The effects of a heat acclimation protocol in persons with spinal cord injury

Trbovich, MB, Kiratli, JB & Price, MJ

Original citation & hyperlink:
Trbovich, MB, Kiratli, JB & Price, MJ 2016, 'The effects of a heat acclimation protocol in persons with spinal cord injury' Journal of Thermal Biology, vol 62, no. A, pp. 56-62.
DOI: 10.1016/j.jtherbio.2016.10.006
https://dx.doi.org/10.1016/j.jtherbio.2016.10.006

DOI 10.1016/j.jtherbio.2016.10.006
ISSN 0306-4565

Publisher: Elsevier

Manuscript Details

Manuscript number	TB_2016_126
Title	The effects of a heat acclimation protocol in persons with spinal cord injury
Article type	Full Length Article

Abstract

Persons without spinal cord injury (SCI) physiologically acclimate between seven to fourteen consecutive days of exercise in the heat. Decreased resting and exercise core temperature, decreased heart rate, increased plasma volume and increased thermal comfort during exercise are changes consistent with heat acclimation. Autonomic dysfunction after SCI impairs heat dissipation through sweating and vasodilation. The purpose of this study is to determine if seven consecutive days of exercise in the heat would result in physiologic changes consistent with heat acclimation in persons with SCI. Ten persons with SCI divided into two groups: tetraplegia (n=5) and paraplegia (n=5) exercised in 35°C using an arm ergometer at 50% Wpeak for 30 minutes followed by 15 minutes rest. This protocol was repeated over seven consecutive days. Heart rate (HR), skin temperature, aural temperature (Taur), rate of perceived exertion (RPE), rate of perceived thermal strain (RPTS), and plasma volume (PV) were measured throughout the protocol. There were no significant differences in resting Taur exercise Taur, mean skin temperature, HR, PV, RPE or RPTS over the 7 days for either the tetraplegic or paraplegic group. Participants with SCI did not demonstrate the ability to dissipate heat more efficiently over 7 days of exercise at 35°C.

Keywords	thermoregulatory dysfunction; heat acclimation; spinal cord injury; thermal strain

Corresponding Author	Michelle Trbovich
Corresponding Author's Institution	UT Health Science Center at San Antonio
Order of Authors	Michelle Trbovich, Jenny Kiratli, Mike Price
Suggested reviewers	Christopher Minson, jill Wecht, Katy griggs, John Handrakis

Submission Files Included in this PDF

File Name [File Type]

thermal biology HA cover letter.docx [Cover Letter]

HA response to reviewers rev 2.docx [Response to reviewers]

HA REVISED manuscript 9.19.16.docx [Manuscript]

Figure 1.tiff [Figure]

Figure 2.tiff [Figure]

Figure 3.tiff [Figure]

Figure 4.tiff [Figure]

michelle trbovich.tiff [Figure]

jenny kiratli.tiff [Figure]

mike price.tiff [Figure]

Table 1.docx [Table]

Table 2.docx [Table]

annotated HA manuscript 9.19.16.docx [Editor-Annotated Manuscript]

Highlights thermal biology HA mt ed 7.19.16.docx [Highlights]

Title Page

Title: The effects of a heat acclimation protocol in persons with spinal cord injury

Author names and affiliations:

Michelle B. Trbovich[1,2,3], Jenny B. Kiratli[1,2], Mike J. Price[1,2,4]

[1]Veterans Affairs Palo Alto Health Care System 3801 Miranda Ave, Palo Alto, CA, USA 94304

[2]Stanford University, Dept of Biology 450 Serra Mall, Stanford, CA, USA 94305

Corresponding Author

Name**:** Michelle Trbovich M.D.

Address: 7703 Floyd Curl Drive, San Antonio, TX 78229

Telephone Number: 210-818-4353

Fax Number: 210-949-3327

Email: michelle.trbovich@va.gov

Present addresses

[3] UT Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229

[4] Coventry University, Priory Street, Coventry, UK CV1 5FB

Response to Reviewers – Revision 2

Minor revisions

1. How do potential sensory deficits, which may be different between the two SCI groups, factor into rate of perceived thermal strain?

Response: We have added to this section to try and discuss reason why RPTS may change in the PP but not the TP. Research in this area is lacking and we have tried to integrate previous studies relating RPE and RPTS to changes in exercise intensity to reduce heat storage. We also suggest that previous experiences of thermal imbalance in the TP may, in conjunction with consistent greater Taur, may override any potential adaptations to RPTS.

2. Care to comment on what it means that the population may not acclimate or adapt to this type of thermal stress?

Response: on the 2nd page of the introduction we proposed a few benefits of heat acclimation that logically would not occur if heat acclimation did not occur. If heat acclimation occurred, it could 1. Help instruct SCI athletes on training protocols for competitions in the heat and 2. May improve the ability of non-athletic SCI persons to undertake activities in the heat without risk of heat related illness and thus improve their quality of life.

We have added more discussion about the consequences of lack of acclimation in the Conclusion section

3. The verbiage associated with heat sink just needs a few small clarifications. A heat sink, in theory, should be able to store more heat prior to needing to dissipate the generated or acquired thermal load.

Response: We have reworded this section for clarity.

4. Section 2.2, last sentence of the page: “which would not have any effect…” consider softening this to something like “which would likely affect...”

Response: This has been changed.

5. Section 4.4, 6th line: Plasma volume itself is not normally assessed by Hct and hemoglobin; rather it is the “change in plasma volume” that can be estimated.

Response: This has been rewritten

The effects of a heat acclimation protocol in persons with spinal cord injury

Abstract

Persons without spinal cord injury (SCI) physiologically acclimate between seven to fourteen consecutive days of exercise in the heat. Decreased resting and exercise core temperature, decreased heart rate, increased plasma volume and increased thermal comfort during exercise are changes consistent with heat acclimation. Autonomic dysfunction after SCI impairs heat dissipation through sweating and vasodilation. The purpose of this study is to determine if seven consecutive days of exercise in the heat would result in physiologic changes consistent with heat acclimation in persons with SCI. Ten persons with SCI divided into two groups: tetraplegia (n=5) and paraplegia (n=5) exercised in 35°C using an arm ergometer at 50% W_{peak} for 30 minutes followed by 15 minutes rest. This protocol was repeated over seven consecutive days. Heart rate (HR), skin temperature, aural temperature (T_{aur}), rate of perceived exertion (RPE), rate of perceived thermal strain (RPTS), and plasma volume (PV) were measured throughout the protocol. There were no significant differences in resting T_{aur} exercise T_{aur}, mean skin temperature, HR, PV, RPE or RPTS over the 7 days for either the tetraplegic or paraplegic group. Participants with SCI did not demonstrate the ability to dissipate heat more efficiently over 7 days of exercise at 35°C.

Key words: thermoregulatory dysfunction; heat acclimation; spinal cord injury; thermal strain

---

Abbreviations: AIS= American Spinal Injury Association Impairment Scale, PP = paraplegia, SCI = spinal cord injury, T_{aur} = aural temperature, T_{skin} = skin temperature, TP = tetraplegia, W_{peak} = peak power output.

1. Introduction

Heat acclimation in able-bodied persons results in improved sweating and cutaneous blood flow at a given core temperature to improve heat dissipation.[1] Heat acclimation therefore results in reduced physiological strain as evidenced by lowered resting core and both reduced core temperature and heart rate (HR) during exercise. [2,3] Furthermore, fluid losses from sweating lead to increased fluid regulatory hormones that raises plasma volume by 25% from minutes to hours after exercise in the heat.[3] These physiologic responses improve thermal comfort, endurance and performance in the heat. [4,5]

Although heat acclimation occurs in able-bodied individuals, spinal cord injury (SCI) results in reduced sweat rates and/or impaired cutaneous vasodilation below the lesion, which may prevent effective heat acclimation. [6,7] Studies in SCI show that both at rest and during exercise, the effectiveness of thermoregulation varies according to residual sympathetic function and the extent to which thermoregulatory reflexes are compromised. [8-10] Due to compromised vasomotor and sudomotor activity below the level of injury, such persons have impaired ability to regulate core temperature during heat exposure.[9,11] Specifically, tetraplegics demonstrate a complete absence of, or significant reduction in, sweating (depending on the completeness of injury) whereas those with paraplegia demonstrate a sweating response that is proportional to the lesion level.[6,11,12] During prolonged exercise in cool conditions (25°C), paraplegic athletes demonstrate whole body sweat losses similar to those for matched able-bodied athletes.[13-15] This suggests that sweating is increased within the smaller sensate surface area so that paraplegic athletes can maintain thermal balance in cool conditions.[9] In contrast, during exercise in cool conditions (25°C), tetraplegic athletes demonstrate greater and continual increases in core temperature when compared to paraplegic athletes, a response accentuated during exercise in hot conditions.[11,13,14,16] Few studies have examined thermoregulatory responses to exercise in hot conditions of SCI persons. In preparation for the upcoming Paralympics in Brazil, a recent review article stated that the lack of knowledge of heat acclimation in SCI athletes makes competition/training recommendations for this population difficult. [17] Furthermore, non-athletic SCI persons often limit or avoid activities involving heat exposure to prevent overheating and the associated discomfort. Effective heat acclimation could not only improve performance in SCI athletes but may also improve the ability of non-athletic SCI persons to undertake activities in the heat and thus improve their quality of life. The aim of this study was to examine thermoregulatory responses of SCI persons during heat acclimation. We hypothesized that persons with tetraplegia would not demonstrate heat acclimation whereas those with paraplegia would demonstrate some degree of heat acclimation responses proportional to their level of injury and sweating capacity.

2. Material and Methods

2.1 Setting

Data collection occurred in an exercise lab and an environmental controlled chamber. All study procedures were approved by the local institutional review board.

2.2 Participants

Participants were recruited from a Veterans Affairs clinic and were included if they utilized a manual wheelchair, could propel an upper extremity ergometer, and carried a diagnosis of chronic (>1 year) SCI. Participants were excluded by a history of heat-related illness, cardiovascular disease, were pregnant (self-report) or had an acute illness. Ten persons volunteered and were divided into two groups: tetraplegia (TP) (n=5) and paraplegia (PP) (n=5). Body mass index (BMI) was measured for each participant. SCI classification and neurological exam from the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) exam was obtained from the medical record. The significant differences between groups were time since injury, which would not likely have any effect on thermoregulatory function; and peak power output that is expected to be less in the TP group given the neurologically impaired arm muscles in this group. (Table 1) All medications of participants are recorded in Table 2. Participants were recreationally active but not highly trained. None had undertaken prior heat acclimation training. All refrained from caffeine and alcohol in the 24 hours prior to visits and fasted for two hours prior to each visit.

Table 1.

Table 2.

2.3 Preliminary tests

An exercise test was conducted to establish individual baseline values for peak power output (W_{peak}). The protocol began with exercise on an arm crank ergometer All tests were undertaken on a specific arm crank ergometer (Ergoline, Ergoselect 400) at 5 watts for 2 minutes (min) with increases of 5 watts every 2 min at a cadence of 60 revolutions per min until volitional exhaustion. W_{peak} of all participants are reported in Table 1.

2.4 Heat acclimation protocol

2-3 days after preliminary testing, participants began the seven consecutive day heat acclimation protocol. Each day, participants arrived at the laboratory having followed the same pre-exercise conditions noted earlier. In addition, participants were asked to arrive euhydrated. Although hydration was not specifically measured participants were provided with fluids following every exercise sessions and were advised to continue drinking throughout the remainder of the day, prior to their next visit. Participants exercised continuously at 50% W_{peak} for 30 min (see Table 1 for HA exercise work rates of each participant) followed by 30 min of passive recovery. Each exercise session was scheduled at the same time of day (± 1 hour) to avoid circadian variations. [18] On arrival at the laboratory participants rested for 15 min in an air-conditioned environment (22.4 ±1.1°C, <40% relative humidity) while thermistors were attached. Aural temperature (T_{aur}) was measured from an aural thermistor inserted into the ear canal and insulated with cotton wool. This method is a reliable indicator for core temperature measurement in SCI persons in thermoregulatory studies. [11,13,14,19,20] Skin temperatures (T_{skin}) were measured at standard anatomical landmarks at the forehead, upper arm, chest, thigh and calf using thermistors taped to the skin. Mean T_{skin} was calculated using the latter four measures according to Ramanathan.[21] HR was continually monitored using a Polar monitor. Rating of perceived exertion (RPE) was determined from a 15-point scale ("no exertion" to "maximum exertion") throghout exercise. Rate of perceived thermal strain (RPTS) was recorded from a 9-point (0-8) Likert scale at baseline and during exercise.[22,23]

After thermistors were applied as above, participants rested for 15 min in cool conditions to enable T_{aur} and T_{skin} to equilibrate. After 15 min, baseline T_{aur}, T_{skin}, HR, and RPTS were recorded. Participants then entered the environmental chamber set at 35°C, ~40% relative humidity. After 5 min of rest, T_{aur}, T_{skin}, HR, and RPTS were documented. Participants then began the 30 min exercise protocol. $T_{aur,}$ T_{skin}, and HR measurements were continuously monitored by the data logger, and recorded every 5 min by an investigator. RPE and RPTS were recorded at 5, 15 and 30 min. On day-1 and day-7, venous blood samples (10 ml) were drawn at 0 and 30 min for measurement of hemoglobin concentration and hematocrit (Unicell DXH800 Coulter Cellular Analysis System) to assess changes in plasma volume (PV) during exercise.[24] In addition, earlobe capillary blood samples (20 μl) were also obtained on day-1 and day-7 to measure blood lactate concentration (BLa; Lactate Pro, Arkay, Japan) at rest, 5, 15 and 30 min. After 30 min of exercise, participants rested for 15 min prior to exiting the chamber. Participants were monitored by a physician throughout and after each exercise session for signs of heat-related illness. Participants were given the opportunity to leave the chamber voluntarily if uncomfortable or if T_{aur} increased by more than 2°C above baseline; however, none chose to do so.

