5-2022

Self-Rated Function and Delayed Onset Muscle Soreness (DOMS) Responses to 6-Week Supplementation with Waters with Anti-Inflammatory Capabilities (WAC)

Kristin Garner
University of Arkansas, Fayetteville

A thesis project for the fulfillment of the requirements for the degree of Bachelor of Science in Exercise Science

Self-Rated Function and Delayed Onset Muscle Soreness (DOMS) Responses to 6-Week Supplementation with Waters with Anti-Inflammatory Capabilities (WAC).

By

Kristin Garner
University of Arkansas
Bachelor of Science in Exercise Science, 2022

To be presented: April 2022
University of Arkansas

_____

Dr. Brendon McDermott
Thesis Director

_____

Dr. Matthew Ganio
Committee Member

I. Introduction

A key phrase of the exercising realm is “no pain, no gain.” While this has some merit concerning the effects of delayed onset muscle soreness (DOMS), it can often mislead individuals new to strenuous exercise, leading them to believe that the only way to train their muscles is by inducing pain. Studies have shown that this statement is not accurate in producing muscle gains, but because of mass media, many individuals fall into this trap (Flann et al., 2011). This can reduce exercise adherence, instigating a fear that becoming physically active is a constant battle of overcoming muscle pain. For this reason, the area of nutritional supplementation for exercise has been sought after by active individuals, and those new to strenuous activity.

Nutritional supplementation for exercise has been used for centuries, but it has developed into an underregulated industry that has grown in popularity within the exercising community. Supplements claim to produce greater muscle gains and prevent muscle fatigue and pain brought on by exercising without enough research done to support these claims. However, most people are uninterested in how these supplements work and use them because of the intriguing statements on the labels. Some independent studies have examined the effects of these supplements, but most researchers investigate more “natural” remedies for DOMS in fruits, like oranges and cherries, which have high anti-inflammatory capabilities. Despite this, research has scarcely examined the effects of anti-inflammatory supplementation of volatile compounds within superfruits.

The most noticeable indicators of exercise-induced muscle damage (EIMD) include muscle pain and self-rated function. Physically active individuals are familiar with these symptoms, especially after returning to exercise after a break. Of particular interest for

individuals who experience DOMS are ways to reduce pain and improve any impaired function. To understand the effectiveness of treatments for DOMS and its related symptoms, it must be recognized that alterations at the cellular level of the inflammatory response are responsible for these side effects.

Statement of the Problem

Current research analyzes the effects of antioxidant and anti-inflammatory supplementation on muscle damage, focusing on individuals who regularly exercise. More research is needed to analyze the effects of this supplementation on EIMD in novice exercisers. In the case of these inexperienced individuals, supplementation has the possibility of improving the transition into a healthier, physically active lifestyle through exercise, by reducing the side effects of EIMD. This is only the case if the inflammatory response brought on by EIMD is reduced by anti-inflammatory supplementation in those who are novice exercisers.

Additionally, few studies have investigated natural sources for antioxidant and anti-inflammatory supplementation. Vitamin C is the most commonly researched vitamin supplementation due to its high antioxidant capabilities, but not all individuals favor the regular consumption of pills for a variety of reasons. For this reason, some studies have examined naturally occurring antioxidant and anti-inflammatory capable foods like fruits and fruit-derived polyphenols. However, with the varying seasons of ripeness and pure motivation of an individual, it is difficult for people to consume an adequate number of fruits that will drastically affect a person's oxidative capacity or overall inflammation. Therefore, it is reasonable to suggest that an easily accessible beverage with high antioxidant and anti-inflammatory capabilities should be analyzed in its ability to reduce inflammation.

The purpose of this study is to identify the effects of waters with anti-inflammatory capabilities (WAC) consumption on self-reported muscle function and DOMS responses. Data includes a pre-participation screening of each participant's one-repetition maximum (1RM) back squat, rating of perceived exertion (RPE) for each participant during exercise, and self-reported muscle function and muscle pain questionnaires following exercise. The results of this study were used to identify if a highly concentrated beverage with anti-inflammatory capabilities perceptually decreases the inflammatory response of a working body to prevent and treat muscle soreness.

Hypothesis

The hypotheses for the research study above are as follows:

1. After drinking the WAC beverages for six weeks, perceived muscle function will be greater 24- and 48-hours post-exercise compared to pre-supplementation.
2. After WAC supplementation for six weeks, perceived muscle soreness will be decreased 24- and 48-hours post-exercise compared to pre-supplementation.

Assumptions

1. All participants will adhere to refraining from consuming berries for 48 hours and drinking alcohol 24 hours before baseline testing.
2. During baseline testing, all participants will reach close to their perceived 1 repetition maximum to ensure the acute heavy resistance exercise protocol induces an inflammatory response.
3. All participants will accurately self-report muscle soreness and muscular function in each questionnaire.

