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Effect of Cupping Therapy on Respiratory Gas Exchange in Trained Endurance Runners

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Trained Endurance Runners

By

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of the Requirements for the Degree
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Maximilian T. Antush

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A Thesis

Presented to

The Faculty of

Western Washington University

In Partial Fulfilment

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Abstract

The objective of this study was to elucidate the effects of myofascial decompression through cupping therapy (CT) on running economy (RE) in well-trained runners. Five minutes of CT or placebo gel (PG) was applied to bilateral hip extensor muscles of 15 well-trained runners (n = 7 female, n = 8 male) after a 10-minute treadmill warm-up. Running economy was measured using two 6-minute steady-state treadmill runs, one at a standardized velocity (females = 3.93 m·s^{-1}; males = 4.47 m·s^{-1}) and the other at 10-km race velocity. Maximal oxygen consumption (VO$_2$ max) test followed the RE tests. All subjects performed both conditions in randomized order. Running economy, respiratory exchange ratio (RER) during steady-state, and VO$_2$ max after CT and PG were compared independently using paired two-sample t-tests. Effect size for all variables was calculated using Cohen's *d*. There was no difference in RE expressed as %VO$_2$ max between CT and PG (standard = 76.9 ± 10.6% of VO$_2$ max vs. 76.6 ± 10.5% of VO$_2$ max, *p* = 0.72, *d* = 0.02; 10-km = 84.2 ± 7.2% of VO$_2$ max vs. 83.7 ± 6.9% of VO$_2$ max, *p* = 0.17, *d* = 0.07). There was also no difference in VO$_2$ max between CT and PG (65.1 ± 9.1 mL·kg^{-1}·min^{-1} vs. 65.0 ± 10.3 mL·kg^{-1}·min^{-1}, *p* = 0.96, d = 0.004); however, RER was significantly increased by CT compared to gel (standard = 0.92 ± 0.06 vs. 0.90 ± 0.04, *p* = 0.04, d = 0.32; 10-km = 0.94 ± 0.04 vs. 0.92 ± 0.03, *p* = 0.02, d = 0.52). Acute CT increases steady-state carbon dioxide expiration in well-trained runners without changing oxygen consumption. This has implications for enhanced buffering from putative increased localized blood.

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Introduction

        Compression therapy, through various forms of massage, has been a common method of therapeutic rehabilitation throughout the history of modern athletics. Compression therapy is commonly applied as manual pressure of human tissue through manual compressions and rhythmic percussions with the purpose to increase blood and lymphatic flow (Nunes et al., 2016). More recently, decompression therapy, applied as negative pressure on human tissue through mechanical manipulation, has been used in the rehabilitation treatment of athletes. The most pervasive form of negative pressure decompression therapy used in athletics is cupping therapy (CT).

        Despite the prevalence of its use, literature on the efficacy of CT is limited to primarily pain management and range of motion. Subjects with non-specific neck pain have reported that CT reduced pain both at rest and during motion and a review of randomized clinical trials of CT concluded that CT reduces pain intensity in chronic and acute pain (Cramer et al., 2011; Lauche et al., 2011; Cao et al., 2014). Cupping therapy used with anterior knee pain patients resulted in significant improvements in both passive and active range of motion and a reduction in pain (Ullah et al., 2007). Additionally, CT increased lower body range of motion in healthy subjects while also increasing electromyography activation of the treated muscles (Kim et al., 2017).

        One potential mechanism of action of CT is increased skeletal muscle blood flow. Arce-Esquivel et al. (2017) applied dry CT on the arm of apparently healthy young adults and microvascular function was evaluated using fingertip digital thermal monitoring of vascular reactivity before and after a 10-minute cupping intervention. Following the 10-minute cupping treatment, the individuals experienced a significant 36% increase in

Vascular Reactivity Index, a measure of microvascular function, with subjects experiencing no complications as a result of the intervention (Arce-Esquivel et al., 2017). Utilizing a 5-minute dry cupping intervention with a conventional cupping set, Li et al. (2017) reported both a prominent drop in deoxy-hemoglobin and significant elevation in oxy-hemoglobin surrounding the cupping site both during cupping and post-treatment as measured by near-infrared spectroscopy.

Running performance is limited by the potential to provide the energy required to cover a given distance via aerobic and anaerobic metabolism (Beneke and Hütler, 2005). Endurance training adaptations, such as increased morphology and functionality of skeletal muscle mitochondria, an increase in oxidative muscle capacity, and hematological changes, have been suggested as factors that act to improve an athlete's running economy (RE) (Barnes and Kilding, 2015). Previous research has indicated that RE is a significant determining factor in endurance running performance when comparing athletes who have a similar maximal oxygen consumption (VO_2max) (Conley and Krahenbuhl, 1980; Ziogas et al., 2011). Conley and Krahenbuhl (1980) found that among a group of top finishers in a nationally prominent 10-km road race, who had similar relative VO_2max values, 65.4% of the variation observed in race performance on the 10-km race can be explained by variation in RE. Running economy has also been used to explain similar running performances between athletes who have a variation in VO_2max (Weston et al., 2000). Weston et al. (2000) compared eight Caucasian runners and eight African runners who, as two groups, achieved similar race times over 10-km. The African runners had a mean relative VO_2max that was 13% lower than the mean relative VO_2max for the Caucasian runners and were 5% more economical when running at a speed of 16.1 km/h (Weston et al., 2000).

Cupping therapy may improve microcirculation, intramuscular vasodilation, endothelial repair, and angiogenesis of local tissue, as well as elevate regional blood oxygen levels, thus clearing metabolic contaminants (Emerich et al., 2014; Li et al., 2017; Look and Look, 1997; Mehta and Dhapte, 2015; Wang et al., 2014; Yoo and Tausk, 2004). The enhancement of localized blood flow has implications of improved RE and respiratory buffering; both key metrics to predict running performance (Barnes and Kilding ,2014; Dollery et al., 1962). However, the direct effect CT has on RE and respiratory buffering has not been researched. Therefore, the purpose of this study is to elucidate the effects of myofascial decompression through CT applied to key hip extensor muscle groups on RE and respiratory gas exchange. We hypothesized that an acute single treatment of CT would improve RE and enhance respiratory buffering in well-trained runners when compared to a placebo.

Methods

Ethical Consideration

Subject participation in this research study was voluntary. Each individual subject was given a verbal and written explanation of the purpose of the study, the methods of data collection, potential risks associated with participation, and their rights as a human subject. Each individual subject signed an informed consent form. At the conclusion of the study, subjects were informed of the deception associated with the placebo condition. This research study was approved by the Western Washington University Institutional Review Board.

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Subjects

Fifteen competitive runners, seven female (29.3 ± 2.1 yrs, 1.68 ± 0.06 m, 60.2 ± 3.4 kg) and eight male (27.5 ± 6.2 yrs, 1.77 ± 0.04 m, 69.1 ± 4.0 kg) were recruited from the greater Bellingham and Western Washington University running community. Subjects were all well-trained distance runners, averaging at least 30 miles per week of running specific training over the previous six weeks, with average 10-km race times of 41.4 ± 4.4 min for females and 33.5 ± 1.2 min for males. Subjects were excluded from participation in this study if they had current injuries that limited their running performance, were unable to perform a maximal hip extension against weighted resistance, or they were unable to complete the prescribed running tests during the study. Each individual subject was instructed to wear their normal running footwear and comfortable attire for running indoors on a treadmill.

Outcome Measures

Oxygen consumption and CO_2 expiration values for RE, respiratory exchange ratio (RER), and VO_2max were determined through the analysis of collected respiratory gas by the TrueOne 2400 Metabolic Measurement System (ParvoMedics, Murray, UT, USA) using a two-way breathing valve. Carbon dioxide expired was recorded as liters per minute ($L \cdot min^{-1}$). Running economy was defined as the steady-state oxygen consumption at a constant workload and expressed as a percentage of VO_2max (Barnes and Kilding, 2015; Conley and Krahenbuhl, 1980; Morgan et al., 1989; Saunders et al., 2004). Subject VO_2max was recorded as milliliters per kilogram per minute ($mL \cdot kg^{-1} \cdot min^{-1}$). Respiratory exchange ratio was expressed as the volume of CO_2 expired to the volume of O_2 consumed.

Experimental Procedure

The study was a two-group crossover design in which subjects were randomly assigned on their first session, via flip of a coin, whether they completed the RE tests with CT or placebo gel (PG). The alternative condition was performed on the second session. Each subject completed two sessions with a minimum of a one-week washout period, but not more than three weeks, between conditions. Subjects were instructed to avoid intense physical activity in the 48 hours prior to testing sessions and to maintain their normal training routines between training sessions. For the placebo condition, a salt-free, chloride-free, water-soluble electrode gel (Spectra 360 Electrode Gel, Parker Laboratories Inc., Fairfield, NJ, USA) was applied to the subject. Subjects were told that the PG had been shown to increase O_2 consumption. For the test protocol, subjects performed a 10-minute jogging warm up on the treadmill (T645 Treadmill, SportsArt Fitness, Mukilteo, WA, USA) at a self-selected velocity followed by either CT or PG protocol. Subjects were given a three-minute rest period and a five-minute self-selected pace jog on the treadmill prior to the RE and VO_2max test protocol.

An FDA-approved medical grade professional Chinese cupping therapy set (Care me, Inc., Norcross, GA, USA) was used to perform CT on the primary hip extensor muscles of the subjects. A clean cupping set was used for each subject. The application sites for each subject were the muscle belly of the gluteus maximus, biceps femoris long head, semimembranosus, and semitendinosus muscles on both the right and left legs (Hamner et al., 2010). A vacuum seal to apply negative pressure was created in the cups using a hand pump with pumps applied until 1.6 cm of skin was elevated into each cup (Markowski et al., 2014). Cups were left on subjects for five minutes (Cramer et al., 2011; Shixi and Yu,

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2006). Cups were removed from subjects in the same order that they were applied. Cupping therapy was performed by a certified athletic trainer experienced in the technique. The PG trial followed the same protocol.

