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**INFLUENCES OF SKIN AND CORE TEMPERATURE ON CARDIOVASCULAR
RESPONSES DURING EXERCISE**

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Thesis

Presented to the Faculty of the Graduate School

of the University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Master of Arts

The University of Texas at Austin

August 2010

Abstract

INFLUENCES OF SKIN AND CORE TEMPERATURE ON CARDIOVASCULAR RESPONSES DURING EXERCISE

By

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The University of Texas at Austin, 2010

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The cardiovascular effects of whole body heat stress during exercise are well established. However the independent contribution of elevated skin temperature (T_{sk}) or core temperature (T_c) on these responses remains unclear. The purpose of this study was to determine how increases in T_{sk} and T_c alone and in combination, impact cardiovascular responses during moderate intensity exercise. To accomplish this goal, eight healthy, recreationally active males were immersed to the neck in a cold (14 - 17°C) or hot (40 – 42.5°C) water bath for 20 to 25 min to alter T_c immediately prior to exercise with either cool T_{sk} (i.e. fans) or warm T_{sk} (i.e. heaters). Conditions during exercise were cool skin and cool core (CC), warm skin and cool core (WC), cool skin and warm core (CW), and warm skin and warm core (WW), and were conducted in a randomized crossover design.

When data was combined (n=16), warm core conditions (CW and WW) were associated with significantly higher average heart rate (HR) and lower stroke volume (SV) during exercise compared to cool core conditions (CC and WC); 168.1 ± 3.2 vs. 152.2 ± 4.0 beats/min and 139.2 ± 7.3 vs. 147.7 ± 9.4 mL/beat, respectively. The approximate 9 mL/beat decline in SV and 16 beat/min increase in HR in warm core conditions tended to increase cardiac output (Q), 23.2 ± 0.6 vs. 22.2 ± 0.7 L/min, $P=0.078$. Similarly, warm T_{sk} conditions (WC and WW) were associated with significantly higher average HR and lower SV during exercise compared to cool T_{sk} conditions (CC and CW); 165.2 ± 3.3 vs. 155.1 ± 3.4 beats/min and 140.8 ± 7.8 vs. 146.0 ± 8.7 mL/beat, respectively. Additionally, there was also a trend for Q to be elevated with warm skin (23.0 ± 0.6 vs. 22.4 ± 0.6 , $P=0.075$). Although combined data indicated that warm T_{sk} conditions significantly lowered average SV by ~ 6 mL/beat, there was no reduction in SV during exercise by warm T_{sk} , when T_{es} was cool (i.e. $<37.0^{\circ}\text{C}$), as evidenced by identical values for SV in CC and WC, 147.7 ± 9.8 vs. 147.7 ± 9.0 mL/beat, respectively. In contrast, SV was significantly lower in WW compared with CW, 133.9 ± 7.0 vs. 144.4 ± 7.8 mL/beat, respectively. Therefore, the major reduction in SV by warm T_{sk} occurred during WW, when T_{es} was elevated (i.e. $>38.0^{\circ}\text{C}$).

Analyzing data independently for precooling and preheating conditions revealed that warm T_{sk} was associated with greater HR drift from 5 to 20 min of exercise, compared to cool T_{sk} , when esophageal temperature (T_{es}) was both cool or warm (23.9 ± 2.2 vs. 17.5 ± 2.3 and 12.3 ± 1.3 vs. 4.6 ± 1.7 beats/min, respectively). These observations demonstrate that both T_{es} and T_{sk} can directly influence cardiovascular

responses during exercise, as indicated by elevations in HR during exercise with warm T_{sk} , with both warm and cool T_{es} . However SV is not compromised by warm T_{sk} if T_{es} is below 37.5°C. Furthermore, when both T_{es} and T_{sk} are elevated simultaneously, cardiovascular strain (i.e. increased HR and reduced SV) is much greater than when either is elevated alone. This is demonstrated by the finding that average HR was 175.8 ± 3.2 beats/min in WW, compared to 149.8 ± 4.0 , 154.7 ± 4.1 , and 160.3 ± 3.5 beats/min, in CC, WC, and CW, respectively, and the fact that SV was lowest during exercise in WW. In conclusion, individuals exercising in the heat should take measures to keep skin cool, especially when T_{es} is 39°C or greater to attenuate the cardiovascular strain that occurs with warm T_{sk} , when T_{es} is elevated.

Table of Contents

INTRODUCTION	1
REVIEW OF LITERATURE	3
Introduction.....	3
External heating increases cardiovascular strain.....	3
Internal temperature influences cardiovascular responses.....	5
Cardiovascular effects of heat stress during exercise.....	7
Influence of skin and core temperature on exercise performance.....	10
METHODS	14
Subjects.....	14
Research protocol.....	14
Manipulation of core temperature.....	15
Exercise protocol.....	16
Manipulation of skin temperature.....	17
Measurements and techniques.....	17
Statistical analysis.....	22
RESULTS	23
Influence of core temperature during exercise.....	23
Influence of skin temperature during exercise.....	23
Combined influences of core and skin temperature during exercise.....	24
Influence of skin temperature when core temperature is cool.....	24
Influence of skin temperature when core temperature is warm.....	26

Rapid skin cooling during exercise with warm core temperatures.....	28
Figure Legends.....	31
Table Legends.....	33
Figures.....	35
Tables.....	40
DISCUSSION.....	43
Primary findings.....	43
Precooling and preheating.....	48
Warm vs. cool skin when core temperature is cool.....	49
Warm vs. cool skin when core temperature is warm.....	51
Warm skin temperature increases perception of effort.....	53
Impact of skin temperature on physical performance.....	54
Rapid skin cooling.....	58
Limitations.....	60
Conclusions.....	61
APPENDIX A.....	62
APPENDIX B.....	66
REFERENCES.....	82
VITA.....	88

INTRODUCTION

High internal body temperature (T_c) can have profound effects on the cardiovascular system during resting conditions [12, 14, 67]. Furthermore, when T_c is elevated above normal levels during exercise, the demands placed on the cardiovascular system can be extreme. In such conditions, the heart must pump blood to both the skin to maintain thermoregulation, and to active skeletal muscle so the demands of oxygen and nutrients are achieved. Cardiovascular responses to heat stress in humans have long been an area of scientific interest [6, 7, 30], due to the large responses elicited to maintain body temperature and blood pressure. Thus, high T_c can have a dramatic influence on performance for elite athletes, recreationally active individuals, and workers exposed to hot environments (i.e. firefighters, soldiers, miners, etc) [48]. The effects of elevated T_c during exercise have been thoroughly investigated, and it is clear that high T_c increases strain on the cardiovascular system and perception of effort at a given exercise intensity, while reducing aerobic exercise capacity [89]. As T_c increases, blood flow is diverted to the skin to facilitate heat removal from the core [69] and cardiac output (Q) must increase to maintain blood pressure and oxygen delivery to the brain. In addition, elevations in T_c can directly increase heart rate (HR) by speeding the firing-rate of action potentials of the sinoatrial node [28], and can increase the force of heart contractions leading to increased stroke volume (SV). High T_c has even been proposed as direct cause of fatigue controlled by the CNS in an effort to prevent the body from reaching dangerously high temperatures. Therefore, it is obvious that high T_c can exert major influences on cardiovascular responses during exercise. However what is less clear, and often times

overlooked, is the role that skin temperature (T_{sk}) can have on cardiovascular strain, perceived exertion, and exercise capacity during exercise at any given T_c . In conditions of high ambient temperature or situations where heavy protective clothing or athletic equipment is worn, T_{sk} can become significantly elevated, leading to a narrowing of the thermal gradient from the core to the skin. In such conditions, adequate heat loss from the body cannot be achieved, leading to an elevation in T_c [69]. In addition, T_{sk} has a direct influence on skin blood flow (SBF) and HR, thereby leading to changes in Q , SV , and mean arterial pressure (MAP).

Therefore, T_{sk} can influence cardiovascular responses during exercise in 2 ways, first by causing direct effects on SBF and HR, and secondly, indirectly increasing T_c by preventing adequate heat loss from the body, leading to exacerbated stress on the cardiovascular system. In conclusion, high T_c and T_{sk} both affect cardiovascular responses during exercise; however, few studies have investigated the relative contribution of each independently. Therefore, the primary aim of this study was to examine how skin and core temperatures, independently and combined, affect cardiovascular responses during exercise.

REVIEW OF LITERATURE

Introduction

The following review of related literature describes the mechanisms by which heat stress, from both external and internal sources, affects cardiovascular function. Additionally, the impact of these heat-induced alterations in cardiovascular function are described as they relate to whole body dynamic exercise (i.e. running or cycling). The objective of this review is to describe the potential of skin and internal temperature to impact cardiovascular responses and explain the significant impact that each may have alone and in combination on exercise capacity.

External heating increases cardiovascular strain

Mechanisms of thermoregulation

Skin blood flow (SBF) is controlled by the net activity of 2 antagonistic branches of the sympathetic nervous system [39]. One branch controls vasoconstriction, mediated through noradrenergic neural control [39]. The other branch controls vasodilation in an active process that is less understood [34]. In response to cold, vasoconstrictor mechanisms are activated, while active vasodilator signal may become completely withdrawn [39], maintaining core temperature by preventing heat loss. Conversely, in response to heating, noradrenergic activity is withdrawn and the active vasodilator system is dominant [39], providing the necessary vasodilation in cutaneous vasculature through a nitric oxide dependent process [40, 90]. Increased SBF promotes heat removal from the body, contingent on skin temperature (T_{sk}) being cooled either by convective or evaporative cooling methods [75]. Interestingly, in normothermic resting humans,

increasing T_{sk} stimulates a reflex increase in SBF primarily mediated by decreased vasoconstrictor activity [57], with only a modest influence from the active vasodilator branch [38]. When core temperature (T_c) is elevated however, increasing T_{sk} stimulates increases in SBF primarily achieved via increased active vasodilator control [57]. This may indicate a graded response, where vasodilator mechanisms are not triggered until T_c reaches some threshold level. Another likely explanation is that active vasodilation is triggered by some combination of elevations in both T_c and T_{sk} .

Blood pressure regulation during heat stress

The delivery of blood and oxygen to the brain is essential, and can become compromised if mean arterial pressure (MAP) declines. MAP is therefore tightly regulated. When humans are passively heated by wearing a special garment that allows hot water to be perfused through tubing cover the entire body surface with the exception of the face, feet and hands, during supine rest, SBF can increase up to 7.5 L/min compared to 300 mL/min during normothermic conditions [65, 69]. Vasodilation of cutaneous vessels decreases total peripheral resistance (TPR) and increases cutaneous vascular conductance (CVC). These changes reduce central blood volume (CBV) and lead to a 2 – 6 mm Hg drop in central venous pressure (CVP) and a 5 – 10 mm Hg decline in MAP [14, 69]. Therefore, in response to large increases in SBF, cardiac output (Q), which is the product of stroke volume (SV) and heart rate (HR), must increase and vascular conductance in other vascular beds must decrease to prevent a decline in MAP and oxygen delivery to the brain [61, 67, 69]. When normal healthy individuals are passively heated vascular resistance in the splanchnic circulation increases, which

provides more blood to be available to perfuse the skin [67, 68, 70]. Additionally, in resting humans Q increases substantially during passive heating, and can approach values upwards of 13 L/min with most of the increase above resting values going to the skin [67]. The rise in Q during passive heating is accomplished primarily by increasing HR; however, it is interesting to note that SV is typically maintained or slightly elevated in these conditions [15, 46, 69, 88]. SBF is influenced not only by temperature, but also by blood pressure. For example if CVP is decreased by 7% during passive heating, SBF declines [13]. However, if CVP is maintained by supine cycling [26] or rapidly infusing saline intravenously [13], SBF will continue to increase with internal temperature. Therefore, cardiopulmonary baroreceptors also are a factor in SBF control during exercise [54].

Internal temperature influences cardiovascular responses

Heart rate response to warm core temperature

Chronotropic control of the heart in humans is controlled by neural and humoral factors [36]. Intrinsic heart rate (IHR) is a term used to describe the heart rhythm in absence of any external neural or humeral stimulation [36]. A single injection containing 0.2 mg/kg of propranolol and 0.04 mg/kg of atropine produces an essentially “isolated” heart model in for living humans, by blocking both adrenergic and cholinergic receptors [36]. Under these conditions the influence of temperature on heart rate has been determined in living humans, indicating that HR increased with a slope of 7 beats for every 1°C increase in T_c [37]. Internal body temperature has been demonstrated to directly impact IHR in baboons [28] with similar results. Gorman et al, determined that

40% of tachycardia, in heat stressed baboons was produced from non-autonomic sources [28, 37]. In addition, after beta adrenergic and cholinergic receptor blockade, it was established that HR increased with a slope of 8 beats for every 1°C increase in T_c [28]. Increased T_c has been proposed to increase HR by warming the blood perfusing the sinoatrial node, thereby increasing the intrinsic rhythm of cardiac pacemaker cells [80]. The influence of T_c on HR is well established [58, 72, 79, 85, 92], however it is less clear how skin temperature (warm or cool), can influence this relationship.

Stroke volume responses to warm core temperatures

SV is determined by CVP or “preload”, inotropic state or “contractility”, and arterial pressure or “afterload”. Furthermore, left ventricular end diastolic cross-sectional diameter alters the ability of the left ventricle to produce force, thereby affecting SV [73]. This concept is known as the Frank-Starling mechanism. Elevated temperature of the heart can increase the inotropic capacity of the cardiac contractile cells, thereby increasing SV [35, 66]. In other words heating the heart can decrease the amount of blood left in the ventricles after a contraction (end systolic volume “ESV”) [12, 14]. Enhanced contractility has been demonstrated to increase SV during passive heating even in the face of reductions in CVP [12, 14], providing some explanation for maintained Q during exercise with warm T_c .

As mentioned above “afterload” exerts an influence on SV, by increasing or decreasing the resistance to blood being ejected from the left ventricle. Heating decreases TPR and therefore afterload, which may explain the maintenance or slight increase in SV in heated subjects with attenuated CVP [86]. On the other hand, increased SBF when T_c

is elevated may reduce SV during exercise in the heat by reducing CBV [63]. This response of high SBF, however has been refuted by studies indicating that the decline in SV over time is not related to increased SBF [25]. In contrast, skin cooling at rest has been demonstrated to decrease conductance in both visceral and cutaneous vascular beds, leading to a 10 mm Hg increase in MAP [87], which would increase afterload potentially altering SV.

Collectively, these results demonstrate that SV is affected by several factors that can change with warm-hot core or skin, and the outcome will depend on the specific condition and given temperature dependent influence on preload, afterload, or contractility.

Cardiovascular effects of heat stress during exercise

Muscle blood flow during exercise in the heat

During dynamic exercise in warm environments, SBF increases in parallel with T_c to mediate heat loss from the body [33]. Elevation in SBF, required to prevent internal temperature from reaching dangerous levels, can increase up to 7.5 L/min and require proportionate increases in Q to maintain MAP [64]. On the other hand, oxygen demands of skeletal and cardiac muscle during dynamic exercise have the potential to require high blood flows [2]. When exercise is limited to a small muscle mass (i.e. quadriceps), rates of blood flow can reach $350\text{-}400\text{ mL}\cdot 100\text{ g}^{-1}\cdot \text{min}^{-1}$ [60, 62]. At these rates, maximal aerobic exercise involving 15 kg of active muscle mass would require cardiac output near 40 L/min [3]. Therefore, when dynamic exercise is conducted in warm-hot environments blood flow requirements for both skin and muscle far exceed the pumping capacity of the

heart [24]. Exercise in the heat has been classically described as a competition between skin and muscle for the available cardiac output [64]. However in the last 30 years data now exist to replace speculation of the past [24]. Studies examining muscle blood flow during exercise in the heat indicate that muscle blood flow is maintained during small [74], and large [23, 52] muscle mass activity. Even if muscle blood flow is maintained in warm-hot conditions, either at the expense of thermoregulation or some other source of blood redistribution, a question remains that remains is how does warm or cool skin in these conditions impact the cardiovascular system and thermoregulation.

Skin blood flow responses to heat stress during exercise

As discussed above, exercise in warm-hot conditions requires SBF to increase in relation to relative intensity of exercise [20], in an effort to remove heat from the body. Increased SBF works in tandem with sweating and evaporative cooling to maintain T_c [48]. During exercise a progressive increase in SBF has classically been considered as the primary cause of reduced SV and increased HR [64]. However the time course for reductions in SV and increases in HR are not temporally related with increases in SBF [25, 27]. Furthermore, SBF typically plateaus at a internal temperature of 38°C during exercise in the heat [10], yet during prolonged exercise when T_c is above this level, SV is progressively reduced and HR increased as temperature continues to increase over time. Therefore, a more likely explanation is that the decline in SV is related to the increase in HR [21], which can decrease SV by shortening the time for diastolic filling [83].