4. All participants will consume two bottles of berry-infused water each day throughout the supplementation phase of the study.
5. All participants will adhere to refraining from consuming berries and drinking alcohol throughout the duration of data collection.

Limitations

1. All data collected by each participant during the self-reporting muscular function questionnaires are perceived by individuals and may not be an accurate reflection of muscular function.
2. Participants who have never endured strenuous resistance training might not accurately determine their actual 1 repetition maximum. This could result in a decrease in DOMS responses and invalidate the effects of WAC consumption.
3. Participants are responsible for consuming the berry-infused water on their own and must self-report that they are consuming them.
4. Participants are responsible for adhering to required dietary limitations throughout the study.

Significance of the Study

The significance of this research is the potential benefits for sedentary individuals seeking to improve their health through exercise initiation. When exercise begins after a lengthy gap, DOMS is inevitable. DOMS can hinder the desire to continue exercise training for any individual new to exercise, but DOMS can especially inhibit those who are trying to implement exercise into their lives. This research study will investigate whether or not waters with anti-inflammatory capabilities (WAC) can reduce the effects of DOMS and improve function in the

initial stages of exercise for sedentary individuals. Further, we will examine if WAC could potentially be used by individuals initiating exercise in facilitating long-term compliance.

II. Literature Review

Delayed Onset Muscle Soreness

Delayed onset muscle soreness (DOMS) is the sensation of muscle fatigue and pain 24 to 48 hours following a bout of exercise. Causes of DOMS have been debated and thoroughly studied, but the most common lines of thought are exercise-induced muscle damage (EIMD) and the immune system response to this damage. As a working muscle contracts, weaker sarcomeres are disrupted by failed reconnection of overstretched myofilaments, leading to damage of the sarcolemma (Peake et al., 2005). This damage can lead to the painful symptoms experienced with DOMS and reduced muscular function following a bout of strenuous exercise. Inflammation at the site of muscle damage is the result of immune system cell migration to repair and replace muscle fiber fragments (Peake et al., 2005). Once the immune system indicates that there is damage to the muscle, pro-inflammatory and anti-inflammatory cells are sent to the site where they will reconstruct and replenish the muscle with stronger and more durable muscle fibers. The purpose of this literature review is to discuss the current literature regarding DOMS, inflammatory response to exercise, antioxidant and anti-inflammatory supplementation.

Biomarkers of Inflammation

The most common biomarkers of inflammation are monocytes, macrophages, and neutrophils. Following a bout of exercise, monocytes increase in circulation and infiltrate damaged muscle tissue where they differentiate into macrophages and aid in satellite cell stimulation to enhance damaged cell survival (Freidenreich & Volek, 2012). This suggests that the increase in circulating monocytes following exercise is an indicator of needed immune interference at the site of muscle damage. Once macrophages enter the tissue, they produce several pro-inflammatory cytokines, which promote the removal of waste and remodeling of

tissue (Kanda et al., 2016). However, these macrophages do not enter the damaged muscle immediately following exercise. The sensation of pain with DOMS may be explained by the delay in macrophage entry into the injured tissue, thus delaying PGE_2 synthesis up to 24- to 48-hours post-exercise (Smith, 1990). PGE_2 is a mediator of inflammation at the site of EIMD. This prostaglandin is responsible for moderating immune cell function in the early stages of muscle damage and ultimately activating satellite cells within the muscle to regenerate impaired muscle fibers (Ho et al., 2017).

Pro-inflammatory cytokines, as mentioned before, play a role in the immune system's response to muscle damage and inflammation at the site. There are a plethora of cytokines involved in inflammation due to exercise, but commonly studied pro-inflammatory cytokines are interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNFα). IL-6 is a cytokine with a variety of functions, and it is typically seen in chronic inflammatory diseases like rheumatoid arthritis and glomerulonephritis (Hirano et al., 1990). IL-6 was initially thought to derive only from B cells, but now studies have shown that IL-6 can be produced by many other cells, including monocytes and macrophages (Scheller et al., 2011). According to Gabay (2006), IL-6 plays a function in acute inflammation as more of a defense mechanism to protect further tissue damage induced by exercise, as well as in chronic inflammation in long-term systemic diseases. IL-6 increases in response to exercise, and it is thought to be secreted by the contracting muscle deeming it a myokine. However, the rate of IL-6 synthesis is dependent on the intensity and duration of the exercise, duration having the greatest effect on systemic IL-6 (Fischer, 2006). Fischer (2006) also found that an IL-6 increases mobilization in circulation and oxidation of free fatty acids in the contracting muscle, as well as systemic adaptations to decrease circulating IL-6 at rest. TNFα is a strong pro-inflammatory cytokine mainly produced by adipose tissue (Coppack, 2001).