Prior to each individual testing session, the metabolic cart was calibrated in accordance with the manufacturer's protocol. Subjects were instrumented and RE was evaluated using an adapted procedure from Daniels and Daniels (1992) and Weston et al. (2000). Each test session included two submaximal, level-grade treadmill runs followed by a constant-speed, increasing-grade treadmill run to determine VO_2max. Submaximal speeds that were used were 3.93 m·s^{-1} for females or 4.47 m·s^{-1} for males and the subject's 10-km race pace. Submaximal speeds used for evaluating RE have ranged from 3.35 m·s^{-1} to 5.81 m·s^{-1}, with speeds determined based on the demographics of the subject pool (Conley and Krahenbuhl, 1980; Daniels and Daniels, 1992; Morgan et al., 1989; Saunders et al., 2004; Weston et al., 2000). Subject's 10-km race pace was determined by the subject's self-reported 10-km race pace on the initial subject demographic questionnaire. Each submaximal test lasted six minutes with gas analysis recorded by the metabolic cart using a 15-second average during the last two minutes of each stage. There was a five-minute recovery period between submaximal tests. Ten minutes following the final submaximal test, a VO_2max test was performed, using the subject's 10-km race pace as the test speed for the max test. The first two minutes of each max test were at 0% grade with 1% grade added to the treadmill each subsequent minute starting with minute three. The test was terminated when the subject decided s/he could not complete another minute or subject VO_2 did not continue to increase with a subsequent increase in treadmill incline. The highest VO_2 recorded was accepted as VO_2max.

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Data Analysis

Running economy for each submaximal test velocity was calculated as the subject's steady-state VO_2 at the test velocity divided by the subject's VO_2max and reported as a percentage of subject's VO_2max. Respiratory exchange ratio was calculated as the volume of CO_2 expired to the volume of O_2 consumed and expressed as such. Subject VO_2max was expressed as $mL \cdot kg^{-1} \cdot min^{-1}$. Paired two-sample t-tests were used to determine differences in RE, RER and VCO_2 during steady-state, and VO_2max between CT and PG conditions. Statistical significance was established at $p < 0.05$. Effect size was calculated, and Cohen's *d* was used to indicate the standardized difference between means; <0.25 = trivial, 0.25-0.50 = small, 0.50-1.0 = moderate, >1.0 = large (Rhea, 2004). Data analysis was performed using Microsoft Excel (Microsoft Corporation, Redmond, WA).

Results

Running Economy and VO_2max

The mean RE values with CT and PG are shown in Figure 1 and mean VO_2max values are shown in Figure 2. There were no significant differences between CT and PG in RE (standard: $76.9 \pm 10.6\%$ of VO_2max vs. $76.6 \pm 10.5\%$ of VO_2max, $p = 0.72$, $d = 0.02$; 10-km: $84.2 \pm 7.2\%$ of VO_2max vs. $83.7 \pm 6.9\%$ of VO_2max, $p = 0.17$, $d = 0.07$) or VO_2max (65.1 ± 9.1 $mL \cdot kg^{-1} \cdot min^{-1}$ vs. 65.0 ± 10.3 $mL \cdot kg^{-1} \cdot min^{-1}$, $p = 0.96$, $d = 0.004$).
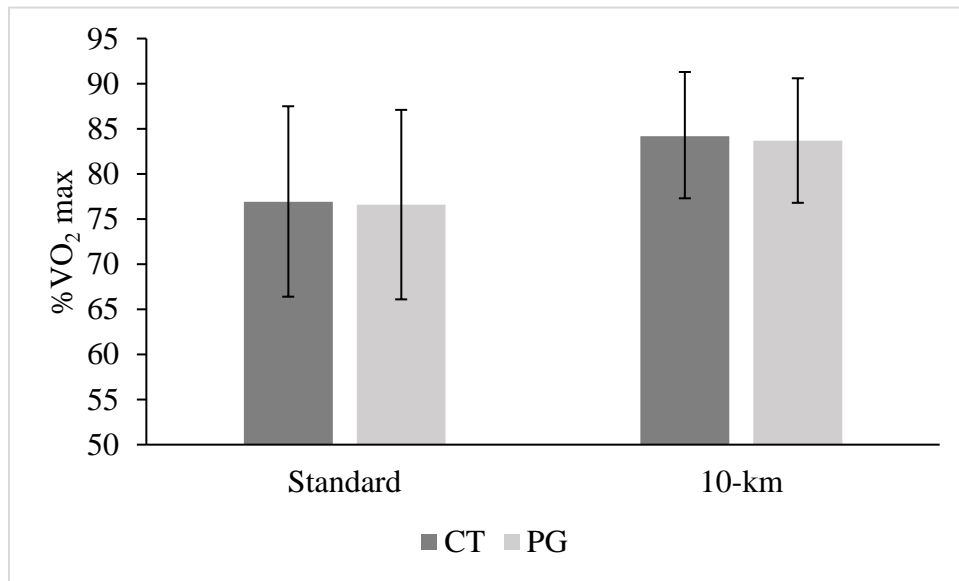
Figure 1. Mean RE values for CT vs. PG at the standardized velocity and subject's 10-km race velocity.
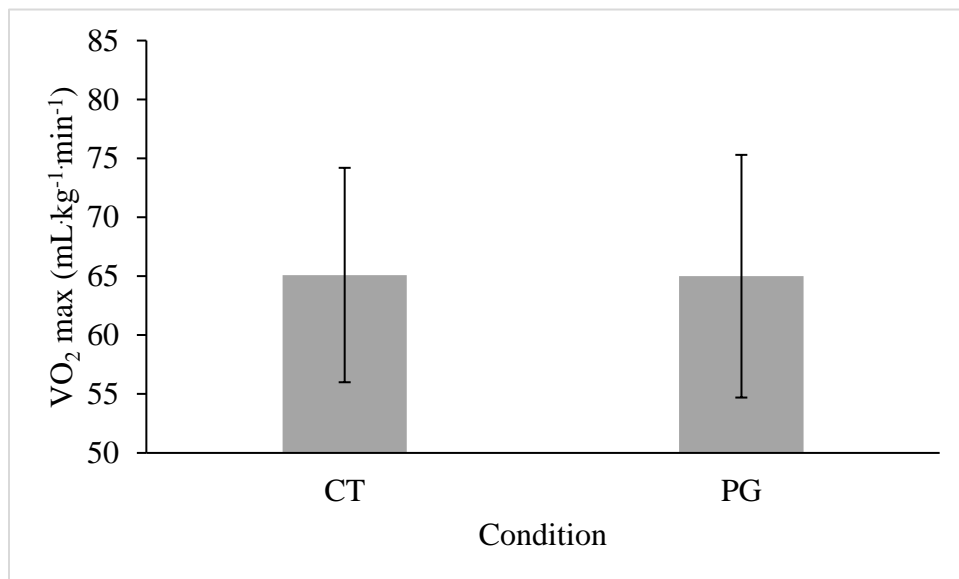
Figure 2. Mean VO$_2$max values for CT vs. PG.

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Respiratory Gas Exchange

The mean RER values during steady-state are shown in Figure 3 and mean CO_2 expiration rate are shown in Figure 4. Cupping therapy significantly increased RER compared to PG at both the standardized running velocity (0.92 ± 0.06 vs. 0.90 ± 0.04, $p = 0.04$, $d = 0.32$) and subject's 10-km running velocity (0.94 ± 0.04 vs. 0.92 ± 0.03, $p = 0.02$, $d = 0.52$). There was no significant difference in CO_2 expiration rate during steady-state between CT and PG at the standardized running velocity (2.93 ± 0.41 L·min^{-1} vs. 2.85 ± 0.34 L·min^{-1}, $p = 0.09$, $d = 0.20$); however, CT significantly increased CO_2 expiration rate during steady-state compared to PG at subject's 10-km running velocity (3.34 ± 0.64 L·min^{-1} vs. 3.24 ± 0.58 L·min^{-1}, $p = 0.01$, $d = 0.16$).
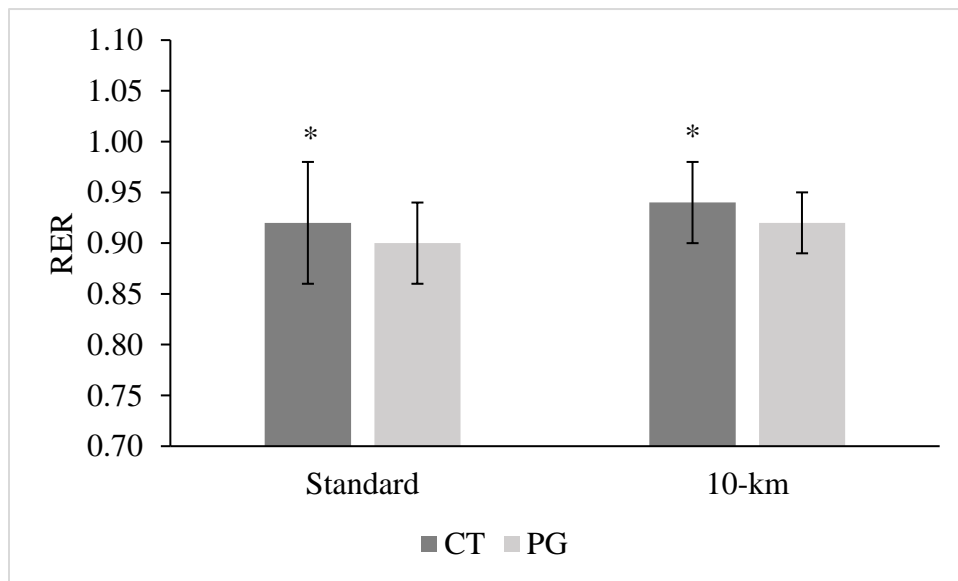
Figure 3. Mean RER values for CT vs. PG at the standard velocity and the subject's 10-km race velocity; *indicates statistically significant result.
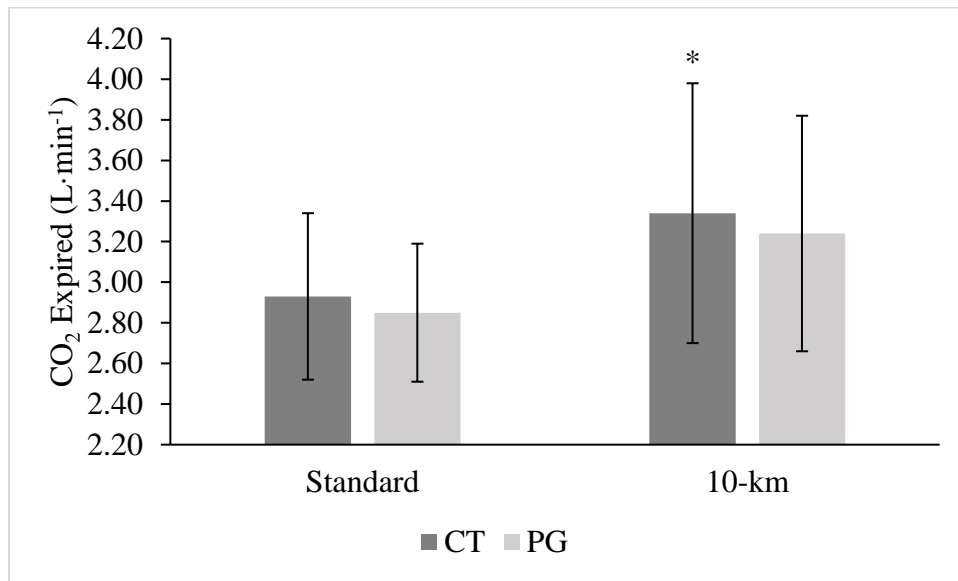
Figure 4. Mean CO_2 expiration rate values for CT vs. PG at the standard velocity and the subject's 10-km race velocity; *indicates statistically significant result.

