PRECOOLING AND WARM-UP EFFECTS ON TIME TRIAL CYCLING PERFORMANCE DURING HEAT STRESS

by

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ABSTRACT

The aim of this study was to investigate the separate and combined effects of precooling and warm-up on a subsequent cycling time trial in a hot environment. Nine healthy men (mean±SD age=24±5 years; body mass=74.7±4.5 kg; height=171.4±7.7 cm; body fat=12.9±5.2%) completed 3 simulated 16.1-km time trials on a cycle ergometer in a hot environment (~33 °C, 45% relative humidity) after: 1) 20 min of fluid ingestion (10 °C) followed by 30 min of ice-slurry ingestion (-1 °C) coupled with ice-vest (PREC), 2) 30 min of ice-slurry ingestion coupled with ice-vest followed by 20 min of warm-up including ice-slurry and ice-vest (COMBO), 3) 30 min of fluid ingestion (10 °C) followed by 20 min of warm-up (WU). At baseline, rectal temperature (T_{re}), mean skin temperature ($\overline{T}_{sk}$), and mean body temperature ($\overline{T}_{b}$) were similar among treatments (all P>0.05). After treatment administration and before the start of the time trial, T_{re} was lower in PREC (36.1±0.3) and COMBO (36.9±0.4) compared to WU (37.6±0.2) (all P<0.05). $\overline{T}_{sk}$ and $\overline{T}_{b}$ were all lower in PREC than COMBO and WU, and were lower in COMBO than WU (all P<0.05). T_{re} remained lower in PREC than WU throughout exercise and was lower in PREC than COMBO for the first 12 km (all P<0.01), while T_{re} in COMBO remained lower than WU for the first 4 km. $\overline{T}_{sk}$ during PREC was lower than COMBO at 4 and 8 km and lower than WU at 0 and 4 km, while during COMBO it was lower than WU at 0 and 4 km (all P<0.05). Heart rate (HR) at baseline was lower in PREC than COMBO and WU (68±10, 106±12, 101±13 beats/min, respectively; P<0.001). During exercise, HR increased similarly among all treatments throughout exercise (all P>0.05). Local sweat rate (SR) was lower in PREC than COMBO and WU for the first 4 km (P <

0.05), but whole-body SR was not different among treatments (all P>0.05). Performance times were not different among treatments (PREC: 31.96±2.05 min; COMBO: 32.64±2.90 min; WU: 33.09±3.09 min; P>0.05). Despite mitigating thermal strain during exercise, precooling alone, or combined with warm-up, did not result in improved performance of a 16.1-km simulated cycling time trial.

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CHAPTER 1

INTRODUCTION

Competitive cycling competitions require a high level of cardiorespiratory endurance and maintenance of high intensities. Races often feature long distances ranging from 5 km to 300 km covered within multiple stages. Heart rate (HR) monitoring during long cycling races has revealed that performance intensity varies between 70%-90% maximal oxygen uptake ($\dot{V}O_{2max}$), with high contributions of aerobic and anaerobic metabolism (Luciá *et al.*, 1999; Fernández-García *et al.*, 2000; Neumayr *et al.*, 2002). Cycling in temperate climates may elevate core temperature (T_c) 1-2 °C depending on exercise intensity (Nybo *et al.*, 2001; Drust *et al.*, 2005; Hausswirth *et al.*, 2012). Elevated T_c results in performance impairments related to non-thermoregulatory mechanisms, including cardiovascular and neurological dysfunction (Abbiss & Laursen, 2005). To further complicate matters, cycling competitions often take place in a hot environment, resulting in increased thermal strain because of elevated heat storage. Elevated body temperature exacerbates cardiovascular strain because of a competition in blood flow distribution between working skeletal muscle and skin for heat dissipation (Rowell, 1974; Johnson, 2010). This can momentarily reduce $\dot{V}O_{2max}$ (Arngrímsson *et al.*, 2003; Wingo *et al.*, 2005), and increase muscular metabolic stress (Febbraio *et al.*, 1994), resulting in performance decrements during cycling while hyperthermic (Febbraio *et al.*, 1994; Galloway & Maughan, 1997; González-Alonso *et al.*, 1999).

Precooling (Marino, 2002) is used by some athletes just prior to a competitive event. Lowering the initial T_c and/or mean skin temperature ($\overline{T}_{sk}$) prior to exercise via precooling has

been shown to enhance exercise time to exhaustion (Lee & Haymes, 1995; González-Alonso *et al.*, 1999; Uckert & Joch, 2007) and distance covered in a given period of time (Booth *et al.*, 1997; Kay *et al.*, 1999; Quod *et al.*, 2008) under hot conditions. Some investigators have speculated that a high critical T_c (~ 40 °C) is a major limiting physiological factor related to impaired performance in hyperthermia, regardless of the baseline T_c at the start of exercise (González-Alonso *et al.*, 1999) and the rate of T_c rise (MacDougall *et al.*, 1974). The decrease in the initial T_c after precooling may explain the mechanism underlying enhanced performance in hyperthermia, through widening the capacity for heat storage and, consequently, increasing the time to reach a critical T_c (Marino, 2002). Alternatively, decreased $\overline{T}_{sk}$ after precooling facilitates a greater thermal gradient between core and skin such that heat storage capacity is increased, thereby attenuating cardiovascular and thermoregulatory strain (Olschewski & Brück, 1988; Lee & Haymes, 1995; Booth *et al.*, 1997).

How is precooling carried out? Several methods have been investigated. Despite the positive impact of some methods, such as water immersion, on body temperatures and performance, implementation of such protocols in a field or sports setting is impractical. This has prompted some to explore more practical alternatives, such as ice-slurry ingestion and use of ice vests/wraps. These methods are practical and easily accessible. Furthermore, ice-slurry ingestion has been shown to reduce T_c prior to exercise and improve cycling (Ihsan *et al.*, 2010; Ross *et al.*, 2011) and running (Siegel *et al.*, 2010) performance.

Notwithstanding the reported benefits of precooling, some precooling protocols have been either detrimental (Bergh & Ekblom, 1979*a*), or ineffective (Ross *et al.*, 2011) to exercise performance. Bergh and Ekblom (1979) indicated that exercise time to exhaustion was reduced after lowering esophageal temperature (T_{es}) from 38.4-34.9 °C, possibly because of reduced peak

oxygen uptake (Bergh & Ekblom, 1979*a*). Furthermore, reduced muscle temperature has also been implicated in performance decrements associated with precooling (Webb & Annis, 1968). Taken together, the evidence touting the benefits of precooling combined with the evidence citing its detriments suggest that precooling provides positive central and peripheral circulatory benefits related to thermoregulation and maintenance of muscle blood flow, but this may occur at the expense of local muscle function. Such muscle function includes twitch tension, maximal force, and rate and velocity of muscle contraction, all of which have been reported to decrease with reduced local temperature (Binkhorst *et al.*, 1977; Sargeant, 1987; Ranatunga *et al.*, 1987; Bigland-Ritchie *et al.*, 1992; Racinais & Oksa, 2010; Cheuvront *et al.*, 2010).

In order to optimize the aforementioned aspects of muscle function prior to exercise, many athletes incorporate a warm-up prior to training and competition. There is a discrepancy about the influence of warm-up on endurance performance, as some studies have reported an improvement (Atkinson *et al.*, 2005), and some an impairment (Uckert & Joch, 2007). Warm-up is suggested to improve performance by increasing muscle temperature in conjunction with decreased muscle and joint stiffness, enhanced nerve transmission, increased energy utilization, and/or elevated baseline $\dot{V}O_2$ (Bishop, 2003). On the other hand, some studies have demonstrated impaired endurance performance through decreased heat storage capacity secondary to elevated initial T_c following a warm-up (Gregson *et al.*, 2002; Uckert & Joch, 2007).

In light of the preceding discussion, it stands to reason that combining precooling and warm-up may result in the benefits of each strategy while avoiding the potential negative consequences. However, to our knowledge, the combination of precooling and warm-up effects on exercise has only minimally been investigated, with mixed results. For instance, many studies

that have investigated the precooling effect on endurance performance in hot conditions, and found improved performance, did not include a warm-up session (Lee & Haymes, 1995; Booth *et al.*, 1997; González-Alonso *et al.*, 1999). Perhaps precooling simply masked the negative effects of a lack of warm-up. Said another way, it may be that precooling fostered positive thermoregulatory responses at the expense of muscle function optimization that might have occurred via a warm-up. One study evaluated the separate effects of precooling and warm-up and found that precooling did not prevent an increase in T_c during warm-up, but nonetheless running time to exhaustion in the heat was improved relative to a trial with warm-up alone, which impaired performance (Uckert & Joch, 2007). In other studies, simultaneous cooling using a cooling vest or head cooling and warm-up resulted in improved 5-km running (Arngrïmsson *et al.*, 2004), but not cycling time trial performance (Levels *et al.*, 2013). Unpublished observations from our laboratory have shown that precooling using gel wraps during an active warm-up in 35 °C did not improve simulated 16.1-km cycling time trial performance. $\overline{T}_{sk}$ was ~2 °C lower, and T_c was unchanged at the end of warm-up with precooling compared to no precooling just prior to commencement of the time trial, but this had no effect on the time to complete 16.1 km. None of the aforementioned studies involved reduced T_c prior to the performance test, which may explain a lack of or small effect of precooling given that reducing T_c has been posited as the main mechanism by which precooling enhances performance (Marino, 2002). However, Kay *et al.* (1999) have shown that reducing T_c prior to exercise is not essential to improve performance if precooling sufficiently reduces $\overline{T}_{sk}$.

Taken together, these studies suggest that the optimal combination of precooling and warm-up remains unclear, especially for cycling. As suggested above, precooling and warm-up may act reciprocally—precooling could possibly mask the detrimental effect of not warming up,

and vice versa, warming up may mask the negative effect of not having precooled. The combination of precooling and warm-up may have an additive, positive impact on endurance cycling time trial performance in a hot environment by reducing thermal strain (from precooling) and accentuating skeletal muscle function (from warming up). This could be attained by reducing T_c while maintaining optimal muscle temperature.

Purpose

In light of the aforementioned background information, the purpose of this study is to investigate the combined and separate effects of precooling and warm-up on subsequent cycling time trial performance in a hot environment.

Hypotheses

Hypothesis 1. Precooling by ice-slurry ingestion while wearing an ice vest will result in improved 16.1-km cycling time trial performance in a hot environment (33 °C) relative to warm-up alone.

Hypothesis 2. Precooling by ice-slurry ingestion while wearing an ice vest followed by warming up for two sets of 3 min at 25% $\dot{V}O_{2max,}$ 5 min at 60% $\dot{V}O_{2max,}$ and 2 min at 80% $\dot{V}O_{2max}$ while wearing an ice vest will improve subsequent 16.1-km cycling time trial performance relative to either condition alone (precooling and warm-up).

Significance of the Study

Data from the proposed study will contribute new information to the existing body of knowledge regarding the combined and independent effects of precooling and warm-up on cycling time trial performance. Results will ascertain whether precooling or warm-up is superior in regards to affecting cycling time trial performance, or whether their effects are additive, or whether perhaps they are ineffective. This information will be valuable for athletes and coaches

to consider when strategizing ways to optimize training for, and performance in, endurance events.

CHAPTER 2

REVIEW OF LITERATURE

The literature for this review was selected from relevant journals from exercise physiology. Related key words were used in several data bases such as PubMed, Science Direct, and EBSCO. The reference lists of related articles were also used to find additional relevant articles.

Prolonged competitive cycling requires performance of a high exercise intensity 70-90% $\dot{V}O_{2max}$ (Luciá et al., 1999; Lucia et al., 2001; Rodríguez-Marroyo et al., 2003). Cycling events that occur in hot environments elevate stress on cyclists and can result in premature exercise impairment (Roberts & Wenger, 1979). In light of the impact of heat stress on cycling performance, this review is focused on cycling and the methods used to improve its performance, such as countermeasures to heat stress. Studies detailing the high intensities at which cyclists perform races are reviewed first in order to show the high level of exertion required to complete the different stages of cycling races. Since many cycling races take place in hot environments, the effect of heat stress on exercise performance and the related physiological factors that limit exercise are reviewed next, along with the mechanisms through which heat stress impairs performance. Then countermeasures used to alleviate the negative effects of heat stress, such as precooling maneuvers, and the mechanisms by which precooling can enhance performance during subsequent cycling in the heat, are reviewed. Finally, warm-up as a technique used prior to exercise is reviewed in the context of the hypothetical combined effect of precooling and warm-up and how performing the two together may be additive in terms of beneficial effects.

