THERMAL PERCEPTION DURING EXHAUSTIVE EXERCISE IN THERMALLY STRESSFUL CONDITIONS

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Department of Kinesiology and Health Science
Abstract

of

THERMAL PERCEPTION DURING EXHAUSTIVE EXERCISE IN THERMALLY STRESSFUL CONDITIONS

by

Max Marvin McLeod Polin

Introduction: The mechanisms of caffeine and other adenosine antagonistic pharmaceuticals are theorized to act primary at the central level. Previous research has shown that depression of rate of perceived exertion (RPE) during exercise has been depressed following caffeine (Caff) consumption. An unchanged perception of thermal score (TS) despite increased core temperature (Tc) has also been shown. Athletes may be prone to heat illness if TS and RPE perception is decreased during exercise in thermally challenging environments. Purpose: To determine the influences of caffeine on perceptual variables and time to exhaustion during continuous, sub-maximal exercise in a heated chamber, using trained cyclists. Methods: Nine competitive cyclists completed a graded exercise test (GXT) and a sub-maximal detection test, identifying VO2peak and the power [watts (W)] at 5% below the first ventilation threshold (VT1), respectively. One control (C) trial was conducted, followed by two double-blind trials using caffeine (5mg/kg bodyweight) or a placebo. All trials were held at constant load exercise at the W reported from the sub-maximal test, and were conducted to exhaustion or terminating criteria. All trails were held in a heated chamber to 35-37°C and 30-40% humidity. Analysis between the trials used a two-way repeating measures ANOVA, with an α-level of .05.
Results: There was no significant effect on time to exhaustion (TTE). End trial Tc for Caff trials was significantly different than placebo (P), $p = .009$. However, no difference was found for average mean Tc between trials. No significant difference was found for TS or RPE. Significant difference in Thirst (THS), $p = .048$, was found between P and C. Strong correlation ($r = .834$, $r = .832$, $r = .916$) existed between time and Tc for C, P, and Caff, respectively. C, P, and Caff were highly correlated for Tc vs. TS, ($r = .872$, $r = .754$, $r = .803$), respectively. For RPE, C, P, and Caff were highly correlated, ($r = .782$, $r = .717$, $r = .876$), respectively. Slope-intercept analysis found Caff to have higher y-intercept for Tc, and depressed y-intercepts for RPE, TS, and THS when compared to P. Conclusion: The results of this investigation found perceptual variables and TTE to not be significantly affected by Caff, although trends towards significance were present. This investigation found the Caff group to have end-trial Tc that was significantly higher than P with no increase in TS or RPE. It may be possible that Caff promotes tolerance of higher Tc due to decreased perceptual feedback at the central level. It may also be possible that the instruments used to collect TS were not appropriate to detect significant difference in TS.

_______________________, Committee Chair
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1. **INTRODUCTION**

Because of its proven ergogenic effects, caffeine, is one of the most widely consumed and commercialized stimulants world-wide, and is abused by endurance athletes during competition (Burke, 2008; Doherty & Smith, 2005). Furthermore, caffeine’s ergogenicity has also been repeatedly examined in a variety of experimental conditions and athletic populations (Hogervorst, et al., 2008; Roti, et al., 2006). Neither caffeine ergogenicity, nor caffeine metabolism (Ganio, et al., 2011), is affected by thermal stress, thus allowing prolonged exercise durations and higher core temperatures to be achieved without a degradation to caffeine’s structure (Roti, et al., 2006). Subjects who have ingested caffeine during thermal stressful exercise have reported identical thermal, thirst scores, and RPE as subjects who reached fatigue sooner and had lower core temperatures (Roti, et al., 2006). The apparent blunting of RPE increasing with thermal temperatures is in opposition to the responses found in non-caffeinated subjects (Nybo & Nielsen, 2001).

These findings indicate that caffeine is acting as an agonist for physical performance and altering perception of thermal stress. Without the reduction in motor-drive from the CNS normally associated to EICF from hyperthermia, consumers of caffeine may be unaware of increasing core temperatures up to and beyond the attainment of critical core temperature (40˚C), placing them in danger of immediate heat illness. Additional cause for concern associated with caffeine ingestion during exercise in thermal conditions is its relationship to decreased sensation of thirst (Smit, & Rodgers, 2001). Caffeine has been found to be a potent ergogenic aid even in the presence of dehydration and decreased food intake during exercise (Del Coso, et al., 2008). Attenuation of thirst response further promotes the accumulation of metabolic heat in the absence of the core cooling effects of fluid intake, and challenges thermal homeostasis.
Thermal homeostasis is critically important during exercise in heat. The hypothalamus, the thermoregulatory center of the body, is responsible for alterations in cognitive and physiological function to maintain thermoregulation during exercise (Griffin, Kaple, Chow, & Boulant, 1996). The primary method of preventing heat excess during exercise is to maintain an appropriate core to skin temperature gradient, which effectively allows for the dissipation of metabolic heat production (Wendt, van Loon, & Lichtenbelt, 2007). Secondarily, inhibition of the Central Nervous System (CNS) motor neuron efferent signals to skeletal muscle fibers may attenuate metabolic heat production (Gonzalez-Alonso, et al., 1999; Nybo & Nielsen, 2001).

In healthy populations, heat related illness is usually avoided and safe thermal gradients within the body are maintained, possibly at the expense of exercise performance (Gonzalez-Alonso, et al., 1999; Nybo & Nielsen, 2001). Thermoregulatory alterations are the direct result of afferent signaling from thermal sensitive nerve endings of the skin, internal organs, and exercising muscles (Wendt, Loon, & Lichtenbelt, 2007). These signals provide information as to the rate of heat storage and potential heat accumulation within the body as well as the environmental conditions in which the body is exercising (Tucker, Rauch, Harley, & Noakes, 2004). This perception of current and potential thermal stress is known as “teleoanticipation” and has been associated with decreased motor-drive from the Central Nervous System (CNS) even prior to the onset of elevated core temperatures (Tucker, Rauch, Harley, & Noakes, 2004). Tucker, et al. reported that in self-paced time trials in heat, reduced motor recruitment was found prior to the attainment of a core temperature of 38°C. Conclusions were made that the thermoregulatory systems of the CNS decreased work output to allow for the completion of the exercise trial and avoid exercise-induced heat illness (Tucker, Rauch, Harley, & Noakes, 2004). This occurrence has been noted in experienced runners in field conditions as well (Ely, et al.,
leading to the belief that peripheral sensory feedback may be as important as core temperature in modifying motor-drive.

The second mechanism for detection of thermal stress is hypothalamic monitoring of cerebral blood temperature (Boulant, 2006). As cerebral arterial blood flows to the hypothalamus, thermosensitive receptor cells monitor blood temperature and rates of heat storage (Cheung, 2010). If arterial blood approaches the proposed critical core temperature of 40°C (Gonzalez-Alonso, et al., 1999), the CNS appears to reduce motor neuron efferent signals to the exercising muscles in efforts to reduce metabolic heat production from skeletal muscle contraction (Nybo, & Nielsen, 2001). This precise overseeing of cerebral blood temperature has been attributed to the high density of thermosensitive neuronal cells of the hypothalamus, which initiate changes to prevent further thermal stress via efferent signaling of muscular contraction intensities (Wendt, Loon, & Lichtenbelt, 2007). This suggests that central perception of cerebral blood temperature signals the reduction of cerebral efferent motor-drive to prevent any further increases in Voluntary Activation (VA), possibly to avoid increasing the heat storage effects of metabolism and potential damage to body tissue (Nybo & Nielsen, 2001; Roelands & Meeusen, 2010).

Such modifications of central control to heat stress have been noted with alterations in cerebral brainwave patterns and plateaus in voluntary activation (VA) of exercising muscles (Nybo, & Nielsen, 2001). Their experiments produced a strong positive correlation between increasing core temperature and electroencephalography (EEG) alterations during submaximal cyclergometry to exhaustion in a heated chamber. These alterations in brain wave amplitude were hypothesized to reflect the increasing difficulty of the pre-frontal cortex to affect motor-drive and match the required work rate of the exercise trial (Nybo, & Nielsen, 2001). Of note is the strong positive correlation found to exist between alterations in EEG and subjective rate of perceived exertion (RPE), as well as between RPE and increased core temperature.
The precise mechanism behind this reduced ability to affect increased muscle recruitment despite the subject’s increased effort to intensify muscular force production remains unclear (Cheung, 2010), but the signaling to the exercising muscle to continue contraction is reduced or blunted in the presence of increased core temperature. If efferent motor-drive from the Central Nervous System (CNS) was allowed to continue to drive exercise without a reduced intensity of contraction of the exercising muscles, the possibility for increased susceptibility to heat illness occurs.

In healthy individuals, the critical core temperature of 40°C suggested to exist by Gonzalez-Alonso has been proposed to act as a “safety brake” (Nielsen & Nybo, 2003) against heat illnesses, such as Exertional Heat Stroke (EHS). EHS is defined as a rectal temperature in excess of 40°C at collapse and by central nervous system changes (Armstrong, et al., 2007). Heat stroke has been associated with discomfort, headache, nausea, vomiting, and when left untreated, can lead to CNS dysfunction, seizure or coma and possibly death (Armstrong, et al., 2007). In healthy individuals EHS is unlikely to occur because of the down-regulation of the central nervous system efferent signaling, known as Exercise Induced Central Fatigue (EICF). Down-regulation of efferent signaling of EICF effectively prevents further accumulation of metabolic heat storage and has been found to lead to volitional fatigue and exercise termination during experimental testing (Nybo & Nielsen, 2001). This effect has been most noted during constant-load or constant-intensity efforts, in which self-pacing strategies and teleoanticipation are excluded (Nybo & Nielsen, 2001).

