The Effects of Pre-Exercise Ginger Supplementation on Muscle Damage and Delayed Onset Muscle Soreness

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Ginger possesses analgesic and pharmacological properties mimicking non-steroidal antiinflammatory drugs. We aimed to determine if ginger supplementation is efficacious for attenuating muscle damage and delayed onset muscle soreness (DOMS) following high-intensity resistance exercise. Following a 5-day supplementation period of placebo or 4 g ginger (randomized groups), 20 non-weight trained participants performed a high-intensity elbow flexor eccentric exercise protocol to induce muscle damage. Markers associated with muscle damage and DOMS were repeatedly measured before supplementation and for 4 days following the exercise protocol. Repeated measures analysis of variance revealed one repetition maximum lift decreased significantly 24 h post-exercise in both groups (p < 0.005), improved 48 h post-exercise only in the ginger group (p = 0.002), and improved at 72 (p = 0.021) and 96 h (p = 0.044) only in the placebo group. Blood creatine kinase significantly increased for both groups (p = 0.015) but continued to increase only in the ginger group 72 (p = 0.006) and 96 h (p = 0.027) post-exercise. Visual analog scale of pain was significantly elevated following eccentric exercise (p < 0.001) and was not influenced by ginger. In conclusion, 4 g of ginger supplementation may be used to accelerate recovery of muscle strength following intense exercise but does not influence indicators of muscle damage or DOMS. Copyright © 2015 John Wiley & Sons, Ltd.

Keywords: Zingiber officinale; exercise; gingerol; antiinflammatory; nutraceutical.

INTRODUCTION

For centuries, ginger (Zingiber officinale Roscoe) has been an important ingredient in Chinese, Ayurvedic, and Tibb-Unani herbal medicines (Ali et al., 2008). The constituents of ginger vary depending on the place of origin and whether rhizomes are fresh or dry. The potent aroma of fresh ginger is highly attributed to the gingerols (Ali et al., 2008). Gingerols can further be converted into shogaol, zingerone, and paradol constituents (Thomson et al., 2002). Ginger and several of its constituents inhibit activity of COX-1 and COX-2, block leukotriene synthesis, and block the production of interleukins, and tumor necrosis factor alpha in activated macrophages (Black et al., 2010; Flynn et al., 1986). Thus, these antiinflammatory actions that may help reduce inflammation, such as the exercise-induced muscle damage, are recognized as a product of participating in unfamiliar or strenuous physical activity (Cooke et al., 2009).

Exercise-induced increases in cytokines, such as TNF-α, are regulated by a variety of stimulators and suppressors within inflammatory pathways, including the COX-prostaglandin cascade (Davis et al., 2007), which leads to further increased inflammatory cytokine production, pain, and performance deficits in muscle function. Muscle damage is indirectly quantified by elevated blood levels of creatine phosphokinase, also referred to as creatine kinase (CK), and lactate dehydrogenase (LDH) (Overgaard et al., 2002; Totsuka et al., 2002). Therefore, CK and LDH measurements may be used as an indicator of muscle fiber damage (Ohtani et al., 2006). Skeletal muscle pain, which may be attributed to the alteration of muscle fiber, followed by ion imbalances and inflammation, is more frequently recognized as delayed onset muscle soreness (DOMS) (Hilbert et al., 2003). DOMS is more commonly recognized by individuals as muscle soreness and tenderness, which develops over many hours and is maximal 1 to 2 days after exercise (Prasartwuth et al., 2005).

Given that ginger has antiinflammatory and analgesic properties, it follows that it may be used to reduce the damage and consequent DOMS following high-intensity exercise. To our knowledge, the current literature does not provide specifications for a suggested dosing of ginger. Past research has utilized a 2 g dosage; this has

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Abbreviations: CK, creatine kinase; COX, cyclooxygenase; DOMS, delayed onset muscle soreness; LDH, lactate dehydrogenase; ROM, range of motion; TNF-α, Tumor Necrosis Factor-α; VAS, visual analog scale

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not demonstrated a consistent link in reducing exercise-induced muscle soreness (Black et al., 2010; Black and O’Connor, 2010). Therefore, the purpose of this study was to investigate ginger as a possible analgesic treatment for inflammatory markers generated from exercise-induced muscle damage. We hypothesized that 5 days of treatment using a 4 g dosage of ginger will demonstrate a substantial reduction in markers of muscle damage following a bout of eccentric exercise.