Petersen and Pedersen (2005) discuss how TNFα has been closely linked to insulin resistance in skeletal muscle, inhibiting glucose entry into muscle fibers for use as an energy source. This is likely the reason why many obese individuals with excess adipose tissue are more likely to develop type 2 diabetes mellitus. During exercise, TNFα levels rise as an acute response to muscle tissue damage resulting from strenuous muscle contractions, but this short spike is quickly limited by an increase in IL-6 that is fifty times greater and inhibits TNFα production (Pedersen et al., 2001). Pedersen et al. (2001) also discuss that there is an apparent relationship between IL-6 and TNFα, describing that both play pro-inflammatory response in the immediate two-hour period following exercise; thereafter TNFα levels decrease, and IL-6 begins to take on an anti-inflammatory role in muscle recovery. Because of the interaction between TNFα and IL-6 during and following an acute strenuous exercise bout, these cytokines could also play a role in symptoms of delayed onset muscle soreness. Croisier et al. (1998) found that the elevated levels of IL-6 immediately after exercise were directly linked to sustained levels of serum myoglobin, which peaked at 48 hours post-exercise matching the time frame of severe DOMS symptoms.

Another factor of inflammation is the flood of oxidants and neutrophils seen closely after exercise. A bout of exercise can increase the number of circulating neutrophils and their migratory activity, indicating that neutrophils may be involved in early muscle damage and inflammatory processes following eccentric exercise (Kanda et al., 2013). Local inflammation in muscular tissue can be caused by this neutrophil migration to the site; in addition, neutrophils produce reactive oxygen species (ROS), which can further tissue damage if overactive (Smith, 1990). Although it is thought that the rate at which ROS are produced is determined by the intensity of exercise, studies have shown that ROS are highly present regardless of the type of exercise. When comparing moderate intensity, high intensity, and low-volume high-intensity

interval exercise, all bouts resulted in significant increases in lipid hyperoxides, indicating the presence of reactive oxygen species (Wadley et al., 2015). This further promotes the neutrophilic activity in the muscle tissue and its necessity to neutralize these ROS.

Nociception and DOMS

Sonkodi, Berkes, and Koltai (2020) hypothesize that when a muscle undergoes repeated eccentric contractions, muscle spindles are excessively stretched, causing fluid within the cavity between the intrafusal fibers and extrafusal fibers to be compressed in such a way that causes micro-injury to the nerve terminals. They note that this disruption of nociceptive sensory fibers is what induces pain. However, the sensation of pain resulting from neural microinjury is not immediate. The nociceptors involved are classified as unmyelinated C fibers, which respond only to mechanical deformation and are slow-acting, and myelinated Aδ fibers, which respond primarily to chemical and thermal stimuli within the muscle in addition to mechanical deformation and are fast-acting (Basbaum et al., 2009). Myelinated Aδ fibers contain low-threshold mechanosensitive receptors (LTM), but both myelinated Aδ fibers and unmyelinated C fibers contain high-threshold mechanosensitive receptors (HTM); together, these receptors provide the sensations of immediate and secondary pain felt when neuromuscular damage is present (Fleckenstein et al., 2017). They found that HTMs were primarily responsible for pain due to EIMD over LTM, but because there is a greater proportion of HTM on unmyelinated fibers than on myelinated fibers, there is significantly greater prolonged pain following damage than acute pain. This augments the notion of slower acting, delayed pain associated with DOMS due to EIMD.

In addition to nociceptors, muscular mechanical hyperalgesia is caused by chain reactions of proteins released within the muscle following EIMD. Murase et al. (2010) found that

bradykinin released during strenuous eccentric contraction is a key mediator of the means that leads to muscular mechanical hyperalgesia. They discovered that injection of a bradykinin receptor antagonist (HOE 140) before eccentric contractions suppressed the development of this hyperalgesia. In addition, they found that upregulation of nerve growth factor (NGF) mRNA and protein (produced in injured skeletal muscle and nerves that results in hyperalgesia by sensitizing nociceptors) was delayed by twelve hours following eccentric contraction and lasted for two days. These findings aid in the understanding of the delay in soreness due to nociceptor sensitivity and activation from EIMD.

Anti-Inflammatory Supplementation

Since inflammation from DOMS comes with many side effects, including muscle pain and fatigue, many individuals turn to supplements in an attempt to recover quicker. Although not much research has investigated the long-term effects on muscle, the most common over-the-counter treatment for DOMS by athletes and other individuals who engage in vigorous exercise are nonsteroidal anti-inflammatory drugs (NSAIDs) (Schoenfeld, 2012). It is thought that NSAIDs relieve pain due to inflammation by inhibiting cyclo-oxygenase enzymes that promote the formation of pro-inflammatory substances (Vane & Botting, 1998). However, these inhibited pathways could potentially decrease the full potential of muscle hypertrophy with long-term use (Schoenfeld, 2012). More natural remedies used are found in common foods. McFarlin et al. (2016) studied curcumin, an ingested natural anti-inflammatory at 5g per day, and found reductions in the concentrations of pro-inflammatory cytokines, including TNFα, and other markers following eccentric exercise. Cherries are high in anthocyanin, a potent antioxidant with anti-inflammatory properties, and reduce and prevent inflammation and EIMD (Coehlo et al., 2015). This offers a low-cost and easily accessible strategy for individuals to treat DOMS and

inflammation succeeding exercise. Anti-inflammatory supplements, especially those from natural sources, have been shown to have positive effects on damaged tissues in resilience and regeneration, as well as the ability of athletes to maintain health and adapt to vigorous exercise (Rawson et al., 2018).

