.e., fruits, vegetables) to determine an association with DOMS.

A contradictory study by Connolly et al. (2006) observed 24 mixed-gender subjects who were untrained in the exercise of intervention. Participants were divided evenly into placebo and supplemented groups, with the supplemented group receiving 3 grams of vitamin C per day for 8 days – 3 days pre-exercise and 5 days post-exercise. Eccentric exercise intervention consisted of 2 sets of 20 bicep curls. No effects on muscle soreness were observed, but this study did not supplement as long as the previous study. In addition, the amount of work completed in the exercise intervention was less in this study than in the previous study, though the relationship between work and DOMS has not been discussed at length.

Amino Acid Supplementation and DOMS

Amino acid supplementation maintains protein synthesis, which may help minimize tissue damage post-exercise (Shimomura et al., 2006). A cross-over study by Shimomura et al. (2006) looked specifically at BCAA supplementation and its effects on DOMS in 30 untrained, mixed-gender subjects, aged 21-24 years. Fifteen minutes pre- eccentric exercise intervention (seven sets of 20 squats), subjects took a solution containing 5 grams of BCAA (isoleucine:leucine:valine ratio of 1:2.3:1.2), 1 gram of green tea, and 1.2 grams of sugar substitute. The latter 2 ingredients were included to mask the BCAA flavor. When compared against the control scores after the cross-over period, female soreness scores post-BCAA intervention were significantly lower at 24, 48, and 72 hours ($p < 0.05$). Males did not report the same results, hypothesized by researchers to be because the solution was not standardized per kilogram for all participants (males received about 77 +/- 3 mg/kg of BCAA, and females received about 92 +/- 2 mg/kg of BCAA), which may have skewed the results. Further research is necessary to determine if BCAA supplementation has beneficial effects in men as well. The use of green tea must be accounted for, as it may also have an effect on DOMS. No dietary recall was taken during this study, so it is unknown whether the groups had significantly different non-supplemental BCAA intake. This information suggests it may be wise to examine the diets of collegiate athletes for BCAA-rich foods (i.e., meat, dairy, eggs) to determine an association with DOMS.

Fluid Intake and DOMS

Cleary et al. (2005) examined the effects of hydration status on DOMS in 10 untrained males (mean age of 21 years). Subjects were split into hydrated and dehydrated groups before onset of physical activity (5-minute warm-up followed by a 60-minute hyperthermic walk and 45-minute normothermic downhill run), with the hydrated group being instructed to drink water *ad libidum* throughout activity. Post-intervention, DOMS was present in both groups, but overall lower-limb soreness was 44% higher in the dehydrated group ($p = 0.03$). The inclusion of a warm-up poses a question of validity, as warm-ups have been shown to decrease perceived pain scores after eccentric exercise (Law & Herbert, 2007).

Nutrition Education: A Possible Link to DOMS Reduction?

In their transition, the college-aged population acquires a larger freedom to make individual food choices (Economos, 2008), but are they well-equipped to handle this freedom? College-aged students rely on the food and nutrition knowledge base built by their environments to support their food choices (Ha, 2009), but is this knowledge base, alone, sufficient enough to help students make healthy food choices? Ha et al. (2009) found that college students (n = 80) who took a semester-long nutrition education class made more healthful food choices. If nutrition education can influence students to make better food choices, it may be beneficial to examine the nutrition education.

EXERCISE REGIMEN AND ALLEVIATION OF DOMS

In addition to eccentric exercise, there are other exercise-related factors that have shown to reduce muscle soreness. Warm-ups and both pre- and post-exercise stretching contribute to lower DOMS.

Warm-ups and DOMS

Similar to the effects of training and conditioning, warming up before eccentric exercise has been shown to reduce muscle soreness. In a study by Law and Herbert (2007), 52 healthy adults (with a mean age of 21 years) were divided into four groups: control, warm-up-only, cool-down-only, and combined warm-up and cool-down. The intervention involved a warm-up and cool-down (10 minutes each) surrounding 30 minutes of backward, downhill walking. At 48 hours post-exercise, the warm-up-only group had a significant reduction in DOMS compared to those who had not completed the warm up (p = 0.03); no reduction in DOMS was seen in the cool-down-only group. Further research is necessary to determine whether warm-up time length affects DOMS.

Stretching and DOMS

Evidence proving that stretching impacts DOMS is lacking. In a Cochrane review, Herbert and de Noronha (2008) examined stretch studies using mixed-gender subjects (n = 10-30 per study) with a mean age of 25 years. These studies used stretching sessions ranging from 40 seconds to 15 minutes, both pre- and post-exercise. Exercise interventions were not solely eccentric. Researchers deduced that pre-exercise stretching only lessened muscle soreness by 0.5 unit on a 100-unit scale ($p < 0.05$), and the reduction from post-exercise was only 1.0 unit on the same scale ($p < 0.05$).

OTHER STRATEGIES USED TO REDUCE DOMS

The effectiveness of heat therapy, cold-temperature therapy, anti-inflammatory medications, and massage have been examined for the alleviation of muscle soreness. Each therapy has demonstrated associations to reduced muscle soreness with the exception of cold-temperature therapies.

Hot/Cold Therapies

Extreme temperature therapy administration has also demonstrated DOMS-reducing effects, though evidence suggests significant results are seen with heat only. The sixteen males aged 23 +/- 3 years that participated in the study by Howatson et al. (2009) were divided into two groups post-eccentric exercise: control and cold water immersion. The latter group was instructed to immerse their legs (up to hip level) in 15°C water for 12 minutes immediately post-exercise and every subsequent day for 3 days. No significant drop in muscle soreness was recorded for the cold water immersion group when compared to the control group.

A study by Mayer et al. (2006) tested heat therapy against cold therapy in 32 mixed-gender subjects, aged 23.5 +/- 6.6 years and untrained in exercises similar to that of the intervention. Heat wrap application for 8 hours at 18 and 32 hours post-eccentric exercise (2 sets of 25 back extensions) provided 138% more pain relief at 24 hours ($p = 0.026$) and significantly more pain relief at 48 hours ($p < 0.05$) than therapy using cold pack application for 15-20 minutes, every 4 hours for 24 hours.

This study also examined heat as a soreness-prevention agent versus a soreness-treatment agent in 34 subjects who underwent the same eccentric exercise mentioned above. When heat wraps were applied for 8 hours (4 hours pre-exercise and 4hours post-exercise), subjects reported pain intensity scores 46.8% lower at 24 hours ($p < 0.001$) and significantly lower scores at 48 hours ($p < 0.05$) than those in the control group. Future experimentation is needed to identify specific temperatures or temperature ranges for effective heat therapy.

Non-Steroidal Anti-Inflammatory Drugs

Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) are of particular interest, because they act on the same pathways through which O3FAs and other PUFAs become anti-inflammatory or inflammatory eicosanoids. NSAIDS inhibit both COX pathways, thereby down regulating or blocking eicosanoid generation (Champe et al., 2008).

It is widely thought that ingestion of NSAIDS leads to pain relief; however, recent evidence does not show that NSAIDS provide relief from DOMS. Rahnama et al. (2005) recruited 44 non-athletic males aged 24.3 +/- 2.4 years, splitting them into 4 intervention groups: physical activity, ibuprofen, combination, and control. Ibuprofen intervention involved administration of a 400 mg dosage 1 hour before physical activity and continued 2 times daily with 400 mg dosages for 48 hours. Physical activity intervention consisted of 2 parts: a warm-up – five-minute jog, 10-minute static stretching session, and 5-minute submaximal concentric movement session – and specific eccentric exercise – a series of 70 eccentric actions on modified, single-arm curl machine using the non-dominant hand. Results post-intervention indicated a significant reduction ($p < 0.001$) in perceived soreness scores from the control score at 24 hours (when perceived soreness was highest) that existed only in the combination and physical activity groups. Due to presence of pre-exercise warm up and to the fact that significant outcomes were the effect of physical activity, the researchers speculate that the warm-up was the source of soreness reduction in this study.

Kreuntz et al. (2008) ran a similar protocol; however, their findings are different. Twelve males and 6 females of college age were recruited for the study. Subjects performed an eccentric exercise intervention that included 6 sets of 4-10 bicep curls on five days a week for 6 weeks, though arms were alternated each day. Supplementation involved giving subjects 400 mg of ibuprofen immediately after the daily exercise intervention, though only on days using one of their arms. After daily exercise intervention using the other arm, subjects received a placebo. Results between arms were compared at the end of the exercise intervention, and no significant differences were found between the placebo and the ibuprofen, nor were any significant changes seen in DOMS. The authors did not include whether subjects were untrained, nor did they mention whether their exercise protocol induced significant muscle soreness, so it is possible that more research is necessary to verify these findings.

Massage

Massage is yet another factor that impacts DOMS. In a study by Frey-Law et al. (2008), 43 mixed-gender subjects aged 23.3 +/- 3.5 years were placed into three treatment groups: control, superficial touch, and deep-tissue massage. After subjects attended the exercise intervention involving three sets of wrist extensor contractions with a 10-pound dumbbell, researchers waited 24-48 hours to put participants through a second massage-centered intervention, whereby they would experience massage for 6 minutes, with the intensity depending on their treatment group. Perceived pain was then tested at rest, during stretch, and with muscle contraction. Researchers found that deep-tissue massage significantly reduced pain perceived during stretch by 48.4% compared with the control group ($p < 0.05$). This treatment demonstrated no significant effects at rest or with contraction, nor was there any significant perceived pain-based effect seen in the superficial massage treatment group.

REFERENCES

Allen, D.G., Whitehead, N.P., & Yeung, E.W. (2005). Mechanisms of stretch-induced muscle damage in normal and dystrophic muscle: role of ionic changes. *The Journal of Physiology, 567*(3):723-735.

American College Health Association (2010). American College Health Association-National College Health Assessment II: Reference Group Data Report Fall 2009. Baltimore: American College Health Association.

Armstrong, LB (1990). Initial events in exercise-induced muscular injury. *Medicine & Science in Sports & Exercise, 22*(4):429–435.

Ayilavarapu, S., Kantarci, A., Fredman, G., Turkoglu, O., Omori, K., Liu, H., Iwata, T., Yagi, M., Hasturk, H., & Van Dyke, T.E. (2010). Diabetes-induced oxidative stress is mediated by ca2+-independent phospholipase a2 in neutrophils. *Journal of Immunology, 184*(3):1507-1515.