2.5. Statistical analysis

Data are presented as mean ±SD. Data were analyzed using SPSS, v17. Normality of data was checked using the Kolmogorov-Smirnov test and by visual inspection of the normality plots. Differences in variables over time within a session (i.e., a Within-Session Time factor, with 2 levels, the initial resting temperature at 0 min and the terminal temperature at 30 min) and over days (i.e., a Days factor, with two levels, day-1 and day-7) were analyzed separately for TP and PP groups using 2-way ANOVA with repeated measures on both factors (Within-Session Time and Days). In addition, a mixed-model ANOVA was conducted which included the between-subject Group factor (with two levels, TP and PP) as well as the within subject Within-Session Time and Days factors. For all tests, the alpha level used to determine significance was set to $p<0.05$. Another mixed-model ANOVA was used to assess the resting T_{aur} over the seven days, assessing Groups (between-subject) and Days (within-subject factor with, in this case, 7 levels). For all within-subject factors, Greenhouse-Geisser corrections were applied as appropriate. To assess differences between two means, t-tests for related measures or t-tests for independent measures were used. As SPSS provides actual probability values, these are reported for each comparison; however, for highly significant results, values are reported as $P<0.001$. The delta T_{aur} (ΔT_{aur}=30-minT_{aur}-0-minT_{aur}) was analyzed using one-way ANOVA for both groups. Changes in PV between day-1 and day-7 were analyzed using paired t-tests. Differences between T_{aur} and T_{skin} at rest between TP and PP groups were analyzed using independent t-tests.

3. Results

Aural temperature for both groups on day-1 and day-7 of the heat acclimation protocol are shown in Figure 1. No significant interaction was observed between Days (day-1 and day-7) and Within-Session Time (0 to 30-min in 5-min intervals) for T_{aur} in either the TP ($P=0.948$) or the PP group ($P=0.576$). Main effects for Within-Session Time were observed in both groups ($P<0.001$). The mixed-model ANOVA also yielded a significant main effect due to Within-Session Time ($P<0.001$) and a significant interaction between Groups and Within-Session Time ($P<0.001$), as T_{aur} of the TP group began at a lower T_{aur}, rose faster and ended at a greater temperature than the PP group. (Figure 1) No other main effects or interactions were significant (Fs< 1.0).

The absolute rise/change in T_{aur} (ΔT_{aur}) from 0 to 30 min of exercise was not significantly different from day-1 to day-7 in the TP (1.6±0.4 vs. 1.6±0.3°C, $P=0.98$) or PP group (0.9±0.1 vs. 0.8±0.3 °C, $P=0.57$) (Figure 2). There was, however, a significant difference in T_{aur} averaged over all 7 days between TP and PP groups; 1.6±0.30°C vs. 0.8±0.10°C, respectively; $P=0.003$. Furthermore, there was no interaction between Group and Days (F< 1.0), meaning that the difference in T_{aur} between groups did not change over the week.

Figure 1

Figure 2

There was no significant difference in resting T_{aur} (i.e.;T_{aur} at 0min) between day-1 and day-7 in either the TP (36.1±0.6 vs 36.2±0.6°C, P=0.85) or PP group (36.6 ±0.5 vs. 36.7±0.3°C, P=0.66). (Figure 3) Incidentally, the TP group showed significantly cooler resting T_{aur} than the PP group throughout the seven days (35.9±0.4 vs. 36.5±0.3°C, respectively; P<0.001), but there was no main effect for Days and no interaction between Group and Days (Fs< 1,0).

Figure 3

The mean T_{skin} responses for both groups are shown in Figure 4. Similar to T_{aur}, there were no differences between day-1 and day-7 for T_{skin} in either group (TP: P=0.991; PP: P=0.999). However, when considering the individual skin sites for the PP group, forehead T_{skin} demonstrated a significant main effect for day (P=0.017) with day-7 eliciting cooler temperature than day-1. Similarly, the TP group demonstrated a significant main effect between day-1 and day-7 for calf T_{skin} (P=0.043) with warmer temperatures on day-7. No other differences were observed between days for the individual T_{skin} sites.

Figure 4

HR for the PP group on day-1 and day-7 at 30 min of exercise were 126±29 and 123 ±32 beats.min^{-1} (P>0.05), while those of the TP group were 94±15 and 95±11 beats.min^{-1}

respectively, ($P>0.05$). Both groups demonstrated a main effect for RPE between days with lower values on day-7 when compared to day-1 (15.4 vs. 13.8 for the TP group, $P=0.041$; 14.2 vs. 13.0 for the PP group, $P=0.038$). The PP group demonstrated a main effect between days for RPTS (5.5 ±0.9 vs. 4.6 ±0.5 at 30 min for day-1 and day-7, respectively; $P=0.003$) whereas TP did not (5.8 ±1.1 vs. 5.8 ±0.8 at 30 min for day-1 and day-7, respectively; $P=0.133$). After 30 min, BLa concentration on day-1 versus day-7 in the TP group was 2.3±0.3 vs. 2.2±1.2 $mmol.l^{-1}$, respectively and for the PP group, 3.3±1.9 vs. 3.4±1.7 $mmol.l^{-1}$ with no significant differences in either group. Neither were there differences in the change in PV between day-1 (-0.56 ±1.97; -0.56 ±2.47) and day-7 (-1.02 ±1.91; -0.74 ±0.51) in either the TP or PP groups, respectively ($P=0.967$).

4. Discussion

We hypothesized that impaired vasomotor and sudomotor function would preclude persons with tetraplegia from acclimating to the heat and that persons with paraplegia would acclimate relative to their level of injury. Physiologic changes consistent with heat acclimation consist of decreased core temperature and HR at a given a exercise intensity, decreased resting core temperature and HR, increased whole body sweat rate, earlier onset of sweating and decreased RPTS. [25,26] Our hypothesis was rejected as neither TP nor PP group, with the exception of lowered RPTS, acclimated to the heat based on these criteria.

4.1 Aural temperature during exercise

The PP and TP groups demonstrated the expected themoregulatory responses for exercise in the heat, those being a gradual increase in T_{aur} for the PP group and a significantly larger increase for the TP groups.[11] Furthermore, increases in T_{aur} were similar to those previously reported for athletes with tetraplegia and high and low levels of paraplegia (2.1,1.1 and 1.4°C, respectively) after 30 min of exercise at a greater intensity (60% W_{peak}) but cooler environmental temperature (31.5°C) [11] so can thus be deemed comparable.

Despite finding the expected differences in ΔT_{aur} *between* TP and PP groups, there was no significant difference in ΔT_{aur} on day-1 and day-7 *within* each group; and ΔT_{aur} did not vary over days within either group. This is reminiscent of Gass et al., who observed no acclimation after 60 min using (resting) warm water immersion (39°C) over five consecutive days in paraplegic men. [27] In contrast, one small study (n=5) of predominately trained PP athletes reported a *partial* heat acclimation response evidenced by decreased resting $T_{aur,}$ decreased T_{aur} (during recovery), HR, RPE and RPTS during each heat acclimation session without changes in sweat rate.[28] Castle et al. utilized mostly (4 of 5) persons with paraplegia who typically have greater vasomotor and sudomotor activity than tetraplegic persons. In addition, participants were trained, so may have undergone some degree of prior acclimation. These facts should not discount their finding of partial heat acclimation; however, it should be clarified that these findings cannot represent the physiology of either the larger untrained SCI population, or those with tetraplegia. To our knowledge, our study is the first to examine heat acclimation in equal groups of untrained persons with tetraplegia (n=5) and paraplegia (n=5) using a traditional heat acclimation protocol.

4.2 Resting aural temperature

The adaptive response of decreased resting core temperature from heat acclimation creates a greater heat storage capacity in anticipation of heat strain so the risk of heat-related injury from hyperthermia is reduced. [29] Neither SCI group demonstrated significant changes in the resting T_{aur} over the seven days of exercise in the heat. However, while resting T_{aur} for the PP group (36.5 ±0.3°C) fell in the range of expected values for able-bodied individuals, the TP group demonstrated significantly cooler resting T_{aur} values (35.9 ±0.4; $P<0.001$).[11,13,14,19] Such subnormal resting core temperature has been observed in up to 66% of persons with SCI.[30] Cooler resting core temperature suggests either lower heat production or increased heat loss. As the drive for heat loss at rest in cool conditions in the current study would be small and would not elicit significant heat loss to the environment, it is likely that lower resting T_{aur} in the TP group results from decreased resting metabolic rate, as previously demonstrated. [31]

The cooler lower body of persons with SCI has been suggested to be a potential site for heat storage[9] and potentially preventing much larger increases in body temperature than may otherwise be expected in the presence of thermal dysfunction. The cooler lower body may therefore act as a *partial* heat sink as thermoregulatory effectors, if present, are concurrently active alongside heat storage occurring, rather than the heat sink activity occurring prior to active thermoregulation. As evidenced by the more 'steady state' T_{aur} response for the PP group than the TP group during exercise any such heat sink properties are more effective when some sudomotor and vasomotor abilities are present. Where these effectors are severely compromised, such as for the TP group, the benefits of such affects may be rendered ineffectual as T_{aur} continues to increase and remain elevated post exercise.[11]

4.3 Skin temperature

Although there were no differences in mean T_{skin} between day-1 and day-7, the calculation of mean T_{skin} includes the arm, chest, thigh and calf skin temperature sites and thus has potential to mask local skin site adaptations, as previously observed.[13] Notable differences in local skin sites are further discussed. For example, forehead T_{skin} of the PP group was cooler on day-7 when compared to day-1. As all PP participants could sweat in this area, it is likely that improved evaporative cooling occurred at the forehead by day-7 when compared to day-1. Increased forehead sweat rates during upper body exercise have been reported previously from our laboratory from a similar heat acclimation protocol.[32] However, any increases in local forehead sweat rate in the current study were not enough to offset increases in T_{aur} in the PP group. Calf T_{skin} was significantly warmer (0.7 °C) on day-7 when compared to day-1 in the TP group, possibly due to cumulative heat storage over the heat exercise protocol. However, this difference is within the observed inter-participant variation. In addition the sum of thigh and calf T_{skin} was unchanged over the seven days, reflecting no change in total body heat storage. Therefore, the difference in calf T_{skin} is unlikely to be an adaptive response to heat acclimation.

4.4 Plasma volume

Recurrent heat stress alters hormones that regulate intravascular fluid, proteins and plasma electrolytes that consequently increase total body water and thus intravascular plasma volume (PV). [3,33] PV increases in able-bodied persons after 3-4 days of repeated heat exposure [34] by to 3-27% [35-37] and is correlated with the extent of fluid losses during exercise. Increased secretion of aldosterone and argninine vasopressin are largely responsible for the expansion of total body water and thus PV during repeated exposure to heat stress.[37,38] Change in PV can be estimated using changes in concentration of blood haemoglobin and haematocrit before and after heat acclimation. [39] Significant decreases in haemoglobin and haematocrit concentrations demonstrate the expanded PV often seen after heat acclimation.[39] Haemoglobin and haematocrit concentrations, and thus PV, remained unchanged in our SCI participants over 7 days of heat acclimation exercise. Reasons for this are likely multifactorial, including, but not limited to: 1) attenuated aldosterone increase during exercise compared to AB persons [40] and 2) impaired sudomotor/sweating activity resulting in less fluid loss to drive the adaptive response of increased PV. Further investigation is needed to fully characterize the changes in fluid regulatory hormones in SCI persons during exercise and heat acclimation, more specifically.

