PRECOOLING AND EXERCISE CAPACITY
DURING HEAT STRESS

by

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A DISSERTATION

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ABSTRACT

Vigorous exercise in a hot and humid environment can substantially impair exercise performance and exercise capacity compared to moderate temperature conditions. Cycling requires high force generation and consequently utilizes a high rate of energy expenditure and often takes place during the hot summer months, which also contributes to heat storage and a rise in core temperature during exercise. There are several factors that could affect exercise performance, however, one explanation for the decrease in exercise performance while working in the heat is the strain put on the cardiovascular system. Part of this strain involves cardiovascular drift (CV drift), defined as a steady increase in heart rate (HR) and a decrease in stroke volume (SV) that occur during an extended bout of constant-rate, submaximal exercise [50-75% of maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\))]. Precooling has been shown in previous studies to diminish cardiovascular strain and thus, improve exercise performance during prolonged exercise lasting between 15 – 45 min in hot ambient temperatures. In order to evaluate the effects of precooling on cycling performance, two studies and a literature review were conducted. The first study evaluated the effects of 20 min of precooling the upper body (torso, head and neck) during an active warm-up, followed by a 15-km simulated time trial. The second study evaluated the effects on CV drift following 20 min of whole body (calve, thigh, torso, neck and head) precooling. Healthy and physically active male cyclists were recruited for both of the studies. A repeated measures, counterbalanced design was used for both of the studies. For both studies, the effect of pre-cooling on time trial performance and CV drift showed very limited differences between the cooling intervention and the control trials. The method of cooling used in both trials may not be the best way to truly decrease the core temperature enough to elicit a true
ergogenic effect in time trial pace cycling and CV drift. More research looking at a practical and convenient way of cooling cyclists before exercising in the heat is warranted.
DEDICATION

I dedicate this dissertation to my best friend and my wife Alissa Marie Katica, who has been extremely supportive and has been an integral part of this dissertation experience. I also dedicate this to my son Bryce Patrick Katica. I hope he will find his path through academia and enjoy his path as I have. I also want to dedicate this project to my parents Robert and Nancy Katica, and my sister Alison Blomgren who have been a great example and very supportive throughout my entire life. Also, I would like to dedicate this dissertation to Jonathan Wingo, Robert and Kelly Pritchett for being such an inspiration, and for all of their help and support. I would not be where I am today without all of your help. Furthermore, I would like to thank my committee members Gary Hodges, Mark Richardson and Ann Godfrey for all of their help throughout this process. Finally, I would like to thank all of my friends and colleagues (Robert Herron, Stacy Bishop, Stephen Carter, Andrew Del Pozzi, Greg Ryan, Elisha Williams, Annie Collins, and Jared Hornsby) for all of their tremendous support.
**LIST OF ABBREVIATIONS AND SYMBOLS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
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<tr>
<td>ANOVA</td>
<td>analysis of variance</td>
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<td>cm</td>
<td>centimeter</td>
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<td>kg</td>
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<td>km</td>
<td>kilometer</td>
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<td>C</td>
<td>Celsius</td>
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<td>P</td>
<td>probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value</td>
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<td>W</td>
<td>watt</td>
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<td>CV</td>
<td>cardiovascular</td>
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<td>GXT</td>
<td>graded exercise test</td>
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<tr>
<td>USG</td>
<td>urine specific gravity</td>
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<tr>
<td>T&lt;sub&gt;re&lt;/sub&gt;</td>
<td>rectal temperature</td>
</tr>
<tr>
<td>Q</td>
<td>cardiac output</td>
</tr>
<tr>
<td>(\dot{V}O_2)</td>
<td>peak oxygen uptake</td>
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<tr>
<td>(\dot{V}O_2)</td>
<td>maximal oxygen uptake</td>
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<tr>
<td>(\dot{V}O_2)</td>
<td>rate of oxygen uptake</td>
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<td>(\bar{T}_{sk})</td>
<td>mean skin temperature</td>
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<td>Hz</td>
<td>hertz</td>
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<td>SV</td>
<td>stroke volume</td>
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<td>HEAD</td>
<td>head and neck cooling</td>
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<td>VEST</td>
<td>ice vest cooling</td>
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<td>RPM</td>
<td>revolutions per minute</td>
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<tr>
<td>CONTROL</td>
<td>no cooling</td>
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<td>HR</td>
<td>heart rate</td>
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<td>HR$_{\text{max}}$</td>
<td>maximal heart rate</td>
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<td>PAR-Q</td>
<td>Physical Activity Readiness Questionnaire</td>
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<td>RPE</td>
<td>rating of perceived exertion</td>
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<td>RTC</td>
<td>rating of thermal comfort</td>
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<td>RH</td>
<td>relative humidity</td>
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<tr>
<td>$SD$</td>
<td>standard deviation</td>
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<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Sciences</td>
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<td>=</td>
<td>equal to</td>
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<td>&lt;</td>
<td>less than</td>
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<td>%</td>
<td>percent</td>
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<tr>
<td>SR$_{\text{local}}$</td>
<td>local sweat rate</td>
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ACKNOWLEDGMENTS

I would like to express my greatest appreciation to Dr. Jonathan Wingo for guiding me through this dissertation experience. Furthermore, I would like to thank my committee members Dr. Gary Hodges, Dr. Mark Richardson, Dr. Robert Pritchett and Dr. Ann Godfrey for helping me through this dissertation journey. Additionally, the quality of this study was greatly enhanced by the assistance of my fellow students Robert Herron, Stacy Bishop, Stephen Carter, Andrew Del Pozzi, Greg Ryan, Elisha Williams, Jason Ng, Annie Collins, and Jared Hornsby for all of their tremendous support.

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CHAPTER I:
INTRODUCTION

Cycling requires high force generation and consequently utilizes a high rate of energy expenditure. Cycling at high intensity can result in a rise in core temperature related to the high rate of skeletal muscle metabolism and concomitant heat storage (1), and doing so in a hot environment will contribute to a more rapid rise in core temperature. Besides a rapid core temperature rise, cycling in hot conditions results in reduced aerobic capacity and performance compared to cycling in more temperate conditions (9, 18, 20).

The decrease in exercise performance during hyperthermia may be partly explained by increased cardiovascular strain. Part of this strain involves cardiovascular drift (CV drift), defined as a steady increase in heart rate (HR) and a decrease in stroke volume (SV) that occurs during an extended bout of constant-rate, submaximal exercise [50-75% of maximal oxygen uptake (VO2max)] (16). The mechanisms which explain the occurrence of CV drift have been studied for several decades and include peripheral displacement of blood volume as well as decreased ventricular filling time related to hyperthermia-induced tachycardia (6-8, 16). CV drift can occur in temperate environments, but greater changes occur in hot environments (12, 15).

In addition to cardiovascular strain, achievement of a critical core temperature limit has also been implicated in explaining diminished exercise capacity and cycling performance (13). Some have argued this may be an artifact of elevated cardiovascular strain associated with high
skin and muscle temperatures and a diminished core-to-skin thermal gradient since the independent effects of a critical core temperature have not been demonstrated (4). Nonetheless, exercise limitations associated with a high core temperature may actually be more related to a critically high hypothalamic temperature (2). The carotid artery is a primary supplier of blood to the brain, and thereby, hypothalamus. Hence, cooling the blood which supplies the hypothalamus may increase the time required to reach a critical hypothalamic temperature limit, and furthermore it may increase heat storage capacity if it reduces the baseline core temperature. Similarly, cooling large muscle groups of the upper and lower body may have a similar attenuation effect on hypothalamic temperature and heat storage throughout the body. Cooling administered prior to high-intensity endurance-type athletic events, commonly referred to as precooling, has become increasingly popular as a method of attenuating performance decrements associated with high-intensity exercise in hot conditions (1, 3, 14, 19). Such precooling strategies include application of ice packs to the torso and neck (1, 14, 19) and immersion in water, each for ~20-30 min before exercise (3, 5, 17).

Surprisingly, no research has investigated the effect of precooling during an active warm-up on cycling time trial performance, and only 2 studies have investigated the effect of precooling during an active warm-up before running. Both studies showed that ice vest precooling administered during a warm-up, compared to a control condition without precooling, significantly (P < 0.05) blunted the rise in core temperature and heart rate, decreased time to completion, and lowered perceptual measures during exercise (1, 14). Despite these positive findings, extrapolation of the data to activities other than 5-km running is tenuous. For example, completion of a 16.1-km (10-mile) cycling time trial requires more time and relies more heavily
on a slightly different muscle mass than a 5-km running race, and therefore the effects of precooling on such an event remain speculative and uncertain.

Like time trial performance, no studies have investigated the effect of precooling on CV drift, or its consequences, during prolonged, submaximal cycling. An associated negative consequence of CV drift is a decrease in \( \dot{V}O_{2\text{max}} \). A series of studies has explored the relationship between CV drift and a decrease in \( \dot{V}O_{2\text{max}} \) and shown that an increase in HR ranging from 12%-16% and a decrease in SV ranging from 10%-16% occur concurrently with a decrease in \( \dot{V}O_{2\text{max}} \) ranging from 9%-19% (11, 15, 22-24). The physiological explanation for these findings is uncertain, but has been speculated to be related to decreased SV associated with CV drift that persists at maximal exertion. To clarify whether the nature of the relationship between CV drift and the decrease in \( \dot{V}O_{2\text{max}} \) during heat stress is causal, the aforementioned investigations manipulated CV drift using hyperthermia (25), hydration (10), power output (21), ambient temperature (15), and body cooling (20). In each case, when the magnitude of CV drift was attenuated, so was the decrease in \( \dot{V}O_{2\text{max}} \). Furthermore, when the magnitude of CV drift was unabated, i.e., larger, the decrease in \( \dot{V}O_{2\text{max}} \) also was larger. Despite these previous findings, more research is necessary to determine if an intervention administered before exercise in the heat, designed to manipulate CV drift, can influence the concomitant decrease in \( \dot{V}O_{2\text{max}} \). Such information will add valuable insight to the body of knowledge regarding the metabolic consequences of CV drift and the nature of the relationship between CV drift and \( \dot{V}O_{2\text{max}} \). Additionally, such research has important practical implications. Maintenance of as high a \( \dot{V}O_{2\text{max}} \) as possible is desirable because maintaining a high \( \dot{V}O_{2\text{max}} \) permits performance of activities at a given work rate at a lower relative metabolic intensity and with a favorable perceived exertion. If \( \dot{V}O_{2\text{max}} \) is decreased, any given workload is performed at a higher
physiological strain and with greater perceived effort. Therefore, countermeasures designed to minimize negative effects on $\dot{V}O_{2\text{max}}$ are important for athletes, firefighters, industrial workers, and soldiers who must frequently perform moderate-intensity exercise for prolonged periods in hot conditions, and which may be followed by high-intensity, maximal or near maximal efforts.

One such countermeasure may be precooling prior to endurance exercise. Precooling has been shown to positively benefit work performance during heat stress, such as increased tolerance time, blunted core temperatures, and decreased perceptual measures (1, 14, 19). However, the extent to which precooling influences the magnitude of CV drift and decrease in $\dot{V}O_{2\text{max}}$ following prolonged, submaximal exercise in the heat is unknown.

