Heat-induced Body Fluid Loss Causes Muscle Cramp during Maximal Voluntary Contraction for the Knee Flexors

Running title: Body Fluid Loss and Muscle Cramp

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Abstract
Exercise associated muscle cramp (EAMC) is common among athletes. However, no study has systematically investigated the effect of the amount of loss of fluid on EAMC. This study was designed to examine the relationship between the amount of decrease in body fluid through sweating and EAMC. A decrease in body fluid by 1%, 2%, and 3% of body mass were induced by sauna exposure on 3 different days, and the occurrence of EAMC was compared between the three conditions and a control (no sauna exposure) condition. Nine young men (age: 22.2 ± 1.4 yrs) who had experienced EAMC during and/or after exercise in the previous year volunteered for the study. A “cramp test”, in which the occurrence of EAMC during maximal voluntary muscle contraction of hamstrings at maximally shortened position for 15 s, was performed before and one hour after the body fluid reduction of 1%, 2%, and 3% of body mass. The cramp test was performed on 2 occasions separated by 3 hours without sauna exposure as the control condition. No EAMC occurred in the control and the 1% condition. Three subjects experienced EAMC in the 2%, and 6 in the 3% condition. These results suggest that body fluid loss of 2% body mass increases the likelihood of EAMC and loss of body fluid is a factor for EAMC.

Keywords: muscle cramp test, sauna, hamstrings, knee flexion, sweating
1. Introduction

Exercise associated muscle cramp (EAMC) is a localized, painful, spasmodic, and involuntary contraction of skeletal muscle that occurs during or after exercise (Schwellnus et al., 1999). Previous studies reported that 18% (Maughan, 1986) or 39% (Manjra et al., 1996) of marathon runners, and 67% of triathletes (Kantorowski et al., 1990) experienced EAMC. Muscles that commonly experience EAMC include triceps surae, hamstrings, and quadriceps femoris (Manjra et al., 1996).

The underlying mechanisms of EAMC are unknown, but it has been documented that loss of body water (Bergeron, 1996) and electrolytes (Stofan et al., 2005; Bergeron, 1996; Hall, 1947) through sweating, and hemodilution induced by overconsumption of water after dehydration (Jung et al., 2005) are associated with EAMC. Dehydration contracts the extracellular fluid compartment of motor nerve terminals (Costill et al., 1976), which may change the concentration of chemical substances such as acetylcholine and potassium of the extracellular spaces and cause mechanical deformation of motor nerve terminals during volitional muscle contraction, resulting in hyperexcitability of motor nerve terminals and EAMC (Layzer, 1994). Early reports have suggested that a major contribution to sweat loss by cycling (Costill et al., 1976) and jumping (Singh et al., 1993) exercise in a hot environment was derived from the interstitial fluid.

Controversy exists concerning the effect of a decrease in body water and electrolytes by sweating on EAMC. Stofan et al. (2005) compared U.S. football players with a history of heat cramps, which can be regarded as EAMC developed in a hot and humid environment, with a cohort group who have no history of cramping. The results showed that the players with a history of cramping lost more than twice the sodium through sweating as the group with no history of cramping during two training sessions per day of 2.5 h each, consisting of a variety of skill drills, playbook execution and game situation conditioning. Horswill et al. (2009) found that loss of sodium in sweat during a morning training session lasting 2.2 h was greater for football players who experienced EAMC compared with those who did not. A case study reported EAMC of a professional tennis player during competition and training was reduced after daily intake of sodium was increased to at least 6-8 g, and drinking about 8 oz or 8 swallows of Gatorade® per changeover during play (Bergeron, 1996). Jung et al. (2005) showed that active men who experienced EAMC during a calf fatiguing exercise in a hot environment had a greater sweat rate than those who did not, and the time to onset of EAMC was longer when they consumed an electrolyte beverage at an amount equivalent to loss of body water compared with the condition without ingestion of water and electrolytes. In contrast, no differences in body mass decrease, plasma volume, and blood electrolytes were reported between distance runners who experienced EAMC during a race and those who did not (Hoffman and Stuempfle, 2015; Kantorowski et al., 1990; Maughan, 1986; Schwellnus et al., 2004; Sulzer et al., 2005). The minimum frequency of stimulation at which the EAMC originates is termed the “threshold frequency,” which is thought to represent one’s propensity to EAMC and lower when susceptibility to EAMC is increased (Miller and Knight, 2009). Miller et al. (2010) observed that 3% body mass loss by cycling exercise using nondominant leg in a hot environment did not change the threshold frequency for electrically inducing EAMC in the flexor hallucis brevis of the dominant leg. Thus, it appears that the association between decrease in body fluid and EAMC is still unclear.