4.5 Training effects

As our participants were recreationally active and not specifically trained it is important top identify whether any training effect occurred due to the seven-day protocol, i.e. reductions in HR, BLa and RPE as observed for able-bodied persons. [3,5,33] As no change was noted in HR and BLa responses between day-1 and day-7, it is unlikely that any appreciable training effect occurred. Regarding RPE, the main effect observed between day-1 and day-7 suggests participants may have simply become more familiar with the exercise. The decrease in values was between 1-2 units representing verbal anchors between ‘Somewhat hard’ and ’Hard’.

4.6 Perceived thermal strain

Participants with paraplegia reported lower RPTS after seven days of heat acclimation without changes in daily ΔT_{aur} or mean skin temperature whereas participants with TP demonstrated no significant changes in either ΔT_{aur}, T_{skin} or the associated RPTS. Studies of able-bodied participants have shown thermal sensation and RPE contribute to adjustments in exercise intensity and activity patterns to reduce heat storage.[41,42] As both the PP and TP showed similar decreases (~1 point) in RPE and only the PP reported lower RPTS, perceptions of effort are unlikely to be the sole contributor to changes in thermal sensation. The rate of heat storage for the TP group is considerably greater than for PP so would likely override any subtle exercise familiarisation effects noted earlier. It is also likely that PP and

TP participants differ in their previous experiences of thermal dysfunction and thermal imbalance. Due to significant loss of afferent thermal sensation for TP, these experiences are potentially more severe and may contribute to poor adaptations to RPTS in hot, exercising conditions whereas PP still have afferent thermal sensation and may thus be able to adjust their sensations more easily, although may be not appropriately. A similar response of decreased RPTS with no change in T_{aur} has been observed in athletes with paraplegia wearing a neck cooling device.[19] These findings collectively suggest that SCI persons with paraplegia have an increased risk of heat injury due to an attenuated perception of thermal strain despite core temperature elevation. Interestingly, although ΔT_{aur} and skin temperatures were consistently greater for the TP, their initial (day 1) RPTS values were similar to that for the PP (5.5 and 5.8, respectively). Thus, the further investigation of the underlying mechanisms and contributors to thermal sensation is warranted.

4.7 Study Limitations

There are a number of limitations that should be considered when interpreting the results of the current study. Firstly, participants had varying levels of injury completeness, making the groups relatively non-homogenous in severity of thermoregulatory dysfunction. However, the range of lesion levels for each group is certainly representative of groups used within the literature cited. Furthermore, four of the ten SCI participants were taking anticholinergic medications, which have potential to attenuate the sympathetic cholinergic activity required for sweating. Of the paucity of data in the literature, some studies state that anticholinergics decrease, but do not abolish sweating while others state that limited data precludes prediction of the effects of anticholinergics on sweating. [43,44] Further research is needed to quantify the effects of anticholinergics on sweat rate.

Although oxygen consumption was not measured in the present study an estimate of the inter-participant heat production may be taken from the absolute exercise intensities undertaken. The TP group undertook exercise at absolute power outputs of approximately 15 to 25 watts whereas the PP group exercised at a wider range of approximately 25 to 65 watts, although this wider range was not evident in the T_{aur} responses. These power outputs are lower than would routinely be expected for trained athletes with SCI[14,45] it is possible that greater absolute power outputs may provide a greater stimulus for heat acclimation. However, in light of the TP group demonstrating continual increases in T_{aur}, this group certainly reached levels of body temperature associated with heat acclimation adaptations. Furthermore, oxygen consumption would have facilitated calculation of exercise efficiency. Wheelchair propulsion exercise is considerably less efficient than arm crank ergometry; however, wheelchair propulsion exercise elicits only local thermoregulatory differences at the arm during prolonged exercise in cool conditions, rather than differences in T_{aur}.[45] How exercise efficiency affects thermoregulatory variables for the SCI population during exercise in the heat has yet to be reported.

It is known that heat acclimation adaptations are related to the exercise intensity, duration, frequency and number of exposures and environmental conditions.[46] Unfortunately, to aid the comparison of our thermal responses to a healthy control group there are, as of yet, no published arm crank ergometry heat acclimation studies reported in the literature. Unpublished data from our laboratory[47] using the same heat acclimation protocol as the current study, but in young able–bodied, arm crank ergometry trained participants, suggests typical acclimation adaptations do occur. i.e. reduced resting rectal temperature, similar acute increases in T_{aur} during exercise to the present study but a reduction by day -7, reduced exercise HR and localized adaptations in sweat rate. Furthermore, heat acclimation responses, especially body temperature responses, do not appear to be affected in the fifth decade, akin to our age groups, in trained participants.[48,49] We are therefore confident that the protocol used can elicit heat acclimation adaptations in participants with fully functioning thermoregulatory systems. However, based on our data, it is possible that persons with SCI may require more days to acclimate or increasingly longer exposures. Interestingly, recent studies have utilized controlled hyperthermia models for heat acclimation which may inducing more complete heat acclimation.[37] Here, core temperature is held at a given level for a set duration of time. Our data suggests that TP and PP persons have increases in T_{aur} of over 1.0°C from rest for approximately half the duration of the protocol with T_{aur} values likely remaining elevated following exercise.[11] Thus, the unique thermal physiology of individuals with SCI may easily replicate and suit a controlled hyperthermia approach.

Finally the small sample size (n=10) limits statistical power; however the probability of a Type I error is quite low as the P values for change in T_{aur} of neither the TP or PP groups (P=0.98 and P=0.57, respectively) approached significance. Chances of finding statistical significance would require a very large sample size and the clinical significance would likely be very small. Recruiting a large population (n=50-100) of persons with SCI who have mobility impairments and multiple medical complications is difficult. Moreover, retaining such a large population for 7 consecutive days of study is likely not feasible. The subject size we studied was the largest feasible within the local veteran population, and the largest of the current literature available.

During this study T_{aur} was taken as an estimate of core temperature due to repeatable measurements being obtained using this measure in persons with SCI [11,13,14,50] and rectal temperature being inappropriate for this population.[51,52] The use of still air and constant environmental temperatures in the heat chamber also contributes to consistent, reliable T_{aur} measures.[20] T_{aur} also represents the site, if not the specific method, used clinically for thermal assessments. As a larger proportion of studies examining thermal responses in SCI have used this method it is thus appropriate both methodologically and for comparison to previous work.

5. Conclusion

This study demonstrated an absence of acclimation to a 7-day heat exercise protocol in ten untrained SCI persons. Specifically, T_{aur}, mean T_{skin}, HR, and PV values were similar on day-1 versus day-7 for both TP and PP groups. Participants with PP demonstrated cooler forehead T_{skin}, which may indicate a limited local adaptation to the heat stress although this was not sufficient to reduce T_{aur}. Furthermore, subnormal resting T_{aur} in persons with TP and impaired changes in RPTS during T_{aur} elevation in both groups have clinical implications that warrant further investigation.

A longer heat acclimation protocol (i.e.; 14 day) should be conducted to more fully investigate the ability of persons with SCI to acclimate to heat. If future trials confirm an inability to acclimate to the heat, there are implications for SCI athletes and non-athletes. For the SCI athlete, lack of heat acclimation can negatively impact performance and endurance especially in warm environments. For the non-athletic SCI person, lack of acclimation can increase risk of heat-related illness in warm environments and thus have a negative impact on quality of life. More data is needed to definitively determine heat acclimation capacity of SCI persons.

Acknowledgements

This material is the result of work supported with resources and the use of facilities at the Palo Alto Veterans Health Care System in Palo, Alto, CA. The Bay Area and Western chapter of the Paralyzed Veterans of Affairs and the Office of Academic Affairs within the Veterans Affairs Health Care System provided direct research costs. Dr. Price was supported by an Applied Research Fellowship from Coventry University, UK.

References

1. Lorenzo S, Minson CT. Heat acclimation improves cutaneous vascular function and sweating in trained cyclists. *J Appl Physiol (1985).* 2010;109(6):1736-1743.
2. Kampmann B, Brode P, Schutte M, Griefahn B. Lowering of resting core temperature during acclimation is influenced by exercise stimulus. *Eur J Appl Physiol.* 2008;104(2):321-327.
3. Garrett AT, Goosens NG, Rehrer NJ, Patterson MJ, Cotter JD. Induction and decay of short-term heat acclimation. *Eur J Appl Physiol.* 2009;107(6):659-670.
4. Sunderland C, Morris JG, Nevill ME. A heat acclimation protocol for team sports. *Br J Sports Med.* 2008;42(5):327-333.
5. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. *J Appl Physiol (1985).* 2010;109(4):1140-1147.
6. Huckaba CE, Frewin DB, Downey JA, Tam HS, Darling RC, Cheh HY. Sweating responses of normal, paraplegic and anhidrotic subjects. *Arch Phys Med Rehabil.* 1976;57(6):268-274.
7. Totel GL, Johnson RE, Fay FA, Goldstein JA, Schick J. Experimental hyperthermia in traumatic quadriplegia. *Int J Biometeorol.* 1971;15(2):346-355.
8. Popa C, Popa F, Grigorian VT, et al. Vascular dysfunctions following spinal cord injury. *J Med Life.* 2010;3(3):275-285.
9. Price MJ. Thermoregulation during exercise in individuals with spinal cord injuries. *Sports Med.* 2006;36(10):863-879.
10. Pritchett RC, Bishop PA, Yang Z, Pritchett KL. Evaluation of artificial sweat in athletes with spinal cord injuries. *Eur J Appl Physiol.* 2010;109:125–131.
11. Price MJ, Campbell IG. Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc.* 2003;35(7):1100-1107.

12. Hopman MT, Oeseburg B, Binkhorst RA. Cardiovascular responses in persons with paraplegia to prolonged arm exercise and thermal stress. *Med Sci Sports Exerc.* 1993;25(5):577-583.

13. Price MJ, Campbell IG. Thermoregulatory responses of paraplegic and able-bodied athletes at rest and during prolonged upper body exercise and passive recovery. *Eur J Appl Physiol Occup Physiol.* 1997;76(6):552-560.

14. Price MJ, Campbell IG. Thermoregulatory responses of spinal cord injured and able-bodied athletes to prolonged upper body exercise and recovery. *Spinal Cord.* 1999;37(11):772-779.

15. Dawson B, Bridle J, Lockwood RJ. Thermoregulation of paraplegic and able bodied men during prolonged exercise in hot and cool climates. *Paraplegia.* 1994;32(12):860-870.

16. Trbovich M, Ortega C, Schroeder J, Fredrickson M. Effect of a cooling vest on core temperature in athletes with and without spinal cord injury. *Top Spinal Cord Inj Rehabil.* 2014;20(1):70-80.

17. Price MJ. Preparation of Paralympic Athletes; Environmental Concerns and Heat Acclimation. *Front Physiol.* 2015;6:415.

18. Winget CM, DeRoshia CW, Holley DC. Circadian rhythms and athletic performance. *Med Sci Sports Exerc.* 1985;17(5):498-516.

19. Goosey-Tolfrey VL, Diaper NJ, Crosland J, Tolfrey K. Fluid intake during wheelchair exercise in the heat: effects of localized cooling garments. *Int J Sports Physiol Perform.* 2008;3(2):145-156.

20. Teunissen LP, de Haan A, de Koning JJ, Clairbois HE, Daanen HA. Limitations of temperature measurement in the aural canal with an ear mould integrated sensor. *Physiol Meas.* 2011;32(9):1403-1416.

21. Ramanathan NL. A New Weighting System for Mean Surface Temperature of the Human Body. *J Appl Physiol.* 1964;19:531-533.
22. Borg GA. Perceived exertion: a note on "history" and methods. *Med Sci Sports.* 1973;5(2):90-93.
23. Young AJ, Sawka MN, Epstein Y, Decristofano B, Pandolf KB. Cooling different body surfaces during upper and lower body exercise. *J Appl Physiol (1985).* 1987;63(3):1218-1223.
24. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol.* 1974;37(2):247-248.
25. Shapiro Y, Hubbard RW, Kimbrough CM, Pandolf KB. Physiological and hematologic responses to summer and winter dry-heat acclimation. *J Appl Physiol Respir Environ Exerc Physiol.* 1981;50(4):792-798.
26. Buono MJ, Numan TR, Claros RM, Brodine SK, Kolkhorst FW. Is active sweating during heat acclimation required for improvements in peripheral sweat gland function? *Am J Physiol Regul Integr Comp Physiol.* 2009;297(4):R1082-1085.
27. Gass EM, Gass GC. Thermoregulatory responses to repeated warm water immersion in subjects who are paraplegic. *Spinal Cord.* 2001;39(3):149-155.
28. Castle PC, Kularatne BP, Brewer J, et al. Partial heat acclimation of athletes with spinal cord lesion. *Eur J Appl Physiol.* 2013;113(1):109-115.
29. Buono MJ, Heaney JH, Canine KM. Acclimation to humid heat lowers resting core temperature. *Am J Physiol.* 1998;274(5 Pt 2):R1295-1299.
30. Khan S, Plummer M, Martinez-Arizala A, Banovac K. Hypothermia in patients with chronic spinal cord injury. *J Spinal Cord Med.* 2007;30(1):27-30.