Therefore, in order to evaluate the effects of precooling on cycling performance and CV drift, 2 studies were conducted. In addition, a review of literature was completed to examine previous studies investigating the effects of precooling. The purpose of the first study was to evaluate the impact of precooling the upper-body (head, neck and torso) during an active warm-up on simulated 16.1-km cycling time trial performance. The torso, neck and head were cooled for 20 min using an ice vest and cold gel packs, respectively, followed by a simulated 16.1-km cycling time trial. This trial was compared to a control trial with no cooling. The hypothesis for the first study was that upper body cooling would result in improved performance (i.e., faster time) compared to no cooling. The purpose of the second study was to determine if precooling mitigates the magnitude of CV drift, and accompanying decrement in $\dot{V}O_{2\text{max}}$, during exercise in the heat. To achieve this goal, participants cycled for either 15 min or 45 min at 60% $\dot{V}O_{2\text{max}}$ followed by a graded exercise test (GXT) in 35 °C, 50% relative humidity. The hypothesis for the second study was that whole body precooling for 20 min would attenuate the magnitude of CV drift and the decrease in $\dot{V}O_{2\text{max}}$ during prolonged, submaximal exercise in a hot
environment. Importantly, these studies elucidated the effects of elevated skin and core temperatures on athletic performance (Study 1) and add further insight into the nature of the relationship between CV drift and $\bar{V}O_{2\text{max}}$ during exercise in the heat (Study 2).
CHAPTER II:
IMPACT OF PRECOOLING THE UPPER BODY ON TIME TRIAL PACED CYCLING IN THE HEAT

Abstract

The purpose of this study was to test the hypothesis that cooling the upper body during a warm-up enhances performance during a subsequent 16.1-km simulated cycling time trial in a hot environment. Eight, trained male cyclists (peak oxygen uptake = 57.8 ± 5.0 mL·kg⁻¹·min⁻¹) completed 2 simulated 16.1-km time trials in a warm environment (30.0 ± 0.5 °C, 43.8 ± 2.0 % relative humidity) each separated by 72 hours. On each occasion participants warmed up for 20 min while either wearing head and neck ice wraps, along with torso cooling using an ice vest (COOLING), or no cooling apparatus (CONTROL). Following warm-up mean skin temperature ($\bar{T}_{sk}$), mean body temperature ($\bar{T}_{b}$) and rating of thermal comfort (RTC) were significantly lower (all $P < 0.05$). However, rectal temperature ($T_{re}$) was unaffected ($P = 0.35$). The effects of precooling on $\bar{T}_{sk}$ and $\bar{T}_{b}$ were not sustained during exercise such that values for COOLING and CONTROL were not different. Likewise, time to completion was not significantly different between trials (30.4 ± 3.5 min vs. 29.3 ± 3.6 for CONTROL and COOLING, respectively, $P = 0.09$). Despite a lack of statistical significance, the time trial was completed faster during COOLING compared to CONTROL by 6 out of 8 (75%) participants with an average time that was 1.06 ± 0.1 min faster. These data suggest that competitive cyclists may experience a modest benefit, if any, while utilizing cooling modalities during an active warm-up before a time trial.

KEY WORDS: cooling, high paced cycling, thermoregulation
Introduction

Time trial cycling requires high force generation and consequently utilizes a high rate of energy expenditure. Such exercise results in a rise in core temperature related to the high rate of skeletal muscle metabolism and concomitant heat storage (1, 15, 25, 26). Furthermore, cycling events often take place during the hot summer months, which also contributes to heat storage and a rise in core temperature during exercise (14). It is well established that exercise capacity and performance are impaired in hot conditions compared to more temperate ones (10, 13, 30-32). This may be related to achievement of a critical core temperature limit (14) or excessive cardiovascular strain, either of which will diminish exercise capacity and cycling performance.

If a critical core temperature limit indeed exists, it likely varies among individuals and may depend on factors such as age, fitness level and body mass (19, 20, 25). In endurance-trained athletes, Gonzalez-Alonso (14) showed this limit to approximate 40 °C, and this has been substantiated by other studies demonstrating a significant decrease in exercise performance or voluntary termination of exercise when core temperature reaches ~40 °C (14, 22, 30). However, the existence of a critical core temperature limit has been challenged in light of runners achieving core temperatures in excess of 40 °C without negative health effects and without performance decrements (12). Nevertheless, in the presence of performance decrements concomitant with heat stress it has been suggested that such decrements may be related to high hypothalamic temperature more than high body temperature per se (7, 8). The carotid artery is a primary supplier of blood to the brain, and thereby, to the hypothalamus. Hence, cooling the blood which supplies the hypothalamus may extend the time required to reach a potentially detrimental, elevated hypothalamic temperature. This may also increase heat storage capacity and attenuate decrements in exercise performance in the heat. Similarly, cooling large muscle
groups of the upper-body may have a similar attenuation effect on hypothalamic temperature and heat storage.

Such an effect may explain the efficacy of precooling prior to endurance exercise bouts in hot conditions (1, 6, 9, 19, 26, 30). Precooling prior to exercise has been shown to improve performance and delay fatigue for vigorous intensity exercise in the heat lasting ~15-40 min (1, 9, 15, 30). Such precooling strategies include application of ice packs to the torso and neck (1, 15, 29, 30) and immersion in water (9, 11, 17, 21, 28) for ~20-30 min before exercise. Neck cooling using gel ice wraps and torso cooling using an ice vest are practical and fairly inexpensive and are typically administered during passive rest (11, 30).

Surprisingly, limited research has investigated the effects of precooling during an active warm-up even though anecdotally, many athletes consider a proper warm-up an important way to optimize neuromuscular function and psychological mind set (1, 15), and empirically, an active warm-up may facilitate rates of muscle contraction (3). Only 2 studies have investigated the effect of precooling during an active warm-up before running. Both studies showed that ice vest precooling administered during a warm-up, when compared to a control condition without precooling, blunted the rise in core temperature and heart rate, decreased time to completion, and lowered perceptual measures during the exercise (1, 15). Despite these positive findings, extrapolation of the data to activities other than 5-km running is tenuous. For example, completion of a 16.1-km cycling time trial requires more time and relies more heavily on a slightly different muscle mass than a 5-km running race, and therefore the effects of precooling on such an event remain speculative and uncertain. Therefore, the purpose of this study was to evaluate the impact of precooling the upper-body (head, neck and torso) during an active warm-up on performance during a simulated 16.1-km cycling time trial. Precooling was administered
for 20 min to the torso using an ice vest and to the neck and head using cold gel packs under one condition, and no cooling was administered under another condition considered a control. The decision to precool both the torso and head/neck simultaneously was based on a desire to maximize the effect of precooling by covering a large surface area. We hypothesized that torso cooling combined with head/neck cooling would result in improved performance (i.e., faster time) compared to no cooling (control).

**METHODS**

**Research design**

A repeated measures research design was used in which all participants were tested under all experimental conditions, which were completed in counterbalanced order. There were a total of 3 exercise sessions. Each exercise session was held in the mid- to late afternoon at the same time of day for a given participant in order to avoid circadian variations in core body temperature. After completion of a peak oxygen uptake (\( \bar{V}O_{2\text{peak}} \)) test, participants completed a familiarization trial, followed by two 16.1-km simulated cycling time trials. Each time trial was preceded by a 20-min warm-up including either 1) head and neck wrapped in gel ice packs and torso covered in an ice vest (COOLING), or 2) no cooling (CONTROL).

**Participants**

Eight healthy, trained male cyclists were recruited from the University’s Cycling and Triathlon Club teams (ages 19 – 40). A power analysis (23) revealed this sample size was adequate to detect a moderate effect size (24) between treatments for time to complete the 16.1-km cycling time trial. Only male participants were utilized because the time frame in which data had to be collected was not sufficient to control for core temperature fluctuations concomitant with the female menstrual cycle. All participants were competitive cyclists, free of any known
diseases as determined by health history questionnaire and performing at least 180 km of cycling every week. The study was approved by the university’s institutional review board in advance, and participants provided written informed consent prior to participation.

Following completion of the consent and health history forms, participants had their height and weight (bike shorts only) measured and body fat percentage estimated from the sum of 3 skinfolds (16). Furthermore, all participants were familiarized with the Velotron Dynafit Pro cycle ergometer (Racer Mate Inc. Seattle, WA) that was used in subsequent visits. The familiarization trial was conducted in a temperate climate (22 °C and 40% relative humidity). Participants were then fitted on the bike and completed a 16.1-km time trial to become familiar with the pace of a time trial and with the testing protocol.

**Determination of \( \dot{V}O_{2\text{peak}} \)**

Following a 72-hour rest period, participants revisited the laboratory having refrained from heavy exercise, ingestion of caffeine, and ingestion of alcohol during the preceding 24 h. Upon arrival, a urine sample was tested for determination of urine specific gravity (USG). USG was used to ensure subjects were adequately hydrated (USG ≤ 1.020). Participants then complete a graded exercise test (GXT) on the cycle ergometer to determine \( \dot{V}O_{2\text{peak}} \) defined as the average of the highest 2 consecutive 30-s averages for oxygen uptake; this value was considered \( \dot{V}O_{2\text{peak}} \) because a plateau in oxygen uptake did not occur for every participant and follow-up testing was not performed to confirm such a plateau. Testing took place in an environment maintained at 22 °C and 40% relative humidity. Participants underwent a 5-min warm-up at a moderate workload. The test commenced at the same workload used for the warm-up, and power output increased 25 W every 2 min until volitional fatigue. Oxygen uptake and other gas exchange measures were measured using open-circuit spirometry (Parvo Medics,
Sandy, Utah) and were averaged every 30 s. Heart rate (HR) was recorded during the final 10 s of every minute of the GXT using a Polar telemetry transmitter unit (Polar, Stamford, CT). Rating of perceived exertion (RPE) was recorded during the final 30 s of each stage (5). Furthermore, maximal heart rate (HR\text{max}) was defined as the highest 5-s value observed at volitional fatigue and maximum power output was defined as the highest value observed over the final 30-s period at volitional fatigue.

**Experimental Trials**

After 72 h, participants revisited the laboratory for the first experimental trial. Upon arrival, USG was measured to ensure adequate hydration (USG ≤ 1.020). Next, participants dressed in bike shorts, socks, cycling shoes, and a cycling jersey. The same ensemble was worn for each experimental trial. They then inserted a flexible rectal thermocouple (model RET-1, Physitemp, Clifton, NJ) approximately 10 cm past the anal sphincter. The rectal probe then was securely taped to the gluteus maximus under the waist band of the shorts. Next, thermocouples were taped to the lateral calf, anterior thigh, lower back, lower abdomen, upper chest and upper back (all on the right side of the body) for measurement of mean skin temperature (T\text{sk}) using the weighted average of the 6 sites. Participants also wore a HR monitor as described previously. Prior to the start of exercise, each participant was fitted to the cycle ergometer (proper seat and handle bar height, etc.), and they were required to stay seated throughout the duration of the test.

All experimental sessions were performed in an environmental chamber maintained at 30 °C and ~ 45\% relative humidity. Participants warmed up for 20 min at 65\% \text{VO}_2\text{peak} and 70-80 revolutions per minute (RPM). COOLING included head and neck cooling with Elasto-Gel ice wraps (Southwest Technologies Inc., North Kansas City, MO) placed around the entire head and around the neck covering the majority of the neck along with torso cooling using a modified
Ironman® reflective vest (World Endurance Sports LLC, Tampa, FL; VEST). Cooling was administered for the entire 20 min. After 10 min, the ice packs were replaced with fresh ice packs. At the end of the 20-min warm-up, all cooling packs were removed. The warm-up was identical during CONTROL trials except that there was no cooling.

Following the 20-min warm up, participants completed a simulated 16.1-km time trial as quickly as possible. During the time trial, HR was monitored continuously and recorded every km, along with measurement of rating of thermal comfort (RTC) and RPE. Furthermore, rectal temperature (T_{re}) and T_{sk} were monitored and recorded continuously at 50 Hz using a data acquisition system (Biopac MP150, Santa Barbara, CA). A fan circulating air at 3.3 m·s\(^{-1}\) was directed at the front of participants during the time trial. Mean body temperature (T_{b}) was calculated from T_{re} and T_{sk} according to the following equation (2):

\[
T_{b} = (0.8 \times T_{re}) + (0.2 \times T_{sk})
\]

**Statistical Analysis**

Data are presented as means ± SD. A paired samples t-test was used to test the significance of mean differences for primary outcome measures such as time to completion of the simulated time trial, subjective ratings, and physiological measures (e.g., HR, T_{re}, T_{sk}, RPE, RTC, and total time) between the COOLING and CONTROL trials. Data were analyzed using SPSS v. 20.0 (IBM, Inc., New York, NY), and p-values less than 0.05 were considered statistically significant.

**Results**

Body mass and urine specific gravity prior to each experimental trial were not different between treatments (both P > 0.05), which suggests hydration status was similar at the beginning
of all tests. Furthermore, ambient temperature ($T_a$) and relative humidity (RH) were not different between trials (both $P > 0.05$).

**Warm-up**

During the warm-up, cooling of the upper body via ice vest, neck collar and head cooling was successful in lowering $T_b$, $T_{sk}$, and RTC (Table 2.1). However, cooling was ineffective in reducing $T_{re}$ during the warm-up ($P = 0.35$). As expected, HR was not different between treatments ($P = 0.70$), because exercise intensity during the warm-up for each treatment was based on a similar $\%\dot{V}O_{2peak}$.