Cycling races and performance

Professional road cycling is a remarkable endurance sport, composed of multiple-stage competitions of variable distance (5-300 km) and intensities. These races involve flat stages, individual time trials, and high mountain ascents. Heart rate (HR) monitoring in two professional stage cycling races revealed that the proportion of the total race time spent at a low (< 70% $\dot{V}O_{2max}$) intensity, moderate (70-90% $\dot{V}O_{2max}$) intensity, and high intensity (>90% $\dot{V}O_{2max}$) was 70%, 25%, and 5%, respectively (Fernández-García *et al.*, 2000). Flat stages are covered predominantly at low-moderate intensity. During high mountain ascents, cyclists are required to overcome high forces resulting from a 5-10% mean gradient, in addition to the rolling resistance from the friction between the bicycle and the rough surface, together presenting multiple forces that require cyclists to ride at a high level of exertion, estimated at close to 90% $\dot{V}O_{2max}$ (Luciá *et al.*, 1999; Lucia *et al.*, 2001; Rodríguez-Marroyo *et al.*, 2003). In staged races, the time trial is considered a crucial stage in the overall outcome of the race, in which cyclists have to ride a fixed distance within the shortest time possible (Lucia *et al.*, 2001; Abbiss & Laursen, 2005). Time trial performance is most often performed individually, and demands maintenance of a high work rate. This is highlighted by some HR data that have shown that cyclists performed an entire time trial at an intensity close to the anaerobic threshold (~ 90% $\dot{V}O_{2max}$) (Padilla *et al.*, 2000; Lucia *et al.*, 2001).

Prolonged high intensity during time trials results in elevated physiological stress (van Ingen Schenau *et al.*, 1992; de Koning *et al.*, 1999; Abbiss & Laursen, 2005). For instance, high cardiovascular strain is evidenced by maintenance of high to near maximal HR (HR_{max}) during time trials (76%-94% HR_{max}), depending on the distance covered in the trial (Palmer *et al.*, 1994, 1996). Furthermore, professional and amateur cyclists have extraordinarily high $\dot{V}O_{2max}$ values

(70-80 ml·kg^{-1}·min^{-1}) (Coyle *et al.*, 1991; Luciá *et al.*, 1999; Fernández-García *et al.*, 2000), and maximal power outputs (400-500 W) (Terrados *et al.*, 1988; Wilber *et al.*, 1997; Padilla *et al.*, 1999). Time trials require cyclists to maintain an extremely high percentage of $\dot{V}O_{2max}$ (e.g., 90% $\dot{V}O_{2max}$) through the majority of the race (Coyle *et al.*, 1991; Luciá *et al.*, 1999; Fernández-García *et al.*, 2000); power outputs at these intensities approximate 350-400 W for high-level cyclists (Padilla *et al.*, 1999, 2000). Sustaining high power output during time trials exacerbates cardiovascular and thermoregulatory strain, and may eventually lead to decrements in performance (Hunter *et al.*, 2002; Gonzalez-Alonso, 2003).

Impact of heat stress on exercise performance

As mentioned above, high intensity cycling in thermoneutral conditions can result in decrements in performance. For instance, cycling time trials in thermoneutral conditions have been shown to increase core temperature (T_c) 1-2 °C depending on exercise intensity (Nybo *et al.*, 2001; Drust *et al.*, 2005; Périard *et al.*, 2011; Hausswirth *et al.*, 2012); this results in elevated skin blood flow (Périard *et al.*, 2011). The increased T_c with a concomitant increase in skin blood flow exacerbates cardiovascular strain via reduced central venous pressure and subsequent reduced stroke volume and increased HR, all of which can impair performance (Roberts & Wenger, 1979).

The physiological responses resulting in elevated cardiovascular strain are intensified when the time trial takes place in a hot environment. Elevated core (T_c) and/or mean skin ($\overline{T}_{sk}$) temperatures are accompanied by impaired aerobic and repeated sprint exercise performance in the heat (Drust *et al.*, 2005; Cheuvront *et al.*, 2010). Likewise, cycling time to exhaustion and self-paced time trial performance have been shown to be attenuated in environmental temperatures higher than neutral (21 - 26 °C) (Schlader *et al.*, 2011). For example, cycling to

exhaustion was differentially influenced by graded ambient temperatures (3.6, 10.5, and 30.2 °C) with the shortest duration reported at the highest temperature (Galloway & Maughan, 1997). Moreover, cycling in a hot condition resulted in reduced time to exhaustion accompanied with higher T_c by 2 °C compared to cycling in a thermoneutral condition (Nybo & Nielsen, 2001*a*).

Studies that have manipulated initial T_c and $\overline{T}_{sk}$ before exercise using cooling and warming maneuvers indicate that total work and time to exhaustion are inversely related to the T_c and $\overline{T}_{sk}$ values before commencing cycling in the heat (Schmidt & Brück, 1981; Kruk *et al.*, 1990; González-Alonso *et al.*, 1999). Self-paced cycling, on the other hand, generally differs from that at a fixed intensity because the cyclist can adjust pace and power output according to feedback that helps regulate the rise in T_c and better maintains thermal homeostasis (Tucker *et al.*, 2004; Marino, 2004; Schlader *et al.*, 2011). Time trial cycling performance, while self-paced, is similar to cycling to exhaustion in that it has been shown to be attenuated in hot compared to thermoneutral environmental conditions, due to factors related to thermoregulatory rather than metabolic factors (Tatterson *et al.*, 2000; Altareki *et al.*, 2009; Abbiss *et al.*, 2010; Peiffer & Abbiss, 2011). For instance, central drive is down-regulated in response to elevated thermal strain (Nybo & Nielsen, 2001*a*; Nielsen & Nybo, 2003). Clearly, heat stress has a negative impact on cycling performance. It is important to understand the physiological factors limiting exercise in the heat in order to design countermeasures. Therefore, literature related to these factors will be reviewed in the next section.

Physiological factors limiting exercise in the heat

Thermoneutral conditions are considered compensable in that heat dissipation mechanisms enable maintenance of thermal homeostasis (Nybo *et al.*, 2001; Drust *et al.*, 2005; Périard *et al.*, 2011; Hausswirth *et al.*, 2012). Generally, performance impairments under such

conditions are thereby the result of issues unrelated to thermoregulation, such as failure of central and peripheral neural drive, diminished response of muscles to neural stimulation (e.g., action potentials), and/or impaired excitation-contraction coupling-related mechanisms (Abbiss & Laursen, 2005). In contrast, exercise in the heat at a high intensity may result in uncompensable heat stress in which thermal homeostasis cannot be achieved and T_c rises over time. Under such circumstances, high body temperature becomes predominant in limiting performance (Nielsen *et al.*, 1993; Kenefick *et al.*, 2007).

How does elevated body temperature ultimately limit exercise during heat stress? Different hypotheses have been explored. One hypothesis asserts that a critical T_c exists at which people voluntarily stop exercise. This has been illustrated in heat acclimation studies which showed a two-fold increase in exercise time to exhaustion in the heat after acclimation, concomitant with lower HR and T_c, and higher sweat rate; however, exhaustion occurred at a similar T_c as before acclimation, but achievement of that T_c took longer because of a slower rate of rise (Nielsen *et al.*, 1993, 1997). To further investigate the principle of the critical T_c, González-Alonso *et al.* (1999) induced different initial body temperatures through 30 min of water immersion and tested cycling time to exhaustion in the heat. The time to exhaustion was inversely related to the initial $\overline{T}_b$, and fatigue occurred at a similar esophageal temperature (T_{es}) regardless of the initial hyperthermia level. Furthermore, different rates of heat storage did not alter the critical body temperature at which subjects reached exhaustion (González-Alonso *et al.*, 1999). More recently, in an attempt to avoid the confounding variables resulting from traditional heating procedures which require a long time to attain a certain initial $\overline{T}_b$, Walters et al. (2000) used microwave irradiation to rapidly heat rats within 3-8 min to induce different initial rectal (T_{re}) and hypothalamic temperatures (T_{hyp}). Exercise time to exhaustion was negatively

correlated with the initial T_{re} and T_{hyp}, and temperature parameters were similar at exercise exhaustion regardless of the initial T_{re} and T_{hyp}. These results further support the concept of a critical T_c (Walters *et al.*, 2000).

The critical T_c has been shown to range from 38-40 °C suggesting a different hyperthermia tolerance among active subjects (González-Alonso *et al.*, 1999). Treating moderately trained subjects with water hyperhydration, glycerol hyperhydration, and euhydration resulted in exercise exhaustion at ~38.5 °C for all treatments (Latzka *et al.*, 1998). In contrast, high level cyclists reached exhaustion at ~40 °C regardless of the initial T_c prior to starting exercise (González-Alonso *et al.*, 1999). Others have found similar results albeit with different values for the critical T_c. For instance, Cheung and McLellan (1998) compared time to exhaustion between highly- and moderately-trained subjects and found that the highly-trained group reached exhaustion at ~39.2 °C while the moderately-trained group reached exhaustion at ~38.8 °C, suggesting that training status influenced hyperthermia tolerance. Also, human-related factors such as motivation, training and hydration status may play a role in the variation seen in hyperthermia tolerance among individuals (Nybo, 2010).

Some have challenged the hypothesis of a critical T_c threshold limiting exercise performance in the heat. Although the critical T_c is suggested to be ~ 40 °C, heating sedated, well-hydrated, unacclimatized men resulted in increased esophageal temperature to 41.6-42 °C without sequelae (Bynum *et al.*, 1978). Furthermore, heat-acclimatized subjects completed an outdoor running race with T_c > 40 °C (Byrne *et al.*, 2006). Ely et al. (2009) have shown that the velocity of the final 600 m during an 8-km running trial was not different among runners with T_c > 40 °C or < 40 °C, which disputes the suggested 40 °C-performance limiting core temperature. Skin temperature varied among treatments, so it may be the combination of elevated core and

skin temperatures that results in impaired performance in the heat, rather than a critically elevated core temperature per se (Ely *et al.*, 2009).

Mechanisms of exercise impairment in the heat

The physiological mechanisms underlying the exercise impairment during heat stress are not completely understood. Research tends to attribute this impairment primarily to central fatigue and cardiovascular strain developed during exercise-induced hyperthermia (Nybo, 2010; Cheuvront *et al.*, 2010). Both systems contribute to fatigue during exercise heat stress dependent upon exercise intensity. Central nervous system-related fatigue seems to be involved during low-moderate intensity prolonged exercise during which endogenous heat generation combined with environmental heat stress results in elevated core and brain temperatures to ~ 40 °C (González-Alonso *et al.*, 1999; Nybo *et al.*, 2002*b*), with no concomitant reduction in cardiac output, leg blood flow, and substrate utilization or availability (Nielsen *et al.*, 1990, 1993). Conversely, cardiovascular-related reduction in oxygen delivery and the subsequent reduced $\dot{V}O_{2max}$ becomes evident during high-intensity exercise in the heat (Nybo *et al.*, 2001; González-Alonso *et al.*, 2008; Nybo, 2010).

Central-related factors

High brain temperature associated with increasing T_c during exercise in the heat has been implicated as the limiting physiological factor leading to exercise impairment (Nielsen & Nybo, 2003). Central fatigue during hyperthermia has been proposed based on a sequence of maximal voluntary contraction (MVC) tests following exercise in the heat (Nybo & Nielsen, 2001*a*). Nybo and Nielsen (2001*a*) had subjects exercise until exhaustion in hot and thermoneutral conditions. Sustained knee extension MVC force reduction after exercise hyperthermia was more pronounced than during normothermia. The percentage of voluntary activation from total MVC

and electrical stimulation was lower after exercise in the heat compared to exercise in a thermoneutral environment; however, the summation of force generated from MVC and electrical stimulation was not different between the two trials. In addition, the same testing procedure using hand grip exercise (non-exercised muscles) resulted in the same outcome as that observed during knee extension (primary working muscles). The reduced voluntary force generation and voluntary activation percentage indicate that the impaired performance in the heat was due to upstream central fatigue (Nybo & Nielsen, 2001*a*).