Fluid reduction expressed in terms of percentage of body mass during dehydration can be used as an indicator of dehydration (Maughan, 1986). It has been reported that the amount of decrease in body fluid for subjects who developed EAMC was 1% during a calf fatiguing...
exercise (Jung et al., 2005) and 1.8% (Bergeron, 1996) or 2.9% (Schwellnus et al., 2004) during long distance races. Moreover, previous research by one of us suggests that 3% body mass loss by an intermittent sauna exposure can cause EAMC during volitional muscle contraction for the toe flexors (Ohno and Nosaka, 2004). In previous studies, sauna-induced body mass loss has been shown to be greater (Caldwell et al., 1984), lower (Kubica et al., 1983), or similar (Costill and Fink, 1974) to a decrease in plasma volume compared with exercise-induced body mass loss. However, sauna exposure can be appropriate for studying the effects of body mass loss on EAMC without muscle fatigue and exercise which may influence the development of EAMC (Ohno and Nosaka, 2004). The present study was designed to investigate how much reduction in body fluid by sauna exposures causes EAMC.

2. Methods

2.1. Participants

Subjects participated in this study after being informed of the testing procedure, possible risk and discomfort, and gave a written informed consent. All subjects had experienced EAMC while performing exercise in the 12 months before participating in the present study. This was confirmed by questionnaires about the situations and frequency that EAMC were experienced. Subjects performed the “muscle cramp test” explained below, and the three subjects who had EAMC during the test were excluded from the study. Nine men who reported that they had experienced EAMC but did not have EAMC during the muscle cramp test were included in this study. Seven of them played football, and two of them played tennis at least once a week. Their mean ± SD age, height, body mass, and percentage body fat were 22.3 ± 1.4 yr, 173.8 ± 9.0 cm, 68.0 ± 6.0 kg, and 16.9 ± 2.8%, respectively.

2.2. Materials and methods

The subjects underwent 4 different conditions in which either 1%, 2%, or 3% decrease in body mass was induced by sauna exposure and no sauna exposure was administered (control condition). The subjects reported to the laboratory on 4 different days and were asked to abstain from strenuous physical activity for 3 days before the test. The order of the 4 conditions was randomized among subjects, and each condition was separated by at least 3 days. Immediately following urination, muscle cramp test, measurements of body mass, and isometric knee flexion torque were conducted before, and repeated one hour after the sauna exposure. For the control condition, the measurements were taken twice separated by 3 hours (0 and 3 hours) because approximately 2 hours were required for the target fluid loss of 3% of body mass in preliminary experiments. Decrease in body mass consisted of the decreases in sweat volume (0-3% of body mass) and urine volume. All tests were conducted at approximately the same time of day between 10am and 5pm for each subject after breakfast or lunch. Participants were required to abstain from eating and drinking during each experimental period.

Decrease in body fluid was achieved by repeated exposures to extreme sauna temperatures (mean temperature 100°C) for 6 min with a 3-min rest until the body mass of subjects reached the target fluid loss (1%, 2%, or 3% of body mass) by measuring body mass using a scale (BC-500, TANITA, Tokyo, Japan) during the break period. Although three of the nine subjects did not sweat sufficiently during sauna exposures to achieve the target fluid loss of 3% body mass loss, their data were included (Table 1). Body mass was measured by a scale with a precision of 10g. Sweating rate was calculated as the decrease in body mass during sauna exposure divided by the total time of sauna exposures required to achieve each of three
fluid loss targets. During sauna exposures and rests, subjects were instructed to sit on the
bench and cool their heads with wet towels. Urination rarely occurred during the sauna
exposures and rests.