Cardiovascular drift

The condition alluded to above (i.e. increased HR, reduced SV, maintained or reduced Q during prolonged exercise) is commonly referred to as cardiovascular drift (CVD) [64]. Cardiovascular drift (CVD) has been proposed to occur from 1) blood pooling in skin causing a reduction in CBV, CVP, and SV [64], 2) increased whole body sympathetic activity [18], and 3) increased motor unit recruitment via central command [76]. However, regardless of proposed method, it appears that an increase in HR is the primary factor driving down SV [21]. Although “pooling” of blood in the cutaneous vessels is not the primary cause of CVD, it may influence SV when T_c and T_{sk} are high.

Progressive increases in SBF may lower SV when T_{sk} above 40°C [71], however T_{sk} is not typically this high and usually peaks within 20 min exercise [21]. During prolonged cycling, CVD is elicited when T_c and T_{sk} are elevated simultaneously [77], and is prevented when either T_{sk} or T_c is lowered [77]. This phenomenon has classically been explained by increased SBF decreasing venous filling pressure thereby lowering SV. It is likely however, that high skin and core temperature together elicit increased HR from increased sympathetic nervous system activity or in response to a decline in MAP as CVC increases, and the accelerated HR reduces time for diastolic filling [83] resulting in a lower SV [21].

Maximal oxygen consumption during exercise in the heat

Peak aerobic power production in trained individuals is reduced in the heat due to decline in Q and MAP resulting in decreased muscle blood flow prior to fatigue [22]. Furthermore, it was recently demonstrated that during maximal intensity, reductions in SV lead to a reduction in systemic oxygen delivery and eventually fatigue, and warm

environments exacerbate this phenomenon [50]. Hyperthermia reduces $\text{VO}_{2\text{max}}$ and increase the relative metabolic intensity at a given work rate [5]. Increased HR may be the primary factor leading to the reduction in oxygen delivery to skeletal muscle prior to fatigue, by restricting diastolic filling time, SV, and Q [22]. This hypothesis is supported by demonstrations that CVD that occurs after 45 min of exercise in the heat, increases HR by 12%, decreases SV by 16%, and reduces $\text{VO}_{2\text{max}}$ by 19% [89].

Influence of skin and core temperature on exercise performance

Perception of effort and fatigue

The role of peripheral (i.e. skin) versus central (i.e. core) temperature on thermoregulation and cardiovascular function has been thoroughly investigated [11, 25, 41, 51, 59, 71, 77, 91, 92], however it remains to be determined the relative influence skin and core temperature during exercise. It is clear that exercise in warm-hot environments impairs performance when T_c is elevated [9, 43], so it is not surprising that athletes can increase performance in the heat when they begin exercise with lower than normal internal temperatures [4, 42, 45, 58, 79, 82]. For example precooling runners' core temperature by 0.37°C prior to high intensity running lowers HR at a given time point during exercise, and improves time to fatigue by ~ 5 min, however at exhaustion, T_c and HR are similar in precooling compared to control conditions [42].

Furthermore, some researchers have put forth the concept that fatigue will result when T_c reaches a critical level (i.e. 40°C). Exercise in warm-hot environments will increase T_c and will lead to fatigue, usually near 40°C , in animals[84] and humans [27].

The central nervous system has been proposed to monitor internal temperature and down-regulate skeletal muscle activation before a dangerously high T_c is reached [44].

Decreased central drive is one mechanism put forth as a cause of premature fatigue during exercise with hyperthermia [44, 53]. Altered brain function in the frontal area is a factor decreasing performance during cycling in warm-hot conditions [53, 56]. Furthermore, the ability to generate force during maximal voluntary contractions in small muscle mass exercises, is impaired during whole body hyperthermia [55], which may offer some explanation for increased perception of effort at a given work rate in warm-hot environments [81].

It is interesting that during exercise in warm-hot climates subjects decrease work output to maintain a similar overall exertion as during exercise in cool environments [16, 82]. Exercise in the heat increases HR for a given work rate [5], thus another possibility is that subjects choose intensities that achieve the same relative HR during exercise warm-hot or cool conditions. Most studies linking fatigue to a high internal body temperature have overlooked the possible exacerbation or attenuation that warm or cool skin, respectively, can impose on exercise when T_c is elevated. For example, runners consistently achieve T_c above 40°C without decrements in performance [1, 17, 51], which is likely due to an effect of cooling the skin. Additionally, it was recently demonstrated that exercise in warm-hot environments decreases total work completed in a 15 min time trial by 17%, and reduces the ability to produce a “end spurt”, even without attainment of severe hyperthermia [16]. This result supports the concept that warm-hot skin can have negative effects before T_c nears the “critical core temperature”. Elevations in skin

temperature can also increase HR at a given core temperature during exercise [77], and have been shown to decrease maximal rates of oxygen consumption [41].

Therefore, it seems likely that some combination of skin and internal temperature limits exercise performance in the heat rather than a “critical” internal temperature alone. Collectively these studies demonstrate that skin temperature can have a major impact on cardiovascular responses and perception of effort during exercise that is often over-looked.

Rapid adjustments to skin temperature during exercise

In 1969, Rowell et al. studied the effects of rapidly changing skin temperature by perfusing water at different temperature through a tight fitting suit with rubbers tubes allow water to pass directly over most of the body surface while subjects exercised on a treadmill. During uninterrupted exercise, subjects were exposed to 21, 45, 10, and 50°C water perfusing the suit, for 30 min for the first 3 temperatures, and 15 min in the last stage. Unfortunately, only values for the beginning and end of each stage are presented and no major statistical analysis was performed in this study. Additionally, SBF was not measured. In this study, HR was very sensitive to changes in skin temperature over time, which also changed T_c making an interpretation of the data difficult. Increasing T_{sk} from 21 to 40°C raised HR by 41 beats over 30 min of exercise, compared to normothermic conditions. In contrast cooling T_{sk} from 40 to 10°C decreased HR back down to levels close to during the initial 30 min of exercise with 21°C water perfusing the suit. Increasing T_{sk} again to 50°C raised HR by 40 beats. It is evident from this study that high

T_{sk} has potential to elevated HR, and vice versa, however the speculation that skin temperature is creating this change in HR, by reducing CBV and SV seems unlikely.

Similarly, Shafftath and Adams reported that during 70 min of exercise when core and skin temperature are both elevated to levels that induce substantial CVD, cooling skin with fans reduces more than half of the drift that occurs over time in 5 min [77]. These authors speculated that skin cooling decreased SBF, thereby increasing CVP and SV, which allowed HR to decrease. The rapid response of HR in these studies is interesting, and demonstrates the important role that T_{sk} has on cardiovascular function during exercise, however the mechanism behind this response remain unclear.

Therefore, the primary purpose of the current investigation is to examine the cardiovascular responses during exercise, while systematically altering skin and core temperature. We hypothesize that both skin and core temperature will substantially impact cardiovascular responses and perception of effort during moderate intensity exercise.

METHODS

Subjects

Eight recreationally active males took part in this study. Subjects' average values for age, weight, height, and maximal oxygen uptake ($\text{VO}_{2\text{peak}}$) were 25.5 ± 1.6 years, 78.5 ± 4.6 kg, 177.4 ± 2.3 cm, and 4.2 ± 0.2 L/min respectively. $\text{VO}_{2\text{peak}}$ was determined at least 3 days prior to the first day of experimental testing during an incremental test to volitional exhaustion on an electromagnetically braked cycle ergometer (Excaliber Sport, Lode, Groningen, The Netherlands). Subjects were instructed to give their best effort, and continue as long as possible. Motivation was given throughout the test. Fatigue was considered to occur when cadence fell below 60 revolutions per min. All subjects were free from any known cardiovascular, musculoskeletal, or metabolic disease, or other medical condition that would impair their ability to perform strenuous exercise. Subjects were informed about all aspects of the study (i.e. equipment, protocol, measurement techniques) as well as any risks associated with their involvement in the study. All subjects gave written informed consent to participate in this study. The Institutional Review Board of The University of Texas at Austin approved all details of this study.

Research protocol

Instrumentation

Upon arriving at the Human Performance Laboratory (HPL), subjects voided their bladders, obtained a nude body weight, and inserted rectal thermistors. Body weights were also obtained post exercise and compared with starting values to determine rates of fluid loss during the exercise. A urine sample was obtained for determination of specific

gravity and hydration status. All subjects had urine specific gravity scores below 1.02 immediately prior to each experimental trial. Subjects then relaxed in a seated position, and inserted the esophageal temperature probe after thorough instruction, and under supervision of the P.I. or experienced member of the HPL.

Resting measurements

Afterward subjects rested while a heart rate (HR) monitor, and skin temperature thermistors were placed on the upper arm, forearm, chest, back, thigh, and calf. Subsequently, subjects mounted the cycle ergometer where resting measurements of oxygen consumption (VO_2), HR, cardiac output (Q), blood pressure, skin temperature, core temperature (T_c) and skin blood flow (SBF) were made. Measurements of resting conditions were collected in a thermoneutral environment (i.e. 22°C, 35-50 %RH).

Manipulation of core temperature

After resting measurements were collected, subjects walked to another room where the water tank for heating or cooling was located.

Pre-cooling (PC) protocol

Subjects entered a tank and were immersed to the level of the neck in cold water. Water temperature at the time when subjects entered was ~14°C and increased over time, as body heat warmed the water. Average water temperature when subjects emerged from water was ~17°C. To prevent local warming of water around the body, the tank was agitated every 3 to 5 min. Subjects sat comfortably in the bath, for 25 min or until esophageal temperature reached 36°C, after which they exited the water bath, dried their skin, changed into cycling clothing, and were helped to the cycle ergometer.

Pre-heating (PH) protocol

As in PC, subjects were immersed in water up to neck level. Water temperature at the time when subjects entered was $\sim 42.5^{\circ}\text{C}$ and decreased over time. Average water temperature when subjects emerged from water was $\sim 40^{\circ}\text{C}$. Subjects sat comfortably in the bath, for 25 min or until esophageal temperature reached 39.5°C , after which they exited the water bath, dried their skin, changed into cycling clothing, and were helped to the cycle ergometer. The magnitude of dehydration during exercise is associated with a proportional increase in core temperature and heart rate during exercise[47], therefore dehydration was prevented by providing Gatorade (The Gatorade Co., Chicago, IL) during PH, in proportion to the level of sweat losses.

Exercise protocol

After either PC or PH subjects mounted the ergometer wearing only spandex cycling shorts and cycling shoes with cleats that secure each foot to the pedal. Subjects were further instrumented with a NIRS probe placed on the vastus lateralis 10-12 cm proximal to the lateral edge of the patella, 2 laser Doppler probes on the dorsal region of the left forearm, and blood pressure cuff and ECG electrodes. Subjects then inserted a silicon mouthpiece and VO_2 was measured for 1-2 min followed by initiation of exercise at $65\% \text{VO}_{2\text{peak}}$. Average work rate for all subjects was 186.2 ± 11.4 watts. Cycling was conducted continuously for 20 min in all trials, while cardiovascular measurements were collected as described below. On the experimental day involving preheating and warming skin with heaters, after the initial 20 min of cycling, heaters were removed and fan cooling was initiated without any disruption of exercise. The purpose is this condition

was to examine the time course of changes in HR, Q, SV, mean arterial pressure (MAP), and skin blood flow (SBF) when skin was rapidly cooled. Measurements collected every 5 min in other trials were the same for the second 20 min of cycling in this condition. Only 4 subjects completed the full 20 min of exercise after the initiation of skin cooling, however all but 1 completed at least 5 min of exercise after fans were turned on. Therefore analysis of rapid changes in skin temperature was compared for these 7 subjects, at the end of the trial and 5 min after. All subjects began cycling <10 min after emerging from water. At the onset of exercise, skin cooling or heating was initiated as described below.

Manipulation of skin temperature

To achieve warm or cool skin respectively, parabolic heaters (Presto, Heat Dish, Eau Claire, WI) or fans were directed at the subject. In each case, a fan or heater was directed at the subject from 4 directions (front-right, front-left, back-right, back-left). Average wind-speed was 1.4 ± 0.1 m/sec. Heaters were placed in the “high” setting, and were placed 1 to 2 meters from the subject. Distance from subject varied depending on subjects’ tolerance to the skin heating, as to prevent burning the skin.

Measurements & Techniques

Testing equipment

Exercise testing was conducted on an upright electromagnetically braked ergometer (Excaliber Sport, Lode, Groningen, The Netherlands) modified with bicycle racing equipment. Saddle and bar height, reach, and seat angle were adjusted for the comfort of each subject. The same position was replicated during each subsequent trial.

Cardiovascular measurements

Impedance cardiography. During $\text{VO}_{2\text{peak}}$ tests, an impedance cardiography device (Physioflow Type PFO5L1, Manatec Biomedical, Macheren, France) was used to determine HR, SV, Q, and cardiac index (C.I.). Validity of this technique has been described previously[49].

Heart rate. Heart rate was collected continuously by a Suunto HR monitor system (Suunto Watches, Sylvan Lake, MI). Data was collected every second on a personal computer with custom software developed by Kevin Christmas. Data was averaged over a 30 sec period, which corresponded to measurements of Q and SBF. Average values for HR were used to calculate SV ($\text{SV} = \text{Q}/\text{HR}$).

Breath-by-breath oxygen consumption. A breath by breath system was used to measure VO_2 during rest and during exercise after PC or PH. Subjects breathed through a two-way non-rebreathing valve (Hans Rudolph, Kansas City, Missouri), with a dead space of 187 mL, connected to a pneumotachometer (Hans Rudolph, Kansas City, Missouri). Levels of oxygen and carbon dioxide gases were determined by a mass spectrometer (Perkin Elmer MGA 1100, St Louis, Missouri). Gas sampled during each breath was collected at the mouthpiece by a 6 foot capillary tube connected directly to an inlet of the mass spectrometer. Gas analysis was determined by custom software (Beck Integrated Physiological Testing Systems “BIPS”) developed by Ken Beck, Ph.D. BIPS and the mass spectrometer were calculated with high precision gases. Volume calibration consisted of pumping a 3-liter syringe (Hans Rudolph) at low, medium, and high rates of

flow. VO_2 was collected for 1-2 min prior to each cardiac output measurement. Values were averaged over a 30 second period.

Acetylene washin cardiac output. Cardiac output was determined via open circuit acetylene washin method as previously described in detail [32], and iterative values were used all calculations. Subjects were instructed to breath normally, and not cough or swallow during a cardiac output measurement, as these issues invalidate the measurement. After a full expiration, a two-way non-rebreathing valve was instantly switched from room air to a bag filled with mixed gases (0.7% acetylene, 9.0% helium, 21% oxygen, and balance nitrogen). Gases were medical grade and safe for use by humans. Subjects breathed from the bag for 8 to 10 breaths. Concentrations of helium and acetylene were monitored continuously via a sample line at the mouthpiece, connected directly by a 6-foot capillary tube to a mass spectrometer inlet. The mass spectrometer was calibrated prior to each trial with gases of known concentrations. Cardiac output was determined by BIPS software using a single alveolar one-compartment model[32]. Optimal time delays were determined for each subject. Q was measured during rest prior to PC or PH and during exercise at 5, 10, 15, and 20 min. During WW, after fans were turned on, additional Q measurements made at 5 min. intervals.

Skin blood flow. SBF was measured continuously by laser-Doppler flowmetry (moorLab, Moor Instruments) at 2 sites on the dorsal side of the left forearm. At 5 min intervals during exercise, for a 1-2 min duration, the left arm was suspended, at heart level, in a sling hanging from the ceiling. Q measurements were made while the arm was

in the sling. Allowing the arm to hang minimized movement artifact, and allowed for a more accurate measurement of skin blood flow. SBF is presented as averaged values for the 2 sites over the last 30 seconds of the period when the arm was in the sling.

Blood pressure. Blood pressure was determined at rest, and every 5 min, immediately after each cardiac output measurement, during cycling. Systolic and diastolic blood pressure was measured by auscultation at the brachial artery of the right arm with a microscope under a blood pressure cuff (Tango+, SunTech Morrisville, NC). Mean arterial pressure was calculated as $DBP + 1/3 \text{ pulse pressure}$. Total peripheral resistance (TPR) was calculated as $TPR = MAP/Q$.

Muscle oxygenation. Near infrared spectroscopy (NIRS) was used to determine muscle oxygenation during exercise. The NIRS probe was placed longitudinally on the vastus lateralis 10 cm proximal to the superior border of the patella. Probe placement was maintained by outlining the area on the skin around the probe. Markings were maintained by each subject between visits to the HPL. The probe was secured with a 2 inch thick elastic strap and covered by the subjects cycling shorts to prevent movement of the probe during cycling and to keep extraneous light from contaminating the NIRS signal. NIRS is a non-invasive technique that determines the percent of muscle oxygenation and deoxygenation at three different depths in the area under the probe, and is an index for muscle oxygen supply in the microvasculature. NIR light absorption characteristics of hemoglobin and myoglobin are indistinguishable; therefore values for [Hb] will represent changes in both myoglobin and hemoglobin, although contribution of myoglobin deoxygenation to the overall signal is small [19].