Cerebral electroencephalogram (EEG) activity, expressed by the power spectrum ratio of α/β where an elevated ratio represents a suppressed arousal, has been used to evaluate the alteration in central activity during exercise in the heat. EEG activity was measured on the frontal cortical brain of cyclists during cycling in hot and cool environments. Cycling to exhaustion in the hot condition resulted in an elevated α/β index compared with no significant elevation during the cool trial (Nielsen *et al.*, 2001). Altered α/β index was also observed in 3 cortex positions after exercise hyperthermia compared to no change after normothermia (Nybo & Nielsen, 2001*b*). Both studies showed a linear relationship between α/β index and the rise in T_c during exercise hyperthermia. Further evidence to support the idea of high brain temperature as a limiting factor during exercise comes from research demonstrating attenuated running performance in goats after elevating hypothalamic temperature (Caputa *et al.*, 1986). Based on these findings, investigators hypothesize that inhibitory signals arise from temperature-sensitive areas in the hypothalamus and attenuate motor activity when body temperature rises (Nybo & Nielsen, 2001*a*; Nybo, 2012). Other hypothetical central mechanisms inducing fatigue in hyperthermia were drawn from studies investigating subjects with multiple sclerosis (a heat sensitive pathology) who were heated passively or through exercise and experienced debilitating

premature fatigue—presumably, due to heat reaction blockade of action potentials in demyelinated neurons (Marino, 2009, 2011).

Other areas of the brain in addition to the hypothalamus also may be affected. Brain cells have been shown to be highly heat sensitive. Brain mitochondria in mice were damaged when heated to 41 °C and were essentially destroyed when heated to 42 °C (Gwóźdź *et al.*, 1978); however, the temperature needed for inducing lethal cell lesions appears to be substantially greater in other tissues (Jung, 1986). Brain temperature regulation has been proposed to be based on local heat production, local blood flow, and temperature of the arterial blood supplying the brain as primary factors, in addition to the importance of maintaining a thermal gradient to transfer heat from the deep grey to the white matter cortex and brain surface (Childs, 2008). Brain heat removal can occur via cerebral blood flow (CBF) as well as heat dissipation through the scalp (Sukstanskii & Yablonskiy, 2006). The temperature of incoming arterial blood to the brain is a determining factor in brain heat removal, which is a part of the extraordinary role of CBF in brain functioning (Childs, 2008). Humans can cool the incoming arterial blood on its passage to the brain through the existence of a temperature gradient between the arterial blood stream and the tissue adjacent to the carotid artery (Rubenstein *et al.*, 1960).

Human brain thermodynamic responses can be measured using the internal jugular venous to carotid arterial blood temperature difference (v-aD_{temp}), cerebral metabolic heat production, and CBF (Nybo *et al.*, 2002*b*). Cerebral heat balance is attained at rest with ~ 0.3 °C v-aD_{temp}, and CBF of ~ 50 ml/100 g cerebral issue)/min (Yablonskiy *et al.*, 2000), in order to cope with an average metabolic rate of ~ 3-3.5 ml O_2/100 g cerebral tissue)/min, which equals a heat production of ~ 0.6 J/g/min (Madsen *et al.*, 1993). During exercise, the balance between cerebral heat production and removal can be disturbed because of increased T_c accompanied with

increased blood temperature; as a result, the brain will be supplied with warmer arterial blood and the v-aD_{temp} will theoretically be diminished. Correspondingly, exercise hyperthermia is accompanied with a reduction in global CBF and elevated metabolic rate of oxygen and glucose at high levels of T_c. Cerebral heat removal is thereby reduced by the lower CBF and heat production is increased by the higher metabolic rate (Nybo *et al.*, 2002*a*).

To determine to what extent brain temperature rises during hyperthermia, cerebral heat removal and brain temperature were investigated during exercise hyperthermia. CBF, indexed by middle cerebral artery mean blood velocity, and v-aD_{temp} responses were compared between exercise hyperthermia and normothermia trials (Nybo *et al.*, 2002*b*). In both conditions, the jugular vein blood temperature was always higher than the arterial blood, indicating a continuous heat removal via CBF. Under such circumstances of reduced CBF during hyperthermia, heat removal was lower by 30%, and more heat was stored in the brain compared to normothermia; however, v-aD_{temp} was not different between trials. Jugular venous blood temperature was higher than the esophagus, tympanic membranes, and arterial blood throughout the trials in both conditions, which highlights the fact that brain temperature is persistently higher than T_c even at high levels of hyperthermia (Nybo *et al.*, 2002*b*).

Cardiovascular-related factors

The cardiovascular-related factors contributing to fatigue during exercise hyperthermia are especially evident during high-intensity activities (González-Alonso *et al.*, 2008; Nybo, 2010). During exercise hyperthermia, skin blood flow increases for heat dissipation (Johnson, 2010). In order to meet the increased demands of blood flow to the skin, the body responds by increasing HR in an attempt to prevent any reduction in cardiac output (Jose *et al.*, 1970; González-Alonso *et al.*, 1997), and inducing splanchnic and renal vasoconstriction (González-

Alonso *et al.*, 2008). However, while HR increases under these conditions, stroke volume is reduced, likely because of reduced ventricular filling time associated with tachycardia as well as peripheral displacement of blood volume (Rowell, 1974; González-Alonso *et al.*, 2000, 2004; Gonzalez-Alonso, 2003; Trinity *et al.*, 2010). This may result in a 2-3 $L \cdot min^{-1}$ reduction in cardiac output (Rowell *et al.*, 1966; González-Alonso *et al.*, 1999; Périard *et al.*, 2011), and this reduction becomes markedly more pronounced if hyperthermia is accompanied with dehydration (González-Alonso *et al.*, 1997). Lower cardiac output during high-intensity exercise in the heat leads to decreased leg blood flow, mean arterial pressure (MAP), and oxygen delivery, which ultimately impairs muscle aerobic capacity (Gonzalez-Alonso, 2003). Generally, muscle blood flow is only compromised during dehydration and/or high-intensity activity (González-Alonso *et al.*, 2008), as would be expected during time trial paced cycling.

In summary, cycling performance is characterized by long duration and high intensity. Hot environments, where events frequently take place, exacerbates cardiovascular and thermoregulatory strain developed during exercise, and performance has been shown to decline under such circumstances. Exercise impairment in heat stress is believed to be attributed to elevated core temperature reaching a critical level at which some people stop exercise voluntarily. Additionally, elevated body temperatures during exercise hyperthermia can impair performance through central- and cardiovascular-related factors. Central factors primarily limit performance via increased brain temperature which inhibits central drive, whereas cardiovascular factors include decreased muscle blood flow at high exercise intensities concomitant with dehydration and/or peripheral displacement of blood flow to skin for heat dissipation. Given the challenges associated with high intensity cycling in the heat, countermeasures have been

developed to attenuate performance impairments. These methods and their associated mechanisms of action will therefore be reviewed in the next section.

Countermeasures to performance reduction in the heat

Precooling

From the preceding discussion it is clear that heat stress impairs aerobic exercise performance and terminates it prematurely. Therefore, countermeasures designed to attenuate elevated T_c are of utmost importance to ameliorate the negative effects of hyperthermia on performance. Literature regarding these countermeasures will be reviewed in the following sections.

One strategy used to mitigate hyperthermia is precooling the body prior to exercise. The goal of precooling is to increase heat storage capacity and thereby reduce the rate of increase in T_c (Marino, 2002; Quod *et al.*, 2006). Generally, precooling techniques, which reduce initial core temperature, skin temperature, or both, have been shown to enhance endurance exercise performance in the heat (Ross *et al.*, 2013).

Precooling methods that lower T_c prior to exercise have shown a positive effect on time to exhaustion and power output during exercise hyperthermia. After either ice slurry ingestion or cold water immersion, running time to exhaustion in the heat (~ 34 °C) was longer compared to control with warm water ingestion only, and authors attributed the improvement to lower T_c in both cooling treatments (Siegel *et al.*, 2012). Compared to cold water ingestion, ice slurry ingestion before exercise resulted in lower T_c before beginning and throughout exercise, higher heat storage, and improved running time to exhaustion in the heat (~ 34 °C) (Siegel *et al.*, 2010; Dugas, 2011). In a hotter environment (40 °C), cycling time to exhaustion performed at 60% $\dot{V}O_{2max}$ was also longer with lower initial T_c induced by cold water immersion (González-Alonso

et al., 1999). In another study, the combination of cold water immersion and cold fluid ingestion that resulted in the lowest initial T_c resulted in the longest time to reach exhaustion after cycling at 80% $\dot{V}O_{2max}$ in the heat (32 °C) (Hasegawa *et al.*, 2006).

Precooling has also been shown to be advantageous in terms of time to exhaustion during exercise performed in cooler environments. Running to exhaustion at 82% $\dot{V}O_{2max}$ in a thermoneutral environment (24 °C) was improved after 30 min of exposure to air flow at 5 °C compared to 24 °C. Cold exposure induced a lower initial T_c and lower T_c throughout exercise, and as a result heat storage was greater (Lee & Haymes, 1995). Exposure to cooler air (0 °C) also increased the work rate and cycling time to exhaustion at a cooler ambient temperature (18 °C), and like the previous study this was accompanied with lower T_c at the beginning and during exercise compared to that of the control group (Schmidt & Brück, 1981; Olschewski & Brück, 1988). Taken together, these studies indicate that precooling with various methods can improve exercise time to exhaustion, most likely through reduced initial body temperature.

The role of skin temperature to improve exercise in the heat, independent of changes in T_c, is unclear. Precooling cyclists using a water-perfused suit resulted in reduced $\overline{T}_{sk}$ (~ 2.1 °C) compared to no precooling, but T_c was unaffected. Reduced $\overline{T}_{sk}$ did not result in improved cycling performance (Levels *et al.*, 2012). In contrast, whole-body water immersion for up to 60 min reduced $\overline{T}_{sk}$ 5-6 °C without a concomitant reduction in T_{re}, and subsequent distance cycled in 30 min in a warm humid environment (31 °C, 60% relative humidity) was increased from 14.9 to 15.8 km (Kay *et al.*, 1999). Likewise, Siegel et al. (2012) showed reduced $\overline{T}_{sk}$ (~ 6 °C) prior to exercise after water immersion improved subsequent performance, despite no change in T_c. In the same study, ice-slurry ingestion resulted in reduced T_c but only a slight decrease in $\overline{T}_{sk}$ (0.2-0.3 °C) prior to exercise; therefore, improved exercise performance was solely attributed to the

reduced T_c (Siegel *et al.*, 2010). Based on the studies mentioned above, treatments which resulted in reduced $\overline{T}_{sk}$ lead to improved performance only if the reduction in $\overline{T}_{sk}$ was large (5-6 °C) (Kay *et al.*, 1999; Siegel *et al.*, 2012) since a small reduction in $\overline{T}_{sk}$ (2.1 °C) was not associated with exercise improvement (Levels *et al.*, 2012). Furthermore, the treatments which induced a large reduction in $\overline{T}_{sk}$ prior to exercise resulted in a lower T_c late in exercise (Kay *et al.*, 1999; Siegel *et al.*, 2012). Precooling that results in reduced $\overline{T}_{sk}$ can be effective in attenuating thermoregulatory and cardiovascular strain during exercise hyperthermia, but only if the reduction is sufficiently large to blunt the rise in T_c during subsequent exercise.

