The Effects of Cooling Core Body Temperature on Overall Strength Gains and Post-Exercise Recovery

Adam Tenforde

Heat is the byproduct of metabolic reactions, with nearly 75% of energy created released as heat. This paper evaluates two studies investigating the effects of cooling core body temperature on overall strength gains and post-exercise recovery. The first study evaluates the effectiveness of cooling before, during and post-exercise on performance and recovery. Results show that lowering core body temperature results in lower overall relative oxygen consumption during prolonged exercise. The second study evaluated the effects of intermittent cooling between bouts of maximal exercise and different conditions of post-exercise conditions of cooling. Results show that a majority of subjects improve on treatment, supporting the theory that temperature is a limiting factor of exercise though responses to cooling treatments are individualistic.

Introduction

Previous studies have demonstrated that elevated temperature can be a source of fatigue. During exercise metabolism, core body temperature, metabolite buildup, and muscle efficiency are significantly affected by elevated core body temperature. Circulation issues related to heat exist in two forms: dehydration and blood redistribution. One main cause of dehydration is loss of fluids through increased sweating in response to demands for greater cooling. Dehydration decreases blood and plasma volume, rate of sweating, muscle strength and capacity, as well as liver glycogen. In addition as the body temperature rises, more blood is shunted to peripheral tissues to aid in cooling and less oxygenated blood is available for muscle tissues, the lungs, heart and other internal organs and tissues. Therefore, there is a direct relationship between exercise performance and esophageal temperature during exercise [12]. Therefore rises in core body temperature will limit performance in cases of prolonged or hypothermic exercise conditions.

There is also a centrally mediated component of fatigue related to heat exposure. A study by Nybo et al. demonstrated that heat-related fatigue was related to changes in cerebral activity [13]. These results demonstrate that there is altered brain activity associated with hyperthermia-induced fatigue, although the actual mechanism was not found [13]. Therefore, it appears that there exists a threshold core temperature of $40°C$ that limits individuals from performing exercise controlled by the central nervous system.

The balance of substrate use may also change as a result of elevated core body temperature, resulting in a decrement of exercise performance. A study by Jentjens et al. demonstrated that more muscle glycogen and less ingested carbohydrates are utilized during submaximal exercise in a heated environment compared to a cooler environment [7]. Prolonged exercise with a corresponding increase in core temperature could lead to substrate-related fatigue.

Metabolic byproducts can have limiting impacts on performance. Elevated temperature compromises the release and uptake of Ca^{2+} in muscles outside of optimal temperatures [1, 8, 9]. Since Ca^{2+} is important in muscle

contractions, changes in Ca^{2+} uptake can inhibit normal muscle function.

In addition, inorganic phosphate (P_i) can have a negative impact on muscle function, as P_i interferes with Ca^{2+} [1, 10]. There is a greater level of metabolites as a result of circulation issues at higher temperatures. Oxygen supply to exercising muscles can become compromised at elevated temperatures, impacting the release of Ca^{2+} as well as changing the composition of metabolites present during physical exertion of muscle groups [6]. Therefore, one possible mechanism of fatigue at the muscle level could be elevated temperatures impacting metabolite buildup and the function of muscles [10].

Purpose and Hypothesis

The purpose of this study was to determine the benefits of cooling on muscle performance. The hypothesis on which this project was based is that one factor producing muscle fatigue is a rise in temperature of the muscles and core body region. Therefore, intermittent cooling during exercise should lead to a greater increase in maximum power output. With small time intervals between sets of maximum exertion, use of this thermoregulation device should result in significantly lower core temperature during exercise. Lowering core temperature of subjects before exercise should increase performance by limiting central fatigue [13]. In addition, application of cooling after exercise should impact recovery rate by reduction in metabolites related to heat. Dehydration effects and substrate utilization can be optimized at lower body temperatures. The factors monitored during exercise that would indicate improvement in performance due to cooling include metabolic gases, lower lactate concentrations and heart rate, and rate of perceived exertion (RPE).

