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Dehydration and Symptoms of Delayed-Onset Muscle Soreness in Normothermic Men

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Context: A dehydrated individual who performs eccentric exercise may exacerbate skeletal muscle damage, leading to structural, contractile, and enzymatic protein denaturation, in addition to the myofiber and connective damage resulting from the eccentric muscle tension.

Objective: To identify the effects of dehydration on 5 physiologic characteristics of delayed-onset muscle soreness (DOMS) in normothermic men after an eccentric exercise perturbation.

Design: Randomized group test-retest design.

Setting: Laboratory.

Patients or Other Participants: Ten healthy male volunteers randomly assigned to either a euhydration (age = 26.2 ± 4.9 years, height = 174.1 ± 6.0 cm, mass = 86.5 ± 15.3 kg) or dehydration (age = 25.8 ± 2.2 years, height = 177.2 ± 3.1 cm, mass = 84.4 ± 3.8 kg) group.

Intervention(s): Subjects performed treadmill walking for 45 minutes in either a thermoneutral (euhydration) or a hot, humid (dehydration) environment. After a rest period to allow for return to the normothermic condition, DOMS was induced with a 45-minute downhill run.

Main Outcome Measures: We assessed 5 physiologic characteristics of DOMS before and at intervals after the eccentric exercise. The characteristics were perceived pain of the bilateral quadriceps and overall body, bilateral punctate tenderness of the superficial quadriceps muscles, bilateral knee-flexion passive range of motion, bilateral thigh circumference, and bilateral isometric quadriceps muscle strength. Thermoregulatory and cardiovascular measures were obtained to monitor participants' heat load during exercise.

Results: The experimental protocol produced a 0.9% increase in body mass of the euhydration group and a significant 2.7% decrease in body mass of the dehydration group. The downhill-running exercise perturbation induced DOMS in both the euhydrated and dehydrated participants, based on increased bilateral quadriceps and overall body perceived pain and punctate tenderness of the bilateral vastus medialis muscle. The signs and symptoms of DOMS after an eccentric exercise perturbation were not exacerbated by moderate dehydration of 2.7% body mass after rest and return to the normothermic condition.

Conclusions: Significantly dehydrated participants who rested and returned to a normothermic condition did not experience increased characteristics of DOMS.

Key Words: euhydration, euthermic, thermal regulation, eccentric exercise

Dehydration is a frequent problem in physically active individuals exercising at high volumes in hot ambient environments, with losses of 6% to 8% of preexercise body mass common.[1,2] As little as 1% to 2% body mass loss in the heat challenges cardiovascular compensatory mechanisms for increased body temperature and reduces exercise capacity.[3] Thermoregulatory shunting of blood from the core to the skin for passive heat loss and sweating provides compensatory mechanisms for cooling but may challenge both total body water and the blood volume needed to optimally perfuse the tissues of the body during exercise,[3] resulting in decreased skeletal muscle perfusion.[3–5]

Normal skeletal muscle function is affected by altered physiologic states such as dehydration. Exercise performance decreases as less blood is available for perfusion of active skeletal muscle. Blood flow to exercising muscles is significantly reduced with dehydration due to reductions in blood pressure and perfusion pressure.[4,5] Sweating is maintained by intracellular water shifting to the extracellular space, resulting in cell dehydration and adversely affecting skeletal muscle cell function.[6] Dehydration negatively affects muscle performance by impeding thermal regulation, altering water movement across cell membranes, and interfering with actin-myosin cross-bridge formation.[7]

Delayed-onset muscle soreness (DOMS) is a clinical model of muscle damage consisting of muscular pain and other symptoms experienced 24 to 48 hours after novel or intense exercise. The signs and symptoms of DOMS include dull, diffuse pain and tenderness; stiffness; swelling; and decreased strength of the exercised muscle.[8] These signs and symptoms typically last 1 to 4 days after the exercise bout.[8–12] The magnitude of DOMS depends on the intensity and duration of the exercise

Figure 1. Research Procedures Flow Chart

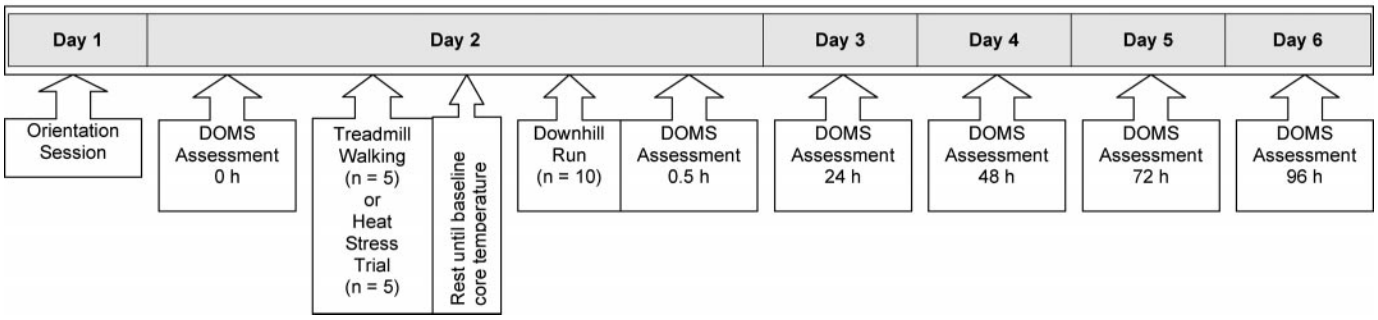

Figure 1. Research procedures flow chart. DOMS indicates delayed-onset muscle soreness.