To test the mechanisms associated with EICF, pharmacological manipulation of the responses to heat stress has been experimentally examined (Nybo & Nielsen, 2001; Roelands & Meeusen, 2010).
Drugs, which act as dopamine reuptake inhibitors, have been found to increase heat tolerance and attenuate decreases in VA (Nybo & Nielsen, 2001; Roelands, & Meeusen, 2010). Bupropion, a dopamine reuptake inhibitor, was found to effectively increase core temperature during time trial performance in high thermal conditions without the expected increase in time to completion, RPE or thermal stress score (Watson, et al., 2005). In addition, Methlyphenidate, a CNS stimulant and dopamine reuptake inhibitor, was found to increase core temperature during time trial performance in heat over placebo (Roelands, et al., 2008). Subjects completed the required amount of work faster than placebo without increased RPE or thermal stress score (Roelands, Hasegawa, & Watson, 2008). The results of these experimental trials led to the proposal that methylphenidate and bupropion may inhibit signaling of increasing core temperatures to the CNS (Roelands, Hasegawa, & Watson, 2008).

A failure of the CNS to detect thermal stress may potentially limit the reduction in voluntary motor-output. A conclusion was made that there may be risk associated with ingestion of methylphenidate for increasing risk of heat illness, and possibly the attainment of core temperatures in excess of 40°C, during exercise in warm conditions (Roelands, Hasegawa, & Watson, 2008).

1.1 Problem

This conclusion may carry considerable implication for caffeine, as caffeine acts similarly to methylphenidate and promotes central dopamine transmission. The perception of thermal stress is attenuated or overridden with dopamine reuptake inhibitors (Nybo & Nielsen, 2001; Roelands & Meeusen, 2010), thus resulting in inhibition of signaling to cease exercise (Maughan, 2010).
1.2 Purpose

It is the purpose of this study is to investigate the influences of caffeine during sub-maximal exercise under thermal stressful conditions on perceived thermal stress, physical thermal stress, and time to exhaustion using competitive cyclists.

1.3 Significance of Thesis

Caffeine has been heavily researched among endurance athletes because of its ergogenic properties in both thermal neutral and thermal hot conditions. This research has yielded data suggesting that perceptual cues of dangerous levels of internal core temperature are dampened with caffeine ingestion. When these effects of caffeine are combined with its psychostimulating properties, the potential for thermal injury is high. Yet, there is an absence of scientific inquiry into the perception of thermal stress markers, such as thermal perception, RPE, and thirst in relation to caffeine dosing during exercise in thermal hot conditions.

1.4 Definition of Terms

Thermal Stress – physiologic alterations within the body caused by high internal and/or external temperatures.

Metabolic Heat – chemical and mechanical factors within the body that cause the production of body heat.

Central Nervous System – representing the division of the nervous system consisting of the brain and the spinal cord.

Down-regulation – the reduction of skeletal muscle signaling from the CNS.

Rate of Heat Storage – the increase in internal core temperature over time.

Teleoanticipation – the product of all sensory feedback information sent to the CNS regarding internal and external temperatures prior to the inset of increased core temperature.

Motor-drive – efferent signals from the CNS to affect skeletal muscle activation.
Hyperthermia – core temperature in excess of 40°C.

Efferent – representative of the CNS signals relayed to the periphery.

Afferent – representative of the peripheral signals relayed to the CNS.

Voluntary Activation – the quantity CNS signaling to reach of muscle fibers ending during exercise.

Electroencephalography – electrical activity within CNS and amplitude of brain waves.

Rate of Perceived Exertion – the perception of stress felt across the entire body.

Dopamine Reuptake Inhibitor – a compound which reduces the removal of dopamine from plasma circulation.

Ergogenic – performance enhancing.

Exercise-Induced Heat Illness- injury to body tissues as a result of metabolic heat production during exercise.

Sub-maximal- exercise that is not maximal intensity. Exercise that is less than 100% VO2max.

Constant-load- exercise that is held at a constant mechanical or resistive work-load.

1.5 Hypotheses

1) When compared to control conditions, there will be no significant decrease in thermal stress score, thirst score, or RPE during constant load exercise in a hot thermal environmental condition after caffeine ingestion.

2) When compared to control conditions, there will be no significant alteration in subject’s internal core temperature relationship with RPE, thermal stress score, and thirst score after caffeine ingestion.

3) When compared to control conditions, there will be no significant effect of caffeine ingestion on attainment of 39.5°C.
4) When compared to control conditions, there will be no significant change in time to exhaustion during sub-maximal, constant-load exercise in a high thermal environment conditions after caffeine ingestion.
2. REVIEW OF LITERATURE

2.1 Introduction

Safe and effective exercise in high thermal conditions necessitates the ability to achieve an effective thermal gradient. In the absence of such temperature equilibrium, the Central Nervous System (CNS) appears to dampen Voluntary Activation (VA) of working skeletal muscle fibers to disallow further metabolic heat production. This decrease in VA has been proposed to act as a preventative measure against the “critical core temperature” of 40°C (Gonzalez-Alonso, et al., 1999) and heat stroke conditions. Research has been conducted to ascertain whether ergogenic CNS stimulants, such as caffeine, can enhance athletic performance and limit VA reduction in both thermal neutral and thermal hot conditions (Costill, Dalsky, & Fink, 1978; Kovacs, Stegen, & Brouns, 1998). The results of caffeine administration have made evident that caffeine blunts the rate of perceived exertion (RPE). Reported data has also shown that in thermal hot environments, there is a strong correlation between core temperature and RPE (Nybo & Nielsen, 2001). Furthermore, results have shown that, in thermal hot environments, a diminished perception of exercise exertion can facilitate heat stroke temperature (>40°C) attainment within exercising subjects. Additionally, Nybo has unreported data in which subjects attained core temperatures in excess of 40°C without an increase in central fatigue after caffeine administration (Nybo, 2008). Yet, to date, there is a lack of data regarding perception of thermal stress indicators during exercise in thermal hot conditions with caffeinated subjects, nor is there research to elucidate correlations between caffeine ingestion and depressed perception of thermal stress.

Little consensus exists concerning the mechanisms of physiological, cognitive, and behavioral changes during exercise under thermal stress (Cheung, 2010), yet it is apparent that the sensation of thermal distress or a threshold in absolute core temperature affects exercise
thermoregulatory responses and promotes central fatigue. Therefore, the successful modulation of exercise intensity to avoid unsafe core temperatures is dependent on the integration and perception of thermal stress cues.

2.2 Absolute Core Temperature and Exercise Performance

A proposed relationship between an absolute core temperature (known as Critical Core Temperature) and volitional fatigue and has been theorized to be an independent regulator of exercise performance (Gonzalez-Alonso, et al., 1999). Gonzalez-Alonso attempted to determine if an increase in absolute core temperature would result in voluntary termination of exercise in hot environments. Subjects received pre-exercise treatment to alter initial core and skin temperatures prior to exposure to exhaustive exercise in a 40˚C heated chamber. Results of the study showed that the esophageal temperature across all subjects at exercise termination (exhaustion) was 40.1± 0.1˚C, regardless of initial core temperature and pre-exercise treatment (Gonzalez-Alonso, et al., 1999). Time to exhaustion was significantly shorter for subjects who were pre-warmed, (28 minutes) to pre-cooled exercise duration (63 minutes). Yet RPE at exercise termination was equally elevated between conditions at 18.5±0.2 to 18.6±0.3 (Gonzalez-Alonso, at al., 1999), which indicates a strong correlation between RPE and rising core temperature.

Measured rates of fuel utilization and cardiac output were not significantly affected at fatigue during the trials, leading to the conclusion that neither metabolic limitations nor cardiovascular limitations were the result of exercise termination (Gonzalez-Alonso, et al., 1999). And although it was not known if high core temperature placed a physiological limit upon exercise or if its perception by the CNS caused reduction in VA, it was likely that exhaustion was directly related to exercise-induced central fatigue (ECIF).
2.3 Perception and Heat Storage