Benson, J., Wilson, A., Stocks, N., & Moulding, N. (2006). Muscle pain as an indicator of vitamin D deficiency in an urban Australian Aboriginal population. *Medical Journal of Australia, 185*(2):76-77.

Bloomer, R.J., Larson, D.E., Fisher-Wellman, K.H., Galpin, A.J., & Schilling, B.K. (2009). Effect of eicosapentaenoic and docosahexaenoic acid on resting and exercise-induced inflammatory and oxidative stress biomarkers: a randomized, placebo controlled, cross-over study. *Lipids in Health and Disease, 8*(36):36-48.

Brunt, A., Yeong, R., & Zhong, L. (2008). Differences in dietary patterns among college students according to body mass index. *Journal of American College Health, 56*(6):629-34.

Bryer, S.C. & Goldfarb, A.H. (2006). Effect of high dose vitamin C supplementation on muscle soreness, damage, function, and oxidative stress to eccentric exercise. *International Journal of Sport Nutrition and Exercise Metabolism, 16*(3):270-80.

Burke, L.M., Loucks, A.B., & Broad, N. (2006). Energy and carbohydrate for training and recovery. *Journal of Sports Sciences, 24*(7):675-85.

Champe, P.C., Harvey, R.A., & Ferrier, D.R. (2008). *Lippincott's Illustrated Reviews: Biochemistry.* 4th ed. Baltimore, MD: Lippincott Williams & Wilkins.

Chapman, D.W., Newton, M., McGuigan, M.R., & Nosaka, K. (2008). Comparison between old and young men for responses to fast velocity maximal lengthening contractions of the elbow flexors. *European Journal of Applied Physiology, 104*(3):531-539.

Cheung, K., Hume, P., & Maxwell, L. (2003). Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine, 33*(2):145– 64.

Cleary, M.A., Sweeney, L.A., Kendrick, Z.V., & Sitler, M.R. (2005). Dehydration and symptoms of delayed-onset muscle soreness in hyperthermic males. *Journal of Athletic Training, 40*(4):288-97.

Close, G.L., Ashton, T., McArdle, A., & MacLaren, D.P.M. (2005). The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comparative Biochemistry and Physiology - Part A: Molecular & Integrative Physiology, 142*(3):257-66.

Connolly, D.A., Lauzon, C., Agnew, J., Dunn, M., & Reed, B. (2006).The effects of vitamin C supplementation on symptoms of delayed onset muscle soreness. *The Journal of Sports Medicine and Physical Fitness, 46*(3):462-7.

Dannecker, E.A., Knoll, V., & Robinson, M.E. (2008). Sex differences in muscle pain: self-care behaviors and effects on daily activities. *The Journal of Pain, 9*(3):200-9.

Driskell, J. & Wolinsky, I. (2006). *Sports Nutrition: Vitamins and Trace Elements*. New York, NY: CRC/Taylor & Francis.

Duchen, M.R. (2004). Mitochondria in health and disease: perspectives on a new mitochondrial biology. *Molecular Aspects of Medicine*, *25*(4):365–451.

Frey-Law, L.A., Evans, S., Knudston, J., Nus, S., Scholl, K., & Sluka, K. (2008). Massage reduces pain perception and hyperalgesia in experimental muscle pain: a randomized, controlled trial. *Journal of Pain, 9*(8):714-21.

Gissel, H. (2005). The role of ca^{2+} in muscle cell damage. *Annals of the New York Academy of Sciences, 1066*(1):166-180.

Ha, E. & Caine-Bish, N. (2009). Effect of nutrition intervention using a general nutrition course for promoting fruit and vegetable consumption among college students. *Journal of Nutrition Education and Behavior*, *41*(2):103-109.

Herbert, R.D. & de Noronha, M. (2007). Stretching to prevent or reduce muscle soreness after exercise. *Cochrane Database of Systematic Reviews*, *1*(4):1-31.

Hinton, P.S., Sanford, T.C., Davidson, M.M., Yakushko, O.F., & Beck, N.C. (2004). Nutrient intakes and dietary behaviors of male and female collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, *14*(4):389-405.

Houston, D.K., Cesari, M., Ferrucci, L., Cherubini, A., Maggio, D., Bartali, B., Johnson, M.A., Schwartz, G.G., & Kritchevsky, S.B. (2007). Association between vitamin D and physical performance: the InCHIANTI study. *Journal of Gerontology, Series A, Biological Sciences and Medical Sciences, 62*(4):440-446.

Howatson, G., Goodall, S., & van Someren, K.A. (2009). The influence of cold water immersions on adaptation following a single bout of damaging exercise. *European Journal of Applied Physiology, 105*(4):615-21.

Ivins, D. (2006). Acute ankle sprain: an update. *American Family Physician, 74*(10):1714–20.

Jubeau, M., Sartorio, A., Marinone, P.G., Agosti, F., Van Hoecke, J., Nosaka, K., & Maffiuletti, N.A. (2008). Comparison between voluntary and stimulated contractions of the quadriceps femoris for growth hormone response and muscle damage. *Journal of Applied Physiology, 104*(1):75-81.

Kreuntz, J.R., Quest, B., Farthing, J.P., Quest, D.W., Chilibeck, P.D. (2008). The effects of ibuprofen on muscle hypertrophy, strength, and soreness during resistance training. *The Journal of Applied Physiology and Nutrition Metabolism, 33*(3):470-475.

Lavender, A.P. & Nosaka, K. (2008). A light load eccentric exercise confers protection against a subsequent bout of more demanding eccentric exercise. *Journal of Science and Medicine in Sport*, *11*(3):291-8.

Law, R.Y.W. & Herbert, R.D. (2007). Warm-up reduces delayed-onset muscle soreness but cool-down does not: a randomized controlled trial. *The Australian Journal of Physiotherapy, 53*(1):91-5.

Lenn, J., Uhl, T., Mattacola, C., Boissonneault, G., Yates, J., Ibrahim, W., & Bruckner, G (2002). The effects of fish oil and isoflavones on delayed onset muscle soreness. *Medicine & Science in Sports & Exercise, 34*(10):1605-1613.

Mackey, A.L., Bojsen-Moller, J., Qvortrup, K., Langberg, H., Suetta, C., Kalliokoski, K.K., Kjaer, M, & Magnusson, S.P. (2008). Evidence of skeletal muscle damage following electrically stimulated isometric muscle contractions in humans. *Journal of Applied Physiology, 105*(5):1620-7.

Mahan, K.L. & Escott-Stump, S. (2008). *Krause's Food & Nutrition Therapy.* 12th ed. St. Louis, MO: Saunders.

Marginson, V., Rowlands, A.V., Gleeson, N.P., & Eston, R.G. (2005). Comparison of the symptoms of exercise-induced muscle damage after an initial and repeated bout of plyometric exercise in men and boys. *Journal of Applied Physiology, 99*(3):1174-81.

Mayer, J.M., Mooney, V., Matheson, L.N., Erasala, G.N., Verna, J.L., Udermann, B.E. & Leggett, S. (2006). Continuous low-level heat wrap therapy for the prevention and early phase treatment of delayed-onset muscle soreness of the low back: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation, 87*(10):1310-7.

Mense, S. & Schmidt, R.F. (1974). Activation of group IV afferent units from muscle by algesic agents. *Brain Research, 72*(2):305–310.

Mickleborough, T.D., & Rundell, K.W. (2005). Dietary polyunsaturated fatty acids in asthma- and exercise-induced bronchoconstriction. *European Journal of Clinical Nutrition*, *59*(12):1335-1346.

Morgan, D.L. & Proske, U. (2004). Popping sarcomere hypothesis explains stretch-induced muscle damage. *Clinical and Experimental Pharmacology & Physiology, 31*(8):541-5.

Nieman, D.C., Henson, D.A., Dumke, C.L., Oley, K., McAnulty, S.R., Davis, J.M., Murphy, E.A., Utter, A.C., Lind, R.H., McAnulty, L.S., Morrow, J.D. (2006). Ibuprofen use, endotoxemia, inflammation, and plasma cytokinesduring ultramarathon competition. *Brain, Behavior, and Immunity, 20*(1):578-584.

Parekh, A.B. & Putney, J.W. (2005). Store-operated calcium channels. *Physiological Reviews*, *85*(2):757–810.

Parr, J.J., Yarrow, J.F., Garbo, C.M., & Borsa, P.A. (2009). Symptomatic and functional responses to concentric- eccentric isokinetic versus eccentric-only isotonic exercise. *Journal of Athletic Training, 44*(5):462-8.

Prasartwuth, O., Taylor, J.L., & Gandevia, S.C. (2005). Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. *The Journal of Physiology,* 567(1):337-48.

Philippou, A., Maridaki, M., & Bogdanis, G.C. (2003). Angle-specific impairment of elbow flexors strength after isometric exercise at long muscle length. *Journal of Sports Science*, *21*(10):859-65.

Pickova, J. (2009). Importance of knowledge on lipid composition of foods to support development towards consumption of higher levels of n-3 fatty acids via freshwater fish. *Physiological Research, 58*(Suppl. 1):S39-S45.

Rahnama, N., Rahmani-nia, F., & Ebrahim, K. (2005). The isolated and combined effects of selected physical activity and ibuprofen on delayed-onset muscle soreness. *Journal of Sports Sciences, 23*(8):843-50.

Shils, M.E., Shike, M., Ross, A.C., Caballero, B., & Cousins, R.J. (2006). *Modern Nutrition in Health and Disease.* 10^{th} ed. Baltimore, MD: Lippincott Williams & Wilkins.

Shimomura, Y., Yamamoto, Y., Bajotto, G., Sato, J., Murakami, T., Shimomura, N., Kobayashi, H. & Mawatari, K. (2006). Nutraceutical effects of branched-chain amino acids on skeletal muscle. *The Journal of Nutrition, 136*(Suppl. 1):529S-32S.

Silva, L.A., Pinho, C.A., Silveira, P.C.L., Tuon, T., De Souza, C.T., Dal-Pizzol, F., & Pinho, R.A. (2010). Vitamin E supplementation decreases muscular and oxidative damage but not inflammatory response induced by eccentric contraction. *The Journal of Physiological Sciences, 60(*1):51-7.