**Responses During the 16.1-km Time Trial**

Despite power output averaging 15.7 W (7.6%) higher during the time trial completed after COOLING relative to CONTROL, results were not significantly different ($P = 0.21$). Comparable power outputs between treatments resulted in average HRs that were not different (COOLING = 170 ± 7 beats/min; CONTROL = 167 ± 8 beats/min; $P = 0.46$). Additionally, the 3.6% (non-significant; $P = 0.16$) higher speed maintained during the time trial after cooling during the warm-up relative to no cooling during the warm-up was apparently insufficient to result in a significantly faster completion time ($P = 0.09$) [Table 2.2 and Figure 1].

Even though $\bar{T}_{sk}$ was 8.3% lower following warm-up during COOLING compared to CONTROL ($P = 0.38$), this difference was not sustained such that $\bar{T}_{sk}$ during the time trial was not different between treatments (COOLING = 33.42 ± 0.33 °C; CONTROL = 33.80 ± 0.26 °C; $P = 0.20$). Apparently this did not alter body heat storage since $T_{re}$ was not different between treatments ($P = 0.13$). The lower $\bar{T}_{sk}$ after the warm-up during COOLING resulted in a lower $\bar{T}_b$ ($P = 0.02$) during warm-up, but during the time trial, since $T_{re}$ and $\bar{T}_{sk}$ were not different between treatments, $\bar{T}_b$ also was not different ($P = 0.80$).
Consistent with the physiological responses, neither of the subjective ratings were significantly different between treatments. RPE throughout the 16.1-km time trial averaged 16 ± 2 for both treatments (\(P = 0.34\)) and RTC averaged 5.5 ± 0.5 for both treatments (\(P = 0.59\)).

**Discussion**

The current study was designed to investigate the efficacy of precooling during a warm-up prior to a simulated cycling time trial. Ice vests and head cooling were used as an ergogenic aid to enhance time trial performance in the heat. The primary finding of the current study is that while COOLING was effective in lowering \(\bar{T}_b\) during warm-up, this did not result in faster completion times.

The current findings are in contrast to 3 previous studies that found improved cycling performance after precooling. Ross et al. (27) investigated simulated time trial performance in hot conditions (32-35 °C and 40-50% RH) after participants either 1) rested in cold water (10 °C) for 10 min followed by wearing a cooling jacket, or 2) consumed an ice slurry beverage. Both cooling techniques were associated with a 3.0% increase in power and a 1.3% improvement in performance time (approximately 1:06 min) compared to the control condition (27). Likewise, Kay et al. (18) observed that participants were able to cycle farther (0.9 km) in 30 min in a warm environment (31.4 °C, 60.2% relative humidity) after whole-body immersion for 60 min in a cold (24 °C) water bath. The third study (11) investigated the effects of precooling using an ice vest and cold air exposure (3 °C) prior to 35 min of cycling in hot conditions (33 °C). Ice vest and cold air precooling reduced physiological and psychophysical strain as well as increased endurance performance.

The reason for the discrepant results relative to the current findings is unclear. Part of the difference may be explained by differences in methodology, e.g., environmental conditions,
cooling modalities, etc. used in the aforementioned studies relative to the current study. Moreover, none of the studies cited above included a warm-up prior to the cycling time trial (11, 27). It may be that a cycling time trial performance that is not preceded by a warm-up is improved by precooling relative to one that is performed without precooling. Additionally, a warm-up prior to a cycling time trial may mask the positive effects of precooling. That said, the ecological validity of completing a cycling time trial without a warm-up is questionable. It remains unclear whether precooling without a warm-up would result in superior cycling performance relative to warm-up alone. Furthermore, resting in cold air or immersion in cold water are not practical in a race setting, so even if they do improve subsequent cycling performance, they are logistically difficult to carry out.

The current study is the first to investigate whether precooling during an active warm-up is an effective way to improve subsequent cycling time trial performance. The cooling vest and head cooling were effective in lowering $T_{sk}$ 2.85 °C during the 20-min active warm-up in the COOLING relative to the CONTROL condition. This decrease was essentially sustained prior to exercise, with no change during the 2-3 min between the end of the warm-up and start of the time trial.

However, the reduced $T_{sk}$ was less effective in blunting the rise in $T_{re}$ during warm-up than the effects reported in the 3 previously mentioned studies (18, 27), or other studies in which ice vests were used for precooling before running events (1, 15). These studies showed a decrease in $T_{re}$ from 0.3 to 1.0 °C during precooling. Only one of these studies featured precooling during a warm-up, but it was before a simulated 5-km running trial instead of cycling (1). They found $T_{re}$ was 0.3 °C lower following the active warm-up with precooling than the active warm-up without precooling, and they attributed improved performance to this lower $T_{re}$.
The decrease in $T_{re}$ in the current study was about half (0.14 °C) that of the Arngrimsson et al. study (1). A minimum threshold decrease in $T_{re}$ during precooling may be necessary in order to observe improved subsequent performance. That said, one study found that a decreased $T_{re}$ is not entirely necessary for precooling to improve performance. Kay et al. (18) found that precooling the entire body by way of water immersion for 1-h decreased $T_{sk}$ by 4.4 °C but did not change $T_{re}$. Subsequent cycling performance improved by 6% (18). Taken together, results of these studies and the current study suggest that a minimum threshold decrease in either $T_{sk}$ or $T_{re}$ may be necessary in order to observe improved subsequent cycling or running performance in a hot environment.

The mechanism by which precooling has been shown to improve performance in hot ambient conditions in other studies (1, 4, 18, 27) is not completely understood. Authors of previous studies showing a performance benefit speculate that performance may be improved by reducing $T_{b}$ before exercise, which in turn could lower cardiovascular strain experienced during exercise. By lowering the cardiovascular strain and body temperature, more work can be performed before a critical level of hyperthermia, possibly associated with fatigue, is reached. Precooling could also aid in the perceptions of thermal discomfort and effort that may influence the exercise intensity which the participant can maintain in the heat (19, 25). The combination of all of these different variables appears to explain the ability to sustain higher intensities during the latter part of the experimental trials in some of these studies. Results showed a decrease in $T_{b}$, rating of thermal comfort and $T_{sk}$ following the warm-up while wearing the ice vest and head cooling garment. These precooling effects have been observed by other researchers (1, 11, 15) and were expected, but the extent to which the precooling strategy would affect physiological outcomes during an active warm-up on a bike was unknown. The beneficial effects of
precooling were negated by the second km of the time trial. The brief period during which precooling effects were sustained likely explains why performance times between treatments were not statistically different. Apparently, for some individuals a greater decrement in body temperatures, perceived exertion, and thermal comfort is necessary to significantly affect performance during a 16.1-km cycling time trial in the heat.

Limitations

A limitation of the present study is that a simulated time trial using a programmable cycle ergometer in a controlled laboratory setting was used instead of an actual time trial on a real bike in a field setting. We used a simulated 16.1-km, flat time trial course to mimic as closely as possible performance on the road but still permit physiological measurements that could provide insight into the mechanisms underlying any performance benefits. The 16.1-km times in the laboratory were slower than self-reported times out on the road for some of the participants, likely because of 1) the use of a different bike and set up of the bike; 2) potential distraction associated with physiological monitoring equipment, e.g., core and skin thermocouples; 3) performing the time trial in the heat; 4) lack of the same magnitude of convective heat loss that would be expected to occur during cycling outdoors; and 4) lack of other competitors who might have affected motivation and pacing. Nonetheless, these limitations likely did not influence the tests of our hypotheses regarding effects of precooling during warm-up on subsequent time trial performance.

Perspectives and Practical Significance

Although not statistically different, the effect of the precooling strategy obtained by using the cooling vest combined with head cooling showed an average 2.2% (COOLING = 29.32 min, CONTROL = 30.38 min) improvement in time to complete the time trial. While the overall time
difference may seem small, this magnitude of improvement is comparable to that found by Arngrimsson et al. (1) in runners (1.1%), in which case 5-km running finishing times were 13 s faster (equivalent to 57 m advantage) after a warm-up with an ice vest. This small difference was statistically different between precooling and control in the Arngrimsson (1) study. Although not statistically different in the current study, both studies show a moderate improvement in performance for some individuals with precooling during the warm-up. Additionally, in competitive cyclists such as the ones who participated in this study, a 1.02% (56-s) improvement translates into a 0.50-km advantage based on the average pace cycled by participants during COOL. This could be practically meaningful for some individuals.

We conclude that wearing a cooling vest and cooling the head during an active warm-up before a cycling time trial is effective in blunting increases in $\bar{T}_b$, rating of thermal comfort and $\bar{T}_{sk}$ during warm-up. However, these effects were not sustained during the time trial, and therefore they do not translate in to an increase in performance time for all individuals. Further research investigating alternative practical cooling methods that can more effectively blunt a rise in core temperature during warm-up is necessary to determine if subsequent performance is affected.
References


# Appendices

Table 2.1

*Cardiovascular, Temperature and Perceptual Measures at the End of the Warm-up*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Cooling</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{re}$ (°C)</td>
<td>37.06 ± 0.20</td>
<td>37.20 ± 0.25</td>
</tr>
<tr>
<td>$T_{sk}$ (°C)</td>
<td>34.51 ± 0.54*</td>
<td>31.66 ± 1.80</td>
</tr>
<tr>
<td>$T_{b}$ (°C)</td>
<td>36.64 ± 0.23*</td>
<td>36.19 ± 0.45</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>127 ± 4</td>
<td>127 ± 5</td>
</tr>
<tr>
<td>RTC</td>
<td>5.0 ± 0.5*</td>
<td>3.5 ± 0.5</td>
</tr>
</tbody>
</table>

Values are means ± SD; $T_{re}$, rectal temperature; $T_{sk}$, mean skin temperature; $T_{b}$, mean body temperature; HR, heart rate; RTC, rating of thermal comfort. *P < 0.05 vs. COOLING.

Table 2.2

*Temperature and Performance Measures During the Last Kilometer of the 16.1-km Time Trial, and Time to Complete the Entire Time Trial.*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Cooling</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{re}$ (°C)</td>
<td>38.23 ± 0.25</td>
<td>38.38 ± 0.34</td>
</tr>
<tr>
<td>Time to Completion (min)</td>
<td>30.38 ± 3.5</td>
<td>29.32 ± 3.6</td>
</tr>
<tr>
<td>Power Output (W)</td>
<td>213.1 ± 69.5</td>
<td>228.7 ± 43.5</td>
</tr>
<tr>
<td>Speed (km·h⁻¹)</td>
<td>36.5 ± 5.61</td>
<td>37.38 ± 4.26</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>183 ± 8</td>
<td>183 ± 7</td>
</tr>
</tbody>
</table>

Values are means ± SD; $T_{re}$, rectal temperature; HR, heart rate.
Figure 1. Individual completion times for the simulated 16.1-km time trial following COOLING and CONTROL treatments administered during a 20-min warm-up.
CHAPTER III:
THE EFFECT OF PRECOOLING ON CARDIOVASCULAR DRIFT AND MAXIMAL OXYGEN UPTAKE DURING HEAT STRESS

Abstract

Hot environmental conditions result in substantial cardiovascular drift (CV drift) during exercise, which is proportional to reductions in maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\)). Continuous cooling during exercise has been shown to mitigate the magnitude of CV drift and thereby the concomitant reduction in \(\dot{V}O_{2\text{max}}\), but it remains unknown if precooling before exercise has the same effect. The purpose of this study was to determine if precooling blunts the magnitude of CV drift and accompanying decrement in \(\dot{V}O_{2\text{max}}\) during prolonged, constant-rate, submaximal exercise in the heat. After a control \(\dot{V}O_{2\text{max}}\) test, 5 men cycled on separate days at 60% \(\dot{V}O_{2\text{max}}\) for 45 min in 35 °C after being cooled for 20 min (45C) or after no cooling treatment (45NC). After the 45 min, they completed a graded exercise test to measure \(\dot{V}O_{2\text{max}}\). \(\dot{V}O_{2\text{max}}\) also was measured after 15 min of cycling at 60% \(\dot{V}O_{2\text{max}}\) on a different day (15max), so that CV drift and \(\dot{V}O_{2\text{max}}\) could be measured over the same points in time. Precooling successfully lowered mean skin temperature just before the start of exercise (mean ± SD, 32.89 ± 0.41 °C vs. 34.48 ± 1.26 for 45C and 45NC, respectively, P < 0.05), but rectal and mean body temperatures were unaffected (both P > 0.05). During subsequent exercise, the magnitude of CV drift was unaffected by precooling, such that \(\dot{V}O_{2\text{max}}\) also was unaffected (both P > 0.05). Precooling of
the head, neck, quadriceps and calf using gel ice wraps and of the torso using an ice vest has no effect on the magnitude of CV drift and decrease in $\dot{V}O_2_{max}$ during subsequent exercise in the heat.

