Oral consumption of electrokinetically modified water attenuates muscle damage and improves postexercise recovery

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Borsa PA, Kaiser KL, Martin JS. Oral consumption of electrokinetically modified water attenuates muscle damage and improves postexercise recovery. J Appl Physiol 114: 1736–1742, 2013. First published April 11, 2013; doi:10.1152/japplphysiol.00083.2013.—The purpose of this study was to assess the effects of consuming electrokinetically modified water (EMW) on attenuating muscle damage and improving functional recovery following a single bout of isokinetic resistance exercise. Subjects were randomly assigned to an EMW (n = 20) or a placebo control (n = 20) group. Subjects consumed EMW or placebo water daily for 23 days. On day 19 subjects completed an exercise protocol for the biceps brachii to induce muscle damage. The protocol consisted of three sets of 20 repetitions using concentric and eccentric contractions of the elbow flexors. Blood draw and clinical measurements were performed preexercise as well as 24, 48, and 96 h postexercise. Clinical measures included maximal isometric strength, muscle soreness, pain with elbow extension, relaxed elbow angle (RANG), and self-report arm disability. Plasma samples were analyzed to determine concentrations of creatine kinase (CK) and high-sensitivity C-reactive protein (hsCRP). Pain with elbow extension and self-report arm disability were significantly higher in the placebo group compared with the EMW group at 48 h (P < 0.01) and 96 h (P < 0.01) after exercise. Oral consumption of EMW significantly reduced exercise-induced muscle damage and inflammation and improved functional recovery.

isokinetic; muscle soreness; hydration; inflammation; charge-stabilized nanostructures

Researchers have recently begun to investigate the anti-inflammatory and cytoprotective effects of electrokinetically modified solutions. RNS60, a modified isotonic saline solution generated through a process involving Taylor-Couette-Poiseuille (TCP) flow (18), has shown potent anti-inflammatory and cytoprotective effects across models of different diseases including allergic asthma and neuroinflammation (9–11; Khasnavis S, Ghosh S, Watson R, Pahan K, unpublished observation). A similar technology has been used to develop electrokinetically modified water (EMW) suitable for human consumption (6). Unlike supplemented “sports” beverages, the EMW has no added traditional active ingredient. Instead, the EMW is produced by treating the fluid with strong, controlled turbulence and cavitation events designed to produce nano-bubble-based physical structures stabilized by an electrical double layer at the liquid/gas interface. The exact nature of these nanostructures is currently under investigation, and the descriptive term “charge-stabilized nanostructures” (CSNs) has been used to refer to these entities. Cell and animal studies have begun to elucidate the mechanistic underpinnings of CSNs (10, Khasnavis S, Ghosh S, Watson R, Pahan K, unpublished observations). In humans, early evidence has shown that oral consumption of EMW containing CSNs can positively influence muscle performance and training adaptations in an athletic population (1, 6).

Strenuous exercise, especially eccentric-based exercise, is known to cause muscle fiber disruption, which activates an acute inflammatory response (3, 22). Although inflammation drives the repair process by synthesizing and releasing chemical mediators locally in the injured muscle, uncontrolled or prolonged inflammation can delay postexercise recovery mechanisms. The inflammatory response has also been shown to produce high levels of oxygen-derived free radicals and proteolytic enzymes that if allowed to proceed unabated can produce a secondary cascade of muscle fiber disruption, thus hindering the recovery process (4). Therefore, impaired or incomplete recovery following high-intensity exercise can negatively affect physical performance and delay functional progression.