Temperature measurements

Core Temperature. Esophageal temperature was determined by a probe (Measurement Specialties, Dayton, Ohio) inserted into the esophagus via the nose and nasal cavity to a depth of $\frac{1}{4}$ standing height, which has been determined as the approximate distance to put the tip of the probe at heart level[45]. This method of determining core temperature is considered a good index of central blood temperature[78]. Subjects' inserted the probe themselves under the supervision and guidance of an experience member of the HPL team. Subjects were instructed about the anatomical structures of the nasal canal and esophagus and a visual demonstration (textbook description) of the anatomical structures for esophageal placement was provided when necessary. To further aid in the insertion process, subjects ingested a bolus of water while passing the probe past the glottis and down the esophagus. This aids in lubricating the upper digestive tract and stimulates the natural swallowing process. Rectal temperature was measured using a wire type thermistor (YSI 401, Ohio) inserted 12 cm past the anal sphincter. Subjects inserted the probe themselves in a private room adjacent to the laboratory after receiving verbal instruction. Lubrication (KY Jelly, Johnson and Johnson) was used to aid the insertion of the probe.

Skin temperature. Skin temperature was measured with surface thermistors (YSI model 409A, Yellow Springs, Ohio) attached to the skin at six sites (upper arm, forearm, chest, back, thigh, and calf). Skin temperatures are presented as a weighted mean as described previously by Hardy and DuBois [29].

Statistical Analysis

Mean, standard error (SE), and coefficient of variation were calculated for descriptive analysis. Data are reported as mean $\pm$ SE. Repeated measured ANOVA was used to analyze VO₂, HR, SV, Q, SBF, MAP, DBP, SBP, and TPR over time. Analysis of HR, SV, MAP, TPR, and SBF were considered planned comparisons. When a significant main effect was found, post hoc analysis was conducted with Tukeys HSD test. Statistical significance was defined as $P < 0.05$.

RESULTS

Influence of core temperature during exercise

Core temperature & cardiovascular variables

Immersion in cold or warm water significantly altered subjects' average T_{es} over the course of 20 min of exercise; 36.8 ± 0.1 and $38.1 \pm 0.2^{\circ}\text{C}$, ($P < 0.001$), for pre-cooled and pre-heated trials respectively. When T_{es} was warm, HR was significantly elevated, compared with cool core conditions, (Figure 2). Warm T_{es} during exercise tended to increase Q compared to cool conditions, 23.2 ± 0.8 vs. 22.2 ± 1.0 L/min, respectively, however this increase was not statistically significant ($P = 0.078$). In contrast, SV was reduced with warm T_{es} during exercise, compared with cool conditions, ($P < 0.05$), (Figure 2). Furthermore, warm T_{es} was associated with significant declines in both in MAP and TPR compared to when T_{es} was cool, 102.9 ± 4.2 vs. 114.7 ± 3.5 mmHg, ($P < 0.05$), and 357.4 ± 7.6 vs. 418.4 ± 18.5 dyne $\cdot\text{sec}^{-1}\cdot\text{cm}^{-5}$, ($P < 0.05$), respectively.

Influence of skin temperature during exercise

Skin temperature & cardiovascular variables

Warming the skin during exercise significantly increased average T_{sk} during exercise compared to fan cooling (wind speed 1.4 ± 0.1 m/sec), 28.7 ± 0.2 vs. $26.0 \pm 0.3^{\circ}\text{C}$. Warm T_{sk} conditions were associated with increased HR, and reduced SV compared to cool conditions, ($P < 0.05$), (Figure 2). Additionally, warm T_{sk} tended to increase Q during exercise compared with cool skin conditions, 23.0 ± 0.6 vs. 22.4 ± 0.6 L/min, ($P = 0.075$), however this was not significant. MAP was not altered by warm T_{sk} , and was 107.7 ± 3.4 and 109.9 ± 4.1 mmHg for warm and cool T_{sk} , respectively. Warm

T_{sk} during exercise lead to significantly reduced TPR compared with cool T_{sk} , 378.0 ± 15.6 vs., 397.8 ± 19.2 dyne*sec⁻¹*cm⁻⁵, ($P < 0.05$).

Combined influences of core and skin temperature during exercise

It is important to note that the T_{sk} and T_{es} were not static during exercise, and therefore cardiovascular responses were not in a steady state. Therefore, to more clearly isolate the cardiovascular responses that occurred as a result of T_{sk} or T_{es} temperature, precooling and preheating conditions are reported separately. Conditions during exercise are reported as; cool skin and cool core (CC), warm skin and cool core (WC), cool skin and warm core (CW), and warm skin and warm core (WW). Treatment effects for CC, WC, CW, and WW are shown in Table 1 as means $\pm$ SE for measurements collected at 5, 10, 15, and 20 min of exercise.

PRE-COOLING

Influence of skin temperature when core temperature is cool

Temperatures. T_{es} during CC and WC was similar at each time points, however T_{sk} was significantly higher in WC compared to CC, ($P < 0.05$). Table 2 shows temperature and cardiovascular data for warm and cool T_{sk} during exercise at 5, 10, 15 and 20 min, after precooling.

Cardiovascular variables. HR during WC compared to CC was 140.4 ± 4.5 , 153.0 ± 4.2 , 160.9 ± 4.2 and 164.3 ± 3.9 , vs. 139.0 ± 4.4 , 149.4 , 154.2 ± 3.7 , and 156.5 ± 4.3 beats/min at 5, 10, 15 and 20 min of exercise, respectively. There was no difference in HR between WC and CC at 5 min, however thereafter HR was significantly elevated in WC compared to CC, ($P < 0.05$), (Figure 3). HR increased similarly over time in both WC

and CC, with values at 10, 15, and 20 min being significantly higher compared to 5 min, ($P<0.05$). In WC, Q was only significantly higher at 20 min compared to CC, ($P<0.05$), (Table 2). Furthermore, Q increased over time in WC and was significantly higher at 15 and 20 min of exercise compared to values at 5 min, ($P<0.05$). In contrast, Q did not change over time in CC. There were no significant differences in SV at any time point between WC and CC (Table 2). Interestingly in CC, SV was significantly lower at 20 min of exercise compared to values at 5 min, ($P<0.05$), (Figure 3). During WC, SV was significantly lower than the 5 min point at 10 and 15, but not 20 min, of exercise, ($P<0.05$). MAP during WC compared to CC was 112.2 ± 3.9 , 116.7 ± 3.8 , 111.5 ± 3.5 , and 111.5 ± 3.5 , vs., 107.7 ± 6.3 , 116.0 ± 4.6 , 121.6 ± 4.3 , and 120.3 ± 4.5 mmHg at 5, 10, 15 and 20 min, respectively, (Figure 3). Compared to CC, MAP was significantly decreased at 15 and 20 of exercise during WC, ($P<0.05$). There were no significant changes in MAP over time in either condition. SBP was significantly lower at 20 min in WC compared to CC, but there were no differences at any other time during exercise (Table 2). In addition, there were no significant changes over time in either WC or CC. DBP tended to be attenuated throughout exercise in WC compared with CC, but was only significantly different at 15 min during exercise, ($P<0.05$), (Table 2). During WC, TPR was significantly reduced at 15 and 20 min, compared to CC, ($P<0.05$), (Table 2). During CC, TPR did not change over the course of exercise. In contrast, during WC TPR was significantly lower at 20 vs. 5 min during exercise, ($P<0.05$). In WC, SBF was significantly higher at 10, 15, and 20 min during exercise compared to CC, ($P<0.05$), (Figure 3). In CC, SBF remained low and relatively constant throughout exercise,

however, in WC SBF increased over time and was significantly elevated at 10, 15, and 20 min compared to values at 5 min, ($P<0.05$), (Table 2). CVC was significantly greater during WC at 10, 15, and 20 min of exercise compared to CC, ($P<0.05$), (Table 2). In WC, CVC increased significantly over time, however in CC CVC remained unchanged.

Perceived effort. There were no significant differences in RPE in WC compared to CC, (Table 2), however, it is interesting to note that RPE was significantly increased during WC at 15 and 20 min vs. the 5 min value, ($P<0.05$).

PREHEATING

Influences of skin temperature when core temperature is warm

Temperatures. T_{es} was only significantly higher in WW compared to CW at 10 and 15 during exercise, ($P<0.05$). Temperature and cardiovascular data for WW and CW at 5, 10, 15, and 20 min of exercise are shown in Table 3. During CW, T_{es} remained relatively constant for the duration of exercise, however in WW, T_{es} increased over time and was significantly elevated at 15 and 20 min, compared to the 5 min value, ($P<0.05$). T_{sk} was significantly higher in WW compared with CW at all time points during exercise, ($P<0.001$). T_{sk} increased over time in both WW and CW, and was significantly higher at 10, 15, and 20 min compared to 5 min, in both conditions, ($P<0.05$).

Cardiovascular measurements. HR during WW compared to CW was 168.7 ± 2.5 , 175.2 ± 3.2 , 178.0 ± 3.7 , and 181.0 ± 3.3 , vs., 157.1 ± 3.4 , 160.0 ± 3.6 , 162.6 ± 3.8 , and 161.7 ± 3.7 beats/min at 5, 10, 15 and 20 min, respectively. In WW compared to CW, HR was significantly higher at each time point during exercise, ($P<0.05$), (Figure 4). In both WW and CW, HR increased significantly over time, and was significantly higher at

10, 15, and 20 min compared with the 5 min value, ($P<0.05$). There were no significant differences in Q between WW and CW at any time point during exercise, (Table 3).

During WW, Q was significantly higher at 20 min of exercise compared to value at 5min, ($P<0.05$). In CW, Q changed over time, and was significantly higher at 10 and 15 min of exercise compared to 5 min ($P<0.05$). SV was significantly lower in WW compared to CW at 5, 15, and 20 min of exercise, ($P<0.05$), (Figure 4). In both WW and WC, SV did not change significantly during exercise. In WW, MAP was significantly lower at 15 and 20 min, compared to CW, ($P<0.05$), (Figure 4). There were no significant changes in MAP over time in either WW or WC. SBP during WW compared to CW was 181.8 ± 8.9 , 188.0 ± 7.7 , 187.8 ± 7.6 , and 185.6 ± 7.7 vs., 168.8 ± 9.8 , 177.3 ± 11.1 , 190.4 ± 10.3 , and 188.9 ± 8.5 mmHg at 5, 10, 15 and 20 min, respectively. During WW, SBP was significantly elevated above the 5 min value at 10 and 15, but not 20 min. DBP during WW compared to CW was 63.3 ± 5.1 , 60.0 ± 4.7 , 59.8 ± 3.2 , and 56.8 ± 2.0 vs., 60.3 ± 6.4 , 63.1 ± 6.0 , 67.3 ± 4.9 , and 67.3 ± 4.3 mmHg at 5, 10, 15 and 20 min, respectively. In WW, DBP was significantly lower at 15 and 20 min, compared to CW, ($P<0.05$). TPR was significantly different at 20 min in WW compared to CW, (Table 3). During WW, TPR decreased slightly over time and was significantly decreased at 20 min of exercise compared to 5 min, ($P<0.05$). SBF was significantly higher in WW compared to CW at 5 and 20 min of exercise, ($P<0.05$), (Figure 4). In WW compared to CW, CVC was slightly elevated at 5, 10, and 15 min of exercise (n.s.), however only reached significance at 20 min, ($P<0.05$).

Perceived effort. In WW, RPE was significantly higher than CW at 15 and 20 min. of exercise, ($P<0.05$), (Table 3). In WW, RPE increased steadily throughout exercise, and was significantly higher at 15 and 20 min compared to values at 5 min, ($P<0.05$). Interestingly, in CW, RPE did not change over time during exercise.

Rapid skin cooling during exercise with warm core temperature

In the WW condition, after 20 min of exercise, 7 subjects ($n=7$) had their skin rapidly cooled by removing heaters and initiating fan cooling. One subject's performance deteriorated severely during this condition and fatigued prior to finishing the initial 20 min, and therefore was excluded from the analysis. Another subject, did not reach volitional fatigue, however his esophageal temperature reached the maximal temperature we allowed in this study at 15 min, thus fan cooling in this subject began at this point rather than at 20 min of exercise as in other subjects. To determine time course of changes in cardiovascular responses to skin cooling, 4 subjects continued cycling for 20 min after the initiation of fan cooling. Due to the small sample size ($n=4$), differences were not significant at any time, however, it is noteworthy that trends for differences in HR, SV, and RPE existed at 5 min, but not at any other time point, compared to min 20 in WW.

Temperatures. As expected, initiation of cooling significantly reduced T_{sk} by $7.0 \pm 0.3^{\circ}\text{C}$, 5 min after skin cooling began compared to the end of WW, ($P<0.001$), (Figure 5). T_{sk} at the end of WW and after 5 min of skin cooling was 33.4 ± 0.3 and $26.4 \pm 0.2^{\circ}\text{C}$, respectively, ($P<0.001$). During this same period, there was a small (n.s.) drop in T_{es} ($0.2 \pm 0.1^{\circ}\text{C}$), from 38.8 ± 0.2 to $38.5 \pm 0.1^{\circ}\text{C}$.

Cardiovascular responses. Rapid cooling of the skin significantly reduced HR by 10.8 ± 1.2 beats/min, from 183.0 ± 0.1 at the end of WW, to 172.2 ± 3.9 after 5 min of fan cooling ($P < 0.05$), (Figure 5). Despite this reduction in HR, there were no statistical differences in Q (23.7 ± 0.5 vs. 23.0 ± 0.9 L/min), or SV (130.0 ± 4.4 vs. 133.9 ± 6.3), for end of WW and 5 min after initiation of fan cooling, respectively. Similarly, values for MAP (99.7 ± 2.9 vs. 99.3 ± 3.4 mm Hg) and TPR (333.3 ± 12.8 vs. 350 ± 20.7 dynes*sec⁻¹*cm⁻⁵) were not different before and after skin cooling, respectively.

Perceived effort. Rapid skin cooling reduced RPE by 1.6 ± 0.5 Borg scale units ($P < 0.05$), from the end of WW compared with 5 min after initiation of skin cooling, 16.0 ± 0.7 vs., 14.4 ± 0.9 Borg scale units, respectively, ($P < 0.05$), (Figure 5).

Skin blood flow. Skin cooling had no effect on SBF, which remained elevated 5 min after initiation of skin cooling, 125.0 ± 7.0 vs. 120.4 ± 11.1 a.u. for end of WW and 5 min after skin cooling, respectively, (Figure 5). CVC was also unchanged by skin cooling (1.3 ± 0.1 vs. 1.2 ± 0.1 volts*mmHg⁻¹, before and after 5 min, respectively).

Time course of cardiovascular changes (n=4). Data presented in this section are only for 4 subjects who completed the entire 20 min of cycling with fans, immediately after WW. At the 20 min point in WW, T_{sk} was $33.5 \pm 1.2^{\circ}\text{C}$, (n=4). All values for T_{sk} after fan cooling began were significantly lower than values at the end of WW, ($P < 0.05$). After the initial decline in T_{sk} , by 5 min, temperatures remained fairly constant, however there was a slight decline after 20 min, ($P < 0.05$). At the end of WW, HR was 182.1 ± 7.8 , and was decreased to 172.0 ± 10.4 , 168.5 ± 12.4 , 168.0 , and 166.9 after 5, 10, 15, and 20 min of skin cooling, respectively, however there were no significant differences due to

the small sample, (n=4). It is noteworthy that in this small sample HR was close to becoming significant, (P=0.053), after 5 min of fan cooling compared to values at the end of WW. In contrast, cooling the skin at the end of WW, did not lead to significant changes in Q. MAP after skin cooling was 94.9 ± 7.6 , 92.3 ± 6.7 , 92.1 ± 9.5 , 94.5 ± 6.7 vs., 96.4 ± 8.0 mmHg at 5, 10, 15, and 20 min of fan cooling, and the end of WW, respectively. There were no significant differences in MAP, TPR, SBF, or CVC after skin cooling compared to values at the end of WW. RPE after skin cooling was reduced however differences were not significant, 12.8 ± 0.5 , 12.8 ± 0.5 , 12.8 ± 0.5 and 12.0 ± 0.7 vs., 15.0 ± 0.8 Borg scale units at 5, 10, 15, and 20 min of fan cooling, and the end of WW, respectively. T_{es} was not statistically different at any time after skin cooling was 38.5 ± 0.2 , 38.2 ± 0.2 , 38.0 ± 0.3 , and 38.1 ± 0.2 vs., $38.6 \pm 0.2^{\circ}\text{C}$ at 5, 10, 15, and 20 min of fan cooling, and the end of WW, respectively.

Figure Legends

Figure 1. Study design used to investigate the cardiovascular responses during exercise with systematic alterations in core and skin temperature. Arrows indicate collapsed comparisons between warm and cool skin conditions (dashed line), and warm and cool core conditions (solid line), (n=16).

Figure 2. Influence of core and skin temperature on A) heart rate and B) stroke volume during exercise. Values are means $\pm$ SE for the average of all measurements made during exercise after preheating (warm core) or precooling (cool core), (n=16). *Significantly different from corresponding cool condition. The 2x2 table to the right, indicates numerical values for cool core conditions (CC and WC), warm core conditions (CW and WW), cool skin conditions (CC and CW), and warm skin conditions (WC and WW). Values are means for data collected at 5, 10, 15, and 20 min of exercise.