31. Mollinger LA, Spurr GB, el Ghatit AZ, et al. Daily energy expenditure and basal metabolic rates of patients with spinal cord injury. *Arch Phys Med Rehabil.* 1985;66(7):420-426.
32. Bottoms L, Price M. The effect of arm training on thermoregulatory responses and calf volume during upper body exercise. *Eur J Appl Physiol.* 2014;114(6):1113-1122.
33. Garrett AT, Rehrer NJ, Patterson MJ. Induction and decay of short-term heat acclimation in moderately and highly trained athletes. *Sports Med.* 2011;41(9):757-771.
34. Sawka MN, Coyle EF. Influence of body water and blood volume on thermoregulation and exercise performance in the heat. *Exerc Sport Sci Rev.* 1999;27:167-218.
35. Bass DE, Kleeman CR, Quinn M, Henschel A, Hegnauer AH. Mechanisms of acclimatization to heat in man. *Medicine (Baltimore).* 1955;34(3):323-380.
36. Senay LC, Kok R. Body fluid responses of heat-tolerant and intolerant men to work in a hot wet environment. *J Appl Physiol.* 1976;40(1):55-59.
37. Periard JD, Racinais S, Sawka MN. Adaptations and mechanisms of human heat acclimation: Applications for competitive athletes and sports. *Scand J Med Sci Sports.* 2015;25 Suppl 1:20-38.
38. Fellmann N. Hormonal and plasma volume alterations following endurance exercise. A brief review. *Sports Med.* 1992;13(1):37-49.
39. Bazett HC. Climatic effects on the volume and composition of blood in man. *Am J Physiol.* 1940;129:69-83.
40. Kawasaki T, Nakamura T, Sasaki Y, et al. Renal function and endocrine responses to arm exercise in euhydrated individuals with spinal cord injury. *Eur J Appl Physiol.* 2012;112(4):1537-1547.

41. Periard JD, Racinais S, Knez WL, Herrera CP, Christian RJ, Girard O. Thermal, physiological and perceptual strain mediate alterations in match-play tennis under heat stress. *Br J Sports Med.* 2014;48 Suppl 1:i32-i38.

42. Tucker R, Marle T, Lambert EV, Noakes TD. The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *J Physiol.* 2006;574(Pt 3):905-915.

43. Previnaire JG, Soler JM, Leclercq V, Denys P. Severity of autonomic dysfunction in patients with complete spinal cord injury. *Clin Auton Res.* 2012;22(1):9-15.

44. Previnaire JG, Soler JM, Hanson P. Skin potential recordings during cystometry in spinal cord injured patients. *Paraplegia.* 1993;31(1):13-21.

45. Price MJ, Campbell IG. Thermoregulatory and physiological responses of wheelchair athletes to prolonged arm crank and wheelchair exercise. *Int J Sports Med.* 1999;20(7):457-463.

46. Periard JD, Travers GJ, Racinais S, Sawka MN. Cardiovascular adaptations supporting human exercise-heat acclimation. *Auton Neurosci.* 2016;196:52-62.

47. Bottoms L. Thermoregulatory responses during upper body exercise, thermal stress, training and heat acclimation. 2008.

48. Best S, Thompson M, Caillaud C, Holvik L, Fatseas G, Tammam A. Exercise-heat acclimation in young and older trained cyclists. *J Sci Med Sport.* 2014;17(6):677-682.

49. Pandolf KB, Cadarette BS, Sawka MN, Young AJ, Francesconi RP, Gonzalez RR. Thermoregulatory responses of middle-aged and young men during dry-heat acclimation. *J Appl Physiol (1985).* 1988;65(1):65-71.

50. Castle PC, Maxwell N, Allchorn A, Mauger AR, White DK. Deception of ambient and body core temperature improves self paced cycling in hot, humid conditions. *Eur J Appl Physiol.* 2012;112(1):377-385.

51. Gass GC, Camp EM, Nadel ER, Gwinn TH, Engel P. Rectal and rectal vs. esophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol (1985)*. 1988;64(6):2265-2271.

52. Gass GC, Camp EM. The maximum physiological responses during incremental wheelchair and arm cranking exercise in male paraplegics. *Med Sci Sports Exerc.* 1984;16(4):355-359.

Table and Figure captions

Table 1. Demographics and peak power outputs of participants

Table 2. Medications of participants

Figure 1. T_{aur} of tetraplegic (TP) and paraplegic (PP) groups on day-1 vs. day-7 of heat acclimation protocol.

Figure 2. ΔT_{aur} (0 to 30 min) in tetraplegic (TP) vs. paraplegic (PP) groups after on day-1 vs. day-7.

Figure 3. Resting T_{aur} in tetraplegic (TP) vs. paraplegic (PP) groups over seven days.

Figure 4. T_{skin} in tetraplegic (TP) and paraplegic (PP) groups on day-1 vs. day-7.

Vitae

Michelle Trbovich, MD

Dr. Trbovich is an Assistant Professor at the University of Texas Health Science Center at San Antonio (UTHSCSA), Department of Rehabilitation Medicine as well as a staff physician at the Veterans Health Care System. She completed an SCI Fellowship at Stanford University and an Advanced Research Fellowship at the Palo Alto VA Health Care System and is board certified in Physical Medicine and Rehabilitation and SCI. Her main research interest is thermoregulation in SCI, with a focus on heat acclimation and cooling techniques. She remains active in lecturing/teaching residents and medical students in clinical and research aspects of SCI.

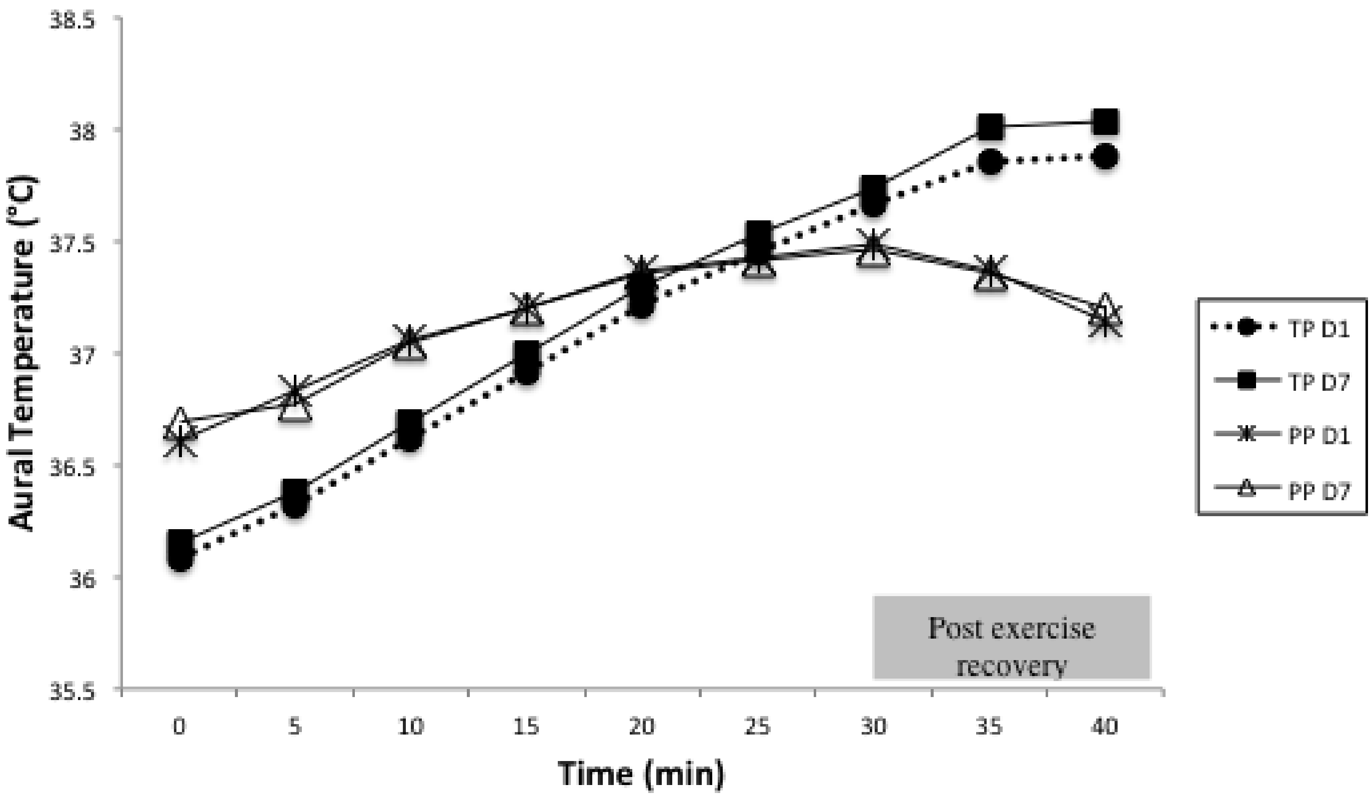
Jenny Kiratli, PhD

Dr. Kiratli serves as the Director of Clinical Research at Palo Alto Veterans Affairs (VA) Health Care System SCI center and has developed a broad-based program of funded research addressing multiple needs of persons with SCI. She has a strong interest in mentoring junior investigators and carries this out as Associate Director of the SCI Medicine Fellowship, Program Director for the VA Advanced SCI Medicine Fellowship Program, and Clinical Associate Professor of Physical Medicine & Rehabilitation at Stanford School of Medicine. She serves on research committees of national SCI organizations and oversees human rights protections in multi-site VA research projects.

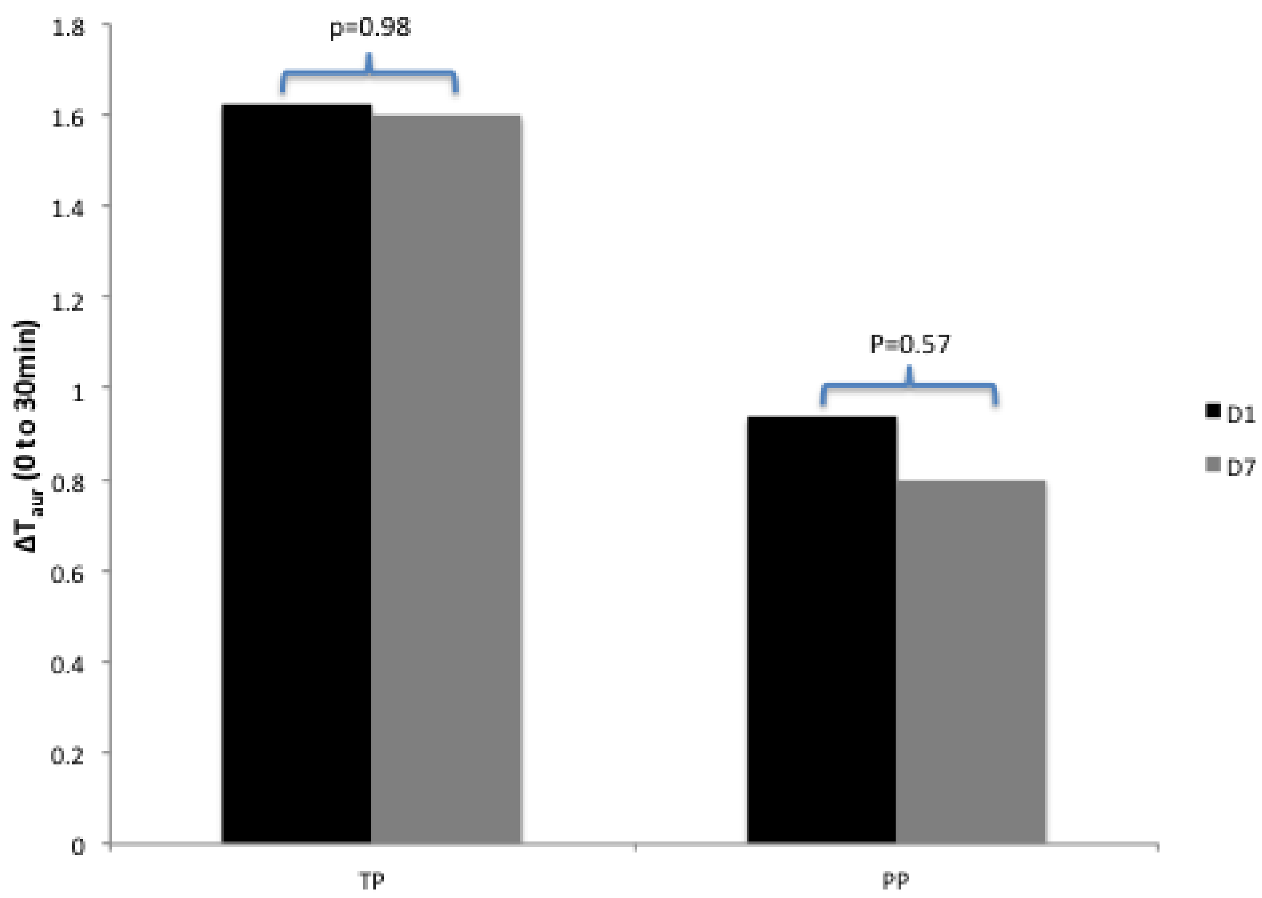
Mike Price, PhD

Dr. Price serves as a Reader in Exercise Physiology in the School of Health and Life Sciences at Coventry University, UK. He obtained his PhD in exercise physiology at The Manchester Metropolitan University, UK and is currently a Fellow of the British Association of Sport and