To assess the occurrence of EAMC of the right and left knee flexors during voluntary
muscle contraction, we developed a test called “muscle cramp test.” For this test, subjects
were asked to isometrically flex their knee joint maximally for 15 seconds by trying to touch
the hip with the heel in a prone posture while keeping their toe flexing dorsally. Subjects were
asked to verbally report and relax immediately when a EAMC was induced. The investigator
checked the occurrence of EAMC determined by participant feedback and visual observation
of sustained knee flexion, then stretched the muscle until resistance force caused by
involuntary muscle contraction subsided. Before the measurement, subjects did not warm-up
because exercise may induce EAMC (Ohno and Nosaka, 2004). The number of subjects who
developed EAMC of the right and/or left knee flexors was counted. The presence or absence
of EAMC in the test was reproducible, and the test was reliable for examining susceptibility
to EAMC in the hamstrings (Ohno, 2015).

Isometric torque of the knee flexors was measured using an isokinetic dynamometer
(CONTREX MJ, CMV AG, Zürich, Switzerland) interfaced with a computer (6843-42J, IBM
Ltd., Tokyo, Japan) in which data acquisition software (human kinetics 1.5.1, CMV AG,
Zürich, Switzerland) was installed. The voltage output from the dynamometer was sampled at
128 Hz and automatically converted to physical units (Nm). Subjects lay in a prone position
on the adjustable bench of the equipment, and the hip and thigh of the dominant leg were
secured to the bench with adjustable straps. The ankle was also secured to the lever arm of the
dynamometer parallel to the lower leg by a strap. The lateral epicondyle was positioned so
that the axis of rotation of the knee joint was aligned with that of the lever arm of the
dynamometer. Gravity correction was conducted by measuring downward torques while the
lever arm connected to the lower leg at 90° (where 0° is horizontal). Isometric knee flexion
torque at 90° over 3 s was measured twice on each occasion (1 min between measurements),
and the peak values were used for further analysis. Before the measurement, subjects did not
conduct a warm-up because muscle cramp occurred as knee flexion force was developed and
at this moment the measurement of muscle cramp was performed.

### 2.3. Statistical Analysis

Percent body mass change and isometric knee flexion torque were each compared between
four levels of body water loss (control vs 1% vs 2% vs 3%) by a one-way repeated measures
analysis of variance (ANOVA). Sweating rate was compared between the three conditions
(1% vs 2% vs 3%) by a one-way repeated measures ANOVA. Change in isometric knee
flexion torque was compared between four levels of body water loss and hydration statuses
using a two-way repeated measures ANOVA. When a significant difference was found,
Bonferroni post hoc test was used for multiple comparisons. Change in number of muscle
cramp occurrence was compared between four levels of body water loss by a Cochran’s Q test.
Multiple post hoc comparisons were performed using McNemar test if a significant difference
was identified. SPSS 17.0 (IBM Statistics, Japan) was used for all statistical analyses.
Statistical significance was set at P < 0.05. The reliability of knee flexion torque before body
mass loss was assessed by calculating the coefficient of variation (CV) using the following
equation: CV = mean/SD*100. The data are shown as mean ± SD.
3. Results

The numbers of subjects who experienced EAMC during the cramp tests were 3 in 2%, 6 in 3% conditions, and 0 in 1% and control conditions (Table 2). The number was significantly (P < 0.05) higher in 3% than 1% and the control condition, respectively. In subjects E and H, EAMC were also induced during the measurement of the knee flexion torque in the 3% condition.