Figure 3. Heart rate, stroke volume, mean arterial pressure, and skin blood flow during exercise with either cooled or warmed skin, after precooling, (n=8). Values are means $\pm$ SE for data collected 5, 10, 15, and 20 min of exercise. *Significantly different from cool core condition. #Significantly different from 5 min value.

Figure 4. Heart rate, stroke volume, mean arterial pressure, and skin blood flow during exercise with either cooled or warmed skin, after preheating, (n=8). Values are means $\pm$ SE for data collected 5, 10, 15, and 20 min of exercise. *Significantly different from cool core condition. #Significantly different from 5 min value.

Figure 5. Heart rate, skin blood flow, Perceived exertion, and skin temperature at the end of WW (warm), and 5 min after removing heaters and initiating fan cooling (cool).

Values are means $\pm$ SE, (n=7). †Significantly different from warm skin, $P<0.001$.

*Significantly different from warm skin, $P<0.05$.

Table Legends

Table 1. Cardiovascular effects of altering skin and core temperature during exercise; conditions during exercise are cool skin & cool core (CC), warm skin & cool core, cool skin & warm core (CW), or warm skin & warm core (WW). Values are means $\pm$ SE for time points 5, 10, 15, & 20 min of exercise in 8 subjects (n=8). T_{es} , esophageal temperature; T_{sk} , mean skin temperature; VO_2 , oxygen consumption; HR, heart rate; Q, cardiac output; SV, stroke volume; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; TPR, total peripheral resistance; SBF, skin blood flow; CVC, cutaneous vascular conductance; RPE, rating of perceived exertion.

*Significantly different from CC. †Significantly different from WW. ‡WC significantly different from CW.

Table 2. Cardiovascular variables during exercise after pre-cooling with either skin cooling (CC) or skin warming (WC). Values are means $\pm$ SE for 8 subjects. T_{es} , esophageal temperature; T_{sk} , mean skin temperature; VO_2 , oxygen consumption; HR, heart rate; Q, cardiac output; SV, stroke volume; MAP, mean arterial pressure; DBP, diastolic blood pressure; TPR, total peripheral resistance; SBF, skin blood flow; CVC, cutaneous vascular conductance; RPE, rating of perceived exertion; Δ , change from 5 to 20 min of exercise. *Significantly different from cool skin condition. #Significantly different from 5 min. †Significantly different from 20 min.

Table 3. Cardiovascular variables during exercise after pre-heating with either skin cooling (CW) or skin warming (WW). Values are means $\pm$ SE for 8 subjects. T_{es} , esophageal temperature; T_{sk} , mean skin temperature; VO_2 , oxygen consumption; HR,

heart rate; Q, cardiac output; SV, stroke volume; SBP, systolic blood pressure; DBP, diastolic blood pressure; TPR, total peripheral resistance; SBF, skin blood flow; CVC, cutaneous vascular conductance; RPE, rating of perceived exertion; Δ , change from 5 to 20 min of exercise. *Significantly different from cool skin condition. #Significantly different from 5 min. †Significantly different from 20 min.

Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5.

Table 1.

Variable	CC	WC	CW	WW
T _{es} , °C	36.7 ± 0.1	36.9 ± 0.1††	37.8 ± 0.2*	38.3 ± 0.2*
T _{sk} , °C	22.3 ± 0.3	29.7 ± 0.4*††	24.6 ± 0.2*†	32.7 ± 0.3*
VO ₂ , L/min	3.0 ± 0.2	2.9 ± 0.2	3.0 ± 0.2†	2.9 ± 0.1
%VO _{2peak}	70.3 ± 1.6	67.7 ± 1.4	70.3 ± 1.7	67.4 ± 1.4
HR, beats/min	149.8 ± 4.0	154.7 ± 4.1*†	160.3 ± 3.5*†	175.8 ± 3.2*
%HR _{max}	77.4 ± 1.4	79.9 ± 1.2*†	82.9 ± 1.0*†	90.9 ± 1.0*
Q, L/min	21.9 ± 1.0	22.6 ± 0.9	23.0 ± 0.8	23.4 ± 0.8
SV, mL/beat	147.7 ± 9.8	147.7 ± 9.0†	144.4 ± 7.8†	133.9 ± 7.0
MAP, mmHg	116.4 ± 4.1	113.0 ± 3.0†	103.4 ± 4.9	102.4 ± 3.8*
SBP, mmHg	191.0 ± 8.8	191.1 ± 8.2	181.3 ± 7.4	185.8 ± 7.8
DBP, mmHg	79.1 ± 4.0	73.9 ± 3.9†	64.5 ± 5.0*	60.0 ± 3.4*
TPR, dyne*sec ⁻¹ *cm ⁻⁵	431.9 ± 22.1	404.8 ± 15.9†	363.7 ± 20.9	351.1 ± 16.3*
SBF, a.u.	30.4 ± 4.0	65.5 ± 5.88*††	107.8 ± 8.5*	128.5 ± 6.2*
CVC, 100 * (V* mmHg ⁻¹)	26.1 ± 2.9	58.5 ± 4.9*††	105.9 ± 7.3*	127.5 ± 6.4*
RPE, Borg scale units	12.5 ± 0.6	13.5 ± 0.7	13.1 ± 0.7†	15.1 ± 0.7*

Table 2.

Variable	Condition	5	10	15	20	Δ
T_{es} , °C	CC	35.9 ± 0.1	36.5 ± 0.1#†	37.0 ± 0.1#†	37.5 ± 0.2#	1.6 ± 0.1
	WC	35.9 ± 0.1	36.6 ± 0.1#†	37.4 ± 0.1*#†	37.7 ± 0.2#	1.8 ± 0.1
T_{sk} , °C	CC	20.5 ± 0.3	21.9 ± 0.3#†	23.0 ± 0.3#†	23.7 ± 0.3#	3.2 ± 0.2
	WC	26.9 ± 0.5*	28.9 ± 0.4*#†	31.0 ± 0.5*#†	32.1 ± 0.4*#	5.2 ± 0.3*
VO_2 , L/min	CC	2.8 ± 0.2	3.0 ± 0.2#	3.0 ± 0.2#	3.1 ± 0.2#	0.2 ± 0.1
	WC	2.7 ± 0.2	2.9 ± 0.2#	3.0 ± 0.2#	3.0 ± 0.2#	0.3 ± 0.1
HR, beats/min	CC	139.0 ± 4.4	149.4 ± 3.8#†	154.2 ± 3.7#†	156.5 ± 4.3#	17.5 ± 2.3
	WC	140.4 ± 4.5	153.0 ± 4.2*#†	160.9 ± 4.2*#†	164.3 ± 3.9*#	23.9 ± 2.2*
Q , L/min	CC	21.3 ± 1.1	22.0 ± 1.4	22.5 ± 0.9	21.7 ± 1.0	0.4 ± 0.6
	WC	21.6 ± 1.1	21.9 ± 0.8†	23.0 ± 1.0#†	23.9 ± 0.8*#	2.4 ± 0.5*
SV, mL/beat	CC	154.8 ± 10.2	148.6 ± 11.8	147.3 ± 8.9†	140.1 ± 9.6#	-14.7 ± 3.9
	WC	155.5 ± 11.6	144.0 ± 7.6#	144.3 ± 9.6#	146.8 ± 8.0	-8 ± 4.9
SBP, mmHg	CC	173.6 ± 12.6	189.0 ± 11.6	200.3 ± 9.3	201.0 ± 9.0	27.4 ± 13.2
	WC	180.0 ± 10.9	197.5 ± 10.3	195.4 ± 8.8	191.6 ± 8.8*	11.6 ± 11.0
DBP, mmHg	CC	74.8 ± 4.6	79.5 ± 4.9	82.3 ± 4.8	80.0 ± 3.6	5.3 ± 4.1
	WC	78.3 ± 4.2	76.3 ± 3.9	69.5 ± 5.3*#	71.5 ± 4.3	-6.8 ± 3.1
TPR, dyne*sec ⁻¹ *cm ⁻⁵	CC	409.9 ± 28.9	432.4 ± 29.5	435.5 ± 19.3	449.6 ± 22.9	39.7 ± 17.9
	WC	424.4 ± 28.2	429.4 ± 17.2†	391.7 ± 16.5*†	373.9 ± 10.7*#	-50.5 ± 21.2*
SBF, a.u.	CC	26.1 ± 3.3	26.6 ± 2.0	29.8 ± 4.2	39.3 ± 7.9	13.2 ± 6.2
	WC	26.9 ± 2.2	43.1 ± 5.7*#†	73.0 ± 11.3*#†	118.9 ± 9.2*#	92.0 ± 8.9*
CVC, 100*(V*mmHg ⁻¹)	CC	24.5 ± 3.1	23.2 ± 1.9	24.3 ± 2.9†	32.5 ± 5.9	8.0 ± 5.9
	WC	24.1 ± 1.8	37.0 ± 4.5*#†	65.6 ± 9.3*#†	107.2 ± 8.8*#	83.2 ± 8.3*
RPE, Borg scale units	CC	12.1 ± 0.5	12.5 ± 0.5	12.6 ± 0.7	12.6 ± 0.8	0.5 ± 0.4
	WC	12.8 ± 0.6	13.4 ± 0.6	13.9 ± 0.7#	14.0 ± 0.8#	1.3 ± 0.3*

Table 3.

Variable	Condition	5	10	15	20	Δ
T_{es} , °C	CW	37.8 ± 0.2	37.8 ± 0.2	37.8 ± 0.2†	37.9 ± 0.2	0.1 ± 0.1
	WW	37.8 ± 0.1	38.3 ± 0.1#†	38.5 ± 0.2*#†	38.7 ± 0.2*#	1.0 ± 0.1*
T_{sk} , °C	CW	24.1 ± 0.2	24.5 ± 0.2#†	24.7 ± 0.2#†	24.9 ± 0.2#	0.8 ± 0.1
	WW	32.2 ± 0.4*	32.7 ± 0.4*#†	32.9 ± 0.4*#†	33.2 ± 0.3*#	1.0 ± 0.2
VO ₂ , L/min	CW	2.8 ± 0.2	3.0 ± 0.1#	3.1 ± 0.2#	3.1 ± 0.2#	0.4 ± 0.1
	WW	2.6 ± 0.2	2.9 ± 0.1#	3.0 ± 0.1#	3.0 ± 0.1#	0.4 ± 0.1
HR, beats/min	CW	157.1 ± 3.4	160.0 ± 3.6#	162.6 ± 3.8#	161.7 ± 3.7#	4.6 ± 1.7
	WW	168.7 ± 2.5*	175.2 ± 3.2*#†	178.0 ± 3.7*#†	181.0 ± 3.3*#	12.3 ± 1.3*
Q, L/min	CW	21.9 ± 1.0	23.0 ± 1.0#	23.7 ± 0.6	23.4 ± 0.9	1.5 ± 0.9
	WW	22.4 ± 1.1	23.5 ± 0.9	23.5 ± 0.8#†	24.3 ± 0.7#	1.9 ± 0.5
SV, mL/beat	CW	140.8 ± 9.3	144.8 ± 9.4	146.7 ± 6.5	145.4 ± 7.7	4.6 ± 5.1
	WW	133.4 ± 8.4*	134.8 ± 7.4	132.9 ± 7.1*	134.7 ± 6.0*	1.4 ± 2.7
SBP, mmHg	CW	168.8 ± 9.8	177.3 ± 11.1	190.4 ± 10.3	188.9 ± 8.5	20.1 ± 12.0
	WW	181.8 ± 8.9	188.0 ± 7.7#	187.8 ± 7.6#	185.6 ± 7.7	3.9 ± 3.8
DBP, mmHg	CW	60.3 ± 6.4	63.1 ± 6.0	67.3 ± 4.9	67.3 ± 4.3	7.0 ± 3.7
	WW	63.3 ± 5.1	60.0 ± 4.7	59.8 ± 3.2*	56.8 ± 2.0*	-6.4 ± 4.8*
TPR, dyne*sec ⁻¹ *cm ⁻⁵	CW	357.5 ± 34.2	356.0 ± 26.9	366.9 ± 17.9	374.4 ± 22.3	17.0 ± 29.7
	WW	372.8 ± 25.0	353.3 ± 19.1	350.8 ± 14.5†	327.3 ± 12.7*#	-45.6 ± 18.7
SBF, a.u.	CW	103.6 ± 9.4	109.7 ± 9.1	118.6 ± 9.9†	99.6 ± 8.8	-4.0 ± 3.9
	WW	143.6 ± 7.1*	121.7 ± 9.3	120.8 ± 10.6	127.9 ± 6.8*	-15.7 ± 7.3
CVC, 100*(V* mmHg ⁻¹)	CW	111.0 ± 12.8	110.8 ± 10.0	109.9 ± 8.3	91.9 ± 6.1	-19.1 ± 9.9
	WW	142.5 ± 11.0	119.1 ± 8.6	118.0 ± 10.4	130.5 ± 6.6*	-12.0 ± 11.7
RPE, Borg scale units	CW	12.9 ± 0.6	13.1 ± 0.6	13.3 ± 0.8	13.3 ± 0.8	0.4 ± 0.5
	WW	14.0 ± 0.7	14.6 ± 0.7	15.5 ± 0.7*#	16.4 ± 0.7*#	2.4 ± 0.4*

DISCUSSION

The purpose of this study was to examine how skin and core temperatures, independently and combined, affect cardiovascular responses during exercise. Specifically this study had 3 primary aims; 1) describe the overall effect of T_{sk} and T_{es} on HR, SV, Q, MAP, and SBF during exercise, 2) test the hypothesis that T_{sk} has profound effects on cardiovascular function and exercise performance when T_c is warm and cool, and 3) evaluate how HR, SV, Q, MAP, SBF, and RPE respond when T_{sk} is rapidly cooled during exercise with elevated T_{es} .

Primary findings

Cardiovascular responses during exercise in warm-hot conditions consistently demonstrate elevations in HR and reductions in SV, however the contribution of skin and core on these responses is not well established. The first major finding of the current study is that warm T_{es} and T_{sk} independently increase HR and reduced SV during 20 min of moderate intensity exercise (Figure 2), however the influence of warm T_{sk} on SV only occurs when T_{es} is elevated (i.e. $>38^{\circ}\text{C}$), Figure 4 . The average HR over 20 minutes of moderate intensity exercise was 10 beat/min higher when skin was warm, and 15 beats/min higher when core was warm, compared cool skin and core respectively. Additionally, SV was 5 mL/beat lower when skin was warmed, and 9 mL/beat lower when core was warm, compared to cool skin and cool core respectively, Figure 2.

Exercise with warm T_{sk} tended to increase Q, however this result was not significant, ($P=0.075$). Additionally, warm T_{sk} significantly reduced MAP and TPR compared with when skin was cool. Furthermore, in warm compared to cool core

conditions, the elevation in HR was associated with significant reductions in SV, TPR, and MAP. Average Q was elevated with warm core conditions, however was not statistically different from cool core, ($P=0.078$). The elevation in HR and reductions in SV when skin was warm, demonstrates that T_{sk} is an important factor contributing to increased cardiovascular strain during exercise.

The second major finding of this study was that warm T_{sk} increases HR and RPE, and reduces MAP during exercise when T_{es} is cool and warm. By comparing the influence of warm vs. cool skin after subjects were preheated or precooled, we were able to demonstrate that warm skin increases HR by 5 to 15 beats/min, with the least influence resulting when T_{es} is lowest and greatest where T_{es} is highest, (Table 1). The only point where warm T_{sk} did not influence HR was at the 5 min point in WC, where T_{es} was only 35.9°C , (Table 2). At this T_{es} , the impact of warm skin may be overpowered by the thermoregulatory and cardiovascular responses that result from such a low internal temperature.

Additionally, RPE increased over time during exercise with warm skin, regardless of whether subjects had been preheated or precooled. This finding demonstrates that when skin is warm exercise at a given intensity “feels” more difficult, regardless of T_{es} . Thus, it must be considered that T_{sk} may play a major part of the premature fatigue that occurs during exercise in the heat. The last major finding of this study is that rapidly reducing T_{sk} from 33.4 ± 0.3 to 26.4 ± 0.2 is associated with a significant decline in HR (10.8 ± 1.2 beats/min, $P<0.001$), and RPE (1.6 ± 0.5 Borg scale units, $P<0.05$), without

any significant change in T_{es} , or SBF. This result indicates that perception of effort and HR are both directly influenced by T_{sk} .