Discussion

The main finding of this study was that there was no difference in RE or VO_2max between CT and PG in well-trained endurance runners at either a standardized velocity or 10-km race velocity; however, there was a significant increase in RER at both velocities and a significant increase in CO_2 expiration rate at 10-km race velocity with CT. Thus, our initial hypothesis that an acute single treatment of cupping therapy would improve RE and enhance respiratory buffering in well-trained runners was partially confirmed.

Although the definition of RE is simple in concept, RE is a multifactorial measure which reflects the combined functioning of the metabolic, cardiopulmonary, musculoskeletal, and neuromuscular systems (Barnes and Kilding, 2015). More specifically, some reported contributing factors to RE include: time of day, gender, age, running environment (i.e. treadmill or over-ground), temperature, fatigue, training

experience, body mass, body and segment mass distribution, running speed, stride length, stride frequency, ground reaction forces, and capacity to generate mechanical power (Morgan et al., 1989). Because of the various factors that influence RE, there is a limit to controls that can be implemented while maintaining external validity. Running economy is a measure of global oxygen consumption during steady-state submaximal running that is influenced by more than just the cardiovascular and respiratory systems. If CT only stimulates localized increases in oxygen consumption, RE might not reflect the potential performance enhancements associated with acute CT.

In well-trained runners, VO₂max has a very low trainability and remains virtually unchanged despite performance improvements (Legaz Arrese et al., 2005). With the subject population of this study, maximal O_2 consumption was a physiological performance variable that may likely see minimal improvements with subsequent training interventions. In exercising humans, O_2 delivery, not skeletal muscle O_2 extraction, is viewed as the primary limiting factor for VO₂max because when O_2 delivery is altered by blood doping, hypoxia, or beta-blockade, VO₂max changes accordingly, the improvement in VO₂max with training results primarily from increasing cardiac output rather than increasing a-v O_2 difference, and when a small muscle mass is oversaturated during exercise, it has an exceptionally high capacity for consuming O_2 (Bassett JR and Howley, 2000). Because CT may only have local blood flow effects that are limited to the region CT was applied, it is possible that CT could affect regional skeletal muscle O_2 extraction and not global O_2 delivery.

The significant increase in steady-state RER and CO_2 expiration rate as a result of CT was consistent with increased localized blood flow clearing metabolic waste products.

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As the rate of blood flow increases, the rate of CO_2 cleared increases (Dollery et al., 1962). There was not a change in workload between the CT and PG conditions, as evidenced by treadmill velocity, duration of treadmill tests, or steady-state O_2 consumption, so enhanced localized microvascular blood flow would likely be attributable to the CT, similar to the findings of Arce-Esquivel et al. (2017). Oxygen delivery to the muscles is dependent on the intake of oxygen during respiration; whereas, CO_2 expiration is dependent on the CO_2 produced as a metabolic waste product during aerobic metabolism and bicarbonate buffering in response to anaerobic metabolism. Because aerobic and anaerobic metabolism take place at the active skeletal muscle level during exercise and the hip extensor muscles to which CT was applied are integral to running, increases in localized blood flow to those muscles would likely increase the rate of metabolic waste product clearance.

There were several limitations to this study. Although the sample in this study was a homogenous demographic, it is a limitation that the findings of this study are based on a relatively small sample size. There is not presently a standardized protocol for CT that is accepted by practitioners and changes in outcomes based on the differences in methodology of CT application, such as time-course of application or magnitude of negative pressure applied, has not been researched. The sites of CT application that were selected were limited to hip extensor muscles; however, those are not the only skeletal muscles that are active during running and results could possibly be influenced by the selection of different or additional muscles as application sites.

Based on the results of this study, future research on the effect CT has on CO_2 clearance should be extended to more diverse population groups. Although there are implications that increased CO_2 expiration rates would improve running performance, more

direct research is necessary on the impact that CT may have on endurance running performance. Additionally, future research measuring oxy-hemoglobin levels and blood CO_2 levels during and post-cupping therapy may provide valuable insight to the precise mechanism of CO_2 expiration rate increases as a result of CT and the potential efficacy of CT impacting changes in O_2 delivery to those particular CT application sites.

Conclusion

The findings of this study suggest that acute cupping therapy increases steady-state carbon dioxide expiration in well-trained runners without changing oxygen consumption. This has implications for enhanced buffering from putative increased localized blood, which could have an impact on endurance running performance.

Declarations

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Review of Pertinent Literature

Decompression Therapy in Athletics

Compression therapy, through various forms of massage, has been a common method of therapeutic rehabilitation throughout the history of modern athletics. Compression therapy is commonly applied as manual pressure of human tissue through manual compressions and rhythmic percussions with the purpose to increase blood and lymphatic flow (Nunes et al., 2016). More recently, decompression therapy, applied as negative pressure on human tissue through mechanical manipulation, has been used in the rehabilitation treatment of athletes. The most pervasive form of negative pressure decompression therapy used in athletics is cupping therapy.

Notably, U.S. Olympic swimmer Michael Phelps displayed a series of circular markings on his back and arms, characteristic of cupping treatment, at the 2016 Rio Olympics. Inquiries about the marks prompted *USA Today* magazine to publish an article that reports cupping therapy as being popular among members of the USA track & field team and an increase in blood flow as being responsible for the resulting skin discoloration (Peter, 2016). Published the same day, an article in *Independent* magazine reported that despite the popularity of cupping therapy in athletics, the treatment benefits were "desperately implausible" (Cockburn, 2016). There currently are no available published studies quantifying improved athletic performance due to cupping therapy.

Application of Cupping Therapy

The primary use of cupping therapy in western culture that has been extensively researched is pain management. Lauche et al. (2011) compared reported pain symptoms of participants with chronic non-specific neck pain who either received cupping therapy

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treatment, N = 22, or were in the waiting list control group, N = 24. Subjects receiving the cupping treatment reported significant improvements in pain at rest, $p = 0.00002$, pain at movement, $p = 0.01$, and Neck Disability Index, $p = 0.002$, when compared to control group subjects. Cramer et al. (2011) compared a treatment group receiving cupping therapy, N = 25, against a control group receiving self-directed standard medical care, N = 25, in subjects with chronic neck pain, reporting a significant difference in the improvement of pain at motion, $p < 0.001$, and functional disability, $p = 0.025$, of the treatment group over the control group. A review presented randomized clinical trials of cupping therapy, 16 trials with a total of 921 participants, and observed that cupping therapy reduces pain intensity in chronic or acute pain (Cao et al., 2014).

In addition to pain management, cupping therapy has been reported to enhance both passive range of motion (PROM) and active range of motion (AROM). Cupping therapy used with anterior knee pain patients significantly increased knee PROM immediately after cupping, $148.45 \pm 7.60° \ p = 0.001$, 1 week after cupping, $150.67 \pm 7.18° \ p = 0.003$, 2 weeks after cupping, $150.58 \pm 6.56° \ p = 0.003$, and 3 weeks after cupping, $151.31 \pm 5.96° \ p = 0.002$, compared to PROM before cupping, $142.64 \pm 11.17°$ (Ullah et al., 2007). Ullah et al. (2007) reported significantly increased knee AROM immediately after cupping, $140.00 \pm 7.26° \ p = 0.022$, 1 week after cupping, $143.33 \pm 7.50° \ p = 0.045$, 2 weeks after cupping, $145.67 \pm 8.50° \ p = 0.005$, and 3 weeks after cupping, $147.24 \pm 7.04° \ p = 0.005$, compared to AROM before cupping, $134.14 \pm 16.53°$. Additionally, pain was reported to be significantly reduced at all time-points after cupping when compared to before cupping, $p < 0.0001$ (Ullah et al., 2007). In patients with subacute low back pain, patients exhibited significantly increased lumbar flexion range of motion (ROM), $p = 0.016$, and decreased

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pain, $p < 0.0001$, post-cupping treatment compared to pre-cupping treatment (Markowski et al., 2014).

Although improved ROM could be attributed to reductions in pain perception in injured patients, improved ROM was exhibited post-myofascial release and post-cupping therapy in healthy subjects as well. Myofascial release of the quadricep with foam rolling significantly increased knee joint range of motion by 10° at 2 minutes post-treatment and 8° at 10 minutes post-treatment, $p < 0.001$, in a physically active, college-aged, healthy male population without a subsequent deficit in muscle force, electromyography (EMG) activation, rate of force development (RFD), or twitch force (MacDonald et al., 2013). Kim et al. (2017) reported that cupping therapy improved PROM, $p < 0.001$, AROM, $p < 0.001$, semitendinosus EMG, $p = 0.01$, and biceps femoris EMG, $p = 0.01$ in 15 healthy male and female subjects in their 20s and 30s. These outcome measures were similar to subjects who performed passive stretching as their therapeutic intervention, indicating that cupping therapy should be considered as another option to treat range of motion and muscle activity in a clinical setting (Kim et al., 2017).

Cupping Methodology

To enhance the potential for an effective cupping treatment, there are some procedural precautions that should be observed. Generally, cups should be applied to areas with thick muscle with a suitable size of cup selected and subjects in a proper body position (Shixi and Yu, 2006). Additionally, cupping should not be applied over areas of skin with dermatitis, ulceration, swelling, or over an artery or the heart (Shixi and Yu, 2006). Although many patients had residual markings after treatment due to engorged blood

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vessels; unlike bruises, these commonly seen marks are not sensitive to touch, and they fade in one to 10 days (Markowski et al., 2014).