Body mass decreased 0.5 ± 0.3% for control, 1.6 ± 0.1%, 2.2 ± 0.2%, and 3.0 ± 0.6% for the 1%, 2%, and 3% condition, respectively (Table 1). The changes in body mass were significantly (P < 0.05) different across the conditions. EAMC occurred following sauna exposures which induced 1.4-2.6 L body mass loss, but not when body mass loss was 1.0-2.5 L.

Sweating rate was different between subjects and conditions, and the mean was 0.5 to 1.1 L/h for each subject (Table 3).

Isometric knee flexion torque was decreased significantly (P < 0.05) by 5.7 ± 8.0% and 6.0 ± 7.0% in the 2% and 3% conditions, respectively, but showed no difference in 1% (3.8 ± 7.8%) and control conditions (-1.4 ± 12.6%) (Table 4). Percent changes in knee flexion torque were not significantly different across the conditions. CV for knee flexion torque before body mass loss was 8.2 ± 3.5% (2.9-16.3%).

4. Discussion

The present study investigated the effects of decreases in body water of 1%, 2%, and 3% of body mass through sweating induced by sauna exposure on EAMC cramp of the hamstrings during the muscle cramp test in nine young men. The number of subjects who experienced EAMC was 3 in the 2% and 6 in the 3% conditions (Table 2). No EAMC occurred in the control and the 1% conditions. These results suggest that a decrease in body water by over 2% of body mass increases occurrence of EAMC.

Our results were inconsistent with the reports from several studies (Kantorowski et al., 1990; Maughan, 1986; Schwellnus et al., 2004; Sulzer et al., 2005) which did not find significant differences in body fluid loss between distance runners who experienced EAMC during a race and who did not. Moreover, Miller et al. (2010) observed that 3% body mass loss by cycling exercise using nondominant leg in a hot environment did not change the minimum stimulation frequency for electrically inducing EAMC in the flexor hallucis brevis of the dominant leg. In these studies, however, exercise and/or muscle fatigue were not excluded. It should be noted that no previous studies except our research have investigated the effects of body fluid loss on EAMC without exercise. Our previous study (Ohno and Nosaka, 2004) showed that 3% of body fluid loss by an intermittent sauna exposure increased the number of subjects who developed EAMC during muscle cramp test in the toe flexors but not in the knee flexors. The result was different from that of the present study in which EAMC was induced in the knee flexors after up to 3% of body mass loss. Because EAMC in the knee flexors was induced more in subjects who had higher frequency of EAMC in their daily life and had experienced EAMC of the hamstrings (Ohno, 2015), the different results might be attributed to individuals’ tendency to develop EAMC.

The present study shows the relationship between body fluid loss and EAMC. The importance of body fluid and electrolytes to prevent EAMC was suggested in studies by Bergeron (1996) and Jung et al. (2005). Bergeron (1996) reported that a male tennis player p. 6
who had suffered from heat cramps during a tennis match was sweating heavily and lost 1.4% of body mass by the end of the match regardless of drinking fluid freely. In the study, EAMC was reduced by increasing the daily intake of sodium to at least 6-8 g and drinking about 8 oz, or 8 swallows, of Gatorade® per changeover during play. The author suggested that the increase in sodium intake plays an important role in replacing a significant portion of sodium that was lost through sweating, which helps to restore body water after the exercise. Jung et al. (2005) developed a calf-fatiguing protocol by which EAMC was induced in the calf muscles. They found that the time to cause EAMC by the exercise in a hot environment was about 20 minutes (150%) longer by maintaining body fluid with an electrolyte beverage at an amount equivalent to loss of body mass than when 1% of body mass was lost with no fluid consumption. The authors concluded that the hydration and electrolytes are related to the EAMC if the intensity of exercise is relatively low. Dehydration would result in increases in chemical substances such as acetylcholine and potassium in the interstitial space and deformation of nerve endings in the space during muscle shortening, resulting in hyperexcitability of selected motor nerve terminals, causing EAMC (Layzer, 1994). It is interesting to note that when 2% of body mass was decreased by cycling exercise in a hot environment, a large fraction (60%) of the water loss was derived from the interstitial fluid, and the decreases in the intracellular (30%) and plasma (10%) water were relatively small (Costill et al., 1976). Similar result was reported in the study by Singh et al. (1993) showing that a major (80%) contribution to sweat loss during jumping exercise in a hot environment was observed from the interstitial fluid compartment up to the 2% level of dehydration, and little water was lost from the intracellular (6%) and plasma (14%) compartment. Thus, it is possible that the development for EAMC observed in the present study was likely due to the decrease in body fluid, especially from the interstitial space. Further study is needed to examine the relationship between reduction in interstitial fluid and EAMC.