Antioxidant Supplementation

Antioxidant supplements are also used to treat symptoms of DOMS and immune system function following exercise. After eight weeks of antioxidant supplementation, both normal-weight and overweight young adults showed a decrease in exercise-induced changes in lipid hyperoxides, possibly due to shifts in cytokines from pro-inflammatory to more anti-inflammatory (Vincent et al., 2006). Studies have investigated various natural sources of antioxidants and their effects on working muscles at various intensities of exercise. Acute and chronic supplementation of fruit-derived polyphenols decreases oxidative stress during prolonged high-intensity exercise when ROS capacity is exceeded (Kashi et al., 2019). In a study done by Cases et al. (2017), when supplemented with a combination of pomegranate, green tea, and grape extract an hour before the start of exercise, total power output was increased by 5% over the placebo group during high-intensity exercise. This was likely due to increased antioxidant capacity from supplementation when without it, antioxidant reserves naturally found in the body would have been depleted (Cases et al., 2017). Vitamins C and E are also sources of antioxidants and could potentially protect damaged muscle tissue when supplemented. Supplementation of vitamins C and E has demonstrated positive effects following exercise, including reduced DNA damage from vitamin E supplementation (due to decreased low-density lipid oxidation), as well as reduced DOMS symptoms from vitamin C supplementation (due to increased antioxidant activity on free radicals produced during exercise) (Kanter, 1998).

Increases with plasmatic vitamin E levels are significantly correlated with increases in plasmatic VLDL-cholesterol and triglycerides as the intensity of exercise is increased (Cases et al., 2006). This shows that vitamin E is important during exercise, although most healthy individuals are typically not deficient in it (Kanter, 1998).

III. Methods

Ethical Approval and Consent

The Institutional Review Board (IRB) of the University of Arkansas approved all procedures described in this report (#2102314312). All subjects provided written consent before participating in the study as part of the recruitment and baseline testing. All personal data were de-identified to ensure that patient confidentiality is upheld.

Study Population

Subjects were recruited from the university campus by announcements through daily newsletters and word of mouth. Exclusion criteria included: regular resistance training, leg muscle or lower limb joint conditions, chronic inflammatory conditions, ingestion of seven servings or more of fruits and vegetables each week, and regular use of NSAIDs. This study included eight participants who met the criteria (Table 1).

Subjects reported to the lab on Day 0 for baseline testing, where each participant's anthropometric measures were taken. This included height, weight, body mass index (BMI), and body composition. Body composition was measured through dual-energy X-ray absorption (DEXA) scans. In addition, each participant's one-repetition maximum (1RM) back squat was determined using standardized protocols set forth by the Nation Strength and Conditioning Association. This was used to calculate each participant's 10-repetition maximum (10RM) that was used for the exercise protocol.

Table 1. Summary of participant characteristics on Day 0.

	n	Minimum	Maximum	Mean	Standard deviation
Height (cm)	8	63.5	70.9	168.3	7.1
Body mass (kg)	8	120.0	257.0	75.4	21.7
BMI	8	20.3	39.2	26.5	6.4
Body fat (%)	8	11.6	49.5	36.7	12.0
1-rep maximum (lbs)	8	95.0	195.0	133.8	34.4

Pre-Supplementation Data Collection

Participants were instructed to return to the lab on Day 7 for the first exercise protocol to ensure participants had adequate rest and recovery time following the 1RM testing. Participants were instructed to abstain from eating berries for 48 hours before and drinking alcohol for 24 hours before reporting for baseline testing and all subsequent lab visits. All lab visits were completed between 7:00 am and 9:00 am to ensure that each participant arrived fasted and has not participated in any type of physical activity before completing the exercise protocol. Participants then underwent a standardized warmup, consisting of five minutes at a self-selected pace on a treadmill, followed by two sets of 10 bodyweight squats, 10 glute bridges, and 10 lunges on each leg. Participants completed an acute heavy resistance exercise protocol (AHREP) following the warmup, consisting of six sets of 10 repetitions at their 10RM back squat with two minutes of rest between sets. The working set load for the 10RM was estimated by taking 75% of the 1RM weight for each participant. This protocol was chosen due to its effectiveness in eliciting a large inflammatory response in other studies. Following each set of squats during the AHREP, each participant's rate of perceived exertion (RPE) – on a scale of 6 to 20 – was collected. After completion of the exercise, participants performed a standardized cool down of walking at a comfortable pace on a treadmill.