Understanding the hydration requirements of athletes engaged in high-intensity exercise training and competition is necessary to maintain peak performance and assist in recovery. Thus the combined fluid replacement/hydration and protective effects of consuming EMW can benefit athletes by keeping them hydrated during and after exercise while concurrently mitigating the effects of physiological stressors such as intense exercise training and competition. We hypothesize that oral consumption of EMW will protect skeletal muscle from exercise-induced damage by reducing the extent of biomechanical stress from inflammation, and facilitate postexercise recovery following high-intensity resistance exercise. Therefore, our
Table 1. Subject characteristics and descriptive statistics between the electrokinetically modified water and placebo-control groups

<table>
<thead>
<tr>
<th></th>
<th>EMW</th>
<th>PLA</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>10 men, 10 women</td>
<td>9 men, 11 women</td>
<td></td>
</tr>
<tr>
<td>Age, yr</td>
<td>23.1 ± 3.0</td>
<td>24.0 ± 4.3</td>
<td>0.333</td>
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<tr>
<td>Height, cm</td>
<td>170.9 ± 9.3</td>
<td>174.8 ± 8.2</td>
<td>0.178</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.5 ± 13.8</td>
<td>72.5 ± 13.2</td>
<td>0.986</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.6 ± 3.0</td>
<td>23.6 ± 3.2</td>
<td>0.317</td>
</tr>
</tbody>
</table>

Values are means ± SD. EMW, electrokinetically modified water; PLA, placebo-control.

The objective was to determine whether oral consumption of EMW can reduce exercise-induced muscle damage and inflammation and improve functional recovery following a single bout of strenuous isokinetic resistance exercise for the biceps brachii muscle.

MATERIALS AND METHODS

Subjects. Forty healthy, nonsmoking, physically active males and females participated in the study. To be included in the study, subjects were required to be free of nutritional or dietary supplements for a minimum of 6 wk. Supplements included but were not limited to ephedra, yohimbine, prohormones, creatine, or anabolics. Subjects were excluded if they reported regular involvement in a weight-training program within the last 6 wk for the upper extremity. The criterion for “regular involvement” was equal to or greater than 2 times per week of weight training. Subjects were also excluded if they had a prior history of injury to the neck, shoulder, or elbow region of the dominant limb, a reported history of a bacterial infection, and/or use of anti-inflammatory medication within the last 6 wk. Subjects were instructed to maintain normal exercise and dietary habits and not to begin any weight loss program throughout their participation in the study. The protocol was approved by the University’s institutional review board, and all subjects gave written informed consent prior to participation. Subject demographics and descriptive statistics are listed in Table 1.

Design. This study used a double-blind, randomized, placebo-controlled, two-arm experimental design to investigate the effects of EMW on attenuating muscle damage and enhancing recovery following a single bout of isokinetic resistance exercise. Subjects were randomly allocated to either an experimental group (n = 20) or placebo control group (n = 20). Subjects allocated to the experimental group consumed EMW while the placebo-control group consumed unprocessed water. Subjects completed a 23-day study trial that included a controlled water consumption regimen, preexercise baseline testing, an exercise protocol to induce muscle damage, and planned follow-up postexercise data collection time points (Fig. 1). Subjects consumed two to six 500-ml bottles of the EMW or unprocessed water daily, as determined by weight categories. On day 19 subjects returned to the laboratory for preexercise (baseline) measurements followed by completion of the exercise protocol for the elbow flexors. The exercise protocol consisted of a single bout of isokinetic resistance exercise using three sets of 20 repetitions of concentric and eccentric contractions of the biceps brachii muscle. Subjects returned to the laboratory on day 20 (24 h postexercise), day 21 (48 h postexercise), and day 23 (96 h postexercise) for follow-up measurements. Preexercise and postexercise measurements consisted of maximal isometric arm strength, muscle soreness, pain with elbow extension, relaxed elbow angle (RANG), and self-report upper limb disability. Subjects also completed a blood draw preexercise and again during postexercise follow-up (days 20, 21, and 23).

Test articles and dosing schedule. Revalesio (Tacoma, WA) supplied the EMW and placebo water. The EMW was manufactured from reverse osmosis water with an added mineral content of 1.84 mg/l calcium chloride, 1.84 mg/ml magnesium chloride, and 1.17 mg/ml potassium bicarbonate and oxygen at a final concentration of ≥45 ppm by processing in a rotor/stator device at 4°C (patent 6,386,751). Chemically the EMW is identical to water with a small amount of dissolved oxygen. Placebo-control water was made from the same source water by adding the same minerals without processing in the rotor/stator device. The EMW and placebo water were packaged in identical 500-ml plastic bottles, and subjects were instructed to consume a prescribed dose of water (bottles/day) based on body weight categories (Table 2).