In the present study, body mass loss was induced by sauna exposure to remove the effects of exercise and muscle fatigue on EAMC. Few studies investigated the differential effects of sauna- and exercise-induced body mass loss on plasma volume. Caldwell et al. (1984) found that decrease in plasma volume was greater after 3.5% of body mass loss by sauna (10.3%) than 2.5% body mass loss by exercise (training, running, or swimming) (0.9%). In that study, however, the amount of body mass loss was different between the two conditions making it difficult to compare the conditions. Kubica et al. (1983) reported a relatively smaller decrease in plasma volume after 2.6% of body mass loss by sauna (6.7%) than 2.5% of body mass loss by prolonged bicycle exercise (7.5%). Costill and Fink (1974) showed no significant difference in plasma volume after 4% of body mass loss between thermal (18%) and exercise-induced dehydration (16%) (running or cycling). Thus, different methods and/or exercise for inducing body mass loss, as well as the amount of dehydration, may contribute differently to the onset of EAMC.

Individual variability likely plays an important role in the causation of EAMC (Jung et al., 2005). Jung et al. (2005) reported greater sweat rate in moderately active men who experienced EAMC during a calf fatiguing exercise in a hot environment (2.0 ± 0.9 L/hour) than those who did not (1.3 ± 0.6 L/hour). In the present study, however, sweat rate was relatively small, and similar between when EAMC occurred (0.5-1.1 L/hour) and did not occur (0.5-1.0 L/hour), and not significantly different across conditions (0.7-0.8 L/hour) (Table 3). In addition, body mass loss during sauna exposures was also similar between when EAMC occurred (1.4-2.6 L) and did not occur (1.0-2.5 L). The result was consistent with the reports from a previous study (Stofan et al., 2005) which found that average sweat loss for a 2.5-h practice during a twice daily training camp was not different between football players
who experienced EAMC and those who did not (crampers: 4.0 ± 1.1 L, noncrampers: 3.5 ± 1.6 L). In the study by Stofan et al (2005), however, sweat sodium loss was higher in crampers (5.1 ± 2.3 g) than noncrampers (2.2 ± 1.7 g). Although in the present study electrolytes were not measured, sweat sodium could be higher in subjects who developed EAMC than those who did not. The importance of the results of present and previous studies is that EAMC occurred in some subjects even when sweating rate and/or body fluid loss were relatively low. Future studies are needed to investigate the occurrence of EAMC when body mass and electrolytes are controlled.

It is important to note that all subjects who experienced EAMC in this study had played football at least once a week and the knee flexors were often used during the activity. Because it is suggested that EAMC develops more in the muscles which are often used (Schwellnus et al., 2004), habitual exercise may affect the susceptibility to development of EAMC following body fluid loss. Another possibility is that cramping subjects lost more fluid in the interstitial space than those who did not. Although Morgan et al. (2002) reported that younger subjects demonstrated greater losses of interstitial fluid and smaller losses of intracellular fluid than older subjects after intermittent cycling exercise for 80 min in a hot environment, no previous study has investigated whether such differences were observed between cramping and noncramping subjects. Further study is needed to investigate the possible individual differences in susceptibility to EAMC.