Materials and Methods

Sub-maximal Exertion Running Study

Seven endurance-based male running subjects in aerobic base training phase were recruited to participate in this study. Two forty-minute bout of sub-maximal running trials were performed at the Sports Medicine Institute International Physiology Laboratory. Subjects were counterbalanced into two groups.

Initially, subjects performed a VO_2 max test designed by Dr. Jack Daniels. Baselines of esophageal (T_{es}) and tympanic (T_{tym}) temperature as well as heart rate measurements were taken prior to beginning exercise.

Subjects were given twenty minutes of active warm-up followed by stretching. During the pre-cooling trial, subjects stretched in a room kept at hyperthermic temperatures (31.3 $\pm$0.7°C). The application of heat lowered T_{es} by an average of 1.2 $\pm$0.4°C.

During each trial subjects performed a sub-maximal exercise set at 82-87% of their VO_{2max}. Data collected included VO_2 data, respiratory exchange ratio (RER), and fingertip blood lactate samples (YSI 9000 lactate analyzer) at 10-minute intervals during the 40-minute exercise bout. These measurements were additionally taken 10-15 minutes immediately following the conclusion of the sub-maximal exertion. Heart rate and RPE values were obtained at five-minute intervals during exercise.

Temperature and Heart Rate Measurements

The subject's esophageal and ear canal temperatures were sampled at 1 sec intervals using thermocouple temperature probes positioned at the approximate level of the heart. The tip of the ear canal temperature probes were self-inserted and fastened to the outer ear. Both temperature measurements were taken using a temperature data recorder recording at one-second intervals.

The heart rate measurements were obtained by use of the Polar heart rate monitor. The heart rate device used a chest strap that positioned it directly above the xyphoid process. Heart rate values were recorded within five seconds immediately preceding each measurement interval.

Cooling Mechanism

The Rapid Thermal Exchange device designed by H. Craig Heller and DA Grahn at Stanford University and produced by AVACORE (RTX Rapid Thermal Exchange System) has been shown to effectively manipulate core temperature [4, 15]. The body core releases excess heat through areas consisting of arteriovenous anastomoses (AVA's) and the subcutaneous venous plexuses found on hairless regions of the body consisting of the hands, feet, face and ears [4]. These specialized vessels constrict and dilate as a response to higher and lower core temperatures, respectively [2, 4, 5, 11, 15]. The result is a change in the venous temperature of the blood returning to the heart, impacting the core temperature. This device creates a thermal gradient using a water-perfused matrix underlying a metal plate that is designed for the curvature of the palm of the hand. The temperature of the water bath can be manipulated to create the desired thermal gradient. In addition, the metal plate is enclosed in a plastic cover with a pressure-cuff designed to create a vacuum around the wrist of the hand set at 15 to 25 in H_2O below atmospheric pressure, allowing a greater volume of blood to enter the hand for heat exchange and, in addition, may prevent constriction of the AVA's to maximize heat exchange with the thermal gradient underlying the palm of the subjects' hands.

Post-Cooling Procedure

At the conclusion of the forty-minute exercise bout, subjects were randomly assigned to receive cooling during one of the two post exercise periods, using the AVACore device set to 22°C. During the recovery period, temperature and VO_2 data was continuously collected with a blood lactate sample at conclusion of exercise.

Data Analysis

The data was collected from each subject and averages for each time period as well as for the entire test were created. Comparisons between each time period between subjects were conducted using a one-sided paired t-test with the significance level set at $p < 0.05$.

Cyclist Study

Ten healthy, well-trained experienced male cyclists in the racing phase

of their season were recruited to participate in this study. On two separate occasions, cyclists performed eight sprints of 0.2 miles in length at the Sport Medicine Institute International Physiology Laboratory at 23°C. Subjects were counterbalanced to perform two sessions of eight time trials to exhaustion with three minutes of passive recovery between sprints. During the intermittent cooling trial, subjects received cooling using AVACore Rapid Thermal Exchange (set at 26°C) during the duration of the three-minute recovery period.