Rate of heat storage has also been implicated in hyperthermia-induced fatigue (Gonzalez-Alonso, et al., 1999). To examine the role of central perception of environmental conditions influencing central modulation of exercise intensity during environmental thermal stress, ten male cyclists performed a 20-kilometer time trial on a stationary cyclergometer in a 35°C heated chamber and in a thermal neutral condition of 15°C (Tucker, et al., 2004). Both trials were standardized to ensure that initial RPE, core temperature (Tc), and heart rate (HR) were not significantly different between environmental conditions (Tucker, et al., 2004). Onset skin temperatures were significantly higher at kilometer zero in the hot than in cool conditions, and remained higher to exercise completion. Yet the initial and ending skin temperatures for the hot trial were not significantly different (Tucker, et al., 2004). Power output was not significantly different between trials for the first 30% of the trial duration, at which point, rate of decline increased with the 35°C subjects in comparison to the 15°C subjects, reaching significance at 80%-100% of the time duration (Tucker, et al., 2004). Both hot and cool end power outputs were significantly higher than the initial starting power, suggesting that peripheral fatigue was not responsible for the reported decline in power output (Tucker, et al., 2004). Furthermore, these results occurred in the absence of a significant between-trial variance in Tc or attainment of 40°C Tc, until 15 kilometers, indicating core temperature was not a limiter during self-paced efforts. Tucker noted that rates of heat storage were not different between environmental conditions. Therefore, it was proposed that thermal afferents regarding environmental temperature and the potential for heat storage rate perceived by the CNS caused a down-regulation of VA and motor recruitment and avoidance of 40°C Tc prior to the completion of the exercise interval (Tucker, et al., 2004).
Central perception of thermal stress, both external and internal, elicits changes to efferent signaling to working muscles, limiting metabolic heat production and increasing in core temperature. Therefore, a 40˚C core temperature appears to be avoidable during exercise performance during self-paced exercise trials in compensable environments, provided perception of thermal stress is inherent, regardless of the mode of sensation. But Tucker’s approach to this subconscious monitoring of the rate of heat storage has been refuted. It has been argued that CNS modulation of VA via calculation of rate of heat storage by perception of internal heat accumulation was highly unlikely (Jay & Kenny, 2009). Furthermore, it was suggested that the only evidence of anticipatory response to thermal stress is derived from changes in skin temperature (Jay & Kenny, 2009). This stance has been supported (Ely, et al., 2009). Tucker’s results showed that skin temperature in hot conditions did not vary throughout the trial. The skin temperatures rose, but appeared to be modulated by forehead temperature. This finding is significant given that physiological modifications and reduced RPE in response to face cooling have been well documented (Mundel, Bunn, Hooper, & Jones, 2007), further suggesting the role of skin’s neurological feedback influence on perception of thermal stress.

Regardless of the conclusion made by Tucker, the significance of the self-selected reduction in power-output in the absence of a critical core temperature in Tucker’s results cannot be overlooked. A sensation of physical effort may be involved in the decision making process of a conscious reduction in voluntary exertion. Marcora argues that sensations that contribute to an overall sensation of effort and physical stress during exercise may directly motivate subjects to reduce the intensity of voluntary muscular effort (Marcora, Staiano, & Manning, 2009). This viewpoint is relevant in that Tucker reported that subjective RPE was near maximal, in both experimental groups at exercise termination, despite the reduction in power output in the 35˚C chamber. Taken together, the results of this study illustrate that the of reduction of VA in thermal
hot conditions arises in the Central Nervous System and not only regulates muscle fiber recruitment during self-paced efforts, but also the potential for excessive heat production as a result of muscular contraction.

2.4 Central Nervous System Perception

Research concerning interconnections between Central Nervous System alterations and heat tolerance has been conducted during constant-intensity exercise under thermal stressful conditions (Nybo & Nielsen, 2001). Nybo and Nielsen investigated whether hyperthermia-induced cerebral electrical changes during exercise would be related to alterations muscle fiber recruitment. Secondly, it was sought to relate cerebral and muscular activity to RPE. Nybo and Nielsen observed subjects exercising at 60%VO2max on a cyclergometer in a heated 40°C chamber to exhaustion, and then made comparisons to subjects performing the same exercise in 18°C. As Tc increased in both trial groups, RPE, alterations in cerebral electrical activity rose concomitantly. In thermal neutral exercise conditions (18°C), the increases in RPE, Tc and alterations in cerebral electrical activity stabilized. Subjects exercising in the thermal hot chamber (40°C) had continually increasing Tc, RPE, and alterations in cerebral electrical activity until exhaustion. Graphical analysis of RPE plotted against core temperature and alterations in cerebral activity reached strong positive linear correlation (r = 0.94, P<0.001) (Nybo & Nielsen, 2001). Nybo and Nielsen suggest that constant-intensity exercise in thermal hot conditions increases core temperature, amplitude in cerebral electrical activity (r = 0.94, P<0.001), and RPE (r = 0.98, P<0.001). These results further suggest the possible causal relationship between elevated Tc and EICF during exercise. This was supported by the lack of alterations in EMG recordings on the vastus lateralis muscle during exercise, even in the presence of maximal RPE and subjects’ attempt to sustain exercise intensity (Nybo & Nielsen, 2001).
CNS electrical alterations and increased RPE may directly impact subjective motivation for continued voluntary efferent signaling to muscles and maintenance of intensity. It has been postulated that motivation and desire to continue a task may be dampened by perception of effort and “mental fatigue” (Marcora, Staiano, & Manning, 2009). Investigative research revealed that mentally fatiguing subjects prior to an exercise bout caused a reduction in voluntary physical effort and a shortened time to exercise exhaustion (Marcora, Staiano, & Manning, 2009). Marcora claimed that his results were in-line with proposed motivational theories, in that, the subjects’ level of willingness to perform intense exercise was inversely related to the degree of mental fatigue they incurred (Marcora, Staiano, & Manning, 2009). This becomes relevant during prolonged exercise in adverse conditions because of the relationship between EICF and VA.

Physiologically, the nucleus accumbens is a region of the brain directly linked to motivation, behavior and sense of effort (Farrar, et al., 2010). Localized inhibitions of adenosine receptors in this region have been found to induce depression of locomotor activity in animal models (Barraco, Martens, Parizon, & Normile, 1993). Given that physical exertion increases levels of adenosine, it may be possible that the nucleus accumbens becomes impaired during exercise, thereby interfering with motivation and voluntary locomotive drive to skeletal muscles. Furthermore, since increased RPE is in part the result of increased sense of effort, it can be speculated that increased RPE may depress motivation to continue a physical task, should the task become too subjectively demanding.

2.5 Caffeine’s Mechanisms and Ergogenicity

Usage of ergogenic supplements that alter RPE and motivation may have greater potential for harm if EICF is attenuated for extended periods of time during hyperthermia. Caffeine, and other dopamine re-uptake inhibiting CNS stimulants, has been heavily abused by
athletic populations because of the proven ergogenic effects, which mitigate decreases in muscle activation, and for its ability to reduce perception of exercise intensity, pain and prolong exercise duration (Burke, 2008; O’Connor, Motl, Broglio, & Ely, 2004; Olson, Thornton, Adam, & Lieberman, 2010). During exercise in hot environmental conditions caffeine has been found to attenuate increases in thermal stress score despite prolonged exercise time and core temperature (Roti, et al., 2006). Furthermore, Ely found subjects to have diminished perception of thermal stress during exercise despite increased core temperature (Ely, Ely, & Cheuvront, 2011). Ely had subjects perform cyclergometry at 50% VO2peak in a 40°C chamber after ingesting a single bolus of caffeine and found there to be an increase of approximately 0.25°C Tc after caffeine ingestion (Ely, Ely, & Cheuvront, 2011). Yet compared to the placebo group, which was in a neutral temperature environment, thermal comfort rating was slightly lower or did not differ at any point during the exercise trial. The changes had been due to a large caffeine dose of 9mg/kg, which may have affected resting and exercise metabolic rate. Moreover, neither exercise intensity nor duration was aggressive enough to promote hyperthermic Tc (~40°C) in any subject. Investigation into potential correlations between caffeine ingestion and perception of thermal stress and actual physiological strain has not been conducted.

Pharmophysiologically, caffeine has been found to affect tissue sites throughout the body, but at the central level it effectively antagonizes adenosine receptor sites, A1 and A2A (Ferre, 2010). This interaction with pre-synaptic adenosine receptor sites allows for a cascade of events of occur within the nervous system that enhance sympathetic neural drive and elevate positive mood states. A byproduct of energy metabolism is adenosine, which is a hormone that promotes restfulness and drowsiness. Caffeine effectively blocks A1 receptor site uptake of adenosine from circulation, and is proposed, therefore, to enhance motivation and arousal (Ferre, 2010). This effect has been
reported by Roti (Roti, et al., 2006) who examined chronic caffeine ingestion in comparison to tolerance of exercise in heat and alteration in fluid-electrolyte balance. The subjects treadmill walked in 37°C room temperature to exhaustion after consuming a bolus of caffeine. Subjects who ingested 3mg/kg caffeine prior to the exercise bout reported enhanced mood states and vigor the day prior to testing, compared to 6mg/kg caffeine subjects and placebo. Data was not presented for the exercise trial, but inferences were made relating enhanced mood scores to exercise performance increases (Roti, et al., 2006). Caffeine has also been heavily linked to the dopaminergic system, which is densely populated with A2A receptor sites (Yoshimura, 2005). As such, caffeine acts secondarily as a dopamine re-uptake inhibitor, which seems to allow for increased efferent signaling and increased motivation in test subjects (Ikemoto, 2007). Such an interaction with the dopaminergic system at the central level, may have implications for altering mental fatigue, perception of effort, and effort-based decision making, to which Marcora alluded (Marcora, Staiano, & Manning, 2009). Postsynaptically, caffeine has been found to play a role in the active down-regulation of GABA receptors, which act as neural transmission inhibitors (De Koninck, & Mody, 1996). Neurologically, caffeine acts as a nervous system stimulant as it maintains neurotransmission across synaptic clefts and also acts as both a promoter of wakefulness and an inhibitor of central fatigue. These actions may be responsible for the increased mood states and motivation that are reported during exercise conditions.