The studies reviewed above showed benefits of precooling but involved standardized exercise intensities (Siegel *et al.*, 2010, 2012). In self-paced exercise, precooling maneuvers have been shown to have mixed effects on performance. Cold water immersion resulted in decreased T_c and $\overline{T}_{sk}$ and increased the distance run in 30 min in a hot and humid condition (32 °C, 60% relative humidity) by ~ 304 m. Mean body temperature was lower at the start and throughout the exercise after precooling, which facilitated a greater rate of heat storage (Booth *et al.*, 1997). In addition to running, cycling performance at a self-selected intensity also benefits from cold water immersion. For instance, lower body cold water immersion before exercise increased cycling power output during 40 min in the heat (33 °C). Body temperatures, such as T_c, $\overline{T}_{sk}$, and muscle temperature, were lower after precooling until 20 min into exercise compared to no precooling. It is noteworthy that performance differences between the two conditions occurred even after differences between physiological measures (e.g., core, muscle, skin, and body temperatures) were no longer apparent (Duffield *et al.*, 2010). Precooling using water immersion, in which only $\overline{T}_{sk}$ was decreased, without a concomitant reduction in T_c, also resulted in increased heat storage and distance cycled in a 30-min self-paced cycling trial in a hot and humid environment (31 °C,

60% relative humidity). $\overline{T}_{sk}$ remained lower throughout exercise, and T_c was lower between 15 and 20 min of exercise after precooling compared to control (Kay *et al.*, 1999). This demonstrates that maintaining a favorable core to skin thermal gradient, independent of lowering T_c before exercise, can positively impact performance. In contrast, another study showed that when applying different cooling procedures prior to a 40-min cycling time trial in the heat, only the procedure (water immersion followed by ice vest) which reduced T_c improved performance; ice vest alone only reduced $\overline{T}_{sk}$ and had no effect on performance (Quod *et al.*, 2008). Furthermore, altering $\overline{T}_{sk}$ per se did not affect intensity during a 7.5-km cycling time trial (Levels *et al.*, 2012). However, wearing a cooling vest and refrigerated band around the head during warm-up resulted in reduced $\overline{T}_{sk}$ but not T_c and improved 20-min cycling time trial performance in a hot and humid environment (29 °C, ~ 80% relative humidity) by an average increase in power output of 17 W compared to control (Gonzales *et al.*, 2014). The authors attributed the improved performance to higher heat storage resulting from the precooling maneuver (Gonzales *et al.*, 2014). Collectively, these studies suggest that perhaps the best strategy is one in which T_c and $\overline{T}_{sk}$ are lowered before exercise so that the rate of heat storage during subsequent exercise is optimized.

Mechanisms by which precooling improves performance in the heat

One of the mechanisms by which precooling is purported to improve performance is by attenuating declines in neural function. Following running to exhaustion in the heat, the torque output during a 2-min sustained MVC was higher after ice slurry than warm liquid ingestion, consumed following running to exhaustion and prior to MVC test (Siegel *et al.*, 2011). Under such circumstances, internal cooling is hypothesized to enhance performance by facilitating central drive to the muscle (Burdon *et al.*, 2010; Siegel *et al.*, 2010) and attenuating inhibitory

afferent feedback associated with hyperthermia (Siegel *et al.*, 2011). The positive effect of precooling on central drive is further supported by research in which EEG was monitored during exercise in a cool environment (19 °C) in which no elevation in the α/β index occurred relative to exercise in the heat during which hyperthermia elevated the α/β index, which elicited a greater suppression of central arousal (Nielsen *et al.*, 2001). Even though that study did not employ precooling per se, T_c (< 38 °C) during exercise in the cool condition was lower relative to that (~39.8 °C) during exercise in the hot condition, which leads one to speculate that the same could also occur with precooling and ultimately lead to attenuated suppression of central arousal.

Another mechanism by which precooling enhances performance is by attenuating an increase in brain temperature, as high levels of brain temperature have been suggested to limit exercise (Nielsen & Nybo, 2003). As alluded to in a previous section, brain temperature is thought to be regulated to some extent through the brain circulation (Childs, 2008). Given that arterial blood temperature appears to be strongly linearly related with T_c (Fulbrook, 1993), it is possible that precooling induces a decrease in T_c with a concomitant lower arterial blood temperature that can consequently elevate heat removal from the brain. This assertion has been illustrated in research in which cooling the adjacent tissue to the carotid artery via a cooling collar showed an improved exercise time to exhaustion in the heat (Tyler & Sunderland, 2011). Investigators speculated this direct cooling of the region surrounding the carotid artery facilitated greater brain heat removal, which likely contributed to improved performance (Tyler & Sunderland, 2011). As CBF is a primary component of brain temperature regulation (Yablonskiy *et al.*, 2000; Sukstanskii & Yablonskiy, 2006), maintenance of CBF is of utmost importance. Heat stress (60 °C ambient temperature exposure for 10 min), however, has been shown to reduce cerebral blood velocity and mean arterial pressure (Wilson *et al.*, 2002). Nonetheless,

when skin cooling was applied, the decreases in cerebral blood velocity and MAP were suppressed (Wilson *et al.*, 2002). Furthermore, during normothermia selective brain cooling increased CBF but selective brain warming decreased CBF (Kuluz *et al.*, 1993). These studies show the potential efficacy of precooling in preventing the fall of CBF during exercise hyperthermia, but validating this hypothesis needs more investigation.

In addition to having positive effects on the brain, precooling is purported to improve performance during exercise in the heat by ameliorating cardiovascular strain developed during hyperthermia. During incremental exercise conducted in a thermoneutral environment, precooling attenuated cardiovascular strain by reducing HR 8-10 beats/min throughout exercise compared with control (Smith *et al.*, 2013). Cardiovascular drift (i.e., a progressive increase in HR and decrease in stroke volume over time during constant rate exercise) during exercise in the heat is attenuated with fan air cooling (Gliner *et al.*, 1975), which also attenuates the reduction in $\dot{V}O_{2max}$ associated with cardiovascular drift (Wingo & Cureton, 2006). While fan air cooling is different from precooling, one could speculate that if precooling is sufficient enough to mitigate thermal and cardiovascular strain, results could be directionally similar, although the magnitude of the effect might be different. More research is necessary to substantiate this hypothesis, however. Other evidence in support of precooling as a countermeasure to cardiovascular strain includes the restoration of cardiac output and central venous pressure to pre-heating levels after skin cooling (Rowell *et al.*, 1969). Further studies are needed to determine whether precooling per se results in similar alleviation of cardiovascular strain during time trial paced cycling exercise in the heat.

Warm-up techniques

Warm-up is one of the most widely used practices by athletes and coaches prior to exercise and competition. The prevailing theory supporting warm-up is that it is an essential requirement to optimize performance. However, evidence to support the advantageous effects of warm-up on performance is lacking, and the results of studies investigating the effect of warm-up are mixed (Bishop, 2003). Short-duration exercise performance (≤ 5 min) appears to be positively affected by warm-up (Bishop, 2003). For example, vertical jump performance was improved when preceded by different types of warm-up (stretching, running in place, or combination), compared to no preliminary exercise (Pacheco, 1957). Testing the effect of warm-up prior to exercise on short-term power output during cycling revealed that 3-6 min of moderate intensity (60% $\dot{V}O_{2max}$) was an optimum duration and intensity to improve subsequent exercise performance (Sargeant & Dolan, 1987). Evaluating different types of warm-up on a 50-yard swimming sprint, however, showed variable effects on performance. Individual data indicated that 19% of participants performed their best trial after a short warm-up, 37% after no warm-up, and 44% after a regular precompetition warm-up (Balilionis *et al.*, 2012). Improvement of short-term exercise performance following warm-up has been attributed to elevated muscle temperature that likely optimizes enzyme kinetics and contractile function (Bennett, 1984; Cheuvront *et al.*, 2010). This speculation is supported by research demonstrating reduced vertical jump height with lower muscle temperatures (Asmussen *et al.*, 1976). Further evidence shows that maximal dynamic strength, power output, jumping, and sprint performance are all directly related to muscle temperature to a point, i.e., reduced performance with lower muscle temperature (Bergh & Ekblom, 1979*b*).

While warm-up has been shown to have a positive impact on short-duration exercise performance, the effect of warm-up on prolonged exercise is equivocal (Bishop, 2003). Studies reporting a benefit of warm-up showed improved 16.1-km cycling time trial performance at two times of day (morning and evening) after warming up for 5 min at 60% of peak power (Atkinson *et al.*, 2005). On the other hand, warm-up (20 min, 70% $\dot{V}O_{2max}$) impaired running time to exhaustion compared to precooling and no warm-up trials (Uckert & Joch, 2007). Also, active warm-up or passively heating the body both reduced running time to exhaustion in a moderate ambient temperature (~ 21°C), presumably because of higher body temperatures experienced at the beginning of exercise after warming up relative to no warm-up (Gregson *et al.*, 2002).

Mechanisms of warm-up effects on performance

Warm-up is purported to improve exercise performance through modification of muscle function. For instance, increases in body and muscle temperatures following a warm-up are thought to increase nerve impulse transmission, and hence increase muscular contraction speed (Woods *et al.*, 2007). Increasing muscle temperature was accompanied with elevation in maximal velocity, maximal power, and an improvement in the force-velocity relationship (Binkhorst *et al.*, 1977). Also, warm-up-induced elevated muscle temperature potentially increases muscle metabolism. Thighs passively heated prior to 2 min of very intense exercise at ~ 115% $\dot{V}O_{2max}$ resulted in greater ATP and inosine 5`-monophosphate degradation during exercise, lower post-exercise muscle glycogen content, and higher post-exercise lactate concentration, indicating elevation in glycogenolysis, glycolysis, and high energy phosphate degradation (Febbraio *et al.*, 1996). In submaximal exercise at 70% $\dot{V}O_{2max}$, one leg was heated and the other was cooled prior to and during exercise using water-perfused cuffs. Leg heating induced higher muscle temperature and thereby resulted in greater net glycogen use than the

cooling treatment, but did not cause a difference in phosphagen metabolism (Starkie *et al.*, 1999). In the case of prolonged exercise, elevated baseline oxygen uptake ($\dot{V}O_2$) after warm-up is most likely the major mechanism by which performance is improved (Bishop, 2003).

Combined precooling and warm-up

The preceding presentation of studies shows the benefits of precooling and warm-up on exercise performance; interestingly, their respective mechanisms of performance enhancement seem at odds with one another. Precooling improves performance by attenuating a rise in body temperature (Marino, 2002) and warm-up improves performance by facilitating muscle function, largely through increased muscle temperature (Bennett, 1984; Cheuvront *et al.*, 2010). That said, impairment of prolonged exercise after warm-up sessions are thought to be attributable to elevated thermoregulatory strain prior to exercise and reduced heat storage capacity during subsequent exercise (Bishop, 2003). Likewise, in some precooling protocols, decrements in performance were attributed to reduced $\dot{V}O_2$ and muscle temperature (Webb & Annis, 1968; Bergh & Ekblom, 1979*a*).

Whether combining the two strategies would result in a net positive outcome, i.e., precooling would attenuate negative increases in body temperature while warm-up would optimize muscle temperature and thereby contractile and metabolic function, remains to be determined. To our knowledge, studies that investigated the effect of precooling on exercise time to exhaustion in the heat did not include a warm-up session prior to exercise commencement. Likewise, few of the studies that examined the effect of precooling on self-paced exercise time trials had the participant complete a warm-up. Two studies included a standard 20-min warm-up session after three different methods of body cooling and prior to completion of a cycling time trial (Quod *et al.*, 2008; Ross *et al.*, 2011). However, the designs utilized do not permit one to

identify the separate and combined effects of precooling and warm-up on subsequent exercise performance. In another study, a comparison was made between the effect of warm-up and precooling on running time to exhaustion in the heat. In that study, incremental running was preceded by a 20-min warm-up, a 20-min period while wearing a cooling vest at rest, or no treatment (Uckert & Joch, 2007). Running time was improved after the use of the cooling vest by 2.2 min compared to control, but was impaired by 3.4 min after warm-up.

As mentioned, few studies have included a warm-up and precooling in the same investigation, and those that have did not permit the separate evaluation of the two. For instance, one study included torso cooling (via a cooling vest) and another included head cooling (via a neoprene-covered silicone cooling cap) during the warm-up, and the cooling vest treatment resulted in improved 5-km running time (Arngrïmsson *et al.*, 2004). However, investigators in the other study utilizing the cooling cap did not observe cycling time trial performance differences (Levels *et al.*, 2013). Similarly, unpublished observations from our laboratory have shown that precooling using gel wraps during an active warm-up in 35 °C did not improve simulated 16.1-km cycling time trial performance. There was a reduction in $\overline{T}_{sk}$ but no change in T_c at the end of warm-up with precooling compared to no precooling. The net reduction of $\overline{T}_{sk}$ in that combined procedure did not alter the performance outcome.

In light of preceding discussion, the optimal combination of precooling and warm-up remains unclear, especially for cycling. Precooling and warm-up may act reciprocally; precooling could possibly mask the detrimental effect of not warming up, and vice versa, warming up may mask the negative effect of not having precooled. The combination of precooling and warm-up may have an additive, positive impact on endurance cycling time trial

performance in a hot environment by reducing thermal strain (from precooling) and accentuating skeletal muscle function (from warming up), but this has not been systematically investigated.