Although there is individual variability in the time course of cupping application, most applications last for a duration of five to 20 minutes. Shixi and Yu (2006) caution that the time for retaining the cup on the skin should not be too long and that five to 10 minutes is generally appropriate for most cases. Lauche et al. (2011) removed cups after 10 to 20 minutes depending on the color of the circular cupping marks, which range from slightly rose to dark pink, in subjects with chronic non-specific neck pain. Cramer et al. (2011) applied cupping therapy to the trapezius muscle for five to 10 minutes following a 10 to 15 minute treatment of pneumatic pulsation therapy in subjects with chronic neck pain. Markowski et al. (2014) applied cups for 10 minutes in subjects with low back pain. Hand pump suction was applied until 1.6 cm of skin was elevated within each cup to standardize the procedure and because previous trials with this magnitude of suction consistently achieved the suction effect without patient discomfort (Markowski et al., 2014). Arce-Esquivel et al. (2017) applied cups for 10 minutes on the forearm of healthy subjects to assess increased blood flow.

Proposed Mechanisms of Cupping Therapy

Cupping therapy is a form of negative pressure, myofascial decompression treatment using vacuum suction cups that was used as an ancient medical technique in European, Asian, and Middle Eastern cultures (Al-Rawi and Fetters, 2012; Lauche et al., 2011). The two primary forms of cupping are wet cupping and dry cupping. Wet cupping is performed by making small punctures to the skin in the treatment area prior to the application of the vacuum cups; whereas, dry cupping does not breach the skin (Cao et al.,

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2012; Michalsen et al., 2009). In Chinese medicine, the most common method of dry cupping generates suction through use of a flame; commonly referred to as "fire cupping" (Mehta and Dhapte, 2015). However, many Western practitioners of dry cupping use manual methods or suction pumps to create a vacuum within the cups. According to Tham et al. (2006), tensile load in the center of the cup and the compression of tissues at the rim of the cup transmits forces through the connective tissue layers to the muscle. Additionally, Tham et al. (2006) reported that larger diameter cups provided greater stress, which exerted more force on the tissue being treated. Although there is a plethora of research that reports various methods of cupping therapy as a treatment for a wide range of ailments, primary research that investigates the physiological mechanism through which cupping therapy acts as a treatment is limited at this time.

The aponeurotic fascia that ensheath skeletal muscle has two or three layers of parallel collagen fiber bundles with each layer separated by a thin layer of loose connective tissue that allows for sliding of the fascial layers over each other; additionally, the aponeurotic fascia is richly innervated, specifically in the superficial sublayer (Tesarz et al., 2011). Epimysial fascia has a similar multilayered collagen structure to that of aponeurotic fascia, gives insertions to aponeurotic fascia, and has a fundamental role in the transmission of muscle generates force toward skeletal levers (Purslow, 1989; Stecco et al., 2013). Between the collagen fibers of epimysial fascia is a ground substance with an abundance of proteoglycans, namely hyaluronic acid, which allows the collagen fibers to slide with little friction (McCombe et al., 2001). In physiological solutions and under tightly contained environments, like within fascial extracellular matrix, hyaluronic acid chains tend to bind to each other and entangle into complex rigid structures (Matteini et al., 2009). Instead of

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being a lubricating component of the myofascial layers, hyaluronic acid becomes adhesive

and the distribution of lines of force within the fascia become altered (Stecco et al., 2013).

Applying mechanical stress to the fascia, through techniques like cupping, increase the

temperature of the tissues and reduces the viscosity of the hyaluronic acid polymers

restoring it lubricative properties (Stecco et al., 2013). Consequently, cupping therapy

might stimulate improved fascial gliding, which would enhance cellular

mechanotransduction (Stecco et al., 2014). Rho-kinase, a key cytoskeleton regulatory

enzyme that's functions include cytoskeletal reorganization and actomyosin contractility,

activated in response to mechanical deformation of the tissues is a mechanism through

which mechanotransduction may stimulate fibroblast proliferation and collagen synthesis

(Amano et al., 2010; Fang, 2014; Gehlsen et al., 1999; Goldman et al., 2013; Langevin et

al., 2006).

Gehlsen et al. (1999) reported that augmented soft tissue mobilization (ASTM)

therapy on rats with Achilles tendonitis not only stimulated fibroblast proliferation, but that

the proliferative response was dependent on the magnitude of the applied ASTM pressure.

Although the Achilles tendonitis rats with no ASTM, light ASTM, and medium ASTM all

exhibited a significantly greater fibroblast cell count than rats not induced with Achilles

tendonitis, $p < 0.05$, tendonitis rats that received extreme ASTM exhibited significantly

greater fibroblast cell counts than all other groups, $p < 0.05$ (Gehlsen et al., 1999). This

suggests that fibroblast proliferation can be stimulated via mechanotransduction, with a

mechanical stimulus applied without breaking the skin surface being sufficient.

Additionally, Langevin et al. (2006) reported that increased preload of rat subcutaneous

tissue to a threshold of 4.9 mN before acupuncture without needle rotations increased

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fibroblast cell cross-sectional area, $p = 0.004$, and that zero to two acupuncture needle rotations in subcutaneous tissue preloaded to 2.9 mN also increased fibroblast cell cross-sectional area to a similar size observed in the greater preload and no needle rotations, $p < 0.001$. However, when fibroblasts in the experimental subcutaneous tissue were incubated in the presence of Rho kinase inhibitors, the proliferated fibroblast cells were significantly diminished in cross-sectional area, $p < 0.05$ (Langevin et al., 2006). These findings suggest that mechanotransduction stimulates fibroblast proliferation and subsequent collagen synthesis through Rho kinase mediated mechanisms.

A mechanical method that has been reported as stimulating an increase in blood flow is negative pressure. A study that investigated the application of intermittent negative pressure to the lower extremities to increase blood flow in the feet found an increase in arterial blood flow velocity in two of their four treatment sequences, from 8.9 cm/sec to 9.6 and 10.1 cm/sec $p < 0.001$, with an increase in peak blood flow velocity of 44% from the onset of negative pressure, $p < 0.001$ (Sundby et al., 2016). Similar results were reproduced in patients with peripheral arterial disease; peak blood flow increasing with intermittent negative pressure from 6.7 cm/sec to 7.5 cm/sec with $p = 0.03$ (Sundby et al., 2017). Another study evaluated physiological responses to lower body negative pressure and concluded that the principal human response to the translocation of a significant portion of the blood volume from the thorax to the lower body segments by lower body negative pressure is a decreased stroke volume and cardiac output (Raven et al., 1984).

Topical negative pressure therapy is a form of negative pressure that is used in wound care. Topical negative pressure therapy consists of a reticulated foam dressing that is inserted in the wound and sealed in place with the use of an adhesive dressing. A suction

force is then applied by a VAC (Vacuum Assisted Closure, Advanced Therapy System, KCI Whitney, Oxon, UK) machine across the wound surface (Jones et al., 2005). One of the mechanisms of healing that topical negative pressure therapy is reported to enhance is capillary blood flow within a wound (Cipolla et al., 2008). Lindstedt et al. (2007) reported that topical negative pressure therapy was used to increase microvascular blood flow in normal myocardium, 14.7 ± 3.9 PU to 25.8 ± 6.1 PU $p < 0.05$, ischemic myocardium, 7.2 ± 1.5 PU to 13.8 ± 2.6 PU $p < 0.05$, and re-perfused myocardium, 10.8 ± 2.0 PU to 19.3 ± 5.6 PU $p < 0.05$. Another study reported microvascular blood flow increases 2.5 cm from the site of application that increased with the magnitude of topical negative pressure therapy applied ranging from -10 mmHg to -80 mmHg, with no additional increases in blood flow as pressure increased (Borgquist et al., 2010). Negative pressure vacuum-assisted wound closure was reported to significantly upregulate mRNA and protein expression of both vascular endothelial growth factor and collagen I, $p < 0.01$ (Wang et al., 2014). This suggests that in addition to increasing blood flow to the area, negative pressure can stimulate the development of new blood vessels.

Cupping therapy, a form of negative pressure, may improve microcirculation, intramuscular vasodilation, endothelial repair, and angiogenesis of local tissue, as well as elevate regional blood oxygen levels, thus clearing metabolic contaminants (Emerich et al., 2014; Li et al., 2017; Look and Look, 1997; Mehta and Dhapte, 2015; Wang et al., 2014; Yoo and Tausk, 2004). Arce-Esquivel et al. (2017) applied dry cupping therapy on the arm of apparently healthy young adults and microvascular function was evaluated using fingertip Digital Thermal Monitoring of vascular reactivity before and after a 10-minute cupping intervention. Following the 10-minute cupping treatment, the individuals

experienced a significant 36% increase in Vascular Reactivity Index, 2.60 ± 0.40 to 3.53 ± 0.42 $p < 0.05$, with subjects experiencing no complications as a result of the intervention (Arce-Esquivel et al., 2017). Utilizing a 5-minute dry cupping intervention with a conventional cupping set, Li et al. (2017) reported both a prominent drop in deoxy-hemoglobin, -19.43 ± 3.83 μM during and -13.70 ± 4.98 μM post $p < 0.05$, and significant elevation in oxy-hemoglobin, 7.73 ± 2.56 μM during and 5.64 ± 2.68 μM post $p < 0.05$, surrounding the cupping site both during cupping and post-treatment as measured by near-infrared spectroscopy.

Running Economy and Running Performance

Running economy (RE) has been defined as the steady-state oxygen consumption (VO_2) at a given velocity, is measured in $L \cdot min^{-1}$ or $mL \cdot kg^{-1} \cdot min^{-1}$, and expressed as a percentage of VO_2 max (Barnes and Kilding, 2015; Conley and Krahenbuhl, 1980; Morgan et al., 1989; Saunders et al., 2004). Runners with good RE use less oxygen than runners with poor RE when running at the same steady-state velocity (Conley and Krahenbuhl, 1980). Although the definition of RE is simple in concept, running economy is a multifactorial measure which reflects the combined functioning of the metabolic, cardiopulmonary, biomechanical, and neuromuscular systems (Barnes and Kilding, 2015). More specifically, some reported contributing factors to RE include: time of day, gender, age, running environment (i.e. treadmill or over-ground), temperature, fatigue, training, body mass, body and segment mass distribution, running speed, stride length, stride frequency, ground reaction forces, and mechanical power (Morgan et al., 1989).

Running performance is limited by the potential to provide the energy required to cover a given distance via aerobic and anaerobic metabolism (Beneke and Hütler, 2005).