Age, Height, Percentage of Body Fat, Resting Heart Rate, and Resting Mean Arterial Pressure of Euhydration and Dehydration Groups (Mean ± SD)

	Age Group* (y)	Height (cm)	Percentage of Body Fat	Resting Heart Rate (beats/min)	Resting Mean of Arterial Pressure (mm Hg)
Euhydration	26.2 ± 4.9	174.1 ± 6.0	19.9 ± 1.5	68.0 ± 11.1	97.7 ± 1.4
Dehydration	25.8 ± 2.2	177.2 ± 3.1	16.3 ± 1.0	65.0 ± 10.6	93.7 ± 9.4

*n = 5 for each group.

perturbation[8,10,11] as well as the physiologic condition of the individual.[13,14]

Eccentric exercise performed when an individual is dehydrated may exacerbate the skeletal muscle damage as a result of reduced intracellular water. Eccentric muscle activity with decreased intracellular water during dehydration has been theorized to lead to structural, contractile, and enzymatic protein denaturation.[6,7] These structural and functional protein alterations might occur in addition to the initial myofiber and connective tissue damage produced by eccentric muscle tension. Rest periods before additional exercise in a thermoneutral environment have been shown to sufficiently redistribute body water and attenuate thermoregulatory and cardiorespiratory responses of dehydrated individuals.[15] Ultimately, dehydration may increase the risk of DOMS in healthy individuals engaged in novel or unaccustomed physical exercise.[12,16] Ostensibly, DOMS has a detrimental impact on athletic performance in that muscle endurance, strength, and power are reduced.[17] The combination of the deleterious effects of dehydration and DOMS may predispose an athlete to the risk of additional injury during physical activity. A paucity of data exists concerning the effects of dehydration on eccentric-biased aerobic exercise performance and the resulting signs and symptoms of DOMS. Our purpose was to determine the effects of dehydration on the characteristics of DOMS.

METHODS

Research Design

The research design consisted of a randomized group test-retest design with 2 factors (group and time) with repeated measures on time (Figure 1). Participants performed 45 minutes of treadmill walking in either a thermoneutral (euhydration) or a hot, humid (dehydration) environment. After a rest period to allow for the return to the normothermic condition, DOMS was induced with a 45-minute downhill run (−12° from horizontal). The euhydration group was allowed water ad libitum, and the dehydration group was fluid restricted. Five physiologic characteristics of DOMS were assessed preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. The study was approved by the institutional review board and was conducted at the Biokinetics Research Laboratory.

Participants

Participants were 10 healthy male students (age = 25.4 ± 3.6 years, height = 175.8 ± 7.6 cm, and body mass = 85.3 ± 3.6 kg) (Table). Males were selected to reduce the variability of hormone levels and substrate utilization between sexes during exercise. Before participating in the study, subjects completed a health history questionnaire and an informed consent form in compliance with institutional review board policies.

Participants were accepted based on the absence of lower extremity eccentric training during the 6 months preceding data collection. Eccentric training was defined as downhill running, running downstairs, jumping, or weight training. Subjects maintained normal activities of daily living and refrained from initiating an exercise program during the course of the study. Additionally, they denied a history of lower limb injury or surgery, predisposing cardiovascular or cardiorespiratory conditions, or heat-related illness within 1 year preceding data collection. Participants were required to abstain from ingestion of alcohol, caffeine, and nonprescription medications for 24 hours before and during the study. They were also instructed to abstain from massaging, stretching, or otherwise treating the exercised limbs during the study.

Instruments

Visual Analog Scale. Bilateral quadriceps and overall body perceived pain were assessed using a visual analog scale (VAS). The VAS consists of a 10-cm line labeled with *no*

soreness on the left and *extremely sore* on the right.[18] Participants rated perceived pain by placing a slash mark on the line best corresponding to their soreness level. Perceived pain was quantified by measuring to the nearest 1 mm from the left end of the line to the slash mark.

Subjects assessed their perceived pain level using a standardized protocol. All determinations were performed twice with a blank VAS for each assessment to minimize bias from previous determinations. The averages of the 2 bilateral quadriceps determinations and the 2 overall body determinations were recorded. The VAS has been used to reliably quantify perceived pain following eccentric exercise.[19–21] The VAS average intratester reliability for the current study was an intraclass correlation coefficient (ICC) (2,1) of 0.99.

Punctate Tenderness Gauge. Bilateral quadriceps punctate tenderness was assessed using the punctate tenderness gauge (PTG) (model 75 force probe gauge; Technical Products, Caldwell, NJ). Spring-loaded probes similar to the PTG have been used to reliably quantify muscular tenderness.[21–25] The PTG has a 2-mm hemispheric probe attached to a force gauge. A 1.5-cm closed-cell foam stopper is attached to the end of the probe to simulate digital palpation.[21–25] We used a plastic grid with 10 holes spaced 10 cm apart to standardize placement of the PTG.

Bilateral quadriceps punctate tenderness was assessed using a standardized protocol. Participants were seated on a plinth with the hips in 0° of abduction and 90° of flexion and the knees in 0° of extension. The plastic grid was positioned over the quadriceps muscle group; its distal corners were secured with self-adhesive strips over the medial and lateral femoral condyles. The gauge was inserted into each hole of the grid and depressed until the participant reported that the sensation of pressure turned to tenderness or pain. A maximum force of 14 lb (6.35 kg) was recorded and interpreted as absence of punctate muscle tenderness.[21] The force required to elicit a response is inversely related to punctate muscle tenderness (ie, the more force applied, the less muscle tenderness present). Measurements were recorded to the nearest 0.25 lb (0.11 kg). Each region of the quadriceps muscle group was assessed. The vastus medialis and the rectus femoris each had 3 data collection levels: proximal, middle, and distal. The vastus lateralis had 4 data collection levels: proximal, middle proximal, middle distal, and distal. Measurements were performed twice for each data collection level and averaged for each muscle. The punctuate tenderness gauge has weights traceable to the National Bureau of Standards with a reliability of $r = 0.99$, $P < .05$ (unpublished data, Donald C. Meserlian, 1987–1991). The average intratester reliability for the gauge in the current study was an ICC (2,1) of 0.76, comparable with previously published studies.[21,22]

Goniometer. Goniometry has been used to assess passive range of motion (ROM) after eccentric exercise.[21,26–28] Bilateral knee-flexion passive ROM was measured using a 12-in (30.48-cm) clear plastic goniometer (No. 137; Orthopedic Equipment Co, Bourbon, IN). The goniometer was marked in 1.0° increments. Bilateral knee-flexion passive ROM was assessed using a standardized protocol. Participants were positioned on a plinth prone with the hips in 0° of abduction and flexion. A folded towel was placed under each distal thigh to stabilize the femur and reduce hip rotation, flexion, and extension. The fulcrum of the goniometer was centered over the lateral femoral epicondyle, and the stationary arm of the goniometer was aligned with the greater trochanter of the femur. The movable arm of the goniometer was aligned with the lateral midline of the fibula, using the lateral malleolus as a reference.[26] The starting position was 0° of knee extension. Bilateral knee-flexion passive ROM was assessed at the end range when participants either reported pain or soreness or no further movement occurred. Measurements were performed twice for each limb. The average of the 2 measurements for each limb was recorded. Passive knee-flexion ROM average intratester reliability was an ICC (2,1) of 0.99.