MATERIALS AND METHODS

Subjects. Male and female subjects were recruited through fliers, e-mail listservs, and word of mouth. Those individuals that responded were further screened to determine eligibility within the study. The subject pool consisted of 20, non-weight trained men and women, with ten randomly assigned to the ginger group and ten to the placebo group. Men and women were evenly distributed within each group (five men and five women in each). Subjects were instructed to maintain a similar activity level and diet for the duration of the study. Descriptive characteristics for the subjects are located in Table 1.

Experimental design. This study was conducted in the Human Physiology Laboratory at Marywood University in Scranton, PA. All subjects underwent written informed consent approved by the university’s Institutional Review Board (225766-3). This study was conducted as a double-blind, randomized placebo-controlled trial.

Subjects participated in the study for a total of 12 days of research procedures, including a 5-day treatment period, as outlined in Fig. 1. On day 1 of the study, baseline measurements (BASE₁) for elbow flexion strength, range of motion, upper arm circumference, skin temperature, and muscle soreness were completed for all participants. Blood samples were also collected for serum CK and LDH measurements. On day 2 (BASE₂), subjects repeated the day 1 procedures and were provided with a container holding a 5-day supply of 4 g ginger supplement or placebo and given instructions for self-administration of their randomly assigned intervention. Subjects began consuming their respective treatment on day 3.

Subjects were asked to consume their assigned treatment once daily for 5 days (days 3–8). A specific time for consumption was not specified, but subjects were asked to be consistent in their administration time and record the time of supplementation in a log provided to them. Subjects were informed they could consume their respective treatment with fluids (e.g. one glass of water), although fluid consumption was not standardized. Supplementation took place over a 5-day period. On day 8 of the study, subjects participated in an eccentric exercise protocol. Subjects underwent testing procedures immediately before (PRE-EX) and after the eccentric exercise protocol (POST₀), and testing procedures every 24 h thereafter for 4 days (POST₂₄, POST₄₈, POST₇₂, and POST₉₆). Visual analog scale of soreness (VAS), one repetition maximum elbow flexion exercise performance (1RM), and blood samples were not collected at POST₀ but were collected for all other post-exercise measurements. All post-exercise tests were performed at a consistent time each day. In most instances, subjects completed the study over a course

Table 1. Demographic data of subjects

<table>
<thead>
<tr>
<th>Age (year)</th>
<th>Placebo (n = 5)</th>
<th>Ginger (n = 5)</th>
<th>Placebo (n = 5)</th>
<th>Ginger (n = 5)</th>
<th>Placebo (n = 5)</th>
<th>Ginger (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>29 ± 4</td>
<td>24 ± 4</td>
<td>27 ± 7</td>
<td>27 ± 5</td>
<td>32 ± 9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>183 ± 5</td>
<td>180 ± 7</td>
<td>165 ± 2</td>
<td>160 ± 7</td>
<td>174 ± 10</td>
<td>170 ± 012</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>85 ± 15</td>
<td>86 ± 15</td>
<td>60 ± 1.0</td>
<td>56 ± 11</td>
<td>72 ± 17</td>
<td>71 ± 20</td>
</tr>
</tbody>
</table>

Figure 1. Research timeline and statistical comparison. ANOVA, analysis of variance.
of 12 consecutive days. If schedule conflicts did not allow consecutive visits, subjects completed BASE1 and BASE2, and supplementation did not begin until 5 days prior to their third visit. All remaining testing sessions then occurred on consecutive days.