These ergogenic properties have been found unchanged regardless of environmental or subject characteristics. Observations were made on trained male cyclists in 12°C and 33°C ambient temperatures exercising at 60%-70% VO2max intensity for 90 minutes (Ganio, et al., 2011). Subjects ingested either a placebo or 2 doses of 3mg/kg caffeine given at time intervals during the exercise trial. Subjects then completed a 15-minute maximal exertion time trial. There was found to be an overall positive effect on work performed after caffeine ingestion for
the 15-minute time trial, independent of ambient temperature (Ganio, et al., 2011). A decrease in kilojoules performed was noted in the 33°C temperature for all subjects compared to 12°C, but not to the effect that caffeine ergogenicity was negated (Ganio, et al., 2011). Ganio reported caffeine to have no significant affect on cardiovascular, metabolic, body temperature or hematological values in either environmental condition, compared to placebo. Ganio reported subjective RPE being identical (20±1) post-time trial in all of the experimental conditions, indicative of maximal effort. Reportedly, no subject reached the critical core temperature of 40°C during any trial, yet Tc data was not provided for the time trial effort. However, Tc appeared to be highest in the 33°C/Caffeine group after 30 minutes of exercise for the duration of the experiment. A further delimitation of this study was that subjects were required to consume 22°C fluid replacement in the amount 80% of the expected sweat loss every 15 minutes during exercise. This likely was able to control Tc increases, which may not be possible during exercise bouts in non-laboratory settings.

Additional investigation into caffeine’s ergogenic action during various exercise conditions has been conducted (Del Coso, et al., 2008). Seven trained male cyclists performed 120 minutes of cycling exercise at 63 ± 5% VO2max in a 36 ±3°C room, while performing intermittent maximal sprints. Six experimental trials were conducted with each subject serving as his own control. The trials included: 1) no fluid placement, 2) caffeine (6mg/kg) ingestion prior to exercise, 3) water replacement only, 4) water replacement plus caffeine (6mg/kg), 5) carbohydrate drink fluid replacement, 6) carbohydrate drink replacement plus caffeine (6mg/kg) (Del Coso, et al., 2008). Subjects in the caffeine only group lost 3.8 ± 0.3% of initial body weight and had peak core temperature readings of 39.4 ± 0.5°C. These readings were higher than trials with fluid replacement. Grouped data across all trials indicated that caffeinated subjects maintained muscle contractile quality pre-exercise to post-exercise and increased maximal
cycling power 3 ± 1% over non-caffeine groups (Del Coso, et al., 2008). Interestingly, the ingestion of caffeine alone allowed subjects to maintain VA significantly more than subjects with water replacement or water plus caffeine (Del Coso, et al., 2008). Results of this study indicate that caffeine may allow for the attainment of higher core temperatures, possibly as a result of VA maintenance and muscle contractile quality. Results occurred even in the presence of dehydration, which has been implicated in the hastened accumulation of heat storage and as a precursor to heat illness (Armstrong, et al., 2007). Interestingly, it also appears caffeine may attenuate the thirst response to dehydration (Roti, et al., 2006; Smit, & Rodgers, 2001).

Diminished perception of thirst may allow for a hastened rate of heat storage, and may be attributed to Marcora’s model of mental fatigue. Caffeine’s mechanisms may be potent enough to allow task motivation to persist even in the presence of extreme physical exertion and known physiological limitations to performance, such as hypohydration and 40°C Tc.

Del Coso provided no RPE or thermal stress data, yet it is apparent that subjective motivation, i.e. improvement in cycling power, was not impaired for the duration of the exercise trials when caffeine was ingested, even during marked dehydration and hyperthermic conditions. Further delimitations of this study were that subjects were acclimatized prior to the experimental trials; therefore, it is unknown how the experimental conditions affected unacclimatized subjects. Furthermore, the sprint bouts were spaced intermittently by 30 minutes, lasted only 4 seconds, and 2 minutes of recovery was given prior to resuming 63% VO2max power. It is possible that with continuous high intensity cycling, caffeinated subjects would have incurred higher core temperatures in excess of 40°C.

2.6 Thermal Tolerance among Endurance Athletes

It has been suggested that highly trained athletes may have a higher set point for thermal distress, in spite of the strong correlation between a 40°C acting as a set point for heat tolerance
and voluntary exercise termination (Cheung & McLellan, 1998). Cheung and McLellan suggested that highly trained populations with low adiposity may achieve greater heat strain and core temperature above 40˚C during exercise. This assumption was made after results showed that trained individuals reported prolonged resistance to exercise in uncompensable environments and at higher core temperatures than untrained, high adiposity populations. All subjects in the trained, low adiposity group had exercise terminated upon reaching the ethical Tc limit of 39.5˚C. It was further suggested that the lower perceived heat strain reported by trained individuals is likely not from a reduced perception of effort, but more from a reduced perception of thermal stress (Tikuisis, McLellan, & Selkirk, 2002). This tolerance of increased Tc and reduction of perceived effort may promote the maintenance of voluntary activation and metabolic heat generation after caffeine dosing, allowing for a dangerous Tc in excess of 40˚C.

2.7 Summary

Research has found that exercise during uncompensable conditions causes accumulation of heat storage within the body as a result of unbalanced thermal gradients. As core, skin and cerebral temperature increase there are cognitive and behavioral alterations that alter central motor drive. These changes preserve homeostasis and, thus, avoidance of hyperthermia and heat illness. The precise mechanism behind these alterations has not been established, however, consensus points to the alterations being caused directly by the hyperthermic condition itself or indirectly via CNS down-regulation of efferent motor-output to exercising muscles, due to decreased motivation and increased RPE. Strong correlations between CNS electrical activity alterations, Tc, and RPE have been made (Nybo & Nielsen, 2001). Therefore, it appears that difficulty maintaining constant-load exercise under thermal stress is the result of decreased signaling from the CNS, disallowing continued, elevated metabolic heat production.
Caffeine ingestion has been found to act as a CNS agonist and promote VA in the presence of thermal stress and EICF, and is associated with reduced RPE. Caffeine’s proposed characteristic of maintaining VA and increased motivation during expected EICF, has resulted in enhanced exercise performance. Yet, in uncompensable environments, this enhanced performance and reduced RPE has resulted in obtainment of hyperthermic core temperatures to be achieved. Most notably, hyperthermia occurs in elite endurance athletes and for extended periods of exercise duration. The abuse of caffeine as a performance-enhancing drug in elite athletes may increase risk of heat stroke temperature in this population because of decreased perception of physical heat strain.
3. METHODS

3.1 Design

There is a lack of research regarding perceptual changes to thermal stress after caffeine ingestion using athletes. The purpose of this study was to examine if there is a significant difference of perception of thermal stress during sub-maximal, constant-load exercise in heat after caffeine ingestion in cyclists. The study design was within subject, experimental and consisted of three separate trials. Each participant completed the three trials in a double-blind, randomized order, using Latin squares design, reducing the external variability of the study. Physiological and perceptual variables were collected pre, during and at the termination of each exercise trial. Physiological variables included: heart rate (HR), water consumption, body weight change, and internal core temperature (Tc). Perceptual variables included: thermal stress (THS), rate of perceived exertion (RPE), and thirst score (TS). Statistical analysis was completed to determine mean differences between the caffeinated group (CG), placebo group (PG), and the control group (CTG).

Subjects performed cycling trials at the same time of day, in respect to their first experimental trial, to limit diurnal variability in core temperature and exercise performance. Furthermore, data collection was conducted from March to May to avoid external heat acclimatization of the participants.

Subjects reported to the laboratory on four separate occasions over a four to five-week period. During Visit 1, informed consent was obtained, explanation of procedures was provided, and medical and athletic history was obtained to ensure inclusion criteria were met. The subjects then performed a maximal graded exercise test (GXT) on a Lode cyclergometer (Netherlands) to volitional fatigue. The results of the GXT were used to determine each subject’s peak
cardiovascular capacity (VO$_2$peak), as well to determine the initial workload for the VO$_2$
detection trial.

Twenty to thirty minutes post-GXT the subjects performed a VO$_2$ detection trial to detect
the power associated with the VO$_2$ (L/min) at 5% below the first ventilation threshold (VT1). At
that power, all experimental trials were conducted.
Visit 2 served as a control trial, whereas visits 3 and 4 were experimental. Seven to nine days
separated each trial to allow for recovery and avoid heat acclimation of subjects. Trials were
conducted in a heated chamber after the treatments were administered. All subjects cycled to
volitional fatigue, a drop in cadence of five revolutions per minute below baseline, or a core
temperature (Tc) of no greater than 39.5°C or unsafe signs and symptoms. End trial data was
collected and subjects exited the chamber and were monitored while pre-test conditions were
reestablished. The subjects were then thanked and allowed to leave the laboratory.

3.2 Subjects

Nine male volunteers volunteered for this study; recruitment occurred using advertising
in local bicycle shops and local cycling and triathlon clubs. Females were excluded to avoid
biological variation in core temperature due to the menstrual cycle. The experimental inclusion
criteria required that all subjects had at least two years of competitive experience in cycling
events. Subjects were also required to be habitual, moderate (1-6mg/kg/day) consumers of
caffeine (Burke, 2008). This was determined during the completion of initial questionnaires.
Cardiovascular inclusion criteria required that all subjects have a relative VO$_2$peak of
$\geq$50ml/kg/min, determined by a graded maximal exercise test (GXT) on a cyclergometer. This
was in place to control for expected heat tolerance and endurance capacity between subjects. All
volunteers met classification for low risk for health complications by the American College of
Sports Medicine Guidelines (Armstrong et al., 2009). Subjects provided signed informed consent
and became fully informed of their expected involvement with the study prior to their enrollment. This methodology met Human Subject approval, as authorized by the Sacramento State University, California Internal Review Board. All testing occurred in the Irvin Faria Exercise Physiology Research Laboratory, CSUS, Sacramento, CA.

