Effect of cooling on muscular health prior to running a marathon

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To examine the effects of a prerace whole-body cold shower on muscle soreness (MS) and on serum creatine kinase (CK) and creatine kinase M B (CK-M B) isoenzyme activities, 16 experienced distance runners were randomly assigned to one of two treatment categories prior to running a marathon: cold shower (n = 8) or without cold shower (n = 8). Venous blood samples were drawn 3 days before the race, 10 minutes before the race, immediately (within 3 minutes) after the race, and at 1, 24, 48, and 96 hours postrace. Nine muscle sites were evaluated for soreness 10 minutes before the race, immediately after the race, and at 24, 48, and 96 hours postrace. The results showed a marked (P < .05) difference between the cold shower group and the group without cold showers for CK-M B/CK ratio, and no difference for CK, CK-M B, and MS. Both CK and CK-M B values peaked at 24 hours postrace. MS occurred most frequently immediately after the race and at 24 hours postrace. The MS was completely resolved in all subjects by 96 hours postrace. The most frequently reported sites of MS were the quadriceps, followed by the gastrocnemius, the soleus, and the tibialis anterior. Severe MS was rated highest at the quadriceps and the soleus, and the least at the gastrocnemius and the tibialis anterior. The data suggest that prerace whole-body cold showers neither prevented the production of serum CK and its M B fraction, nor attenuated MS after a marathon. Peak serum CK and CK-M B activity was not associated with the onset of MS.

(Key words: marathon, serum creatine kinase, CK-M B isoenzyme, muscle soreness)

Little information is available regarding the use of cold treatment prior to strenuous endurance exercise for the prevention or reduction of exercise-induced muscle soreness (MS). The major signs and symptoms of this condition are muscular stiffness, tenderness, pain, and range of motion limitations in the corresponding joints. The pathophysiologic mechanisms that might be behind this condition remain unclear, although many theories exist.1-7

Application of cold treatment after exercise has been systematically examined, and the results have not shown that cold therapy will reduce MS.8 There are traces of CK-M B in many body tissues, but the ratio of CK-M B to CK is highest in cardiac muscle. After skeletal muscle injury, both CK and CK-M B are released in the serum; however, the percentage that is CK-M B remains low.2

Researchers have performed experiments to determine the effects of cooling on physical work and athletic performance, examining such measures as whole-body cold showers or baths, cold sprays or cold packs on the abdominal area, cold towels over the neck or head, water-cooled suits, and cold air circulating over the whole body.7,13-16 Although precompetition warm-up has been shown to improve performance by athletes in primarily sprint and jumping events,13 athletes competing in endurance events of an aerobic nature often benefit from the anti-inflammatory effect resulting from a reduction in intramuscular bleeding8 or antibody formation.9 The application of cryotherapy to treat muscular injury or soreness resulting from exercise has been an accepted practice in sports medicine.

Elevations of total creatine kinase (CK) and its M B isoenzyme (CK-M B) after exercise and contact sports have been reported.10-12 The detection of CK in the circulating blood after strenuous exercise suggests injury to the skeletal muscle.10-12 Muscle damage is related to MS following intense eccentric exercise.4 The characteristics of exercise-induced muscle damage include elevated plasma CK level, inflammatory cell infiltration, a reduced capacity for glycogen storage, MS, and loss of muscle function.13,14 Post–exercise-induced increase in circulating CK appears to be closely related to the type and intensity of the exercise.15

The efflux of muscle enzymes following high-intensity eccentric exercise suggests that there is irreversible destruction of muscle fibers.3 The probable cause of cellular destruction includes physical shearing of membranes or filaments by excessive physical loads, disturbances in cell volume or in energy state, or transient ischemia from force development.15 However, the rise in plasma CK level following intense exercise is not a sensitive indicator of skeletal muscle damage.14,15 There are traces of CK-M B in many body tissues, but the ratio of CK-M B to CK is highest in cardiac muscle. After skeletal muscle injury, both CK and CK-M B are released in the serum; however, the percentage that is CK-M B remains low.2

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application of cold. The various methods of external body cooling are often used during exercise or competition to reduce thermal loads that can impair performance and predispose the athlete to heat injury. 

Marathon and triathlon races are occasionally conducted on cold, windy, or rainy days in northern America. However, because little information is available for sports medicine physicians and runners regarding the effect of cold rain or cold water (for example, triathlon) on the athletes’ muscular health, it is essential that a full study be conducted to collect data on this important topic, under controlled experimental conditions. The purpose of this study was to obtain preliminary data by examining the effect of a cold shower prior to a marathon on the production of CK and CK-MB and on the development of MS. It was hypothesized that a prerace cold shower prior to a marathon might delay or reduce the appearance of CK or CK-MB in the blood and/or attenuate exercise-induced MS.