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Endurance training adaptations, such as increased morphology and functionality of skeletal muscle mitochondria, an increase in oxidative muscle capacity, and hematological changes, have been suggested to be factors that act to improve an athlete's running economy (Barnes and Kilding, 2014). Previous research has indicated that running economy is a significant determining factor in endurance running performance when comparing athletes who have a similar maximal VO_2 (Conley and Krahenbuhl, 1980; Ziogas et al., 2011). Conley and Krahenbuhl (1980) found that among a group, $N = 12$, of top finishers in a nationally prominent 10-km road race, who had similar relative VO_2 max values, 71.70 ± 2.80 mL$^.$kg$^{-1.}$min^{-1}, 65.4% of the variation observed in race performance on the 10-km race can be explained by variation in running economy. Running economy has also been used to explain similar running performances between athletes who have a variation in VO_2 max (Weston et al., 2000). Weston et al. (2000) compared eight Caucasian runners and eight African runners who, as two groups, performed similar race times over 10-km, 32.0 ± 2.5 min and 32.8 ± 2.8 min. The African runners had a mean relative VO_2 max that was 13% lower than the mean relative VO_2 max for the Caucasian runners, 61.9 ± 6.9 mL$^.$kg$^{-1.}$min^{-1} compared to 69.9 ± 5.4 mL$^.$kg$^{-1.}$min^{-1} with $p = 0.01$, and were 5% more economical, 47.3 ± 3.2 mL$^.$kg$^{-1.}$min^{-1} compared to 49.9 ± 2.4 ml/kg/min with $p < 0.05$, when running at a speed of 16.1 km$\cdot$h^{-1} (Weston et al., 2000).

Due in part to the relatively low metabolic demand of force production and transmission via non-contractile elements, it is likely that enhanced fascial contributions to lower body force production when running may improve running economy. Storen et al. (2008) reported that there was a significant correlation between half-squat one-repetition maximum and RE, $r^2 = 0.38$, $p < 0.01$. This suggests that muscle force production,

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particularly of the hip extensor muscle group, may have a role in the improvement of running economy.

VO_2 max, the most commonly used physiological parameter used to define cardiovascular fitness, shares some common determinants with RE; however, a high running economy is not necessarily related to a high VO_2 max. There is only a moderate correlation, $r = 0.33$, between running economy and VO_2 max in highly trained male runners with more than 85% of the variance in these parameters unexplained by their relationship (Shaw et al., 2015). A study of international level middle distance runners reported that running economy, $r = 0.9$ and $p < 0.01$, at race pace had a much stronger relationship with race performance than VO_2 max, $r = 0.05$ (Ferri et al., 2012). The dominant mechanism for the increase in VO_2 max with training is an increase in blood flow; and consequently, oxygen delivery (Bassett and Howley, 2000). If there is greater oxygen delivery due to an increase in blood flow, then the oxygen cost of running at a given sub-maximal velocity would be decreased; i.e. an increase in running economy.

Measurement of Running Economy

Protocols for evaluating running economy at submaximal running speeds standardly consist of having subjects run at a given submaximal speed and measuring the volume of oxygen consumed while running at that speed (Barnes and Kilding, 2015; Conley and Krahenbuhl, 1980; Daniels and Daniels, 1992; Foster and Lucia, 2007; Jones et al., 2003; Saunders et al., 2004; Weston et al., 2000). Daniels and Daniels (1992) established a protocol for testing running economy in 65 elite distance runners. Each test session included a series of submaximal, level-grade treadmill runs, followed by a constant-speed, increasing-grade treadmill run to determine VO_2 max (Daniels and Daniels, 1992). Each

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submaximal test lasted for six minutes with expired-air samples being collected for analysis the final two minutes. The series of tests performed by each male subject began at 268 m/min and followed in sequence to 290 m·min^{-1}, 310 m·min^{-1}, 330 m·min^{-1}, and 350 m·min^{-1}, with a few subjects completing additional stages of 370 m·min^{-1} and 390 m·min^{-1} (Daniels and Daniels, 1992). Five to 10 minutes following the final submaximal treadmill run, a VO$_2$ max test was performed using each subject's final treadmill velocity (not exceeding 350 m·min^{-1}) with the first two minutes being run at 0% grade and 1% grade being added to the treadmill with each subsequent minute beginning at three minutes (Daniels and Daniels, 1992). The test was terminated when the subject decided that he could not complete another minute, tests generally lasting six to seven minutes, and the highest VO$_2$ recorded was accepted as VO$_2$ max.

Weston et al. (2000) used a different protocol for testing running economy in 16 well-trained male subjects. Subjects performed a peak treadmill velocity test with concurrent measurement of VO$_2$, minute ventilation (VE), and respiratory exchange ratio (RER) (Weston et al., 2000). On a separate day, subjects warmed up at 14 km·h^{-1} on the treadmill for five minutes and then performed two submaximal work-loads, the first at 16.1 km·h^{-1} and the second at current 10-km race pace, with VO$_2$, VE, RER, and HR being measured continuously (Weston et al., 2000). Each workload was six minutes in duration with five minutes of recovery between workloads.

Hip Extensor Muscle Action and Running Performance

In running performance, marginal physiological and biomechanical gains may have a meaningful impact on race results. An increase in stride length and/or stride frequency is the biomechanical method through which running velocity is increased in humans (Dorn et

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al., 2012; Swanson and Caldwell, 2000). An increase in running velocity is accompanied by an increase in hip extension and force production of the primary hip extensor muscles, gluteus maximus, biceps femoris long head, semimembranosus, and semitendinosus, is positively associated with improved running performance (Blazevich and Jenkins, 1998; Dorn et al., 2012; Lieberman et al., 2006; Mero and Komi, 1994; Swanson and Caldwell, 2000; Zafeiridis et al., 2005).

Blazevich and Jenkins (1998) found that stepwise multiple regression analysis equations for predicting both 20-m maximum velocity run time and 20-m acceleration time may be calculated with an error of less than 0.05 seconds using only isokinetic and squat strength data. The researchers used a Cybex 6000 isokinetic dynamometer to evaluate concentric isokinetic hip flexor and extensor strength and a modified squat machine that only allowed the weighted bar to move in the vertical plane (Blazevich and Jenkins, 1998). Although this research was specific to sprinting, it highlights the importance of hip musculature in the action of running.

Lieberman et al. (2006) concluded, through electromyographic and kinematic analyses, that the gluteus maximus muscle plays an important role in the running capabilities of humans. Walking and running electromyographic data was collected with surface electrodes over the center of the gluteus maximus and stance phase kinematics were assessed using footswitches to determine timing of heel-strike, foot-flat, heel-off, and toe-off (Lieberman et al., 2006). Lieberman et al. (2006) reported that normalized activity of the gluteus maximus was greater during running than both level and uphill walking and that the magnitude of muscle activity at the biceps femoris and gluteus maximus and time of onset for both muscles were quite similar, $p = 0.705$ and $p = 0.961$, during the propulsive

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phase of running. These results indicate that gluteus maximus is critical to human running and functions in conjunction with the hamstring muscle group as a hip extensor.

Contractile and Noncontractile Force Enhancement

Enhancement of hip extension force production when running has historically been accomplished through traditional strength training of the gluteus maximus and hamstring muscle group in runners (Blazevich and Jenkins, 1998; Delecluse, 1997; Mero and Komi, 1994; Zafeiridis et al., 2005). Common methods of strength training analyzed in running performance research include hamstring curls, weighted squats, bounding and jumping exercises, and weighted sled-pulling, which are focused primarily on active force development and enhancement (Blazevich and Jenkins, 1998; Mero and Komi, 1994; Zafeiridis et al., 2005). Despite the effectiveness of these training methods, force production contributions from non-contractile elements, e.g. fascia, may provide evidence suggesting myofascial manipulation, such as myofascial decompression, through cupping techniques might be an alternative method of enhancing force production (Balcioglu et al., 2015; Drost and Hesselink, 2000; Kim et al., 2017; MacDonald et al., 2013; Stecco et al., 2013; Stecco et al., 2013; Tham et al., 2005; Williams et al., 2010; Williams et al., 2013).

Alterations of protein conformations, a proposed mechanism of cupping therapy, may have implications on non-contractile force production that are related to the structure of myofilament lattice structure. Williams et al. (2010) found that the rate at which cross-bridges bind and generate force decreases as lattice spacing grows and Williams et al. (2013) reported that the radial distance between the actin and myosin filaments, the filament lattice spacing, is responsible for between 20% and 50% of the change in force seen between sarcomere lengths of 1.4 and 3.4 µm. Additionally, Balcioglu et al. (2015)

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found that altering the relative abundance of fibronectin-binding integrins in cell-matrix adhesions affects the spatiotemporal organization of force transmission. These findings suggest that if protein conformations can be restructured through mechanical decompression therapy, like cupping, it may influence passive force production.

Quantification of Hip Extensor Force Production

Hip extension force production in athletes is typically quantified using a using a multimodal dynamometer (Boling et al., 2009; Negahban et al., 2013; San Juan et al., 2018; Taylor-Haas et al., 2014; Souza et al., 2009). Hip extensor torque was measured by Boling et al. (2009), Negahban et al. (2013), and Taylor-Haas et al. (2014) using the Biodex System 4 Pro isokinetic dynamometer and Biodex Software Package (Biodex Medical Systems, Inc., Shirley, NY, USA). The Biodex is considered to be the gold standard of dynamometry (Kollock et al., 2010). Boling et al. (2009) and Negahban et al. (2013) positioned subjects for strength assessment of the hip according to manufacturer guidelines and tested at an angular velocity of $60°·s^{-1}$ because greater concentric force is produced at slower isokinetic velocities.

Summary

The popularity of cupping therapy in high performance athletics is increasing and is being utilized by top level Olympic athletes. Despite the prevalence of its use, literature on the efficacy of cupping therapy is limited to primarily pain management and range of motion. Potential mechanisms of action of cupping therapy include increased skeletal muscle blood flow and the restructuring of fascial protein conformations through mechanotransduction. The enhancement of localized blood flow and manipulation of fascial protein structures has implications of improved running economy and hip extensor

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force production; both are key metrics to predict running performance. Therefore, the purpose of this study is to elucidate the effects of myofascial decompression through cupping therapy applied to key hip extensor muscle groups on running economy and hip extensor force production in well-trained runners.

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Appendix A: Journal Guidelines to Authors

Journal of Sports Science and Medicine

Journal guidelines to authors:

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    Exercise-induced oxidative stress and muscular performance in healthy women: role

    of vitamin E supplementation and endogenous estradiol. *European Journal of*

    Applied Physiology **84,** 141-147.

Journal of Bodywork and Movement Therapies

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    4: 21-27. https://doi.org/10.1054/jbmt.2000.0206

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Appendix B: Secondary Manuscript

EFFECT OF CUPPING THERAPY ON HIP EXTENSOR FORCE PRODUCTION

IN TRAINED ENDURANCE RUNNERS (DRAFT)

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ABSTRACT

Background

Literature on the efficacy of cupping therapy (CT) is limited. Potential mechanisms of action of cupping therapy include restructuring of fascial protein conformations through mechanotransduction, which has implications for improved hip extensor force production (HEFP). Hip extensor force production is positively associated with improved running performance.