3.3 Procedure

3.3a. Maximal Graded Exercise Test

Visit 1 included a GXT and a Detection Trial. Subjects arrived after a four-hour fast and twenty-four hours without vigorous exercise. These conditions were assessed by written recall. Informed consent and ACSM low risk stratification was obtained, as well as height, weight, blood pressure, and heart rate. Prior to testing, subjects adjusted the cyclergometer to their comfort and attached their own pedals. Subjects were fitted with a Polar Heart Rate chest strap and then sat atop the cyclergometer. A ten-minute active warm-up was given at 150 watts of resistance. At the conclusion of the warm-up, subjects were fitted with a nose-clip, headgear and two-way breathing valve (Hans-Rudolf, Kansas City, MO), which was connected to the pneumotach with a large-bore flexible plastic breathing hose. Respiratory gases were measured continuously during exercise using a ParvoMedics Trueone 2400 metabolic measurement system (Sandy, Utah), calibrated using medically certified gas of known concentration.

A 1-minute stage protocol was utilized for the GXT. Stage 1 began at 70 watts; the power was increased by 35 watts for each successive stage. Subjects were instructed to pedal at a cadence that is most reflective of their natural pedaling rate, but at a cadence of at least eighty-five revolutions per minute. Testing was terminated upon volitional fatigue. Rate of perceived exertion (RPE), using a 6-20 BORG scale (Borg, 1982), and HR were recorded at ten seconds prior the completion of each stage. Signs and symptoms were monitored continuously. In the case of abnormal signs and symptoms, the GXT was terminated.
3.3b. Detection Trial

The results of the GXT were used to determine VT1 using the ventilatory equivalents method of expired gases (Beaver, et al., 1986; Caiozzo, et al., 1982). Also noted was the GXT power associated with VT1. The VO$_2$ (L/min) 5% below VT1 was then calculated in addition to the power at 15% below VT1 watts from the GXT. The power at 15% below VT1 power served as the first stage power for the detection trial. Twenty to thirty minutes post GXT the subjects began cycling at this power (15% prior to VT1 wattage). Respiratory gases were continuously collected. After a period of four minutes, and a steady state in oxygen has been established (≤2ml/kg/min), the wattage was increased by five watts. This four-minute protocol was repeated until a steady state in oxygen was established at 5% VO$_2$ (L/min) below VT1. This repetition occurred until the subject’s average VO$_2$ exceeded the goal VO$_2$ (L/min) for a stage. The wattage prior to exceeding the VO2 then served as the power for all heat trials.

3.3c. Experimental Trials and Control Trial

Two experimental trials and one control trial began within 7 days after Visit 1 and 5-7 days apart from each other to ensure there was no acute heat acclimation due to the testing procedures. All subjects had Visit 1 serve as a control trial. The subsequent trials were in a randomized order and identical in design except for the independent variable of caffeine or placebo. Subjects arrived at the same time of day for each of the experimental trials, and 7-8 hours after ingesting a thermal transmitter (Mini Mitter, VitalSense, Bend, Or). Subjects were instructed to abstain from vigorous exercise for twenty-four hours, and abstain from caffeine and alcohol for forty-eight hours prior to testing. A two-day dietary and three-day physical activity recall were collected to confirm subjects meet pre-test conditions. A third-party member (with no direct interaction with the participant or the data collection procedures) prepared the experimental treatment prior to the subject’s arrival. The third party member then supplied the
investigator with the proper treatment on the day of the trial. The treatments were: water (4mL/kg body weight) for the control trials, 4mL/kg of water plus a placebo mix, or 4mL/kg of water plus a placebo mix with 5mg/kg anhydrous caffeine. Placebo mix was a single packet of Crystal Light, flavored powder. Each packet contained 10 calories. During the control trial, subjects were given 4mL/kg of plain water. Only after data collection was complete, was the treatment content and order of trials revealed to the investigator.

After administering the treatment, subjects then sat comfortably in neutral room conditions for twenty-five minutes. The subjects were then asked to void and nude weight (kg) was then collected on a weight scale (Seca, Germany) and reported by the subject. The subjects then dressed in cycling shorts and shoes. A Polar heart rate chest strap was placed around the chest at approximately the level of the xiphoid process. Subjects then entered the prepared heat chamber set to 35-37°C, 30-40% humidity, 0km/hr wind speed and sat for approximately twenty minutes to adjust to the conditions. In total, forty-five minutes passed between administration of treatment and start of exercise. This has been found to be an appropriate length of time to achieve peak plasma caffeine concentration and fully saturate the cytochrome P450 system in the liver (Graham, 2001). Baseline resting measurements were taken during this time.

The subject then began cycling at the power derived from the Detection Trial. Subjects were instructed to hold a cadence range of ±5 revolutions per minute (RPM) of their average cadence from the Detection Trial. This intensity was held until the subject reached volitional fatigue, experienced a cadence loss of more than five revolutions per minute from their required average cadence, until unsafe Tc (≥39.5°C) or signs and symptoms occurred. Subjects were provided plain water (kept in the heat chamber) ad libitum throughout the trial. Unsafe signs and symptoms of thermal stress included: pale skin, loss of visible sweating, angina, muscular pain,
dizziness, nausea, difficulty breathing, poor heart rate response, drop in systolic blood pressure (\(-10\text{mmHg}\)), or inability to respond to verbal questions.

3.4 Trial Measurements

Initial blood pressure, thermal score (TS), room conditions, THS, HR, Tc and RPE were recorded. These measurements were also be taken at 37°C Tc and then at every 0.5°C increase in Tc, and at test termination. Additionally, time was recorded at each 0.5°C increase in Tc. At ten-minute intervals respiratory gases were collected for a period of two minutes to assess metabolic conditions. Thermal score, room conditions, THS, HR, Tc, BP and RPE were collected post-gas interval as well. Total water consumption was measured by volume (mL) at exercise termination for comparison between trials. Post-trial nude weight (kg) was collected on the weight scale, after which the subjects were allowed to shower, rest and consume food and drink while pre-test Tc were reestablished.

3.5 Statistical Analysis

A Two-way repeated measures ANOVA was used to discover mean differences between independent variables (treatments, time and Tc) in respect to the dependent variables (thermal score, thirst score, water intake, body weight change, and RPE). A Two-way repeated measures ANOVA was used to determine differences in total water consumption. Scheffe’s Post Hoc test was used when statistical significance was found. Pearson Correlation was used to find correlation between experimental conditions and effect on RPE, Tc, TS, and THS. For all values a significance level of \(p<0.05\) was used.
4. RESULTS

4.1 Introduction

All nine male subjects met the inclusion criteria [30.33 ± 8.14 yrs, 75.67 ± 7kg, 179.89 ± 6.25 cm, 2.17 mg/kg daily caffeine consumption & 3.88 ± 1.76 yrs cycling experience] and completed all experimental trials. Subject characteristics are represented in Table 1. One subject experienced a malfunction in the thermal transmitter and was asked to reschedule. As a result, one experimental trial occurred eleven days after the previous trial day. This was determined to not have effect on the trial’s outcome, and the subject was allowed to continue participation. Additionally, one subject experienced an unforeseen circumstance as the ad libitum water was used as a cooling mechanism and poured onto the back. Water consumption comparison, however, was found to not be significant enough to preclude the subject for completing the remaining trials, and the subject was allowed to continue. One of the nine subjects was unable to establish a steady-state in VO$_2$ post-GXT and was asked to return on a subsequent day for the Detection Trial. Upon the return visit, Detection Trial procedures resulted in the desired outcome and experimental testing proceeded normally.
Table 1. Inclusion Characteristics from Nine Subjects.

<table>
<thead>
<tr>
<th>n</th>
<th>Age (yr)</th>
<th>BMI</th>
<th>VO2peak</th>
<th>Caffeine (mg/wk)</th>
<th>Training (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>30.33 ± 8.14</td>
<td>23.45 ± 2.72</td>
<td>4.32 ± 0.29</td>
<td>57.33 ± 5.04</td>
<td>1155 ± 521</td>
</tr>
</tbody>
</table>
4.2 Graded Exercise Test & Detection Trial

Peak values in VO$_2$ (absolute and relative), HR$_{\text{max}}$, and Power$_{\text{Max}}$ are presented in Table 2. Eight of the nine subjects completed the Detection Trial without interruption 20-30 minutes post-GXT. One subject returned two days post-GXT and performed the Detection Trial without complication. Additionally, percent VO$_2$$_{\text{peak}}$ at -5% VT1, and power (watts) at -5% VT1 are presented in Table 2.
Table 2. GXT and Detection Trial Performance and Physiologic Data.

\(HR_{\text{max}}\), \(P_{\text{max}}\), indicate heart rate maximum and peak watts during the final stage of the VO\(_2\text{peak}\) trial. \(P_{\text{trial}}\) indicates the wattage calculated at -5\% VT1 for each subject.