On Day 8, approximately 24 hours after completion of the first AHREP (AHREP1), participants returned to the lab to complete muscle soreness and muscular function questionnaires. Self-reported muscle soreness was measured using a pain scale of 0 to 10, with a score of "0" equating to no pain and "10" equating to severe pain. Muscular function was evaluated using a horizontal 100mm line with no other visual markers other than the two endpoints. Participants were asked to draw a vertical line on the diagram to indicate their level of function. 0mm indicated no muscular function, and 100mm indicated complete muscular function. On Day 9, approximately 48 hours after completion of AHREP1, the participants returned to the lab to complete the same muscle soreness questionnaire and muscular function questionnaire. Before leaving the lab, participants were given pre-bottled berry-infused water and were instructed to consume two bottles each day.

Supplementation Period

After completing the 48-hour muscle soreness and muscular function questionnaires, participants were given 42 bottles containing 16.9 oz of berry-infused water, during which they were instructed to drink two bottles per day starting after they left the lab. This first day of supplementation was denoted as S0. Participants were instructed to self-report consumption of the berry-infused water and any other consumption of fruits and vegetables or alcohol although they were asked to exclude them. On Day 29 of the study (S21), participants were scheduled to return to the lab for a compliance check and to receive the remaining 42 bottles of berry-infused water for the study. The supplementation period continued until the end of data collection, totaling six weeks.

Post-Supplementation Data Collection

On Day 50 of the study, participants returned to the lab to complete another exercise protocol. The participants performed the same standardized warmup as on Day 7, and following the warmup completed a second AHREP (AHREP2). The same RPE scale was used to measure RPE after each set of back squats. Following completion of the exercise, participants performed the same standardized cool down on a treadmill. On Day 51, approximately 24 hours after completion of AHREP2, participants returned to the lab for the same muscle soreness and muscular function questionnaires. On Day 52, approximately 48 hours after completion of AHREP2, participants returned to the lab for the final muscle soreness and muscle function questionnaires. At this point, the participation in the study was concluded, and participants were compensated for their efforts.

Statistical Analysis

Each participant's RPE for each AHREP was used to compare the effectiveness of the protocol and ensure that the exertional workload of each AHREP remained consistent. The muscle soreness and muscular function questionnaires were used to quantify each participant's muscle pain and mobility. The data were then organized by participant and occurrence in relation to the supplementation period – denoted as pre-supplementation or post-supplementation. RPE after each set for both AHREP1 and AHREP2 were analyzed using a repeated-measures ANOVA with appropriate Greenhouse-Geisser correction when sphericity was violated. Data collected from the questionnaires were compared using paired-samples t-tests to examine pre- versus post-supplementation differences in muscle pain and self-rated function. Significance was set a-priori as $\leq .05$.

IV. Results

All eight participants completed the prescribed number of sets and repetitions for both exercise protocols. There were no significant trial differences in RPE regardless of time ($p=0.56$). There were no significant time point differences in RPE, regardless of the trial ($p=0.64$). Lastly, there was not a significant trial x time interaction ($p=0.53$).

Table 1 compares the self-reported muscle soreness measurements 24- and 48 hours following each trial. Of the eight participants, seven completed the muscle soreness questionnaires for both 48-hour measurements.

Table 1. Muscle soreness 24- and 48-hours following AHREP1 and AHREP2

	n	**Mean ± Std**
24-hour soreness post-AHREP1	8	2 ± 1
24-hour soreness post-AHREP2	8	3 ± 1
48-hour soreness post-AHREP1	7	3 ± 2
48-hour soreness post-AHREP2	7	4 ± 2

There were no significant differences in muscle soreness 24-hours following the AHREP before and after the supplementation period ($p=0.554$). There were also no significant differences in muscle soreness 48-hours following the AHREP before and after the supplementation period ($p=0.379$).

Table 2 compares the self-reported muscular function 24- and 48-hours following each trial. Along with the muscle soreness questionnaires, seven out of the eight participants completed both 48-hour muscular function questionnaires.

Table 2. Self-rated function 24- and 48-hours following AHREP1 and AHREP2.

	n	**Mean ± Std**
24-hour function post-AHREP1	8	64.25 ± 22.08
24-hour function post-AHREP2	8	51.75 ± 24.71
48-hour function post-AHREP1	7	54.57 ± 33.32
48-hour function post-AHREP2	7	35.29 ± 23.05

There were no significant differences in muscular function 24-hours following the AHREP before and after the supplementation period ($p=0.417$). There were also no significant differences in muscular function 48-hours following the AHREP before and after the supplementation period ($p=0.291$).

V. Discussion

Summary of Findings

Before analyzing the results, we hypothesized that the consumption of the WAC beverage would lower self-reported muscle soreness and improve self-reported muscular function following the AHREP. In this study, we found that there were no significant differences in muscle soreness and muscular function before and after the supplementation period. This finding was somewhat expected since there was a small group of participants within this study.