Exercise protocol. Arm exercise consisted of sustained near-maximal concentric and eccentric actions of the biceps brachii muscle group. An isokinetic testing and exercise device (Kin-Com 125 AP, Isokinetic International, Harrison, TN) provided resistance during arm exercise. Prior to the exercise protocol each subject completed a series of stretching exercises involving all of the major muscle groups of the upper extremity. Subjects were then seated in the Kin-Com with their dominant arm secured at their side in 90° elbow flexion. Initially, the subject’s maximal voluntary contraction (MVC) was measured (N-m) and then the subject began the exercise protocol. The protocol required subjects to perform repeated maximal shortening (concentric) and lengthening (eccentric) actions for the biceps brachii muscle group. The angular velocity was set at 45°/s for concentric actions and 60°/s for eccentric actions. Each subject completed three sets of 20 repetitions. Subjects were given a 1-min recovery period between sets. Each subject was instructed to perform arm repetitions “as hard as
that they can.” Verbal encouragement was provided by the investigator during the exercise protocol.

**Muscle strength.** Peak torque was assessed using the Kin-Com dynamometer. Peak torque is the maximum voluntary isometric torque produced during a static muscle contraction and was used as the criterion measure for isometric strength. Subjects were seated with their dominant arm placed at their side in 90° elbow flexion. Each subject performed three maximal voluntary isometric contractions of the dominant arm. Each contraction was held for 5 s. A 30-s recovery period was provided between contractions. The most forceful contraction of the three values was recorded as peak torque in newton-meters.

**Muscle soreness.** Muscle soreness was measured by applying focal pressure to a targeted area of the biceps muscle using an instrumented algometer (Force Ten FDX, Wagner Instruments, Greenwich, CT). Pressure was applied to the muscle at a rate of 1 kg/s until the pressure turned to pain. The subject was instructed to indicate at what point the pressure becomes painful. We referred to this point as the mechanical pain threshold (MPT). The MPT was performed at three sites on the biceps (proximal, midbelly, and distal region). The test was performed three times at each site and the average of the three scores was recorded as the criterion measure.

**Range of motion.** Relaxed elbow angle (RANG) was used to quantify limitations in elbow range of motion as a result of the resistance exercise. The RANG was measured using a standard two-arm plastic goniometer by having the participants stand with their dominant arm relaxed in a supinated position at their side. The RANG was measured in degrees (°).

**Pain with elbow extension.** Subjects were asked to subjectively rate their level of perceived pain after actively extending their exercised arm. Pain was quantified using a visual analog scale (VAS). The VAS contains a line from 0–10 cm with 0 representing no pain or soreness and 10 representing extreme pain or soreness. Subjects were asked to draw a single slash on the line at the location that most ideally represents their perceived pain level as a function of extending their dominant arm.

**Self-report upper limb disability.** Upper limb disability was measured using the QuickDASH self-report questionnaire. The QuickDASH is designed to focus on the subject’s ability to use the affected arm during activities of daily living. Subjects rated their symptoms and ability to perform specific tasks using a 5-pt hierarchical Likert scale. Scores were obtained by summing circled responses, dividing the total by the number of items answered, subtracting 1, and then multiplying that figure by 25. A score of zero represents no disability at all, while higher scores represent more limitations in self-reported function, with a score of 100 representing more limitations in self-reported function, with a score of 100 representing their perceived pain level as a function of extending their dominant arm.

**Blood collection.** Blood samples were collected at baseline (preexercise) and again at 24 h, 48 h, and 96 h postexercise via venipuncture in the early morning (between 7 and 10 am) after a 10-h fast. After separation, all specimens were aliquoted into the appropriate number of cryovials based on the proposed number of assays. Blood was taken from the antecubital vein and collected into a 10-ml Vacutainer tube containing ethylenediaminetetraacetic acid (K2EDTA; 8.4 mg/Vacutainer) and one 10-ml serum collection tube. Blood samples were centrifuged at 4°C at 1,500 × g for 15 min. Samples were stored immediately at −80°C in 4 to 6 aliquots. Samples (~1 ml) were thawed only once and immediately analyzed for a specific biomarker. Cryovial labels were coded by subject ID number, date, visit, and cryovial number. Each sample was logged into an Excel spreadsheet by a lab technician and then frozen at −80°C in locked freezers.

