Effect of Two Regimes of Vitamin C on Delayed Onset Muscle Soreness

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Abstract

The aim of present study was to investigate the physiological effects of two regimes of vitamin C on the delayed onset muscle soreness (DOMS). Thirty seven non-athletic female volunteers (age, 22.02 ± 1.54 yrs) were randomly allocated into 4 groups; Group 1: consumed 100 mg vitamin C (n=9); Group 2: consumed 200 mg vitamin C (n=10); Group 3: consumed placebo (n=9); Group 4: control (n=9). The treatment groups received vitamin C 1 hr before eccentric actions up to 47 hr after actions. The placebo group consumed identical capsules that contained 100 mg lactose. Muscle soreness was induced by performing 70 eccentric contractions of the triceps muscle of the non-dominant side on a modified arm curl machine. The time interval every action was 3 s with a 10 s rest between them and there was 1 min rest between every 10 actions. Perceived muscle soreness (PMS), maximal eccentric contraction (MEC), creatine kinase (CK) enzyme values and elbow range of motion (EROM) were assessed, 1 hr before, and 1 hr, 24 hr, and 48 hr after the eccentric actions. Data were analyzed by two-way analysis of variance (ANOVA) with repeated measures for the time component of the experiment and the following results were concluded: Peak muscle soreness in all groups in the study occurred 48 hr after the contraction except in the group 1 and there was not a significant difference between the 4 groups in muscle soreness. CK activity increased in all groups after eccentric contraction and there was not a significant difference in CK levels between the 4 studied groups. A non-significant reduction of elbow joint ROM was also indicated in all groups of study. There was not significant reduction in MEC in any groups. Therefore consumption 100 or 200 mg vitamin C dose not affect on (DOMS).

Key words: Muscle soreness, Anti oxidant, Vitamin C, Free radical

Introduction

Delayed onset muscle soreness (DOMS) results from high-intensity work involving eccentric exercise contraction (2,7,8,13). It is associated with loss of strength, muscle shortening, elevation of serum CK and muscle disruption (1,4,11).

Muscle soreness may have a number of different causes, including the adverse effects of free radicals (14,15,16). Production of reactive oxygen species during exhaustive exercise well established and the subsequent removal depends on the capacity of the scavenger and antioxidant system (10). If the rise in the level of free radicals exceeds the antioxidant defence capacity of the cells lipid peroxidation will occur (14). A great number of human studies have shown supplementation with antioxidant vitamins has favourable effects on the process of lipid peroxidation (3,5,14).

Vitamin C is a powerful antioxidant scavenging free radicals and ingestion of large amount of vitamin C offers some protection against exercise-induced muscle soreness and lipid peroxidation (16).

Several studies have reported positive effects of vitamin C supplementation on muscle soreness and muscle damage before and after exercise. For example Kaminski and Boal reported a reduction in muscle soreness after consumption 3000 mg vitamin
C for 3 days (6). McBride and coworkers reported a reduction in past – exercise plasma CK activity following a resistance exercise when subjects consumed 1,200 IU of vitamin E for 2 weeks before exercise (9). Shafat et al suggested that 37 days of supplementation with vitamin C and vitamin E reduced the deficit in muscle function normally experienced during and after bouts of eccentric muscle contraction (15).

In contrast Peterson and etal found daily consumption of 500 mg vitamin C and 400 mg vitamin E for 14 days prior to a down hill run didn’t reduce muscle damage when compared with a control group(2). Also acute supplementation (1000 mg) 2 hr prior to exercise or consumption a single dose of 200 mg no effect in reducing muscle soreness or indices of damage (16,18).Therefore the effectiveness of vitamin supplementation on reducing exercise–induced muscle damage has not been consistently demonstrated (3, 5).

Recent studies have shown that excessive doses of vitamin C can lead to toxicity. Furthermore, high vitamin C intakes appear to be occasionally associated with diarrhea and intestinal discomfort possibly due to bacterial fermentation of unabsorbed vitamin C (17). On the other hand vitamin C is water soluble and availability may be increased after a single dose and there may be no need for prolonged supplementation (16).

Therefore the aim of present study was to identify consumption 100 or 200 mg vitamin C before and after eccentric contraction would affect on DOMS.