Before beginning exercise, esophageal (T_{es}) and tympanic (T_{tym}) temperature and heart rate measurements were recorded. Subjects mounted the cycle ergometer and were given a ten-minute warm-up prior to beginning each trial. Each cyclist used his own bicycle fastened in a stationary position equilibrated to ensure accurate measurements.

Computrainer software (RacerMate CompuTrainer 3D Software) provided readings of the total time to complete each trial, as well as data on the maximum wattage, average wattage, maximum speed and average speed. Data collected included VO_2 data, RER, respiratory metabolic information as well as lactate measurements taken throughout the duration of the eight sprints. Measurements were continued for ten to fifteen minutes during passive recovery immediately following the eighth sprint to monitor recovery.

Results

Sub-maximal Exertion Running Study

Six of the seven subjects were used in the analysis of the data. One subject was excluded for heat illness during both trials. Temperature data is presented in Figure 1. Subjects experienced an initial drop in the starting temperature of the pre-heat experiment compared to control conditions (37.00°C versus 37.72°C, $p < 0.05$) although the average temperature reached at the conclusion of trials was similar (38.44°C to 38.56°C).

The results of each performance measure are listed in Table 1. Although the respiratory exchange ratios (RER) showed no differences between pre-heating and control trials (0.895 versus 0.896), the average VO_2 values for pre-heating were significantly lower than the control condition (53.7 versus 55.0 mL O_2/kg, $p < 0.01$). The rate of perceived exertion (RPE) values were on average lower for the pre-heating trial compared to the control trial (12.92 versus 13.28) but failed to reach statistical significance ($p = 0.13$).

The average blood lactate levels between cooling and control trials showed no major differences (3.46 versus 3.60) although the rate of increase of blood lactate accumulation was less than half as rapid for the pre-heating trial compared to the control trial (0.0308 versus 0.0644, Figure 2). No significant trends were observed in average heart rate trends between the pre-heating and control trial (162.3 versus 161.2 bpm).

The post-cooling results are in Figure 3. There was no significant trend over the 3-minute time intervals of RER with and without cooling, although the RER value at the 12 minute time interval was significantly lower ($p < 0.05$) than the control.

Cyclist Study

Seven of ten subjects were used in the analysis of data. Three subjects were excluded for non-compliance to the protocol. The remaining subjects were divided into responders (n = 5) and non-responders (n = 2) (Figure 4). The responders (n = 5) had consistently lower core temperature throughout the duration of the experiment (38.29°C versus 38.56°C, $p < 0.01$, Figure 5). A second criterion for being labeled a responder was an increase in performance measures (time to trial completion, average and maximum wattage performed) during the cooling trial. One subject was placed in the responder group after only meeting the second condition, although he had no change in T_{es} temperature. The non-responder group (n = 2) was composed of subjects who displayed a decrement in performance, measured in the time to complete each trial, the average wattage per trial, and the maximum wattage performed per trial, although their temperature was higher for the control condition for the duration of the experiment (Figure 6).

The results of each performance measure are summarized in Table 2. The responders displayed a lower average time over the eight trials (32.98 versus 33.71 seconds, $p < 0.05$), a higher average wattage output (426.0 versus 404.7 watts, $p < 0.05$), and a higher maximum wattage output (678.4 versus 648.2 watts, $p < 0.01$). The non-responder group had decrements in performance during the cooling trial compared to the control condition, with higher average time trial values (34.58 versus 34.34 seconds), lower average wattage output (383.3 versus 386.9 watts), and lower maximum wattage output (520.1 versus 536.1 watts). Upon comparison between trials, none of the three performance measurements in the control group reached a significance level of $p < 0.05$.