Smith, LL (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine & Science in Sports Exercise, 23*(5):542-551.

Soper, C. & Hume, P.A. (2004). Towards an ideal rowing technique for performance: the contributions from biomechanics. *Sports Medicine*, *32*(12):825-48.

Tartibian, B., Maleki, B.H., & Abbasi, A. (2009). The effects of ingestion of omega-3 fatty acids on perceived pain and external symptoms of delayed onset muscle soreness in untrained men. *Clinical Journal of Sport Medicine, 19*(2):115-119.

Tiidus, P.M. (2003). Influence of estrogen and gender on muscle damage and repair. *Exercise and Sport Sciences Reviews, 31*(1): 40–44.

CHAPTER THREE

MANUSCRIPT

The Relationship between Omega-3 Fatty Acid Intake and Muscle Soreness in Collegiate Rowers

Danica Carswell, BS, Dr. Dan Benardot, Ph.D., DHC, RD, LD, FACSM, Dr. Anita Nucci, Ph.D., RD, LD, and Dr. Walter Thompson, Ph.D., FACSM, FAACVPR

The primary objective of this study was to determine if the intake of omega-3 fatty acids (O3FA), from self-selected foods and supplements, was associated with a reduction in muscle soreness in collegiate rowers. Several groups of collegiate rowers were identified as potential subjects and, using an IRB-approved protocol, 61 volunteers from this subject pool agreed to participate in the study. At the end of the first week of fall practice, subjects were asked to provide information on age, weight, height, ethnicity, rowing level, sun exposure, exercise regimen, pre- and peak-soreness and exertion levels, hydration status, and food/supplement intake. A Talag scale was used to determine the severity of muscle soreness, and a modified Borg scale was used to determine the exertion intensity level. A food frequency questionnaire using a 10-point Likert scale was used to assess food and supplement intake, with a special focus on O3FA. Subjects with a higher intake of EPA/DHA rich bottled supplemental fish oil, taken by spoon, had significantly higher soreness levels than those with lower intakes ($p = 0.028$). Higher soreness was also significantly associated with higher intake of EPA/DHA rich fish oil capsules ($p < 0.001$). Lower soreness was significantly associated with higher fruit intake ($p = 0.030$). These findings suggest that high intakes of O3FA from fish oils, regardless of liquid or capsule form, may be contraindicated for collegiate rowers wishing to reduce muscle soreness. Recommending fruit intake may be warranted for rowers wishing to reduce muscle soreness.

Keywords: omega-3 fatty acids, nutrient intake, muscle soreness, athletes

Introduction

Muscle soreness is commonly experienced by athletes and is among several factors that contribute to poor athletic performance. In recent literature, muscle soreness has been shown to have negative effects on athletic performance, because of its association with extreme reductions in range of motion and muscle strength (Cleary et al., 2005; Parr et al., 2009). Muscle soreness referred to as “delayed-onset muscle soreness” (DOMS) is that which peaks between 24 and 48 hours post-exercise and continues 2 to 4 days (Cleary et al., 2005; Frey-Law et al., 2008; Mayer et al., 2006; Rahnama et al., 2005; Tartibian et al., 2009).

DOMS is defined as a grade I muscle strain injury which represents a stretching and/or minor tearing of the muscle, but not partial (grade II) or complete (grade III) muscle tearing (Cheung et al., 2003; Ivins, 2006). It is thought to originate from inflammation secondary to eccentric exercise (EE): Tartibian et al. (2009) found increases in limb circumference accompanied by DOMS after EE intervention. EE is defined as a form of exercise that lengthens (or stretches) the muscle with contraction (Prasartwuth et al., 2005). Lengthened contractions (versus shortened ones) have been shown to increase muscle damage, which is triggers the inflammatory response.

The inflammatory response is initiated by the calcium-mediated production of eicosanoids (prostaglandins, thromboxanes, and leukotrienes) from polyunsaturated fatty acids (PUFA), and their production is upregulated by the production of cytokines [tumor necrosis factor-alpha (TNF-alpha), interleukin-1 (IL-1), and interleukin-6 (IL-6)] in response to damage or already present inflammation (Mahan & Escott-Stump, 2008). These eicosanoids control growth factor release, protease release, and attraction of phagocytic cells in response to cell injury or damage (Mahan & Escott-Stump, 2008). This increased movement of inflammatory particles to an area of damage and edema associated with inflammation are thought to increase intramuscular pressure, stimulating the group III and IV afferent sensory receptors that terminate in the myofibrils (Smith, 1991; Armstrong, 1990). Cheung et al. (2003) showed evidence of this, finding an association between decreased DOMS-linked inflammation and lower intramuscular pressure.

The inflammatory nature of eicosanoids is determined by the fatty acids from which they are derived (Figure 1). Omega-6 fatty acids become pro-inflammatory eicosanoids, but omega-3 fatty acid (O3FA) become anti-inflammatory 3-series prostaglandins and thromboxanes via the cyclooxygenase-2 (COX-2) pathway and 5-series leukotrienes via the lipooxygenase (LOX) pathway (Shils et al., 2006). Tartibian et al. (2009) have found O3FA supplementation to significantly decrease muscle soreness.

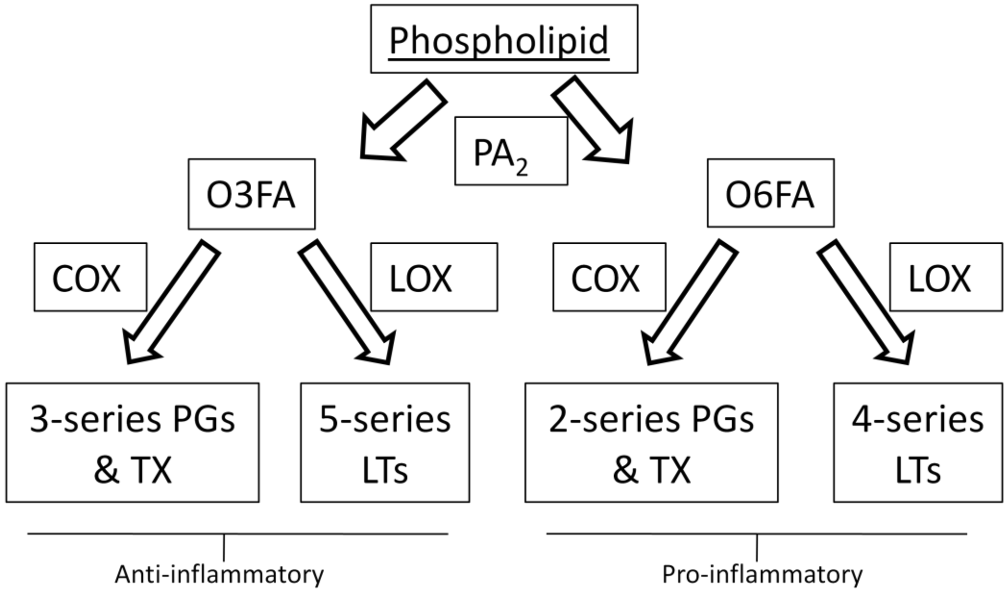

Figure 1 – Eicosanoid production (source: Champe et al.; 2008: 215).

Because O3FA have been shown to reduce muscle soreness through anti-inflammatory action, it is important to investigate their intake. Intake of O3FA in collegiate athletes is of particular interest, because they have a higher risk for developing muscle soreness than other age groups, which may reduce athletic performance (Chapman et al., 2008; Marginson et al., 2005). They have also been shown to have low intakes of O3FA-rich foods like fish, nuts, and seeds (Brunt et al., 2008). In addition, there are aspects of the rowing technique that involve inflammatory-inducing eccentric contractions vastus medialis action prevents further forward motion by stopping knee flexion at the catch of the stroke, and rectus femoris action prevents further backward motion by slowing hip extension at the stroke finish (Figure 2) (Soper & Hume, 2004).

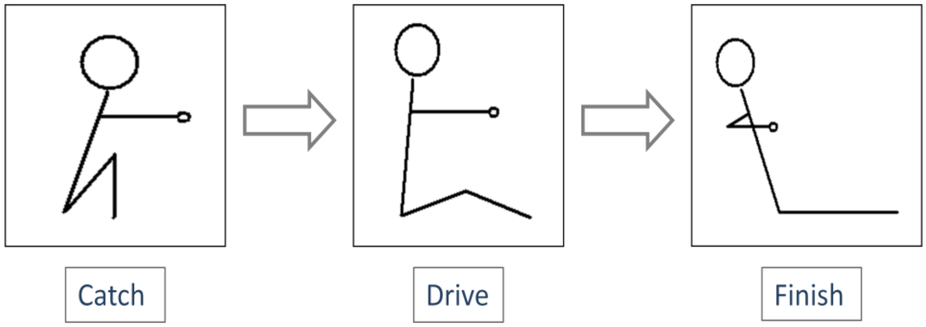

Figure 2 – The rowing technique. The catch represents the beginning of the stroke, where the oar blade is dropped into the water. The drive represents the oar being pulled through the water. The finish represents the end of the stroke, where the oar blade is lifted out of the water.

Only two studies by Lenn et al. (2002) and Tartibian et al. (2009) have recently examined the effects of O3FA on muscle soreness. Results of these studies were conflicting, and neither study dealt with athletes or, specifically, rowers. The major purpose of this study is to assess whether different levels of O3FA consumption from both foods and supplements was associated with muscle soreness in collegiate rowers. Ultimately, this information may help determine whether higher levels of O3FA intake would be useful to collegiate rowers who currently experience high levels of muscle soreness. It is hypothesized that higher O3FA intake is associated with reduced muscle soreness in collegiate rowers.

Methods

Data were collected via one-time surveys during regular Saturday team practice hours (vary by team) at Stone Mountain and Chattahoochee (Saint Andrew's Church) boathouses on August 28th, 2010. Collection was performed at the Chattahoochee boathouse pre-practice for females at 8:00 a.m. and post-practice for males at 12:00 p.m. Collection was performed at the Stone Mountain boathouse for both males and females at 11:00 a.m., though the time was post-practice for males and pre-practice for females. Investigators were not previously made aware of the order in which the different genders would practice.