**KEY WORDS:** cooling, $\dot{V}O_2_{max}$, heart rate, thermoregulation, cycling
Introduction

Exercising in a hot environment can substantially impair exercise performance and exercise capacity (28) compared to moderate temperature conditions (15, 20, 28). One explanation for the decrease in exercise performance while working in the heat is the strain put on the cardiovascular system. Part of this strain involves cardiovascular drift (CV drift), defined as a steady increase in heart rate (HR) and a steady decrease in stroke volume (SV) that occur during an extended bout of constant-rate, submaximal exercise [50-75% of maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\))] (24). The mechanisms which explain the occurrence of CV drift have been studied for several decades and include peripheral displacement of blood volume as well as decreased ventricular filling time related to hyperthermia-induced tachycardia (10, 12, 14, 24). CV drift can occur in temperate environments, but greater changes occur in hot environments (17, 20).

A series of investigations has explored whether CV drift has negative consequences, such as a reduction in \(\dot{V}O_{2\text{max}}\). An increase in HR ranging from 12%-16% and a decrease in SV ranging from 10%-16% have been shown to be related to a decrease in \(\dot{V}O_{2\text{max}}\) ranging from 9%-19% (16, 20, 31, 32, 35, 37). The physiological explanation for these findings is uncertain, but has been speculated that decreased SV associated with CV drift persists during maximal exertion such that \(\dot{V}O_{2\text{max}}\) is compromised. To clarify whether the nature of the relationship between CV drift and the decrease in \(\dot{V}O_{2\text{max}}\) during heat stress is causal, the aforementioned investigations manipulated CV drift using hydration (16), power output (32), ambient temperature (20), and body cooling (31). In each case, when the magnitude of CV drift was attenuated, so was the decrease in \(\dot{V}O_{2\text{max}}\). Furthermore, when the magnitude of CV drift was unabated (i.e., larger), so
was the decrease in \( \dot{V}O_{2\text{max}} \). These findings strongly support a causal relationship between CV drift and a decrease in \( \dot{V}O_{2\text{max}} \).

All of the manipulations of CV drift cited above occurred during exercise. It is currently unknown if an intervention administered before exercise in the heat can manipulate CV drift during the subsequent exercise to an extent that would influence the decrease in \( \dot{V}O_{2\text{max}} \). Such information will add valuable insight to the body of knowledge regarding the metabolic consequences of CV drift and the nature of the relationship between CV drift and \( \dot{V}O_{2\text{max}} \). Furthermore, such research has important practical implications. Maintenance of as high a \( \dot{V}O_{2\text{max}} \) as possible is desirable because maintaining a high \( \dot{V}O_{2\text{max}} \) permits performance of activities requiring a given work rate at a lower relative metabolic intensity and with a favorable perceived exertion. If \( \dot{V}O_{2\text{max}} \) is decreased, then any given workload is performed at a higher physiological strain and with greater perceived effort. Therefore, countermeasures designed to minimize negative effects on \( \dot{V}O_{2\text{max}} \) are important for athletes, firefighters, industrial workers, and soldiers who must frequently perform moderate-intensity exercise for prolonged periods in hot conditions, and which may be followed by high-intensity, maximal or near maximal efforts.

One such countermeasure may be precooling prior to endurance exercise. Precooling has been shown to have positive benefits on subsequent performance of work in the heat, such as increased tolerance time, blunted core temperature, and improved perception of effort (2, 7, 18, 29). However, the extent to which precooling influences the magnitude of CV drift and decrease in \( \dot{V}O_{2\text{max}} \) following prolonged, submaximal exercise in the heat is unknown. Therefore, the purpose of this study was to determine if precooling prior to exercise in the heat mitigates the magnitude of CV drift and whether this consequently mitigates a decline in \( \dot{V}O_{2\text{max}} \). We hypothesized that whole body precooling for 20 min would attenuate the magnitude of CV drift,
and thus the decrease in $\dot{V}O_{2\text{max}}$, following prolonged, submaximal exercise in a hot environment.

**Methods**

**Research Design**

A repeated measures research design was used in which all participants were tested under all experimental conditions which were completed in counterbalanced order. There were a total of 4 exercise sessions. Each exercise session was held in the mid to late afternoon and at the same time of day for each respective participant to avoid circadian variations in core body temperature. Following completion of a control $\dot{V}O_{2\text{max}}$ test, participants completed 3 submaximal cycling bouts that included the following: 1) precooling of the head, neck, torso, thigh and calf for 20 min followed by 45 min of cycling at 60% $\dot{V}O_{2\text{max}}$; 2) condition 1 with no precooling; 3) no precooling followed by 15 min of cycling at 60% $\dot{V}O_{2\text{max}}$. All trials were followed by a graded exercise test (GXT) to determine $\dot{V}O_{2\text{peak}}$. The purpose of the separate 15- and 45-min trials was to measure $\dot{V}O_{2\text{peak}}$ during the same time interval in which CV drift occurred. $\dot{V}O_2$ values obtained during the GXT following experimental trials were referred to as $\dot{V}O_{2\text{peak}}$ because the verification procedure to ensure a plateau in $\dot{V}O_2$, performed during the control $\dot{V}O_{2\text{max}}$ test (see below), was not performed.

**Participants**

Following approval by the university’s Institutional Review Board, 5 healthy, male cyclists were recruited from the University’s cycling team, triathlon team and Kinesiology department (ages 19 - 40). A power analysis (22) revealed a sample size of $n = 5$ was sufficient to detect a large effect size (23) among the measures of $\dot{V}O_{2\text{max}}$. Originally, 7 males were recruited, however, data was corrupted and 2 participants data had to excluded from the study.
All participants were healthy active males, free of any known diseases, as determined by health history questionnaire, and performing at least 6 h of exercise every week. Additionally, participants provided written informed consent prior to participation.

**Determination of Control \( \dot{V}O_2_{\text{max}} \)**

Participants arrived after having refrained from heavy exercise, caffeine, and/or alcohol during the previous 24 h. They reported to the laboratory at least 2 h postprandial, but well hydrated. To verify adequate hydration, a urine sample was analyzed for urine specific gravity (USG; ≤ 1.020 for adequate hydration). Participants then completed a graded exercise test (GXT) on a Velotron Dynafit Pro cycle ergometer (Racer Mate Inc. Seattle, WA) to determine \( \dot{V}O_2_{\text{max}} \) (defined as the average of the highest 2 consecutive 30-s averages). Testing took place in an environment maintained at ~22 °C and 40% relative humidity. Participants underwent a 5-min warm-up at a self-selected intensity. The GXT began at 200 W and increased 25 W every 2 min until participants could no longer maintain at least 40 rpm or until they stopped on their own. Oxygen uptake and other gas exchange variables were measured using open-circuit spirometry (Parvo Medics, Sandy, Utah) and were averaged every 30 s. Heart rate (HR) was measured and recorded every 5 s using a Polar HR monitor (Polar, Stamford, CT). Rating of perceived exertion (RPE) was recorded during the last 30 s of each stage. Maximal heart rate (HR\(_{\text{max}}\)) was defined as the highest 5-s value observed at volitional fatigue, and maximum power output was defined as the highest value observed over the last 30-s period at volitional fatigue.

Twenty minutes after the end of the GXT, participants completed an additional bout of cycling in order to ensure a plateau in \( \dot{V}O_2 \) was achieved. Power output was set to that of the last stage of the GXT (if < 1 min was completed during the last stage of the GXT) or to a power output 25 W higher than that of the last stage of the GXT (if ≥ 1 min was completed during the
last stage of the GXT). A modified version of the criterion emphasized by Taylor et al. (26) was used to define a plateau in ŶO₂max between 2 successive stages as an increase in ŶO₂ of less than half (135 mL·min⁻¹) of the expected increase (270 mL·min⁻¹) based on the American College of Sports Medicine (1) metabolic equation for gross ŶO₂ during leg ergometry: ŶO₂ = (10.8 × W × M⁻¹) + 7, where ŶO₂ is gross oxygen uptake in mL·kg⁻¹·min⁻¹, W is power output in watts, and M is body mass in kg.

Twenty minutes after the follow-up procedure to ensure a plateau in ŶO₂max, participants cycled for ~10 min while the power output corresponding to the intensity (60% ŶO₂max) that was utilized during the experimental trials was verified. Minor adjustments to power output were made as needed to elicit 60% ŶO₂max.

**Experimental Trials**

After at least 48 h but no more than 7 d, participants returned to the laboratory for their first experimental trial. Upon arrival, USG was measured to ensure that participants were adequately hydrated (USG ≤ 1.020). Then participants measured nude body weight on a digital scale (Tanita Corporation, Tokyo, Japan) and dressed in bike shorts, socks, and cycling shoes that were also used for the subsequent experimental trials. Participants were then asked to insert a flexible rectal thermocouple (model RET-1, Physitemp, Clifton, NJ) approximately 10 cm past the anal sphincter. The rectal probe was securely taped to the gluteus maximus under the waist band of the shorts. A HR monitor was worn around the chest as described previously. Next, a flexible venous catheter was inserted into a forearm vein and kept patent with 0.9% normal saline (45-min trials only). Then, thermocouples were taped to the lateral calf, anterior thigh, lower back, lower abdomen, upper chest and upper back (all on the right side of the body) for measurement of mean skin temperature (T̄sk) using the weighted average of the six sites (27).
Rectal temperature ($T_{re}$) and $T_{sk}$ were measured continuously during exercise and were used to calculate mean body temperature ($T_b$) as $[(0.8 \cdot T_{re}) + (0.2 \cdot T_{sk})]$.

Participants then sat upright outside of the heat chamber for 20 min while a probe was taped to the right posterior forearm for measurement of skin blood flow using laser-Doppler flowmetry (MoorVMS-LDF2, Moor Instruments, Devon, UK). Furthermore, a capsule was taped adjacent to the skin blood flow probe for measurement of local sweat rate ($SR_{local}$) using capacitance hygrometry. Additionally, electrodes were attached to the back for measurement of cardiac output ($\dot{Q}$) using impedance cardiography (19, 30) (Biopac MP150, Goleta, CA). For 1 of the 45-min trials, during the 20-min preparation period participants underwent cooling of the head, neck, torso, quadriceps and calf (45C) using Elasto-Gel ice wraps (Southwest Technologies Inc., North Kansas City, MO). The other 45-min trial was identical but did not involve any precooling (45NC). The 15-min trial (15max) did not involve precooling before the trial; only $T_{sk}$, $T_{re}$, and HR were measured during exercise; and the GXT to measure $\dot{V}O_{2peak}$ started after 15 min instead of after 45 min.

After precooling, participants entered an environmental chamber maintained at 35 °C and 40-50% relative humidity. They then mounted the cycle ergometer, and resting $\dot{V}O_2$, skin blood flow, systolic and fourth phase diastolic blood pressure using auscultation of the brachial artery, and $T_{re}$ and $T_{sk}$ were measured, followed by acquisition of a 2-mL blood sample into a Vacutainer containing EDTA. Systolic and diastolic blood pressures were used to calculate mean arterial pressure as $1/3$ pulse pressure plus diastolic pressure. Blood samples were used to measure hemoglobin and hematocrit, which were then used with exercising values to estimate changes in plasma volume using the Dill and Costill equation (11).
After all resting measures were obtained, participants began cycling at a power output corresponding to 60% \( \dot{V}O_{2\text{max}} \) for either 15 or 45 min, immediately followed by a GXT to measure \( \dot{V}O_{2\text{peak}} \). During the 45-min trials, rectal and skin temperatures, skin blood flow, and local sweat rate were recorded continuously. Additionally, oxygen uptake, blood pressure, RPE, and rating of thermal comfort (RTC) were measured, and a 2-mL blood sample was collected, in that order, between 10-15 and 38-45 min of exercise. The rate of change of the impedance signal (\( dZ/dt \)) from impedance cardiography was measured continuously. However, after all other measures at each respective time point were completed, the participant was asked to keep the torso as stable as possible for ~ 30 s while still cycling so a clean \( dZ/dt \) signal tracing could be obtained. The maximum \( dZ/dt \) at each respective time point was then used to calculate SV offline using the following equation: 

\[
SV = R \times \left( L^2/Z_0^2 \right) \times T \times dZ/dt_{\text{max}} \tag{19, 30}
\]

Where SV = stroke volume, \( R \) = resistivity of blood (147 Ohms·cm used as a constant), \( L \) = length between electrodes (cm), \( T \) = systolic left ventricular ejection time (derived from the electrocardiogram tracing), and \( dZ/dt_{\text{max}} \) = magnitude of the largest impedance change during systole (Ohms/s).