Associated with performance, lactate and heart rate measurements were also compared between and within groups. The average lactate values of the responder group were lower during the cooling trial (16.15 versus 17.52, $p < 0.05$, Figure 7). The non-responder group had higher average lactate values during cooling (12.45 versus 10.82, $p < 0.01$) as well as had higher lactate values recorded at the end of each of the eight trials during cooling versus control (Figure 8). The heart rate values for both groups demonstrated no significant trends for either responders (169.3 versus 168.2 bpm) or non-responders (157.8 versus 159.2 bpm) between cooling and control trials, respectively. The cooling and control values of each subject were collected together and are presented in Figure 8. The RER values for cooling on three-minute intervals were significantly lower for cooling over control at $p < 0.05$ levels. The post-exercise temperatures were also significantly lower for cooling over control, with the 3, 6, 9, and 12-minute interval values for temperature at a $p < 0.01$ level and the 15-minute interval value at a $p < 0.05$ level (Figure 9).

Discussion

Sub-maximal Exertion Running Study

The results suggest that pre-heating may have slight performance-enhanc-

ing benefits. The VO_2 values were lower for the pre-heating trial although the same pace was maintained for both trials. In addition, the higher rates of rise of lactate for the control trials compared to the preheating condition suggests that the body was more efficient at managing metabolite accumulation in the pre-heating condition. Lower RPE values were consistent with results that suggest pre-heat trials perceived an easier workload, although not reaching statistical significance. These three findings support the theory that temperature manipulation can improve performance.

Although the subjects entered a hot room for the pre-heating trial, the temperature measurements suggest that the subjects experienced a drop in T_{es} of 1.2 $\pm$0.4°C. One potential reason for a lowering of core temperature monitored as a result of the body redistributing heat in the body to the periphery in order to exchange heat and avoid a future rise in core temperature. A second reason for the observed decrease in may be due to recording failure of the OMNI-3000 device at higher ambient temperatures.

A second reason for the increase in performance for preheated subjects may be due to the impacts of pre-heating as aiding in pre-exercise warm-ups. After concluding a 20-minute warm-up on the treadmill, subjects in both the pre-heated and control conditions maintained a relatively static position. As a result, subjects may not have been able to perform an adequate warm-up routine and may have been aided by the warmer environment to prepare the muscles for the 40-minute pre-exercise bout.

Finally, the subjects in the control were given a placebo 'cooling' by placing one hand in the AVACore device set at 12°C. The thermal gradient created by this setting resulted in relatively no change in T_{es} over the duration of the 15-minute period between the warm-up and sub-maximal exercise bout. One possible explanation for this observation is that the temperature was set low deliberately to cause a local vasoconstriction for subjects in order to maintain a higher core temperature. As a result, the normal vaso-regulatory mechanisms in the periphery may have

been activated and led to general peripheral constriction that may have lasted through the beginning of exercise, resulting in less efficient heat exchange for the control subjects and less effective mechanisms to deal with metabolites, as evidenced by the higher rate of lactate accumulation in control conditions.

Post-exercise cooling appears to have no effect for sub-maximal exercise, as evidenced by Figure 3. The lack of change in RER values is not surprising, as the subjects were working at a sub-maximal exercise level and therefore did not generate significant levels of metabolites or disturb the acid-base balance of the individual. Without changes in either metabolism or acid-base balance, it is not surprising to note no effects of cooling on RER values.

Cycling Study

Based on the results of the responder group, it appears that cooling is an effective means to increase performance in maximal exertion exercises, as the cooling trials for the responder group resulted in faster time trials, as well as higher average and maximum wattage compared to control conditions. The lactate and heart rate values do not give any additional support for cooling as a performance enhancer, though there may be reasons behind these findings. Lactate is an expected result of anaerobic work, and therefore should be high in both cooling and control environments, regardless of cooling. In addition, heart rate should be elevated in both trials. The results seem to be consistent to a theory of a central mechanism of fatigue related to elevated temperatures [21, 22].

Although the subjects in the control group exhibited higher T_{es} during the control trials over the cooling trials, the difference between the first and eight trials was 0.60°C for cooling and 0.48°C for control (Figure 6). This suggests that although the non-responders had lower T_{es} values for the duration of the study, the temperature was rising at a faster rate for the cooling trial. This trend was not observed in the responder group, as the responder group had a net increase of 0.08°C for cooling com-

pared to 0.32°C for control.