To accomplish an effective combination of precooling and warm-up, two criteria must be taken into consideration: 1) the combination reduces core temperature while maintaining or increasing muscle temperature, and 2) the precooling method can be used practically in an actual athletic setting and not only in a laboratory setting. One way this might be accomplished is by utilizing ice-slurry ingestion. Ice-slurry ingestion and cold water immersion precooling resulted in improved running times to exhaustion in the heat compared to control, but these separate precooling treatments were not different from each other, indicating that both techniques are comparable (Siegel *et al.*, 2012). Furthermore, in another study that included water immersion and ice-slurry ingestion prior to a cycling time trial in the heat, both treatments resulted in improved performance compared to control, but ice-slurry had a larger effect on power output during one stage of the race and resulted in better overall performance (3.0%) than cold water immersion (1.1%) (Ross *et al.*, 2011). Furthermore, other studies have shown an improvement in running time to exhaustion by ~10 min and cycling time trial performance by 6.5% accompanied with lower T_c after ice-slurry ingestion compared with cold or tap water ingestion (Siegel *et al.*, 2010; Ihsan *et al.*, 2010). These studies have demonstrated that ice-slurry ingestion is comparable with other, effective precooling methods.

Use of ice-slurry is practical in that a relative small quantity is sufficient to reduce T_c compared to larger fluid volumes. The theory of enthalpy of fusion states that the conversion of ice from the solid to the liquid requires larger heat energy than that required to increase the temperature of a liquid (Merrick *et al.*, 2003). This implies that ice ingestion will absorb more heat energy in order to equilibrate with body temperature, and less ice-slurry (compared to fluid)

is needed to induce the same magnitude of temperature reduction. Indeed, the efficacy of ice-slurry ingestion was tested during an outdoor 10-km running time trial. The ice-slurry group had a faster running performance by an average of 15 s, which was associated with a lower T_c at the commencement of the trial compared to subjects who ingested the same amount of a drink at a warmer temperature (Yeo *et al.*, 2012).

Based on the preceding discussion, it may be that ice-slurry and warm-up are a potential combination that can satisfy the conditions needed to achieve optimum performance, i.e., lower T_c and optimally elevated muscle temperature at the start of exercise, and practical for use in athletic field settings.

Summary

Competitive cycling is a prolonged high intense exercise that frequently takes place in hot conditions. Such conditions elevate heat stress and exacerbate the cardiovascular and thermoregulatory strain during exercise. Some evidence implicates attainment of a critical core temperature (~ 40 °C) as a limiting factor during exercise. Other mechanisms through which exercise can be impaired in the heat are classified as central- and cardiovascular-related factors, which occur primarily via increased brain temperature and reduced cardiac output, respectively. Precooling is a strategy used to attenuate thermoregulatory and cardiovascular strain during exercise hyperthermia. Precooling improves performance if it is associated with reduced core temperature prior to exercise. Lowered skin temperature in conjunction with precooling also may play a role if it is sufficiently reduced prior to exercise (5-6 °C) and blunts the rise in T_c during subsequent exercise. Warm-up is also commonly utilized by athletes before exercise and believed to enhance performance, but evidence for its efficacy is lacking, and the effects on performance are mixed. Warm-up is thought to influence performance by accentuating muscle

function and elevating $\dot{V}O_2$ kinetics prior to exercise. The combination of warm-up and precooling may result in combining the advantageous effects of warm-up (muscle accentuation and $\dot{V}O_2$ kinetics), and precooling (attenuation of thermoregulatory and cardiovascular strain), and thereby result in improved performance relative to either strategy alone. Further research is necessary to substantiate this hypothesis, however.

CHAPTER 3

METHODOLOGY

Research Design

A repeated measures research design was utilized in which all participants were tested under all conditions. Participants completed 4 visits to the Exercise Physiology Laboratory at the University of Alabama. The first visit involved measurement of maximal oxygen uptake ($\dot{V}O_{2max}$) using a graded exercise test. The subsequent 4 visits involved the following treatments, in counterbalanced order: 1) precooling alone, 2) precooling + warm-up, and 3) warm-up alone. The counterbalanced treatment orders were assigned to participants in random order. After each treatment, participants completed a 16.1-km simulated cycling time trial in a hot environment (33 °C, ~40-50% relative humidity).

Participants

Following approval by the University's Institutional Review Board and after providing written informed consent, 9 healthy, recreationally active men (performing aerobic exercise 2-3 days per week for at least 20 minutes each day) were recruited. Descriptive statistics (means ± SD) were as follows: age = 24 ± 5 years; body mass = 74.7 ± 4.5 kg; height = 171.4 ± 7.7 cm; body mass index = 25.4 ± 1.5 kg/m^2; body fat estimated from skinfolds = 12.9 ± 5.2%. All participants were free of any cardiovascular or metabolic disease as determined by health history questionnaire. A power analysis revealed 8 – 10 subjects would be necessary to detect a moderate effect among treatments for time to completion of the 16.1 km cycling time trial,

assuming power ~ 0.8 and correlation among the repeated measurements of time to completion ~ 0.9 (Park & Schutz, 1999; Potvin & Schutz, 2000). Women were excluded from participation because fluctuation in internal body temperature and sex hormones concomitant with the female menstrual cycle affect thermoregulatory responses, which could have impacted the primary outcomes (Hessemer & Brück, 1985).

Procedures

Control $\dot{V}O_{2max}$

On the first visit, participants reported to the laboratory after a 3-h fast, but well hydrated. They were instructed to avoid the consumption of caffeine, alcohol, and nonprescription drugs during the day before and the day of testing and vigorous exercise for 2 days prior to testing. Upon arrival, every participant completed a 24-h history questionnaire in order to verify adherence to pre-test instructions. Next, height, weight, and percent body fat from the sum of 3 skinfolds (Jackson & Pollock, 2004) were measured, and a urine sample was provided for assessment of hydration status using urine specific gravity. Urine specific gravity values ≤ 1.020 were considered adequately hydrated (Kavouras, 2002).

Participants then completed a graded exercise test (GXT) on an electronically-braked cycle ergometer (Velotron Dynafit Pro, Racer Mate Inc. Seattle, WA) in an environment maintained at 23 °C and 40%-50% relative humidity. After a 5-10-min warm-up at a self-selected intensity, participants began the GXT at a power output selected during the warm-up based on their perceived exertion and heart rate (140-160 W). Thereafter, power output was 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. The highest 2 consecutive 30-s values were averaged for $\dot{V}O_{2max}$. Heart rate

(HR) was monitored continuously during the GXT using a Polar telemetry transmitter unit (Polar, Stamford, CT). Rating of perceived exertion (RPE) was recorded during the last 30 s of each stage (Borg, 1998). Furthermore, maximal heart rate (HR_{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. Following the GXT and an appropriate rest (typically ~ 20 min), participants completed a simulated 16.1-km cycling time trial to familiarize them with the experimental trials, and to improve the reliability and reproducibility of the measurements during the subsequent trials (Laursen *et al.*, 2003; Zavorsky *et al.*, 2007; Sporer & McKenzie, 2007). The familiarization trial was performed in the same environmental conditions as the GXT (23 °C and 40%-50% relative humidity).

Experimental Trials

Three to four days after the first visit, participants returned to the laboratory for the first experimental trial. They were given the same pre-test instructions as the first visit. Upon arrival, they completed the same 24-h history questionnaire as before. Then, urine specific gravity and nude body mass were measured. Participants then inserted a flexible rectal thermocouple (model RET-1, Physitemp, Clifton, NJ) ~12 cm past the anal sphincter for measurement of rectal temperature (T_{re}) and clothed in cycling shorts and a cycling jersey. Additional thermocouples were then taped to the skin at the lateral deltoid, upper chest, quadriceps, and lateral gastrocnemius for measurement of mean skin temperature ($\overline{T}_{sk}$), according to the following equation (Ramanathan, 1964):

$$\overline{T}_{sk} = 0.3(T_1 + T_2) + 0.2(T_3 + T_4),$$

where T_1, T_2, T_3, and T_4 are lateral deltoid, upper chest, quadriceps, and lateral gastrocnemius skin temperatures, respectively. Mean body temperature ($\overline{T}_b$) was calculated from $\overline{T}_{sk}$ and T_{re} using the following formula (Lenhardt & Sessler, 2006):

$$\overline{T}_b = 0.64 \cdot T_{re} + 0.36 \cdot \overline{T}_{sk}.$$

T_{re} and $\overline{T}_{sk}$ were monitored and recorded continuously at 50 Hz using a computerized data acquisition system (Biopac MP150, Santa Barbara, CA). After instrumentation with thermocouples, a small plastic capsule was taped to the posterior forearm approximately mid-way between the wrist and elbow in order to measure local sweat rate via capacitance hygrometry.

The next set of procedures depended on the treatment. For precooling (PREC), participants sat quietly for 10-15 min inside an environmental chamber set at 33 °C, 40% relative humidity while baseline temperature and HR measurements were recorded. Then, they ingested 4 g/kg body mass of carbohydrate-electrolyte beverage at 10 °C over the course of 20 min, followed by ingesting 14 g/kg of ice-slurry in 2 boluses over the course of 30 min (1 bolus every 15 min) (Ross *et al.*, 2011). During the slurry ingestion, participants wore an ice vest (World Endurance Sports LLC, Tampa, FL) over the cycling jersey, and Elasto-Gel ice wraps (Southwest Technologies, Inc., North Kansas City, MO) were placed around the head, neck, and both legs at the level of the quadriceps.

After precooling, participants voided their bladders and the ice vest and gell wraps were removed. Then the participant completed a 16.1-km simulated time trial as quickly as possible in the environmental chamber (33 °C, 40% relative humidity), as was practiced on the first visit.

For the warm-up trial (WU), participants sat quietly in an environmental chamber (33 °C, 40% relative humidity) for 10-15 min while baseline temperature and HR measurements were

recorded. Then they ingested 14 g/kg of carbohydrate-electrolyte beverage at 10 °C to match the same volume of fluid ingested during the precooling part of PREC. Next, they completed a 20-min warm-up on the cycle ergometer, which consisted of 2 sets of 3 min at 25% $\dot{V}O_{2max,}$ 5 min at 60% $\dot{V}O_{2max,}$ and 2 min at 80% $\dot{V}O_{2max}$, as was practiced by elite Australian cyclists in the same conditions (Ross *et al.*, 2011). During the warm-up, participants ingested 4 g/kg body mass of the carbohydrate-electrolyte beverage at 10 °C, matching the volume of fluid ingestion during the 20-min period before precooling in PREC. After the warm-up, participants voided their bladder, and then completed a 16.1-km time trial as before.

For the warm-up + precooling trial (COMBO), the procedures were the same except both the precooling treatment and the warm-up were performed. Ice-slurry was ingested during both the precooling (14 g/kg body mass) and warm-up (4 g/kg body mass) segments. Additionally, during precooling and warm-up, an ice vest was worn over the cycling jersey and Elasto-Gel ice wraps were placed around the head, neck, and both legs at the level of the quadriceps, in an effort to sustain the effect of precooling. Participants were exposed to the precooling treatments for a longer duration during COMBO compared to PREC because of inclusion of the cooling methods during the warm-up portion of COMBO. After finishing the treatments, participants voided their bladders and the ice vest and gel wraps were removed, and the 16.1-km time trial was completed as before. There was a 5-min transient time between the end of the first part and the start of the second part of all treatments. In all trials at the beginning of exercise, a fan was placed in front of the participant at a distance that produced air velocity of 10 km/h at the position of the bike, to simulate the wind convective effect in outdoor cycling.

During the treatments, $T_{re,}$ $\overline{T}_{sk,}$ $\overline{T}_{b}$, local sweat rate, and HR were recorded continuously. The simulated 16.1-km time trial was segmented into 4 intervals of 4-km each. At the end of

each interval, T_{re}, $\overline{T}_{sk}$, $\overline{T}_{b}$, HR, rating of perceived exertion (RPE), rating of thermal comfort (RTC), local sweat rate, average power output, and completion time were recorded. Experimental trials were separated by 48-72 hours.