The possibility that other factors such as changes in muscle strength and body temperature, rather than water loss, affected EAMC cannot be excluded. The present study showed that isometric torque of the knee flexors was significantly decreased 5.7% and 6.0% after 2% and 3% of body mass loss, respectively, although development of EAMC during the measurement could affect the decrease in torque in some subjects (E, H) in the 3% condition (Table 4). Viitasalo et al. (1987) reported a decrease in maximal isometric leg extension strength after 3.4% of body mass loss by sauna exposure. The elevated muscle temperature may have played a role in decreasing isometric muscle strength since force has been shown to decline with increased muscle temperature (Rome and Kushmeric, 1983). In addition, an in vitro study demonstrated that with increased muscle temperature, an increased stimulation frequency is necessary to develop the same force (Segal et al., 1986). In the present study, muscle temperature might be higher after 2% and 3% of body mass loss in some subjects although the measurements were conducted one hour after the target body mass loss. However, no previous study examined the relationship between force decline and EAMC. Thus, it is unclear whether the decrease in knee flexion torque directly affected EAMC in the present study. Regarding body temperature, Maughan (1986) reported that athletes who experienced EAMC during a marathon race had a similar rectal temperature after the race to those who did not. Rectal temperature was found to be increased (39.3°C) at a loss of 4% of body mass by sauna and was still slightly high (37.8°C) 60 min after sauna compared with the prevalue (37.2°C) (Costill and Fink, 1974). However, it is suggested that temperature-dependent changes have no effect on the neuromuscular transmission at the motor endplate (Rutkove, 2001). Therefore, it seems to be plausible that body fluid loss is the main factor inducing EAMC observed in present study.

In present study, CV for knee flexion torque before body mass loss was 8.2%. This result was the same as our unpublished data in other subjects and greater than the decrease in knee flexion torque after body mass loss in present study (3.8-6.0%). The time and order of the experiment had little effect on the difference because all tests were conducted at about the same time of day for each subject and the order of the 4 conditions was randomized. In contrast, subjects did not conduct a warm-up before the knee flexion torque because knee
flexion force was developed at the time of the muscle cramp test conducted before the measurement. It may be that conducting a warm-up before the measurements of knee flexion torque increases reliability of the torque.

5. Conclusion

In summary, the present study showed that a decrease in body mass of 2% and 3% induced EAMC to 3 and 6 out of 9 subjects respectively, by sauna exposure, but no EAMC occurred in the 1% loss of body mass and control conditions. These results suggested that the occurrence of EAMC increases when body fluid is reduced by 2% of body mass in some athletes.

6. Acknowledgments

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References

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● Japanese Society of Education and Health Science
● Japan Academy of Learning Disabilities
Table 1: Percent change in body mass (%) for the control (0 hour, 3 hour), 1%, 2%, and 3% condition (pre and post sauna exposure). * a significant difference from the other conditions

<table>
<thead>
<tr>
<th>Subject</th>
<th>Con</th>
<th>1%</th>
<th>2%</th>
<th>3%</th>
<th>Mean ± SD</th>
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<td>-0.8</td>
<td>-1.9</td>
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<td>-3.4</td>
<td>-2.0 ±1.0</td>
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<td>B</td>
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<td>-1.7</td>
<td>-2.0</td>
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<td>C</td>
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<td>-3.1</td>
<td>-1.9 ±1.1</td>
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<td>D</td>
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<td>-2.3</td>
<td>-3.5</td>
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<tr>
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<td>-2.0</td>
<td>-2.2</td>
<td>-1.6 ±0.7</td>
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<td>G</td>
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<td>H</td>
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<td>-2.3</td>
<td>-2.5</td>
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<td>I</td>
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<td>-2.0</td>
<td>-3.6</td>
<td>-2.0 ±1.2</td>
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<td>Mean ± SD</td>
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<td>-1.6 ± 0.1*</td>
<td>-2.2 ± 0.2*</td>
<td>-3.0 ± 0.6*</td>
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Table 2: Development of muscle cramp of each subject for the control, 1%, 2%, and 3% conditions.