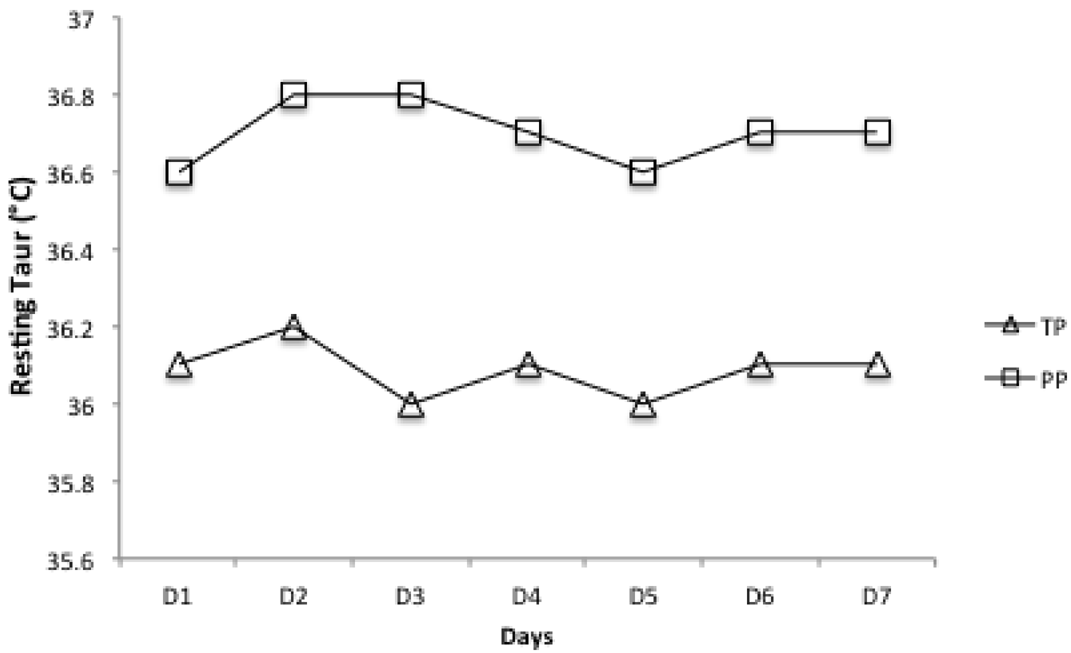
Exercise Sciences (BASES). He has a strong research focus in the area of thermoregulation as it relates to persons with and without SCI and their athletic performance. He is well published in this area and actively mentors students, trainees and post-doctoral students in his laboratory.



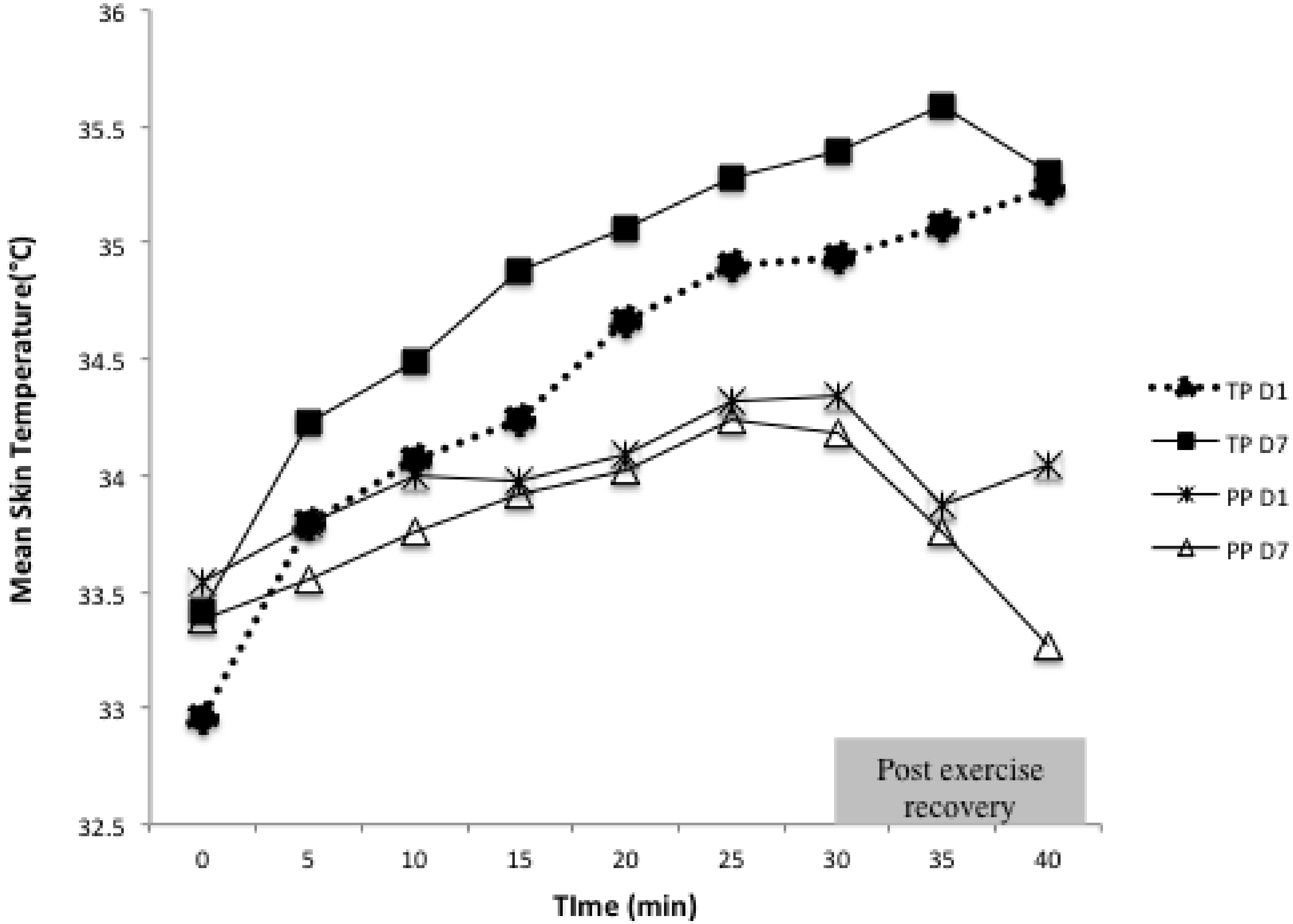
Aural Temperature (°C)
38.5
38
37.5
37
36.5
36
35.5
0
5
10
15
20
25
30
35
40
Time (min)
Post exercise recovery
TP D1
TP D7
PP D1
PP D7



p=0.98
P=0.57
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
ΔT_{aur} (0 to 30min)
TP
PP
D1
D7



37
36.8
36.6
36.4
36.2
36
35.8
35.6
Resting Taur (°C)
D1
D2
D3
D4
D5
D6
D7
Days
TP
PP



Mean Skin Temperature(°C)
36
35.5
35
34.5
34
33.5
33
32.5
0
5
10
15
20
25
30
35
40
Time (min)
TP D1
TP D7
PP D1
PP D7
Post exercise recovery

Table 1. Demographics and peak power outputs of participants

Participant	Sex	Age	ISNCSCI classification	Etiology of SCI	Years since injury	BMI	Peak power (Watts)
TP1	M	57	C6 AIS B	MVA†	34	20.3	45W
TP2	F	37	C7 AIS D	MVA	16	17.1	30W
TP3	M	46	C7 AIS B	MVA	16	25.1	40W
TP4	M	47	C5 AIS A	MVA	10	25.8	50W
TP5	M	41	C7 AIS A	spina bifida	34	34.9	40W
Mean (SD)		45.6 (7.5)			22.0 (11.2)	24.6 (6.8)	40 (9.4)
PP1	M	41	T7 AIS A	transverse myelitis	6	26.7	110W
PP2	M	50	T11 AIS C	bicycle accident	1.5	26	120W
PP3	M	60	L3 AIS D	MVA	12	27.7	100W
PP4	M	55	L1 AIS A	fall	17	22.2	130W
PP5	M	19	T12 AIS A	MVA	2	24.9	50W
Mean (SD)		45.0 (16.1)			7.7 (6.7)	25.5 (2.1)	98 (39.6)
Between group P value		P=0.94			P=0.04	P=0.80	P=0.03

† MVA: Motor Vehicle Accident

Table 2. Medications of participants

Participant	anticholinergics	other
TP1	n/a	synthroid oxycodone
TP2	oxybutynin	baclofen
TP3	oxybutynin	tizanadine diazepam
TP4	n/a	n/a
TP5	n/a	n/a
PP1	oxybutynin nortriptylene	baclofen omeprazole
PP2	n/a	dantrolene
PP3	n/a	cyclobenzaprine fexofenadine synthroid wellbutrin
PP4	n/a	n/a
PP5	oxybutynin	docusate sennosides gabapentin

The effects of a heat acclimation protocol in persons with spinal cord injury

Abstract

Persons without spinal cord injury (SCI) physiologically acclimate between seven to fourteen consecutive days of exercise in the heat. Decreased resting and exercise core temperature, decreased heart rate, increased plasma volume and increased thermal comfort during exercise are changes consistent with heat acclimation. Autonomic dysfunction after SCI impairs heat dissipation through sweating and vasodilation. The purpose of this study is to determine if seven consecutive days of exercise in the heat would result in physiologic changes consistent with heat acclimation in persons with SCI. Ten persons with SCI divided into two groups: tetraplegia (n=5) and paraplegia (n=5) exercised in 35°C using an arm ergometer at 50% W_{peak} for 30 minutes followed by 15 minutes rest. This protocol was repeated over seven consecutive days. Heart rate (HR), skin temperature, aural temperature (T_{aur}), rate of perceived exertion (RPE), rate of perceived thermal strain (RPTS), and plasma volume (PV) were measured throughout the protocol. There were no significant differences in resting T_{aur} exercise T_{aur}, mean skin temperature, HR, PV, RPE or RPTS over the 7 days for either the tetraplegic or paraplegic group. Participants with SCI did not demonstrate the ability to dissipate heat more efficiently over 7 days of exercise at 35°C.

Key words: thermoregulatory dysfunction; heat acclimation; spinal cord injury; thermal strain

---

Abbreviations: AIS= American Spinal Injury Association Impairment Scale, PP = paraplegia, SCI = spinal cord injury, T_{aur} = aural temperature, T_{skin} = skin temperature, TP = tetraplegia, W_{peak} = peak power output.