**Methods**

37 apparently healthy females with no history of vitamin C use participated in this study voluntarily. All subjects in this investigation participated in a familiarization session. During the familiarization session, subjects were informed as to the experimental procedures, completed a personal/medical history form, and signed informed consent statement in adherence with the human subject’s guidelines of Guilan University. Subject’s descriptive characteristics were(mean ±SD) 21±3yrs, 56±5kg, and 158±4cm. Using a single blind manner, the subjects were randomly assigned to the following groups:

- **Group 1**(100 mg vitamin C supplementation, n=9)
- **Group 2**(200 mg vitamin C supplementation, n=10)
- **Group 3**(Placebo, n=9)
- **Group 4**(Control group without supplementation, n=9)

Subjects were instructed to maintain their regular eating habits during the investigation period and ingest the supplements just before exercise, 24 hr and 48 hr after exercise. The supplementation consisted of capsules containing 100 mg and 200 mg of vitamin C, whereas the placebo consisted of similar capsules containing 100mg lactose. To induce DOMS, subjects performed 7 sets of 10 repetitions using 80 % of their eccentric one repetition maximum and only the non-dominant arm (as used previously by Rahmani- Nia et al., 2004). Duration of contraction was 3seconds. Subjects were given 10 seconds rest periods between sets. Dominant arm was determined by dynamometer (Laffayatte, USA). The exercise-testing apparatus consisted of a modified arm curl machine (11). The subject’s arm was place on a designed padded support table that is joined to the arm curl machine. A cuff was placed around the subject’s wrist and the cuff was attached to the strain gauge.1RM using of arm-extension maximal eccentric contraction (MEC) of elbow extensors on the arm curl machine was recorded. Elbow range of motion was measured goniometrically by the researcher. Subjects were seated with shoulder into full extension (11) either to the physiological limitation or to pain tolerance. The measurement of elbow extension on a180 degree goniometric scale was recorded. This procedure was repeated and the two measures were averaged to produce a mean measurement of elbow extension that was used for data analysis.
Perceive muscle soreness was measured at the same time. Upon reaching full elbow extension, the subject indicated the degree of soreness experienced by a 30-point scale (1=normal and 30=very very sore). Blood was taken from Cubital vein of the involved arm. Serum was separated and frozen at −20 °C for subsequent analysis of CK. Total CK was determined using spectrophotometrically at 30 °C using commercially available kit, 47 UV (Sigma Diagnostic, St. Louis Mo). Creatine-kinase, MEC, range of motion (ROM) of elbow joint and perceived muscle soreness were evaluated in four different times:

- On the first day (1hr before the exercise)
- On the first day (1hr after the exercise)
- On the second day (2hr after supplementation)
- On the third day (2 hr after supplementation)

Data were analyzed by two-way analysis of variance (ANOVA) with repeated measures for the time component of the experiment with LSD post-hoc procedures for all daily measurements. Statistical significance was determined as P<0.05. Data are presented as means ±SD.

Results

Results of CK, perceived muscle soreness, ROM of elbow and MEC are presented in table 1,2,3 and 4 respectively. No significant differences (P>0.05) were found between groups 1,2,3 and 4 in 3days.

Table1. Comparison of CK between four groups (mean ±SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1h before exs.</th>
<th>1h after exs.</th>
<th>Second day</th>
<th>third day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group1</td>
<td>94.33±29.97</td>
<td>107.77±29.67</td>
<td>120.77±33.35</td>
<td>101.33±27.50</td>
</tr>
<tr>
<td>Group2</td>
<td>105.8±21.80</td>
<td>112.2±26.93</td>
<td>121.5±25.51</td>
<td>97.8±18.73</td>
</tr>
<tr>
<td>Group3</td>
<td>108±28.46</td>
<td>117.33±37.77</td>
<td>138.66±41.97</td>
<td>96±27.73</td>
</tr>
<tr>
<td>Group4</td>
<td>89±19.29</td>
<td>98.66±26.01</td>
<td>108.66±27.34</td>
<td>82±25.31</td>
</tr>
</tbody>
</table>

Table2. Comparison of perceived muscle soreness between four groups (mean ±SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1h before exs.</th>
<th>1h after exs.</th>
<th>Second day</th>
<th>third day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group1</td>
<td>-</td>
<td>4.66±3.9</td>
<td>13.11±8.71</td>
<td>6.77±3.9</td>
</tr>
<tr>
<td>Group2</td>
<td>-</td>
<td>2.9±2.56</td>
<td>8.5±7.28</td>
<td>9.8±7.03</td>
</tr>
<tr>
<td>Group3</td>
<td>-</td>
<td>2.88±2.68</td>
<td>8.55±5.67</td>
<td>9±5.67</td>
</tr>
<tr>
<td>Group4</td>
<td>-</td>
<td>6±5.73</td>
<td>10.22±8.76</td>
<td>10.33±6.54</td>
</tr>
</tbody>
</table>