<table>
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<td>3%</td>
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<td>0</td>
<td>0</td>
<td>3</td>
<td>6*</td>
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– indicates no occurrence of muscle cramp, and X indicates a muscle cramp occurred during the cramp test. The number of subjects who had muscle cramp is shown in the bottom row of “n.” * a significant difference from control and 1% conditions.
Table 3: Sweating rate (L/hour) of each subject during sauna for 1%, 2%, and 3% conditions (pre and post sauna exposure).

<table>
<thead>
<tr>
<th>Subject</th>
<th>1%</th>
<th>2%</th>
<th>3%</th>
<th>Mean ± SD</th>
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<td>0.9</td>
<td>0.8</td>
<td>0.8 ± 0.1</td>
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<td>0.9</td>
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<td>1.0</td>
<td>1.1</td>
<td>0.9 ± 0.2</td>
</tr>
<tr>
<td>E</td>
<td>0.7</td>
<td>0.7</td>
<td>0.9</td>
<td>0.8 ± 0.1</td>
</tr>
<tr>
<td>F</td>
<td>0.7</td>
<td>0.8</td>
<td>0.5</td>
<td>0.7 ± 0.1</td>
</tr>
<tr>
<td>G</td>
<td>0.8</td>
<td>0.9</td>
<td>0.6</td>
<td>0.8 ± 0.2</td>
</tr>
<tr>
<td>H</td>
<td>0.7</td>
<td>0.7</td>
<td>0.6</td>
<td>0.7 ± 0.1</td>
</tr>
<tr>
<td>I</td>
<td>0.9</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1 ± 0.1</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>0.7 ± 0.1</td>
<td>0.8 ± 0.2</td>
<td>0.8 ± 0.2</td>
<td></td>
</tr>
</tbody>
</table>
Table 4: Percent change in isometric knee flexion torque (%) at 90° for the control (0 hour, 3 hour), 1%, 2%, and 3% condition (pre and post sauna exposure).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Con</th>
<th>1%</th>
<th>2%</th>
<th>3%</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>14.8</td>
<td>-7.1</td>
<td>-10.8</td>
<td>0</td>
<td>-0.8 ±11.3</td>
</tr>
<tr>
<td>B</td>
<td>8.9</td>
<td>-11.8</td>
<td>0.2</td>
<td>2.4</td>
<td>-0.1 ± 8.6</td>
</tr>
<tr>
<td>C</td>
<td>-22.2</td>
<td>8.8</td>
<td>-19.3</td>
<td>-1.2</td>
<td>-8.5 ± 14.8</td>
</tr>
<tr>
<td>D</td>
<td>-9.7</td>
<td>-7.9</td>
<td>-1.3</td>
<td>-12.5</td>
<td>-7.8 ± 4.7</td>
</tr>
<tr>
<td>E</td>
<td>19.4</td>
<td>2.4</td>
<td>-9.8</td>
<td>-3.7</td>
<td>2.1 ± 12.6</td>
</tr>
<tr>
<td>F</td>
<td>2.5</td>
<td>0.4</td>
<td>7.0</td>
<td>-20.0</td>
<td>-2.5 ± 12.0</td>
</tr>
<tr>
<td>G</td>
<td>-3.9</td>
<td>-0.8</td>
<td>-5.8</td>
<td>-8.6</td>
<td>-4.8 ± 3.3</td>
</tr>
<tr>
<td>H</td>
<td>-0.2</td>
<td>-1.7</td>
<td>-0.2</td>
<td>-3.3</td>
<td>-1.3 ± 1.5</td>
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<tr>
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<td>-16.9</td>
<td>-11.5</td>
<td>-6.9</td>
<td>-8.2 ± 8.2</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>1.4 ± 12.6</td>
<td>-3.8 ± 7.8</td>
<td>-5.7 ± 8.0</td>
<td>-6.0 ± 7.0</td>
<td></td>
</tr>
</tbody>
</table>