Objective

The purpose of the study was to elucidate the effects of myofascial decompression through CT on HEFP in well-trained runners.

Methods

Five minutes of CT or placebo gel (PG) was applied to bilateral hip extensor muscles of 15 well-trained runners (n = 7 female, n = 8 male) after a 10-minute treadmill warm-up. Maximal HEFP was measured immediately post CT or PG using an isokinetic dynamometer. All subjects performed both conditions in randomized order separated by at least 1 week, but not more than 3 weeks. Maximal HEFP after CT and PG was compared independently using paired two-sample t-tests. Effect size was calculated using Cohen's d.

Results

Maximal HEFP was not significantly different between conditions (CT: 1.63 ± 0.47 Nm·kg^{-1}; 1.51 ± 0.40 Nm·kg^{-1}, $p = 0.18$, $d = 0.29$). Additionally, there was no significant difference in maximal hip extension force production for female subjects ($p = 0.17$, $d = 0.23$) or male subjects ($p = 0.40$, $d = 0.32$) between CT and PG.

Conclusion

Well-trained runners did not demonstrate statistically significant differences in hip

extensor force production after an acute single-session CT treatment compared to PG.

EFFECT OF CUPPING THERAPY ON HIP EXTENSOR FORCE PRODUCTION IN

TRAINED ENDURANCE RUNNERS

1. Background

Throughout the history of modern athletics, various techniques of massage have

been a common application of using compression therapy as therapeutic rehabilitation.

Compression therapy is typically administered as manual pressure applied to human tissue

through manual compressions and rhythmic percussions with the intention of increasing

blood and lymphatic flow (Nunes et al 2016). In recent years, the use of negative pressure

to elicit decompression of human tissue through mechanical manipulation has become more

common in athlete rehabilitation. The most prevalent method of decompression therapy

using negative pressure in athletics is cupping therapy (CT).

Despite the pervasive use, research on the effectiveness of CT is generally focused

on range of motion and pain management. Cupping therapy used with patients suffering

from anterior knee pain resulted in significant passive and active range of motion

improvements and an abatement of pain (Ullah et al 2007). Furthermore, CT improved

lower body range of motion in healthy subjects while also increasing electromyography

activation of the treated muscles (Kim et al 2017). Subjects with non-specific neck pain

reported that CT decreased pain during both motion and rest and a review of randomized

clinical trials of CT concluded that CT reduced pain intensity for subjects with both acute

and chronic pain (Cramer et al 2011; Lauche et al 2011; Cao et al 2014).

One potential mechanism of action of CT is the restructuring of fascial protein

conformations through mechanotransduction. The aponeurotic fascia that ensheath skeletal

muscle has two or three layers of parallel collagen fiber bundles with each layer separated

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by a thin layer of loose connective tissue that allows for sliding of the fascial layers over each other; additionally, the aponeurotic fascia is richly innervated, specifically in the superficial sublayer (Tesarz et al 2011). Epimysial fascia has a similar multilayered collagen structure to that of aponeurotic fascia, gives insertions to aponeurotic fascia, and has a fundamental role in the transmission of muscle generates force toward skeletal levers (Purslow 1989; Stecco et al 2013). Between the collagen fibers of epimysial fascia is a ground substance with an abundance of proteoglycans, namely hyaluronic acid, which allows the collagen fibers to slide with little friction (McCombe et al 2001). In physiological solutions and under tightly contained environments, like within fascial extracellular matrix, hyaluronic acid chains tend to bind to each other and entangle into complex rigid structures (Matteini et al 2009). Instead of being a lubricating component of the myofascial layers, hyaluronic acid becomes adhesive and the distribution of lines of force within the fascia become altered (Stecco et al 2013). Applying mechanical stress to the fascia, through techniques like cupping, increase the temperature of the tissues and reduces the viscosity of the hyaluronic acid polymers restoring it lubricative properties (Stecco et al 2013). Consequently, cupping therapy might stimulate improved fascial gliding, which would enhance cellular mechanotransduction (Stecco et al 2014).

Enhancement of hip extension force production when running has historically been accomplished through traditional strength training of the gluteus maximus and hamstring muscle group in runners (Blazevich and Jenkins, 1998; Delecluse, 1997; Mero and Komi, 1994; Zafeiridis et al., 2005). Common methods of strength training analyzed in running performance research include hamstring curls, weighted squats, bounding and jumping exercises, and weighted sled-pulling, which are focused primarily on active force

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development and enhancement (Blazevich and Jenkins, 1998; Mero and Komi, 1994; Zafeiridis et al., 2005). Despite the effectiveness of these training methods, force production contributions from non-contractile elements, e.g. fascia, may provide evidence suggesting myofascial manipulation, such as myofascial decompression, through cupping techniques might be an alternative method of enhancing force production (Balcioglu et al., 2015; Drost and Hesselink, 2000; Kim et al., 2017; MacDonald et al., 2013; Stecco et al., 2013; Stecco et al., 2013; Tham et al., 2005; Williams et al., 2010; Williams et al., 2013).

In running performance, marginal physiological and biomechanical gains may have a meaningful impact on race results. An increase in stride length and/or stride frequency is the biomechanical method through which running velocity is increased in humans (Dorn et al., 2012; Swanson and Caldwell, 2000). The manipulation of fascial protein structures has implications of increased hip extensor force production. Therefore, the purpose of this study is to elucidate the effects of myofascial decompression through CT applied to key hip extensor muscle groups on hip extensor force production in well-trained runners. We hypothesized that an acute single treatment of CT would increase hip extensor force production in well-trained runners.

2. <u>Methods</u>

2.1. subjects

Fifteen well-trained competitive distance runners were recruited from the greater Bellingham and Western Washington University running community; demographics listed in Table 1. Subjects were excluded from participation in this study if they had current injuries that limited their running performance or ability to perform a maximal hip extension against weighted resistance. Each individual subject was instructed to wear their

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normal running footwear and comfortable attire for running indoors on a treadmill and performing a maximal hip extension.

2.2. outcome measures

Concentric torque of the hip extensors, normalized to subject body mass and expressed as Newton meters per kilogram ($Nm \cdot kg^{-1}$), was assessed using a BIODEX System 4 Pro (Biodex Medical Systems, Inc., Shirley, NY, USA).

2.3. experimental procedure

The study was a two-group crossover design in which subjects were randomly assigned on their first session, via flip of a coin, whether they completed the maximal hip extensor strength test with CT or placebo gel (PG). The alternative condition was performed on the second session. Each subject completed two sessions with a minimum of a one-week washout period, but not more than three weeks, between conditions. Subjects were instructed to avoid intense physical activity in the 48 hours prior to testing sessions and to maintain their normal training routines between training sessions. For the placebo condition, a salt-free, chloride-free, water-soluble electrode gel (Spectra 360 Electrode Gel, Parker Laboratories Inc., Fairfield, NJ, USA) was applied to the subject. Subjects were told the PG had been shown to enhance force production. For the test protocol, subjects performed a 10-minute jogging warm up on the treadmill (T645 Treadmill, SportsArt Fitness, Mukilteo, WA, USA) at a self-selected velocity followed by either CT or PG. Subjects then completed the maximal hip extensor strength test.

An FDA approved medical grade professional Chinese cupping therapy set (Care me, Inc., Norcross, GA, USA) was used to perform CT on the primary hip extensor muscles of the subjects. A clean cupping set was used for each subject. The application sites for

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each subject were the muscle belly of the gluteus maximus, biceps femoris long head, semimembranosus, and semitendinosus muscles on both the right and left legs (Hamner et al 2010). A vacuum seal to apply negative pressure was created in the cups using a hand pump with pumps applied until 1.6 cm of skin was elevated into each cup (Markowski et al 2014). Cups were left on subjects for five minutes (Cramer et al 2011; Shixi & Yu 2006). Cups were removed from subjects in the same order that they were applied. Cupping therapy was performed by a certified athletic trainer experienced in the technique. The maximal hip extensor torque test protocol was completed immediately following cupping. The PG trial followed the same protocol.

Prior to each test session, a calibration verification of the BIODEX System 4 Pro was performed. The maximal hip extensor torque test was performed with subjects positioned consistent to the hip extension test position used by San Juan et al. (2018). Subjects were asked to stand in front of the Biodex chair while flexing their trunk to 45° with respect to the ground and with the tested hip flexed to 30°. The dynamometer lever arm axis of rotation was aligned with the greater trochanter of the femur on the test leg. The thigh of the tested leg was secured distally above the medial and lateral epicondyle using the thigh attachment that came with the dynamometer. The subject's contralateral foot was kept flat on the ground. The subject was instructed to exert maximal force against the dynamometer in the direction of hip extension while keeping the knee flexed at approximately 90°. The dynamometer was programmed to move in an arc of approximately 30 degrees, from approximately 30° of hip flexion to approximately neutral (Boling et al 2009; Taylor-Haas et al 2014). Each subject was allowed three submaximal practice repetitions prior to testing. Subjects performed three maximal test repetitions of hip

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extension at a testing velocity of $60°·s^{-1}$ (Boling et al 2009; Negahban et al 2013). A rest interval of 30 seconds was given between submaximal practice repetitions and maximal test repetitions.

2.4. data analysis

Maximal hip extensor torque was reported as the peak torque achieved from the three test trials. Paired two-sample t-tests were used to determine differences in maximal hip extension torque. Statistical significance was established at $p < 0.05$. Effect size was calculated, and Cohen's d was used to indicate the standardized difference between means. Data analysis was performed using Microsoft Excel (Microsoft Corporation, Redmond, WA).

3.   Results

The mean maximal hip extension force production values with CT and PG are summarized in Table 2. There was no significant difference in maximal hip extension force production ($p = 0.18$, $d = 0.29$) between CT and PG. Additionally, there was no significant difference in maximal hip extension force production for female subjects ($p = 0.17$, $d = 0.23$) or male subjects ($p = 0.40$, $d = 0.32$) between CT and PG.

4.   Discussion

The main finding of this study was that there was no difference in maximal hip extension torque between CT and PG in well-trained endurance runners. Thus, our initial hypothesis that an acute single treatment of CT would increase maximal hip extension torque in well-trained runners was not supported.