**Supplementation.** Subjects were randomly assigned to the ginger group or placebo group using a computerized randomization scheme. Supplement powder was measured using a high-precision balance analyzer and filled in capsules using a commercialized capsule preparation system by the principal investigator of the study. Both ginger and placebo supplementation were administered in containers as identical capsules, with subjects and data collection team members blinded to treatment group. Subjects in the experimental group ingested 4 g of ginger once a day for 5 days (20 g⋅d⁻¹ for 5 days); whereas, subjects in the control group ingested 4 g of placebo (dextrose) once a day for 5 days (20 g⋅d⁻¹ for 5 days). The 5-day loading period was selected based on creatine supplementation studies, during which creatine is administered for 5 days to enhance muscular performance (Bemben and Lamont, 2005). Subjects were provided with directions for supplementation consumption. Because this study aimed to examine the effects of a 5-day loading dose of ginger, subjects completed their respective treatment period on the day prior to the eccentric exercise protocol in attempt to minimize potential confounding factors that could influence exercise performance (i.e. acute effect of ginger supplementation).

**Muscle function, circumference, and muscle soreness tests.** Muscle soreness development may vary with changes in muscle strength and range of motion, upper arm circumference, and plasma CK activity (Zainuddin et al., 2005). Therefore, indirect assessment markers of muscle damage were gathered to determine the depth of muscle damage for each subject within the study.

Concentric one repetition maximum (1RM) of the elbow flexors was measured on days 2, 8 (pre-exercise only), and 9–12. For each 1RM measurement, the subject was required to perform a warm-up of one set of 10–12 repetitions at a load equal to 30% of their 1RM. Participants were seated on a preacher curl bench with feet flat on the floor. The upper arm was placed on a pad of the bench with the shoulder positioned at approximately 45° of flexion. After completion of the warm-up, a 2-minute rest was provided, and the resistance was adjusted to the level recorded on the previous visit (for days 8–12); if necessary, the resistance was decreased (Smith et al., 1994). 1RM was determined utilizing the guidelines set by the National Strength and Conditioning Association (Baechle and Earle, 2008).

Range of motion was measured as a surrogate of inflammation and neuromuscular dysfunction of the elbow flexors. Active range of motion was measured for both flexion (ROM\textsubscript{FLEX}) and extension (ROM\textsubscript{EXT}). A goniometer was used to measure the joint angle of the elbow, which was defined as the midpoint of the wrist, the lateral humeral epicondyle, and the acromion process (Black et al., 2010). For ROM\textsubscript{FLEX}, subjects were asked to attempt to fully flex their elbow, attempting to touch their shoulder with the palm of the hand, such that a lower number represented a smaller joint angle and thus a greater range of motion. Measurements for active ROM\textsubscript{EXT} were performed such that subjects were asked to maximally extend the elbow joint of the non-dominant arm. Subjects were instructed to keep their elbow joint of the non-dominant arm close to their body throughout the duration of both movements. Range of motion was measured on days 1, 2, 8 (pre-exercise and post-exercise), and 9–12.

Arm circumference was determined with the use of a spring tape measure on days 1, 2, 8 (pre- and post-exercise), and 9–12. The tape was then used to measure swelling at four sites: 4, 6, 8, and 10 cm above the elbow joint as the subject stood in a relaxed position with arms placed by his or her side (Stay et al., 1998). The mean value of these measurements was utilized for arm circumference assessment. A permanent marker was used to mark the four sites to be measured. Measurements were recorded to the nearest 0.1 cm.

Muscle soreness was assessed using a visual analog scale (VAS) of a 100 mm line, where 0 mm indicated no pain and 100 mm indicated extremely painful. VAS is a widely utilized method for determining pain intensity (Black et al., 2010; Rawson et al., 2001). Muscle soreness was evaluated on days 1, 2, 8 (pre-exercise and post-exercise), and 9–12.