The current finding that warm T_{es} increased HR and reduced SV was not surprising, and corroborates previous findings. In 1966 Rowell et al, found that during exercise on a treadmill, healthy untrained men, not acclimatized to the heat, exhibited elevations in HR, and reductions in SV while walking at 3.5 mph at 4 incremental grades (7.5, 10.0, 12.5, and 15%) [63]. In their study SV was substantially reduced in 43.3°C compared with 25.6°C environmental temperatures, which lead to reductions in Q, even with extremely elevated HR. It was concluded that the hot conditions elicited high SBF and low CBV, which reduced SV, leading to increased HR in an effort to maintain Q. Similar to our current finding, these authors found that exercise in warm conditions increased HR and reduced SV. However unlike our present study where we observed slightly higher Q when T_{es} was warm, Rowell observed reductions in Q by 1.0 to 1.2L in the 43.3°C environment [63]. In addition, Nadel et al, found that HR was increased and SV was decreased during exercise in warm conditions when T_{es} and T_{sk} was elevated [51]. In their study, recreationally active subjects conducted exercise on a cycle ergometer at 40 and 70 % VO_{2max} in 3 different environmental conditions (i.e. 20, 26, and 36°C), which produced mean T_{sk} of 32.0, 33.6, and 35.5°C, respectively. In support of our findings, these authors demonstrated that Q was either maintained or elevated in the heat depending on the relative intensity of exercise. Interestingly, during low intensity exercise in the 36°C environment, Q was elevated by 1.3 L/min, however at the higher work rates at this temperature there was no difference. These authors concluded that at

the higher intensities it becomes increasingly difficult to increase Q, as HR is near maximal and SV is attenuated due to reduced cardiac filling pressure.

Our current results seem to indicate a similar phenomenon, because although we observed large differences in HR between conditions ($77.4 \pm 1.4 \%HR_{\max}$ in CC compared to $90.0 \pm 1.0 \%HR_{\max}$ in WW), we did not observe any significant differences between Q between any treatments. This indicates that subjects were working at or near their maximal Q, for the given condition, by the end of WW where T_{sk} and T_{es} were both elevated throughout exercise and reached 38.7 ± 0.2 and $33.2 \pm 0.3^{\circ}C$, respectively, by the 20 min point in exercise, (Table 3). Therefore it seems clear that increased environmental temperature can increase HR and reduce SV, most likely by increasing both T_{es} and T_{sk} . The contrasting Q response observed by Rowell et al, compared to our current findings and those of Nadel, may be related to the different exercise modality used by Rowell (i.e. treadmill walking). Walking on a treadmill may further attenuate CVP due to the upright posture compared to cycling, thereby lowering Q during exercise in the heat. Additionally, treadmill walking may elicit less activation of the muscle pump to facilitate venous return compared to cycling exercise and therefore may be another reason why Q was decreased in there study. Furthermore, in their study, Rowell et al had subjects exercise in a $43.3^{\circ}C$ environment that produced rectal temperatures of 37.9, 38.6, 39.1, and 39.4 during walking at 3.5 mph on a treadmill at 7.5, 10.0, 12.5, and 15% grade, respectively. These temperatures are similar to values we observed in the current investigation after preheating, however their cool environment (i.e. $25.6^{\circ}C$) produced internal temperatures that were much higher than our current investigation (37.6, 38.0,

38.0 and 38.2°C at 7.5, 10.0, 12.5, and 15% grade, respectively compared with our average T_{es} of $36.8 \pm 0.1^\circ\text{C}$). Therefore our “cool conditions” are more than 1°C lower than those of Rowell et al, which may be another factor when comparing the differences between “warm” and “cool” conditions in their trial and our current one. These authors speculated that increased SBF would substantially reduce CVP and SV in the heat and this condition would be reversed in cool conditions. Based on this theory, exercise in cool conditions, such as our current study, would produce low SBF thereby maintaining CVP, SV, and Q. In direct contrast, our current results show that SV is not reduced when SBF increases while T_{es} is cool, and furthermore may actually facilitate the maintenance in SV by reducing after load, Figure 3. The lack of a significant difference in Q during exercise in CC and WC, in our present study indicates that large differences in SBF did not have a significant affect on SV during exercise.

In support of this finding, Gonzalez-Alonso demonstrated that euhydrated subjects have significantly higher HR, Q, and SBF during exercise in a 35°C environment compared to exercise in a 8°C environment, however SV is not reduced[25]. This study demonstrates that increased T_c is associated with increased HR, however if euhydration and thus, blood volume are maintained, SV is not reduced during exercise in warm conditions, even with high SBF. In contrast, in the present investigation we observed reductions in SV, which may be related to the higher T_{es} we achieved ($38.7 \pm 0.2^\circ\text{C}$), or the fact that our subjects were not endurance trained. Together these previous studies support the finding that high environmental conditions can increase HR and reduce SV, however they do not isolate contribution that T_{es} or T_{sk} may play. In contrast our current

investigation, provides substantial evidence that T_{sk} alone has major impacts on cardiovascular function during exercise alone and can exacerbate responses from high T_c .

Precooling and Preheating

In our present study, HR was reduced during exercise after precooling, compared to when subjects were preheated (Figure 2), as expected based on results from other precooling studies [4, 42]. Cooling athletes' core temperatures prior to exercise can increase performance during exercise in normothermic and warm climates, by increasing heat storage capacity, and thereby decreasing cardiovascular strain and perceived exertion associated with elevated T_c [42]. Lee et al, demonstrated that precooling lowered HR for the first 20 min of exercise in a thermoneutral environment and increased endurance by 21% [42]. These authors concluded that decreased stress on the cardiovascular system and increased heat storage capacity was the major factor leading to increased performance.

Over the course of exercise in our current investigation, T_{es} and HR increased similarly indicating a close relationship between internal body temperature and cardiac function. Conversely, Gonzalez-Alonso demonstrated preheating subjects decreases heat storage capacity, increases cardiovascular strain, and decreases time to fatigue during exercise in warm environments[27]. In these conditions HR is substantially elevated to near maximal levels, due to high T_c and T_{sk} similar to what observed in the WW condition in the current investigation (i.e. 93% of max), Figure 4.

Collectively these results indicate that exercise in a given environment can clearly affect cardiovascular responses during exercise, however what is left uncertain from these

investigations is the impact that skin and core temperature independently have on these responses. The interaction of core and skin temperature during exercise is complex and can significantly impact the cardiovascular responses over the duration of exercise. Therefore to gain further insight into the contributions of skin and core on these responses it is useful to determine the effects of warm or cool skin during exercise, when T_c is either warm or cool.

Warm vs. cool skin during exercise when core temperature is cool

Overall, T_{es} was not different during exercise after precooling and averaged 36.7°C with cool skin (CC), and 36.9°C with warm skin (WC), (Table 1). Over the duration of exercise T_{es} increased similarly in WC and CC conditions. In contrast, T_{sk} was significantly higher at each time point during exercise in WC compared to CC (Table 2). When skin was warmed during exercise after precooling HR was 4 beats/min higher by 10 min into exercise and remained higher thereafter (Table 2). SBF also increased over time, and was significantly higher at 10, 15, and 20 min of exercise, (Figure 3). The lack of a difference in HR and SBF at 5 min could be due to the extremely low T_{es} (i.e. 35.9°C in both CC and WC) at this time, which may override any increase in SBF by local warming of the skin. Additionally, at 10 min of exercise T_{es} increased to 36.5 and 36.6°C, for CC and WC, respectively, (Table 1) which may be high enough to remove some of the inhibition on SBF.

Therefore at 10 min, T_{es} may have been high enough to allow local warming to trigger some elevations in SBF. and stimulate an increase in HR to maintain MAP as TPR declines. This concept is supported by the fact that HR was significantly higher in WC

compared to CC after SBF began to increase, at the same T_{es} , (Figure 3). Conversely, thermoreceptors in the skin may directly increase activity of the sympathetic and therefore HR, by sending afferent signals to the brain, leading to the elevated HR. The elevated HR when skin is warmed demonstrates that T_{sk} can contribute to cardiovascular strain during exercise, even when T_c is cool. In addition, MAP declines when skin is warm during exercise even when T_{es} is cool. Figure 3 shows that MAP is reduced in WC compared to CC, at 15 and 20 min of exercise. It is likely that MAP begins to decline when SBF becomes substantially elevated and reduces TPR.

The similarity of T_{es} at each time point during exercise along with the substantial differences in T_{sk} , indicate that the reduction in MAP that occurs at 15 and 20 min is related to high T_{sk} . However, differences in MAP between WC and CC at 15 and 20 min must be considered in terms of the interaction of T_{es} and T_{sk} because there are no significant changes over time in either condition and the differences that exist at 15 and 20 min seem to be equally attributed to increased values when skin is cool and decreased values when skin is warm, (Figure 3). Furthermore, because T_{sk} remains fairly constant over the duration of exercise, but T_{es} increases steadily, it appears that MAP is not affected until T_{es} reaches 37.4°C , with a T_{sk} of $\sim 31^{\circ}\text{C}$, (Table 2). In CC, MAP increased over the course of exercise, (Figure 3), likely due to the elevation in SBP (191.0 ± 8.8 mmHg) and low SBF (30.4 ± 4.0 a.u.), (Table 1). Additionally, over the course of exercise after precooling there were no differences in SV at any time between WC and CC, (Figure 3). It is important to note that the lack of a difference in SV at any time point in CC compared to WC, is at odds with our initial finding that warm T_{sk} reduces SV,

(Figure 2). One explanation for this discrepancy is that cool core temperature lowers HR such that diastolic filling time, and thus SV, is not yet impaired. The lack of difference in SV between CC and WC, indicate that the overall reductions we see with warm skin (Figure 2) are primarily due to the large reduction in SV during WW. Also noteworthy is the fact that elevations in SBF during WC did not reduce SV, supporting the hypothesis that increased SBF is not the major cause for the reduction in SV during exercise in the heat. Furthermore, it seems possible that the increased CVC when T_{sk} was warmed in WC may have facilitated the maintenance of SV by reducing afterload.

In support of this explanation, after 20 minutes of exercise in CC, SV was reduced compared to the 5 min value. One explanation for this observation is that the increase in DBP at 15 and 20 min during CC, increased afterload leading to increased resistance of blood leaving the left ventricle and therefore a reduction in SV. In support of this finding, Wilson et al, demonstrated that skin surface cooling in resting humans, increases resistance in cutaneous and visceral vascular beds and was associated with elevations in MAP [87]. In contrast to our present findings, Wilson et al, demonstrated that the increased MAP had no effect on SV and Q. These differences may be due to different hemodynamics during exercise compared to rest. The finding that SV was maintained when T_{sk} was warm and while SBF was increasing, indicates that in conditions where T_{es} remains cool, there appears to be a SV reserve which may be due to the lower HR and increased time for diastolic filling.

Warm vs. cool skin during exercise when core temperature is warm

During exercise with warm skin after being preheated, the average HR for the duration of exercise was $90.9 \pm 1.0\%$ of the maximal HR achieved during preliminary maximal exercise tests (Figure 4). Such an elevated HR response during moderate intensity exercise is not surprising, as it is well documented that exercise with high T_c and T_{sk} places extreme demands on the cardiovascular system [25, 48, 51, 63]. A novel aspect of the current study is the isolation of core temperature during exercise with drastically different T_{sk} .

In support of the increased HR, and reduced SV we observed in the present investigation, Gonzalez-Alonso et al demonstrated that preheating subjects decreases heat storage, increases cardiovascular strain, and decreases time to fatigue during exercise in warm environments [27]. Furthermore, these authors concluded that a critical core temperature limits exercise performance, because subjects always fatigued at a T_c of 40.1 to 40.3°C, despite beginning exercise after precooling, or preheating. It is important to consider that all exercise was conducted in the heat (i.e. 40°C) therefore subjects had not only elevated T_c but also high T_{sk} . It is likely that fatigue was a factor of the combination of high T_c and T_{sk} , rather than T_c alone. In fact, in their study, mean T_{sk} at the time of fatigue was 37.2 ± 0.2 , 37.2 ± 0.1 , and 37.0 ± 0.1 °C during precooling, control, and preheating, respectively. In our investigation, mean T_{sk} after 20 min of exercise in skin heating conditions was only 32.7 ± 0.3 °C, yet had substantial cardiovascular consequences, (Table 3).

Our current findings that HR was elevated at 5 and 10 minutes in WW compared to CW, although T_{es} was not statistically different indicate that warm T_{sk} can increase HR

by 11 to 15 beats/min when T_{es} is $\sim 38^{\circ}\text{C}$, (Table 3). Furthermore, our findings indicate that perception of effort increases over time when skin is warmed, in both cool core and warm core conditions.

Warm skin temperature increases perception of effort

In the present investigation warm T_{sk} significantly increased average RPE during exercise when T_{es} was cool and warm, however significance was only achieved with warm T_{es} (i.e. WW), (Table 1), ($P < 0.05$). RPE increased from 5 to 20 min of exercise, when skin warm in both warm and cool core conditions (1.3 ± 0.3 and 2.4 ± 0.4 Borg Scale Units in WC and WW, respectively). In contrast when T_{sk} was cooled during exercise RPE remained constant over time, with both warm and cool T_{es} . Together these results indicate that warm T_{sk} increased RPE independent of T_{es} .

In support for the finding that T_{sk} increases perception of effort independent of T_{es} , Tucker et al. recently used a “fixed” perception of effort exercise model to investigate the differences in exercise performance in cool and hot conditions [81]. Subjects adjusted their work output during cycle ergometry to maintain a fixed, predetermined, level of perceived effort. In this study, exercise in the hot environment (i.e. 35°C) reduced power output more quickly (2.35 ± 0.73 vs. 1.63 ± 0.70 W/min), and fatigue occurred in sooner (34.0 ± 10.4 vs. 50.2 ± 16.3 min) compared to exercise in cool conditions (15°C). By reducing their work rate subjects’ maintained similar T_c over the course of exercise in all conditions. These authors concluded that afferent feedback from elevated T_{sk} and higher rates of heat storage increase RPE, and lead to reductions in work output so subjects achieve similar T_c in cool and hot climates.

Although we did not make any measures of performance in the present investigation the increase in RPE over the course of exercise only when skin was warm, is evidence that T_{sk} is an important factor in the perception of effort. It is important to note that over time, as RPE became significantly elevated WW conditions, there were also significant increases in T_{es} compared to CW. Therefore, we cannot be certain that warm T_{sk} *per se* increases RPE during exercise, however this finding indicates that both T_{sk} and T_c play a role in perception of effort. Another possibility is that RPE increases when T_{es} reaches a given T_{es} temperature (i.e. 38.4°C in the current study). This seems less likely, however because during cool T_{es} conditions we observed increased RPE at 15 and 20 min of exercise in WC compared to values at 5 min, while in CC there were no changes over the course of exercise.

Impact of skin temperature on physical performance

Many studies have examined the thermoregulatory and cardiovascular effects of exercising in warm-hot environments and it is clear that performance declines, as the cardiovascular system has to deliver oxygen and nutrients to active skeletal muscle as well as increase thermoregulatory mechanisms in an effort to keep body temperature within normal limits. What is not clear however, is the contribution of the skin and core temperatures, independently on thermoregulatory and cardiovascular responses, as well as performance.

The concept of a “critical core temperature” where fatigue will occur to protect individuals from achieving dangerous internal body temperatures has been proposed. Gonzalez-Alonso et al demonstrated that fatigue occurs at a T_c of 40 °C, during exercise

in the heat regardless of internal temperature at the beginning of exercise [27]. In their investigation, subjects' T_c was manipulated prior to exercise by resting in either 40, 36 or 17 °C water for 30 min, to increase, maintain, or decrease T_c , respectively, during subsequent exercise in the heat. Subjects exercised longer after being precooled, however fatigue always occurred when T_c was close to 40°C. In this study, exercise was conducted in 40°C, 19% R.H producing T_{sk} close to 35°C, which is warmer than T_{sk} achieved in our present study during warm skin conditions ($33.2 \pm 0.3^\circ\text{C}$). Therefore their data indicate that for a given warm-hot skin temperature, subjects can exercise longer when T_c is lowered prior to exercise. However, if warm T_{sk} can increase HR, RPE, and reduce SV, as shown in our current investigation, this could also contribute to fatigue. Furthermore, it is likely that subjects' could have continued to exercise with T_c of 40°C if their skin had been cooled during exercise.

In a recent study designed to test the influence of T_{sk} on the concept of a critical core temperature, Ely et al demonstrated that runners can achieve T_c above 40°C without any decrement in performance when T_{sk} is cool [17]. In their study runners completed 8 kilometer running time trial in either warm conditions or cool conditions. In cool conditions average T_c for the runners was 39.84° compared to 40.32°C in warm conditions, however there were no observed changes in running velocity. Several runners were able to increase their speed at the end of the event, despite a T_c well above the “critical” limit. This finding indicates that cool T_{sk} either decreases total cardiovascular strain, or decreases perception of effort, thereby allowing athletes to continue exercising at high intensities with very high T_c . It is noteworthy that runners in the warm

environments had slightly slower finishing times compared with runners in the cool environment, and had less of a finishing surge in the last few min of the event. In our current study we demonstrated that HR at a given intensity is elevated when T_{es} is warm. Therefore, one explanation for the slightly slower finishing times is that this alteration in the HR vs. work relationship reduced peak oxygen consumption, and therefore performance.

In agreement with this explanation, Arngrimsson et al demonstrated that elevations in HR during exercise in the heat were associated with a reduction in VO_{2peak} [5]. This study found that VO_{2peak} measured after 20 min of walking on a treadmill was significantly lower compared to thermoneutral conditions by 4% at 35°C, 9% at 40°C, and 17% at 45°C. An important finding in our current study was that HR was significantly different in every treatment, and increased in a graded manner, from CC (149.8 ± 4.0), to WC (154.7 ± 4.1), followed by CW (160.3 ± 3.5), and lastly WW (175.8 ± 3.2). Thus, it could be argued that the increase in HR with warm T_{sk} independent of T_{es} may contribute the reductions in VO_{2peak} during exercise in the heat.