Subject Recruitment and Description

The protocol was approved by the Georgia State University Institutional Review Board. Prior to, they had to acquire permission for volunteer recruitment. Collegiate rowing coaches in the local area were contacted. The coaches were informed about the study and were asked to sign permission forms that allowed surveying by the PI on one day during practice time. By signing this form, coaches also agreed that there was to be no coercion related to subject recruitment and that there would be no penalty for rowers who either did or did not participate in the study. Coaches allowed the student PI access to potential study participants on a weekend day after their first week of fall-season practice/ conditioning. Data were collected after the first week of conditioning to reduce memory bias.

Before survey administration, the student PI met with the rowers and asked if they wished to volunteer. This involved explaining the study protocol and risks/benefits. Those who volunteered were asked to review and sign a consent form. The student PI then gave consented volunteer subjects a 72-item survey that asked about anthropometric information, daily habits, dietary intake, exercise regimen, and muscle soreness (see Appendix A), but did not ask for any personally identifying information. Instructions for the survey were both written in the survey and given verbally by the student PI.

Sixty-one rowers currently competing for a collegiate team in Georgia were recruited as volunteer subjects. Exclusions included current injury (which would prevent them from training) and age under 18 years. Of the 61 rowers who filled out surveys, 13 completed every answer on the survey, though the lowest *N* for any one variable was 51.

Survey Methodology

Extensive research on factors that may contribute to muscle soreness was used to create the questions of the survey. The questions on sun exposure were part of a vitamin D survey developed by Dr. Kumaravel Rajakumar, MD, Children's Hospital of Pittsburgh of UPMC, Primary Care Center, 2010. The exertion scale used is a modified Borg rating of perceived exertion (RPE) scale, found to be the most sensitive subjective scale for overall fatigue from exercise (Grant et al., 1999). The soreness scale used is a Talag soreness scale used by the most recent research on the effects of O3FA on muscle soreness (Tartibian et al., 2009). Hydration was measured using a urine color chart that has been validated by the International Journal of Sport Nutrition, and color values were indicated as such: 1-3 represent “good hydration;” 4-6 represent “moderate hydration;” and 7-8 represent “dehydration” (Armstrong et al., 1998). The food frequency questionnaire (FFQ) uses a 10-point Likert scale, which has been validated by the journal, *Nutrition* (Swierk et al., 2010). The quantitative FFQ format using small, medium, and large serving sizes was deemed accurate for measuring group nutrient intake by the European Journal of Clinical Nutrition (Nes et al., 1992). The marine O3FA items of the FFQ were used by the American Journal of Epidemiology (Folsom & Demissie, 2004).

Statistical Analysis

The Statistical Program for the Social Sciences was used to run all data analyses (version 15.0, SPSS, Inc., Chicago IL, USA). To input data, all categorical variables were assigned numerical values. All “yes” answers were coded as “1,” and all “no” answers were coded as “2.” “Number of seasons completed on a collegiate team” responses were used to create another variable, categorizing subjects into “varsity” (rowing more than 2 seasons) and “novice” (rowing in a first or second season) before giving numerical values.

So as to test whether we could reject the null hypothesis, we ran the Pearson product-moment correlation coefficient test, one-way analysis of variance (ANOVA), paired *t*-tests, and independent *t*-tests. The Pearson product-moment correlation coefficient test was used to find correlations between perceived soreness levels and other variables. For this test, each food or supplement variable was represented by an individual FFQ score derived from multiplication of intake frequency and serving size. There were not enough strong, significant correlations between peak soreness and other variables to perform a regression analysis.

ANOVA was used to compare dietary, exertion, and soreness data. To ensure that groups had adequate data to run ANOVA and Bonferroni *post hoc* tests, it was necessary to place data into tertiles, though none of these tests proved significant. Dietary tertiles were created by first dividing food and supplement variables into plant- and fish-based types and then dividing the overall FFQ score range of each type by three. Dietary tertiles were also created for each individual food and supplement variable in the same manner. Exertion tertiles were derived from the combination of scores into the following groups: 0-2 (“nothing at all” to “light”), 3-6 (“moderate” to “more than hard”), and 7-10 (“very hard” to “very, very hard”). Soreness tertiles were derived from the combination of scores into the following groups: 1-2 (“no pain” to “dull vague ache”), 3-4 (“slight persistent pain” to “more than slight pain”), and 5-7 (“painful” to “unbearably painful”).

Paired *t*-tests were used to compare mean FFQ scores by omega-3 fatty acid source (fish versus plant) and to compare mean soreness, exertion, and hydration levels over time. Independent *t*-tests were used to compare mean peak soreness levels within each variable. When using individual food and supplement intake for independent *t*-tests, mean scores were those of the combined frequency and serving size scores used in the Pearson product-moment correlation coefficient tests. When comparing soreness of macronutrient intake, gram calculations were based off of diabetic exchanges. To compare mean frequencies of food and supplement intake by soreness level, independent *t*-tests were also utilized. Independent *t*-tests were also run to determine statistical significance of the subject characteristic differences between genders. Statistical significance was accepted at p =< 0.05, and equal variances were not assumed within parametric tests. Significance (p =< 0.05) of a *t*-test or ANOVA signifies rejection of the null hypothesis.

Results

Subject Characteristics

Subject characteristics include anthropometric data, gender, rowing level, and ethnicity.

Anthropometric Data

Table 1 Anthropometric data of collegiate rowers

Variable	*N*	Female, mean (SD)	Male, mean (SD)
Age (y)	59	20.4 (1.20)	20.0 (1.37)
Height (in)	60	67.9 (2.90)	73.3 (2.34)
Weight (lb)	60	149.5 (20.67)	175.0 (17.49)
BMI (kg/m^2)	60	22.7 (2.00)	22.9 (1.89)

Note. BMI = body mass index; SD = standard deviation

Subject anthropometric data are shown in Table 1. The mean age of males was 20.0 years with a standard deviation of 1.37 years. The mean age of females was 20.4 years with a standard deviation of 1.20 years. Mean height of males was 73.3 inches and mean weight of subjects was 175.0 pounds, giving males a mean BMI of 22.9 kg/m^2. Mean height of females was 67.9 inches and mean weight of subjects was 149.5 pounds, giving females a mean BMI of 22.7 kg/m^2. There were no significant anthropometric differences between genders.

Gender and Rowing Level

Thirty-two males and 29 females volunteered for this study. Of those rowers, 44 were of varsity level (signifying having rowed more than 2 seasons), and 14 were of novice level (signifying having rowed fewer than 2 seasons). There was no significant difference in rowing level between genders. Neither gender nor rowing level had significant effects on muscle soreness in this study.

Ethnicity

A majority of subjects (83.6%) were of the non-Hispanic Caucasian ethnicity. There were no subjects of the non-Hispanic Black or American Indian ethnicities.

Subject Daily Habits

Subject daily habits include sun exposure, hydration status throughout practice time, and use of soreness-reducing strategies.

Sun Exposure

A majority of subjects (62.3%, all male) typically had their torso exposed to the sun. More than 95% of subjects typically had their arms, hands, and legs exposed. Mean length of sun exposure per day was “2-2.5 hours.” Mean SPF of sunscreen used most was “1-15 SPF.” None of the sun exposure variables were found to be factors of muscle soreness.

Hydration Status

Table 2 Hydration status of collegiate rowers measured before, during, and after practice

Status	Before, *n* (%)	During, *n* (%)	After, *n* (%)
Good	51 (83.6)	48 (78.7)	33 (54.1)
Moderate	9 (14.8)	10 (16.4)	28 (45.9)
Dehydration	1 (1.6)	0 (0.0)	0 (0.0)

Note. Hydration status was determined by a validated, 8-color urine color chart with the first 3 colors representing "good," the second 3 colors representing "moderate," and the last 2 colors representing "dehydration."

Subjects' hydration status data is presented in Table 2. The percentage of subjects with good hydration status dropped from 83.6% before practice to 54.1% after practice, where they fell into moderate hydration status. Only one subject reported dehydration based on urine color, but only before practice, not during or after practice. There were no significant differences in soreness levels during the first week of practice based on hydration status.

When hydration status was compared by time, the mean urine color score was significantly higher post-practice than pre-practice ($p = 0.001$). This indicates that rowers were more dehydrated after practice, though both mean urine color scores fell under the category of "good hydration."

Use of Soreness-Reducing Strategies

Seventy-two percent of subjects did not use massage, 88.5% did not use heat, 88.5% did not use ice, and 62.3% did not use NSAIDS to relieve muscle soreness; however, the strategy used by the most subjects to reduce muscle soreness was the use of NSAIDS.

There were no significant differences in soreness levels found between the use and non-use of these strategies.

Subject Nutritional Intake

Subject intake information includes the influence of school nutrition services, the frequency of specific food group intake, the frequency of specific types of O3FA-rich foods and supplements, and the relationships between these factors and soreness levels.

Dietary Correlations to Peak Soreness Levels

Table 3 Dietary correlations to peak soreness levels of collegiate rowers

Variable	*N*	r (*p*)
Use of school nutrition services	29	0.262 (0.170)
Non-fish meat consumption	58	-0.143 (0.283)
Grain consumption	58	0.002 (0.990)
Vegetable consumption	57	0.146 (0.278)
Fruit consumption	58	-0.226 (0.088)
Fast food consumption	53	-0.017 (0.905)
Dark fish consumption	56	-0.113 (0.412)
Canned tuna consumption	55	-0.035 (0.799)
Other fish consumption	55	0.036 (0.792)
Crustacean consumption	56	0.066 (0.630)
Nuts/seeds consumption	57	-0.036 (0.791)
Flaxseed consumption	56	0.044 (0.746)
Plant oil consumption	58	0.047 (0.726)
Flaxseed oil consumption	55	-0.124 (0.366)
Fish oil capsule consumption	51	0.138 (0.339)
DHA supplement consumption	50	-0.095 (0.511)
Bottled fish oil consumption	55	0.176 (0.199)

Note. r = Pearson's correlation coefficient; *p* = probability (two-tailed); peak soreness levels refer to the highest perceived soreness during the first week of practice; dark fish include mackerel, salmon, sardines, bluefish, fresh tuna, and swordfish; bottled fish oil refers to free, non-encapsulated oil (i.e., cod liver oil).