At the end of each submaximal cycling bout, participants began a GXT to measure \( \dot{V}O_{2\text{peak}} \) with no cessation of cycling. Power output was instantly increased 25 W, with 25-W increases every 2 min until volitional exhaustion. Oxygen uptake and HR were recorded continuously, and RPE was obtained at the end of each 2-min stage. Each participant was instructed to stay seated throughout the duration of the test. Three minutes after the end of the GXT, an additional blood sample was obtained, and all instrumentation except the skin blood flow probe was removed. Then, the participant dismounted the cycle ergometer, exited the chamber, towed off, re-measured nude body weight. All experimental sessions were separated by 72 h.
**Statistical Analysis**

Resting measures taken after precooling in 45C (or an equivalent 20-min period during 45NC) but before the start of exercise (e.g., T_{sk}, T_{re}, T_{b}, and HR) were compared between treatments using paired samples t-tests. During exercise, thermoregulatory measures, such as T_{re}, T_{sk}, T_{b}, skin blood flow (% change from rest), and local sweat rate, were acquired continuously at a sampling rate of 50 Hz using a data acquisition system (Biopac MP150, Santa Barbara, CA). A 1-way repeated measures analysis of variance (ANOVA) was used to test the significance of mean differences in \( \dot{V}O_{2\text{peak}} \) among treatment conditions (control, 15max, 45C, and 45NC). For other outcome measures, such as HR, subjective ratings, and physiological measures (e.g., T_{sk}, T_{re}, etc.), 2-way (treatment × time) repeated measures ANOVAs were conducted to test the significance of mean differences. For all ANOVAs, in the event of a significant F statistic in which the sphericity assumption was upheld, paired samples t-tests with Tukey’s Honestly Significant Difference were used to determine individual differences between treatments and time points. Data were analyzed using SPSS v. 20 (IBM, Inc., New York, NY), and p-values less than 0.05 were considered statistically significant. All data are presented as means ± SD.

As mentioned before, after data were collected, dZ/dt_{max} data were used to calculate SV. This measure is sensitive to movement artifact. Despite our attempt to obtain a clear tracing by requesting participants hold their torsos as stable as possible for ~ 30 s at each respective time point, the signal to noise ratio was too small to render the data useable. Therefore, SV data from dZ/dt were not obtainable. In order to get an index of SV, oxygen pulse (O_{2} pulse) data from 6 published studies (31, 32, 34) and 1 unpublished study using a similar exercise protocol and similar environmental conditions were regressed to predict SV. O_{2} pulse has been shown to
predict SV within ~ 20% (3, 13). While this margin of error is not ideal, we felt it worthwhile to utilize this method in order to present the trends in the SV data, with the understanding that the actual values may be inaccurate. Furthermore, lack of accurate SV data should not have affected the tests of our hypotheses, i.e., the HR data can be used to represent the magnitude of CV drift. The resultant regression equation was $SV = 5.278 \times \text{O}_2 \text{ pulse} + 23.563$, $R^2 = 0.63$, SEE = 12.61.

Results

Urine specific gravity was not different between the NC and C trials (pre-trial USG = 1.013 ± 0.005 and 1.015 ± 0.005 for NC and C, respectively, $P = 0.19$). Additionally, pre-trial body mass was not significantly different among Control, 15min, 45C and 45NC trials ($P = 0.88$). Taken together, these measurements indicated that participants began each trial with a similar hydration status. Because of sweat losses, post-trial body mass was lower than pre-trial body mass regardless of treatment (pre-trial 45C = 76.1 ± 6.5 kg, 45NC = 76.1 ± 6.7 kg, vs. post-trial 45C = 75.0 ± 6.0 kg, 45NC = 75.1 ± 6.6 kg, $P = 0.006$ for main effect of time), so cooling did not differentially affect the change in body mass (-1.44 ± 0.70% vs. -1.32 ± 0.45% for 45C and 45NC, respectively, $P = 0.72$) or whole-body sweat rate during 45C (1,243.5 ± 804.4 mL/h) relative to 45NC (1,095 ± 444.7 mL/h), $P = 0.63$. Plasma volume decreased (Table 1) from rest during the 45-min of submaximal exercise in both 45NC and 45C. While there was no treatment × time interaction ($P = 0.40$), the decrease was larger in 45NC (treatment main effect, $P = 0.002$).

Responses to Precooling

Table 2 shows the responses to precooling prior to the start of exercise in the 45C and 45NC trials. Precooling was insufficient to lower $T_{re}$, but $T_{sk}$ was ~ 1.8 °C lower on average after precooling during 45C compared to after the baseline period during 45NC. However, by
the 15-min time point this difference was negated. Despite the difference in $\bar{T}_{sk}$ between treatments before the onset of exercise, $\bar{T}_{b}$ was unaffected.

**Responses during Submaximal Exercise**

Cardiovascular, perceptual and gas exchange responses to the submaximal exercise between the 15-min time point and the 45-min time point within each treatment are presented in Table 1. A substantial CV drift occurred in both experimental trials as evidenced by a 12.5% increase in HR between 15 and 45 min during 45C and an 11.3% increase in HR between 15 and 45 min during 45NC ($P < 0.001$ for main effect of time). $O_2$ pulse and predicted SV were higher at 15 min compared to 45 min in 45NC only ($P < 0.05$), but did not change over time between 15 and 45 min in 45C. Even though the work rate on the cycle ergometer was kept constant for each participant during each experimental trial, $\dot{V}O_2$ increased over time during 45C ($P = 0.02$). Despite the lower $T_{sk}$ at the onset exercise during 45C, there were no differences between treatments between 15 and 45 min of exercise ($P = 0.88$). Likewise, precooling did not attenuate the rise in $T_{re}$ since $T_{re}$ increased to the same extent over time under both treatments ($P = 0.11$).

No difference was found between 45C and 45NC in regards to $SR_{local}$ ($0.62 \pm 0.08$ and $0.48 \pm 0.02$ mg·cm$^{-2}$·min$^{-1}$ for 45NC and 45C 15-min time point values, respectively; $0.79 \pm 0.15$ and $0.67 \pm 0.11$ mg·cm$^{-2}$·min$^{-1}$ for 45NC and 45C 45-min time point values, respectively; $P = 0.88$), but $SR_{local}$ increased significantly over time across both treatments ($P = 0.002$ for main effect of time). Interestingly, during the 45C trials sweating began ~2.5 min after that during 45NC. Finally, RPE increased significantly over time during the exercise bout ($P = 0.001$ comparing 15 with 45 min across both treatments), but the increase was not different between 45C and 45NC.
Responses during Maximal Exercise

Responses to maximal exercise are presented in Table 3. $\dot{V}O_{2\text{max}}$ after the control test and after the 15-min trial were not different ($P > 0.05$), but despite this only the control $\dot{V}O_{2\text{max}}$ value was greater than $\dot{V}O_{2\text{peak}}$ after 45C and 45NC (11.5% and 13.8% decrease from control for 45C and 45NC, respectively, $P < 0.05$). The non-significant decrease in $\dot{V}O_{2\text{peak}}$ after 45 min in 45C and 45NC compared to 15 min may have practical significance, however, given these decreases amounted to 6.3% and 8.7% for 45C and 45NC, respectively. Similar to the submaximal bout of exercise, $T_{re}$, $\overline{T}_{sk}$ and $\overline{T}_{b}$ were not different between 45C and 45NC at the end of the GXT (all $P > 0.05$). Like the $\dot{V}O_{2\text{peak}}$ data, max test duration was not statistically different between the GXT after 15 min and after 45 min in the experimental trials ($P > 0.05$), despite being 21% and 27% lower after 45C and 45NC, respectively. Peak power output was not different during the GXT after 45C and 45NC ($P > 0.05$), but it was significantly lower during the GXT in 45NC compared to the GXT after 15 min ($P < 0.05$). Even though $\dot{V}O_{2\text{peak}}$ was lower after the experimental trials relative to control, it appears a maximal effort was provided by participants as evidenced by $HR_{\text{max}}$, RER, and RPE values that were not different across conditions at maximum (all $P > 0.05$). Likewise, blood lactate values at maximum were only different after 45NC compared to after 15 min ($P < 0.05$). This likely reflects the lower power output reached during that trial, rather than lack of effort.

Discussion

This study assessed whether whole-body precooling for 20 min would reduce the decline in $\dot{V}O_{2\text{max}}$ related to CV drift during submaximal exercise in the heat. As expected, the submaximal exercise protocol elicited considerable CV drift (as reflected by greater than 10% increases in HR over time in both experimental trials). However, contrary to our hypothesis,
precooling did not mitigate the magnitude of CV drift. Therefore, consistent with other studies (16, 31, 35), CV drift was accompanied by a subsequent reduction in $\dot{V}O_{2\text{max}}$.

The lack of precooling effect on CV drift is in contrast to prior studies that incorporated fan cooling during exercise in the heat (25, 31). Likewise, lack of a precooling effect on CV drift also resulted in a lack of effect on the decrease in $\dot{V}O_{2\text{max}}$, which is also in contrast to a previous study involving fan cooling during exercise. Wingo et al. (31) reported that fan cooling at 4.5 m/s beginning after about 10 min during 45 min of cycling at 60% $\dot{V}O_{2\text{max}}$ resulted in a decline in $\dot{V}O_{2\text{max}}$ between 15 and 45 min that was about one third (6%) the decline observed without fan cooling (18%). The reductions in $\dot{V}O_{2\text{max}}$ in the current study were smaller than the reduction in $\dot{V}O_{2\text{max}}$ in the no fan condition of the aforementioned study (31) and others with similar methodology (32, 33, 35), but the magnitude of CV drift was smaller as well, providing further support for the hypothesis posited elsewhere that suggests the magnitude of CV drift during heat stress is proportional (and perhaps causes) the decrease in $\dot{V}O_{2\text{max}}$ observed over the same time period (33). Apparently, precooling has no effect on mitigating CV drift and thereby attenuating a decrease in $\dot{V}O_{2\text{max}}$.

Precooling was effective in decreasing $\bar{T}_{sk}$ prior to the start of exercise. This was expected to delay the combined negative effects of elevated core and skin temperatures on cardiovascular strain (8). However, by ~2 min of exercise, the benefits of precooling were no longer apparent as $T_{sk}$ was no longer different between treatments from that point till the end of exercise. We speculate the rapid decrease in $\bar{T}_{sk}$ after the onset exercise in 45NC was because of skin cooling associated with the onset of sweating. This deduction is consistent with our data indicating an ~ 2.5 minute delay in sweating in the 45C condition relative to 45NC. In the precooling condition, $T_{sk}$ was ~32.7 °C at the start of exercise. Lowered skin temperature has
been shown to attenuate local sweating, independent of skin blood flow (36). So, even though skin blood flow was not different between conditions (Table 1), the benefits of decreased \( \bar{T}_{sk} \) may have been offset by a delay in local sweating associated with precooling. That said, though, whole-body sweat rate was not different during 45C compared to 45NC. Furthermore, other studies investigating precooling have reported greater sweat evaporation following precooling (75%) compared to a control condition (57%) (5, 21), so it remains unclear why the reduction in \( \bar{T}_{sk} \) with precooling in 45C was offset by a sharp decrease in \( \bar{T}_{sk} \) at the onset of exercise in 45NC.

The effects of precooling in the present study were short-lived. This is in contrast to studies reporting effects of precooling lasting as many as 30-40 min during prolonged endurance exercise in the heat (4, 6, 9). The explanation for this difference is uncertain but we speculate different exercise protocols and precooling protocols may explain the discrepant results.

Even though the present results do not support a beneficial effect of precooling on CV drift and subsequent \( \dot{V}O_{2\text{max}} \), they offer further support to a growing body of evidence relating CV drift to a decrease in \( \dot{V}O_{2\text{max}} \) (16, 31-33, 35, 37). The fact that \( \dot{V}O_{2\text{max}} \) decreased over the same time interval that CV drift occurred supports previous assertions (33) that the relationship between CV drift and a decrease in \( \dot{V}O_{2\text{max}} \) during exercise in the heat is causal in nature. We speculate the decreased SV that occurs as part of CV drift persists during maximal exercise such that \( SV_{\text{max}} \) is not attained, and thereby \( \dot{V}O_{2\text{max}} \) is reduced. This has implications for exercise prescription because this means the increased HR occurring in conjunction with CV drift reflects increased relative metabolic intensity. Furthermore, when CV drift occurs, performance is likely to be compromised because of the concomitant decrease in \( \dot{V}O_{2\text{max}} \). Future studies should
determine countermeasures to attenuate the magnitude of CV drift and subsequent reduction in 
\( \dot{V}O_{2\text{max}} \).