Anthropometric Measuring Tape. Bilateral thigh circumference was measured using a Gullick anthropometric measuring tape (Fabrication Enterprises, Everton, NY). The measuring tape has a spring-loaded gauge for improved standardization of applied tension. Bilateral thigh circumference was assessed using a standardized protocol. We measured the length of the thigh from the anterior superior iliac spine to the superior pole of the patella. Distal thigh circumference was measured at 10%, 20%, 30%, and 40% (distal, middle distal, middle proximal, and proximal sites, respectively) of the length of the thigh. Two measurements were performed at each site, and the average of the 2 measurements for each site was recorded. Limb circumference has been used to reliably assess muscle swelling after eccentric exercise.[27,28] Bilateral thigh circumference intertester and average intratester reliabilities for the current study were ICC (2,1) of 0.99 and 0.99, respectively.

Isokinetic Dynamometer. Isokinetic dynamometry has been used to assess isometric strength after eccentric exercise.[21,28–30] Bilateral quadriceps isometric strength was measured using the Biodex B-2000 isokinetic dynamometer (Biodex Corp, Shirley, NY), which is a multijoint testing and rehabilitation system. The dynamometer was calibrated before each data collection session in accordance with the manufacturer's guidelines.

Bilateral quadriceps isometric strength was assessed using a standardized protocol. Participants were seated on the bench with the hips and knees at 90° of flexion. They crossed their arms over their chest, and we secured straps over their arms, pelvis, and femur. The knee attachment was secured to the tibia just proximal to the talocrural joint, allowing full plantar flexion and dorsiflexion. The dynamometer axis was aligned with the joint line of the knee. The dynamometer arm was positioned at 60° (knee extension) and confirmed goniometrically. Subjects performed five 5-second submaximal isometric warm-up repetitions. Testing consisted of the participants applying as much force as possible against the immovable dynamometer arm in the direction of knee extension. Participants performed five 5-second maximal isometric repetitions for each limb with a 5-second rest between repetitions. The highest and lowest values were eliminated, and the remaining 3 values were averaged and recorded. Bilateral quadriceps isometric strength average intratester reliability was an ICC (2,1) of 0.95.

Experimental Protocols

As safety precautions, participants' rectal and skin temperatures, heart rate, mean arterial pressure, and rating of perceived exertion responses were recorded preexercise and at 15-minute intervals during treadmill walking and downhill running. Thermoregulatory response to exercise was determined via rectal probe (model 401; YSI, Yellow Springs, OH) and 4 skin thermistors (model 409A; YSI) connected to a te-

lethermometer (model 43; YSI). Cardiovascular response to exercise was determined by measuring heart rate using the SensorMedics Max-1 12-lead electrocardiogram (Yorba Linda, CA). Participants' blood pressures were assessed standing and during exercise using a mercury sphygmomanometer (American Diagnostics, West Babylon, NY) and stethoscope (American Diagnostics). Perceived exertion was measured using the Borg Rating of Perceived Exertion Scale.[31] Exercise rectal temperature and the electrocardiogram were monitored every 5 minutes.

Euhydration. The euhydration protocol consisted of participants reporting to the laboratory fully hydrated on the morning of data collection. Subjects were instructed to consume a breakfast consisting of a bagel or toast and 4 oz (118.29 mL) of orange juice. Euhydrated participants consumed water ad libitum to maintain preexercise body mass throughout exercise and data collection. To mitigate the confounding effects of exercise on DOMS, the euhydrated subjects performed 45 minutes of treadmill walking (model Q55; Quinton Instruments, Seattle, WA) in a thermoneutral environment. The treadmill walking protocol commenced with a 5-minute warm-up at 3.0 mph (4.83 kph). Treadmill speed was then increased, and the participants walked for 40 minutes at 60% to 70% of their age-predicted heart rate range. A 60-second rest was administered after every 15 minutes of walking.

Dehydration. The dehydration protocol consisted of participants reporting to the laboratory fully hydrated on the morning of data collection. The dehydration criterion was a decrease in preexercise body mass of at least 3.0%, consistent with a moderate level of dehydration.[3] Subjects were dehydrated by walking on a treadmill for 45 minutes in a hot, humid environmental chamber (Tenney Engineering, Union, NJ) with fluid restriction. The treadmill was located inside the environmental chamber, which is a stainless steel vault measuring 10 $\times$ 5 $\times$ 6 m and capable of producing and maintaining temperatures up to 40°C and 85% relative humidity. The treadmill walking protocol commenced with a 5-minute warm-up at 3.0 mph (4.83 kph). Treadmill speed was then increased, and the participants walked for 40 minutes at 60% to 70% of their age-predicted heart rate range. A 60-second rest was administered after every 15 minutes of walking. Conditions inside the environmental chamber were maintained at an ambient temperature of 40.0 $\pm$ 1.7°C, a relative humidity of 66.5 $\pm$ 12.4%, and sea level ambient pressure. The first 2 environmental conditions were confirmed using a dry bulb thermometer (Bacharach, Pittsburgh, PA) and sling psychrometer (Bacharach).

Delayed-Onset Muscle Soreness Inducement. After treadmill walking, participants rested to allow rectal temperature to return to preexercise, or normothermic, levels (30 to 60 minutes) to attenuate the cardiovascular compensatory effects of exercise in the heat. We induced DOMS in all participants by having them run downhill on a treadmill elevated $-12°$ from horizontal by placing wooden blocks under the rear of the treadmill. Downhill running was performed in a thermoneutral room and commenced with a 5-minute warm-up at 4.0 mph (6.44 kph). Treadmill speed was then increased, and subjects ran for 40 minutes at 60% of their age-predicted heart rate range.[32] A 60-second rest period was administered after every 15 minutes of downhill running. At the conclusion of the data collection session, dehydrated participants were required to orally rehydrate with cool water until they returned to within 2% of their preexercise body mass.