Dietary correlations to soreness levels are shown in Table 3. No significant correlations were found between food consumption (regular or O3FA-based) and peak soreness levels during the first week of practice. There was no correlation between nutrition education via use of nutrition services and peak soreness levels.

Presence and Use of Free or Low-Cost Nutrition Services

A majority of subjects (52.5%) were not sure whether they had access to free or low-cost nutrition services at school. Of those subjects who were aware that their school offered nutrition services, a majority (79.3%) did not utilize them.

When O3FA-rich food intake was compared based on subject use of free or low-cost school nutrition services, there was no significant difference found between the O3FA-rich food intakes of rowers who did or did not use the free or low-cost nutrition services provided to them by their schools, nor was any significant difference found between use or non-use of school nutrition services and peak soreness during the first week of practice.

Frequency of Nutrient Intake

Frequencies of both O3FA-rich and non-O3FA-rich foods and supplements were examined.

Frequency of O3FA-Rich Intake

Table 4 Frequency of plant-based, omega-3-rich food consumption in collegiate rowers

Frequency	Flaxseed *n* (%)	Nuts/seeds *n* (%)	Flaxseed oil *n* (%)	Plant oil *n* (%)
<1x/month	47 (77.0)	6 (9.8)	49 (80.3)	9 (14.8)
1x/month	1 (1.6)	1 (1.6)	5 (8.2)	2 (3.3)
2x/month	0 (0)	7 (11.5)	1 (1.6)	0 (0)
3x/month	2 (3.3)	4 (6.6)	2 (3.3)	6 (9.8)
1x/week	3 (4.9)	5 (8.2)	0 (0)	12 (19.7)
2x/week	3 (4.9)	10 (16.4)	2 (3.3)	10 (16.4)
3x/week	3 (4.9)	14 (23.0)	0 (0)	8 (13.1)
Every other day	1 (1.6)	5 (8.2)	1 (1.6)	9 (14.8)
1x/day	1 (1.6)	6 (9.8)	1 (1.6)	5 (8.2)
2x+/day	0 (0)	3 (4.9)	0 (0)	0 (0)

The frequency of plant-based O3FA-rich food consumption is shown in Table 4. A majority of subjects consumed flaxseed (77.0%) and flaxseed oil (80.3%) less than once per month. More rowers consumed nuts and seeds 3 times a week (23.0%) and consumed plant oil once a week (19.7%) compared to other frequencies.

Table 5 Frequency of fish-based, omega-3-rich food consumption in collegiate rowers

Frequency	Dark fish *n* (%)	Canned tuna *n* (%)	Other fish *n* (%)	Crustaceans *n* (%)
<1x/mo	30 (49.2)	41 (67.2)	37 (60.7)	40 (65.6)
1x/mo	8 (13.1)	7 (11.5)	9 (14.8)	7 (11.5)
2x/mo	11 (18.0)	6 (9.8)	6 (9.8)	7 (11.5)
3x/mo	3 (4.9)	5 (8.2)	3 (4.9)	4 (6.6)
1x/wk	7 (11.5)	1 (1.6)	5 (8.2)	3 (4.9)
2x/wk	1 (1.6)	1 (1.6)	1 (1.6)	0 (0)
3x/wk	0 (0)	0 (0)	0 (0)	0 (0)
Every other day	0 (0)	0 (0)	0 (0)	0 (0)
1x/d	0 (0)	0 (0)	0 (0)	0 (0)
2x+/d	0 (0)	0 (0)	0 (0)	0 (0)

Note. Dark fish include mackerel, salmon, sardines, bluefish, fresh tuna, and swordfish.

The frequency of fish-based O3FA-rich food consumption is presented in Table 5. A majority of rowers consumed fish-based omega-3 foods less than once per month. No subjects consume these foods more than 2 times per week.

Table 6 Frequency of omega-3-rich supplement consumption in collegiate rowers

Frequency	Fish oil capsules *n* (%)	DHA capsules *n* (%)	Bottled fish oil *n* (%)
<1x/month	53 (86.9)	59 (96.7)	53 (86.9)
1x/month	1 (1.6)	0 (0)	1 (1.6)
2x/month	1 (1.6)	0 (0)	2 (3.3)
3x/month	0 (0)	0 (0)	3 (4.9)
1x/week	0 (0)	0 (0)	1 (1.6)
2x/week	0 (0)	0 (0)	1 (1.6)
3x/week	2 (3.3)	0 (0)	0 (0)
Every other day	0 (0)	1 (1.6)	0 (0)
1x/day	2 (3.3)	0 (0)	0 (0)
2x+/day	0 (0)	0 (0)	0 (0)

Note. Bottled fish oil refers to free, non-encapsulated oil (i.e., cod liver oil).

The frequency of O3FA-rich supplement consumption is shown in Table 6. Subjects were surveyed on consumption of both liquid and encapsulated supplements. A majority of subjects consume bottled (86.9%) and encapsulated (86.9%) fish oils and DHA capsules (96.7%) less than once per month.

Frequency of Non-O3FA-Rich Intake

More subjects (27.9%) consumed non-fish meat once per day compared to other frequencies. A majority of subjects (54.1%) consumed grains 2 or more times per day. More rowers consumed fruits (36.1%) and vegetables (34.4%) 2 or more times (each) per day compared to other frequencies. More rowers (37.7%) consumed fast food less than once per month compared to other frequencies.

Breakdown of Subject O3FA Intake by Source

Rowers consumed significantly more plant-derived O3FA-rich foods than fish-derived ($p < 0.001$). The mean FFQ score for plant-based O3FA was 24.4, while that of fish-based O3FA was 14.4. FFQ scores represent the average serving size combined with the average frequency.

Breakdown of Subject Intake by Macronutrient

The average rower consumed 18.7 g of protein (standard deviation of 13.5 g), 2.0 g of fat (standard deviation of 3.8 g), and 108.3 g of carbohydrate (standard deviation of 45.3 g) per day. There were no significant differences found between individual macronutrient intakes of rowers with above- and below-average peak soreness levels.

Associations between Soreness Levels and O3FA-Rich Intake

There were no significant differences found between peak soreness levels above and below the mean fish-based O3FA-rich food intake. There were also no significant differences found between fish-based O3FA-rich food intake levels above or below the mean soreness level.

There were no significant differences found between peak soreness levels above and below the mean plant-based O3FA-rich food intake. There were also no significant differences found between plant-based O3FA-rich food intake levels above or below the mean soreness level.

Table 7 Comparison of peak soreness levels based on above- and below-average omega-3-rich supplement intake in collegiate rowers

Food type	***n***	**Intake**	**Soreness mean (SD)**	***p***
Fish oil capsules	10	>= 838.00	4.10 (1.523)	0.524
	45	< 838.00	3.76 (1.417)	
DHA capsules	1	>= 1.00	3.00 (n/a)	*****
	49	< 1.00	3.979 (1.464)	
Bottled fish oil	4	>= 1.31	4.75 (0.500)	**0.026**
	46	< 1.31	3.87 (1.470)	

Note. SD = standard deviation; *p* = probability; peak soreness levels refer to the highest perceived soreness during the first week of practice; supplemental fish oil refers to free, non-encapsulated oil (i.e., cod liver oil); only 1 subject consumed DHA capsules, making comparative statistical analysis impossible; capsule intake was based on milligram amount and frequency of consumption, while bottled fish oil intake was based on number of teaspoons and frequency of consumption.

A comparison of peak soreness levels based on above- and below-average O3FA-rich supplement intake is presented in Table 7. Rowers who consumed bottled fish oil above the mean had significantly higher mean peak soreness levels ($p = 0.026$). Average intake of bottled fish oil was represented by a 1 teaspoon serving size consumed at a frequency of less than once per month.

Table 8 Comparison of omega-3-rich supplement intake frequencies based on above- and below-average peak soreness levels in collegiate rowers

Food type	***n***	**Soreness level**	**Frequency mean (SD)**	***p***
Fish oil capsules	33	>= 3.92	1.94 (2.344)	**0.028**
	26	< 3.92	1.00 (<0.001)	
DHA capsules	34	>= 3.92	1.21 (1.200)	0.325
	26	< 3.92	1.00 (<0.001)	
Bottled fish oil	34	>= 3.92	1.65 (1.368)	**0.015**
	27	< 3.92	1.04 (0.192)	

Note. SD = standard deviation; *p* = probability; peak soreness levels refer to the highest perceived soreness during the first week of practice; supplemental fish oil refers to free, non-encapsulated oil (i.e., cod liver oil).

The mean frequency of O3FA-rich supplement intake in rowers with above- and below-average peak soreness levels is presented in Table 8. Rowers who experienced above-average peak soreness levels during the first week of practice had significantly higher frequencies of fish oil capsule intake ($p < 0.028$) and bottled fish oil intake ($p < 0.015$) than those with below-average levels.

A significant difference between fish oil capsule intakes for above- and below-average soreness levels was not found when frequency was combined with serving sizes, and the same was found between bottled fish oil intakes. DHA capsules did not show significant intake frequency differences above and below mean soreness levels.

Associations between Soreness Levels and Non-O3FA-Rich Intake

Table 9 Comparison of non-omega-3-rich food intake frequencies based on above- and below-average peak soreness levels in collegiate rowers

Food type	*n*	Soreness level	Frequency mean (SD)	*P*
Non-fish meat	34	>= 3.92	7.82 (2.455)	0.894
	27	< 3.92	7.74 (2.363)	
Grains	33	>= 3.92	9.06 (1.248)	0.206
	27	< 3.92	9.41 (0.844)	
Fruits	34	>= 3.92	8.32 (1.902)	**0.030**
	27	< 3.92	9.15 (0.907)	
Vegetables	34	>= 3.92	8.65 (1.455)	0.871
	27	< 3.92	8.70 (1.265)	
Fast food	34	>= 3.92	3.12 (2.100)	0.551
	27	< 3.92	2.81 (1.841)	

Note. SD = standard deviation; *p* = probability; peak soreness levels refer to the highest perceived soreness during the first week of practice.