Skin temperature was determined using an electronic surface thermometer (Thermalert Model TH-8, Physitemp Instruments, Inc., Clifton, NJ). Each temperature probe was connected to a separate gauge on the system, and ambient temperature calibration of the temperature probes was performed before each reading. The control arm (non-exercised) was used to compare the effects of supplementation on this arm compared with the exercised arm, as well as to validate that the exercise protocol was effective (i.e. greater temperature in the exercised arm). To ensure consistent readings, the mid-belly of the arm was defined as the point measured halfway between the acromion and the lateral epicondyle and a mark was made on each arm with a permanent marker. The temperature probe was placed on the mark and taped in place. Subjects sat in a temperature-regulated room (20 °C) to ensure a standardized environment for each visit. Measurement of the skin temperature took place for 5 min while the subjects sat with both arms relaxed on top of their thighs. Gauge 1 was standardized as the control (dominant) arm for each subject. Following the 5 min time span, the temperature of each arm was recorded. Skin temperature was evaluated on days 1, 2, 8 (pre-exercise and post-exercise), and 9–12.

**Blood samples.** Blood samples were collected into serum separator tubes from the antecubital veins during each visit on days 1, 2, and 8 and every 24 h starting with day 9 for 4 days subsequently. Tubes were centrifuged for 15 min at 2500 rpm and chilled to 4 °C and then analyzed by commercial laboratory (Proven Diagnostics, Geisinger Wyoming Valley Medical Center, Wilkes-Barre, PA) for CK and LDH measurements. Normal reference ranges for men were 30–259 U/L (CK) and 90–250 U/L (LDH). Normal ranges for women were 34–174 U/L (CK) and 90–250 U/L (LDH).
Eccentric exercise muscle damage protocol. Subjects sat on a preacher curl bench with the arm positioned anterior to the torso with the posterior aspect of the upper arm placed on a padded support. The subject’s forearm was supinated with the wrist resting on the bench. The use of the preacher bench ensured that the elbow flexors were isolated as biceps curls were performed. Repeated eccentric contractions of the non-dominant arm were used to induce DOMS in the elbow flexors. Subjects performed four sets of concentric/eccentric actions of ten repetitions or to failure at 80% of 1RM. Subjects then performed four sets of eccentric actions of ten repetitions or to failure at 100% of 1RM. These procedures were similar to others that exhibited a generation of DOMS between 80% and 100% of 1RM (Black et al., 2010; Ciccone et al., 1991; Dudley et al., 1997; Stay et al., 1998). For each eccentric contraction, subjects were instructed to slowly lower the dumbbell from a fully flexed to a fully extended position over 5s while the researcher verbally counted. The process was repeated until control over the speed of descent could no longer be attained. To emphasize the eccentric contraction, the weight was returned to a starting position of full elbow flexion by the investigator. A rest period of 1 min was provided between sets (Stay et al., 1998).

Statistical analysis. All statistical procedures were performed using SPSS v20.0 (Armonk, NY). Statistical significance was set at \( p < 0.05 \) a priori.

Normality tests and data transformations. Raw data were examined for normality using the Kolmogorov–Smirnov test. Variables that were not normally distributed were natural log (ln) transformed.

Measurement error and reproducibility of dependent variables. Measurement error and reproducibility parameters were computed to provide a reference standard for interpreting changes following the supplementation periods and eccentric exercise protocol. The measurement error was calculated as the residual mean square error obtained from the repeated measures analysis of variance (Bland and Altman, 1996). The measurement error is the common within subject standard deviation (Bland and Altman, 1996). The measurement error was multiplied by 2.77 to obtain the reproducibility. The reproducibility is defined as the difference between two measurements obtained on two different days for the same subject is expected to be less than 2.77 \times \) the measurement error for 95% of pairs of observations (Bland and Altman, 1996). Thus, any pair of changes that is larger than the reproducibility value has a 97.5% chance that it is a true and meaningful change (Hopkins, 2000). However, the reproducibility value may be too stringent of a criterion for change, so the smallest measureable change (SMC) is also calculated. The SMC is half the reproducibility (Hopkins, 2000), and any pair of changes that is equal to or larger than the SMC value has an 84% probability that the change observed is a real meaningful and measureable change. Pearson’s \( r \) and Spearman’s \( \rho \) (along with the 95% confidence interval) were performed to look at the association between both baseline measurements.

Comparisons of subject characteristics. Independent \( t \)-tests were performed to determine if any differences in subject characteristics existed between groups.