Experimental Procedures. Participants reported to the Biokinetics Research Laboratory 24 hours before data collection for nude weighing and baseline data collection. The following day, they reported to the Laboratory and euhydrated body mass was confirmed as $\pm$1% of baseline. Subjects were randomly assigned (n = 5) to perform treadmill walking in either a thermoneutral environment with oral rehydration to elicit the euhydration (control) condition or a hot, humid environment (40.0 $\pm$ 1.7°C, 66.5 $\pm$ 12.4% relative humidity) with fluid restriction to elicit the dehydration (experimental) condition. The criterion for dehydration was 3% body mass loss, consistent with moderate levels of dehydration. After treadmill walking, all participants rested (30 to 90 minutes) to reduce rectal temperatures to preexercise, or normothermic, levels in order to mitigate the fatiguing effect of lower extremity exercise on DOMS. They then performed 45 minutes of downhill running to induce DOMS. During all exercise sessions (treadmill walking and downhill running), heart rate, blood pressure, rating of perceived exertion, rectal temperature, and skin temperature were recorded at 15-minute intervals. After downhill running, participants were again weighed nude. At 0.5 hours postexercise, the DOMS-dependent variables were measured and recorded. Five characteristics were assessed: bilateral quadriceps and overall body perceived pain, bilateral quadriceps punctate tenderness at 3 muscle sites, bilateral knee-flexion passive ROM, bilateral thigh circumference at 4 sites, and bilateral quadriceps isometric strength. Subjects returned to the laboratory 24, 48, 72, and 96 hours postexercise for follow-up measurements of the DOMS dependent variables. At least 96 hours after exercise, the participants' body compositions were determined via hydrostatic weighing (underwater weighing scale; Chatillon Creative Health Products, Plymouth, MI) and population-specific formulae for conversion of body density to percentage of body fat.[32] Subjects also performed a graded treadmill test using the SensorMedics Vmax 229 metabolic cart to determine cardiovascular fitness level. Data collection was conducted in May and July (mean maximum ambient temperature = 27.9 $\pm$ 4.8°C, National Weather Service, Mount Holly, NJ).

Data Analysis

Statistical analyses were conducted on the participants' physical characteristics and DOMS-dependent variables. Physical characteristics and hydration status were compared between groups using independent *t* tests. The DOMS-dependent variables were analyzed using separate 2 (group: euhydration and dehydration) $\times$ 6 (time: preexercise and 0.5, 24, 48, 72, and 96 hours postexercise) analysis of variance with repeated measures on the second factor. When applicable, bilateral data for the DOMS-dependent variables were averaged and used as the criterion measure for the data analyses. When significant interactions existed, tests of simple main effects were performed. The appropriate post hoc *t* test with a Bonferroni adjustment for multiple comparisons was performed to reveal the location of the significant differences. Data were analyzed using the SPSS 10.0 for Windows Statistical Package (SPSS Inc, Chicago, IL). Significance was set at $P \leq .05$ for all statistical analyses.

RESULTS

Physical Characteristics

No significant differences existed between the euhydration and dehydration groups for age ($t_9 = 0.17$, $P = .87$), height

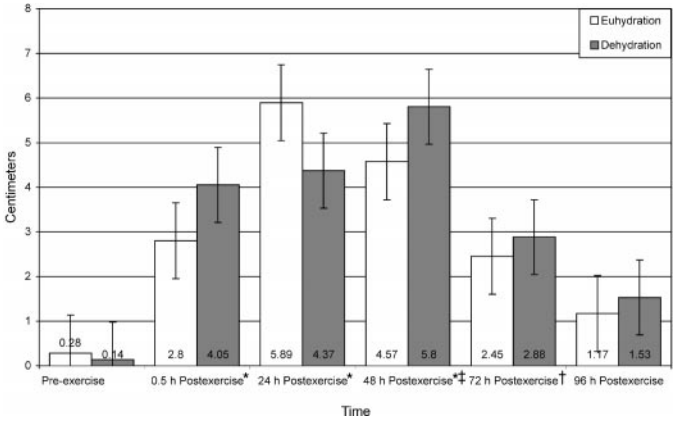

Figure 2. Perceived pain of the bilateral quadriceps muscles of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. *Significantly greater at 0.5, 24, and 48 hours postexercise than at preexercise ($P \leq .001$). †Significantly greater at 24, 48, and 72 hours postexercise than at 96 hours postexercise ($P < .003$, $P \leq .001$, and $P = .002$, respectively). ‡Significantly greater at 48 hours postexercise than at 72 hours postexercise ($P = .002$). Nonsignificant for group ($F_{1,8} = 0.087$, $P = .776$, power = .058).

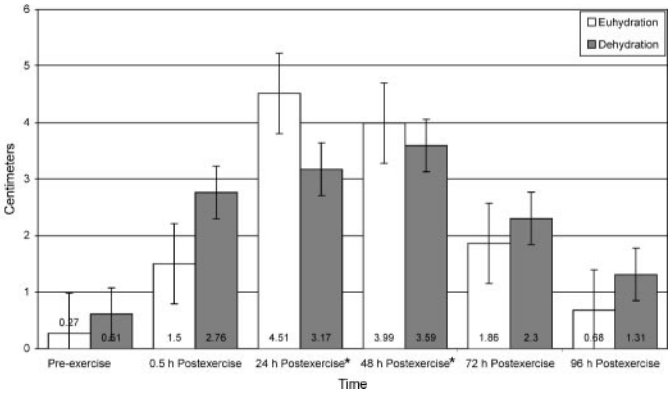

Figure 3. Perceived pain of the overall body of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. *Significantly higher at 24 and 48 hours postexercise than at preexercise ($P = .002$). Nonsignificant for group ($F_{1,8} = 0.024$, $P = .882$, power = .052).