<table>
<thead>
<tr>
<th>Subject</th>
<th>HR(_{\text{max}}) (bpm)</th>
<th>(P_{\text{max}}) (watts)</th>
<th>VO(_2)\text{max}</th>
<th>P(_{\text{trial}}) (watts)</th>
<th>Trail % VO(_2)Peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>HS_35</td>
<td>182</td>
<td>350</td>
<td>3.96</td>
<td>50.1</td>
<td>154</td>
</tr>
<tr>
<td>HS_47</td>
<td>176</td>
<td>385</td>
<td>4.23</td>
<td>55.7</td>
<td>203</td>
</tr>
<tr>
<td>HS_51</td>
<td>192</td>
<td>385</td>
<td>4.28</td>
<td>66.9</td>
<td>169</td>
</tr>
<tr>
<td>HS_281</td>
<td>162</td>
<td>385</td>
<td>4.73</td>
<td>52</td>
<td>220</td>
</tr>
<tr>
<td>HS_102</td>
<td>184</td>
<td>385</td>
<td>4.29</td>
<td>59.9</td>
<td>238</td>
</tr>
<tr>
<td>HS_110</td>
<td>171</td>
<td>350</td>
<td>3.8</td>
<td>54.4</td>
<td>164</td>
</tr>
<tr>
<td>HS_77</td>
<td>189</td>
<td>420</td>
<td>4.48</td>
<td>57.3</td>
<td>179</td>
</tr>
<tr>
<td>HS_89</td>
<td>191</td>
<td>385</td>
<td>4.53</td>
<td>60.8</td>
<td>159</td>
</tr>
<tr>
<td>HS_111</td>
<td>189</td>
<td>420</td>
<td>4.33</td>
<td>58.5</td>
<td>238</td>
</tr>
</tbody>
</table>
4.3 Effect of Treatment on Exercise Performance

There was no significant difference found in end trial time due to an effect of treatment, p=.22. This is illustrated in Figure 1. End trial mean times were 29.66, 28.27, and 31.04 (minutes) for Control, Placebo, and Caffeine, respectively. Since this was a constant load design, there was no difference in mechanical load between trials for each subject.
Figure 1. End Trial Mean Time for all Conditions.

There was no significant effect found, p > .05. Vertical bars denote 0.95 confidence intervals.
4.4 Main Effect of Treatment

There was a Main Effect of Treatment found for core temperature versus time, p=.032. Scheffe post hoc analysis found there to be a significant difference, p=.009, between mean end placebo core temperature and mean end caffeine core temperature. Mean values and results are found in Table 3.

There was no significant difference, p>.05, found for effect of treatment on mean core temperature, and this is illustrated in Figure 3. However, analysis found there to be a significant effect of time on core temperature, p<.000, for all treatments. This indicates that significant heat stress was placed upon the subjects during all trials, yet not different enough to cause variance.
Table 3. Core Temperature Magnitude of Change Across Time.

** indicates a significant difference with placebo being different from control, p=.012.

* indicates caffeine being significantly different than placebo, p=.009, but not from control.

α=0.05 for all conditions. End trial time ranged from 17.6-46.4 minutes. Temperatures are displayed in degrees Celsius.

<table>
<thead>
<tr>
<th>Trial</th>
<th>Pre-Trial</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>End-Trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>36.85 ± 0.46</td>
<td>37.37 ± 0.48</td>
<td>38.08 ± 0.51</td>
<td>38.99 ± 0.65</td>
</tr>
<tr>
<td>Placebo</td>
<td>36.79 ± 0.39</td>
<td>37.21 ± 0.46</td>
<td>37.89 ± 0.38</td>
<td>38.54 ± 0.57**</td>
</tr>
<tr>
<td>Caffeine</td>
<td>36.86 ± 0.29</td>
<td>37.39 ± 0.19</td>
<td>38.13 ± 0.33</td>
<td>39 ± 0.53*</td>
</tr>
</tbody>
</table>
Figure 2. Comparison of Mean Core Temperature for each Condition.

There was no significant effect found, p>.05. Vertical bars denote 0.95 confidence intervals.
4.5 Effect of Treatment on Heart Rate Response

Analysis found that across all trials, heart rate was significantly different, $p<.000$, at end trial when compared to pre-test. However, there was found to be no significant difference, $p>.05$, in heart rate response as an effect of treatment. Treatment versus time analysis found there to be a trend towards significance, $p=.067$, for heart rate response. Results are displayed in Table 4. Further analysis with Scheffe post hoc analysis revealed no significant difference at any time check between the three treatments. End trial means were 169.75, 168.13, and 172.56 beats per minute for Control, Placebo, and Caffeine, respectively. The magnitude of the average heart rate increases for the three treatments are displayed in Table 4.
Table 4. Data from Heart Rate Change Across Time. No significant effect found for treatment, p=.067. * indicates significant effect of time with End being significantly larger than Pre-trial, p<.000. α=0.05 for all conditions. End trial time ranged from 17.6-46.4 minutes. Heart rates are displayed in beats per minute.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>End</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>66.7 ± 15.5</td>
<td>146.6 ± 9.76</td>
<td>157.8 ± 9.4</td>
<td>168.6 ± 8.6*</td>
<td>134.3 ± 42.5</td>
</tr>
<tr>
<td><strong>Placebo</strong></td>
<td>73.5 ± 12.4</td>
<td>151.0 ± 11.2</td>
<td>159.7 ± 9.7</td>
<td>167.2 ± 11.6*</td>
<td>137.9 ± 39.6</td>
</tr>
<tr>
<td><strong>Caffeine</strong></td>
<td>65.2 ± 14.7</td>
<td>151.5 ± 10.2</td>
<td>163.4 ± 11.0</td>
<td>172.5 ± 11.6*</td>
<td>138.2 ± 44.9</td>
</tr>
</tbody>
</table>
4.6 Perceptual Responses

There were significant differences, $p<.000$, found as an effect of time for all perceptual variables: Rate of Perceived Exertion (RPE), Thirst (TS), and Thermal Perception (THS), which are represented in Tables 5-7. Results indicate that the trial conditions were significant enough to produce the desired variability in pre and post perceptual variables for all treatments. As an effect of treatment, mean RPE had a trend towards significance, $p=.084$, favoring a reduced mean RPE in the Caffeine group, compared to Control and Placebo. Results are displayed in Figure 5. As an effect of Treatment versus Time, there was found to be no significant difference, $p>.05$, between the three conditions.

There was found to be no significant difference, $p>.05$, in Thermal Perception as an effect of treatment or upon comparison of THS for time versus treatment, $p>.05$. These results are illustrated in Table 5.

Effect of Treatment on mean TS, represented in Table 8, between treatments produced a significant difference, $p=.048$. Scheffe post hoc analysis found there to be a trend towards significance, $p=.056$, between the mean Placebo group and mean Control group. No significant difference was found as an effect of caffeine, $p>.05$. No significant difference, $p>.05$, was found for TS when comparing time versus treatment.
Table 5. Thirst Perception Data Presented Across Time. * indicates significant effect of time with End being significantly larger than Pre-trial, p<.000. ** indicates a significant effect, p=.048, with placebo mean average begin less than control. α=0.05 for all conditions. End trial time ranged from 17.6-46.4 minutes.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>End</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>2.33 ± 0.86</td>
<td>3.67 ± 1.22</td>
<td>4.63 ± 1.9</td>
<td>5.78 ± 1.92*</td>
<td>4.10 ± 1.46</td>
</tr>
<tr>
<td><strong>Placebo</strong></td>
<td>1.89 ± 0.78</td>
<td>3.22 ± 1.48</td>
<td>4.11 ± 1.69</td>
<td>5.00 ± 2.48*</td>
<td>3.56 ± 1.32**</td>
</tr>
<tr>
<td><strong>Caffeine</strong></td>
<td>2.44 ± 1.13</td>
<td>3.33 ± 1.22</td>
<td>4.00 ± 1.5</td>
<td>4.78 ± 2.38*</td>
<td>3.64 ± 0.99</td>
</tr>
</tbody>
</table>
Table 6. Thermal Perception Data Presented Across Time. * indicates significant effect of time with End being significantly larger than Pre-trial, p<.000. α=0.05 for all conditions. End trial time ranged from 17.6-46.4 minutes.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>End</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td>4.89 ± 0.41</td>
<td>6.11 ± 0.65</td>
<td>6.81 ± 0.59</td>
<td>7.78 ± 0.36*</td>
<td>6.38 ± 1.18</td>
</tr>
<tr>
<td><strong>Placebo</strong></td>
<td>4.83 ± 0.43</td>
<td>5.94 ± 0.46</td>
<td>6.94 ± 0.95</td>
<td>7.72 ± 0.36*</td>
<td>6.36 ± 1.23</td>
</tr>
<tr>
<td><strong>Caffeine</strong></td>
<td>4.78 ± 0.61</td>
<td>6.00 ± 0.43</td>
<td>6.67 ± 0.66</td>
<td>7.67 ± 0.35*</td>
<td>6.27 ± 1.17</td>
</tr>
</tbody>
</table>
Table 7. Rate of Perceived Exertion Data Presented Across Time. * indicates significant effect of time with End being significantly larger than Pre-trial, p<.000. α=0.05 for all conditions. End trial time ranged from 17.6-46.4 minutes.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>End</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6.0 ± 0</td>
<td>13.50 ± 1.45</td>
<td>15.25 ± 1.48</td>
<td>17.89 ± 1.36*</td>
<td>13.12 ± 4.64</td>
</tr>
<tr>
<td>Placebo</td>
<td>6.0 ± 0</td>
<td>13.33 ± 1.80</td>
<td>15.89 ± 1.76</td>
<td>17.89 ± 1.36*</td>
<td>13.27 ± 4.76</td>
</tr>
<tr>
<td>Caffeine</td>
<td>6.0 ± 0</td>
<td>12.78 ± 0.97</td>
<td>14.89 ± 1.05</td>
<td>17.78 ± 1.64*</td>
<td>12.86 ± 4.52</td>
</tr>
</tbody>
</table>
Figure 3. Comparison of Mean Rate of Perceived Exertion for Each Condition

There was no significant effect found, p=.084. Results indicate a trend for the caffeine group to be reduced compared to the placebo group. Vertical bars denote 0.95 confidence intervals.
4.7 Body Weight Loss and Water Consumption

There was found to be a trend towards significance, $p=0.068$, between pre and post body mass (kg) measurements. However, there was found to be no significant difference, $p=0.384$, in mean water consumption between the three treatments. Mean values are presented in Table 8.
Table 8. Comparison of Water Consumption and Body Weight Loss. No significant effect found for body weight loss between conditions, $p=.068$, or for water consumption, $p=.384$. 