Precooling the entire body using icepacks did not diminish the magnitude of CV drift during a 45-min submaximal exercise bout in the heat and thus did not have an effect on \( \dot{V}O_{2\text{max}} \) when compared to a condition without precooling. While practical, under the conditions of this study, this precooling approach is likely ineffective in increasing heat storage, attenuating CV drift, and mitigating reduced \( \dot{V}O_{2\text{max}} \) associated with CV drift during prolonged exercise in the heat.
References


### Table 3.1

*Responses During Submaximal Cycling at 60% $\dot{V}O_{2max}$ after Different Conditions of Cooling Pre-exercise (mean ± SD).*

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th>15-min</th>
<th>45-min</th>
<th>15-min</th>
<th>45-min</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$ (L·min$^{-1}$)</td>
<td>45NC</td>
<td>2.35 ± 0.3</td>
<td>2.41 ± 0.2</td>
<td>2.28 ± 0.3*</td>
<td>2.49 ± 0.2</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (% control $\dot{V}O_{2max}$)</td>
<td>45C</td>
<td>60.6 ± 8.4</td>
<td>62.1 ± 7.8</td>
<td>59.4 ± 3.2*</td>
<td>65.0 ± 2.9</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)**</td>
<td></td>
<td>152 ± 12</td>
<td>171 ± 10</td>
<td>154 ± 16</td>
<td>171 ± 11</td>
</tr>
<tr>
<td>SV (mL·beat$^{-1}$)</td>
<td></td>
<td>105.7 ± 12.6*</td>
<td>98.4 ± 9.0</td>
<td>101.9 ± 9.4</td>
<td>100.4 ± 5.6</td>
</tr>
<tr>
<td>Oxygen Pulse (mL·beat$^{-1}$)</td>
<td></td>
<td>15.6 ± 2.3*</td>
<td>14.2 ± 1.7</td>
<td>14.8 ± 1.8</td>
<td>14.6 ± 1.1</td>
</tr>
<tr>
<td>SR$_{local}$ (mg·cm$^{-2}$·min$^{-1}$)**</td>
<td></td>
<td>0.62 ± 0.08</td>
<td>0.79 ± 0.15</td>
<td>0.48 ± 0.02</td>
<td>0.67 ± 0.11</td>
</tr>
<tr>
<td>$\Delta$SkBF from rest (%)</td>
<td>45NC</td>
<td>428.8 ± 303.1</td>
<td>512.4 ± 412.6</td>
<td>504.8 ± 261.7</td>
<td>566.0 ± 343.8</td>
</tr>
<tr>
<td>$\Delta$PV from rest (%)†</td>
<td>45C</td>
<td>-11.4 ± 4.5</td>
<td>-11.0 ± 5.0</td>
<td>-7.4 ± 3.3</td>
<td>-8.80 ± 5.8</td>
</tr>
<tr>
<td>$T_{re}$ (°C)**</td>
<td></td>
<td>37.1 ± 0.3</td>
<td>38.2 ± 0.3</td>
<td>37.4 ± 0.4</td>
<td>38.2 ± 0.3</td>
</tr>
<tr>
<td>$\bar{T}_{sk}$ (°C)</td>
<td></td>
<td>35.0 ± 0.7</td>
<td>35.1 ± 0.6</td>
<td>35.2 ± 0.6</td>
<td>35.1 ± 0.7</td>
</tr>
<tr>
<td>$T_{b}$ (°C)**</td>
<td></td>
<td>36.7 ± 0.4</td>
<td>37.6 ± 0.3</td>
<td>36.9 ± 0.3</td>
<td>37.6 ± 0.3</td>
</tr>
<tr>
<td>RPE**</td>
<td></td>
<td>12 ± 1</td>
<td>14 ± 1</td>
<td>11 ± 1</td>
<td>14 ± 1</td>
</tr>
</tbody>
</table>

45NC no cooling trial, 45C precooling trial, $\dot{V}O_2$ oxygen uptake, SV stroke volume, SR$_{local}$ local sweat rate, $\Delta$SkBF change in skin blood flow, $\Delta$PV change in plasma volume, $T_{re}$ rectal temperature, $\bar{T}_{sk}$ mean skin temperature, $\bar{T}_{b}$ mean body temperature, RPE rating of perceived exertion. *P < 0.05 vs. 45-min time point within the same condition; **P < 0.05 for main effect of time; †P < 0.05 for main effect of treatment.
Table 3.2

Responses following 20 min of whole-body precooling (45C) or quiet sitting (45NC) (mean ± SD).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>45NC</th>
<th>45C</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{sk}$ (°C)</td>
<td>34.48 ± 1.26</td>
<td>32.89 ± 0.41*</td>
</tr>
<tr>
<td>$T_{b}$ (°C)</td>
<td>36.26 ± 0.35</td>
<td>36.26 ± 0.41</td>
</tr>
<tr>
<td>$T_{re}$ (°C)</td>
<td>36.71 ± 0.12</td>
<td>36.97 ± 0.28</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)</td>
<td>70 ± 14</td>
<td>74 ± 12</td>
</tr>
</tbody>
</table>

45NC no cooling trial, 45C precooling trial, $T_{re}$ rectal temperature, $T_{sk}$ mean skin temperature, $T_{b}$ mean body temperature, HR heart rate. *$P < 0.05$ vs. 45NC.
Table 3.3

Responses during the maximal exercise bouts (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>15max</th>
<th>45NC</th>
<th>45C</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ (L·min⁻¹)</td>
<td>3.84 ± 0.29</td>
<td>3.62 ± 0.30</td>
<td>3.30 ± 0.24*</td>
<td>3.38 ± 0.25*</td>
</tr>
<tr>
<td>VO₂ (ml·kg⁻¹·min⁻¹)</td>
<td>50.4 ± 3.5</td>
<td>48.0 ± 6.0</td>
<td>43.7 ± 5.0*</td>
<td>44.7 ± 4.8*</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>194 ± 5</td>
<td>193 ± 5</td>
<td>197 ± 3</td>
<td>197 ± 7</td>
</tr>
<tr>
<td>RPE</td>
<td>19 ± 1</td>
<td>20 ± 1</td>
<td>20 ± 1</td>
<td>20 ± 1</td>
</tr>
<tr>
<td>ΔPV from rest (%)</td>
<td>-</td>
<td>-</td>
<td>-12.2 ± 4.2**</td>
<td>-9.7 ± 3.1</td>
</tr>
<tr>
<td>Tₛk (°C)</td>
<td>-</td>
<td>-</td>
<td>34.9 ± 0.7</td>
<td>34.9 ± 0.8</td>
</tr>
<tr>
<td>T_b (°C)</td>
<td>-</td>
<td>-</td>
<td>37.7 ± 0.4</td>
<td>37.7 ± 0.3</td>
</tr>
<tr>
<td>T_re (°C)</td>
<td>-</td>
<td>-</td>
<td>38.5 ± 0.4</td>
<td>38.4 ± 0.3</td>
</tr>
<tr>
<td>SRₗocal (mg·cm⁻²·min⁻¹)</td>
<td>-</td>
<td>-</td>
<td>0.89 ± 0.16**</td>
<td>0.70 ± 0.12</td>
</tr>
<tr>
<td>Test Duration (min)</td>
<td>11.4 ± 2.6</td>
<td>9.7 ± 1.4</td>
<td>7.1 ± 2.0*</td>
<td>7.8 ± 2.2*</td>
</tr>
<tr>
<td>Power Output (W)</td>
<td>295.0 ± 27.4</td>
<td>284.0 ± 25.1</td>
<td>251.0 ± 32.9*†</td>
<td>259.0 ± 28.6*†</td>
</tr>
<tr>
<td>RER</td>
<td>1.11 ± 0.04</td>
<td>1.08 ± 0.07</td>
<td>1.07 ± 0.07</td>
<td>1.05 ± 0.07</td>
</tr>
<tr>
<td>Blood lactate (mmol·L⁻¹)</td>
<td>6.58 ± 2.03</td>
<td>6.75 ± 1.24</td>
<td>4.04 ± 1.94†</td>
<td>4.77 ± 1.52</td>
</tr>
</tbody>
</table>

45NC no cooling trial, 45C precooling trial, VO₂ oxygen uptake, HR heart rate, RPE rating of perceived exertion, ΔSkBF change in skin blood flow, ΔPV change in plasma volume, Tₛk mean skin temperature, T_b mean body temperature, RER respiratory exchange ratio. *P < 0.05 vs. control; **P < 0.05 vs. 45C; †P < 0.05 vs. 15max.
CHAPTER IV:
PRECOOLING REVIEW OF LITERATURE

Literature reviewed for this dissertation was selected from relevant journal articles and supplementary reference lists. Sources were obtained from searching electronic databases, which included PubMed/Medline and Google Scholar using keywords pertaining to cooling, performance and cardiovascular drift.

Several physiological changes that occur in the human body when exercising in hot ambient conditions can have an effect on athletic performance and exercise capacity. When exercise begins, metabolic heat production increases in proportion to the intensity of activity. During intense physical activity, an individual’s metabolic rate may increase 10 to 20 fold, equating to between 750 and 1,500 kcal per hour, and it may remain elevated for several hours following physical activity (38, 54). Exercising or working in a hot and/or humid environment can have a serious impact on performance and possibly even result in development of a heat illness (74, 81). In order to blunt a rise in core temperature and avoid these maladies, several physiological responses occur (81).

These responses include increases in sweating and skin blood flow (32). When superimposed on elevated muscle blood flow associated with exercise, the increase in skin blood flow taxes the cardiovascular system. Accordingly, the decrease in exercise performance during hyperthermia may be partially explained by increased cardiovascular strain. Elevated strain may
be manifested as cardiovascular drift (CV drift), defined as a progressive increase in heart rate (HR) and decrease in stroke volume (SV) during an extended bout of constant-rate, submaximal exercise [50-75% of maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\))] (70). The mechanisms which explain the occurrence of CV drift have been studied for several decades and include peripheral displacement of blood volume as well as decreased ventricular filling time related to hyperthermia-induced tachycardia (16, 21, 28, 70). CV drift can occur in temperate environments, but greater changes occur in hot environments (22, 31, 46). CV drift occurring in hot environments has been shown to be associated with decreased \(\dot{V}O_{2\text{max}}\) and thereby maximal performance capacity (30, 46, 82-88).

In addition to reducing performance because of elevated cardiovascular strain, elevated body temperature during exercise heat stress also may decrease performance by reaching a so-called “critical core temperature limit” that causes premature fatigue and voluntary cessation of activity (34). It is difficult to isolate the independent effects of elevated core temperature on performance because elevated core temperature is accompanied by tremendous cardiovascular strain (12). Nevertheless, some research suggests the existence of a critical core temperature limit at which point exercise performance/capacity is diminished (34, 66, 67).

Regardless of the mechanism by which prolonged exercise in the heat reduces performance and exercise capacity, cooling administered prior to high-intensity endurance-type athletic events, commonly referred to as precooling, has become increasingly popular as a method of attenuating performance decrements (4, 10, 40, 78). Such precooling strategies include application of ice packs to the torso and neck (4, 40, 78) and immersion in water, each (10, 15, 74) for ~20-30 min before exercise. Given that exercise heat stress increases body temperature, which then exacerbates cardiovascular strain and reduces performance, and given
the potential for precooling to mitigate these effects, the following sections will briefly review pertinent articles investigating cardiovascular and body temperature responses, as well as the ergogeneity of precooling, during exercise heat stress.

**Exercise Performance in Hot Ambient Conditions**

Performing work in a hot or humid environment is more physiologically stressful than working in a neutral or mild climate. For example, working in a hot or humid environment can cause premature fatigue (32, 34, 67). A study conducted by Gonzalez-Alonso et al. (34) found that participants with high core temperatures fatigue sooner than people with cooler core temperatures during prolonged exercise in the heat. The core temperature limit at which fatigue ensued that was investigated in this study (34) approximated 40 °C, and this has been substantiated by other studies demonstrating a significant decrease in exercise performance or voluntary termination of exercise when core temperature reaches ~ 40 °C (34, 60, 77, 78).

