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Firefighter pre-frontal cortex oxygenation and hemodynamics during rapid heat stress

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ABSTRACT

This study evaluated the impact of rapid heat stress on prefrontal cortex (PFC) oxygenation and hemodynamics. Previous work has demonstrated that heat stress affects cerebral oxygenation and hemodynamics. Fourteen male subjects performed a graded exercise test to a termination criterion (volitional maximum, core temperature = 39.5 °C, or a 2-hour time cap) with (GEAR) and without (NOGEAR) firefighting gear in a laboratory with an ambient temperature of 25–26 °C. Changes in oxyhemoglobin (O_2Hb), total hemoglobin (tHb), and tissue oxygen saturation index (TSI %) were monitored in the left and right PFC using near-infrared spectroscopy (NIRS). Significant NIRS results were a plateau in the left-side O_2Hb and tHb at 80 % of the time to termination (TTT) in NOGEAR, and 60 % of TTT in GEAR. These TTT points were when the subject's core temperature (Tc) was equal to 38 °C. Additionally, there was higher left-side PFC activation during GEAR, as indicated by a significant decrease in TSI % from start to end of exercise and double the reduction in TSI % per minute in PPE compared to NOGEAR. There were no significant differences during the NOGEAR session. These data suggest that a rapid heat stress scenario (GEAR) causes altered cerebral oxygenation and hemodynamic response in the left-side PFC. The left PFC could be working harder to prevent fatigue in GEAR. This could affect cognitive processes during or following exercise in the heat while wearing personal protective equipment. Our results also support previous research demonstrating that NIRS is a sensitive metric of fatigue.

1. Introduction

Humans can deal with large fluctuations in environmental temperatures and exercise conditions while maintaining thermal homeostasis. During heat stress, the human body relies predominantly on thermoregulation from increased circulation to the periphery, sweating, and subsequent evaporation to the external environment. For example, if sweating did not occur, the human body would reach unsafe core temperatures within 10 min of moderate exercise (Kenney & Johnson, 1992).

Problems arise when humans are subject to rapid heat stress. Rapid heat stress is acute heat (Fig. 1.) stress in which heat storage accumulates at twice the rate of a normothermic condition (Coehoorn et al., 2020). In many occupations (e.g., firefighters, hazardous waste disposal, and military) and athletic settings (e.g., American football, auto racing, cricket, and goalkeeping in field hockey), personal protective equipment (PPE) is required to protect the individual from environmental hazards or bodily injury. The PPE has limited air and water vapor permeability, resulting in inadequate exchange to the outer environment. The PPE creates a microclimate with its own temperature and humidity through which metabolically generated heat must cross before escaping into the external environment (Renberg et al., 2022). This microenvironment is the initial environmental layer the body interacts with upon heat dissipation (Cheung et al., 2000). The PPE creates problems in conditions of high exertion, high ambient temperatures, and high relative humidity (Cheung et al., 2000). The PPE is also very cumbersome. Due to added weight, this PPE will also cause an increase in the energy cost of physical performance. In addition, the PPE causes excess metabolic heat production above average values, which can exacerbate the problems associated with heat production and removal when the microenvironment created by the PPE exists (Coehoorn et al., 2020; Coehoorn et al., 2022).

During heat stress of any sort, there are changes in cerebral oxygenation and hemodynamics. Transcranial Doppler readings during

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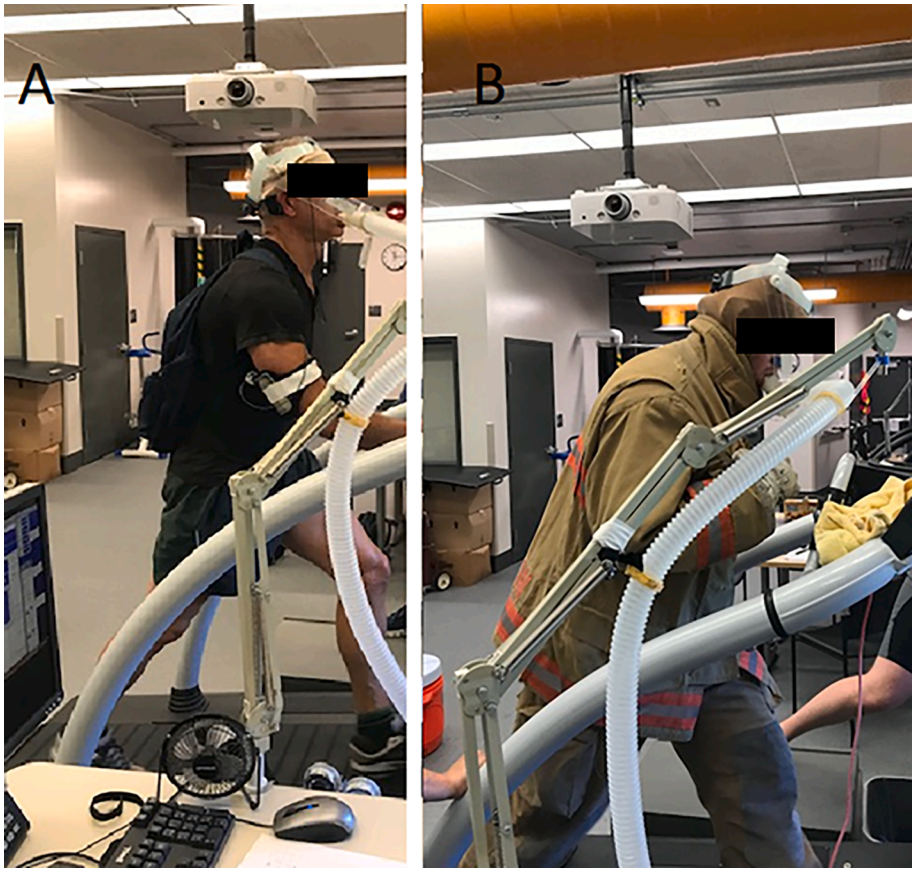