The mean frequency of non-O3FA-rich food intake in rowers with above- and below-average peak soreness levels is presented in Table 9. Rowers who experienced above-average peak soreness levels during the first week of practice had a significantly lower frequency of fruit intake than those with below-average levels ($p = 0.030$). A significant difference between fruit intakes for above- and below-average soreness levels was not found when frequency was combined with serving sizes. No other non-O3FA-rich foods in this study showed significant intake frequency differences above and below mean soreness levels. There were no significant differences found between peak soreness levels above and below the mean non-O3FA-rich food intake.

Subject Exercise Regimen

Subject exercise regimen data consist of warm-up and practice regimen and participation including performance of eccentric exercises and exertion levels. The relationships between these factors and subject soreness levels are also examined.

Table 10 Age, gender, and exercise correlations to peak soreness levels of collegiate rowers

Variable	*N*	r (*p*)
Age	59	-0.161 (0.224)
Gender	61	-0.130 (0.317)
Summer training	61	0.063 (0.631)
Warm-up performance	61	-0.214 (0.098)
Pre-practice stretching	61	-0.067 (0.607)
Post-practice stretching	61	-0.202 (0.118)
Number of practices/week	61	0.137 (0.292)
Practice' time of day	60	0.108 (0.410)
Time length of practice	61	**0.267 (0.038)**
Time length of warm-up	61	0.035 (0.787)
Performing backward, downhill running	61	0.119 (0.360)
Performing bicep curls	61	0.116 (0.372)
Performing lunges	61	**-0.257 (0.045)**
Performing back extensions	61	-0.053 (0.684)
Performing forward, downhill running	61	0.051 (0.697)
Performing squats	61	-0.226 (0.080)
Performing step-ups	61	-0.232 (0.072)
Performing wrist extensions	61	-0.099 (0.450)
Pre-season exertion level	61	-0.092 (0.483)
Peak practice exertion level	61	**0.439 (<0.001)**

Note. r = Pearson's correlation coefficient; *p* = probability (two-tailed); peak soreness levels refer to the highest perceived soreness during the first week of practice.

Age, gender, and exercise correlations to soreness levels are shown in Table 10. A moderate, positive relationship exists between peak soreness levels and peak exertion levels of rowers ($p < 0.001$). There is a weak, positive relationship between rowing practice length and peak soreness levels of rowers ($p = 0.038$).

A weak, negative relationship exists between performing lunges by rowers during the first week of rowing practice and peak soreness of rowers ($p = 0.045$). Performance of specific exercises was entered as either 1 (yes) or 2 (no), so lower values signify that the exercise was done.

Subject Warm-up and Practice Regimen

A majority of subjects reported typically performing a warm-up (90.2%), pre-practice stretching (72.1%), and post-practice stretching (73.8%).

Peak soreness levels during the first week of practice were significantly higher in rowers who performed a warm-up versus rowers who did not ($p = 0.039$). Rowers who performed warm-ups had a mean soreness level of 4.0, while those who did not perform warm-ups had a mean soreness level of 3.0.

In this sample, an average rowing practice was held in the afternoon/ evening time about six times a week for a mean time of 100.7 minutes (standard deviation of 27.89 minutes). Of those who indicated performing a warm-up, subjects' average warm-up length was 9.5 minutes with a standard deviation of 5.12 minutes.

Peak soreness levels during the first week of practice were significantly higher in rowers whose practice lengths were above the mean (100.7 minutes) versus below the mean ($p < 0.026$). Rowers with above-average practice lengths had a mean soreness level of 4.38 with a standard deviation of 1.329, while those who had below-average practice lengths had a mean soreness level of 3.57 with a standard deviation of 1.420. Warm-up length had no significant effects on muscle soreness.

In the first week of practice, a majority of subjects reported not participating in backward and downhill running (96.7%), bicep curls (59.0%), back extensions (54.1%), forward and downhill running (77.0%), step-ups (80.3%), and wrist extensions (98.4%). A majority of subjects did report participation in lunges (59.0%) and squats (65.6%) in the first week of practice.

Table 11 Comparison of peak soreness levels based on eccentric exercise performance of collegiate rowers

Exercise performed	*n*	Mean soreness level	*t*	*P*
Backward, downhill running	2	3.000	-0.423	0.718
No backward, downhill running	59	3.949		
Bicep curls	25	3.720	-0.930	0.356
No bicep curls	36	4.056		
Lunges	36	4.222	2.049	**0.046**
No lunges	25	3.480		
Back extensions	28	4.000	0.405	0.687
No back extensions	33	3.849		
Forward, downhill running	14	3.786	-0.344	0.735
No forward, downhill running	47	3.957		
Squats	40	4.150	1.741	0.090
No squats	21	3.476		
Step-ups	12	4.583	2.097	**0.049**
No step-ups	49	3.755		
Wrist extensions	1	5.000	0.761	*****
No wrist extensions	60	3.900		

Note. p = probability; two-tailed significance; peak soreness levels refer to the highest perceived soreness during the first week of practice; only 1 subject performed wrist extensions, making comparative statistical analysis impossible.

A comparison of peak soreness levels based on performance of specific eccentric exercises is shown in Table 11. Those rowers who performed either lunges or step-ups had significantly higher soreness levels during the first week of practice than those who did not ($p < 0.05$). No significant differences were found for any other eccentric exercise.

Subject Soreness Levels

Table 12 Frequency of collegiate rowers with different pre-season and peak season soreness levels

Soreness levels	Pre-season, *n* (%)	Peak season, *n* (%)
1-No pain	44 (72.1)	2 (3.3)
2-Dull vague ache	8 (13.1)	8 (13.1)
3-Slight persistent pain	4 (6.6)	17 (27.9)
4-More than slight pain	2 (3.3)	11 (18.0)
5-Painful	1 (1.6)	12 (19.7)
6-Very painful	1 (1.6)	11 (18.0)
7-Unbearably painful	0 (0.0)	0 (0.0)

Note. Pre-season soreness levels refer to perceived soreness the day before the first day of practice; peak soreness levels refer to the highest perceived soreness during the first week of practice.

Subject pre-season and peak season soreness levels are presented in Table 12. A majority (72.1%) of subjects perceived no pain pre-season. More subjects (27.9%) perceived "slight persistent pain" in the first week of practice than any other level. Between the preseason and the first week of practice, 14.7% more rowers reported "more than slight pain" soreness, 18.1 % more reported "painful" soreness, and 16.4% more reported "very painful" soreness. No subjects perceived "unbearably painful" soreness.

Soreness levels were significantly higher during the first week of practice than in the preseason ($p < 0.001$). The mean preseason soreness level was 1.5, while the mean peak soreness level in the first week of practice was 3.9.

Subject Exertion Levels

Table 13 Frequency of collegiate rowers with different pre-season and peak season exertion levels

Exertion levels	Pre-season, *n* (%)	Peak season, *n* (%)
1-Nothing at all	8 (13.1)	0 (0.0)
2-Very, very light	0 (0.0)	0 (0.0)
3-Very light	3 (4.9)	0 (0.0)
4-Light	5 (8.2)	4 (6.6)
5-Moderate	14 (23.0)	1 (1.6)
6-Somewhat hard	10 (16.4)	6 (9.8)
7-Hard	9 (14.8)	6 (9.8)
8	4 (6.6)	8 (13.1)
9-Very hard	7 (11.5)	15 (24.6)
10	0 (0.0)	11 (18.0)
11	0 (0.0)	7 (11.5)
12-Very, very hard	1 (1.6)	3 (4.9)

Note. Pre-season exertion levels refer to average perceived exertion during the week before the first day of practice; peak exertion levels refer to average perceived exertion during the first week of practice.

Subject pre-season and peak season exertion levels are presented in Table 13. Eight subjects (13.1%) perceived no exertion level in the preseason, and there were no subjects who perceived no exertion level during the first week of practice. More subjects (23.0%) perceived moderate exertion levels compared to other levels. More subjects (13.1%) perceived very hard exertion levels during the first week of practice than in the preseason. Only 1 subject (1.6%) perceived very, very hard exertion in the preseason, and 3 subjects (4.9%) perceived this same level during the first week of practice.

Mean exertion levels were significantly higher during the first week of practice than in the preseason ($p < 0.001$). The mean preseason exertion level was 3.7, while that of the first week of practice was 6.5.

A comparison of mean peak soreness levels above and below mean exertion levels is shown in Table 36. During the first week of practice, mean soreness levels are significantly higher in rowers with above-average exertion levels ($p < 0.001$). No significant differences in soreness levels were found for pre-season exertion levels.

Summary of Major Findings

Mean peak soreness levels during the first week of practice were found to be nearly 2.5 levels higher than in the pre-season ($p < 0.001$). Intake of O3FA-rich foods had no effect on soreness levels. Lower peak soreness levels were significantly associated with below-average bottled fish oil intake ($p = 0.026$), and lower frequencies of fish oil capsule ($p = 0.028$) and bottled fish oil ($p = 0.015$) intakes were significantlyassociated with below-average peak soreness levels. Intake of non-O3FA-rich foods had a reducing effect on soreness levels: those subjects with below-average peak soreness levels were significantly associated with higher frequency of fruit intake ($p = 0.030$). Concerning nutrition education, a majority of subjects did not utilize school-offered nutrition counseling and no significant effects were seen in between nutrition counseling, O3FA-rich food intakes, and mean peak soreness levels.

Significantly higher mean peak soreness levels were found in subjects who had above-average practice lengths ($p = 0.026$), who performed pre-practice warm-ups ($p = 0.039$), who performed lunges during practice (p = 0.046), who performed step-ups during practice ($p = 0.049$), and who had above-average exertion levels in the first week of practice ($p < 0.001$).

Discussion

In this study, the dietary intake and exercise regimen of collegiate rowers were examined to determine the effects, if any, of the consumption of food and supplemental O3FA on delayed-onset muscle soreness in rowers. To induce muscle soreness in these athletes, performance of unaccustomed eccentric exercise was necessary. Between the preseason and the first week of practice, perceived soreness levels in rowers significantly increased. Dietary and other factors were shown to have various effects on these levels. There were no differences found between genders or rowing levels.