Comparison of Findings to Other Studies

The lack of effect of the WAC beverage is somewhat unexpected when compared to other studies analyzing the effectiveness of polyphenol-rich foods on DOMS and muscular function, namely tart cherry juice. A study examining the effects of Montmorency tart cherry concentrate found that muscle soreness ratings were significantly lower in the supplemented group compared to the placebo group after performing prolonged, repeated sprints (Bell et al., 2016). Compared to the present study, this study used a similar visual analog scale to rate muscle soreness, but instead of self-rating muscle function, the study utilized quantifiable tests of functional performance. This could more accurately depict muscle function following anti-inflammatory supplementation; however, the supplementation period of this study lasted only seven consecutive days – four days prior to the exercise protocol and on each trial day. Similarly, Tanabe et al. (2022) reviewed dietary supplementation for EIMD and DOMS, reporting that tart cherry juices had positive effects on muscular function following EIMD but little to no effect on DOMS. This study investigated eight studies with an average supplementation period of approximately eight days, concluding that tart cherry juice favors improved recovery when used four to five days prior to exercise or two to three days after. A meta-analysis reviewing the

effects of supplementation through polyphenol-rich foods, juices, and concentrates, most including tart cherry juice and for a similar duration, found moderate to very low certainty that these supplements accelerate muscle function and reduce muscle soreness after EIMD (Rickards et al., 2021). This could indicate that tart cherry juices and substances with comparable antioxidant capabilities, like the WAC beverage, might be better suited for acute treatment of EIMD and DOMS although the evidence is inconsistent and minimal to create conclusions.

The study design could have masked the effect of the WAC beverage. AHREP1 was completed seven days after 1RM testing, whereas AHREP2 was completed after six weeks of no activity. It is known that neuromuscular adaptations are the body's first attempts to increase force production in response to a new stimulus (Škarabot et al., 2020). This is accomplished by the recruitment of more motor units, greater motor unit synchronization, and increased neural activation (Hughes et al., 2018). These adaptations can be seen as early as two to four weeks or sooner after the onset of strength training (Škarabot et al., 2020). Because the participants of the present study were untrained and unfamiliar with resistance training, the 1RM testing could have been a large enough stimulus to evoke neural adaptations during AHREP1. The six-week gap in resistance training could have been enough to remove the neural adaptations made from the earlier sessions. Detraining, or a period of an insufficient training stimulus, has been marked as the reversal of training adaptations, including neuromuscular adaptations (Mujika & Padilla, 2001). Mujika and Padilla (2001) indicated that within eight weeks of the cessation of strength training, muscular strength can be lost by up to 7-12%, more specifically noting an 11.6% decrease in force production for the back squat. Although the gap in the present study was only six weeks, the stoppage in stimulus for the novice lifters could have been enough to lower their force production capabilities for AHREP2. This would explain the slight increase in self-reported

DOMS and the decrease in self-rated muscular function although these values were not significant. With this in mind, the impact of the WAC beverage on reducing inflammation may not have been accurately seen.

The WAC beverage is different from other polyphenol-containing substances because it contains a higher concentration of polyphenols. The WAC beverage contains one part of berry essences to 12 parts water, which is higher than the current manufactured berry essence-infused waters. Other manufactured fruit drinks contain fruit juices, which lose part of their antioxidant and anti-inflammatory capabilities in the volatile compounds released as essences. By creating a fluid that is flavored with natural components of berries and that contains a high capacity for combatting inflammation, further studies could be initiated to assess the capabilities of the beverage within the human body.

Limitations

A limitation of this study was the reliance of participants to accurately report their consumption of fruits and vegetables. Data from dietary recall is also prone to underreporting when it comes to studies heavily excluding a particular type of food. Participants may have reported eating fewer servings of fruits and vegetables than what they realistically consumed. This could influence the effects of the WAC beverage on inflammation if the participant were consuming greater than seven servings of fruits and vegetables.

This study was also reliant on the accuracy of participants to quantify their perceptual feelings and rating of function. Given the nature of subjective muscle soreness and perceived muscular function, these measures tend to have a lower effect size than more objective biological measurements. This also relates to the determination of 1RM for each participant. For those who

have never performed a squat before, determining a true 1RM may exceed their comfort level, as they are not accustomed to squatting heavy loads.

Conclusion

In summary, this study analyzed the effectiveness of anti-inflammatory supplementation, through berry essence infused water, on DOMS responses to acute exercise. This study did not show that after six weeks of supplementation muscle soreness nor function was altered following a bout of DOMS-inducing exercise. Further research should be conducted on inflammatory biomarkers and if there are any alterations in their responses to exercise following berry-essence supplementation. The inclusion of placebo groups could also prevent bias within the subjects while reporting perceptual data. Along with this, a larger sample size consisting of both males and females should be included to present an accurate representation of the population.