Data Analysis

Data are presented as means ± SD. Data collected during exercise were averaged every 4 km. A one-way repeated measures analysis of variance (ANOVA) was used to test the significance of mean differences among the treatment conditions for time to complete the simulated time trial. For other outcomes, such as temperature measures and HR, two-way (treatment × distance) repeated measures ANOVAs were performed. For significant ANOVA results, paired samples t-tests with a Bonferroni-adjusted alpha level to control experiment-wise error rate were performed to detect differences at each 4-km point during exercise. Data were analyzed using SPSS v. 19.0 (SPSS, Inc., Chicago, IL), and all significance tests used an α level of 0.05.

Further analyses of individual performance times were conducted to evaluate single participant responses separate from group effects. The smallest mean time difference between PREC and WU and between COMBO and WU that would be considered statistically significant was determined using a paired samples t-test with α of 0.05 and β of 0.8 and specialized statistical software (Piface) (Lenth, 2006-9). Participants whose performance times for either PREC or COMBO were lower (i.e., faster) than WU by an amount equal to or greater than the least significant time difference were then classified as positive responders. Participants whose performance times for either PREC or COMBO were higher (i.e., slower) than WU by an amount equal to or greater than the least significant difference were classified as negative responders. Participants whose performance times for either PREC or COMBO were equal to

that of WU or different by an amount less than the least significant difference were considered non-responders.

CHAPTER 4

RESULTS

As expected, ambient temperature was not different among experimental trials and averaged 33.9 ± 0.1 °C, 33.8 ± 0.1 °C, and 33.8 ± 0.1 °C for PREC, COMBO, and WU, respectively (P = 0.96).

Maximum oxygen uptake, hydration, and body mass changes

The graded exercise test showed participants had an average $\dot{V}O_{2max}$ of 43.0 ± 5.2 $mL \cdot kg^{-1} \cdot min^{-1}$, and HR_{max} of 187.7 ± 9.6 beats/min. After fluid and ice-slurry ingestion, pre-exercise USG was < 1.020 in all treatments, but was significantly lower in warm-up (WU) (1.007 ± 0.005) than precooling (PREC) (1.015 ± 0.008) and combination of warm-up + precooling (COMBO) (1.015 ± 0.005) (P = 0.02). USG values post-exercise were not different among treatments (1.011 ± 0.007; 1.006 ± 0.003; and 1.005 ± 0.002 for COMBO, WU, and PREC, respectively; P = 0.06). Fluid and ice-slurry ingestion prior to exercise resulted in a gain in body mass in all treatments (PREC = 1.2 ± 0.3 kg, COMBO = 1.1 ± 0.3 kg, WU = 0.9 ± 0.3 kg), but the gain was not different among treatments (P = 0.09). Furthermore, body mass remained similar among treatments prior to exercise (PREC = 76.1 ± 5.0 kg; COMBO = 76.0 ± 5 kg; WU = 74.7 ± 3.7 kg; P = 0.8). Exercise resulted in comparable decreases in body mass across treatments (PREC = 75.5 ± 5.0 kg; COMBO = 75.2 ± 5.0 kg; WU = 74.1 ± 3.6 kg; P = 0.8).

Time trial performance and power output

The exercise was segmented into 4 intervals of 4 km each. As shown in Table 1, time to complete each 4-km interval was not different between treatments (P = 0.81 for treatment × 4-

km interval interaction), and so not surprisingly, time to complete the overall time trial was not different among treatments, either (PREC: 31.96±2.05 min; COMBO: 32.64±2.90 min; WU: 33.09±3.09 min; P>0.05). There was a general trend of reduced power output over time until the final interval in each treatment, but there was no treatment × 4-km interval interaction (Table 1; P = 0.98). Practically speaking, power output was higher during each 4-km interval in PREC, and overall performance time was shorter by 3.4% compared to WU and 2% compared to COMBO, but these differences were not statistically significant (P = 0.35 for power output and P = 0.67 for performance time).

Table 1. Performance time, power output, and perceptual responses during each 4-km interval of a 16.1-km cycling time trial after precooling, warm-up, and a combination of both (mean ± SD) (n = 9).

		Distance (km)				
Treatment	Variable	Baseline	0 – 4	4 – 8	8 – 12	12 – 16.1
PREC	Time (min)	—	7.9 ± 0.4	7.9 ± 0.5	8.0 ± 0.6	7.9 ± 0.7
	PO (W)	—	165.0 ± 17.9	162.5 ± 18.7	160.5 ± 19.6	161.7 ± 21.9
	RPE	8 ± 3	12 ± 1*	14 ± 1*	16 ± 1*	17 ± 2*
	RTC	3 ± 1†	4 ± 1*†§	5 ± 1*†§	6 ± 1*	6 ± 1*
WU	Time (min)	—	8.0 ± 0.7	8.2 ± 0.8	8.0 ± 0.9	8.6 ± 2.0
	PO (W)	—	160.0 ± 32.0	153.6 ± 28.9	149.7 ± 27.0	151.9 ± 26.8
	RPE	7 ± 2	13 ± 1*	15 ± 2*	16 ± 1*	18 ± 1*
	RTC	4 ± 1	5 ± 1*	6 ± 0*	6 ± 1*	7 ± 1*
COMBO	Time (min)	—	8.1 ± 0.8	8.2 ± 0.8	8.3 ± 0.8	8.0 ± 0.7
	PO (W)	—	159.2 ± 27.5	154.4 ± 26.4	151.6 ± 25.9	154 ± 25.6
	RPE	7 ± 1	13 ± 1*	15 ± 1*	16 ± 1*	17 ± 2*
	RTC	3 ± 1†	5 ± 1*	6 ± 1*	6 ± 1*	6 ± 1*

PREC = precooling treatment; WU = warm-up treatment; COMBO = combination treatment. PO = power output; RPE = rating of perceived exertion; RTC = rating of thermal comfort. *P < 0.05 vs. baseline, †P < 0.05 vs. WU, §P < 0.05 vs. COMBO.

The smallest mean difference in performance time between PREC and WU and between COMBO and WU that would be considered statistically different (i.e., the least significant difference) was determined to be 2.7 min. Only 1 person was a positive responder, i.e., completed the 16.1 km faster during PREC than WU by 3.7 min (Figure 1). The rest of the participants were non-responders (i.e., the difference between PREC and WU and COMBO and WU was less than 2.7 min).

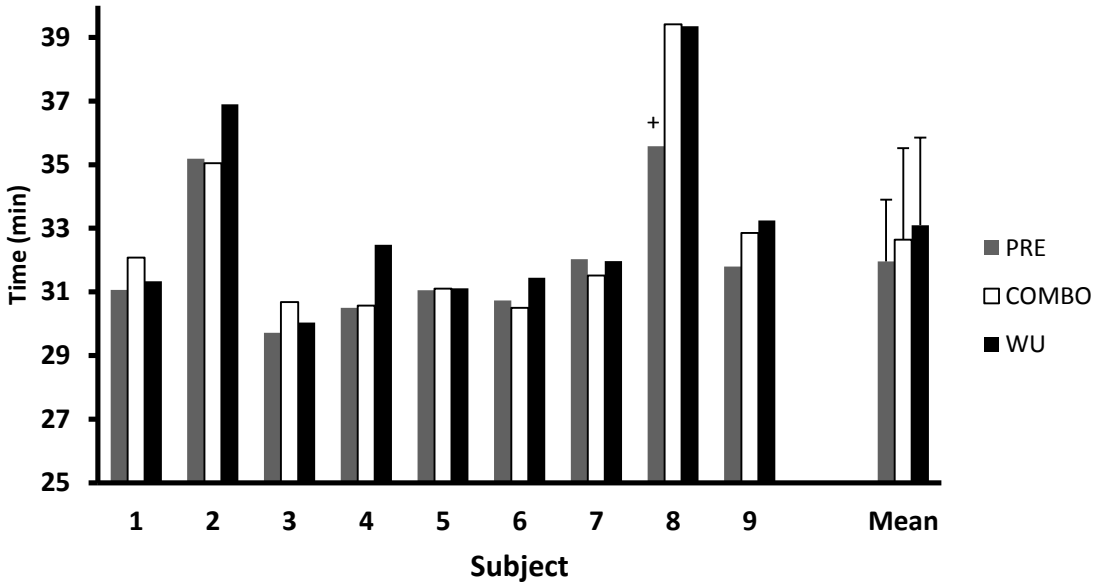

Figure 1. Individual and mean ± SD performance times to complete a 16.1-km cycling time trial after precooling (PREC), warm-up (WU), and a combination of both (COMBO). Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled

with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest. Approximately 5-10 min separated the end of a given treatment and the start of exercise. [+] One person was considered a positive responder (i.e., PREC was faster than WU by an amount greater than the least significant difference, 2.7 min). The other participants were considered non-responders (i.e., the difference in PREC and WU and COMBO and WU was less than the least significant difference).

Heart rate, perceived exertion, and thermal sensation

Baseline heart rate (HR), rating of perceived exertion (RPE), and rating of thermal comfort (RTC) were measured when participants finished the treatments and right before starting the time trial; HR also was measured continuously during the time trial, and values were then averaged every 4 km (Figure 2). Not surprisingly, baseline HR was lower after PREC than after COMBO and WU since PREC did not involve any exercise during treatment administration. The addition of PREC to WU (COMBO) did not attenuate the elevation of HR during the warm-up (Figure 2). Averaged across all 4-km intervals, HR was lower during PRE than during COMBO and WU (main effect of treatment; $P = 0.03$), but upon inspection differences in HR among treatments did not appear to be of any practical significance.

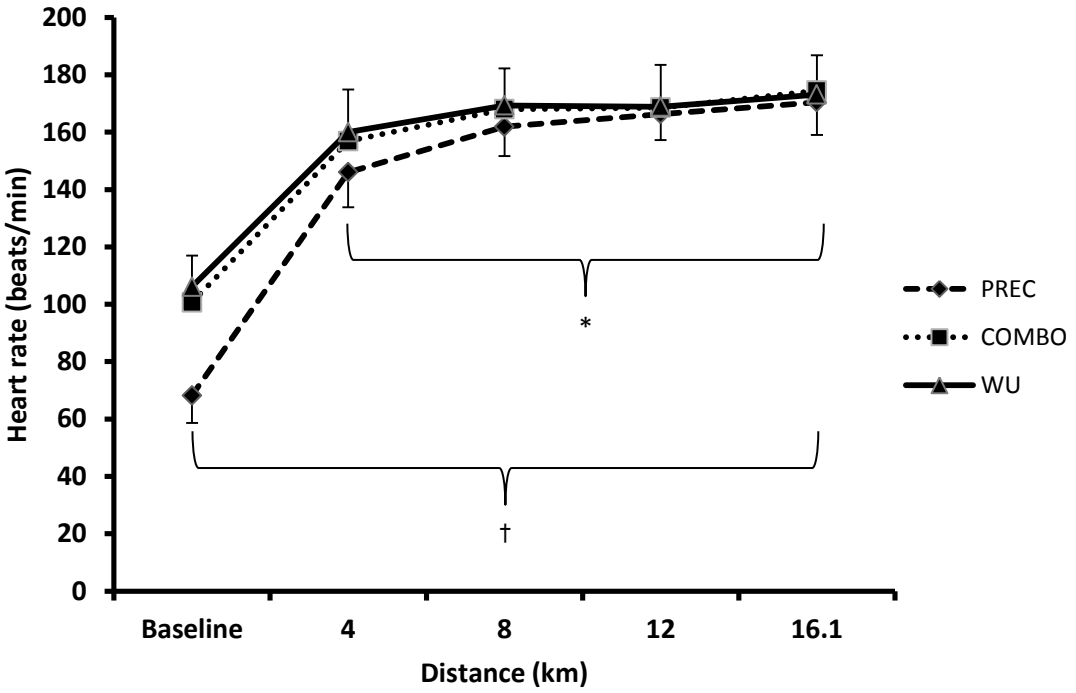

Figure. 2. Heart rate responses (mean ± SD) during completion of a 16.1-km cycling time trial after precooling (PREC), warm-up (WU), and a combination of both (COMBO). Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest. Approximately 5-10 min separated the end of a given treatment and the start of exercise. SD bars for COMBO have been omitted for clarity. Heart rate values represent the average over each 4-km interval of the time trial. Baseline represents the time point after treatment administration just prior to the start of exercise. *$P < 0.05$ vs. baseline across all treatments; $^{\dagger}P < 0.05$ PREC vs. COMBO across all 4-km intervals ($n = 9$).