The difference in responses between individuals deserves mentioning. A few different reasons may account for the division of the seven individuals into two group responses. The two subjects in the non-responder group did not show improvements between the control and cooling trials, as observed by a consistently lower average and maximum wattage compared with the responder group for both cooling (383.4 versus 426.0 watts; 678.4 versus 520.1 watts) and control periods (386.9 versus 426.0 watts; 536.1 versus 648.2 watts) for average and maximum wattage, respectively. These values suggest that the composition of groups may have been divided into power athletes tending to fit into the responder group and the non-responders being more endurance-oriented. In addition, the non-responders were unable to generate the same lactate levels that would further support this finding.

One other interesting finding was the effects of post-exercise cooling on the RER values of individuals. Over the fifteen-minute post-exercise period, the RER values were significantly lower for individuals during cooling (0.97 versus 1.03, p<0.01). Interesting to note that the T_{es} values for subjects were also suppressed during the post-exercise cooling bout (37.83°C versus 38.05°C, p<0.01), suggesting that temperature was a causal link in changing the RER values. These preliminary findings suggest that cooling could have an impact on recovery following maximal exercise.

Future research needs to be conducted to determine if the RER differences are due to a change in the post-exercise metabolism of individuals, acid-base balance, or the rate of respiration. In addition, future research needs to address the performance discrepancy between individuals during maximal exercise bouts. Possible areas to consider include developing better procedures and methods to find the constriction threshold for each individual and setting an appropriate temperature gradient to optimize heat exchange.

References

1. Allen DG, Kabbara AA, and Westerblad H. Muscle fatigue: the role of intracellular calcium stores. *Can J Appl Physiol* 27: 83-96, 2002.
2. Bergersen TK, Eriksen M, and Walloe L. Local constriction of arteriovenous anastomoses in the cooled finger. *Am J Physiol* 42: R880-R886, 1997.
3. Brooks GA, Fahey TD, White TP, and Baldwin KM. Exercise Physiology: Human Bioenergitics and Its Applications: Mayfield Publishing, 2000.
4. Grahn DA, Brock-Utne JG, Watenpaugh DE, and Heller HC. Recovery from mild hypothermia can be accelerated by mechanically distending blood vessels in the hand. *J Appl Physiol* 85: 1643-1648, 1998.
5. Hales JR, Jessen C, Fawcett AA, and King RB. Skin AVA and capillary dilatation and constriction induced by local skin heating. *Pflugers Arch* 404: 203-207, 1985.
6. Hepple RT. The role of O2 supply in muscle fatigue. *Can J Appl Physiol* 27: 56-69, 2002.
7. Jentjens RL, Wagenmakers AJ, and Jeukendrup AE. Heat stress increases muscle glycogen use but reduces the oxidation of ingested carbohydrates during exercise. *J Appl Physiol* 92: 1562-1572, 2002.
8. Kaminski HJ and Richmonds CR. Extraocular Muscle Fatigue. *Ann NY Acad Sci* 956: 397-398, 2002.
9. Lamb GD. Voltage-sensor control of Ca^{2+} release in skeletal muscle: insights from skinned fibers. *Front Biosci* 7: 834-842, 2002.
10. McCully KK, Authier B, Olive J, and Clark BJr. Muscle fatigue: the role of metabolism. *Can J Appl Physiol* 27: 70-82, 2002.
11. Midttun M and Sejrsen P. Cutaneous blood flow rate in areas with and without arteriovenous anastomoses during exercise. *Scand J Med Sci Sports* 8: 84-90, 1998.
12. Montain SJ and Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 73: 1340-1350, 1992.
13. Nybo L and Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* 91: 1055-1060, 2001.
14. Nybo L and Nielsen B. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *J Appl Physiol* 91: 2017-2023, 2001.
15. Soreide E, Grahn DA, Brock-Utne JG, and Rosen L. A non-invasive means to effectively restore normothermia in cold stressed individuals: a preliminary report. *J Em Med* 17: 725-730, 1999.