**Biochemical analysis.** Blood samples were analyzed from serum at each time point to determine concentrations of creatinine (CRK) and high-sensitivity C-reactive protein (hsCRP). Creatinine kinase (CK) levels were analyzed using a commercially available enzymatic assay kit (MaxDiscovery; B100 Scientific; Austin, TX; catalog no. 3460–07) and reported in units per liter (U/l). High-sensitivity C-reactive protein levels were analyzed using a commercially available enzymatic-linked immunosorbent assay kit (BioQuant, San Diego, CA; catalog no. BQ017C) and reported in milligrams per liter (mg/l).

**Data analysis.** All data analyses were performed using IBM SPSS Statistics for Windows 19.0 (IBM, Armonk, NY). Data were analyzed using a 2 × 4 [group (EMW or placebo) × time (preexercise, postexercise 24 h, postexercise 48 h, and postexercise 96 h)] ANOVA with repeated measures on the second factor. Pairwise comparisons (least significant difference) were used in the presence of significant interaction effects to reveal where differences occurred. Statistical significance was set a priori at $P < 0.05$.

## RESULTS

**Preexercise (baseline) measures.** There were no significant between-group differences in baseline scores for any outcome measure.

**Muscle strength.** There was no significant difference in maximal isometric arm strength between the group that consumed EMW and the group that consumed unprocessed water at any time point during the recovery process (interaction effect $F_{1,38} = 2.08; P = 0.157$) (Table 3). Strength loss at 24 h and 48 h postexercise was not significantly different between groups, with an average strength loss of 22% of preexercise levels at 48 h postexercise. At 4 days (96 h) postexercise, strength recovery was incomplete for each group with both groups displaying a residual 13% strength deficit.

**Muscle soreness.** There was no significant difference in muscle soreness between the group that consumed EMW and the group that consumed unprocessed water at any time point during the recovery process (interaction effect $F_{1,38} = 1.11; P = 0.299$) (Table 3). The mechanical pain threshold (MPT) decreased significantly for both groups at 24 h and 48 h postexercise (main effect for time; $P < 0.001$) and recovered to near preexercise levels by day 4 (96 h) postexercise ($P = 0.195$).

**Range of motion.** As a result of completing the exercise protocol RANG significantly declined for both groups at 24 h and 48 h postexercise. There was a significant between-group difference in RANG at 48 h and 96 h postexercise (interaction effect $F_{1,38} = 2.33; P = 0.10$).

## Table 2. Weight categories and bottles consumed per day

<table>
<thead>
<tr>
<th>Weight Category</th>
<th>Bottles Consumed Per Day</th>
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<tbody>
<tr>
<td>&lt; 59.1</td>
<td>2</td>
</tr>
<tr>
<td>59.1–72.7</td>
<td>3</td>
</tr>
<tr>
<td>72.7–86.4</td>
<td>4</td>
</tr>
<tr>
<td>86.4–100</td>
<td>5</td>
</tr>
<tr>
<td>&gt; 100</td>
<td>6</td>
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</table>

Weight recorded in kg.

Table 3. Outcome measures comparing preexercise vs. postexercise recovery scores for muscle strength and soreness