Comparisons of responses across time and between treatments. Repeated measures analysis of variance (ANOVA) was utilized to evaluate differences in dependent variables between test sessions and treatment groups. Test session (e.g. BASE1 and BASE2) was the repeated factor. Treatment group (placebo vs. ginger) was the between-subjects factor. When the assumption of sphericity was violated, the Greenhouse–Geisser correction was used when epsilon \(< 0.75\), and the Huynh–Feldt correction was used when epsilon \(\geq 0.75\). Two separate series of RM ANOVA were performed for each dependent variable (Fig. 1).

The first series of RM ANOVA (ANOVA-1) included BASE1, BASE2, POST1A, and POST1B for the repeated factor and represented the pre-DOMS data. This allowed for determination of baseline differences between treatment groups (i.e. ginger BASE1 vs. placebo BASE1), verified that dependent variables were remained stable before each respective treatment (i.e. BASE1 vs. BASE2 for each group), evaluated whether the intervention influenced dependent variable responses prior to exercise (i.e. BASE1 and BASE2 vs. PRE-EX), and determined acute changes in dependent variables following the exercise protocol (i.e. PRE-EX vs. POST0).

The repeated factors in the second series of RM ANOVA (ANOVA-2) were data from POST24, POST48, POST72, and POST96 and represented the DOMS data. This allowed for the time course of post-exercise changes in dependent variables to be evaluated (e.g. POST24 vs. POST96 within the ginger group), and how this differed between groups (e.g. placebo POST72 vs. ginger POST72).

RESULTS AND DISCUSSION

The present study revealed that 5 days of ginger supplementation may accelerate the recovery of maximal strength following a high-load eccentric exercise protocol. As demonstrated in Table 2, the dependent variables measured were generally very reproducible.

A summary of statistical findings for each dependent variable can be found in Table 3. Strength is expected to be initially disrupted when participation in unaccustomed exercise is performed, and this was observed with significant differences in 1RM between baseline and post-exercise 1RM \( (p < 0.001) \). A gradual recovery in strength was observed, such that 1RM was significantly \( (p < 0.005) \) lower at POST34 than all other post-exercise measurements across both groups. Ginger did influence recovery, as evidenced by a significant time \( \times \) treatment interaction \( (p = 0.048) \) for 1RM (Fig. 2A). The ginger group showed a significant improvement in 1RM during the early stages of DOMS (POST24 to POST48) \( (p = 0.002) \), but this did not occur in the placebo group \( (p = 0.089) \). Interestingly, the placebo group continued to display improvements in
EFFECT OF GINGER ON EXERCISE-INDUCED MUSCLE SORENESS

Table 2. Reproducibility of the measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>Measurement error</th>
<th>Reproducibility</th>
<th>SMC (95% CI for the −)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK (IU/L)</td>
<td>0.94 (0.84–0.98)</td>
<td>0.96 (0.90–0.99)</td>
<td>0.98 (0.94–0.99)</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>0.75 (0.66–0.90)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAS (mm)</td>
<td>0.65 (0.29–0.85)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circumference (cm)</td>
<td>0.98 (0.94–0.99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROM – flexion (deg)</td>
<td>0.95 (0.89–0.99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROM – extension (deg)</td>
<td>0.95 (0.90–0.99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin temp – non-dominant arm (°C)</td>
<td>0.95 (0.90–0.99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin temp – dominant arm (°C)</td>
<td>0.95 (0.90–0.99)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CV, Coefficient of Variation; SMC, smallest measureable change; CI, confidence interval; CK, creatine kinase; LDH, lactate dehydrogenase; VAS, visual analog scale; ROM, range of motion; NS, non-significant.

No values were determined for 1RM as this measure only had one baseline value. CV for VAS could not be determined due to multiple individuals having identical values for repeated baseline measurements.