RPE and RTC were measured at the end of every 4-km interval (Table 1). Like HR, RTC was lower at baseline after PREC and COMBO compared to WU because of the effect of the precooling. Values increased over time but remained lower in PREC compared to the other treatments until the third 4-km interval, after which point treatments were not different. RPE values at baseline and throughout exercise were not different among treatments (all $P > 0.05$).

Thermal responses

As shown in Figures 3, 4, and 5, before treatment administration, T_{re}, $\overline{T}_{sk}$, and $\overline{T}_{b}$ were not different across treatments ($P = 0.88$, 0.86, and 0.38, respectively). The administration of ice-slurry coupled with ice-vest during the precooling part of COMBO and PREC resulted in lower T_{re}, $\overline{T}_{sk}$, and $\overline{T}_{b}$ than WU at the end of the treatments, despite the slight temperature increase that occurred during the warm-up segment of COMBO. Additionally, PREC resulted in lower $\overline{T}_{sk}$ and $\overline{T}_{b}$ than COMBO. As expected, WU resulted in increases in all 3 temperature parameters relative to baseline. While ice-slurry and ice-vest resulted in lower temperatures at the end of treatment and early in exercise in PREC and COMBO, the differences among treatments for the 3 temperature parameters narrowed as the exercise progressed.

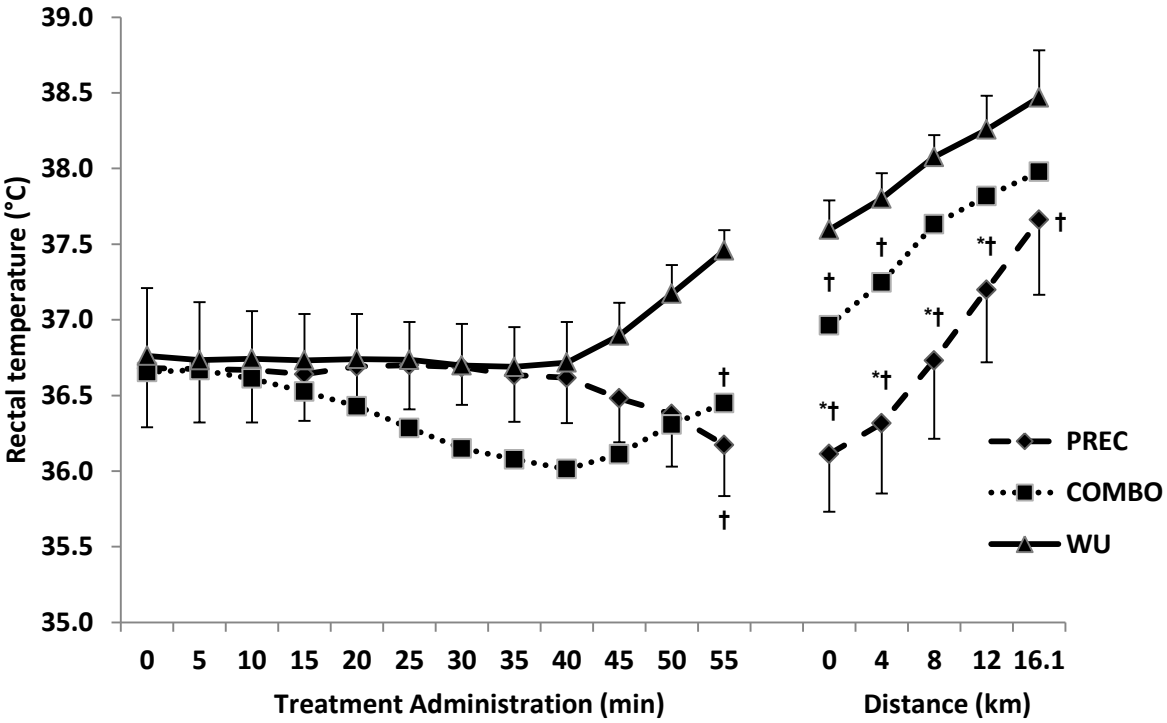

Figure3. Rectal temperature (T_{re}) responses (mean ± SD) over time during treatment administration [precooling (PREC), warm-up (WU), and a combination of both (COMBO)] and averaged every 4 km during completion of a 16.1-km cycling time trial. Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest. Approximately 5-10 min separated the end of a given treatment and the start of exercise. SD bars for COMBO have been omitted for clarity. $^{*}P < 0.05$ vs. COMBO; $^{\dagger}P < 0.05$ vs. WU (n = 9).

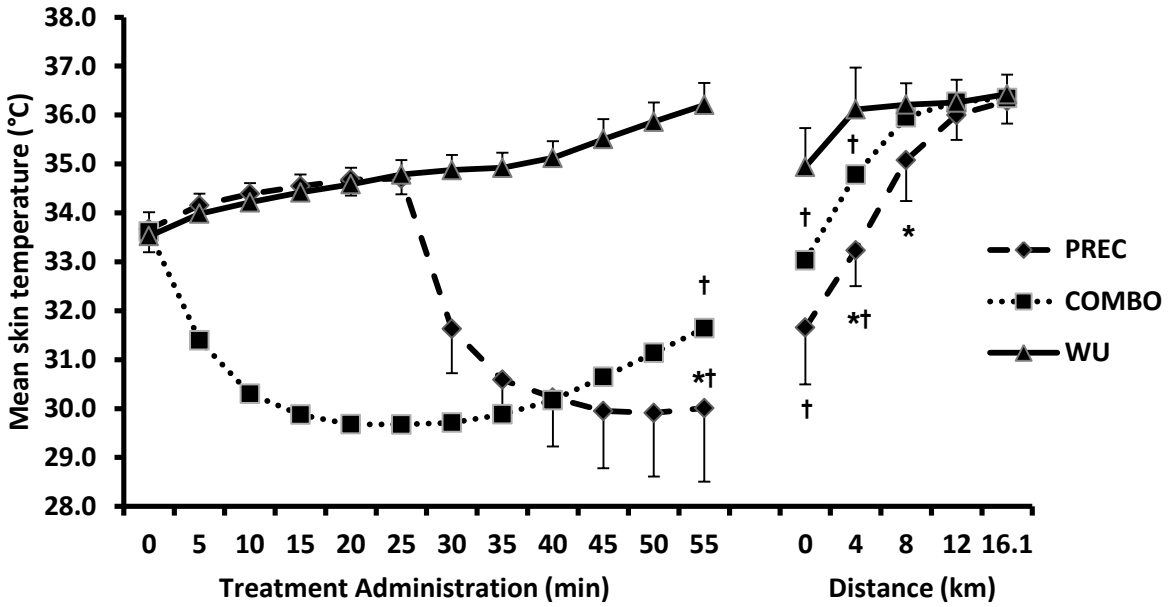

Figure 4. Mean skin temperature responses (mean ± SD) over time during treatment administration [precooling (PREC), warm-up (WU), and a combination of both (COMBO)] and averaged every 4 km during completion of a 16.1-km cycling time trial. Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest. Approximately 5-10 min separated the end of a given treatment and the start of exercise. SD bars for COMBO have been omitted for clarity. $^{*}P < 0.05$ vs. COMBO; $^{\dagger}P < 0.05$ vs. WU (n = 9).

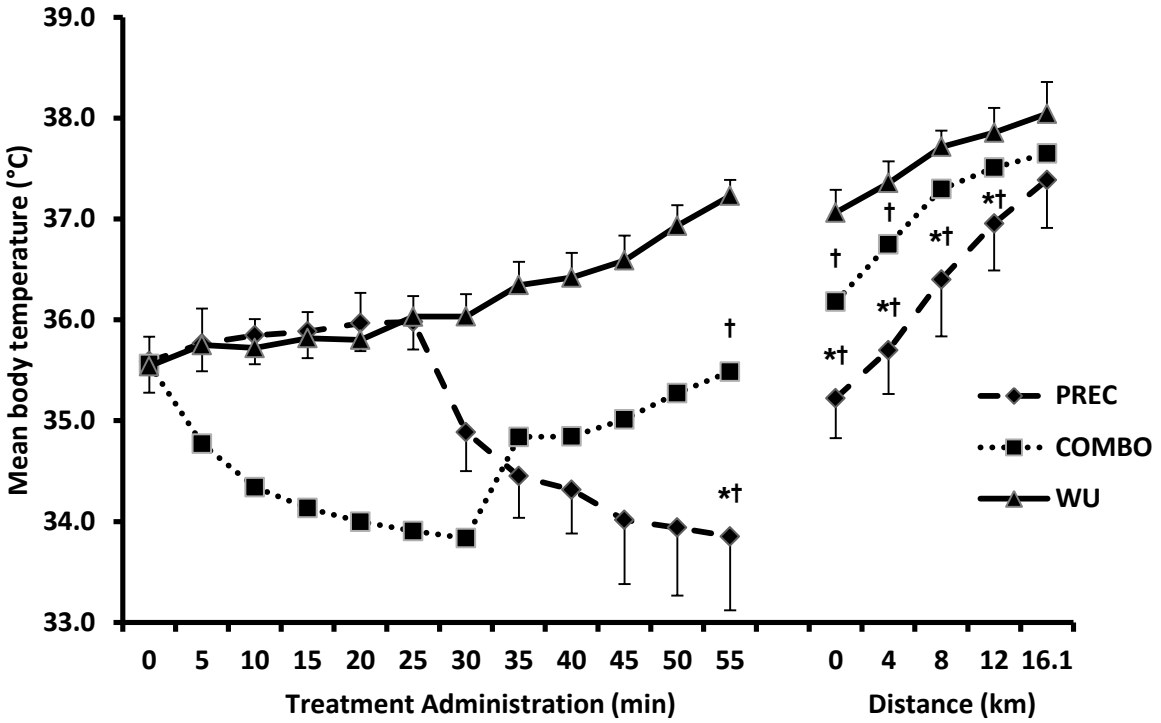

Figure 5. Mean body temperature responses (mean ± SD) over time during treatment administration [precooling (PREC), warm-up (WU), and a combination of both (COMBO)] and averaged every 4 km during completion of a 16.1-km cycling time trial. Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest. Approximately 5-10 min separated the end of a given treatment and the start of exercise. SD bars for COMBO have been omitted for clarity. $^{*}P < 0.05$ vs. COMBO; $^{\dagger}P < 0.05$ vs. WU (n = 9).

Local and whole-body sweat rate

As illustrated in Figure 6, local sweat rate was essentially negligible during the first 40 min of treatment administration. During the warm-up phase of WU, local sweat rate increased

and remained higher than both PREC and COMBO until the start of the time trial. Local sweat rate also was higher during COMBO than PREC at the start of the time trial. After exercise started, local sweat rate remained higher in COMBO and WU for the first 4 km, but thereafter treatments were not different ($P > 0.05$). Likewise, whole-body sweat rate was not different among treatments (1.1 ± 0.2 L/h, 1.03 ± 0.5 L/h, and 1.22 ± 0.38 L/h for PREC, COMBO, and WU, respectively) ($P = 0.54$).