<table>
<thead>
<tr>
<th></th>
<th>Preexercise Baseline</th>
<th>24 h</th>
<th>48 h</th>
<th>96 h</th>
</tr>
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<tbody>
<tr>
<td>MVC N·m</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMW</td>
<td>38.5 ± 19.0</td>
<td>30.0 ± 14.0</td>
<td>28.7 ± 14.2</td>
<td>30.1 ± 12.6</td>
</tr>
<tr>
<td>Placebo</td>
<td>46.5 ± 26.0</td>
<td>38.4 ± 19.0</td>
<td>37.8 ± 24.5</td>
<td>40.4 ± 26.0</td>
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<tr>
<td>Muscle soreness, kg/s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMW</td>
<td>5.1 ± 2.0</td>
<td>3.6 ± 2.0</td>
<td>4.0 ± 2.0</td>
<td>4.8 ± 2.0</td>
</tr>
<tr>
<td>Placebo</td>
<td>4.5 ± 1.5</td>
<td>3.2 ± 1.1</td>
<td>3.7 ± 1.3</td>
<td>4.1 ± 1.2</td>
</tr>
</tbody>
</table>

Values are means ± SD. MVC, maximal voluntary contraction.
effect $F_{1,38} = 4.22; P = 0.047; \eta^2 = 0.1$. RANG was significantly greater in the group that consumed EMW compared with the group that consumed unprocessed water at 48 h ($P < 0.01$) and 96 h ($P < 0.01$) after exercise (Fig. 2). The group that consumed EMW recovered nearly full preexercise range of motion at 96 h postexercise compared with the group who consumed unprocessed water who still had an 8° deficit in RANG at 96 h postexercise.

**Pain with elbow extension.** There was a significant between-group difference in pain with active elbow extension at 48 h and 96 h postexercise (interaction effect $F_{1,38} = 9.27; P = 0.004; \eta^2 = 0.2$). The level of pain perceived with active elbow extension was significantly higher in the group that consumed the unprocessed water compared with the group that consumed EMW at 48 h ($P < 0.01$) and 96 h ($P < 0.01$) postexercise (Fig. 3).

**Upper limb disability.** The level of upper limb disability significantly increased for both groups as a result of the exercise protocol at 24 h, 48 h, and 96 h (main effect for time; $P < 0.001$) postexercise. There was a significant between-group difference in upper limb disability at 48 h and 96 h postexercise (interaction effect $F_{1,38} = 6.09; P = 0.018; \eta^2 = 0.14$). Self-report arm disability was significantly higher in the group that consumed unprocessed water compared with the group that consumed EMW at 48 h ($P < 0.001$) and 96 h ($P < 0.001$) after exercise (Fig. 4).

**Markers of muscle damage and inflammation.** Postexercise plasma levels for CK and hsCRP were significantly elevated at 48 h and 96 h compared with preexercise levels for both groups, indicating that the exercise protocol was effective in producing muscle damage and a postexercise inflammatory response.

Plasma concentrations for CK (interaction effect $F_{1,38} = 7.74; P = 0.008; \eta^2 = 0.17$) were significantly lower in the group that consumed EMW compared with the group that consumed unprocessed water at 48 h ($P < 0.05$) and 96 h ($P < 0.05$) after exercise (Fig. 5). Similarly, plasma concentrations for hsCRP (interaction effect $F_{1,38} = 4.76; P = 0.035; \eta^2 = 0.11$) were significantly lower in the group that consumed EMW compared with the group that consumed unprocessed water at 48 h ($P < 0.05$) and 96 h ($P < 0.01$) after exercise (Fig. 6).

**DISCUSSION**

The major findings of this study support our hypothesis that oral consumption of EMW reduces muscle damage and facilitates postexercise functional recovery following high-intensity resistance exercise. The group that consumed EMW for 18 days prior to, the day of, and 4 days after completing a bout of strenuous arm exercise demonstrated significant improvements in functional recovery compared with the group that consumed
the placebo water. Subjects who consumed the EMW had less muscle damage and demonstrated a blunted inflammatory response at 48 h and 96 h postexercise compared with preexercise. Functionally, these same subjects reported significantly less muscle pain during active elbow extension and were found to have significantly greater range of motion and less upper limb disability at 48 h and 96 h postexercise.

Muscle fiber disruption is known to occur as a direct result of high-intensity resistance exercise, especially if the subject is unaccustomed to the exercise and the exercise utilizes lengthening (eccentric) muscle actions (3, 7, 22). Common clinical signs and symptoms of muscle fiber disruption include diminished strength, a delayed onset of muscle soreness (DOMS), stiffness in the affected muscles, and elevated levels of inflammatory molecules in the blood (3, 22, 23). Muscle damage occurs in two distinct phases: primary and secondary. Primary muscle damage occurs as a direct result of the mechanical stress from exercise and secondary damage is caused by oxidative stress and the cumulative effects of the inflammatory cascade (7, 14).