1. Introduction

Heat acclimation in able-bodied persons results in improved sweating and cutaneous blood flow at a given core temperature to improve heat dissipation.[1] Heat acclimation therefore results in reduced physiological strain as evidenced by lowered resting core and both reduced core temperature and heart rate (HR) during exercise. [2,3] Furthermore, fluid losses from sweating lead to increased fluid regulatory hormones that raises plasma volume by 25% from minutes to hours after exercise in the heat.[3] These physiologic responses improve thermal comfort, endurance and performance in the heat. [4,5]

Although heat acclimation occurs in able-bodied individuals, spinal cord injury (SCI) results in reduced sweat rates and/or impaired cutaneous vasodilation below the lesion, which may prevent effective heat acclimation. [6,7] Studies in SCI show that both at rest and during exercise, the effectiveness of thermoregulation varies according to residual sympathetic function and the extent to which thermoregulatory reflexes are compromised. [8-10] Due to compromised vasomotor and sudomotor activity below the level of injury, such persons have impaired ability to regulate core temperature during heat exposure.[9,11] Specifically, tetraplegics demonstrate a complete absence of, or significant reduction in, sweating (depending on the completeness of injury) whereas those with paraplegia demonstrate a sweating response that is proportional to the lesion level.[6,11,12] During prolonged exercise in cool conditions (25°C), paraplegic athletes demonstrate whole body sweat losses similar to those for matched able-bodied athletes.[13-15] This suggests that sweating is increased within the smaller sensate surface area so that paraplegic athletes can maintain thermal balance in cool conditions.[9] In contrast, during exercise in cool conditions (25°C), tetraplegic athletes demonstrate greater and continual increases in core temperature when compared to paraplegic athletes, a response accentuated during exercise in hot conditions.[11,13,14,16] Few studies have examined thermoregulatory responses to exercise in hot conditions of SCI persons. In preparation for the upcoming Paralympics in Brazil, a recent review article stated that the lack of knowledge of heat acclimation in SCI athletes makes competition/training recommendations for this population difficult. [17] Furthermore, non-athletic SCI persons often limit or avoid activities involving heat exposure to prevent overheating and the associated discomfort. Effective heat acclimation could not only improve performance in SCI athletes but may also improve the ability of non-athletic SCI persons to undertake activities in the heat and thus improve their quality of life. The aim of this study was to examine thermoregulatory responses of SCI persons during heat acclimation. We hypothesized that persons with tetraplegia would not demonstrate heat acclimation whereas those with paraplegia would demonstrate some degree of heat acclimation responses proportional to their level of injury and sweating capacity.

2. Material and Methods

2.1 Setting

Data collection occurred in an exercise lab and an environmental controlled chamber. All study procedures were approved by the local institutional review board.

2.2 Participants

Participants were recruited from a Veterans Affairs clinic and were included if they utilized a manual wheelchair, could propel an upper extremity ergometer, and carried a diagnosis of chronic (>1 year) SCI. Participants were excluded by a history of heat-related illness, cardiovascular disease, were pregnant (self-report) or had an acute illness. Ten persons volunteered and were divided into two groups: tetraplegia (TP) (n=5) and paraplegia (PP) (n=5). Body mass index (BMI) was measured for each participant. SCI classification and neurological exam from the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) exam was obtained from the medical record. The significant differences between groups were time since injury, which would not likely have any effect on thermoregulatory function; and peak power output that is expected to be less in the TP group given the neurologically impaired arm muscles in this group. (Table 1) All medications of participants are recorded in Table 2. Participants were recreationally active but not highly trained. None had undertaken prior heat acclimation training. All refrained from caffeine and alcohol in the 24 hours prior to visits and fasted for two hours prior to each visit.

Table 1.

Table 2.

2.3 Preliminary tests

An exercise test was conducted to establish individual baseline values for peak power output (W_{peak}). The protocol began with exercise on an arm crank ergometer All tests were undertaken on a specific arm crank ergometer (Ergoline, Ergoselect 400) at 5 watts for 2 minutes (min) with increases of 5 watts every 2 min at a cadence of 60 revolutions per min until volitional exhaustion. W_{peak} of all participants are reported in Table 1.

2.4 Heat acclimation protocol

2-3 days after preliminary testing, participants began the seven consecutive day heat acclimation protocol. Each day, participants arrived at the laboratory having followed the same pre-exercise conditions noted earlier. In addition, participants were asked to arrive euhydrated. Although hydration was not specifically measured participants were provided with fluids following every exercise sessions and were advised to continue drinking throughout the remainder of the day, prior to their next visit. Participants exercised continuously at 50% W_{peak} for 30 min (see Table 1 for HA exercise work rates of each participant) followed by 30 min of passive recovery. Each exercise session was scheduled at the same time of day (± 1 hour) to avoid circadian variations. [18] On arrival at the laboratory participants rested for 15 min in an air-conditioned environment (22.4 ±1.1°C, <40% relative humidity) while thermistors were attached. Aural temperature (T_{aur}) was measured from an aural thermistor inserted into the ear canal and insulated with cotton wool. This method is a reliable indicator for core temperature measurement in SCI persons in thermoregulatory studies. [11,13,14,19,20] Skin temperatures (T_{skin}) were measured at standard anatomical landmarks at the forehead, upper arm, chest, thigh and calf using thermistors taped to the skin. Mean T_{skin} was calculated using the latter four measures according to Ramanathan.[21] HR was continually monitored using a Polar monitor. Rating of perceived exertion (RPE) was determined from a 15-point scale ("no exertion" to "maximum exertion") throghout exercise. Rate of perceived thermal strain (RPTS) was recorded from a 9-point (0-8) Likert scale at baseline and during exercise.[22,23]

After thermistors were applied as above, participants rested for 15 min in cool conditions to enable T_{aur} and T_{skin} to equilibrate. After 15 min, baseline T_{aur}, T_{skin}, HR, and RPTS were recorded. Participants then entered the environmental chamber set at 35°C, ~40% relative humidity. After 5 min of rest, T_{aur}, T_{skin}, HR, and RPTS were documented. Participants then began the 30 min exercise protocol. $T_{aur,}$ T_{skin}, and HR measurements were continuously monitored by the data logger, and recorded every 5 min by an investigator. RPE and RPTS were recorded at 5, 15 and 30 min. On day-1 and day-7, venous blood samples (10 ml) were drawn at 0 and 30 min for measurement of hemoglobin concentration and hematocrit (Unicell DXH800 Coulter Cellular Analysis System) to assess changes in plasma volume (PV) during exercise.[24] In addition, earlobe capillary blood samples (20 μl) were also obtained on day-1 and day-7 to measure blood lactate concentration (BLa; Lactate Pro, Arkay, Japan) at rest, 5, 15 and 30 min. After 30 min of exercise, participants rested for 15 min prior to exiting the chamber. Participants were monitored by a physician throughout and after each exercise session for signs of heat-related illness. Participants were given the opportunity to leave the chamber voluntarily if uncomfortable or if T_{aur} increased by more than 2°C above baseline; however, none chose to do so.

2.5. Statistical analysis

Data are presented as mean ±SD. Data were analyzed using SPSS, v17. Normality of data was checked using the Kolmogorov-Smirnov test and by visual inspection of the normality plots. Differences in variables over time within a session (i.e., a Within-Session Time factor, with 2 levels, the initial resting temperature at 0 min and the terminal temperature at 30 min) and over days (i.e., a Days factor, with two levels, day-1 and day-7) were analyzed separately for TP and PP groups using 2-way ANOVA with repeated measures on both factors (Within-Session Time and Days). In addition, a mixed-model ANOVA was conducted which included the between-subject Group factor (with two levels, TP and PP) as well as the within subject Within-Session Time and Days factors. For all tests, the alpha level used to determine significance was set to $p<0.05$. Another mixed-model ANOVA was used to assess the resting T_{aur} over the seven days, assessing Groups (between-subject) and Days (within-subject factor with, in this case, 7 levels). For all within-subject factors, Greenhouse-Geisser corrections were applied as appropriate. To assess differences between two means, t-tests for related measures or t-tests for independent measures were used. As SPSS provides actual probability values, these are reported for each comparison; however, for highly significant results, values are reported as P<0.001. The delta T_{aur} (ΔT_{aur}=30-minT_{aur}-0-minT_{aur}) was analyzed using one-way ANOVA for both groups. Changes in PV between day-1 and day-7 were analyzed using paired t-tests. Differences between T_{aur} and T_{skin} at rest between TP and PP groups were analyzed using independent t-tests.

3. Results

Aural temperature for both groups on day-1 and day-7 of the heat acclimation protocol are shown in Figure 1. No significant interaction was observed between Days (day-1 and day-7) and Within-Session Time (0 to 30-min in 5-min intervals) for T_{aur} in either the TP (P=0.948) or the PP group (P=0.576). Main effects for Within-Session Time were observed in both groups (P<0.001). The mixed-model ANOVA also yielded a significant main effect due to Within-Session Time (P<0.001) and a significant interaction between Groups and Within-Session Time (P<0.001), as T_{aur} of the TP group began at a lower T_{aur}, rose faster and ended at a greater temperature than the PP group. (Figure 1) No other main effects or interactions were significant (Fs< 1.0).

The absolute rise/change in T_{aur} (ΔT_{aur}) from 0 to 30 min of exercise was not significantly different from day-1 to day-7 in the TP (1.6±0.4 vs. 1.6±0.3°C, P=0.98) or PP group (0.9±0.1 vs. 0.8±0.3 °C, P=0.57) (Figure 2). There was, however, a significant difference in T_{aur} averaged over all 7 days between TP and PP groups; 1.6±0.30°C vs. 0.8±0.10°C, respectively; P=0.003. Furthermore, there was no interaction between Group and Days (F< 1.0), meaning that the difference in T_{aur} between groups did not change over the week.

Figure 1

Figure 2

There was no significant difference in resting T_{aur} (i.e.;T_{aur} at 0min) between day-1 and day-7 in either the TP (36.1±0.6 vs 36.2±0.6°C, P=0.85) or PP group (36.6 ±0.5 vs. 36.7±0.3°C, P=0.66). (Figure 3) Incidentally, the TP group showed significantly cooler resting T_{aur} than the PP group throughout the seven days (35.9±0.4 vs. 36.5±0.3°C, respectively; P<0.001), but there was no main effect for Days and no interaction between Group and Days (Fs< 1,0).

Figure 3

The mean T_{skin} responses for both groups are shown in Figure 4. Similar to T_{aur}, there were no differences between day-1 and day-7 for T_{skin} in either group (TP: P=0.991; PP: P=0.999). However, when considering the individual skin sites for the PP group, forehead T_{skin} demonstrated a significant main effect for day (P=0.017) with day-7 eliciting cooler temperature than day-1. Similarly, the TP group demonstrated a significant main effect between day-1 and day-7 for calf T_{skin} (P=0.043) with warmer temperatures on day-7. No other differences were observed between days for the individual T_{skin} sites.

Figure 4

HR for the PP group on day-1 and day-7 at 30 min of exercise were 126±29 and 123 ±32 beats.min^{-1} (P>0.05), while those of the TP group were 94±15 and 95±11 beats.min^{-1}

respectively, (P>0.05). Both groups demonstrated a main effect for RPE between days with lower values on day-7 when compared to day-1 (15.4 vs. 13.8 for the TP group, P=0.041; 14.2 vs. 13.0 for the PP group, P=0.038). The PP group demonstrated a main effect between days for RPTS (5.5 ±0.9 vs. 4.6 ±0.5 at 30 min for day-1 and day-7, respectively; P=0.003) whereas TP did not (5.8 ±1.1 vs. 5.8 ±0.8 at 30 min for day-1 and day-7, respectively; P=0.133). After 30 min, BLa~~A~~ concentration on day-1 versus day-7 in the TP group was 2.3±0.3 vs. 2.2±1.2 mmol.l^{-1}, respectively and for the PP group, 3.3±1.9 vs. 3.4±1.7 mmol.l^{-1} with no significant differences in either group. Neither were there differences in the change in PV between day-1 (-0.56 ±1.97; -0.56 ±2.47) and day-7 (-1.02 ±1.91; -0.74 ±0.51) in either the TP or PP groups, respectively (P=0.967).

4. Discussion

We hypothesized that impaired vasomotor and sudomotor function would preclude persons with tetraplegia from acclimating to the heat and that persons with paraplegia would acclimate relative to their level of injury. Physiologic changes consistent with heat acclimation consist of decreased core temperature and HR at a given a exercise intensity, decreased resting core temperature and HR, increased whole body sweat rate, earlier onset of sweating and decreased RPTS. [25,26] Our hypothesis was rejected as neither TP nor PP group, with the exception of lowered RPTS, acclimated to the heat based on these criteria.

4.1 Aural temperature during exercise

The PP and TP groups demonstrated the expected themoregulatory responses for exercise in the heat, those being a gradual increase in T_{aur} for the PP group and a significantly larger increase for the TP groups.[11] Furthermore, increases in T_{aur} were similar to those previously reported for athletes with tetraplegia and high and low levels of paraplegia (2.1,1.1 and 1.4°C, respectively) after 30 min of exercise at a greater intensity (60% W_{peak}) but cooler environmental temperature (31.5°C) [11] so can thus be deemed comparable.

Despite finding the expected differences in ΔT_{aur} *between* TP and PP groups, there was no significant difference in ΔT_{aur} on day-1 and day-7 *within* each group; and ΔT_{aur} did not vary over days within either group. This is reminiscent of Gass et al., who observed no acclimation after 60 min using (resting) warm water immersion (39°C) over five consecutive days in paraplegic men. [27] In contrast, one small study (n=5) of predominately trained PP athletes reported a *partial* heat acclimation response evidenced by decreased resting $T_{aur,}$ decreased T_{aur} (during recovery), HR, RPE and RPTS during each heat acclimation session without changes in sweat rate.[28] Castle et al. utilized mostly (4 of 5) persons with paraplegia who typically have greater vasomotor and sudomotor activity than tetraplegic persons. In addition, participants were trained, so may have undergone some degree of prior acclimation. These facts should not discount their finding of partial heat acclimation; however, it should be clarified that these findings cannot represent the physiology of either the larger untrained SCI population, or those with tetraplegia. To our knowledge, our study is the first to examine heat acclimation in equal groups of untrained persons with tetraplegia (n=5) and paraplegia (n=5) using a traditional heat acclimation protocol.