Support for a reduction in VO_{2peak} by T_{sk} is demonstrated in the study by Arngrimsson et al, where at the end of 20 min of submaximal exercise HR was significantly elevated with environmental temperature, however T_{es} was only different in the 45°C environment, which increased T_{es} to 37.8°C. Therefore the elevation in HR, and subsequent reduction in VO_{2peak} , seems to be more related to T_{sk} , which increased steadily with environmental temperature [5].

Other studies have found similar reductions in $\text{VO}_{2\text{max}}$ with increased HR during exercise in the heat. For example, Wingo et al demonstrated that cardiovascular drift that occurred after 45 min cycling in a 35°C environment reduced $\text{VO}_{2\text{max}}$ [89]. In this study $\text{VO}_{2\text{max}}$ was determined after 15 min and 45 min of moderate intensity cycling on different days, to determine the influence that the CVD, which occurred between those time periods had on $\text{VO}_{2\text{peak}}$. In this study, exercise in a 35°C environment elevated HR from 151.1 ± 8.5 at 15 min to 169.3 ± 9.7 after 45 min. There was a relatively small change in T_{sk} during this period (35.9 ± 0.4 to 36.2 ± 0.5), however based on our current findings, T_{sk} at these values can increase HR by 5 to 15 beats above cool conditions. Furthermore T_{es} increased dramatically during this time period by greater than 1°C, and presumably contributed to the majority of increase in HR. However, the 1°C increase in T_{es} during this period would only be expected to increase HR by ~ 7 - 8 beats/min based solely on the effects of increased T_{c} , [72], thus the 20 beat/min increase must be related to some other factor.

In our present investigation, MAP decreased over the course of exercise when T_{sk} was warm, in both warm and cool T_{es} conditions, therefore some of the additional increase in HR in the study by Wingo et al, may be related to a baroreceptor response to maintain blood pressure. Another explanation for the increased HR may be that sympathetic activity due to warm T_{sk} , T_{es} , or both.

Another similar study from this research group supports our finding that skin and core can both influence cardiovascular responses during exercise. Lafrenz et al, demonstrated that the magnitude of CVD and the related reduction in $\text{VO}_{2\text{max}}$ is greater in

a warm (35°C) compared to a cool (22°C) environment [41]. In warm conditions, HR increased 11% and SV decreased by 11%, which produced a 15% reduction in $\text{VO}_{2\text{max}}$. In contrast, exercise in cool conditions elevated HR by 1-2%, and reduced SV by 1-2%, and did not impact $\text{VO}_{2\text{max}}$ [41].

In conclusion, these studies combined with data from the current investigation, provide evidence that fatigue during exercise in the heat is not solely due to elevated T_c , as cool T_{sk} can attenuate the reductions in performance with high T_c . Furthermore, our results indicate that warm T_{sk} can negatively affect performance even with cool core temperatures.

Rapid skin cooling

As expected, WW produced to highest values for HR, Q, and SBF, and the lowest values for SV, MAP, and TPR, indicating that this condition was stressing subjects' cardiovascular systems to near maximal capacity. At this point, subjects' had their skin cooled rapidly by removing the heaters and circulating air towards subjects with 4 fans. Interestingly, HR was reduced almost instantly, and after 5 min of exercise values were significantly lower than at the end of WW, before cooling began. This result was not entirely unexpected, as others have made similar observations[71, 77, 93].

Shaffrath and Adams demonstrated that conditions of CVD were only elicited when skin and temperature were both warmed, and cooling of either attenuated the responses[77]. In this study, a single subject who was better trained than the others, exercised with no fan cooling (i.e. warm skin), which produced significant CVD. At the end of this ride, fans were turned on, which lead to a significant reduction in HR, and

reversed nearly half of CVD that had occurred over the previous 70 min of exercise. These authors concluded that when fans were turned on, a reflex response from skin cooling and speculated declines in SBF, lead to increased venous return and SV, resulting in decreased HR.

Similar to the results of Shaffrath and Adams, Rowell showed that when skin temperature was cooled rapidly during exercise on a treadmill CBV and SV increased, and HR decreased [71]. In this study it was concluded that decreases in peripheral blood volume after skin cooling redistributed blood centrally leading to increased CBV and SV, causing the reduction in HR. However, in this study SBF was not measured, additionally the time course of increases in CBV were not temporally related to increases in SV. Collectively these studies demonstrate that rapidly cooling the skin will significantly reduce HR, however the mechanisms behind this response are speculative, and based on the premise that SBF will decrease substantially when fans are turned on. In the current investigation we observed similar significant reductions in HR after rapidly cooling the skin. Furthermore, we measured SBF and found no change 5 min after the initiation of skin cooling. Additionally, there were no significant differences in Q or SV, although most subjects displayed slightly increased SV (n.s.).

Our results indicate that the reductions in HR after rapid skin cooling is not an effect of decreased SBF and increased SV. It is possible that, decreased afferent feedback from thermoreceptors in the skin, may play a role in the reduction of HR after rapid skin cooling [31]. After 5 min of exercise with skin cooling, T_{es} was not different from values at the end of WW, thus it is unlikely that the reduction in HR was due to lower core

temperature. It is interesting to note that even though there were no significant reductions T_{es} or Q , the decline in HR after skin cooling was associated with a significant reduction in RPE. The finding that RPE is reduced while T_{es} remains elevated, is supported by studies indicating that high core temperatures can be maintained in runners without any reduction in performance when T_{sk} is kept cool[17]. Along these lines Tucker et al, demonstrated that when subjects conduct self paced exercise in warm conditions they reduce power output in order to maintain core temperatures that are similar to normothermic conditions [82]. Similarly, Ely et al recently demonstrated that exercise in warm conditions decreases exercise performance without reaching severe levels of hyperthermia [16].

Collectively these studies and the present investigation support the notion that warm skin increases perception of effort during exercise at a given work rate, independent of high internal temperatures.

Limitations

Laser Doppler probes on the dorsal region of the left forearm were used to measurement SBF, while subject's rested their arm in a sling hanging from the ceiling to reduce variability from arm movement. At all other times during exercise, subjects removed their arm from the sling and adopted a normal cycling position, with both hands resting on the handlebars. During skin warming trials, it is possible that some regions of the skin received more radiant heat than others; therefore we cannot be certain that our measurements are representative of whole body SBF. Additionally, it is possible that whole body SBF early during exercise (i.e. 5 min) in WC was higher than our

measurements indicated because the heaters may not have warmed skin on the arm as much as other parts of the body. Another limitation of the current investigation is that all subjects were young males (25.5 ± 1.7 years), therefore our results may not be applicable to older populations, females, or individuals with lower fitness levels.

It is quite possible that the relative influence of warm T_{sk} is greater than that observed in the current investigation, due to overpowering influence of T_c on thermoregulatory and cardiovascular responses [8, 64, 92]. Although, T_c is the primary regulator of thermoregulatory and cardiovascular responses, our data indicate that T_{sk} exerts a substantial influence on these variables and is often overlooked.

Conclusions

The primary finding of the current investigation is that both T_{es} and T_{sk} contribute cardiovascular strain and perception of effort during exercise. Furthermore, a novel aspect of the current investigation is that warm T_{sk} can increase HR and perceived exertion, and reduce MAP during exercise when T_{es} is either cool or warm. Additionally, it appears that SV can be maintained, or even increased, in the face of elevations in T_{sk} and SBF when T_{es} remains cool. These findings are important for elite or recreational athletes, physical laborers (especially when protective clothing is worn), or individuals with limited cardiovascular capacity (elderly, cancer or cardiovascular disease patients) in warm-hot conditions where T_{sk} may become elevated concurrently with T_c . Therefore, measures should be employed to keep the skin cool during exercise or activity in a warm-hot environment, especially when T_c increases above 38°C , in order to attenuate the cardiovascular strain and perception of effort associated with warm T_{sk} .

APPENDIX A

Supplemental figures.

Legends for Supplemental Figures

Supplemental Figure 1. Skin temperature and esophageal temperature during exercise with either cooled (CC) or warmed (WC) skin, after precooling, (n=8). Values are means $\pm$ SE for data collected 5, 10, 15, and 20 min of exercise. *Significantly different from cool core condition. #Significantly different from 5 min value.

Supplemental Figure 2. Skin temperature and esophageal temperature during exercise with either cooled (CW) or warmed (WW) skin, after preheating, (n=8). Values are means $\pm$ SE for data collected 5, 10, 15, and 20 min of exercise. *Significantly different from cool core condition. #Significantly different from 5 min value.

Supplemental Figure 1.

Supplemental Figure 2.

APPENDIX B

Raw data for all trials.

COOL SKIN & COOL CORE (CC)

<i>Subject</i>	Tsk pre (rest)	5min	10min	15min	20min	Δ	Tes pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	27.3	19.8	20.7	21.6	22.4	2.6	36.7	36.2	36.9	37.4	37.9	1.7
<i>JK</i>	29.8	21.3	22.8	23.9	24.7	3.4	36.9	36.1	36.8	37.4	38.0	1.9
<i>KC</i>	30.3	19.7	20.9	22.2	23.2	3.5	36.7	35.7	36.3	36.9	37.4	1.6
<i>JT</i>	29.6	19.4	21.4	22.4	23.1	3.7	36.5	36.0	36.3	36.8	37.1	1.1
<i>NY</i>	27.6	19.9	21.3	22.2	23.0	3.0	37.1	36.2	36.8	37.3	37.8	1.6
<i>TC</i>	29.5	21.0	22.3	23.4	23.8	2.9	36.3	35.7	36.4	37.1	37.8	2.1
<i>BM</i>	29.2	21.1	22.6	24.0	25.0	3.9	36.1	35.1	35.7	36.2	36.4	1.3
<i>NV</i>	29.3	21.8	23.1	23.9	24.3	2.6	36.7	36.1	36.6	37.1	37.6	1.5
<i>Mean</i>	29.1	20.5	21.9	22.9	23.7	3.2	36.6	35.9	36.5	37.0	37.5	1.6
<i>S.D.</i>	1.1	0.9	0.9	1.0	0.9	0.5	0.3	0.4	0.4	0.4	0.5	0.3

WARM SKIN & COOL CORE (WC)

<i>Subject</i>	Tsk pre (rest)	5min	10min	15min	20min	Δ	Tes pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	29.8	25.6	28.1	28.8	30.0	4.4	36.7	36.1	36.8	37.2	37.4	1.2
<i>JK</i>	28.3	29.9	30.8	33.0	33.6	3.7	36.9	36.1	36.8	37.9	38.2	2.1
<i>KC</i>	29.6	26.9	29.7	32.0	32.7	5.8	36.5	35.6	36.3	37.1	37.5	1.8
<i>JT</i>	30.4	26.3	28.5	30.2	31.1	4.8	36.7	36.0	36.6	37.4	37.7	1.8
<i>NY</i>	30.5	25.6	27.3	29.5	31.6	6.1	37.1	36.4	36.8	37.8	38.2	1.7
<i>TC</i>	30.1	27.7	30.2	32.3	33.3	5.7	36.6	35.9	36.7	37.5	37.9	2.0
<i>BM</i>	29.3	27.0	28.8	31.3	32.9	5.9	36.4	35.4	36.1	36.6	36.9	1.5
<i>NV</i>	28.8	26.5	27.8	30.8	31.7	5.2	36.6	36.3	36.9	37.6	38.1	1.8
<i>Mean</i>	29.6	26.9	28.9	31.0	32.1	5.2	36.7	36.0	36.6	37.4	37.7	1.8
<i>S.D.</i>	0.7	1.4	1.2	1.4	1.2	0.8	0.2	0.3	0.3	0.4	0.5	0.3

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	Tsk pre (rest)	5min	10min	15min	20min	Δ	Tes pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	27.9	24.5	24.8	24.8	24.8	0.2	36.7	37.4	37.7	37.6	37.7	0.3
<i>JK</i>	29.7	24.2	24.5	25.1	25.5	1.2	36.7	37.6	37.6	37.4	37.6	0.0
<i>KC</i>	29.7	23.5	24.0	24.0	24.2	0.7	36.4	37.6	37.7	37.8	37.8	0.3
<i>JT</i>	29.6	24.4	24.7	24.8	24.9	0.5	36.6	37.6	37.7	37.7	37.7	0.1
<i>NY</i>	28.8	23.3	23.6	23.7	23.9	0.6	37.1	38.2	37.8	38.2	38.3	0.1
<i>TC</i>	30.2	24.2	24.9	25.1	25.2	1.0	36.7	38.1	38.1	38.1	38.2	0.1
<i>BM</i>	29.5	24.1	24.4	24.9	25.0	1.0	36.3	36.9	36.8	36.7	36.8	-0.1
<i>NV</i>	29.8	24.7	25.3	25.4	25.6	0.9	37.3	38.8	38.8	38.8	38.8	0.0
<i>Mean</i>	29.4	24.1	24.5	24.7	24.9	0.8	36.7	37.8	37.8	37.8	37.9	0.1
<i>S.D.</i>	0.7	0.5	0.5	0.6	0.6	0.3	0.3	0.6	0.6	0.6	0.6	0.1

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	Tsk pre (rest)	5min	10min	15min	20min	Δ	Tes pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	29.4	31.1	31.3	31.6	32.1	1.0	36.6	37.1	37.8	37.9	37.9	0.8
<i>JK</i>	29.7	33.4	34.2	33.9	34.2	0.8	36.8	38.1	38.3	38.5	38.7	0.7
<i>KC</i>	29.3	33.2	33.7	34.5	34.7	1.5	36.4	37.6	38.0	38.1	38.6	1.1
<i>JT</i>	29.2	32.3	32.8	33.0	33.3	1.1	36.9	37.8	38.3	38.5	38.6	0.8
<i>NY</i>	29.6	32.8	33.1	33.0	33.0	0.2	37.5	38.3	39.0	39.4	39.5	1.2
<i>TC</i>	31.5	32.5	33.1	33.3	33.5	1.0	37.5	38.1	38.5	38.8	39.1	1.0
<i>BM</i>	29.7	31.3	31.8	31.9	32.0	0.7	36.3	37.4	37.9	38.0	38.5	1.1
<i>NV</i>	28.4	30.7	31.5	32.1	32.8	2.1	37.2	37.8	38.2	38.7	39.0	1.1
<i>Mean</i>	29.6	32.2	32.7	32.9	33.2	1.0	36.9	37.8	38.3	38.5	38.7	1.0
<i>S.D.</i>	0.9	1.0	1.1	1.0	0.9	0.6	0.5	0.4	0.4	0.5	0.5	0.2

COOL SKIN & COOL CORE (CC)

<i>Subject</i>	VO2 pre (rest)	5min	10min	15min	20min	Δ	RPE pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	0.6	3.2	3.3	3.5	3.5	0.3		12.0	12.0	12.0	12.0	0.0
<i>JK</i>	0.4	2.7	2.9	2.9	2.9	0.1		11.0	11.0	11.0	11.0	0.0
<i>KC</i>	0.3	2.8	3.0	3.1	3.2	0.3		11.0	11.0	11.0	11.0	0.0
<i>JT</i>	0.4	2.7	2.7	2.7	2.8	0.1		14.0	15.0	16.0	16.0	2.0
<i>NY</i>	0.4	2.6	2.9	3.1	3.1	0.5		13.0	13.0	14.0	15.0	2.0
<i>TC</i>	0.3	3.0	3.2	3.1	3.0	0.0		13.0	13.0	12.0	12.0	-1.0
<i>BM</i>	0.4	3.7	3.8	3.8	3.8	0.2		13.0	14.0	14.0	14.0	1.0
<i>NV</i>	0.3	1.9	2.1	2.1	2.1	0.2		10.0	11.0	11.0	10.0	0.0
<i>Mean</i>	0.4	2.8	3.0	3.0	3.1	0.2		12.1	12.5	12.6	12.6	0.5
<i>S.D.</i>	0.1	0.5	0.5	0.5	0.5	0.2		1.4	1.5	1.8	2.1	1.1

WARM SKIN & COOL CORE (WC)

<i>Subject</i>	VO2 pre (rest)	5min	10min	15min	20min	Δ	RPE pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	0.6	2.8	3.1	3.2	3.3	0.5		11.0	12.0	12.0	13.0	2.0
<i>JK</i>	0.4	2.6	2.7	2.9	2.7	0.1		12.0	12.0	12.0	12.0	0.0
<i>KC</i>	0.3	2.7	3.0	2.9	3.0	0.3		12.0	13.0	13.0	13.0	1.0
<i>JT</i>	0.4	2.7	3.0	3.0	3.0	0.4		17.0	17.0	18.0	19.0	2.0
<i>NY</i>	0.3	2.2	2.7	2.9	3.1	0.8		13.0	14.0	15.0	15.0	2.0
<i>TC</i>	0.4	2.7	2.9	2.9	2.8	0.1		12.0	12.0	12.0	12.0	0.0
<i>BM</i>	0.4	3.7	3.8	3.8	3.8	0.1		13.0	14.0	15.0	15.0	2.0
<i>NV</i>	0.3	1.9	2.1	2.1	2.2	0.2		12.0	13.0	14.0	13.0	1.0
<i>Mean</i>	0.4	2.7	2.9	3.0	3.0	0.3		12.8	13.4	13.9	14.0	1.3
<i>S.D.</i>	0.1	0.5	0.5	0.5	0.5	0.2		1.8	1.7	2.1	2.3	0.9