An increase in running velocity is accompanied by an increase in hip extension and force production of the primary hip extensor muscles, gluteus maximus, biceps femoris

long head, semimembranosus, and semitendinosus, is positively associated with improved running performance (Blazevich and Jenkins, 1998; Dorn et al., 2012; Lieberman et al., 2006; Mero and Komi, 1994; Swanson and Caldwell, 2000; Zafeiridis et al., 2005). Although there were no statistically significant differences and small effect sizes, CT may have an influence on skeletal muscle force enhancement under different protocols than the one used in this study.

Some limitations to the present study must be considered. The study used a homogenous sample population; however, the sample size was relatively small. The measurement of maximal hip extension torque is a strength measure and there was no control for strength training that subjects performed in addition to their running volume. Additionally, there is not a standardized CT protocol currently that is utilized by practitioners and the appropriate magnitude of negative pressure, number of treatment sessions, or length of time cups are applied to elicit a physiological response has not yet been determined.

The p-values and small effect sizes in this study imply that future research on CT and skeletal muscle force production should be conducted (Rhea 2004). The restructuring of fascial protein conformation through mechanotransduction does not likely occur within several minutes following a single session of CT, so increasing the number of sessions or the time between CT and muscle strength testing in future studies may result in different findings. Additionally, future research quantifying potential muscular and fascial structure and arrangement changes in response to CT may provide valuable insight into the efficacy of CT and decompression therapy in therapeutic rehabilitation.

5.	<u>Conclusion</u>

Well-trained runners did not demonstrate statistically significant differences in hip extensor torque after an acute single-session CT treatment compared to PG. The p-values and small effect in this study has implications for future research.

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Table 1. Subject Demographics

Sex	Age (yrs)	Height (m)	Mass (kg)	10-km race time (min)
Female (n = 7)	29.3 ± 2.1	1.68 ± 0.06	60.2 ± 3.4	41.4 ± 4.4
Male (n = 8)	27.5 ± 6.2	1.77 ± 0.04	69.1 ± 4.0	33.5 ± 1.2

Table 2. Mean ± SD maximal hip extension force production.

Sex	CT (Nm·kg^{-1})	PG (Nm·kg^{-1})	P-value	Cohen's d
Female	1.63 ± 0.46	1.53 ± 0.45	0.17	0.23
Male	1.63 ± 0.50	1.49 ± 0.38	0.40	0.32
Combined	1.63 ± 0.47	1.51 ± 0.40	0.18	0.29

Subject Demographics:

Sex	Height (m)	Mass (kg)	Age (y)	10-km Time (min)
F	1.74	64.0	31.0	35.00
F	1.70	57.7	27.0	46.60
F	1.70	60.5	31.0	45.05
F	1.74	63.6	28.0	45.00
F	1.58	62.7	27.0	42.00
F	1.68	55.9	29.0	39.35
F	1.65	56.8	32.0	37.00
M	1.70	61.6	33.0	33.83
M	1.79	74.1	36.0	35.00
M	1.84	69.1	19.0	34.00
M	1.78	69.0	24.0	33.00
M	1.78	74.1	24.0	32.00
M	1.78	67.7	22.0	32.00
M	1.68	69.1	28.0	35.00
M	1.80	67.7	34.0	33.13
Average	1.73	64.9	28.3	37.20
Standard Deviation	0.07	5.8	4.7	5.09

Running economy for standard treadmill velocity:

Subject	CT (%VO$_2$max)	PG (%VO$_2$max)
F1	72.9	75.5
F2	77.5	81.4
F3	93.9	93.8
M4	76.4	77.3
M5	67.4	68.4
F6	100.2	95.1
F7	89.2	86.5
M8	73.9	76.5
F10	84	85.5
M11	68.5	66.9
F12	73.5	74.9
M13	71.5	70.8
M14	61.8	58.3
M15	68	63.7
M16	74.3	74.6
Average	76.9	76.6
Standard Deviation	10.6	10.5

Running economy for 10-km race pace treadmill velocity:

Subject	CT (%VO_2max)	PG (%VO2max)
F1	92.3	91.1
F2	70.9	72.6
F3	88	88.7
M4	90.4	90.4
M5	78.4	77.4
F6	94.4	91.7
F7	91.4	90.4
M8	80.1	81.2
F10	91.8	91.9
M11	77.1	76.2
F12	87.4	87.7
M13	83	81.7
M14	77.1	74.6
M15	78.5	79.1
M16	81.7	80.7
Average	84.2	83.7
Standard Deviation	7.1	6.9

Maximal oxygen consumption:

Subject	CT (ml·kg·min⁻¹)	PG (ml·kg·min⁻¹)
F1	59.8	59.6
F2	54.1	57.4
F3	54.7	51.2
M4	78	76.2
M5	72.4	76.6
F6	51.6	47.2
F7	57.8	55
M8	63.7	63.9
F10	62.1	60.7
M11	70.4	69.8
F12	58	59.4
M13	77	75.4
M14	78.4	81.5
M15	67.8	71.9
M16	70.4	69.8
Average	65.1	65.0
Standard Deviation	9.1	10.3

Steady-state respiratory exchange ratio during standard treadmill velocity:

Subject	CT	PG
F1	0.88	0.86
F2	0.935	0.89
F3	0.986	0.97
M4	0.935	0.87
M5	0.885	0.89
F6	1.051	0.99
F7	0.9	0.89
M8	0.919	0.9
F10	0.979	0.96
M11	0.922	0.9
F12	0.839	0.89
M13	0.905	0.9
M14	0.856	0.84
M15	0.856	0.86
M16	0.884	0.89
Average	0.915	0.899
Standard Deviation	0.056	0.043

Steady-state respiratory exchange ratio during 10-km race pace treadmill velocity:

Subject	CT	PG
F1	1.003	0.97
F2	0.918	0.89
F3	0.958	0.94
M4	0.956	0.89
M5	0.894	0.89
F6	1.039	0.95
F7	0.899	0.9
M8	0.923	0.9
F10	1.015	0.99
M11	0.941	0.93
F12	0.915	0.95
M13	0.927	0.94
M14	0.934	0.89
M15	0.916	0.92
M16	0.896	0.89
Average	0.942	0.922
Standard Deviation	0.045	0.033

Steady-state CO_2 expiration during standard treadmill velocity:

Subject	CT ($L \cdot min^{-1}$)	PG ($L \cdot min^{-1}$)
F1	2.45	2.48
F2	2.4	2.41
F3	2.86	2.8
M4	3.68	3.45
M5	2.81	2.88
F6	3.31	2.83
F7	2.77	2.66
M8	3.21	3.27
F10	2.79	2.8
M11	3.05	2.91
F12	2.08	2.24
M13	3.35	3.3
M14	3.19	2.94
M15	2.83	2.66
M16	3.17	3.18
Average	2.93	2.85
Standard Deviation	0.41	0.34

Steady-state CO_2 expiration during 10-km race pace treadmill velocity:

Subject	CT (L·min^{-1})	PG (L·min^{-1})
F1	3.53	3.36
F2	2.15	2.14
F3	2.61	2.58
M4	4.46	4.15
M5	3.31	3.24
F6	2.94	2.62
F7	2.84	2.79
M8	3.5	3.47
F10	3.16	3.08
M11	3.5	3.41
F12	2.7	2.8
M13	4.01	3.99
M14	4.36	4.02
M15	3.5	3.55
M16	3.53	3.47
Average	3.34	3.24
Standard Deviation	0.64	0.58

Hip extension torque (Combined):

Subject	CT ($Nm \cdot kg^{-1}$)	PG ($Nm \cdot kg^{-1}$)
F1	1.598	1.642
F2	1.676	1.744
F3	1.499	1.256
F6	0.961	0.987
F7	1.764	1.349
F10	2.494	2.376
F12	1.408	1.324
M4	2.623	1.476
M5	1.234	1.584
M8	1.495	1.382
M11	2.197	2.282
M14	1.467	1.595
M13	1.348	0.964
M15	1.328	1.347
M16	1.373	1.285
Average	1.631	1.506
Standard Deviation	0.465	0.398

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Hip extension torque (Female):

Subject	CT ($Nm \cdot kg^{-1}$)	PG ($Nm \cdot kg^{-1}$)
1	1.598	1.642
2	1.676	1.744
3	1.499	1.256
6	0.961	0.987
7	1.764	1.349
10	2.494	2.376
12	1.408	1.324
Average	1.629	1.525
standard Deviation	0.462	0.451

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Hip extension torque (Males):

Subject	CT ($Nm \cdot kg^{-1}$)	PG ($Nm \cdot kg^{-1}$)
4	2.623	1.476
5	1.234	1.584
8	1.495	1.382
11	2.197	2.282
14	1.467	1.595
13	1.348	0.964
15	1.328	1.347
16	1.373	1.285
Average	1.633	1.489
Standard Deviation	0.499	0.378

Appendix D: Statistical Analysis

Standard treadmill velocity running economy:

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.253333
	CT	*PG*		Pooled SD	10.55244
Mean	76.86666667	76.61333333		d	**0.024007**
Variance	112.4780952	110.2298095			
Observations	15	15			
Pearson Correlation	0.967663093				
Hypothesized Mean Difference	0				
df	14				
t Stat	0.365333953				
P(T<=t) one-tail	0.360161278				
t Critical one-tail	1.761310136				
P(T<=t) two-tail	**0.720322556**				
t Critical two-tail	2.144786688				
CT		*PG*			
Mean	76.86666667	Mean	76.61333333		
Standard Error	2.738346158	Standard Error	2.710840085		
Median	73.9	Median	75.5		
Mode	#N/A	Mode	#N/A		
Standard Deviation	10.60556907	Standard Deviation	10.4990385		
Sample Variance	112.4780952	Sample Variance	110.2298095		
Kurtosis	0.385264104	Kurtosis	-0.375282594		
Skewness	0.969707287	Skewness	0.227601135		
Range	38.4	Range	36.8		
Minimum	61.8	Minimum	58.3		
Maximum	100.2	Maximum	95.1		
Sum	1153	Sum	1149.2		
Count	15	Count	15		

10-km race pace treadmill velocity running economy:

t-Test: Paired Two Sample for Means					Effect Size - Cohen's d	
					Mean Difference	0.473333333
	CT	PG			Pooled SD	6.994494434
Mean	84.16666667	83.69333333			d	0.067672273
Variance	50.76380952	47.08209524				
Observations	15	15				
Pearson Correlation	0.984513351					
Hypothesized Mean Difference	0					
df	14					
t Stat	1.456801635					
P(T<=t) one-tail	0.083616266					
t Critical one-tail	1.761310136					
P(T<=t) two-tail	0.167232531					
t Critical two-tail	2.144786688					
CT		PG				
Mean	84.16666667	Mean	83.69333333			
Standard Error	1.839634194	Standard Error	1.771667675			
Median	83	Median	81.7			
Mode	77.1	Mode	90.4			
Standard Deviation	7.124872597	Standard Deviation	6.861639399			
Sample Variance	50.76380952	Sample Variance	47.08209524			
Kurtosis	-1.137098791	Kurtosis	-1.594431411			
Skewness	-0.167058217	Skewness	-0.170616913			
Range	23.5	Range	19.3			
Minimum	70.9	Minimum	72.6			
Maximum	94.4	Maximum	91.9			
Sum	1262.5	Sum	1255.4			
Count	15	Count	15			