Materials and methods

Sixteen marathoners, 10 males and 6 females, participated in this study. A total of 10 runners (5 in the cold shower [CS] group and 5 in the without cold shower [WOS] group) had completed at least one previous marathon. Their recent (within the past 3 years) best marathon finishing times were as follows: CS group mean, 242.0 ± 42.3 minutes (n = 8); WOS group mean, 230.2 ± 25.2 minutes (n = 8). The protocol for this study was reviewed and approved by the Institutional Review Board, and each subject gave informed consent. Subjects older than 40 years were given a 12-lead ECG-monitoring grade exercise tolerance test before the race for all subjects. Following the marathon, blood samples were also collected in the following sequences: immediately after the race and at 1, 24, 48, and 96 hours postrace. Five milliliters of venous blood were drawn from an antecubital vein into chilled in crushed ice. Within 2 hours, the samples were centrifuged and stored at -20°C until assayed. The serum was used for determination of total CK using the modified ultraviolet enzymatic method described by Oliver and others. For the assay of CK-MB, the immunoenzymatic assay method described by Witherspoon and others was applied, using the Photon Immunoassay Analyzer as the measuring device (Tandem-E CKM B, Hybritech Inc, San Diego, Calif). Unlike electrophoresis and ion exchange column chromatography methods, this assay procedure is not affected by increased CK-M or CK-BB concentrations and is specific for CK-MB. The unit for total CK is expressed in units per liter (U/L) and in ng/mL for CK-MB. Because the ratio of CK-MB to total CK in skeletal muscle is considerably lower than in the cardiac muscle, the CK-MB result is also expressed as a ratio to total CK. The ratio is calculated using percent relative index: %RI = (CK-M B/total CK) × 100.

Two-way analysis of variance (groups × sampling times) with repeated measures was used to determine statistically significant differences for CK, CK-MB, CK-M B/CK ratio, and M S. In the presence of a significant F ratio, Tukey’s post hoc test was used to locate the source of the difference. The χ² test was used to examine the difference in frequency of severity of M S. The .05 level was used for all tests of statistical significance. Values are reported as means plus or minus standard error (SEM).

Results

Fifteen of the 16 subjects completed the study. One subject dropped out of the race due to exhaustion after having completed approximately 20 miles. Consequently, our results were analyzed using n = 15. The physical characteristics and marathon racing information of the 15 subjects are presented in Table 1.

The effects of prerace cold shower on serum CK, CK-MB isoenzyme, and CK-M B/CK ratio are shown in Figure 1, and results are presented in Figure 2. Group differences were significant (P < .05) for the CK-M B/CK ratio only, and...
not significant for CK, CK-MB, and M S. The WOS group exhibited a higher mean value of CK-MB/CK ratio than the CS group. At different sampling times (with combined group values), the mean differences were significant (P < .05) for CK, CK-MB, CK-MB/CK ratio, and M S. With combined group values, the serum CK peaked at 24 hours postrace (2437 ± 759 U/L), and the increase was significantly different (P < .05) from the values obtained prerace (102 ± 17 U/L), immediately postrace (593 ± 133 U/L), 1 hour postrace (792 ± 179 U/L), and 96 hours postrace (327 ± 89 U/L; Figure 1). The differences in CK values between 24 and 48 hours postrace were not statistically significant. Peak values for CK-MB (combined group values) were also significantly different (P < .05) from the values obtained prerace (48 ± 0.9 ng/mL), 1 hour postrace (4.6 ± 0.9 ng/mL), 48 hours postrace (3.4 ± 1.1 ng/mL), and 96 hours postrace (2.8 ± 0.9 ng/mL; Figure 1). The values for CK-MB/CK ratio after the race were significantly (P < .05) elevated at 24 hours postrace (7.9 ± 1.7 ng/mL) compared with prerace (1.5 ± 0.0 ng/mL), 1 hour postrace (4.6 ± 0.9 ng/mL), 48 hours postrace (3.4 ± 1.1 ng/mL), and 96 hours postrace (2.8 ± 0.9 ng/mL; Figure 1). The values for CK-MB/CK ratio after the race were significantly (P < .05) lowered at 10 minutes (0.61 ± 0.01%), 1 hour (0.50 ± 0.07%), 24 hours (0.49 ± 0.14%), 48 hours (0.35 ± 0.05%), and 96 hours (1.00 ± 0.20%) relative to the prerace value (1.97 ± 0.26%; Figure 1).