($t_9 = -1.04$, $P = .33$), body mass ($t_9 = 0.30$, $P = .11$), percentage of body fat ($t_9 = 1.81$, $P = .12$), resting heart rate ($t_9 = 0.44$, $P = .67$), or resting mean arterial pressure ($t_9 = .94$, $P = .37$) (see Table). However, percentage change in body mass data from preexercise to postexercise revealed significant differences between groups. The dehydration group lost significantly more body mass ($t_9 = 7.92$, $P \leq .001$) than the euhydration group. Treadmill walking in a hot, humid environment with fluid restriction resulted in a 2.7% decrease in body mass of the dehydrated participants (2.3 ± 0.1 kg). Treadmill walking in a thermoneutral environment with oral rehydration resulted in a 0.9% increase in body mass of the euhydrated participants (0.7 ± 0.4 kg).

Delayed-Onset Muscle Soreness Assessment

Bilateral Quadriceps Perceived Pain. The quadriceps perceived pain data (Figure 2) revealed a significant time main effect only. Quadriceps perceived pain was significantly higher at 0.5, 24, and 48 hours postexercise than at preexercise ($t_9 = -4.80$, $P \leq .001$; $t_9 = -5.48$, $P \leq .001$; and $t_9 = -6.65$, $P \leq .001$, respectively). Bilateral quadriceps perceived pain was also significantly higher at 24, 48, and 72 hours postexercise than at 96 hours postexercise ($t_9 = 4.02$, $P = .003$; $t_9 = 5.95$, $P \leq .001$; and $t_9 = 4.46$, $P = .002$, respectively) and significantly higher at 48 hours postexercise than at 72 hours postexercise ($t_9 = 4.24$, $P = .002$). No interaction effects were observed for bilateral quadriceps perceived pain.

Overall Body Perceived Pain. A significant time main effect only existed for overall body perceived pain (Figure 3). Overall body perceived pain was significantly higher at 24 and 48 hours postexercise than at preexercise ($t_9 = -4.31$, $P = .002$ and $t_9 = -4.44$, $P = .002$, respectively). No interaction effects were observed for overall body perceived pain.

Bilateral Quadriceps Punctate Tenderness. The punctate tenderness data for the vastus medialis (Figure 4) revealed a significant time main effect only. Punctate tenderness of the vastus medialis was significantly higher at 24 hours postexercise than at preexercise and 0.5 hours postexercise for both groups ($t_9 = 5.36$, $P \leq .001$ and $t_9 = 4.70$, $P \leq .001$, respectively). Punctate tenderness data for the vastus lateralis revealed a significant interaction between group and time. Tests of simple main effects demonstrated that punctate tenderness of the vastus lateralis was 18.5% and 10.1% higher for the euhydrated participants than for the dehydrated participants at 72 and 96 hours postexercise, respectively ($t_9 = -3.64$, $P = .007$ and $t_9 = -3.69$, $P = .006$). No significant interaction or main effects existed for the rectus femoris.

Bilateral Knee-Flexion Passive Range of Motion. Bilateral knee-flexion passive ROM (Figure 5) revealed no significant interaction or main effects.

Bilateral Thigh Circumference. Distal bilateral thigh circumference data (Figure 6) revealed a significant time main effect only. Distal bilateral thigh circumference was significantly greater for both groups at 0.5 hours postexercise than at preexercise ($t_9 = -4.63$, $P \leq .001$). No significant interaction or main effects existed for middle distal, middle proximal, or proximal bilateral thigh circumferences.

Bilateral Quadriceps Isometric Strength. Quadriceps isometric strength data (Figure 7) revealed a significant time main effect only. Quadriceps isometric strength was significantly greater at 96 hours postexercise than at 0.5 hours postexercise for both groups ($t_9 = -4.72$, $P \leq .001$). No interaction effects were observed for bilateral quadriceps isometric strength.

DISCUSSION

We demonstrated that the downhill running exercise perturbation induced DOMS in both the euhydrated and dehydrated participants, based on increased bilateral quadriceps and overall body perceived pain and punctate tenderness of the bilateral vastus medialis muscle. All 3 variables were significantly higher at 24 hours postexercise than at preexercise. Bilateral quadriceps perceived pain was also significantly higher at 48 hours postexercise than at preexercise. Compared to its zenith at 48 hours postexercise, bilateral quadriceps perceived pain was significantly reduced at 72 and 96 hours postexercise. This classic model of DOMS has been consistently reported in the literature.[10,11,33–36]

Pathophysiology of Delayed-Onset Muscle Soreness

Primarily, DOMS is caused by exercise that incorporates heavy loads with passive lengthening during muscle contrac-