Table 3. Summary of RM ANOVA p-values from pre-DOMS data (ANOVA-1) and delayed post-exercise data (ANOVA-2)

<table>
<thead>
<tr>
<th>Variable</th>
<th>ANOVA-1</th>
<th>ANOVA-2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time</td>
<td>Time × treatment</td>
</tr>
<tr>
<td>CK</td>
<td>0.499</td>
<td>0.953</td>
</tr>
<tr>
<td>LDH</td>
<td>0.417</td>
<td>0.370</td>
</tr>
<tr>
<td>1RM</td>
<td>&lt;0.001*</td>
<td>0.682</td>
</tr>
<tr>
<td>VAS</td>
<td>&lt;0.001*</td>
<td>0.596</td>
</tr>
<tr>
<td>Circumference</td>
<td>&lt;0.001*</td>
<td>0.237</td>
</tr>
<tr>
<td>ROM – flexion</td>
<td>&lt;0.001*</td>
<td>0.203</td>
</tr>
<tr>
<td>ROM – extension</td>
<td>0.120</td>
<td>0.795</td>
</tr>
<tr>
<td>Skin Temp – ND</td>
<td>&lt;0.001*</td>
<td>0.306</td>
</tr>
<tr>
<td>Skin Temp – D</td>
<td>0.238</td>
<td>0.232</td>
</tr>
</tbody>
</table>

ANOVA, analysis of variance; DOMS, delayed onset muscle soreness; CK, creatine kinase; LDH, lactate dehydrogenase; RM, repetition maximum; VAS, visual analog scale; ROM, range of motion; ND, non-dominant; D, dominant.

*Statistically significant at p < 0.05 level.

strength after POST48 (POST48 vs. POST72; p = 0.021; POST72 vs. POST96; p = 0.044), yet the ginger group showed no other significant improvements (POST48 vs. POST72; p = 0.821; POST72 vs. POST96; p = 0.834). Thus, it appears that ginger accelerated the initial improvement in muscle strength but did not influence strength 72 and 96h after the exercise bout. Indications as to why the ginger group did not continue to improve may be due to the initial potency or bioavailability of necessary constituents no longer being available, given that supplementation had ceased during the recovery period.

Ginger supplementation also appeared to increase indicators of muscle damage, as evidenced by increased plasma CK and impaired flexibility (Fig. 2B and C). There was a significant time main effect for CK (p = 0.015) and ROMFLEX (p < 0.001), with a significant time × treatment interaction (p = 0.028 for each). Pairwise comparisons revealed no significant differences in CK between groups for any of the days, although there was a strong trend (p = 0.056) for CK to be greater in the ginger group than placebo for POST96 when one extreme value was included, although this trend was not apparent when the extreme value was removed. However, pairwise comparisons revealed CK significantly increased from POST48 to POST72 (p = 0.006) and POST72 to POST96 (p = 0.027) and, thus, an overall increase from POST34 to POST96 (p = 0.010) in the ginger group even with the extreme values removed. In other words, after POST34, CK steadily increased in the ginger group but not the placebo group. Pairwise comparisons revealed ROMFLEX was significantly greater for ginger than placebo on POST34 (p = 0.014) but not on any other days. There were significant decreases in ROMFLEX between POST34 versus POST72 (p = 0.029) and POST96 (p = 0.005) in the placebo group, but not POST34 versus POST48 (p = 0.081). In the ginger group, there were significant differences from ROMFLEX between POST34 and all other days (p < 0.005), as well as POST48 versus POST72 (p = 0.013).

The multitude of changes indicative of muscle damage confirmed the protocol used was effective in inducing muscle damage and therefore is valid for studying the effects of ginger supplementation on muscle damage. Significant increases in VAS, circumference, and TEMPND at POST0 (p < 0.001 for each) are consistent with expected physiological changes in muscle following an acute bout of exercise. Post-exercise, VAS was reduced each day compared with that of the previous day (p ≤ 0.002 for each, Fig. 2D), except for POST48, which was not different than POST34 (p = 0.228). This would be expected as DOMS typically peaks 24 to 48h after exercise, and some individual differences would

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be expected. The lack of time × treatment effect for VAS (p = 0.145) indicates ginger does not influence muscle soreness itself.