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	VO2 pre (rest)	5min	10min	15min	20min	Δ	RPE pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	0.5	3.0	3.1	3.4	3.4	0.4		14.0	13.0	12.0	12.0	2.0
<i>JK</i>	0.4	2.6	2.9	2.9	2.9	0.3		12.0	12.0	12.0	12.0	0.0
<i>KC</i>	0.4	2.9	3.2	3.4	3.5	0.6		12.0	12.0	12.0	12.0	0.0
<i>JT</i>	0.5	2.5	2.9	2.9	3.0	0.5		15.0	16.0	18.0	18.0	3.0
<i>NY</i>	0.4	2.7	3.1	3.3	3.2	0.5		13.0	14.0	14.0	14.0	1.0
<i>TC</i>	0.3	2.8	2.9	3.0	3.0	0.2		12.0	12.0	12.0	12.0	0.0
<i>BM</i>	0.3	3.5	3.6	3.7	3.8	0.3		15.0	15.0	15.0	15.0	0.0
<i>NV</i>	0.4	2.0	2.2	2.1	2.2	0.2		10.0	11.0	11.0	11.0	1.0
<i>Mean</i>	0.4	2.8	3.0	3.1	3.1	0.4		12.9	13.1	13.3	13.3	0.4
<i>S.D.</i>	0.1	0.4	0.4	0.5	0.5	0.1		1.7	1.7	2.3	2.3	1.4

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	VO2 pre (rest)	5min	10min	15min	20min	Δ	RPE pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	0.6	3.1	3.2	3.2	3.3	0.2		12.0	13.0	13.0	13.0	1.0
<i>JK</i>	0.3	2.5	2.8	3.0	2.9	0.4		13.0	13.0	14.0	15.0	2.0
<i>KC</i>	0.4	2.8	3.2	3.3	3.3	0.5		13.0	14.0	14.0	15.0	2.0
<i>JT</i>	0.4	2.3	2.7	2.7	2.8	0.5		15.0	15.0	18.0	19.0	4.0
<i>NY</i>	0.4	2.4	2.9	3.0	3.1	0.7		14.0	15.0	16.0	17.0	3.0
<i>TC</i>	0.3	2.3	3.0	2.9	3.0	0.7		14.0	15.0	15.0	16.0	2.0
<i>BM</i>	0.5	3.3	3.5	3.4	3.5	0.2		18.0	19.0	19.0	19.0	1.0
<i>NV</i>	0.4	1.9	2.1	2.1	2.1	0.2		13.0	13.0	15.0	17.0	4.0
<i>Mean</i>	0.4	2.6	2.9	3.0	3.0	0.4		14.0	14.6	15.5	16.4	2.4
<i>S.D.</i>	0.1	0.5	0.4	0.4	0.4	0.2		1.9	2.0	2.1	2.1	1.2

COOL SKIN & COOL CORE (CC)

<i>Subject</i>	HR pre (rest)	5min	10min	15min	20min	Δ	Q pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	62.1	124.1	138.1	145.3	146.3	22.1	10.2	21.8	22.4	23.4	23.6	1.8
<i>JK</i>	65.0	141.2	149.5	151.9	152.7	11.4	13.6	20.5	23.6	22.1	22.4	1.9
<i>KC</i>	69.2	124.6	141.1	148.3	150.4	25.8	10.5	20.5	17.1	21.0	20.6	0.1
<i>JT</i>	70.5	130.0	141.2	146.9	150.8	20.8	9.8	21.1	23.3	23.3	22.6	1.4
<i>NY</i>	89.5	156.0	167.1	172.2	177.8	21.8	6.3	18.1	18.1	21.8	18.5	0.4
<i>TC</i>	58.0	153.2	160.7	164.7	167.0	13.7	6.5	19.5	20.2	21.4	20.7	1.2
<i>BM</i>	55.7	135.9	141.9	142.8	142.2	6.3	9.6	28.1	30.3	28.1	26.6	-1.6
<i>NV</i>	76.0	147.0	155.6	161.1	165.0	18.0	9.7	20.6	21.0	19.0	18.2	-2.4
<i>Mean</i>	68.3	139.0	149.4	154.2	156.5	17.5	9.5	21.3	22.0	22.5	21.7	0.4
<i>S.D.</i>	10.9	12.4	10.7	10.6	12.1	6.5	2.3	3.0	4.1	2.7	2.7	1.6

WARM SKIN & COOL CORE (WC)

<i>Subject</i>	HR pre (rest)	5min	10min	15min	20min	Δ	Q pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	60.0	128.0	138.9	147.3	149.7	21.7	12.2	21.6	20.3	23.5	23.7	2.2
<i>JK</i>	69.0	144.0	151.3	158.8	163.8	19.7	9.4	21.0	21.3	21.5	22.8	1.8
<i>KC</i>	72.5	130.0	149.8	159.2	165.2	35.2	7.4	20.8	21.8	21.2	22.2	1.4
<i>JT</i>	59.1	126.3	144.2	153.4	158.4	32.1	7.8	21.8	22.0	24.2	24.7	2.9
<i>NY</i>	79.8	156.3	169.5	178.7	179.1	22.8	6.7	17.6	19.9	21.1	23.0	5.5
<i>TC</i>	58.0	153.3	163.1	171.3	172.0	18.7	7.1	20.8	22.4	21.3	22.8	2.0
<i>BM</i>	53.8	131.9	141.1	146.3	150.4	18.6	10.0	28.9	26.7	29.3	29.4	0.4
<i>NV</i>	76.6	153.7	166.1	171.8	175.9	22.2	8.2	20.0	20.6	21.5	22.9	2.9
<i>Mean</i>	66.1	140.4	153.0	160.8	164.3	23.9	8.6	21.6	21.9	23.0	23.9	2.4
<i>S.D.</i>	9.6	12.8	11.8	12.0	11.0	6.3	1.8	3.3	2.1	2.8	2.3	1.5

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	HR pre (rest)	5min	10min	15min	20min	Δ	Q pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	60.0	156.5	158.5	163.9	160.3	3.8	11.5	22.2	22.0	23.9	24.8	2.6
<i>JK</i>	65.0	157.7	157.6	156.5	155.8	-1.9	12.3	22.0	23.8	23.1	23.3	1.3
<i>KC</i>	61.2	156.1	162.1	170.0	166.8	10.7	11.3	21.7	23.4	24.9	25.2	3.4
<i>JT</i>	70.0	145.1	151.3	156.3	156.8	11.7	9.9	21.6	23.1	25.1	26.3	4.6
<i>NY</i>	96.2	172.7	179.8	181.1	178.8	6.2	5.6	17.3	19.0	23.5	20.0	2.7
<i>TC</i>	59.1	160.9	161.3	163.3	165.9	5.0	7.8	22.2	21.3	21.9	23.9	1.7
<i>BM</i>	50.0	143.2	145.1	144.4	143.0	-0.2	8.2	27.8	29.0	26.4	24.3	-3.5
<i>NV</i>	66.1	164.7	164.3	164.9	166.3	1.7	10.2	20.5	22.1	20.8	19.1	-1.4
<i>Mean</i>	65.9	157.1	160.0	162.6	161.7	4.6	9.6	21.9	22.9	23.7	23.3	1.4
<i>S.D.</i>	13.6	9.7	10.1	10.8	10.5	4.9	2.3	2.9	2.9	1.8	2.5	2.7

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	HR pre (rest)	5min	10min	15min	20min	Δ	Q pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	60.0	167.1	171.5	173.1	177.9	10.8	10.6	22.4	23.1	22.6	24.3	1.9
<i>JK</i>	72.0	165.1	170.4	172.7	173.6	8.4	8.6	23.0	27.0	24.9	24.9	1.9
<i>KC</i>	63.5	171.6	179.1	182.6	186.2	14.7	7.3	22.8	22.2	24.1	25.3	2.5
<i>JT</i>	65.7	164.7	174.0	177.9	179.1	14.4	8.1	22.0	24.5	24.2	24.5	2.5
<i>NY</i>	90.3	177.2	188.7	193.4	196.0	18.8	5.9	17.9	20.6	21.2	21.7	3.8
<i>TC</i>	74.1	164.8	169.3	169.2	177.7	13.0	7.6	22.2	23.7	22.3	22.8	0.6
<i>BM</i>	58.5	159.2	162.2	163.7	167.0	7.8	8.3	28.9	26.7	28.1	28.0	-0.9
<i>NV</i>	87.1	180.1	186.8	191.5	190.8	10.7	6.8	19.9	19.9	20.6	22.7	2.8
<i>Mean</i>	71.4	168.7	175.2	178.0	181.0	12.3	7.9	22.4	23.5	23.5	24.3	1.9
<i>S.D.</i>	12.0	7.1	9.1	10.5	9.4	3.6	1.4	3.1	2.6	2.4	2.0	1.4

COOL SKIN & COOL CORE (CC)

<i>Subject</i>	SV pre (rest)	5min	10min	15min	20min	Δ	SBF pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	164.5	175.9	161.9	161.2	161.6	-14.2	15.9	19.1	27.6	30.6	38.9	19.8
<i>JK</i>	209.3	144.8	157.7	145.3	146.6	1.8	17.4	38.0	29.5	33.4	29.6	-8.4
<i>KC</i>	152.0	164.4	121.5	141.7	137.1	-27.4	15.0	14.7	20.9	23.3	26.7	12.0
<i>JT</i>	139.0	162.6	165.0	158.9	149.7	-12.9	16.7	26.1	19.9	18.5	29.2	3.1
<i>NY</i>	70.0	116.0	108.3	126.4	104.1	-11.9	10.7	19.8	21.9	20.8	30.5	10.7
<i>TC</i>	111.4	127.3	125.6	130.0	124.2	-3.1	15.1	29.0	30.8	30.8	44.2	15.2
<i>BM</i>	171.8	206.8	213.4	197.0	186.7	-20.1	24.9	40.7	36.2	56.2	91.8	51.1
<i>NV</i>	127.3	140.4	135.2	117.8	110.6	-29.8	7.3	21.5	25.8	24.6	23.6	2.1
<i>Mean</i>	143.2	154.8	148.6	147.3	140.1	-14.7	15.4	26.1	26.6	29.8	39.3	13.2
<i>S.D.</i>	42.0	29.0	33.4	25.2	27.3	10.9	5.1	9.3	5.6	11.9	22.2	17.7

WARM SKIN & COOL CORE (WC)

<i>Subject</i>	SV pre (rest)	5min	10min	15min	20min	Δ	SBF pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	203.4	168.5	146.2	159.5	158.6	-9.9	19.4	22.9	37.8	87.8	95.5	72.5
<i>JK</i>	136.3	145.9	140.5	135.4	139.2	-6.8	18.9	29.6	35.1	60.5	137.0	107.4
<i>KC</i>	101.9	159.8	145.3	133.4	134.3	-25.6	18.3	22.1	38.6	72.5	152.4	130.3
<i>JT</i>	132.9	172.5	152.5	158.1	155.9	-16.7	14.0	25.1	30.6	54.6	80.4	55.3
<i>NY</i>	84.2	112.3	117.2	118.2	128.6	16.4	20.8	20.0	25.3	34.1	95.4	75.4
<i>TC</i>	122.1	135.5	137.6	124.6	132.4	-3.2	10.8	38.7	53.7	90.9	132.8	94.2
<i>BM</i>	186.4	219.5	188.9	200.3	195.2	-24.3	29.0	24.1	76.1	135.7	141.7	117.6
<i>NV</i>	107.6	130.3	123.8	125.2	130.4	0.1	29.8	32.7	47.4	47.7	115.8	83.1
<i>Mean</i>	134.3	155.5	144.0	144.3	146.8	-8.7	20.1	26.9	43.1	73.0	118.9	92.0
<i>S.D.</i>	41.3	32.9	21.6	27.2	22.7	13.8	6.6	6.3	16.1	31.9	26.1	25.2

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	SV pre (rest)	5min	10min	15min	20min	Δ	SBF pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	192.3	141.8	139.0	146.1	154.7	12.9	22.8	76.3	79.0	84.6	81.5	5.2
<i>JK</i>	189.1	139.7	151.1	147.4	149.9	10.2	17.7	136.2	131.7	131.2	127.1	-9.0
<i>KC</i>	184.2	139.3	144.2	146.6	150.8	11.5	12.5	74.9	94.8	118.4	80.4	5.5
<i>JT</i>	142.1	149.1	152.7	160.7	167.5	18.4	19.9	77.6	118.9	136.1	78.2	0.6
<i>NY</i>	57.7	100.2	105.4	129.8	111.8	11.6	9.8	89.7	75.0	72.6	82.1	-7.7
<i>TC</i>	131.2	137.9	131.9	134.3	144.0	6.2	13.9	121.3	120.7	119.6	109.7	-11.6
<i>BM</i>	165.0	194.0	199.5	182.5	169.7	-24.4	30.2	135.1	149.8	160.2	144.0	8.8
<i>NV</i>	154.4	124.3	134.3	126.2	114.6	-9.6	15.2	117.3	107.3	125.7	93.5	-23.8
<i>Mean</i>	152.0	140.8	144.8	146.7	145.4	4.6	17.7	103.6	109.6	118.5	99.6	-4.0
<i>S.D.</i>	44.1	26.3	26.6	18.3	21.7	14.3	6.5	26.7	25.8	28.1	24.9	11.0

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	SV pre (rest)	5min	10min	15min	20min	Δ	SBF pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	176.1	134.0	134.6	130.5	136.4	2.5	19.3	130.5	101.4	83.7	92.1	-38.4
<i>JK</i>	119.9	139.2	158.2	144.1	143.2	4.1	16.9	168.7	170.2	126.7	135.1	-33.5
<i>KC</i>	114.5	132.8	124.0	131.7	136.0	3.2	13.2	168.0	113.9	149.3	141.0	-27.0
<i>JT</i>	123.0	133.4	140.8	136.1	136.7	3.3	25.1	151.2	96.9	67.1	142.4	-8.8
<i>NY</i>	65.1	101.1	109.3	109.6	110.7	9.6	15.0	139.8	119.1	120.1	122.0	-17.8
<i>TC</i>	102.2	134.5	139.9	131.9	128.2	-6.3	14.0	108.6	133.1	137.5	132.7	24.1
<i>BM</i>	141.7	181.4	164.7	171.5	167.7	-13.8	25.3	148.3	144.1	155.0	155.0	6.7
<i>NV</i>	77.8	110.5	106.6	107.7	118.8	8.3	14.4	133.8	94.5	127.3	109.7	-24.1
<i>Mean</i>	115.0	133.4	134.8	132.9	134.7	1.3	17.9	143.6	121.7	120.8	128.8	-14.9
<i>S.D.</i>	35.0	23.6	20.9	20.0	17.1	7.7	4.9	20.0	26.2	30.7	20.1	21.3

COOL SKIN & COOL CORE (CC)

	MAP						DBP					
<i>Subject</i>	pre (rest)	5min	10min	15min	20min	Δ	pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	87.0	81.0	89.0	104.3	101.7	20.7	73.0	60.0	59.0	67.0	61.0	1.0
<i>JK</i>	99.3	93.7	110.7	140.3	135.0	41.3	71.0	69.0	92.0	104.0	90.0	21.0
<i>KC</i>	102.3	99.7	107.3	108.0	103.3	3.7	91.0	68.0	72.0	78.0	75.0	7.0
<i>JT</i>	122.7	93.3	122.7	119.7	131.3	38.0	83.0	60.0	66.0	62.0	82.0	22.0
<i>NY</i>	98.0	120.3	125.7	122.0	124.3	4.0	89.0	95.0	100.0	94.0	94.0	-1.0
<i>TC</i>	102.0	119.7	120.0	123.7	120.0	0.3	87.0	82.0	82.0	80.0	76.0	-6.0
<i>BM</i>	103.7	129.3	128.0	135.3	131.0	1.7	91.0	75.0	77.0	86.0	83.0	8.0
<i>NV</i>	87.3	124.7	124.7	119.3	116.0	-8.7	74.0	89.0	88.0	87.0	79.0	-10.0
<i>Mean</i>	100.3	107.7	116.0	121.6	120.3	12.6	82.4	74.8	79.5	82.3	80.0	5.3
<i>S.D.</i>	11.1	17.9	13.1	12.2	12.7	18.6	8.5	13.0	13.7	13.7	10.1	11.7

WARM SKIN & COOL CORE (WC)