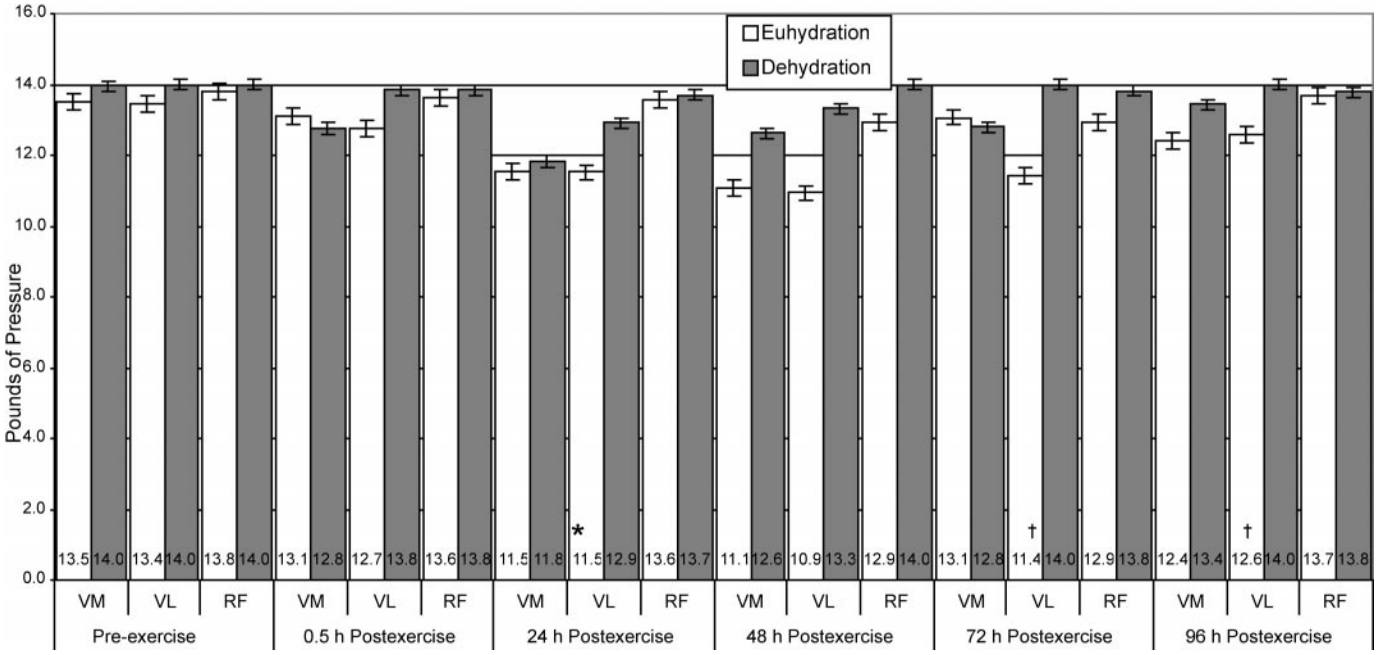

Figure 4. Punctate tenderness of the bilateral quadriceps of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. Values are inversely related to tenderness, ie, the lower the value, the more tenderness reported. *Significantly greater ($P \leq .001$) than preexercise and 0.5 hour postexercise. †Significantly higher ($P \leq .007$) than dehydration group for each time. Vastus medialis nonsignificant for group ($F_{1,8} = 0.633$, $P = .449$, power = .108). Rectus femoris nonsignificant for time ($F_{5,40} = 1.016$, $P = .421$, power = .323) and group ($F_{1,8} = 1.235$, $P = .299$, power = .166). Muscles tested: VM indicates vastus medialis; VL, vastus lateralis; and RF, rectus femoris.

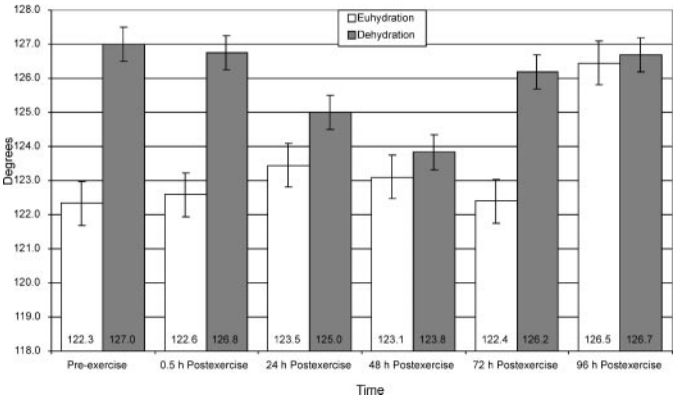

Figure 5. Knee-flexion range of motion of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. Nonsignificant for group ($F_{1,8} = 0.242$, $P = .242$, power = .072) and time ($F_{5,40} = 1.155$, $P = .348$, power = .313).

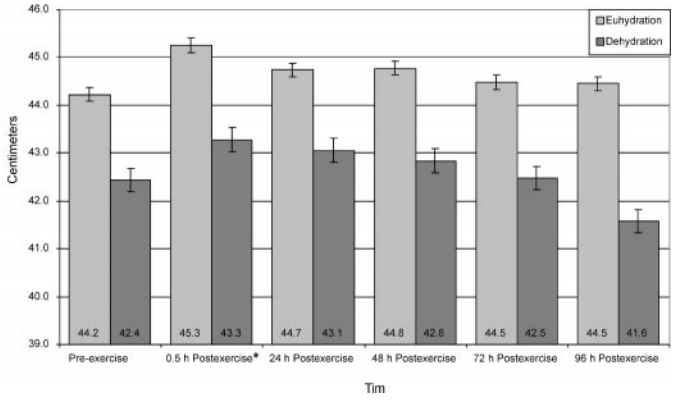

Figure 6. Distal thigh circumference of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. *Significantly greater at 0.5 hour postexercise than at preexercise ($P \leq .001$). Nonsignificant for group ($F_{1,8} = 1.711$, $P = .227$, power = .211).

tion or production of eccentric muscle tension.[33–35] Eccentric muscle tension is produced during lowering weight against gravity, downhill walking, and downhill running.[34,35] Acute bouts of eccentric muscle tension produce microscopic lesions of the myofiber often referred to as ''microdamage.''[36–38] Microdamage affects the sarcolemma, sarcoplasmic reticulum, Z-lines,[11,37,38] contractile components of the myofiber, and surrounding connective tissue.[39–43] Skeletal muscle microdamage produced by eccentric muscle tension is subcellular and leads to concomitant events that result in the signs and symptoms associated with DOMS.[44–48]

The signs and symptoms of DOMS are attributed to subcellular alterations of the sarcolemma, or phospholipid membrane, as a result of skeletal muscle microdamage.[49–53] The sarcolemma loses its ability to retain potassium, creatine kinase, and myoglobin, which are released into the extracellular fluid, plasma, and urine.[33,36,49–53] Efflux of intramuscular ions and proteins leads to increased osmolarity of the extracellular fluid and fluid shifts out of the cell.