References

Basbaum, A. I., Bautista, D. M., Scherrer, G., & Julius, D. (2009). Cellular and molecular mechanisms of pain. *Cell, 139*(2), 267-284. https://doi.org/10.1016/j.cell.2009.09.028

Bell, P., Stevenson, E., Davison, G., & Howatson, G. (2016). The effects of montmorency tart cherry concentrate supplementation on recovery following prolonged, intermittent exercise. *Nutrients*, *8*(7), 441. https://doi.org/10.3390/nu8070441

Cases, J., Romain, C., Marin-Pagan, C., Gaillet, S., Prost-Camus, E., Prost, M., & Alcaraz, P. E. (2017). Supplementation with a polyphenol-rich extract, Perfload, improves physical performance during high-intensity exercise: A randomized, double blind, crossover trial. *Nutrients*, *9*, 421–433. https://doi.org/10.3390/nu9040421

Cases, N., Sureda, A., Maestre, I., Tauler, P., Aguilo, A., Cordova, A., Roche, E., Tur, J. A., & Pons, A. (2006). Response of antioxidant defenses to oxidative stress induced by prolonged exercise: antioxidant enzyme gene expression in lymphocytes. *European Journal of Applied Physiology*, *98*, 263–269. https://doi.org/10.1007/s00421-006-0273-y

Coppack, S. W. (2001). Pro-inflammatory cytokines and adipose tissue. *Proceedings of the Nutrition Society, 60,* 349-356. https://doi.org/10.1079/pns2001110

Coehlo Rabello Lima, L., Oliveira Assumpcao, C., Sergio Denadai, B., & Prestes, J. (2015). Consumption of cherries as a strategy to attenuate exercise-induced muscle damage and inflammation in humans. *Nutricion Hospitalaria*, *32*(5), 1885–1893. https://doi.org/10.3305/nh.2015.32.5.9709 x

Croisier, J. L., Camus, G., Venneman, I., Deby-Dupont, G., Juchmès-Ferir, A., Lamy, M., ... & Duchateau, J. (1999). Effects of training on exercise-induced muscle damage and interleukin 6 production. *Muscle & Nerve: Official Journal of the American Association*

of Electrodiagnostic Medicine, *22*(2), 208-212. https://doi.org/10.1002/(sici)1097-4598(199902)22:2<208::aid-mus8>3.0.co;2-b

Fischer, C. P. (2006). Interleukin-6 in acute exercise and training: what is the biological relevance. *Exercise Immunology Review*, *12*(6-33), 41.

Flann, K. L., LaStayo, P. C., McClain, D. A., Hazel, M., & Lindstedt, S. L. (2011). Muscle damage and muscle remodeling: no pain, no gain? *Journal of Experimental Biology, 214,* 674-697. https://doi.org/10.1242/jeb.050112

Fleckenstein, J., Simon, P., Konig, M., Vogt, L., & Banzer, W. (2017). The pain threshold of high-threshold mechanosensitive receptors subsequent to maximal eccentric exercise is a potential marker in the prediction of DOMS associated impairment. *PLOS ONE, 12*(10). https://doi.org/10.1371/journal.pone.0185463

Freidenreich, D. J., & Volek, J. S. (2012). Immune response to resistance exercise. *Exercise Immunology Review*, *18*, 8–41.

Gabay, C. (2006). Interleukin-6 and chronic inflammation. *Arthritis Research & Therapy*, *8*(2), 1-6. https://doi.org/10.1186/ar1917

Hirano, T., Akira, S., Taga, T., & Kishimoto, T. (1990). Biological and clinical aspects of interleukin 6. *Immunology Today*, *11*, 443-449. https://doi.org/10.1016/0167-5699(90)90173-7

Ho, A. T., Palla, A. R., Blake, M. R., Yucel, M. D., Wang, Y. X., Magnusson, K. E., Holbrook, C. A., Kraft, P. E., Delp, S. L., & Blau, H. M. (2017). Prostaglandin E2 is essential for efficacious skeletal muscle stem-cell function, augmenting regeneration and strength. *Proceedings of the National Academy of Sciences of the United States of America, 114*(26), 6675-6684. https://doi.org/10.1073/pnas.1705420114

Hughes, D. C., Ellefsen, S., & Baar, K. (2017). Adaptations to endurance and strength training. *Cold Spring Harbor Perspectives in Medicine*, *8*(6). https://doi.org/10.1101/cshperspect.a029769

Kanda, K., Sugama, K., Hayashida, H., Sakuma, J., Kawakimi, Y., Miura, S., Yoshioka, H., Mori, Y., & Suzuki, K. (2013). Eccentric exercise-induced delayed-onset muscle soreness and changes in markers of muscle damage and inflammation. *Exercise Immunology Review*, *19*, 72–85.