We hypothesized that oral consumption of EMW would protect skeletal muscle from exercise-induced damage by reducing the extent of biochemical stress from inflammation. In the present study, subjects who consumed the EMW were found to have lower levels of CK and hsCRP at 48 h and 96 h postexercise compared with the group that consumed the placebo water. At 24 h postexercise, CK levels are likely to reflect the effects of primary damage from the initial mechanical insult to the muscle as well as the early effects of secondary damage from the inflammatory response. The continued elevation of CK levels at 48 h and 96 h postexercise is more indicative of secondary damage from inflammatory amplifiers and reactive oxygen species.

Recent research has shown a reduction in the extent of exercise-induced muscle damage and inflammation in subjects who consumed capsules or beverages supplemented with fruit, berry, and/or vegetable extracts prior to completing strenuous resistance exercise (2, 5, 12, 13, 24). Phytochemicals derived from the fruit extracts were speculated to have anti-inflammatory and cytoprotective properties that helped improve recovery from high-intensity resistance exercise (2, 24). Although the exact molecular and cellular mechanisms mitigating the anti-inflammatory and cytoprotective effects of the EMW remain to be determined, they are likely based on the presence of “charged stabilized nanostructures” (CSNs), which in the case of RNS60 has been linked to effects on intracellular signaling pathways that influence inflammation, cell death, and survival (9–11, 18; Khasnavis S, Ghosh S, Watson R, Pahan K, unpublished observation).

With particular relevance to cytoprotection, it has been observed (Jana A, Ghosh S, Watson R, Pahan K, unpublished) that RNS60 protected cultured neurons challenged with amyloid-beta peptides, known to cause Alzheimer’s disease. Using a mouse model of Parkinson’s disease, it has also been observed (Khasnavis S, Ghosh S, Watson R, Pahan K, unpublished observation) that RNS60 reduced inflammation and neuronal degeneration in animals challenged with methylphenyl-tetrahydropyridine intoxication compared with control saline. In an in vitro study, Khasnavis et al. (18) were able to show that RNS60 inhibited microglial activation of NF-κB. In this particular study, active glial cells exposed to RNS60 responded by expressing lower levels of proinflammatory mediators than cells exposed to a placebo saline. NF-κB activation is known to play a role in amplifying the inflammatory response after eccentric contraction-induced muscle damage by instigating the synthesis and release of proinflammatory mediators such as TNF-α, IL-1, and IL-6 (19, 20). In a study using humans, Hyldeahl et al. (15) were able to show that repeated eccentric muscle actions can stimulate NF-κB activation in and around damaged myofiber. EMW may be able to downregulate the inflammatory response and improve recovery after strenuous arm exercise by a similar mechanism; however, more research is needed to identify a direct link between EMW consumption and its effects on inhibiting NF-κB activation.

Our functional recovery findings did not show any significant differences in muscle strength or soreness between the experimental and placebo-control groups after exercise. Both the EMW and placebo-control groups did demonstrate strength deficits of ~22% at 48 h, followed by an equal recovery with residual strength deficits of 12–13% at 96 h postexercise. Subjects who consumed EMW, however, reported less pain with active elbow extension than subjects who consumed the placebo water. The disparity in clinical and statistical significance between the scores for muscle soreness using pressure algometry and the scores for pain with active elbow extension may be explained by the manner in which soreness was assessed. Pain with active elbow extension required subjects to rate their level of pain during an active lengthening or stretch of the biceps muscle, while muscle soreness ratings required subjects to rate their level of soreness as a result of focal pressure being applied to the biceps muscle to elicit the mechanical pain threshold. Mechanical pain threshold measures the subject’s sensitivity to pressure applied to the muscle using a compressive force with the muscle relaxed at a fixed position or length, while active elbow extension places a tensile or stretch load to the muscle. The active test may be a more functional test and one that is specific to strain-induced injury from resistance exercise. Future research should be aimed toward determining the efficacy of rating muscle pain and soreness using compressive or applied forces to a muscle as opposed to a lengthening or stretch load.