Increased osmolarity of the extracellular fluid enhances potassium release by cells and, in a counterproductive way, further increases extracellular fluid osmolarity and potassium concentrations. As plasma osmolarity increases, intracellular water leaves the cell because of the osmotic pressure gradient across the cell membrane. Intracellular water shifts out of the cell until the intracellular fluid osmolarity equals the extracel-

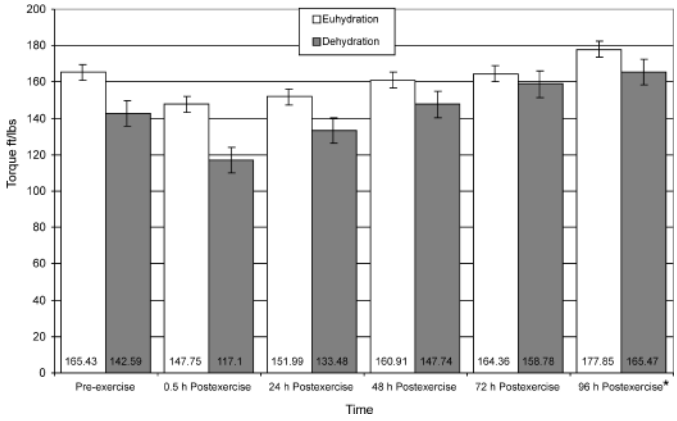

Figure 7. Isometric strength of euhydrated and dehydrated participants measured preexercise and at 0.5, 24, 48, 72, and 96 hours postexercise. *Significantly greater than at 0.5 hour postexercise ($P \leq .001$). Nonsignificant for group ($F_{1,8} = 4.572$, $P = .065$, power = .469).

lular fluid osmolarity. This loss of intracellular water shrinks (crenates) cells, causing the intracellular potassium concentration to rise, providing a driving force for potassium efflux from cells, further increasing extracellular fluid and plasma potassium concentration.[54]

During exercise, more potassium is released from skeletal muscle cells than during rest.[54] Release of potassium during the recovery phase of the action potential and the ensuing plasma hyperkalemia depends on the degree of exercise. Plasma potassium concentration increases up to 0.3 mEq/L with slow walking and up to 2.0 mEq/L with heavy exercise. The extent to which these conditions alter plasma potassium concentration depends on the integrity of the sarcolemma. Under normal conditions, plasma potassium concentration is regulated by homeostatic mechanisms, such as the secretion and action of epinephrine, insulin, and aldosterone.[54]

Dehydration and Its Effects on Skeletal Muscle Function

During dehydration, plasma hyperosmolarity is exacerbated as water is redistributed from the intracellular to the extracellular compartments of skeletal muscle in an attempt to maintain normal blood osmolarity. Muscle proteins affected most by dehydration are those involved in electrolyte distribution across the sarcolemma (ie, sodium-potassium and calcium adenosine triphosphatases), calcium release and reuptake by the sarcoplasmic reticulum, and components of the mitochondrial respiratory chain.[7] Cardiovascular compensatory mechanisms for thermoregulatory blood pooling in the skin determine tolerance to dehydration and exercise in the heat. Exercise performance decreases as less blood is available for perfusion of active skeletal muscle. Blood flow to exercising muscles is significantly reduced with dehydration due to reductions in blood pressure and perfusion pressure.[55]

Pain Associated With Delayed-Onset Muscle Soreness

Signs and symptoms of DOMS may be attributed to increased extracellular fluid osmolarity, which leads to swelling, edema, decreased ROM, and elevation of intramuscular pressure.[56,57] Group IV sensory afferent neurons terminate in the connective tissue between myofibers and may be sensitive to the increased osmotic pressure produced by leakage of cellular contents. Activation of these neurons would result in the sensation of dull, diffuse pain associated with DOMS.[58]

Damage to the sarcolemma leads to the inability of the cell to modulate cellular functions and ultimately contributes to the signs and symptoms of DOMS. The reduced ability of the sarcolemma to contain and regulate cellular contents allows extracellular calcium to enter the cell, following its concentration gradient.[59,60] Increased intracellular free calcium in the sarcoplasm may also result from damage or impairment of the sarcoplasmic reticulum, decreasing its ability to resequester calcium.[60,61] Eccentric muscle tension produces sarcolemma damage and increased intracellular free calcium in the sarcoplasm. Increased intramuscular free calcium activates phospholipase A_2,[36,62] stimulating the inflammatory response associated with DOMS and promoting further degradation of cellular structures. Within 12 hours of the initial damage, cellular infiltration of neutrophils is followed by monocytes,[17,51] lymphocytes, granulocytes, and interleukins.[63–65] Further damage to cellular structures is caused by free radicals produced by infiltrated monocytes and calcium-stimulated proteases such as calpains.[59] Eccentric muscle tension producing sarcolemma impairment leads to extensive skeletal muscle microdamage and DOMS.[12,13,66] Altered osmotic pressure gradients at the tissue-capillary interface result in fluid and solute shifts in and out of the cell. Osmotic pressure gradient alterations may contribute to increased plasma osmolarity (hyperosmolarity or hemoconcentration). Fluid shifts associated with plasma hyperosmolarity adversely affect the ability of the body to regulate temperature and subsequently decrease skeletal muscle function.[6]

Muscle Tenderness Associated With Delayed-Onset Muscle Soreness

The increased perceived pain and punctate tenderness after downhill running were likely the result of microdamage from mechanical stress on the active muscles. Although not directly measured in this study, eccentric exercise-induced mechanical stress results in microdamage to the sarcolemma and contractile elements of the myofiber.[37,38] The damaged sarcolemma allows efflux of intramuscular contents, increasing the osmolarity of the extracellular fluid.[49–52] Increased osmolarity of the extracellular fluid stimulates group IV sensory afferent nerve fibers terminating near the myofiber, resulting in increased perceived pain.[58] A damaged sarcolemma would have also resulted in increased intramuscular pressure, which sensitized sensory afferents terminating in the connective tissues between myofibers.[36,30,41] Increased punctate tenderness is attributed to increased sensitivity to palpation as a result of increased intramuscular pressure.[39,67]

Of the 3 quadriceps muscles assessed for punctate tenderness, only the vastus medialis showed a significant time effect. Newham et al[10,68] reported increased punctate tenderness in the vastii muscles with the rectus femoris being spared 24 to 48 hours after bench stepping.[10,34,68] Schwane et al[35] reported increased punctate tenderness in the quadriceps and other postural and accessory muscles of the lower extremity 24 to 48 hours after downhill running. In a related study, Cleary et al[69] reported increased punctate tenderness of the vastii muscles 24 to 72 hours after downhill running and no effect on the rectus femoris. Ciccotti et al[70] reported that the vastus medialis had

significantly greater motor unit recruitment than the vastus lateralis and rectus femoris during the loading phase of running and downhill walking. Accordingly, the vastus medialis is an important patellofemoral joint stabilizer during ambulation, which leads to increased mechanical tension during the eccentric phase of the gait cycle and the corresponding increased punctate tenderness. The signs and symptoms of DOMS were not significantly different between the euhydrated and dehydrated groups, with one exception. Punctate tenderness of the bilateral vastus lateralis muscle was higher for the euhydrated than the dehydrated participants at 72 and 96 hours postexercise. This finding is an enigma and is counter to what was anticipated.