4.2 Resting aural temperature

The adaptive response of decreased resting core temperature from heat acclimation creates a greater heat storage capacity in anticipation of heat strain so the risk of heat-related injury from hyperthermia is reduced~~does not occur~~. [29] Neither SCI group demonstrated significant changes in the resting T_{aur} over the seven days of exercise in the heat. However, while resting T_{aur} for the PP group (36.5 ±0.3°C) fell in the range of expected values for able-bodied individuals, the TP group demonstrated significantly cooler resting T_{aur} values (35.9 ±0.4; P<0.001).[11,13,14,19] Such subnormal resting core temperature has been observed in up to 66% of persons with SCI.[30] Cooler resting core temperature suggests either lower heat production or increased heat loss. As the drive for heat loss at rest in cool conditions in the current study would be small and would not elicit significant heat loss to the environment, it is likely that lower resting T_{aur} in the TP group results from decreased resting metabolic rate, as previously demonstrated. [31]

The cooler lower body of persons with SCI has been suggested to be a potential site for heat storage[9] and potentially preventing much larger increases in body temperature than may otherwise be expected in the presence of thermal dysfunction. The cooler lower body may therefore act as a *partial* heat sink as thermoregulatory effectors, if present, are concurrently active alongside heat storage occurring, rather than the heat sink activity occurring prior to active thermoregulation. As evidenced by the more 'steady state' T_{aur} response for the PP group than the TP group during exercise any such heat sink properties are ~~is~~ more effective when some sudomotor and vasomotor abilities are present. Where these effectors are severely compromised, such as for the TP group, ~~any effect~~ the benefits of such ~~a heat sink~~ affects may be rendered ineffectual as T_{aur} continues to increase and remain elevated post exercise.[11]

4.3 Skin temperature

Although there were no differences in mean T_{skin} between day-1 and day-7, the calculation of mean T_{skin} includes the arm, chest, thigh and calf skin temperature sites and thus has potential to mask local skin site adaptations, as previously observed.[13] Notable differences in local skin sites are further discussed. For example, forehead T_{skin} of the PP group was cooler on day-7 when compared to day-1. As all PP participants could sweat in this area, it is likely that improved evaporative cooling occurred at the forehead by day-7 when compared to day-1. Increased forehead sweat rates during upper body exercise have been reported previously from our laboratory from a similar heat acclimation protocol.[32] However, any increases in local forehead sweat rate in the current study were not enough to offset increases in T_{aur} in the PP group. Calf T_{skin} was significantly warmer (0.7 °C) on day-7 when compared to day-1 in the TP group, possibly due to cumulative heat storage over the heat exercise protocol. However, this difference is within the observed inter-participant variation. In addition the sum of thigh and calf T_{skin} was unchanged over the seven days, reflecting no change in total body heat storage. Therefore, the difference in calf T_{skin} is unlikely to be an adaptive response to heat acclimation.

4.4 Plasma volume

Recurrent heat stress alters hormones that regulate intravascular fluid, proteins and plasma electrolytes that consequently increase total body water and thus intravascular plasma volume (PV). [3,33] PV increases in able-bodied persons after 3-4 days of repeated heat exposure [34] by to 3-27% [35-37] and is correlated with the extent of fluid losses during exercise. Increased secretion of aldosterone and argninine vasopressin are largely responsible for the expansion of total body water and thus PV during repeated exposure to heat stress.[37,38] Change in PV can be ~~measured~~ estimated using~~via~~ changes in concentration of blood haemoglobin and haematocrit before and after heat acclimation. [39] ~~with~~ Significant d~~d~~ecreases in~~d~~ ~~concentrations~~ haemoglobin and haematocrit concentrations demonstrate the~~seen in~~ expanded PV often seen after heat acclimation.[39] Haemoglobin and haematocrit concentrations, and thus PV, remained unchanged in our SCI participants over 7 days of heat acclimation exercise. Reasons for this are likely multifactorial, including, but not limited to: 1) attenuated aldosterone increase during exercise compared to AB persons [40] and 2) impaired sudomotor/sweating activity resulting in less fluid loss to drive the adaptive response of increased PV. Further investigation is needed to fully characterize the changes in fluid regulatory hormones in SCI persons during exercise and heat acclimation, more specifically.

4.5 Training effects

As our participants were recreationally active and not specifically trained it is important top identify whether any training effect occurred due to the seven-day protocol, i.e. reductions in HR, BLa and RPE as observed for able-bodied persons. [3,5,33] As no change was noted in HR and BLa~~A~~ responses between day-1 and day-7, it is unlikely that any appreciable training effect occurred. Regarding RPE, the main effect observed between day-1 and day-7 suggests participants may have simply become more familiar with the exercise. The decrease in values was between 1-2 units representing verbal anchors between 'Somewhat hard' and 'Hard'.

4.6 Perceived thermal strain

Participants with paraplegia reported lower RPTS ~~thermal strain ratings~~ after seven days of heat acclimation without changes in daily ΔT_{aur} or mean skin temperature~~,~~ ~~while~~ whereas participants with TP demonstrated no significant changes in either ΔT_{aur}, T_{skin} or the associated RPTS. Studies of able-bodied participants have shown thermal sensation and RPE contribute to adjustments in exercise intensity and activity patterns to reduce heat storage (Tucker et al, Periard et al). As both the PP and TP showed similar decreases (~1 point) in RPE and only the PP reported lower RPTS, perceptions of effort are unlikely to be the sole contributor to changes in thermal sensation. The rate of heat storage for the TP group is considerably greater than for PP so would likely override any subtle exercise familiarisation effects noted earlier. It is also likely that PP and TP participants differ in their previous experiences of thermal dysfunction and thermal imbalance. Due to significant loss of afferent thermal sensation for TP, these experiences are potentially more severe and may contribute to poor adaptations to RPTS in hot, exercising conditions whereas PP still have afferent thermal sensation and may thus be able to adjust their sensations more easily, although may be not appropriately. A similar response of decreased RPTS with no change in T_{aur} has been observed in athletes with paraplegia wearing a neck cooling device.[19] These findings collectively suggest that SCI persons with paraplegia have an increased risk of heat injury due to an attenuated perception of thermal strain despite core temperature elevation. Interestingly, although ΔT_{aur} and skin temperatures were consistently greater for the TP, their initial (day 1) RPTS values were similar to that for the PP (5.5 and 5.8, respectively). Thus, the further investigation of the underlying mechanisms and contributors to thermal sensation is warranted.~~Thus, the underlying mechanisms and contributors to thermal sensation in this population, warrantsing further investigation~~.

4.7 Study Limitations

There are a number of limitations that should be considered when interpreting the results of the current study. Firstly, participants had varying levels of injury completeness, making the groups relatively non-homogenous in severity of thermoregulatory dysfunction. However, the range of lesion levels for each group is certainly representative of groups used within the literature cited. Furthermore, four of the ten SCI participants were taking anticholinergic medications, which have potential to attenuate the sympathetic cholinergic activity required for sweating. Of the paucity of data in the literature, some studies state that anticholinergics decrease, but do not abolish sweating while others state that limited data precludes prediction of the effects of anticholinergics on sweating. [41,42] Further research is needed to quantify the effects of anticholinergics on sweat rate.

Although oxygen consumption was not measured in the present study an estimate of the inter-participant heat production may be taken from the absolute exercise intensities undertaken. The TP group undertook exercise at absolute power outputs of approximately 15 to 25 watts whereas the PP group exercised at a wider range of approximately 25 to 65 watts, although this wider range was not evident in the T_{aur} responses. These power outputs are lower than would routinely be expected for trained athletes with SCI[14,43] it is possible that greater absolute power outputs may provide a greater stimulus for heat acclimation. However, in light of the TP group demonstrating continual increases in T_{aur}, this group certainly reached levels of body temperature associated with heat acclimation adaptations. Furthermore, oxygen consumption would have facilitated calculation of exercise efficiency. Wheelchair propulsion exercise is considerably less efficient than arm crank ergometry; however, wheelchair propulsion exercise elicits only local thermoregulatory differences at the arm during prolonged exercise in cool conditions, rather than differences in T_{aur}.[43] How exercise efficiency affects thermoregulatory variables for the SCI population during exercise in the heat has yet to be reported.

It is known that heat acclimation adaptations are related to the exercise intensity, duration, frequency and number of exposures and environmental conditions.[44] Unfortunately, to aid the comparison of our thermal responses to a healthy control group there are, as~~,~~ of yet, no published arm crank ergometry heat acclimation studies reported in the literature. Unpublished data from our laboratory[45] using the same heat acclimation protocol as the current study, but in young able–bodied, arm crank ergometry trained participants, suggests typical acclimation adaptations do occur. i.e. reduced resting rectal temperature, similar acute increases in T_{aur} during exercise to the present study but a reduction by day -7, reduced exercise HR and localized adaptations in sweat rate. Furthermore, heat acclimation responses, especially body temperature responses, do not appear to be affected in the fifth decade, akin to our age groups, in trained participants.[46,47] We are therefore confident that the protocol used can elicit heat acclimation adaptations in participants with fully functioning thermoregulatory systems. However, based on our data, it is possible that persons with SCI may require more days to acclimate or increasingly longer exposures. Interestingly, recent studies have utilized controlled hyperthermia models for heat acclimation which may inducing more complete heat acclimation.[37] Here, core temperature is held at a given level for a set duration of time. Our data suggests that TP and PP persons have increases in T_{aur} of over 1.0°C from rest for approximately half the duration of the protocol with T_{aur} values likely remaining elevated following exercise.[11] Thus, the unique thermal physiology of individuals with SCI may easily replicate and suit a controlled hyperthermia approach.

Finally the small sample size (n=10) limits statistical power; however the probability of a Type I error is quite low as the P values for change in T_{aur} of neither the TP or PP groups ($P=0.98$ and $P=0.57$, respectively) approached significance. Chances of finding statistical significance would require a very large sample size and the clinical significance would likely be very small. Recruiting a large population (n=50-100) of persons with SCI who have mobility impairments and multiple medical complications is difficult. Moreover, retaining such a large population for 7 consecutive days of study is likely not feasible. The subject size we studied was the largest feasible within the local veteran population, and the largest of the current literature available.

During this study T_{aur} was taken as an estimate of core temperature due to repeatable measurements being obtained using this measure in persons with SCI~~.~~ [11,13,14,48] and rectal temperature being inappropriate for this population.[49,50] The use of still air and constant environmental temperatures in the heat chamber also contributes to consistent, reliable T_{aur} measures[20] ~~and rectal temperature are inappropriate for this population.[49,50]~~ T_{aur} also represents the site, if not the specific method, used clinically for thermal assessments. As a larger proportion of studies examining thermal responses in SCI have used this method it is thus appropriate both methodologically and for comparison to previous work.

5. Conclusion

This study demonstrated an absence of acclimation to a 7-day heat exercise protocol in ten untrained SCI persons. Specifically, T_{aur}, mean T_{skin}, HR, and PV values were similar on day-1 versus day-7 for both TP and PP groups. Participants with PP demonstrated cooler forehead T_{skin}, which may indicate a limited local adaptation to the heat stress although this was not sufficient to reduce T_{aur}. Furthermore, subnormal resting T_{aur} in persons with TP and impaired changes in RPTS during T_{aur} elevation in both groups have clinical implications that warrant further investigation.

A longer heat acclimation protocol (i.e.; 14 day) should be conducted to more fully investigate the ability of persons with SCI to acclimate to heat. If future trials confirm an inability to acclimate to the heat, there are implications for SCI athletes and non-athletes. For the SCI athlete, lack of heat acclimation can negatively impact performance and endurance especially in warm environments. For the non-athletic SCI person, lack of acclimation can increase risk of heat-related illness in warm environments and thus have a negative impact on quality of life. More data is needed to definitively determine heat acclimation capacity of SCI persons.

Acknowledgements

This material is the result of work supported with resources and the use of facilities at the Palo Alto Veterans Health Care System in Palo, Alto, CA. The Bay Area and Western chapter of the Paralyzed Veterans of Affairs and the Office of Academic Affairs within the Veterans Affairs Health Care System provided direct research costs. Dr. Price was supported by an Applied Research Fellowship from Coventry University, UK.