Opponents of the critical core temperature hypothesis posit that excessive cardiovascular strain present under the same circumstances eliciting a core temperature of ~ 40 °C (e.g., elevated skin temperature and redistribution of a large portion of the blood volume to the cutaneous circulation for heat dissipation) could be the primary cause of diminished exercise capacity instead of a critical core temperature per se (12). This is supported by studies that have demonstrated runners completing races at core temperatures in excess of 40 °C without negative health effects and without performance decrements (12, 23). It may be that both excess cardiovascular strain and elevated hypothalamic temperature (reflected by elevated core temperature) are responsible for performance decrements in hot conditions. Regardless of the exact cause, it is clear that exercise performance is indeed markedly reduced in hot conditions.
Other mechanisms that may contribute to performance decrements in the heat involve metabolic alterations associated with circulatory adjustments during heat stress (25-27). For example, intense physical activity in a hot environment leads to an increased rate of muscle glycogenolysis (24), an increased lactate production and an early accumulation of lactate compared to work performed in cooler temperatures (51).

**Cardiovascular Drift and Circulatory Adjustments During Exercise in the Heat**

When humans are exposed to a hot environment, the anterior portion of the hypothalamus receives afferent feedback from thermoreceptors that detect elevated core body and skin temperatures, and then integrates this information to coordinate a response that includes circulatory adjustments (13). These circulatory adjustments include cutaneous vasodilation in order to redirect a portion of the cardiac output to the skin for heat dissipation. Vasodilation is defined as the relaxation of the lumen (smooth muscles) which make up the walls of the vessels (81). When cutaneous vasodilation occurs during constant-rate exercise, especially if the exercise is of a moderate to high intensity, it increases cardiovascular strain because the heart must balance cardiac output (\(\dot{Q}\)) between the skin and working skeletal muscle. The end result is a progressive increase in HR and decrease in SV over time. As mentioned earlier, this phenomenon is termed CV drift (83, 87).

The specific mechanisms which explain the occurrence of CV drift have been studied for several decades. A couple of different primary hypotheses have emerged regarding the causes of CV drift. One hypothesis reported by Rowell (70) states that CV drift occurs due to a progressive increase in blood flow to the periphery and a concomitant drop in central venous pressure, which leads to a drop in SV. Rowell (70) argues that while SV is falling, HR increases indirectly most likely due to a baroreceptor reflex initiated to maintain \(\dot{Q}\) and blood pressure.
In contrast, other studies have shown that the blood flow to the periphery and more specifically to the forearm actually can plateau approximately 20-30 min into a constant bout of prolonged moderate intensity exercise in a temperate climate, even though SV continues to decline (28, 57). At the same time, elevated temperature can directly increase sinoatrial node firing and sympathetic nervous system activity, which increases HR (6, 35, 41, 45, 65). This has led some to speculate that the decline in SV results from decreased ventricular filling time associated with tachycardia (16, 17). Fritzche et al. (28) reported that participants cycling at a moderate intensity for 55 min experienced an 11% rise in HR that was accompanied by a 13% decline in SV between 15 and 55 min. In another trial, a β-blocker was used to attenuate the rise in HR over time. Skin blood flow was similar between trials, but the decline in SV was markedly reduced when the rise in HR was attenuated, thereby supporting the idea that blood displaced to the periphery is not responsible for the decline in SV. Furthermore, Nassis (57) reported a decline in SV while participants ran on a treadmill at 60% $\dot{VO}_{2\text{max}}$ while skin blood flow plateaued, or declined throughout the trial. During the trial HR drifted upward around 15 beats/min, once again supporting the notion that SV can decrease without the diversion of blood to the periphery.

Regardless of the cause of CV drift, several factors can affect its magnitude. These factors include, but are not limited to, environmental temperature, core body temperature, dehydration, and duration of exercise. Each of these will be discussed in the following paragraphs.

Environmental temperature has been shown to have a substantial impact on CV drift. Studies have shown that considerable CV drift occurs when ambient temperature exceeds 30 °C (5, 31, 32, 37, 55, 87). However, only a few studies have actually assessed the differences in CV
drift between hot and cold ambient temperatures (31, 46). Each study comparing hot and cold environments found that CV drift was greater in the hot environment compared to the cooler environment.

Exercise in hot environments will undoubtedly lead to higher core body temperature ($T_c$) in an exercising individual. High $T_c$ has been shown to increase skin blood flow as well as HR, which as mentioned previously reduces SV (71, 72). Therefore, higher $T_c$ results in greater CV drift (31, 32, 48, 87).

Hydration status can also have a significant impact on CV drift and fatigue during exercise (29, 32, 36, 56, 58). Studies have found that CV drift may be greater depending on the severity of dehydration (33, 56). Gonzalez-Alonso et al. (33) reported that dehydration of 4% of the participant’s body weight decreased SV and increased HR similarly to that of hyperthermic conditions. Additionally, dehydration combined with hyperthermia resulted in even greater effects on HR and SV (33). However, there may be a threshold level of dehydration at which the magnitude of CV drift is differentially affected during exercise in the heat. For instance, Wingo et al. (85) reported that dehydration of 0.3% and 2.5% did not differentially affect the magnitude of CV drift during 45 min of cycling in 35 °C. Apparently, a percentage of dehydration greater than 2.5% is necessary before the magnitude of CV drift is differentially affected during exercise in the heat.

All of the previously mentioned factors that could affect CV drift are exacerbated by the duration of exercise (16, 17, 30, 62). A longer duration of exercise compounds the effects of environmental temperature, core temperature, and hydration on CV drift.
Effects of Cardiovascular Drift on Performance

Until recently, it was unknown whether CV drift was a benign phenomenon or whether it had any detrimental consequences. Several studies have observed a negative effect of CV drift on \( \dot{V}O_{2\text{max}} \) in a hot environment (30, 46, 83-87). Wingo et al. (87) reported that CV drift occurred between 15 and 45 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in a 35 °C, and approximately 40% relative humidity ambient environment. The 12% increase in HR and 16% decrease in SV were related to a 19% decrease in \( \dot{V}O_{2\text{max}} \) (87). Similarly, a study conducted by Ganio et al. (29) investigated the effects of fluid ingestion versus no fluid ingestion on CV drift and \( \dot{V}O_2\text{peak} \) in 30 °C and found that the magnitude of CV drift was similar to studies investigating the effect of ambient temperature (46), hyperthermia (87), and body cooling (82) on CV drift and \( \dot{V}O_{2\text{max}} \).

However, the decrease in \( \dot{V}O_{2\text{max}} \) was only about half as large as in the studies manipulating CV drift using ambient temperature, hyperthermia, and body cooling (46, 82, 87), likely because thermal strain (as indicated by mean body temperature) was lower in the Ganio study (29) compared to the other studies. Nonetheless, similar to previous studies, maximum power output declined by 12.7% and the \( \dot{V}O_{2\text{max}} \) test duration decreased by \( \sim 3.3 \) min (27%) (29).

Another study using a similar protocol showed the impact of CV drift on cycling performance (83). Participants cycled for 45 min at 60% \( \dot{V}O_{2\text{max}} \) in a 35 °C environment. In one condition, power output was decreased in order to keep HR at a constant level and mitigate the magnitude of CV drift (83). During the control trial in which power output was not manipulated, the researchers reported an increase in HR of 13%, a decrease in SV of 10% and a decrease in \( \dot{V}O_{2\text{max}} \) of 15% between 15 and 45 min. During the experimental trial the wattage had to be decreased by 37% (\( \sim 157 \) W to \( \sim 98 \) W) to keep HR constant (83). The researchers reported that there was a 20% decrease in SV, but they attributed it to the decreased \( \dot{Q} \) associated with a lower
metabolic demand because of the lower wattage needed to keep HR constant rather than CV drift. This mitigation of CV drift resulted in half the decrease in $\dot{V}O_{2\text{max}}$ (7.5%) observed in the control trial.

While several factors could affect one’s exercise capacity and performance, collectively the studies described in this section demonstrate the negative impact that CV drift has on cycling performance because of its detrimental effect on $\dot{V}O_{2\text{max}}$.

**Natural Defenses against Hyperthermia**

As detailed above, given that elevated body temperature exacerbates CV drift, as well as reduces exercise capacity and performance, it is important to optimally dissipate heat in an effort to prevent an excessive rise in core temperature. The body has 4 main avenues by which it exchanges heat when exposed to a hot environment. Conduction is defined as the loss of body heat through the transfer of molecules to a cooler object that is applied to the surface of the skin (63). The rate of conductive heat loss depends on two factors: 1) the temperature gradient between the skin and the environment or object placed on the skin and 2) the thermal qualities of the objects in contact with one another (51, 63). Another avenue of heat exchange, convection, is defined as heat exchange associated with movement of water or air over the surface of the skin (64). Wingo and Cureton (82) assessed the effects of fan cooling on CV drift and subsequent effects on $\dot{V}O_{2\text{max}}$ after 45 minutes of cycling in a 35 °C, 60% RH environment. Fan cooling blunted the rise in core temperature and CV drift during exercise, and it also attenuated the decrease in $\dot{V}O_{2\text{max}}$ observed during a graded exercise test performed after the 45-min bout.

In addition to conduction and convection, heat exchange can occur via radiation, which is defined as heat exchange through infrared rays (63). This type of heat exchange can occur without any physical contact between the objects exchanging heat with one another. At rest the
human body loses up to 60% of heat from radiation because surrounding surfaces are typically cooler than the human body (51, 52, 64), which creates a favorable thermal gradient for heat exchange from the body to its surroundings. When performing work or exercising in a hot environment, the ability of the human body to cool itself through radiation, convection and conduction may be impeded because of clothing (51, 59). Furthermore, humans can actually gain heat via these avenues of heat exchange if the surrounding environment or object in contact with the body is hotter than the skin temperature (43). For example, the body can actually gain heat by radiative heat gains from the sun and nearby hot surfaces (43). Furthermore, convective heat loss is reduced or eliminated when the ambient temperature reaches temperatures close to, or above skin temperatures [(31-33 °C) (51, 61)].

When the body is exposed to hot environments that exceed skin temperature, dry heat exchange is compromised and therefore the evaporation of sweat becomes the primary avenue by which heat is lost. Throughout the dermis there is anywhere from 2 to 4 million sweat glands (14, 18). When the body temperature increases and cooling is needed, sweat glands are stimulated by sympathetic nerve fibers to secrete a saline solution to the surface of the skin (14, 18). In an environment with low humidity (and thereby low vapor pressure), sweat on the surface of the skin will readily evaporate. As sweat changes phase from a liquid to a gas, heat is dissipated. This cools the surface of the skin, which in turn begins to cool the blood that was diverted to the surface. The blood, which then circulates back to the deep body core, will be cooler, which enables it to pick up more heat from the deep core areas. Evaporative cooling depends on 3 factors: 1) ambient temperature and relative humidity, 2) convective currents around the surface of the skin, 3) the surface area of exposed skin (1, 3, 49). Cyclists performing time trials have high air flow (i.e., convective currents) over the surface of the skin.
Furthermore, the amount of skin surface area exposed for cyclists is usually pretty high. The main concern for cyclists, then, is the ambient temperature and the relative humidity. During humid and high ambient temperatures, relative humidity has the biggest impact on evaporative heat loss (51). Environments with high relative humidity reduce the vapor pressure gradient between the ambient air and the moist skin (2). When this occurs the ability to dissipate heat through evaporation diminishes. Under these circumstances, sweat will tend to more readily drip off the surface of the skin without evaporating. When evaporation is reduced then excessive hyperthermia can more easily result which can then lead to performance decrements.

**Theoretical Benefits of Precooling Before Exercise**

Given the challenges of exercising in hot and humid environments detailed above, cooling strategies have been studied in order to limit the premature termination of exercise and performance decrements associated with exercise in the heat. Some studies have shown that starting exercise with a lower core temperature delays the onset of fatigue and ultimately aids in performance (42, 51, 86, 87), possibly because of a delay in achievement of a critical core temperature. Recently, several studies have investigated the effects of cooling the human body in a hot environment on exercise performance, but the effects of cooling in general have been studied for over 7 decades (7). Bazett was one of the first researchers to assess the effects of cooling on circulatory adjustments and oxygen consumption, but not necessarily on performance. The precursor to precooling and exercise may have come from the work of Webb (80) and Veghte (79). Both researchers assessed the effects of water submersion and an air cooling suit on exposure time in the heat and found that when exposed to a hot ambient temperature (41 °C), tolerance time was inversely related to the initial core temperature (79, 80).
Additionally, one of the first studies investigating precooling and exercise was conducted by Bergh and Ekblom (8). The researchers precooled their participants by having them swim in cold water (13-15 °C) for 15-25 min to reach an esophageal temperature ranging from 35.8 °C – 34.9 °C (8). Following the precooling participants were asked to complete a fatiguing arm and leg exercise until fatigue. Participants’ time to exhaustion averaged 6 min (8). Rather than increasing time to exhaustion, they found that precooling actually decreased time to exhaustion. The researchers concluded that the decrease in time to exhaustion was caused by the decrease in peak oxygen consumption (8). However, there were several methodological concerns in this early precooling study that may have confounded the results. Participant’s core temperatures were considered hypothermic and could have contributed to early fatigue because of altered substrate utilization for thermogenesis. Furthermore, swimming for 15-25 min may have prematurely fatigued participants and had an effect on the time to exhaustion test. Finally, the low body temperatures during the precooling trial may have negatively affected muscle function (12).