$\alpha=0.05$ for all conditions.

<table>
<thead>
<tr>
<th></th>
<th>Water Consumption (ml)</th>
<th>Body Weight Loss (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>320.5 ± 234.2</td>
<td>0.97 ± 0.31</td>
</tr>
<tr>
<td>Placebo</td>
<td>273.8 ± 142.4</td>
<td>0.74 ± 0.25</td>
</tr>
<tr>
<td>Caffeine</td>
<td>318.8 ± 198.5</td>
<td>0.75 ± 0.18</td>
</tr>
</tbody>
</table>
4.8 Relationships Between Perceptions and Tc

Pearson Correlation was conducted for core temperature versus time. Analysis resulted in correlational values of $r = .834$, $r = .832$, $r = .916$ for control, placebo, and caffeine, respectively. Correlations were conducted for core temperature versus thermal perception, resulting in correlational values of $r = .872$, $r = .754$, $r = .803$ for control, placebo, and caffeine, respectively. Slope-intercept analysis showed Y-intercepts of 33.54, 34.56, and 34.15 for control, placebo, and caffeine, respectively. Correlations were conducted for core temperature versus RPE, resulting in correlation values of $r = .782$, $r = .717$, $r = .876$ for control, placebo, and caffeine, respectively. Slope-intercept analysis showed Y-intercepts of 35.77, 35.85, and 35.63 for control, placebo, and caffeine, respectively. Correlations were conducted for core temperature versus thirst perception, resulting in correlation values of $r = .698$, $r = .634$, $r = .588$ for control, placebo, and caffeine, respectively. Slope-intercept analysis showed Y-intercepts of 36.52, 36.76, 36.66 for control, placebo, and caffeine, respectively. Correlation graphs are presented for RPE, TS, and THS in Figures 4-6.
Figure 4. Rate of Perceived Exertion versus Core Temperature

Control Slope-Intercept Equation: $y=0.155x + 35.77$

Placebo Slope-Intercept Equation: $y=0.125x + 35.85$

Caffeine Slope-Intercept Equation: $y=0.168x + 35.63$
Figure 5. Thermal Perception versus Core Temperature

Control Slope-Intercept Equation: \( y = 0.667x + 33.54 \)

Placebo Slope-Intercept Equation: \( y = 0.464x + 34.56 \)

Caffeine Slope-Intercept Equation: \( y = 0.585x + 34.15 \)
Figure 6. Relationship Between Core Temperature and Thirst Perception.

Control Slope-Intercept Equation: \( y = 0.313x + 36.52 \)

Placebo Slope-Intercept Equation: \( y = 0.211x + 36.76 \)

Caffeine Slope-Intercept Equation: \( y = 0.307x + 36.66 \)
5. DISCUSSION

5.1 Introduction

The current investigation principally evaluated the influence of caffeine on subjective perceptual variables, while exercising in thermal stressful conditions. Secondarily, physiological variables were analyzed with respect to TTE to determine effect of treatments. The subjects used in the current investigation were similar in description to those in previous studies (Nybo & Nielsen, 2001; Tucker, et al., 2004). The environmental conditions (36-37°C and 30-50% humidity) used in the current investigation were thermally stressful, but did not meet the criteria for uncompensable heat stress (40°C and 60% humidity). Previous research has used caffeine in concentrations ranging from 6-9mg/kg. However, the current research used a bolus of caffeine (5mg/kg), which has been documented to have significant ergogenic effects on performance (Graham, 2001).

In the current study, end-trial core temperature in the caffeine group was the highest of the three treatments, and was significantly different than placebo. A possible reason for this difference is found in TTE differences between groups. End-trial mean times for the three groups were 29.66, 28.27, and 31.04 minutes for control placebo, and caffeine, respectively. However, given that only placebo end-trial temperature was different than control and caffeine groups, it seems likely that the increased core temperature in the caffeine and control groups stems from the increased exercise duration.
5.2 Reasoning for Increased final Tc in Caffeine Group

Mean THS was found to be statistically different in that placebo was less than control, yet not different from caffeine. And although water consumption was lowest in the placebo group, there were no statistical differences between the groups. Body weight loss had a trend for significance, \( p=.068 \), for placebo and caffeine to be lower than control. It is possible that the prolonged TTE in the caffeine group is also due to a diminished perception of THS and increased total water consumption, compared to placebo. Even in the presence of a noticeably lower body weight change compared to control, the caffeine group had a prolonged TTE. This is in agreement with the results of Del Coso and Roti (Del Coso, et al., 2008; Roti, et al., 2006). Subjects in the caffeine group may have experienced increased task-motivation, and thus tolerated more extreme physiological stress. In the current investigation, increased task-motivation is supported by a trend for significance, \( p=.084 \), in RPE begin lowest in the caffeinated group. Furthermore, at all core temperatures, RPE in the caffeinated group is depressed in linear relationship compared to placebo. Perceptual score of TS and THS were also found to hold this relationship. Despite no significant differences being established, \( p=.79 \), for TS and THS, TS was found to display this relationship at \( T_c > 38^\circ C \) (as displayed in Figure 10, 11). It is possible that using a multiple regression model to analyze the data that significant differences in slope and relationships could be found between groups. These relationships should be examined in future research.

The current investigation found HR to have a trend towards significance, \( p=.067 \), for caffeine to have increased HR over time compared to placebo and control. Mean
End-trial HR for the caffeine group (172.56 bpm) was 94.9% of the mean maximum HR obtained from the GXT. This indicates a extreme cardiovascular response during the experimental trials, as a result of testing conditions and possibly CNS stimulation by caffeine. Control and placebo end-trial HR means were 93.1% and 92.2% of GXT peak HR, respectively. Although expired gases analysis was not conducted for this study, and, therefore, metabolic indices cannot be studied, the high HR and high Tc are further evidence of exertional exercise thermal stress. It is likely that caffeine not only acted as a cardiovascular stimulant, but also reduced the perceived effect of such physical stress during exercise as indicated by the RPE data.

5.3 Caffeine’s Influence on Heat Illness Potential

Results from the present study do not support the hypothesis that, using constant-load exercise, all caffeinated athletes will continue voluntary exercise up to and past the point of 40°C core temperature. Seven out of nine caffeinated subjects voluntarily ceased exercise prior to our ethical cut-off of 39.5°C with exercise cessation occurring due to drop in cadence or reaching the point or volitional fatigue. Therefore, within the limits of this study, it does not appear that ingesting a moderate dose of caffeine (5mg/kg) will augment VA significantly enough to counteract EICF. Thus, it may be possible for athletes to avoid the attainment of heat stroke temperature when exercising in the heat even after a moderate dose of caffeine. Yet, it is note-worthy that two caffeinated subjects reached 39.5°C and expressed no desire to discontinue exercise, further stating a desire to have continued. Therefore, it is possible that caffeine’s ergogenic properties affect elite level cyclists in a manner that may lead to heat illness. This potential should
be investigated in the future with an emphasis on heat tolerant cyclists with known caffeine sensitivity.

5.4 Recommendations for Future Research

It was found within this investigation that subjective thermal stress perception was not significantly different, as an effect of treatment, at end trial or during previous data collection intervals. This finding, while in agreement with previous research (Roti, et al., 2006; Ely, Ely, & Cheuvront, 2011), may be flawed due to the data tools and analysis of data used in the current investigation. Future researchers should use data analysis techniques that limit the amount of noise due to the variability of resting measurements. To that effect future researchers may wish to investigate the change in resting values (HR, TTE, and Tc) to end-trial measurements. The change determined between these values and within each group would reduce the internal variability and account for the large standard deviations seen in the present data.

An additional limitation of the current study was the use of constant-load intensity. As seen in previous research, when performing a self-paced time trial in a high thermal environment, caffeine has a positive effect on performance (Ganio, et al., 2011). It is possible that with caffeine, exercise time and core temperature tolerance may have been increased, beyond the current investigation, if subjects were allowed to self-pace to a set distance or time trial. A difference between this study and that of Ganio was Ganio used 22°C water to replenish sweat loss, whereas, this study used warmed water, ad libtum.
A possible reason for the lack of significance for RPE and TS may be the limited sample size, resulting in a lower power for the study. Given the trends for significance found with RPE and HR, it is reasonable that a larger sample size may yield significant results. Also a limiting factor was the wide range of TTE for the experimental trials. Trial termination occurred for multiple reasons, including ethical trial termination and subject volitional fatigue. Although the present study screened subjects for cardiovascular and training experience, it is advised that future studies look to enroll participants with similar expected heat tolerances and similar expected TTE.

5.5 Conclusion

As demonstrated in this study, ingestion of caffeine (5mg/kg) was found to significantly elevate end-trial Tc, significantly depress THS, and result in depression of perceptual variables (RPE and TS) in trained cyclists exercising in thermal hot, compensable conditions to exhaustion.