~~Dr. Trbovich was supported by the Bay Area and Western chapter of the Paralyzed Veterans of Affairs and the Office of Academic Affairs within the Veterans Affairs Health Care System. Dr. Price was supported by an Applied Research Fellowship from Coventry University.~~

References

1. Lorenzo S, Minson CT. Heat acclimation improves cutaneous vascular function and sweating in trained cyclists. *J Appl Physiol (1985).* 2010;109(6):1736-1743.
2. Kampmann B, Brode P, Schutte M, Griefahn B. Lowering of resting core temperature during acclimation is influenced by exercise stimulus. *Eur J Appl Physiol.* 2008;104(2):321-327.
3. Garrett AT, Goosens NG, Rehrer NJ, Patterson MJ, Cotter JD. Induction and decay of short-term heat acclimation. *Eur J Appl Physiol.* 2009;107(6):659-670.
4. Sunderland C, Morris JG, Nevill ME. A heat acclimation protocol for team sports. *Br J Sports Med.* 2008;42(5):327-333.
5. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. *J Appl Physiol (1985).* 2010;109(4):1140-1147.
6. Huckaba CE, Frewin DB, Downey JA, Tam HS, Darling RC, Cheh HY. Sweating responses of normal, paraplegic and anhidrotic subjects. *Arch Phys Med Rehabil.* 1976;57(6):268-274.
7. Totel GL, Johnson RE, Fay FA, Goldstein JA, Schick J. Experimental hyperthermia in traumatic quadriplegia. *Int J Biometeorol.* 1971;15(2):346-355.
8. Popa C, Popa F, Grigorian VT, et al. Vascular dysfunctions following spinal cord injury. *J. Med. Life.* 2010;3(3):275-285.
9. Price MJ. Thermoregulation during exercise in individuals with spinal cord injuries. *Sports Med.* 2006;36(10):863-879.
10. Pritchett RC, Bishop PA, Yang Z, Pritchett KL. Evaluation of artificial sweat in athletes with spinal cord injuries

. *Eur. J. Appl. Physiol.* 2010;109:125–131.

11. Price MJ, Campbell IG. Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc.* 2003;35(7):1100-1107.
12. Hopman MT, Oeseburg B, Binkhorst RA. Cardiovascular responses in persons with paraplegia to prolonged arm exercise and thermal stress. *Med Sci Sports Exerc.* 1993;25(5):577-583.
13. Price MJ, Campbell IG. Thermoregulatory responses of paraplegic and able-bodied athletes at rest and during prolonged upper body exercise and passive recovery. *Eur J Appl Physiol Occup Physiol.* 1997;76(6):552-560.
14. Price MJ, Campbell IG. Thermoregulatory responses of spinal cord injured and able-bodied athletes to prolonged upper body exercise and recovery. *Spinal Cord.* 1999;37(11):772-779.
15. Dawson B, Bridle J, Lockwood RJ. Thermoregulation of paraplegic and able bodied men during prolonged exercise in hot and cool climates. *Paraplegia.* 1994;32(12):860-870.
16. Trbovich M, Ortega C, Schroeder J, Fredrickson M. Effect of a cooling vest on core temperature in athletes with and without spinal cord injury. *Top Spinal Cord Inj Rehabil.* 2014;20(1):70-80.
17. Price MJ. Preparation of Paralympic Athletes; Environmental Concerns and Heat Acclimation. *Front Physiol.* 2015;6:415.
18. Winget CM, DeRoshia CW, Holley DC. Circadian rhythms and athletic performance. *Med Sci Sports Exerc.* 1985;17(5):498-516.
19. Goosey-Tolfrey VL, Diaper NJ, Crosland J, Tolfrey K. Fluid intake during wheelchair exercise in the heat: effects of localized cooling garments. *Int J Sports Physiol Perform.* 2008;3(2):145-156.
20. Teunissen LP, de Haan A, de Koning JJ, Clairbois HE, Daanen HA. Limitations of temperature measurement in the aural canal with an ear mould integrated sensor. *Physiol Meas.* 2011;32(9):1403-1416.
21. Ramanathan NL. A New Weighting System for Mean Surface Temperature of the Human Body. *J Appl Physiol.* 1964;19:531-533.

22. Borg GA. Perceived exertion: a note on "history" and methods. *Med Sci Sports.* 1973;5(2):90-93.
23. Young AJ, Sawka MN, Epstein Y, Decristofano B, Pandolf KB. Cooling different body surfaces during upper and lower body exercise. *J Appl Physiol (1985).* 1987;63(3):1218-1223.
24. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol.* 1974;37(2):247-248.
25. Shapiro Y, Hubbard RW, Kimbrough CM, Pandolf KB. Physiological and hematologic responses to summer and winter dry-heat acclimation. *J Appl Physiol Respir Environ Exerc Physiol.* 1981;50(4):792-798.
26. Buono MJ, Numan TR, Claros RM, Brodine SK, Kolkhorst FW. Is active sweating during heat acclimation required for improvements in peripheral sweat gland function? *Am J Physiol Regul Integr Comp Physiol.* 2009;297(4):R1082-1085.
27. Gass EM, Gass GC. Thermoregulatory responses to repeated warm water immersion in subjects who are paraplegic. *Spinal Cord.* 2001;39(3):149-155.
28. Castle PC, Kularatne BP, Brewer J, et al. Partial heat acclimation of athletes with spinal cord lesion. *Eur J Appl Physiol.* 2013;113(1):109-115.
29. Buono MJ, Heaney JH, Canine KM. Acclimation to humid heat lowers resting core temperature. *Am J Physiol.* 1998;274(5 Pt 2):R1295-1299.
30. Khan S, Plummer M, Martinez-Arizala A, Banovac K. Hypothermia in patients with chronic spinal cord injury. *J Spinal Cord Med.* 2007;30(1):27-30.
31. Mollinger LA, Spurr GB, el Ghatit AZ, et al. Daily energy expenditure and basal metabolic rates of patients with spinal cord injury. *Arch Phys Med Rehabil.* 1985;66(7):420-426.
32. Bottoms L, Price M. The effect of arm training on thermoregulatory responses and calf volume during upper body exercise. *Eur J Appl Physiol.* 2014;114(6):1113-1122.
33. Garrett AT, Rehrer NJ, Patterson MJ. Induction and decay of short-term heat acclimation in moderately and highly trained athletes. *Sports Med.* 2011;41(9):757-771.
34. Sawka MN, Coyle EF. Influence of body water and blood volume on thermoregulation and exercise performance in the heat. *Exerc Sport Sci Rev.* 1999;27:167-218.
35. Bass DE, Kleeman CR, Quinn M, Henschel A, Hegnauer AH. Mechanisms of acclimatization to heat in man. *Medicine (Baltimore).* 1955;34(3):323-380.
36. Senay LC, Kok R. Body fluid responses of heat-tolerant and intolerant men to work in a hot wet environment. *J Appl Physiol.* 1976;40(1):55-59.
37. Periard JD, Racinais S, Sawka MN. Adaptations and mechanisms of human heat acclimation: Applications for competitive athletes and sports. *Scand J Med Sci Sports.* 2015;25 Suppl 1:20-38.
38. Fellmann N. Hormonal and plasma volume alterations following endurance exercise. A brief review. *Sports Med.* 1992;13(1):37-49.
39. Bazett HC. Climatic effects on the volume and composition of blood in man. *Am J Physiol.* 1940;129:69-83.
40. Kawasaki T, Nakamura T, Sasaki Y, et al. Renal function and endocrine responses to arm exercise in euhydrated individuals with spinal cord injury. *Eur J Appl Physiol.* 2012;112(4):1537-1547.
41. Previnaire JG, Soler JM, Leclercq V, Denys P. Severity of autonomic dysfunction in patients with complete spinal cord injury. *Clin Auton Res.* 2012;22(1):9-15.
42. Previnaire JG, Soler JM, Hanson P. Skin potential recordings during cystometry in spinal cord injured patients. *Paraplegia.* 1993;31(1):13-21.
43. Price MJ, Campbell IG. Thermoregulatory and physiological responses of wheelchair athletes to prolonged arm crank and wheelchair exercise. *Int J Sports Med.* 1999;20(7):457-463.
44. Periard JD, Travers GJ, Racinais S, Sawka MN. Cardiovascular adaptations supporting human exercise-heat acclimation. *Auton Neurosci.* 2016;196:52-62.

45. Bottoms L. Thermoregulatory responses during upper body exercise, thermal stress, training and heat acclimation. 2008.
46. Best S, Thompson M, Caillaud C, Holvik L, Fatseas G, Tammam A. Exercise-heat acclimation in young and older trained cyclists. *J Sci Med Sport*. 2014;17(6):677-682.
47. Pandolf KB, Cadarette BS, Sawka MN, Young AJ, Francesconi RP, Gonzalez RR. Thermoregulatory responses of middle-aged and young men during dry-heat acclimation. *J Appl Physiol (1985)*. 1988;65(1):65-71.
48. Castle PC, Maxwell N, Allchorn A, Mauger AR, White DK. Deception of ambient and body core temperature improves self paced cycling in hot, humid conditions. *Eur J Appl Physiol*. 2012;112(1):377-385.
49. Gass GC, Camp EM, Nadel ER, Gwinn TH, Engel P. Rectal and rectal vs. esophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol (1985)*. 1988;64(6):2265-2271.
50. Gass GC, Camp EM. The maximum physiological responses during incremental wheelchair and arm cranking exercise in male paraplegics. *Med Sci Sports Exerc*. 1984;16(4):355-359.

Périard,J.D. Racinais, S., Knez, W.L, Herrera, C.P., Christian, R.J., Girard, O Thermal, physiological and perceptual strain mediate alterations in match-play tennis under heat stress. Br J Sports Med. 2014 Apr; 48(Suppl 1): i32–i38. doi: 10.1136/bjsports-2013-093063

Tucker, R., Marle, T., Lambert, E.V., Noakes, T.D. The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion J Physiol. 2006 Aug 1; 574(Pt 3): 905–915. Published online 2006 Feb 23. doi: 10.1113/jphysiol.2005.101733

Table and Figure captions

Table 1. Demographics and peak power outputs of participants

Table 2. Medications of participants

Figure 1. T_{aur} of tetraplegic (TP) and paraplegic (PP) groups on day-1 vs. day-7 of heat acclimation protocol.

Figure 2. ΔT_{aur} (0 to 30 min) in tetraplegic (TP) vs. paraplegic (PP) groups after on day-1 vs. day-7.

Figure 3. Resting T_{aur} in tetraplegic (TP) vs. paraplegic (PP) groups over seven days.

Figure 4. T_{skin} in tetraplegic (TP) and paraplegic (PP) groups on day-1 vs. day-7.

Vitae

Michelle Trbovich, MD

Dr. Trbovich is an Assistant Professor at the University of Texas Health Science Center at San Antonio (UTHSCSA), Department of Rehabilitation Medicine as well as a staff physician at the Veterans Health Care System. She completed an SCI Fellowship at Stanford University and an Advanced Research Fellowship at the Palo Alto VA Health Care System and is board certified in Physical Medicine and Rehabilitation and SCI. Her main research interest is thermoregulation in SCI, with a focus on heat acclimation and cooling techniques. She remains active in lecturing/teaching residents and medical students in clinical and research aspects of SCI.

Jenny Kiratli, PhD

Dr. Kiratli serves as the Director of Clinical Research at Palo Alto Veterans Affairs (VA) Health Care System SCI center and has developed a broad-based program of funded research addressing multiple needs of persons with SCI. She has a strong interest in mentoring junior investigators and carries this out as Associate Director of the SCI Medicine Fellowship, Program Director for the VA Advanced SCI Medicine Fellowship Program, and Clinical Associate Professor of Physical Medicine & Rehabilitation at Stanford School of Medicine. She serves on research committees of national SCI organizations and oversees human rights protections in multi-site VA research projects.

Mike Price, PhD

Dr. Price serves as a Reader in Exercise Physiology in the School of Health and Life Sciences at Coventry University, UK. He obtained his PhD in exercise physiology at The Manchester Metropolitan University, UK and is currently a Fellow of the British Association of Sport and Exercise Sciences (BASES). He has a strong research focus in the area of thermoregulation as it relates to persons with and without SCI and their athletic performance. He is well published in this area and actively mentors students, trainees and post-doctoral students in his laboratory.

Highlights

- Aural temperature of persons with tetraplegia rises significantly higher than persons paraplegia during heat stress
- Tetraplegic and paraplegic persons have impaired perception of thermal strain during aural temperature elevation
- Resting aural temperature in persons with tetraplegia was significantly lower than persons with paraplegia
- Cooler forehead skin temperature in paraplegic persons may indicate a local adaptation to the heat stress
- Physiologic changes consistent with heat acclimation did not occur in spinal cord injured persons