Several of the early and recent precooling studies have used air cooling as the intervention. Schmidt and Bruck (73) used an environmental chamber that was set at 0 °C for a precooling intervention before exercise. During the study participants were cooled for 100 minutes while sitting in the cold room (73). Following the cooling session, participants were warmed to increase thermal comfort and decrease shivering (73). Following warming, participants performed a graded exercise test until exhaustion on a cycle ergometer in a temperate environment (18 °C). Time to exhaustion was extended after cooling relative to no cooling (73). Similarly, 2 other studies conducted with the same cooling and warming protocols observed similar performance benefits from precooling (39, 68). Lee and Haymes (47) assessed
the effects of cooling participants in a room set at 5 °C for a total of 40 minutes without a warming procedure. Following the precooling intervention participants ran on a treadmill at 82% of \( \dot{V}O_{2\text{max}} \) until exhaustion. Like the other precooling studies mentioned so far, participants ran longer before reaching exhaustion after precooling. Investigators attributed the improvement to an increased capacity for heat storage (47).

As previously mentioned, some researchers have also assessed the effects of water immersion as a precooling strategy. The rationale for using water immersion instead of air exposure is that heat loss by way of water immersion is 25 times greater when compared to air at the same temperature (51, 76). Drust et al. (19) used precooling by exposing participants to a cold shower until core temperature decreased 0.6 °C (19). The reduction in core temperature resulted in better performance times in soccer specific timed sprints (19). In addition to improving sports performance, water immersion has been used to cool firefighters during active duty (42). Firefighters were cooled either by immersing their hands and forearms or immersing their feet and lower legs in cool water (17 °C) for 20 minutes, followed by a test of work tolerance time in a 36 °C environment (42). Although not significantly different, the researchers observed 5 min (24%) longer exposure time in the heat following leg submersion compared to forearm submersion (42). The authors concluded that cooling could have prolonged the tolerance time by decreasing thermal strain, and improving the participants’ perception of effort.

**Precooling Effects on Heat Storage during Exercise**

One advantage of cooling the body before exercise is the increased capacity of the body to store heat (51, 69). Heat storage is dependent upon body size, exercise intensity and metabolic heat production (51, 69). Furthermore, as mentioned earlier the ambient temperature can have an impact on the heat storage capacity of the exercising human (51, 86, 87). Cooling
the body before exercise increases heat storage capacity and thereby may theoretically delay the attainment of a critical core temperature. It is likely that the warmer the ambient temperature the bigger the potential effect of cooling on increasing heat storage capacity.

Some of the early cooling studies calculated heat storage. Olschewski and Bruck (68) calculated heat storage following a testing protocol which used a graded exercise test to fatigue. The amount of heat storage ranged from 380 kJ/m² for precooling to 263 kJ/m² for the control condition (68). Since the 1988 study by Olschewski and Bruck (68) many researchers have used heat storage as a way to express the importance of precooling by calculating heat storage at specified time intervals (e.g., 10 min) throughout exercise. These studies have shown heat storage to be significantly higher under cooling conditions, but this did not result in a greater exercise capacity in the heat (51). The researchers concluded that the high heat storage could be caused by low muscle temperature, which could negatively impact muscle performance since muscle function is impacted by temperature (12). Apparently, for precooling to be effective, a balance must be achieved between beneficial cooling effects on circulatory and thermoregulatory outcomes and negative effects on muscle contractile function.

Lee and Haymes (47) reported heat storage as a rate per unit time and found that heat storage was higher immediately after precooling, but during exercise it decreased substantially. Under temperate conditions (24 °C, 51% RH) heat storage was 144 W/m² for the precooling trial and 119 W/m² for the control trial at the end of exercise (47). The researchers reported that the participants had greater sweat evaporation following precooling (75%) when compared to the control (57%) and worked at a higher intensity following precooling. These results suggest that following precooling participants experienced more economical heat dissipation through evaporation following the precooling treatment (47). In the same study heat storage was also
measured during exercise in a hot environment (32 °C, 60% RH), and investigators found results similar to those in the temperate conditions in terms of heat storage and sweating following the precooling treatment. A similar study conducted by Booth et al. (9) also found that after 30 minutes of running the precooling group increased heat storage from 113 W/m² before precooling to 250 W/m² following precooling (9). The researchers concluded that precooling can aid performance during endurance exercise by reducing thermal strain while increasing the capacity for heat storage. Furthermore, the researchers noted that after precooling and during the first 20 minutes of exercise the rate of increase in skin temperature was high (9). Marino (51) noted that in the majority of cooling studies heat storage is higher in precooled participants than participants who are not precooled. Furthermore, Marino noted that it does not seem to matter what type of precooling intervention (water immersion, cool air, ice packs) is utilized, the results are similar (51). This point is further highlighted by a study conducted by Kay et al. (44). The researchers assessed cooling the skin followed by measurement of skin and core temperatures during exercise in warm and humid conditions (44). Core temperature was lower during exercise after precooling and the authors attributed this to a favorable thermal gradient because of the reduction in skin temperature associated with precooling (44). Reducing the skin temperature and concomitantly attenuating the rise in core temperature reduced the likelihood of reaching a critical core temperature (44). These studies support the notion that precooling increases heat storage capacity and thereby delays the time to reach a critical core temperature, which ultimately may increase time to exhaustion and improve performance.

**Precooling Effects on Exercise Performance**

Several exercise modalities have been used to assess the effects of precooling on exercise performance. Marsh and Sleivert (53) assessed the effects of 30 min of precooling before 70
minutes of aerobic and anaerobic cycling efforts in 29 °C and 80% relative humidity. Participants cycled with intermittent sprints throughout the cycling bout. Time to exhaustion increased 3.3% following the 30-min precooling treatment compared to a control treatment without precooling. The investigators concluded that the improvements may have been caused by vasoconstriction of the periphery, which facilitated venous return and resulted in an increase in central blood volume (53). The researchers speculated that increasing central blood volume increased blood flow available to working skeletal muscle for oxygen delivery and metabolite clearance, which could enable maintenance of a higher intensity during exercise (53). This hypothesis is supported by studies of short bouts of high intensity exercise that showed no effect of precooling (43, 50, 75). Such short bouts of high intensity exercise would not rely as heavily on oxidative metabolism and clearance of muscle metabolites. Cheung and Robinson (11) assessed the effects of upper body precooling on repeated sprint performance. The protocol consisted of 30 min of cycling in 22 °C and 40% relative humidity at 50% VO_{2peak} with 10-s bouts of high intensity sprints interspersed every 5 min (11). Precooling was administered via 5 °C fluid circulated through a garment placed on the upper body for 20 min. Precooling significantly decreased mean skin temperature and heart rate following the cooling treatment, but did not have an effect on overall peak power (11). Likewise, upper body precooling of male rugby players with an ice jacket applied for 10 min before a sprinting protocol in 30 °C and 60% relative humidity failed to affect peak power, mean power, skin temperature, rectal temperature, heart rate, sweat loss, lactate concentration, and rating of perceived exertion during the intermittent sprint protocol (20). Apparently, wearing an ice vest longer than 10-20 minutes is needed to have an effect in the heat, or it may simply be that precooling is not efficacious for
anaerobic sprint events because the purported ergogenic effects are not relevant to short-duration, sprint exercise.

While precooling has limited benefit for short duration exercise, because of its potentially positive effects on cardiovascular and thermoregulatory function, it may be beneficial in prolonged exercise during which increases in body temperature can influence exercise performance and work capacity. Continuous cooling via fan airflow during exercise has been shown to attenuate CV drift and its negative impact on VO$_{2\max}$ (82). However, whether precooling can have a similar effect is unknown because this has not been tested. Furthermore, limited research has investigated the effects of precooling during an active warm-up even though many athletes consider a proper warm-up an important way to optimize neuromuscular function and psychological mind set (4). Only 2 studies have investigated the effect of precooling during an active warm-up before running. Both studies showed that ice vest precooling administered during a warm-up, when compared to a control condition without precooling, blunted the rise in core temperature and heart rate, decreased time to complete a set distance, and lowered perceptual measures during the exercise (4, 40). Despite these positive findings, extrapolation of the data to activities other than 5-km running is tenuous. For example, completion of a 16.1-km (10-mile) cycling time trial requires more time and relies more heavily on a slightly different muscle mass than a 5-km running race, and therefore the effects of precooling on such an event remain speculative and uncertain. Moreover, several elite athletes and professional cycling teams precool the upper body during an active warm-up prior to time trial performances, so the efficacy of this practice needs empirical testing.
Summary

This review has shown evidence that endurance exercise in the heat can have an effect on exercise performance. The decline in performance can be attributed to increased cardiovascular strain (partly because of CV drift), neuromuscular fatigue and attainment of a “critical core temperature”. Studies mentioned in this review have argued that precooling could diminish the negative effects on performance when exercising in the heat. While precooling has been shown to have limited or no impact on short-duration sprint performances, several studies have found improved performance during prolonged exercise in the heat following precooling. These improvements are likely related to effects on cardiovascular and thermoregulatory function because of increased heat storage capacity associated with precooling. Precooling also may positively impact phenomena such as CV drift, which have been shown to negatively affect VO$_{2\text{max}}$, but this has not been tested.
References


CHAPTER V:
CONCLUSIONS

Competitive cycling requires maintenance of a high rate of energy expenditure, which results in elevated metabolic heat production and concomitant heat storage, especially when the exercise occurs in a hot environment and dry heat loss is compromised. Elevated heat storage results in a rise in core temperature that may limit exercise capacity and performance because of potential effects on central drive and/or oppressive cardiovascular strain, which can be manifested as cardiovascular drift (CV drift). Cooling before exercise, known as precooling, has been shown to blunt the rise in core temperature and the decrements in performance in some circumstances, but this has been demonstrated most commonly in studies that did not involve an active warm-up. The studies that investigated precooling and included a warm-up involved running, so extrapolation of findings to cycling is unsubstantiated.

Therefore, the primary purpose of this series of studies was to determine if precooling impacts cycling performance in the heat. Two studies and literature review were completed. The first study assessed the effects of a 20-min precooling intervention during a warm-up on a subsequent simulated 16.1-km cycling time trial in 30 °C. The second study assessed the effects of whole body precooling on CV drift and \( \dot{V}O_2_{\text{max}} \) during subsequent cycling in 35 °C. The participants were cooled for 20-min, then performed a 45 min submaximal cycling bout at 60% \( \dot{V}O_2_{\text{max}} \). Following the 45-min submaximal effort participants completed a graded exercise test (GXT) to achieve \( \dot{V}O_2_{\text{max}} \). \( \dot{V}O_2_{\text{max}} \) also was measured after 15 min of cycling in 35 °C in order
to measure $\dot{V}O_{2\text{max}}$ during the same time interval that CV drift occurred. The literature review found several studies that reported improved performance during prolonged exercise in the heat, following a precooling intervention. However, based on the literature reviewed, precooling does not affect performance of short bouts of high intensity cycling or running.

Precooling with a cooling vest and head/neck wrap blunted the rise in mean body temperature ($T_b$) and facilitated better thermal comfort during the active warm-up, but these outcomes were not sustained during the time trial. Finishing times for the 16.1-km time trial after precooling were non-significantly 2.2% shorter relative to no precooling. Likewise, precooling did not affect the magnitude of CV drift or subsequent reduction in $\dot{V}O_{2\text{max}}$ after 45 min of cycling in 35 °C.

While the studies in this dissertation do not support the use of precooling under the circumstances tested to optimize performance, further research is warranted to determine if more aggressive precooling protocols impact subsequent cycling performance in the heat.
References