Another common symptom of DOMS is muscle and joint stiffness, which is likely to occur as a result of intramuscular microedema. The extent of muscle stiffness was quantified in...
our study by measuring the angle of the elbow while the arm was held relaxed at the side of the body. The group that consumed the EMW lost only 5° of elbow extension (3% deficit) while the group that consumed the placebo beverage lost 17° of extension (10% deficit) at 48 h postexercise. At 96 h postexercise the EMW group recovered almost all range of motion while the placebo group demonstrated a residual extension deficit of 8.2° (5% deficit). It is likely that the blunted inflammatory response in the EMW group reduced the amount of edema within the affected muscles resulting in reduced stiffness and quicker recovery compared with the placebo group.

Upper limb disability was self-reported using the DASH questionnaire. Subjects who consumed EMW reported less functional limitations during activities of daily living at 48 h and 96 h postexercise. Lower levels of upper limb disability in the EMW group can be attributable to the reports of less muscle pain and stiffness that likely resulted from less exercise-induced muscle damage and inflammation.

The potential benefits of consuming EMW were shown by the level of agreement between the extent of active pathology, symptomatic response, and functional impairment in the subjects following the bout of resistance exercise, and how these manifestations dictated the course of recovery especially in the subjects that consumed the EMW compared with the group that consumed the unprocessed water. The group that consumed the EMW was shown to incur less muscle damage and postexercise inflammation in the days following the bout of intense exercise, and this was expressed explicitly by these subjects in their reporting of lower levels of pain and disability than the group that consumed the unprocessed water.

A unique feature of the EMW is its versatility with regard to applications in an athletic environment. Athletes can consume the EMW not only as an aid to maintain hydration before, during, and after exercise, but additionally with the intent of reducing muscle damage and inflammation, thus improving recovery after intense exercise. The combined fluid replacement/hydration and recovery effects of consuming EMW can benefit athletes by effectively mitigating the effects of physiological stressors such as intense exercise training and competition.

Besides controlling the oral consumption of the EMW or placebo water, we did not control the intake of additional water during the course of the study, and this was a potential study limitation. We assume, however, that any variability induced by additional water consumption is of negligible effect. While the subjects did obtain some additional water from their meals (e.g., fruits and vegetables) they likely did not find the need to consume large quantities of water in addition to the daily allotment of water consumed as part of the study protocol. Another limitation of the study was the use of a small number of biomarkers for muscle damage and inflammation. Other limitations include the use of only one dosing schedule for subjects, and the use of a small muscle mass for testing (biceps brachii). The study participants were physically active; however, they were not considered to be elite or highly trained competitive athletes, thus limiting the generalizability of the results. Future studies should be designed to incorporate the use of larger muscle groups, and thus larger muscle mass, as well as using more dynamic resistance exercises such as Wingate cycle ergometry testing. We also recommend expanding the number and types of outcome measures as well as designing studies that focus more closely on the benefit as it relates to the volume of the EMW consumed.

Conclusion. Consuming EMW significantly reduced exercise-induced muscle damage and inflammation and improved functional recovery postexercise. Our data provide evidence that EMW may help prevent muscle damage by reducing postexercise inflammation; however, the EMW has not been tested in competitive athletes undergoing intense bouts of training. Further research will be necessary to determine whether consuming EMW is effective in reducing exercise-induced muscle damage and improve recovery time in athletes during high-level training maneuvers.

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GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: P.A.B. conception and design of research; P.A.B. and J.S.M. analyzed data; P.A.B. interpreted results of experiments; P.A.B. prepared figures; P.A.B., K.L.K., and J.S.M. drafted manuscript; P.A.B., K.L.K., and J.S.M. edited and revised manuscript; P.A.B., K.L.K., and J.S.M. approved final version of manuscript; K.L.K. and J.S.M. performed experiments.

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