Accelerated recovery in muscle strength could be useful for athletes competing in events where multiple bouts of maximal exercise must be performed in a short period of time (e.g., a qualifying round of competition preceding a final round by 48 h). However, post-exercise ROMFLEX was actually impaired following ginger supplementation, and therefore, athletes who rely on flexibility (e.g., gymnasts) may be negatively impacted by ginger. Ultimately, athletic performance represents the complex interaction of multiple factors, and therefore, future studies examining ginger and other nutraceutical compounds as ergogenic aids should attempt to incorporate more holistic measures of athletic performance whenever possible (e.g., sprinting times, agility tests, and sport-specific movements). Indeed, recent evidence suggests that 6 weeks of 1.5 g of ginger per day may attenuate exercise-associated inflammatory markers in male distance runners (Zehsaz et al., 2014).

The mechanism behind this accelerated recovery may be related to ginger delaying actual muscle damage itself, as evidenced by a more gradual increase in CK compared with placebo. While CK is indeed associated with muscle damage and DOMS, it must be emphasized that the etiology of DOMS is multifactorial and may be dissociated from changes in performance (Lewis et al., 2012). As such, it is not surprising that CK was significantly altered by ginger, whereas VAS was not. It is possible that ginger loading may have increased muscular function, which allowed individuals to perform more physical work during the eccentric muscle damage protocol, which may have led to more muscle damage. It is also possible that the ginger constituents have a limited window of anti-inflammatory function, such that termination of ginger supplementation removed the protective effect against muscle damage. This is consistent with data from Black (Black and O’Connor, 2010), whereby individuals who received 2 g of ginger 24 h after an eccentric muscle damage protocol exhibited reduced pain the following day, whereas individuals ingesting placebo did not. While previous studies found ginger supplementation relieved pain associated with eccentric damage (Black et al., 2010; Black and O’Connor, 2010), the VAS data from this study did not. It is possible that continued ginger supplementation is necessary for an analgesic effect. However, it must be considered that the greater number of eccentric contractions performed in this study likely induced...
more muscle damage than other studies, as suggested by comparatively greater VAS values than in previous studies.

There are a few limitations to this study that must be recognized. It must be noted that this study specifically recruited individuals with limited resistance training experience, as eccentric training in this population would likely cause a more extreme response than that in the trained population. This is a common strategy for muscle damage studies (Black et al., 2010; Rawson et al., 2011), but nonetheless, caution must be used in extrapolating these findings to athletes without further study. Lack of strength training experience may explain why a slight learning effect was observed, such that 1RM data was slight, although significantly higher at PRE-EX than BASE2, and suggests the need for greater familiarization of such procedures in future studies. It is also important to note that statistical analysis did not reveal significant differences in these factors between groups (e.g. values for ginger group were not significantly different than placebo group), but there were significant differences within the ginger group not observed in the placebo group (e.g. that for 1RM). As such, some caution should be observed in these interpretations. However, lack of between-groups differences may be an artifact of multiple sampling points combined with a relatively small sample size. Finally, the study was not designed to determine the time course needed before all values returned to baseline, although such full recovery is not typically measured in muscle damage studies (Black et al., 2010; Rawson et al., 2011).

In summary, these findings indicate that a 5-day loading period of 4 g of ginger per day supplement may be a more effective dosage than previously studied 2 g dosages for accelerating recovery of muscle strength following muscle damage. It appears that this ginger supplementation protocol delayed muscle damage but did not prevent it. Termination of the ginger supplementation may explain the lack of effect on DOMS compared with that previous studies and may potentially explain the lack of overall effect on markers of muscle damage. Although there remain many unknowns, the results of this study, combined with multiple others, makes it clear that ginger does indeed have an influence on basic measures of muscle function in relation to exercise, and further studies in this area are needed. Future studies should examine the dose–response relationship of ginger on muscle function, evaluate the effects on trained individuals, and seek to further differentiate the effects of pre- and post-muscle damage ginger supplementation.

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Conflicts of Interest

No conflicts of interest exist for the authors of this study.

REFERENCES