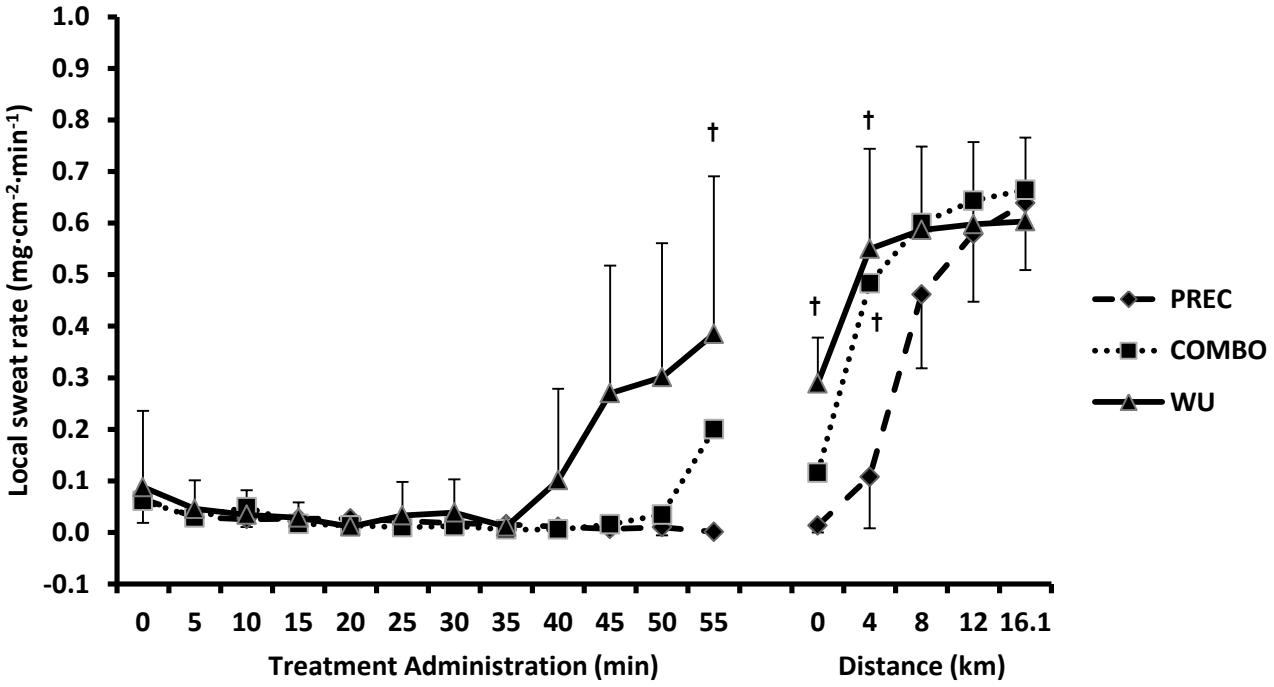

Figure 6. Local sweat rate responses (mean ± SD) over time during treatment administration [precooling (PREC), warm-up (WU), and a combination of both (COMBO)] and averaged every 4 km during completion of a 16.1-km cycling time trial. Treatments were administered over 55 min before the time trial, in the following manner: PREC, 20 min of fluid ingestion, 5 min of transient time, and 30 min of ice-slurry ingestion coupled with ice-vest; WU, 30 min of fluid ingestion, 5 min of transient time, and 20 min of warm-up; COMBO, 30 min of ice-slurry ingestion coupled with ice-vest, 5 min of transient time, and 20 min of warm-up coupled with ice-slurry and ice-vest.

Approximately 5-10 min separated the end of a given treatment and the start of exercise. SD bars for COMBO have been omitted for clarity. $^{*}P < 0.05$ vs. COMBO; $^{\dagger}P < 0.05$ vs. PREC (n = 9).

CHAPTER 5

DISCUSSION

This study investigated the separate and combined effects of warm-up and precooling on a subsequent self-paced 16.1-km cycling time trial. Based on previous studies which have investigated warm-up and precooling independently, it was speculated that precooling alone may mask the detrimental effects of lack of warm-up, and warm-up alone may mask the detrimental effects of lack of precooling. Therefore, it was hypothesized that the combination of the two would be additive and result in faster cycling performance in the heat compared to either treatment alone. Contrary to that hypothesis, the primary finding was that the combined warm-up and precooling treatment did not result in a faster cycling time trial performance, whether expressed as overall time or as time to complete each 4-km segment of the 16.1 km. Despite a lack of impact on performance time, precooling and the combination of precooling and warm-up attenuated thermoregulatory strain during exercise.

In terms of the individual effects of precooling, the present findings are in contrast to some studies which have shown significant improvements in performance following ice-slurry ingestion (Ihsan *et al.*, 2010; Ross *et al.*, 2011; Yeo *et al.*, 2012). Differences in participants and methodology probably explain the discrepant results. For example, consumption of 14 g of ice-slurry per kg of body mass while applying iced towels improved cycling time trial performance in the heat by 1.3% compared to consumption of cold water (Ross *et al.*, 2011). The exercise was meant to simulate the course characteristics of the Beijing Olympic games, which equals ~ 46 km, and was performed by highly trained cyclists with average $\dot{V}O_{2max}$ of 71.6 $mL \cdot kg^{-1} \cdot min^{-1}$. In

the present study, the distance covered was ~ 65% shorter (16.1 km) and the subjects were recreationally active with lower average $\dot{V}O_{2max}$ (~ 42.9 mL·kg^{-1}·min^{-1}). If the current study had utilized a longer race distance then perhaps results may have been more similar. Furthermore, fluid ingestion during exercise was provided ad libitum in the Ross et al. (2011) study. Conversely, no fluid was provided during exercise in the present study. Another methodological difference between the present study and that of Ross et al. (2011) is that pre-exercise fluid and carbohydrate intake were not similar between groups in the Ross et al. (2011) study, whereas in the present study, the volume of fluid ingestion was standardized across treatments in order to assure the same amount of fluid and carbohydrate was administered during all treatments before the commencement of the time trial. Although the authors concluded that the slight difference in hydration and carbohydrate intake between groups was unlikely to have affected performance, it is possible there was an effect.

In another study, 40-km cycling time trial performance was faster after ice-slurry ingestion compared to tap water consumption prior to exercise (Ihsan *et al.*, 2010). These positive findings are not limited to cycling. Ice-slurry ingestion improved 10-km outdoor running time trial performance compared with fluid at ambient temperature (Yeo *et al.*, 2012). Approximately 45 min, on average, was required to complete the trials. Taken together with the present results, these studies and the Ross et al. (2011) study mentioned above suggest that ice-slurry may be efficacious for events of longer distances.

This notion is supported by a study in which participants completed a 15-km cycling time trial, similar to the distance used in the present study, and found no performance improvement with ice ingestion (Levels *et al.*, 2013). Specifically, ice ingestion with or without head cooling reduced T_c prior to exercise compared to warm-up or warm-up combined with head cooling.

Likewise, cold flavored water ingestion, which successfully reduced T_c, did not induce a difference in a 30-min self-paced cycling time trial compared with a warmer control fluid (Byrne *et al.*, 2011). The volume of exercise in the two studies mentioned above was comparable with the volume of exercise used in our study (15-km and 30-min compared to 16.1-km and ~ 32 min, respectively), and effects on performance were also comparable. As mentioned in the previous paragraph, this suggests that the overall heat stress (function of intensity and distance/duration) may require a minimal threshold in order to elicit an effect of precooling. Furthermore, aerobic fitness level and acclimatization status also may influence the efficacy of precooling on performance, since the studies that showed improved performance after precooling had used well trained athletes (Ihsan *et al.*, 2010; Ross *et al.*, 2011; Yeo *et al.*, 2012), and the studies that lacked a performance benefit, like ours, used recreationally active subjects (Byrne *et al.*, 2011; Levels *et al.*, 2013).

To this point the discussion has focused on the results of the precooling treatment in this study in relation to other, similar studies. Besides precooling, however, another important purpose of the present study was to investigate the combined effect of precooling and warm-up on subsequent self-paced cycling time trial performance in the heat. Our findings are difficult to compare to those of other studies, however, because previous studies in this area either: 1) did not include a warm-up session following the precooling treatment (Kay *et al.*, 1999; Siegel *et al.*, 2010, 2012; Ihsan *et al.*, 2010; Byrne *et al.*, 2011), 2) compared precooling and warm-up separately (Uckert & Joch, 2007; Levels *et al.*, 2013), or 3) combined a warm-up session with all treatments so the combination effect of precooling and warm-up cannot be determined (Ross *et al.*, 2011; Yeo *et al.*, 2012). That said, two studies used methods that combined cooling and warm-up in such a way that comparison with the present study can be made. In one of these

studies, the effect of wearing a cooling vest and gel wraps during warm-up on subsequent 5-km running performance was explored (Arngrïmsson *et al.*, 2004). In the other study, the effect of external precooling (ice vest, gel wraps around the neck, head, and legs) during warm-up on subsequent 16.1-km cycling time trial performance in the heat was investigated (unpublished data from our laboratory). Running performance was improved (Arngrïmsson *et al.*, 2004), but cycling performance was unaffected (unpublished observations). Neither study found a reduction in core body temperature below baseline at the start of exercise, and no comparisons between precooling alone and the combination of precooling and warm-up were made. The novel finding of our study was that combining precooling and warm-up did not result in greater performance than either treatment alone.

Despite the lack of a difference in performance among treatments, there were differences in thermoregulatory strain, which could have implications for performance during more prolonged exercise. During the precooling segments of PREC and COMBO, there was a significant reduction in body temperatures (T_{re}, $\overline{T}_{sk}$, $\overline{T}_{b}$); however, after warm-up and prior to exercise, T_{re}, $\overline{T}_{sk}$, and $\overline{T}_{b}$ returned to near baseline levels during COMBO. Nonetheless, compared to WU, T_{re} remained lower in PREC throughout exercise and during COMBO during the first 4 km; $\overline{T}_{sk}$ was lower for the first 4 km of PREC and COMBO; and $\overline{T}_{b}$ was lower for the first 12 km of PREC and the first 4 km of COMBO. This indicates that, relative to WU, the PREC and COMBO treatments successfully blunted thermal strain, especially early in exercise, which may have positively impacted performance during longer duration exercise.

Interestingly, precooling alone resulted in even less thermoregulatory strain than either of the other two treatments, but performance was unaffected. While not statistically significant, performance times were faster in 5 of the 9 participants after PREC. If the data in Figure 3 are

extrapolated, and T_{re} remains lower in PREC, it may be that excess thermoregulatory strain would eventually affect performance in the other treatments. One possible interpretation is that precooling is more important than warm-up prior to prolonged cycling in the heat, but further research is warranted to substantiate this claim.

The magnitude of the effect of PREC on T_{re} and $\overline{T}_{sk}$ is likely the result of both the ice slurry ingestion and the cooling vest/gel packs applied to the skin. Previous studies using external cooling (other than water immersion) without simultaneous internal cooling (ice-slurry) showed reduced skin temperature but not core temperature (Arngrïmsson *et al.*, 2004, unpublished observations). This suggests the optimal precooling strategy may involve both internal and external methods since the combination of elevated core and skin temperatures has been shown to negatively impact aerobic exercise performance (Cheuvront *et al.,* 2010). Indeed, a cooling technique that induces lower $\overline{T}_{sk}$ combined with lower T_c at the initiation of exercise maintained lower T_c for a longer duration during subsequent exercise (Kay *et al.,* 1999; Quod *et al.*, 2008; Duffield *et al.*, 2010).

Limitations. A possible limitation of this study was that there was only one familiarization session, in a temperate environment, before the experimental treatments. The reproducibility of cycling time trial performance was tested on competitive cyclists in two studies involving 3 repeat bouts. Investigators found that performance time for the first trial was significantly longer than performance times for the second and third trials (Zavorsky *et al.*, 2007), and when including the first trial in the reliability assessment, within-subject coefficient of variation was increased (Laursen *et al.*, 2003). In the present study, regardless of the treatment, the completion time was not different between the first, second and third trials. Nonetheless, there was an non-significant improvement in mean completion time of the third

trial compared to the first and second trials, so it is possible there was a small familiarization and/or learning effect (e.g., improved pacing, etc., regardless of treatment); however, the trials were counterbalanced. Another possible limitation is that the sample in this study was from a non-athletic population. This may have impacted the results in that well-trained athletes may have benefited more from the potential ergogenicity of the treatments, especially if they were to exercise at higher absolute intensities, which would have resulted in greater heat production. Moreover, during self-paced exercise, such as that incorporated in this study, pace strategy could interact with treatments in that athletes may manage their pace differently than non-athletes when presented with a given treatment.

Perspectives. While there were no statistically significant differences among treatments for performance time, the average time trial after precooling alone using ice-slurry ingestion and ice vest was faster than that after warm-up alone or a combination of precooling and warm-up by 3.2% and 2%, respectively. This may have practical significance given that elite performances are often decided by a smaller margin. Six subjects had their best performance after precooling, and the effects ranged from very small (a few seconds) to large (~ 4 min), while the remaining 3 subjects completed their fastest trials after the combination treatment; improvements ranged from a few seconds to ~ 2 min. Taken together, one might question the efficacy of warm-up before a short-distance cycling time trial and instead focus time and energy on precooling.

In summary, precooling, warm-up, and combining the two did not result in different 16.1-km cycling time trial performance. Nonetheless, thermoregulatory strain was lower after the treatments involving precooling compared to warm-up. Future studies should determine the effects of these treatments on performance of cycling events involving longer distances and heat exposure.

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