	MAP						DBP					
<i>Subject</i>	pre (rest)	5min	10min	15min	20min	Δ	pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	89.3	100.3	108.0	100.7	99.7	-0.7	74.0	70.0	73.0	60.0	64.0	-6.0
<i>JK</i>	102.0	104.0	119.0	117.0	114.0	10.0	87.0	78.0	60.0	60.0	62.0	-16.0
<i>KC</i>	99.0	103.0	93.7	105.0	103.3	0.3	87.0	87.0	64.0	77.0	75.0	-12.0
<i>JT</i>	104.2	101.7	126.7	102.7	110.3	8.7	87.0	68.0	78.0	48.0	60.0	-8.0
<i>NY</i>	99.3	125.3	122.0	123.3	124.3	-1.0	88.0	97.0	94.0	92.0	92.0	-5.0
<i>TC</i>	108.0	117.7	122.0	106.7	105.7	-12.0	89.0	67.0	76.0	58.0	59.0	-8.0
<i>BM</i>	101.3	119.3	124.0	127.0	128.0	8.7	86.0	68.0	78.0	81.0	81.0	13.0
<i>NV</i>	84.7	126.0	118.0	109.3	107.0	-19.0	71.0	91.0	87.0	80.0	79.0	-12.0
<i>Mean</i>	98.5	112.2	116.7	111.5	111.5	-0.6	83.6	78.3	76.3	69.5	71.5	-6.8
<i>S.D.</i>	7.7	11.0	10.8	9.8	10.0	10.4	7.0	11.9	11.1	15.0	12.0	8.8

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	MAP pre (rest)	5min	10min	15min	20min	Δ	DBP pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	94.0	101.0	94.7	92.7	96.3	-4.7	77.0	62.0	56.0	56.0	59.0	-3.0
<i>JK</i>	101.0	77.3	117.7	116.0	107.7	30.3	81.0	51.0	69.0	62.0	57.0	6.0
<i>KC</i>	96.0	85.7	86.7	86.0	91.0	5.3	83.0	48.0	51.0	52.0	57.0	9.0
<i>JT</i>	105.0	77.0	76.3	108.7	107.3	30.3	84.0	41.0	51.0	56.0	58.0	17.0
<i>NY</i>	101.7	125.3	118.0	115.0	115.0	-10.3	90.0	95.0	91.0	90.0	90.0	-5.0
<i>TC</i>	99.0	105.3	107.7	113.7	112.3	7.0	81.0	60.0	64.0	68.0	66.0	6.0
<i>BM</i>	103.7	121.3	128.0	130.7	126.0	4.7	90.0	78.0	82.0	84.0	78.0	0.0
<i>NV</i>	83.3	78.3	80.3	103.7	106.7	28.3	66.0	47.0	41.0	70.0	73.0	26.0
<i>Mean</i>	98.0	96.4	101.2	108.3	107.8	11.4	81.5	60.3	63.1	67.3	67.3	7.0
<i>S.D.</i>	7.0	19.8	19.4	14.1	10.8	16.2	7.7	18.2	16.9	13.7	12.1	10.4

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	MAP pre (rest)	5min	10min	15min	20min	Δ	DBP pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	90.0	84.7	85.3	88.3	88.0	3.3	76.0	50.0	46.0	48.0	46.0	-4.0
<i>JK</i>	95.3	100.3	106.3	104.3	106.7	6.3	75.0	51.0	60.0	54.0	55.0	4.0
<i>KC</i>	94.0	83.3	87.0	89.0	93.0	9.7	82.0	49.0	49.0	52.0	58.0	9.0
<i>JT</i>	112.0	109.7	108.7	101.0	97.0	-12.7	95.0	66.0	60.0	56.0	56.0	-10.0
<i>NY</i>	101.3	115.3	114.7	108.3	99.7	-15.7	91.0	87.0	85.0	74.0	56.8	-30.2
<i>TC</i>	103.7	102.7	98.0	108.7	107.0	4.3	86.0	54.0	47.0	58.0	60.0	6.0
<i>BM</i>	101.7	124.3	115.3	118.3	99.7	-24.7	87.0	77.0	62.0	68.0	56.8	-20.2
<i>NV</i>	95.0	101.7	106.0	101.3	98.0	-3.7	84.0	72.0	71.0	68.0	66.0	-6.0
<i>Mean</i>	99.1	102.8	102.7	102.4	98.6	-4.1	84.5	63.3	60.0	59.8	56.8	-6.4
<i>S.D.</i>	7.0	14.1	11.5	10.1	6.4	12.3	6.9	14.4	13.3	9.2	5.6	13.5

COOL SKIN & COOL CORE (CC)

<i>Subject</i>	SBP pre (rest)	5min	10min	15min	20min	Δ	TPR pre (rest)	5min	10min	15min	20min	Δ
DM	115.0	123.0	149.0	179.0	183.0	60.0	681.0	296.8	318.4	356.5	344.0	47.2
JK	137.0	143.0	148.0	213.0	225.0	82.0	584.1	366.4	375.5	508.3	482.6	116.2
KC	125.0	163.0	178.0	168.0	160.0	-3.0	777.9	389.1	501.0	411.0	401.1	12.0
JT	165.0	160.0	236.0	235.0	230.0	70.0	1002.0	353.1	421.2	410.1	465.2	112.1
NY	116.0	171.0	177.0	178.0	185.0	14.0	1251.4	531.9	555.4	448.3	537.7	5.8
TC	132.0	195.0	196.0	211.0	208.0	13.0	1263.2	490.7	475.7	461.9	463.0	-27.7
						-						
BM	129.0	238.0	230.0	234.0	227.0	11.0	866.5	368.1	338.2	384.9	394.7	26.5
NV	114.0	196.0	198.0	184.0	190.0	-6.0	722.1	483.2	474.1	503.0	508.6	25.4
Mean	129.1	173.6	189.0	200.3	201.0	27.4	893.5	409.9	432.4	435.5	449.6	39.7
S.D.	16.8	35.7	32.9	26.4	25.4	37.3	256.6	81.8	83.4	54.5	64.7	50.7

WARM SKIN & COOL CORE (WC)

<i>Subject</i>	SBP pre (rest)	5min	10min	15min	20min	Δ	TPR pre (rest)	5min	10min	15min	20min	Δ
DM	120.0	161.0	178.0	182.0	171.0	10.0	585.6	372.3	425.5	342.8	335.9	-36.4
JK	132.0	156.0	237.0	231.0	218.0	62.0	867.6	395.8	447.7	435.5	400.2	4.4
KC	123.0	135.0	153.0	161.0	160.0	25.0	1072.3	396.6	344.3	395.4	372.8	-23.8
JT	138.5	169.0	224.0	212.0	211.0	42.0	1062.0	373.1	460.6	338.8	357.4	-15.7
NY	122.0	182.0	178.0	186.0	189.0	7.0	1183.3	571.3	491.1	467.4	431.7	-139.6
						-						
TC	146.0	219.0	214.0	204.0	199.0	20.0	1219.6	452.9	434.8	399.9	371.2	-81.6
BM	132.0	222.0	216.0	219.0	222.0	0.0	808.8	329.8	372.2	346.8	348.6	18.8
						-						
NV	112.0	196.0	180.0	168.0	163.0	33.0	821.6	503.5	459.0	406.6	373.4	-130.1
Mean	128.2	180.0	197.5	195.4	191.6	11.6	952.6	424.4	429.4	391.6	373.9	-50.5
S.D.	11.0	30.8	29.1	25.0	24.8	31.1	217.3	79.8	48.6	46.5	30.2	60.0

COOL SKIN & WARM CORE (CW)

<i>Subject</i>	SBP pre (rest)	5min	10min	15min	20min	Δ	TPR pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	128.0	179.0	172.0	166.0	171.0	-8.0	651.6	364.0	343.6	309.6	310.7	-53.3
<i>JK</i>	141.0	130.0	215.0	224.0	209.0	79.0	657.4	280.8	395.4	402.2	369.0	88.1
<i>KC</i>	122.0	161.0	158.0	154.0	159.0	-2.0	681.2	315.2	296.5	276.1	289.5	-25.7
<i>JT</i>	147.0	149.0	127.0	214.0	206.0	57.0	844.6	284.7	264.4	346.1	326.9	42.3
<i>NY</i>	125.0	186.0	172.0	165.0	165.0	-21.0	1464.1	579.6	498.2	391.3	460.2	-119.3
<i>TC</i>	135.0	196.0	195.0	205.0	205.0	9.0	1021.9	379.8	404.8	414.7	376.0	-3.8
<i>BM</i>	131.0	208.0	220.0	224.0	222.0	14.0	1005.4	349.4	353.7	396.6	415.6	66.2
<i>NV</i>	118.0	141.0	159.0	171.0	174.0	33.0	653.9	306.3	291.3	398.4	447.5	141.2
<i>Mean</i>	130.9	168.8	177.3	190.4	188.9	20.1	872.5	357.5	356.0	366.9	374.4	17.0
<i>S.D.</i>	9.8	27.8	31.3	29.2	24.1	34.1	285.4	96.7	76.0	50.6	63.2	83.9

WARM SKIN & WARM CORE (WW)

<i>Subject</i>	SBP pre (rest)	5min	10min	15min	20min	Δ	TPR pre (rest)	5min	10min	15min	20min	Δ
<i>DM</i>	118.0	154.0	164.0	169.0	172.0	18.0	681.5	302.5	295.8	313.0	290.0	-12.5
<i>JK</i>	136.0	199.0	199.0	205.0	210.0	11.0	883.6	349.4	315.5	335.3	343.3	-6.1
<i>KC</i>	118.0	152.0	163.0	163.0	163.0	11.0	1034.7	292.7	313.5	296.0	293.8	1.2
<i>JT</i>	146.0	197.0	206.0	191.0	179.0	-18.0	1107.6	399.3	354.8	333.7	316.9	-82.4
<i>NY</i>	122.0	172.0	174.0	177.0	179.0	7.0	1380.3	514.9	444.7	408.8	367.4	-147.4
<i>TC</i>	139.0	200.0	200.0	210.0	201.0	1.0	1096.1	370.6	331.2	389.6	375.8	5.1
<i>BM</i>	131.0	219.0	222.0	219.0	219.0	0.0	980.6	344.4	345.5	337.2	284.8	-59.7
<i>NV</i>	117.0	161.0	176.0	168.0	162.0	1.0	1121.7	408.8	425.6	393.1	346.0	-62.8
<i>Mean</i>	128.4	181.8	188.0	187.8	185.6	3.9	1035.8	372.8	353.3	350.8	327.2	-45.6
<i>S.D.</i>	11.2	25.2	21.7	21.5	21.7	10.8	202.3	70.6	54.0	41.1	35.8	53.0

WARM SKIN & WARM CORE (WW) - FANS ON

<i>Subject</i>	Tsk				Tes			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	25.8	25.1	25.0	25.1	38.1	37.6	37.2	37.8
<i>JK</i>	26.1	25.7	25.5	25.6	38.3	38.2	38.2	38.2
<i>KC</i>	27.0	26.5	26.3	26.3	38.4	38.1	38.0	37.9
<i>JT</i>	26.7	n.d.	n.d.	n.d.	38.5	n.d.	n.d.	n.d.
<i>NY</i>	25.5	n.d.	n.d.	n.d.	38.7	n.d.	n.d.	n.d.
<i>TC</i>	26.7	26.5	n.d.	n.d.	38.7	38.6	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	26.7	26.3	26.3	26.3	39.0	38.8	38.7	38.6
<i>Mean</i>	26.4	26.0	25.8	25.8	38.5	38.3	38.0	38.1
<i>S.D.</i>	0.5	0.6	0.6	0.6	0.3	0.4	0.6	0.4

<i>Subject</i>	VO2				RPE			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	3.4	3.1	3.2	3.4	12.0	13.0	13.0	13.0
<i>JK</i>	2.9	2.9	2.9	3.0	12.0	12.0	12.0	12.0
<i>KC</i>	3.3	3.3	3.4	3.4	14.0	14.0	13.0	13.0
<i>JT</i>	2.9	n.d.	n.d.	n.d.	18.0	n.d.	n.d.	n.d.
<i>NY</i>	3.1	n.d.	n.d.	n.d.	16.0	n.d.	n.d.	n.d.
<i>TC</i>	3.0	3.1	n.d.	n.d.	16.0	16.0	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	2.1	2.1	2.1	2.2	13.0	12.0	11.0	10.0
<i>Mean</i>	3.0	2.9	2.9	3.0	14.4	13.4	12.3	12.0
<i>S.D.</i>	0.4	0.5	0.6	0.6	2.3	1.7	1.0	1.4

WARM SKIN & WARM CORE (WW) - FANS ON

<i>Subject</i>	HR				Q			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	166.5	160.1	162.2	163.3	24.3	20.7	22.6	24.9
<i>JK</i>	161.0	157.1	156.5	154.6	22.5	22.0	22.7	22.8
<i>KC</i>	175.8	172.3	169.4	168.3	26.7	24.8	25.6	26.5
<i>JT</i>	170.6	n.d.	n.d.	n.d.	23.5	n.d.	n.d.	n.d.
<i>NY</i>	186.1	n.d.	n.d.	n.d.	22.3	n.d.	n.d.	n.d.
<i>TC</i>	161.1	161.8	n.d.	n.d.	22.3	22.9	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	184.6	184.3	184.0	181.4	19.2	21.4	21.7	23.1
<i>Mean</i>	172.3	167.1	168.0	166.9	23.0	22.3	23.1	24.3
<i>S.D.</i>	10.3	11.2	11.9	11.2	2.3	1.6	1.7	1.7

<i>Subject</i>	SV				SBF			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	146.2	129.1	139.3	152.7	84.2	77.3	84.2	90.0
<i>JK</i>	139.7	139.9	145.0	147.7	143.4	134.9	127.7	134.0
<i>KC</i>	152.0	144.1	151.0	157.4	152.3	198.5	164.6	173.4
<i>JT</i>	137.8	n.d.	n.d.	n.d.	154.3	n.d.	n.d.	n.d.
<i>NY</i>	119.7	n.d.	n.d.	n.d.	95.0	n.d.	n.d.	n.d.
<i>TC</i>	138.3	141.5	n.d.	n.d.	113.9	121.5	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	103.8	115.9	117.7	127.3	98.6	79.3	80.8	82.0
<i>Mean</i>	133.9	134.1	138.3	146.3	120.3	122.3	114.3	119.9
<i>S.D.</i>	16.6	11.6	14.5	13.3	29.4	49.6	39.7	42.4

WARM SKIN & WARM CORE (WW) - FANS ON

<i>Subject</i>	MAP				DBP			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	85.7	84.7	88.7	88.7	50.0	49.0	48.0	50.0
<i>JK</i>	103.3	100.7	101.0	100.3	56.0	51.0	53.0	57.0
<i>KC</i>	92.3	90.3	80.3	88.7	55.0	57.0	47.0	53.0
<i>JT</i>	99.0	n.d.	n.d.	n.d.	52.0	n.d.	n.d.	n.d.
<i>NY</i>	113.7	n.d.	n.d.	n.d.	79.0	n.d.	n.d.	n.d.
<i>TC</i>	103.0	101.7	n.d.	n.d.	59.0	56.0	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	98.3	93.3	98.3	100.3	65.0	65.0	66.0	70.0
<i>Mean</i>	99.3	94.1	92.1	94.5	59.4	55.6	53.5	57.5
<i>S.D.</i>	8.9	7.1	9.5	6.7	9.9	6.2	8.7	8.8

<i>Subject</i>	SBP				TPR			
	25min	30min	35min	40min	25min	30min	35min	40min
<i>DM</i>	157.0	156.0	170.0	166.0	281.5	327.8	314.0	284.5
<i>JK</i>	198.0	200.0	197.0	187.0	367.4	366.6	356.0	351.4
<i>KC</i>	167.0	157.0	147.0	160.0	276.4	291.2	251.2	267.8
<i>JT</i>	193.0	n.d.	n.d.	n.d.	336.9	n.d.	n.d.	n.d.
<i>NY</i>	183.0	n.d.	n.d.	n.d.	408.0	n.d.	n.d.	n.d.
<i>TC</i>	191.0	193.0	n.d.	n.d.	369.8	355.3	n.d.	n.d.
<i>BM</i>	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
<i>NV</i>	165.0	150.0	163.0	161.0	410.6	349.4	363.4	347.7
<i>Mean</i>	179.1	171.2	169.3	168.5	350.1	338.1	321.2	312.8
<i>S.D.</i>	16.0	23.4	20.9	12.6	54.8	29.8	51.4	42.9

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VITA

Joshua Floyd Lee was born in Temple, Texas. He completed high school at Temple High School in 1999. He later attended Texas State University in San Marcos Texas where he earned the degree of Bachelor of Science. During his undergraduate work and years following, Josh was an active member in the triathlon and endurance sports community, racing as a top age-group athlete in over 10 Half-Ironman, 6 Ironman, 3 marathons, and numerous other multisport and cycling events. His Ironman racing experience includes 4 finishes at the World Championship event contested in Kailua-Kona Hawaii, where he placed 2nd in the Mens 20 – 24 age division in 2005. Josh worked as a coach and sports nutrition consultant for beginner and elite triathletes and cyclist. In 2008, Josh enrolled at The University of Texas at Austin to pursue a Master of Arts in Kinesiology, under direction of Edward F. Coyle, Ph.D. While earning this degree he worked as a Graduate Research Assistant in the Human Performance Laboratory and a Teaching Assistant for Physiology and Anatomy in the Department of Biology.

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