Table3. Comparison of ROM of elbow between four groups (mean ±SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1h before exs.</th>
<th>1h after exs.</th>
<th>Second day</th>
<th>third day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group1</td>
<td>145.66±2.22</td>
<td>139.55±4.54</td>
<td>137.55±7.13</td>
<td>142.88±2.88</td>
</tr>
<tr>
<td>Group2</td>
<td>143.2±4.31</td>
<td>137.7±7.5</td>
<td>141.2±6.68</td>
<td>142.5±6.28</td>
</tr>
<tr>
<td>Group3</td>
<td>145.22±3.10</td>
<td>139.88±6.15</td>
<td>137.44±3.83</td>
<td>142.33±3.49</td>
</tr>
<tr>
<td>Group4</td>
<td>146.22±2.67</td>
<td>140.88±7.81</td>
<td>140.55±5.94</td>
<td>144.66±2.67</td>
</tr>
</tbody>
</table>
Table 4. Comparison of MEC between four groups (mean ±SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1h before exs.</th>
<th>1h after exs.</th>
<th>Second day</th>
<th>Third day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>10.5±1.92</td>
<td>8.27±1.76</td>
<td>9.30±1.99</td>
<td>10.50±1.92</td>
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<tr>
<td>Group 2</td>
<td>10.75±1.48</td>
<td>8.37±1.80</td>
<td>9.30±1.54</td>
<td>10.60±1.62</td>
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<tr>
<td>Group 3</td>
<td>9.08±1.08</td>
<td>6.97±1.03</td>
<td>7.86±0.90</td>
<td>9.05±1.09</td>
</tr>
<tr>
<td>Group 4</td>
<td>10.44±1.75</td>
<td>8±1.77</td>
<td>9.05±1.78</td>
<td>10.02±1.89</td>
</tr>
</tbody>
</table>

Discussion and summary

The purpose of this study was to investigate whether vitamin C consumed 1 hr before exercise up to 48 hr after that would influence the extent of muscle soreness following eccentric contraction.

The muscle soreness experienced by the subjects coupled with the reduction in MEC, in ROM and increase in CK activity and would suggest that the eccentric contractions had induced muscle damage. The results of this study indicate that supplementation for 3 days with vitamin C had no effect on DOMS. Similar results have been reported previously (16,8), although in all of these studies only measured biochemical (creatine kinase, myoglobin, malondialdehyde) markers of muscle damage. It has been suggested that the best measure of muscle damage is not biochemical marker such as CK (3,18). Other studies thought have shown positive effects of vitamin supplementation on DOMS (3,9,15).

Differences in the type and duration of exercise performed, duration and kind of vitamin consumed and the fitness of the subjects used maybe important factors in explaining these contrasting results. There are several explanations for the lack of effect of vitamin C supplementation in the present study. There have been some reports that mixed antioxidant supplementation, including vitamin C and E offer some benefits in terms of lipid peroxidation and muscle damage after exercise. Vitamin C is hydrophilic, accumulates in the cytosol and extra-cellular fluid, whereas vitamin E is hydrophobic, and accumulates in structures such as membranes (17). These vitamins are able to protect different cellular compartment. Therefore the combination of vitamin C and E together may provide more protection from oxidative damage than a single nutrient supplementation. Moreover, vitamin C can also act to reduce oxidized vitamin E antioxidant function (15,17).

It has been suggested that all type of leukocyte, including lymphocytes become saturated at vitamin C intakes of 100-200 mg per day (18,19), although it is unclear whether this is also the case for concentration vitamin C in skeletal muscle (19). Therefore another possible explanation is that increased availability of vitamin C led to further uptake by these cells and maybe the plasma do not become saturated. This is important because extra cellular oxidative stress maybe responsible for the amplification of muscle damage. Another possible explanation is that the antioxidant defenses of our subjects were not adequate, so increased availability of vitamin C offered no additional benefit. Active individuals appear to exhibit better vitamin C status than less active people (16), and the subject in the present study were less active. In summary, the results of the present study suggest that daily consumption of 100 or 200 mg vitamin C dose not affect on DOMS.
References