A significant increase in peak soreness levels was accompanied by a significantly higher intake of plant-based O3FA-rich foods than fish-based ones; however, no individual plant-based food significantly affected perceived soreness levels, and no significant correlation was found between plant-based O3FA-rich food intake and soreness levels. Plant-based O3FA-rich foods contain the fatty acid alpha-linolenic acid (ALA), which must be converted to EPA and DHA to be used to create anti-inflammatory eicosanoids. There is no current literature on the effects of ALA on muscle soreness. The issue is that the conversion rates of ALA to EPA and DHA are 0.29% and <0.01%, respectively (Hussein et al., 2005), so a large amount of ALA must be consumed to have similar effects of EPA and DHA. Even if ALA were consumed in large amounts, its individual beneficial effects do not match those of EPA and DHA (Mozaffarian et al., 2005), though the effects of EPA and DHA on muscle soreness are currently mixed.

These plant-based findings are not coherent with literature on vitamin E, commonly found in plant-based O3FA-rich foods – plant oils, nuts, and seeds. Silva et al. (2010) found significantly lower soreness scores in subjects who were supplemented with 800 IU for 2 weeks pre-exercise intervention and 1 week post-exercise intervention compared with those in the placebo group.

Fish-based O3FA-rich supplements were shown to have a small effect on perceived muscle soreness, though not ameliorating. Rowers with below-average consumption (n = 46) of bottled fish oil were found to have significantly lower peak muscle soreness than those with above-average consumption (n = 4). Average intake of supplemental fish oil in this study was about 1 teaspoon taken less than 1 time per month. The accuracy of these results is questionable, as the subject number of each group is very different. Compared with rowers who perceived below-average peak soreness levels, those with above-average levels in the first week of practice were found to have a significantly higher frequency of fish oil capsule and bottled fish oil intakes – almost 1 time per month versus less than 1 time per month – though intakes of fish oil capsules and supplemental fish oil were not significant when serving size was factored in. These results are contrary to that of Tartibian et al. (2009), who found significant decreases in muscle soreness after supplementing 324 mg EPA and 216 mg DHA once per day for 30 days pre-exercise intervention and for 2 days post-exercise intervention.

DHA-only supplementation did not have a significant impact on soreness in this study, even after examining overall O3FA-rich supplement effects when combined with the fish oil supplements. Nor were there any significant effects on perceived muscle soreness by other O3FA-rich foods caused significant effects, which is similar to the findings of Lenn et al. (2002). These researchers found no significant differences in subject perceived soreness after supplementing with 1.8 g of fish oil for 30 days prior to exercise protocol and in the week during the exercise protocol.

Non-O3FA-rich foods were also found to have a small effect on perceived soreness levels. Compared with rowers who perceived below-average peak soreness levels, those with above-average levels had a significantly lower frequency of fruit intake – every other day versus 1 time per day – though intake of fruit was not significant when serving size was factored in. One explanation for this could be vitamin C content. A 2006 study by Bryer and Goldfarb found significantly lower perceived pain scores in subjects supplemented with 3 grams of Vitamin C per day for 2 weeks prior to soreness-inducing protocol and during the 4 days following the exercise protocol compared with those who took a placebo; however, another study from the same year with a similar supplementation protocol did not find significant effects on muscle soreness (Lauzon et al., 2006).

Exercise factors were not associated with reduced muscle soreness. Rowers who had longer practice lengths, who had higher exertion levels, who performed eccentric lunges and step-ups, and who performed warm-ups in the first week of practice were all found to have significantly higher soreness levels than rowers with shorter practices and who did not perform warm-ups. A study by Law and Herbert (2007) proved opposing effects of warm-up on muscle soreness. In their study, those subjects who participated in a 10-minute warm-up recorded significantly lower mean soreness scores than those who only performed a 10-minute cool-down. There is currently no literature on the length of exercise bouts and muscle soreness, though it is possible that longer bouts allow time for additional eccentric exercise to be performed.

Concerning other soreness-reducing strategies, no significant differences in mean peak soreness levels were found in those who used hot/cold therapies, massage, or NSAIDS. The lack of effects by NSAIDS is interesting in that they work on the same pathways through which O3FA function (Champe et al., 2008). Though intakes of O3FA and NSAIDS were low compared with other food/supplement intake, if neither NSAIDS nor O3FA had an ameliorating effect on muscle soreness, perhaps this indicates that alleviation occurs through other mechanisms.

Several limitations should be taken into consideration regarding the evidence in this study. First, the rowers should have been surveyed at the same time in relation to practice time: all subjects should either be surveyed before or after the practice session. Instead, all females were surveyed before practice, and all males were surveyed after practice, all due to researcher miscommunication in scheduling. Second, the FFQ was very limiting, and did not allow enough serving sizes or frequencies of consumption from which subjects could choose. Another issue was that vitamins and dairy products were not included on the FFQ, which may have provided more insight into subject nutrition status.

In the future, rowers should be asked to keep an exercise record in the first week of practice and record daily soreness levels to see a proper soreness trend and reduce memory bias. Supplementation of O3FA in addition to the FFQ would also give more accurate results. In addition, rowers should be asked why they do or do not take supplements to determine the beliefs of their population and whether it has an effect on O3FA intake. Subjects should also be asked to record daily fluid intake, especially in regards to before, during, and after practice, which may be a better indicator of hydration than the urine color chart. Lastly, this study focuses on delayed-onset muscle soreness, and while the methodology would have to be changed, it may be interesting to examine effects of O3FA on soreness caused by overtraining, of which there currently minimal evidence.

In summary, overall intake of O3FA had minimal effects on delayed-onset muscle soreness in this study, though intake of fish oil, bottled or capsule form, may be contraindicated in collegiate rowers wishing to reduce muscle soreness. These conclusions may be applied to a larger population and suggest it may be unwise to recommend O3FA supplements to any type of collegiate athlete, not only rowers, or possibly even to recreationally active people of college age for the specific purpose of muscle soreness reduction; however, it may be warranted to recommend fruit intake for college-age populations wishing to reduce muscle soreness. Further research is needed to validate these findings and subsequent assumptions.

Acknowledgments

The authors would like to thank the rowers who volunteered to participate in the study and the coaches who gave up a portion of practice time to allow for our recruitment and surveying of athletes.

References

Allen, D.G., Whitehead, N.P., & Yeung, E.W. (2005). Mechanisms of stretch-induced muscle damage in normal and dystrophic muscle: role of ionic changes. *The Journal of Physiology, 567*(3):723-735.

Armstrong, L.E., Soto, J.A., Hacker, F.T., Casa, D.J., Kavouras, S.A., & Maresh, C.M. (1998). Urinary indices during dehydration, exercise, and rehydration. *International Journal of Sport Nutrition, 8*(4):345-355.

Ayilavarapu, S., Kantarci, A., Fredman, G., Turkoglu, O., Omori, K., Liu, H., Iwata, T., Yagi, M., Hasturk, H., & Van Dyke, T.E. (2010). Diabetes-induced oxidative stress is mediated by ca2+-independent phospholipase a2 in neutrophils. *Journal of Immunology, 184*(3):1507-1515.

Bryer, S.C. & Goldfarb, A.H. (2006). Effect of high dose vitamin C supplementation on muscle soreness, damage, function, and oxidative stress to eccentric exercise. *International Journal of Sport Nutrition and Exercise Metabolism, 16*(3):270-80.

Champe, P.C., Harvey, R.A., & Ferrier, D.R. (2008). *Lippincott's Illustrated Reviews: Biochemistry*. 4^{th} ed. Baltimore, MD: Lippincott Williams & Wilkins.

Chapman, D.W., Newton, M., McGuigan, M.R., & Nosaka, K. (2008). Comparison between old and young men for responses to fast velocity maximal lengthening contractions of the elbow flexors. *European Journal of Applied Physiology, 104*(3):531-539.

Cheung, K., Hume, P., & Maxwell, L. (2003). Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine, 33*(2):145– 64.

Cleary, M.A., Sweeney, L.A., Kendrick, Z.V., & Sitler, M.R. (2005). Dehydration and symptoms of delayed-onset muscle soreness in hyperthermic males. *Journal of Athletic Training, 40*(4):288-97.

Duchen, M.R. (2004). Mitochondria in health and disease: perspectives on a new mitochondrial biology. *Molecular Aspects of Medicine*, *25*(4):365–451.

Folsom, A.R. & Demissie, Z. (2004). Fish intake, marine omega-3 fatty acids, and mortality in a cohort of postmenopausal women. *American Journal of Epidemiology, 160*(10):1005-1010.

Frey-Law, L.A., Evans, S., Knudston, J., Nus, S., Scholl, K., & Sluka, K. (2008). Massage reduces pain perception and hyperalgesia in experimental muscle pain: a randomized, controlled trial. *Journal of Pain, 9*(8):714-21.

Gissel, H. (2005). The role of ca^{2+} in muscle cell damage. *Annals of the New York Academy of Sciences, 1066*(1):166-80.

Grant, S., Aitchison, T., Henderson, E., Christie, J., Zare, S., McMurray, J., & Dargie, H. (1999). A comparison of the reproducibility and the sensitivity to change of visual analogue scales, borg scales, and likert scales in normal subjects during submaximal exercise. *Chest, 116*(5):1208-1217.

Hussein, N., Ah-Sing, E., Wilkinson, P., Leach, C., Griffin, B.A., & Millward, D.J. (2005). Relative rates of long chain conversion of 13C linoleic and a-linolenic acid in response to marked changes in their dietary intake in male adults. *Journal of Lipid Research, 46*(2), 269–280.

Ivins, D. (2006). Acute ankle sprain: an update. *American Family Physician, 74*(10):1714–20.

Law, R.Y.W. & Herbert, R.D. (2007). Warm-up reduces delayed-onset muscle soreness but cool-down does not: a randomized controlled trial. *The Australian Journal of Physiotherapy, 53*(1):91-5.

Lenn, J., Uhl, T., Mattacola, C., Boissonneault, G., Yates, J., Ibrahim, W., & Bruckner, G (2002). The effects of fish oil and isoflavones on delayed onset muscle soreness. *Medicine & Science in Sports & Exercise, 34*(10):1605-1613.

Mahan, K.L. & Escott-Stump, S. (2008). *Krause's Food & Nutrition Therapy.* 12th ed. St. Louis, MO: Saunders.

Marginson, V., Rowlands, A.V., Gleeson, N.P., & Eston, R.G. (2005). Comparison of the symptoms of exercise-induced muscle damage after an initial and repeated bout of plyometric exercise in men and boys. *Journal of Applied Physiology, 99*(3):1174-81.