Kanter, M. (1998). Free radicals, exercise and antioxidant supplementation. *Proceedings of the Nutrition Society*, *57*, 9–13. https://doi.org/10.1079/pns19980004

Kashi, D. K., Shabir, A., Da Boit, M., Bailey, S. J., & Higgins, M. F. (2019). The efficacy of administering fruit-derived polyphenols to improve health biomarkers, exercise performance, and related physiological responses. *Nutrients*, *11*, 2189–2201. https://doi.org/10.3390/nu11102389

McFarlin, B. K., Venable, A. S., Henning, A. L., Best Sampson, J. N., Pennel, K., Vingren, J. L., & Hill, D. W. (2016). Reduced inflammatory and muscle damage biomarkers following oral supplementation with bioavailable curcumin. *BBA Clinical*, *5*, 72–78. https://doi.org/10.1016/j.bbacli.2016.02.003

Mujika, I., & Padilla, S. (2001). Muscular characteristics of detraining in humans. *Medicine and Science in Sports and Exercise, 33*(8), 1297-1303. https://doi.org/10.1097/00005768-200108000-00009

Murase, S., Terazawa, E., Queme, F., Ota, H., Matsuda, T., Hirate, K., Kozaki, Y., Katanosaka, K., Taguchi, T., Urai, H., & Mizumura, K. (2010). Bradykinin and nerve growth factor play pivotal roles in muscular mechanical hyperalgesia after exercise (delayed-onset

muscle soreness). *Journal of Neuroscience, 30*(10), 3752-3761. https://doi.org/10.1523/JNEUROSCI.3803-09.2010

Peake, J., Nosaka, K., & Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans. *Exercise Immunology Review*, *11*, 64–85.

Pedersen, B. K., Steensberg, A., Fischer, C., Keller, C., Ostrowski, K., & Schjerling, P. (2001). Exercise and cytokines with particular focus on muscle derived IL-6. *Exercise Immunology Review*, *7*, 18-31.

Petersen, A. M. W., & Pedersen, B. K. (2005). The anti-inflammatory effect of exercise. *Journal of Applied Physiology*, *98*(4), 1154-1162. https://doi.org/10.1152/japplphysiol.00164.2004

Rawson, E. S., Miles, M. P., & Larson-Meyer, D. E. (2018). Dietary supplements for health, adaptation, and recovery in athletes. *International Journal of Sport Nutrition and Exercise Metabolism*, *28*(2), 188–199. https://doi.org/10.1123/ijsnem.2017-0340

Rickards, L., Lynn, A., Harrop, D., Barker, M. E., Russel, M., & Ranchordas, M. K. (2021). Effect of polyphenol-rich foods, juices, and concentrates on recovery from exercise induced muscle damage: a systematic review and meta-analysis. *Nutrients, 13*(9), 2988. https://doi.org/10.3390/nu13092988

Scheller, J., Chalaris, A., Schmidt-Arras, D., & Rose-John, S. (2011). The pro-and anti-inflammatory properties of the cytokine interleukin-6. *BBA Molecular Cell Research*, *1813*(5), 878-888. https://doi.org/10.1016/j.bbamcr.2011.01.034

Schoenfeld, B. J. (2012). The use of nonsteroidal anti-inflammatory drugs for exercise-induced muscle damage. *Sports Medicine*, *42*(12), 1017–1028. https://doi.org/10.1007/bf03262309

Sonkodi, B., Berkes, I., & Koltai, E. (2020). Have we looked in the wrong direction for more than 100 years? Delayed onset muscle soreness is, in fact, neural microdamage rather than muscle damage. *Antioxidants, 9*(3), 212. https://doi.org/10.3390/antiox9030212

Smith, L. L. (1990). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *American College of Sports Medicine*, *23*(5), 542–551.

Tanabe, Y., Fujii, N., & Suzuki, K. (2021). Dietary supplementation for attenuating exercise-induced muscle damage and delayed-onset muscle soreness in humans. *Nutrients*, *14*(1), 70. https://doi.org/10.3390/nu14010070

Vane, J. R., & Botting, R. M. (1998). Anti-inflammatory drugs and their mechanism of action. *Inflammation Research*, *47*, 78–87. https://doi.org/10.1007/s000110050284

Vincent, H. K., Bourguignon, C. M., Vincent, K. R., Weltman, A. L., Bryant, M., & Taylor, A. G. (2006). Antioxidant supplementation lowers exercise-induced oxidative stress in young overweight adults. *Obesity*, *14*(12), 2224–2235. https://doi.org/10.1038/oby.2006.261

Wadley, A. J., Chen, Y. W., Lip, G. Y. H., Fisher, J. P., & Aldred, S. (2015). Low volume–high intensity interval exercise elicits antioxidant and anti-inflammatory effects in humans. *Journal of Sport Sciences, 34*(1), 1-9. https://doi.org/10.1080/02640414.2015.1035666

Škarabot, J., Brownstein, C. G., Casolo, A., Del Vecchio, A., & Ansdell, P. (2020). The knowns and unknowns of neural adaptations to resistance training. *European Journal of Applied Physiology*, *121*(3), 675–685. https://doi.org/10.1007/s00421-020-04567-3