Swelling Associated With Delayed-Onset Muscle Soreness

Distal bilateral thigh circumference was significantly greater at 0.5 and 24 hours postexercise than at preexercise for the euhydrated and dehydrated participants. The metabolic demands of the active quadriceps muscles require increased vascular perfusion for oxygen and glucose delivery and metabolic waste removal, resulting in the increased distal thigh circumference at 0.5 hours postexercise.[5,57] However, this was not the causative factor for the increase at 24 hours postexercise, which was attributed to muscle swelling associated with the inflammatory response.[39,51] Smith et al[63–65] reported increased circulating indices of inflammation 12 to 24 hours after downhill running, which they attributed to microdamage of the exercised myofibers. Using radionuclide-labeled leukocytes, MacIntyre et al[71] confirmed the presence of inflammatory cells throughout the quadriceps 24 hours after eccentric quadriceps exercise.

Reduced passive ROM and isometric strength after an eccentric exercise perturbation have been frequently reported in the DOMS literature.[30,72] Bilateral passive knee-flexion ROM was not affected by downhill running, a finding also noted by Cleary et al.[69] This finding is in contrast to that of Ebbeling and Clarkson[28] and Nosaka and Clarkson,[73] who reported reduced passive ROM after eccentric free-weight loading of the elbow flexor muscles resulted in increased tension per motor unit area. Differences in study outcomes are attributed to differences in the mode of eccentric loading and the size of the musculature tested.

Strength Loss Associated With Delayed-Onset Muscle Soreness

We demonstrated that bilateral quadriceps isometric strength recovers by 96 hours postexercise compared with its nadir at 0.5 hours postexercise. Other researchers[30,71–73] have reported reduced isometric strength up to 6 days after isokinetic eccentric quadriceps exercise. Sargeant and Dolan[72] found reduced quadriceps isometric strength up to 96 hours after exhaustive downhill walking. Cleary et al[69] noted that 45 minutes of downhill running resulted in significantly lower isometric strength at 0.5 hours postexercise than at preexercise and significantly higher isometric strength at 96 hours postexercise than at preexercise. The intensity and duration of the downhill running exercise perturbation and the multiple strength assessments in the current study may have been sufficient to elicit the significant strength increase. The increase is likely the result of improved neural efficiency in motor unit recruitment.[74]

Dehydration Alone Does Not Affect Delayed-Onset Muscle Soreness

Athletic trainers and other practitioners should be aware that treadmill walking at a moderate intensity in a hot, humid environment with fluid restriction resulted in a 2.7% body mass reduction. Researchers[2] have demonstrated that body mass losses as low as 1% to 2% compromise physical performance as a result of cardiovascular compensations for heat load. The signs and symptoms of DOMS after an eccentric exercise perturbation were not exacerbated by moderate dehydration of 2.7% body mass with rest and a return to the normothermic condition. Although the goal had been to dehydrate the participants to 3%, this was not accomplished due to the participants' negative physiologic responses to the dehydration protocol.

Heat load in the current study was attenuated in the dehydrated participants during the 30- to 90-minute rest period before downhill running. Heat load indices were reduced before the downhill run, as demonstrated by no significant differences between the euhydrated and dehydrated participants' thermoregulatory, cardiorespiratory, and rating of perceived exertion responses. In our study, the effects of lower extremity exercise and moderate dehydration were attenuated by rest. The attenuated effects may not have been severe enough to elicit deficits in muscle function.

We acknowledge the limitations of the current study. Power for nonsignificant findings between groups ranged from .052 to .469, which may be attributed to our small sample size or a less than anticipated effect size. Acclimation probably was not a factor, as the average maximum ambient temperature during the data collection period was 27.9 $\pm$ 4.8°C and participants did become moderately dehydrated. It is also possible that downhill running in a hot, humid environment or dehydration levels greater than 2.7% may elicit a threshold sufficient to exacerbate the signs and symptoms of DOMS.

Water lost via sweating is thought to be replaced by movement of intracellular water from inactive tissues to defend plasma volume and active skeletal muscle during dehydration. This outcome has been substantiated in prior research.[15,75] Greiwe et al[76] reported that dehydration of 3.8% body mass and attenuation of heat load indices by a 3-hour rest had no significant effect on isometric quadriceps muscle strength. Nielsen et al[5] noted that 60 minutes of uphill walking in the heat without dehydration increased intramuscular temperatures but did not affect muscle blood flow. Moderate dehydration without a concomitant heat load does not exacerbate eccentric exercise-induced skeletal muscle microdamage.

Practitioners should be aware that dehydration combined with heat load from the environment and from metabolic heat generated during eccentric exercise may potentiate the signs and symptoms of DOMS in dehydrated participants and warrants further investigation. Cleary et al[69] demonstrated more perceived pain and punctate quadriceps tenderness 24 to 72 hours after 45 minutes of downhill running in hyperthermic participants who were 3.3% dehydrated than in hyperthermic participants who were euhydrated. Eccentric exercise-induced microdamage and the concomitant signs and symptoms of DOMS may be exacerbated by reduced tissue perfusion, heat

load, metabolic heat, reduced muscle strength, and denaturation of structural and functional proteins.[7]

We demonstrated that a treadmill walking protocol in a hot, humid environment resulted in a 2.7% reduction in body mass and the downhill running eccentric exercise perturbation resulted in a classic pattern of the signs and symptoms of DOMS. Athletes participating with DOMS may be susceptible to further injury as a result of pain, swelling, and decreased muscle function. Although dehydration to 2.7% of preexercise body mass with rest and attenuation of heat load had no deleterious effects on the signs and symptoms of DOMS, the extent to which this is true at dehydration levels greater than 2.7% is unknown and warrants further study.

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