Maximal oxygen consumption:

t-Test: Paired Two Sample for Means					Effect Size - Cohen's d	
	CT	*PG*			Mean Difference	0.04
Mean	65.08	65.04			Pooled SD	9.701178132
Variance	82.55885714	105.6668571			d	**0.004123211**
Observations	15	15				
Pearson Correlation	0.967512476					
Hypothesized Mean Difference	0					
df	14					
t Stat	0.056596578					
P(T<=t) one-tail	0.47783319					
t Critical one-tail	1.761310136					
P(T<=t) two-tail	**0.95566638**					
t Critical two-tail	2.144786688					

CT		*PG*		
Mean	65.08	Mean	65.04	
Standard Error	2.34604429	Standard Error	2.654139624	
Median	63.7	Median	63.9	
Mode	70.4	Mode	69.8	
Standard Deviation	9.086190464	Standard Deviation	10.27943856	
Sample Variance	82.55885714	Sample Variance	105.6668571	
Kurtosis	-1.360486278	Kurtosis	-1.059454926	
Skewness	0.122888309	Skewness	-0.088452657	
Range	26.8	Range	34.3	
Minimum	51.6	Minimum	47.2	
Maximum	78.4	Maximum	81.5	
Sum	976.2	Sum	975.6	
Count	15	Count	15	

Steady-state respiratory exchange ratio during standard treadmill velocity:

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.016066667
	CT	*PG*		Pooled SD	0.050086116
Mean	0.915466667	0.8994		d	**0.320780844**
Variance	0.003188838	0.0018284			
Observations	15	15			
Pearson Correlation	0.877097252				
Hypothesized Mean Difference	0				
df	14				
t Stat	2.225913303				
P(T<=t) one-tail	0.021479368				
t Critical one-tail	1.761310136				
P(T<=t) two-tail	**0.042958736**				
t Critical two-tail	2.144786688				
CT		*PG*			
Mean	0.915466667	Mean	0.8994		
Standard Error	0.014580439	Standard Error	0.011040531		
Median	0.905	Median	0.893		
Mode	0.935	Mode	#N/A		
Standard Deviation	0.056469798	Standard Deviation	0.042759794		
Sample Variance	0.003188838	Sample Variance	0.0018284		
Kurtosis	1.001505363	Kurtosis	0.415896679		
Skewness	0.977460607	Skewness	0.932789502		
Range	0.212	Range	0.155		
Minimum	0.839	Minimum	0.835		
Maximum	1.051	Maximum	0.99		
Sum	13.732	Sum	13.491		
Count	15	Count	15		

Steady-state respiratory exchange ratio during 10-km race pace treadmill velocity:

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.0202
	CT	PG		Pooled SD	0.039096888
Mean	0.942266667	0.922066667		d	0.51666516
Variance	0.001986495	0.001070638			
Observations	15	15			
Pearson Correlation	0.742013561				
Hypothesized Mean Difference	0				
df	14				
t Stat	2.618177352				
P(T<=t) one-tail	0.010123873				
t Critical one-tail	1.761310136				
P(T<=t) two-tail	0.020247747				
t Critical two-tail	2.144786688				
	CT		PG		
Mean	0.942266667	Mean	0.922066667		
Standard Error	0.011507954	Standard Error	0.008448424		
Median	0.927	Median	0.922		
Mode	#N/A	Mode	0.888		
Standard Deviation	0.044570116	Standard Deviation	0.032720607		
Sample Variance	0.001986495	Sample Variance	0.001070638		
Kurtosis	0.250079012	Kurtosis	-0.701815183		
Skewness	1.083528722	Skewness	0.607017399		
Range	0.145	Range	0.101		
Minimum	0.894	Minimum	0.888		
Maximum	1.039	Maximum	0.989		
Sum	14.134	Sum	13.831		
Count	15	Count	15		

Steady-state CO_2 expiration during standard treadmill velocity:

t-Test: Paired Two Sample for Means			
	CT	*PG*	
Mean	2.93	2.854	
Variance	0.172114286	0.117168571	
Observations	15	15	
Pearson Correlation	0.927711737		
Hypothesized Mean Difference	0		
df	14		
t Stat	1.832624201		
P(T<=t) one-tail	0.044104723		
t Critical one-tail	1.761310136		
P(T<=t) two-tail	**0.088209447**		
t Critical two-tail	2.144786688		
CT		*PG*	
Mean	2.93	Mean	2.854
Standard Error	0.107118092	Standard Erro	0.088381
Median	2.86	Median	2.83
Mode	#N/A	Mode	2.8
Standard Deviation	0.414866588	Standard Dev	0.342299
Sample Variance	0.172114286	Sample Varia	0.117169
Kurtosis	0.081247197	Kurtosis	-0.4837
Skewness	-0.32040428	Skewness	0.044475
Range	1.6	Range	1.21
Minimum	2.08	Minimum	2.24
Maximum	3.68	Maximum	3.45
Sum	43.95	Sum	42.81
Count	15	Count	15
Effect Size - Cohen's d			
Mean Difference	0.076		
Pooled SD	0.380317537		
d	**0.199833015**		

Steady-state CO_2 expiration during 10-km race pace treadmill velocity:

t-Test: Paired Two Sample for Means			
	CT	*PG*	
Mean	3.34	3.244666667	
Variance	0.406757143	0.335398095	
Observations	15	15	
Pearson Correlation	0.980774278		
Hypothesized Mean Difference	0		
df	14		
t Stat	2.779901412		
P(T<=t) one-tail	0.007376122		
t Critical one-tail	1.761310136		
P(T<=t) two-tail	**0.014752244**		
t Critical two-tail	2.144786688		
CT		*PG*	
Mean	3.34	Mean	3.244667
Standard Error	0.164672836	Standard Erro	0.149532
Median	3.5	Median	3.36
Mode	3.5	Mode	3.47
Standard Deviation	0.637775151	Standard Dev	0.579136
Sample Variance	0.406757143	Sample Varia	0.335398
Kurtosis	-0.17713948	Kurtosis	-0.56935
Skewness	0.083150326	Skewness	-0.16641
Range	2.31	Range	2.01
Minimum	2.15	Minimum	2.14
Maximum	4.46	Maximum	4.15
Sum	50.1	Sum	48.67
Count	15	Count	15
Effect Size - Cohen's d			
Mean Difference	0.095333333		
Pooled SD	0.609161406		
d	**0.156499299**		

Hip extension torque (Combined):

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.1248
	CT	*PG*		Pooled SD	0.432993088
Mean	1.631	1.5062		d	**0.28822631**
Variance	0.216250571	0.158715457			
Observations	15	15			
Pearson Correlation	0.692763359				
Hypothesized Mean Difference	0				
df	14				
t Stat	1.405418136				
P(T<=t) one-tail	0.09084938				
t Critical one-tail	1.761310136				
P(T<=t) two-tail	**0.18169876**				
t Critical two-tail	2.144786688				

CT		*PG*	
Mean	1.631	Mean	1.5062
Standard Error	0.120069583	Standard Error	0.102864136
Median	1.495	Median	1.382
Mode	#N/A	Mode	#N/A
Standard Deviation	0.465027495	Standard Deviation	0.398391086
Sample Variance	0.216250571	Sample Variance	0.158715457
Kurtosis	0.637898258	Kurtosis	1.056608943
Skewness	1.107520558	Skewness	1.043202371
Range	1.662	Range	1.412
Minimum	0.961	Minimum	0.964
Maximum	2.623	Maximum	2.376
Sum	24.465	Sum	22.593
Count	15	Count	15

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Hip extension torque (Females):

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.103142857
	CT	*PG*		Pooled SD	0.456491642
Mean	1.628571429	1.525428571		d	**0.225946869**
Variance	0.213597286	0.203171952			
Observations	7	7			
Pearson Correlation	0.926299725				
Hypothesized Mean Difference	0				
df	6				
t Stat	1.554008745				
P(T<=t) one-tail	0.08558826				
t Critical one-tail	1.943180281				
P(T<=t) two-tail	**0.17117652**				
t Critical two-tail	2.446911851				

CT		*PG*	
Mean	1.628571429	Mean	1.525428571
Standard Error	0.174682277	Standard Error	0.170365973
Median	1.598	Median	1.349
Mode	#N/A	Mode	#N/A
Standard Deviation	0.462165864	Standard Deviation	0.450745995
Sample Variance	0.213597286	Sample Variance	0.203171952
Kurtosis	2.399777908	Kurtosis	1.584460624
Skewness	0.804417005	Skewness	1.116166714
Range	1.533	Range	1.389
Minimum	0.961	Minimum	0.987
Maximum	2.494	Maximum	2.376
Sum	11.4	Sum	10.678
Count	7	Count	7

Hip extension torque (Males):

t-Test: Paired Two Sample for Means				Effect Size - Cohen's d	
				Mean Difference	0.14375
	CT	PG		Pooled SD	0.442717158
Mean	1.633125	1.489375		d	**0.32469941**
Variance	0.249406696	0.142590268			
Observations	8	8			
Pearson Correlation	0.484536057				
Hypothesized Mean Difference	0				
df	7				
t Stat	0.888837283				
P(T<=t) one-tail	0.201799409				
t Critical one-tail	1.894578605				
P(T<=t) two-tail	**0.403598819**				
t Critical two-tail	2.364624252				

CT		PG	
Mean	1.633125	Mean	1.489375
Standard Error	0.176566806	Standard Error	0.133505743
Median	1.42	Median	1.429
Mode	#N/A	Mode	#N/A
Standard Deviation	0.499406344	Standard Deviation	0.377611266
Sample Variance	0.249406696	Sample Variance	0.142590268
Kurtosis	1.204721854	Kurtosis	3.082634498
Skewness	1.543363805	Skewness	1.21098126
Range	1.389	Range	1.318
Minimum	1.234	Minimum	0.964
Maximum	2.623	Maximum	2.282
Sum	13.065	Sum	11.915
Count	8	Count	8