Muscle soreness for the nine muscle sites revealed that the mean differences between various observation times (with combined group values) were significant (P < .05; Figure 2). Immediately postrace, most runners felt the soreness in the gastrocnemius, and at 24 and 48 hours postrace most of them felt soreness in the quadriceps (Figure 2). The effects of prerace cold shower on the severity of M S for the entire observation period showed no statistical difference between the two groups. Regardless of the severity of M S, the sum of all types of soreness for all muscle sites (with combined group values) was observed to be greatest immediately postrace (55 or 43%; P < .05) and at 24 hours postrace (53 or 44%; P < .05), relative to the prerace and 96 hours postrace values (Figure 2).

Observing only the various sites of M S for the entire 96-hour postrace period, we found that the most frequently reported sites for soreness were in the quadriceps (21%), followed by the gastrocnemius (14%), the soleus (13%), and the tibialis anterior (13%). When observation was made only for the severe type (grade 3) of M S for the 96-hour postrace period, the following muscle groups were reported: quadriceps (42% for combined group values), soleus (23%), gastrocnemius (16%), and tibialis anterior (7%). Other reported sites of severe soreness were the iliotibial tract (7%) and tendo calcaneus (Achilles tendon; 7%).

When comparing the severity of M S (for example, severe, moderately severe, and mild soreness) between the CS and WOS groups, using the χ² statistic, we observed significantly (P < .05) more instances of moderately severe and mild M S (Table 2). Only at 24 hours postrace were there significantly (P < .05) more runners in the CS group with moderately severe soreness than in the WOS group.

Four of the seven runners in the CS group had at least once participated in a previous marathon. The data collected in this study show that rectal temperature after the cold shower was not changed from the baseline, suggesting that peripheral vasoconstriction or increased thermogenic activity may have occurred. The occurrence could be attributed to initial physiologic response to cold exposure by preventing heat loss via conduction.16

It is known that body heat production can be accomplished via the shivering mechanism.21 In this experiment, the subjects did not shiver while taking the cold shower. It was expected that by cool-

<table>
<thead>
<tr>
<th>Table 1 Physical and physiologic characteristics of the subjects</th>
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<tbody>
<tr>
<td>Group*</td>
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<tr>
<td>Age (y)</td>
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<tr>
<td>Height (cm)</td>
</tr>
<tr>
<td>Weight</td>
</tr>
<tr>
<td>Resting heart rate (beats/min)</td>
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<tr>
<td>Blood pressure (mm Hg)</td>
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<tr>
<td>Systolic</td>
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<tr>
<td>Diastolic</td>
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<tr>
<td>No. of weeks in training</td>
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Values are means, SD is in parentheses.

*No statistically significant difference between groups.
Figure 1. Effects of cold shower treatment and no treatment on postrace serum creatine kinase (A), creatine kinase-MB isoenzyme (B), and relative index for creatine kinase-MB/creatine kinase (C). Values are mean and standard error. *P < .05, compared to 24-hour postrace value. †P < .05, compared to 72-hour prerace value.
ing the whole body for 15 minutes, body temperature would be lowered. Once the race began, the exercise-induced thermogenic process would increase body temperature and eventually neutralize the effect of the cold shower. In the present experiment, whole-body cold shower manipulation did not lower rectal temperature. Perhaps invisible shivering may have occurred. Also, a cold shower may induce other physiologic responses to cold stress, such as increased sympathetic nervous activity to maintain normal body temperature. Perhaps other modes of cooling the body should be explored for prerace cold exposure.

That one of the subjects in the CS group dropped out of the race after completing approximately 20 miles is noteworthy. The subject felt weak, chilled, and dizzy, and required immediate medical attention. The diagnosis made by the race medical director was hypotension (blood pressure of 80/50 mm Hg) that was accompanied by shivering and cold extremities (with the impression of hypothermia). The incident could have been attributed to the prerace cold shower or to windchill factor and cold ambient temperature during the first 18 miles of the race.

The generally accepted values for positive CK-MB elevation, indicative of myocardial injury, are a CK-MB value greater than 9 ng/mL or a CK-MB/CK ratio greater than 4%. Our results showed that after the marathon, the serum CK-MB concentrations of 5 (33%) runners were greater than 9 ng/mL, ranging from 9.4 to 18.6 ng/mL. However, none of the values for the ratio (ranging from 0.2% to 2.1%) approached the critical value for positive myocardial injury. The results suggest that elevated serum CK-MB activity in these well-conditioned endurance runners appears to arise primarily from the skeletal muscle source. The use of the CK-MB/CK ratio has been suggested for making clinical diagnosis of suspected cardiac events in athletes.