Mayer, J.M., Mooney, V., Matheson, L.N., Erasala, G.N., Verna, J.L., Udermann, B.E. & Leggett, S. (2006). Continuous low-level heat wrap therapy for the prevention and early phase treatment of delayed-onset muscle soreness of the low back: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation, 87*(10):1310-7.

Mozaffarian, D., Ascherio, A., Hu, F.B., Stampfer, M.J., Willett, W.C., Siscovick, D.S., & Rimm, E.B. (2005). Interplay between different polyunsaturated fatty acids and risk of coronary heart disease in men. *Circulation, 111*(2):157-164.

Nes, M., Frost, A.L., Solvoll, K., Sandstad, B.E., Husvedt, A., Lovo, A., & Drevon, C.A. (1992). Accuracy of a quantitative food frequency questionnaire applied in elderly Norwegian women. *European Journal of Clinical Nutrition, 46*(11):809-821.

Parr, J.J., Yarrow, J.F., Garbo, C.M., & Borsa, P.A. (2009). Symptomatic and functional responses to concentric- eccentric isokinetic versus eccentric-only isotonic exercise. *Journal of Athletic Training, 44*(5):462-8.

Prasartwuth, O., Taylor, J.L., & Gandevia, S.C. (2005). Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. *The Journal of Physiology,* 567(1):337-48.

Philippou, A., Maridaki, M., & Bogdanis, G.C. (2003). Angle-specific impairment of elbow flexors strength after isometric exercise at long muscle length. *Journal of Sports Science, 21*(10):859-65.

Pickova, J. (2009). Importance of knowledge on lipid composition of foods to support development towards consumption of higher levels of n-3 fatty acids via freshwater fish. *Physiological Research, 58*(Suppl. 1):S39-S45.

Rahnama, N., Rahmani-nia, F., & Ebrahim, K. (2005). The isolated and combined effects of selected physical activity and ibuprofen on delayed-onset muscle soreness. *Journal of Sports Sciences, 23*(8):843-50.

Shils, M.E., Shike, M., Ross, A.C., Caballero, B., & Cousins, R.J. (2006). *Modern Nutrition in Health and Disease.* 10th ed. Baltimore, MD: Lippincott Williams & Wilkins.

Silva, L.A., Pinho, C.A., Silveira, P.C.L., Tuon, T., De Souza, C.T., Dal-Pizzol, F., & Pinho, R.A. (2010). Vitamin E supplementation decreases muscular and oxidative damage but not inflammatory response induced by eccentric contraction. *The Journal of Physiological Sciences, 60(*1):51-7.

Soper, C. & Hume, P.A. (2004). Towards an ideal rowing technique for performance: the contributions from biomechanics. *Sports Medicine, 32*(12):825-48.

Swierk, M., Williams, P.G., Wilcox, J., Russell, K.G., & Meyer, B.J. (2010). Validation of an Australian electronic food frequency questionnaire to measure polyunsaturated fatty acid intake. *Nutrition.* Article in press, corrected proof.

Tartibian, B., Maleki, B.H., & Abbasi, A. (2009). The effects of ingestion of omega-3 fatty acids on perceived pain and external symptoms of delayed onset muscle soreness in untrained men. *Clinical Journal of Sport Medicine, 19*(2):115-119.

APPENDIX A

Research Survey
Division of Nutrition, Georgia State University

General Directions: Please complete all of the questions below. These surveys are anonymous, so please be honest and as accurate as possible! in all of your responses.

Part I: Preliminary Information

ID code: _______________
Gender: (Check one) [ ] (1) Male [ ] (2) Female
Ethnicity: (Check one)
[ ] (1) Hispanic [ ] (2) Non-Hispanic Caucasian [ ] (3) Non-Hispanic Black
[ ] (4) Asian [ ] (5) American Indian [ ] (6) Pacific Islander
[ ] (7) Other
Seasons *completed* on a collegiate team: _________
Age: ______ years Ht: _______ feet ________inches Wt: _________ lbs

II. Daily Habits

1. On average, how much time *per day* do you spend in the sun? Check one:
   - [ ] (1) 1-30 minutes
   - [ ] (2) 30 minutes-1 hour
   - [ ] (3) 1-1.5 hours
   - [ ] (4) 1.5-2 hours
   - [ ] (5) 2-2.5 hours
   - [ ] (6) 2.5-3 hours
   - [ ] (7) more than 3 hours
2. Check the body parts typically exposed when you are in the sun each day:
   - [ ] (1) Torso
   - [ ] (2) Arms
   - [ ] (3) Hands
   - [ ] (4) Legs
3. What sunscreen SPF do you typically wear when you are in the sun?
   - [ ] (1) None
   - [ ] (2) 1-15 SPF
   - [ ] (3) 16-30 SPF
   - [ ] (4) 31-45 SPF
   - [ ] (5) 46-60 SPF
   - [ ] (6) 61-75 SPF
   - [ ] (7) Over 75 SPF

Part III: Examining Exercise Regimen

1. Please circle "YES" or "NO" for the following questions:

Did you train this past summer for the fall season?	□ (1) YES	□ (2) NO
Do you typically warm up before practice?	□ (1) YES	□ (2) NO
Do you typically stretch before practice?	□ (1) YES	□ (2) NO
Do you typically stretch after practice?	□ (1) YES	□ (2) NO

2. How many times per week do you have organized team practice? _____ times/week
3. Check the normal time of day in which you practice:
   [ ] (1) Mid-morning [ ] (2) Mid-day [ ] (3) Afternoon/Evening
4. What is the average duration of practice? _____ minutes
5. What is the average duration of your typical warm-up? _____ minutes
6. In addition to rowing on both the water and the ergometer, check any cross training activities listed below in which you participated this past week:
   - [ ] (1) Backward downhill running
   - [ ] (2) Bicep curls
   - [ ] (5) Forward downhill running
   - [ ] (6) Squats

[ ] (3) Lunges
[ ] (4) Low back extensions
[ ] (7) Step-ups
[ ] (8) Wrist extensions

7. Please check any strategies you used to reduce muscle soreness from exercise this week:
   [ ] (1) Massage
   [ ] (2) Heating pads
   [ ] (3) Ice packs
   [ ] (4) Ibuprofen or aspirin
8. Please use the three scales below to answer the following questions by ranking either exertion, soreness, or urine color and placing the number in the right column labeled "**Rank/Number**":

Question	Rank/Number
What was your average perceived *exertion* during exercise in the week before the fall season? (If you did not perform exercise the week before fall season practice, write "0" in the blank.)	
What was your average perceived *exertion* during practice this past week?	
What is the highest perceived *soreness* you experienced this past week?	
Based on the scale, rate your perceived *soreness* on the day before fall season practice began.	
On average, what color was your *urine* immediately **before** practice this past week?	
On average, what color was your *urine* **during** practice this past week?	
On average, what color was your *urine* immediately **after** practice this past week?	

Rank	Exertion Level
0	Nothing at all
0.5	Very, very light
1	Very light
2	Light
3	Moderate
4	Somewhat hard
5	Hard
6	
7	Very hard
8	
9	
10	Very, very hard

Rank	Soreness Level
1	No pain
2	Dull vague ache
3	Slight persistent pain
4	More than slight pain
5	Painful
6	Very painful
7	Unbearably painful

Rank	Urine Color
1	
2	
3	
4	
5	
6	
7	
8	

Part IV: Food Frequency Questionnaire

Directions: After answering the preliminary questions, please use the following scale to rate how often you typically consume the foods/supplements below by choosing numbers that represent how often you typically consume each food and writing those numbers in the blanks provided under "**Frequency**" in the *second* of the larger tables. For serving size, indicate how much you eat at one time by circling one of the letters provided that represent "small," "medium," and "large." The given examples represent "medium" servings. If you eat less than the given example amount at one time, circle "S." If you eat more than the given example amount at one time, circle "L." If you take a supplement, indicate the amount in milligrams on the designated blank line under serving size.

1. Please check “YES,” “NO,” or “UNSURE” for the following questions:

Does your school provide free or low-cost nutrition counseling? [ ]-(1) YES [ ]-(2) NO [ ]-(3) UNSURE
If so, have you used those services? [ ]-(1) YES [ ]-(2) NO

1	2	3	4	5	6	7	8	9	10
<1x/mo	1x/mo	2x/mo	3x/mo	1x/wk	2x/wk	3x/wk	Every other day	1x/day	2x+/day

Food	Medium Serving Examples
Fish (dark fish, canned tuna, other fish)	2 oz
Crustaceans (crabs, shrimp, lobster, scallops)	2 oz
Fish oil	2 teaspoons
Plant oils (canola, flax, soy, olive, etc.)	2 teaspoons
Flax/Linseed oil	2 teaspoons
Nuts and seeds	2 tablespoons
Flax/Linseeds	2 tablespoons
Meat (non-fish)	2 oz
Grains (pasta, oatmeal, bread, crackers, cereal, etc.)	2 pieces bread, 2 cups cereal, 1 cup cooked oats, 2/3 cup cooked rice/pasta, 2 cups crackers/pretzels
Vegetables	2 cups raw or 1 cup cooked/canned
Fruits	2 cups raw, 1 cup canned, ½ cup dried
Fast food meals	1 entrée plus medium side and medium soda

Food	Frequency (Use scale above)	Serving Size (Examples above)
Dark fish (mackerel, salmon, sardines, bluefish, fresh tuna, swordfish)		**S M L**
Canned tuna		**S M L**
Other fish		**S M L**
Crustaceans (crabs, shrimp, lobster, scallops)		**S M L**
Fish oil		**S M L**
Plant oils (canola, soy, olive, vegetable, etc.)		**S M L**
Flax/Linseed oil		**S M L**
Nuts and seeds		**S M L**
Flax/Linseeds		**S M L**
Meat (non-fish)		**S M L**
Grains (pasta, oatmeal, bread, crackers, cereal, etc.)		**S M L**
Vegetables		**S M L**
Fruits		**S M L**
Fast food meals		**S M L**
Fish oil supplement (capsule form)		___________ mg
DHA supplement (capsule form)		___________ mg

Thank you for participating! Please make sure all answers are complete and accurate!