These results are applicable only to moderately trained cyclists of 3-4 years of experience, and who have a moderate daily caffeine intake. How the current study’s experimental conditions would influence elite level cyclists remains unknown. Highly trained athletes have shown the capacity to tolerate higher Tc than was found in the current investigation. Future investigation into heat illness potential using elite level cyclists should be conducted.
Appendix A

Informed Consent

Research investigator (California State University, Sacramento) Dr. Roberto Quintana, Ph.D. and Max Polin, B.S. invite you to participate in a research study to understand caffeine and its influence on performance in high heat conditions. We are interested in the effects of caffeine ingestion on athletic performance in hot conditions (100°F and 60% humidity). Caffeine has been associated with increased tolerance of exercise in thermally challenging conditions. You were selected as a possible participant for this study because of your current health status, competitive running status, and your interest in participating in this research.

Explanation of the Treatments and Tests to Be Administered

If you decide to participate, we will require you to report to the Human Performance Research Laboratory (HPRL) on four separate occasions over the course of sixteen days. Each visit will be approximately 1-2 hours in duration. The total time commitment to this study is approximately ten hours. The procedures involved are explained as follows:

A. You will report to the HPRL to fill out a medical history questionnaire, 3-day physical activity recall, a 48 hour dietary recall, and have vital signs measured to determine whether you are suitable for enrollment in the study. Also during this time you will be able to become familiar with the procedures of the study and equipment that will be used. During this visit, if you are determined to be suitable for the study, you will perform a maximal graded exercise test (GXT) on a cyclergometer. This test is designed to be progressive and elicit maximal exercise capacity within 12-15 minutes of exercise.

Twenty minutes post GXT, you will perform a cycling test designed to determine power associated with 5% prior to your first ventilation threshold (VT1).
B. You will then return within 2-7 days and perform the first of three experimental trials in a heated chamber (100°F and 60% humidity). You will receive either the treatment (5mg/kg body weight of caffeine in a flavored water solution), or a placebo, or no treatment (control). Prior to the exercise testing, a core temperature pill will be ingested. The temperature capsule will allow the investigators to safely monitor your thermal stress response. The temperature capsule will eventually be passed through the body by a normal bowel movement.

All testing will occur in the HPRL. Nude weight will be measured both pre and post trial. The exercise test is a cycling trial to volitional fatigue (exhaustion) under hot, humid conditions (100°F and 60% humidity) at the power corresponding to -5% VT1 VO2. Your heart rate (HR) and core temperature will be measured continuously during the test. RPE, core temperature, thermal score, and thirst score will be recorded periodically. You will also be allowed to drink water at all times during the testing. At the end of the trial, your nude weight will be measured again. All nude weights will be measured in a private location.

This series of testing will be repeated every 5-7 days and you will receive a different treatment.

C. The total time commitment for the study will be approximately ten hours over sixteen days.

Risks From Procedures

Exercise tests to the point of fatigue are associated with a risk of death (<0.01%) and complications with the heart (<0.1%) (i.e irregular heart rhythm, inadequate blood to the heart, and heart attack). The risk of incidents occurring is much less for individuals who are young, exercise regularly, and are in good health. Completion of the subject history and subject medical history questionnaire prior to the beginning the study will help minimize the risks of any cardiac event. Also associated with an exercise test of this nature are leg and breathing discomfort (100%), as well as increases in body temperature (100%). The caffeine dosing you will receive is
based upon your body weight, with the experimental dose amount being 5 milligrams (mg) of caffeine per kilogram (kg) of body weight. For example: a person weighing 60kg (132lbs) will receive a caffeine dose of 300mg. This is the equivalent of 2.5 cups of caffeinated coffee. A 100kg person will receive a caffeine dose of 500mg. Caffeine in the amounts given is generally safe for adults without a history of adverse reactions to caffeine. There is increased risk of adverse reactions with caffeine consumption if you have had an ulcer previously with caffeine or a history of ulcers. Participation in this study if you have had an ulcer previously will be entirely at the discretion of the lead researcher, Dr. Roberto Quintana, Ph.D.

Your core temperature will be monitored continuously during the trials to avoid a core temperature in excess of 40°C. Water will be provided to you during exercise to help control your core temperature. Additionally, your thermal stress perception, RPE and thirst score will be monitored. Upon cessation of the trials, you will be removed from the heat chamber and allowed to drink fluids or enter a cooled water tank. The investigator will monitor your physiological values until they have returned to normal. If any adverse reactions occur due to exercise testing, you will be referred to your personal physician or the CSUS Student Health Center if you are a student. In case of severe or acute signs and symptoms, we will follow CSUS Guidelines for Emergencies and when necessary Adult CPR/automated external defibrillator procedures.

Benefits of Participation

The benefits to yourself for participating in this study include the difference in your maximal ability to exercise under hot conditions with under normal conditions and with caffeine treatment. This information can be used to help optimize your training and understand your body’s response to exercise in the heat.

Your Rights and Confidentiality
If you decide to participate, you are free to withdraw your consent and to stop participation at any time with no penalty to you. Any information which is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. The data will be identified only with numeric codes, not the names of the participants.

Questions

If you have any questions, please feel free to call XXXXXXXX between 9am and 5pm, or XXXXXXXXXXX anytime.

Statement of Permission

You are making a decision whether to participate or not participate. Your signature indicates that you have decided to participate having read the information provided. Your signature also affirms that the medical history you have provided is complete and true to the best of your knowledge. You will be given a copy of this form to keep. You understand that you will not receive any compensation for participating in this study.

_________________________________  ______________________________
Date                                  Signature of Participant

_________________________________  ______________________________
Date                                  Signature of Investigator
Appendix B

Sac State Human Performance Research Laboratory Subject

Information and Medical History

NAME:_______________________________________________________
DATE_______________________________
ADDRESS:____________________________________________________
PHONE:_____________________________
EMAIL:______________________________
OCCUPATION:_________________________________________________________________
GENDER:  M__  F___  AGE_______yrs  DATE OF  BIRTH_____________________
TOTAL CHOLESTEROL___________mg/dL  HDL_______ mg/dL  LDL________mg/dL  TG___________mg/dL
FASTING BLOOD GLUCOSE _________________mg/dL  Other blood results:____________________

We will take the following 4 measurements (do not answer):
WEIGHT__________kg  HEIGHT_________cm  BP____/____mmHg
HR_________beats/min

MEDICAL HISTORY: (Please Circle your Answer/s)
Are you currently taking any medications: Yes or No:
If yes, please list:___________________________________________________________________________
Please list all medical conditions (e.g. ulcers, arthritis, mono, hepatitis, HIV, musculoskeletal injury)?_____________________
_______________________________________________________________________________________

Please list any hospitalizations and/or surgeries?

Have you ever been diagnosed with a breathing problem such as asthma? Yes or No:
If yes, please explain:________________________________________________________________________

Have you ever been diagnosed with a heart problem or condition? Yes or No:
If yes, please explain:________________________________________________________________________

Do you have any of the following symptoms at rest or with low to moderate physical activity?
Yes or No:

- Lightheadedness
- Shortness of Breath
- Chest Pain
- Numbness
- Fatigue
- Coughing
- Wheezing
- Other__________________

If yes, please explain:________________________________________________________________________

Do you have any of the following cardiovascular disease risk factors? Yes or No

- Family History of Heart Attacks
- Hypertension
- High Cholesterol
- Sedentary Lifestyle (refer to next page)
- Diabetes
- Current cigarette smoker
- Obesity (Calculate BMI=_______kg/m²)

If yes, please explain:________________________________________________________________________

Do you have an immediate family member with any of the following diseases? Yes or No

- Diabetes
- Hypertension
- High Cholesterol
- Obesity

If yes, please explain:________________________________________________________________________

Are there any other conditions that might affect your health/exercise ability? Yes or No:

If yes, please explain:________________________________________________________________________

**Training History**

What type of athlete are you? Please circle the best answer:

A) Professional-National class  B) Competitive at Regional-Local level  C) Age or Class Competitor  D) Well Trained  E) Other:___________________________________________________________

How many years have you been training competitively?________________________________________

Over the last year, what has been your weekly mileage?_______________________________________
Over the last year, what percentage of your overall training is at a pace faster than “somewhat hard” or \( \geq 70\% \) of VO2max?

What are your 3 best performances and include date and event/course?
1:
2:
3:

Please give your best performance over the last 18 months include date, time and course?

These questions concern your training over the 20 weeks:
What is the average number of exercise sessions per week?
What is the average duration of your exercise sessions?
What is the average intensity of your exercise bouts?

Could you give us the respective volume of easy, moderate (= “somewhat hard” or 70\% VO2max) and hard workouts (= “Hard” or 85\% VO2max) per week (miles per week)?

What is the total volume of your workouts per week (miles per week)?

Any recent significant injuries which have limited your training?

Caffeine History:
Please list the amount and type of caffeinated beverages you consume per day (previous 3 months):

________________________________________________________________________
Have you had any adverse reactions to caffeine ingestion (loss of focus/motivation, dizziness, nausea, digestive distress, ulcers, other) in the previous 3 months?

______________________________________________________________________________
______________________________________________________________________________

Additional Information:
How have you ever performed a fitness or maximal exercise test? Yes or No:
If yes, what were the results of your tests? Protocol VO2
max
Speed/Power Lactate Threshold
Overall
Interpretation:

COMMENTS & OBSERVATIONS:

OVERALL RISK STRATIFICATION:

EXERCISE & EXERCISE TEST RECOMMENDATIONS:

APPROVED BY: Dr. Roberto Quintana, Ph.D.

_________________________ Max Polin
References