It is not clear as to why the WOS group exhibited a higher level (P < .05) of CK-MB/CK ratio relative to the CS group at 96 hours postrace. The physiologic or clinical significance of increased CK-MB/CK ratio in response to physical stress is associated with the source of CK-MB production. For instance, high CK-MB/CK ratios would suggest that tissue damage is more likely to be of cardiac origin. The mechanism involved in the efflux of muscle CK and CK-MB into the circulating blood may be related to cell membrane function, integrity or permeability, and fiber necrosis. As none of the runners had signs and symptoms (that is, Q wave ECG and non-Q wave ECG changes) that would have indicated myocardial damage during the 96-hour postrace period or during the 6 months following the race, the release of serum CK-MB was from the muscle source.

At different times after the race, 10 (67%) runners from both groups experienced severe MS, of which 7 (47%) runners felt the soreness at 24 hours postrace, and 3 (20%) immediately after the race. At 48 hours postrace, only 3 (20%) runners still had severe soreness. No complaint of soreness of any degree was observed at 96 hours postrace. When comparison was made between different muscle sites, severe (grade 3) soreness was most often felt in the muscles with predominantly slow-oxidative (type I) fibers, such as the quadriceps and the soleus. The gastrocnemius, composed primarily of fast-glycolytic (type IIb) fibers and fast-glycolytic-oxidative (type IIa) fibers, also was reported to exhibit severe soreness. Whole-body cold shower prior to a marathon significantly increased the incidence of “moderately severe” MS at 24 hours postrace.
involving multiple muscle sites, for example, the quadriceps, soleus, gastrocnemius, tibialis anterior, and flexor digitorum brevis (Table 2; Figure 2).

The cause of the exercise-induced acute (immediate postrace) MS and delayed onset (> 1 hour postrace) MS observed in the present study was strenuous endurance running. The mechanism(s) for exercise-induced MS may have resulted from overuse injury to the skeletal muscle.3,6 Available evidence suggests that concentric exercise-induced MS (as opposed to eccentric exercise) is probably caused by excessive or repetitive mechanical forces exerted on muscle and connective tissue.3,5,7 Other possible mechanisms for exercise-induced MS are interstitial space edema and increased muscle temperature, both of which can aggravate sensory nerve endings resulting in pain or soreness.3,15 Further possibilities include myofibrillar disturbances associated with the Z bands (the contractile elements), leading to interstitial space edema or soft tissue inflammation.4,15

Exposure to cold shower before a marathon apparently did not cause any ill effects on the runners’ health, except for the one subject. Exposure to cold shower immediately before the race also apparently did not cause any significant effect on the runner’s muscular health. Prolonged exercise has been shown to have a minimal effect on immune function in trained athletes.17 No untoward effects of the cold shower were observed in these endurance athletes’ muscular health at a follow-up physical examination 6 months later. The peak-time serum CK and CK-MB activities were not associated with the onset of “severe” MS, nor were they associated with the sum of all types of MS immediately postrace or at 24 hours, 48 hours, or 96 hours postrace. These findings suggest that MS may not be related to maximal appearance of serum enzyme CK and CK-MB concentrations in these athletes. Because of large intersubject variability in the postrace rise in CK activity, we raise a question concerning CK’s usefulness as a predictor of skeletal muscle injury. Our results agree with those of Manfredi and colleagues,13 from their study of plasma CK activity and exercise-induced muscle damage, that postexercise rise in plasma CK level is a manifestation of muscle damage (that is, soreness). We concur with Clarkson and Ebbeling14 and Evans and Cannon15 that CK activity is not a direct indicator of MS. Further study should be conducted to pinpoint the mechanism for exercise-induced MS associated with long-distance running, as well as to explore different modes of cold exposure prior to a race.

We conclude that whole-body cold shower prior to an exhaustive long-distance race did not influence serum enzyme...
Table 2
Degree of severity of muscle soreness between the cold shower group and without cold shower group at postrace intervals

<table>
<thead>
<tr>
<th>Time postrace</th>
<th>Severe CS</th>
<th>WOS</th>
<th>Moderate CS</th>
<th>WOS</th>
<th>Mild CS</th>
<th>WOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediately</td>
<td>36</td>
<td>50</td>
<td>27</td>
<td>55</td>
<td>55</td>
<td>40</td>
</tr>
<tr>
<td>24 h†</td>
<td>40</td>
<td>25</td>
<td>51</td>
<td>9†</td>
<td>35</td>
<td>60</td>
</tr>
<tr>
<td>48 h</td>
<td>24</td>
<td>25</td>
<td>22</td>
<td>36</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>96 h</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

CS, cold shower group; WOS, without cold shower group. Values are sum of muscle soreness from all sites in percent.
†P < .05, compared to severe muscle soreness.
*P < .05, compared to 48- and 96-hour postrace intervals.
**P < .05, compared to the CS group.

CK and CK-MB production, exercise-induced skeletal MS immediately postrace or 96 hours after the race, or the health of the runners immediately postrace or 6 months after the race.

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References