Fig. 1. The difference between the attire in NOGEAR and GEAR. (A) The subject is wearing shorts, *t*-shirt, and the backpack representing the weight of the firefighter PPE. (B) The subject is wearing the full firefighter PPE. The NIRS probes are under a black headband, which is secured by a tensor bandage.

passive heat stress demonstrate that cerebral perfusion as measured by cerebral artery blood velocities is decreased (Brothers et al., 2009; Fan et al., 2008; Fujii et al., 2008). The degree to which cerebral perfusion is decreased is temperature-dependent. Passive heat stress that results in an internal temperature increase of 0.5 to ~ 1.2 °C has little to no effect on mean cerebral perfusion (Low et al., 2009; Wilson et al., 2002). While, passive heat that elevates internal temperatures by 1.5 °C or more results in 20 – 30 % reductions in mean cerebral perfusion (Fan et al., 2008; Fujii et al., 2008; Nelson et al., 2011; Ross et al., 2012). Heat stress resulting from moderate exercise increases left common carotid artery and left internal carotid artery blood flow by 33 % and 17 %, respectively. Middle cerebral artery blood velocity (MCA V) increased by 14 % during moderate exercise (Hellstrom et al., 1996). As exercise continues in the heat to volitional maximum, the middle cerebral artery mean blood velocity (MCA V (mean)) decreases. Nybo and Nielsen (2001) showed that prolonged moderate exercise in the heat until volitional maximum caused a marked reduction in MCA V(mean) by 26 +/- 3 %. After the first 90 s of maximal exercise, there is a decrease in left and right MCA V. This is accompanied by an increase in brain extraction of O_2, glucose, and lactate (Gonzalez-Alonso et al., 2004). There is a 45 % increase in brain extraction of O_2 following the first 90 s of maximal exercise. This shows that although cerebral perfusion declines following the first 90 s of maximal exercise, there are mechanisms to maintain brain function and metabolism. The brain's large oxygen reserve primarily meets the physiological repercussions of reductions in brain perfusion as one approaches exhaustion. Cerebral oxygenation changes have not been studied in rapid heat stress situations. Potential alterations in brain oxygenation and hemodynamics above that which would occur in normothermic scenarios could cause an impairment in maximal neuronal activation. Impairment in maximal neuronal activation leads to a condition known as central fatigue (Nybo & Secher, 2004). Central fatigue affects cognitive function processes, such as decision-making (Smith et al., 2016).

The present study aimed to understand the impact of exercising during rapid heat stress. Specific focus was placed on evaluating cerebral oxygenation and hemodynamics. Prefrontal cortex (PFC) oxygenation and hemodynamics were measured using near-infrared spectroscopy (NIRS). Based on previous literature demonstrating the effect of heat stress on cerebral oxygenation and hemodynamics (Hellstrom et al., 1996; Nybo & Nielsen, 2001), we hypothesized that rapid heat stress would significantly change PFC oxygenation and hemodynamics.

2. Methods

2.1. Participants

Fourteen healthy male (33.6 ± 12.1 years) adults (10 career firefighters and 4 college athletes) participated in this study. The subjects in this study are a subset of a larger study by our laboratory that evaluated many variables related to rapid heat stress (Coehoorn et al., 2020). The subjects had a mean height of 180.8 ± 7.6 cm, a mean pre-exercise body mass of 84.3 ± 9.2 kg, and a mean VO_{2max} of 52.3 ± 5.9 mL kg^{-1} min^{-1}. The participants were considered healthy and had no barriers to physical activity based on the Physical Activity Readiness Questionnaire (PAR-Q). Additionally, it was determined that the participants did not have esophageal constriction, which could contribute to the inability to swallow a core temperature capsule (VitalSense, Health Canada License # 70240). One participant was excluded from the study because of esophageal constriction. The Human Research Ethics Board at the University of Victoria approved this research study (Ethics Protocol #: 17–236), and each participant provided written informed consent.

2.2. Experimental design

Each research subject attended the laboratory on three separate occasions (VO_{2max} determination, NOGEAR, and GEAR). The NOGEAR and GEAR training sessions were the experimental sessions (random, counterbalanced order to prevent crossover effects), and the VO_{2max} session determined pre-experimental fitness status. Subjects needed a minimum VO_{2max} of 35 mL kg $^{-1}$ min^{-1} to participate. Previous research has demonstrated that the firefighter population averages a VO_{2max} of 39.6 kg $^{-1}$ min^{-1} (Storer et al., 2014). Therefore, we established a minimum VO_{2max} of 35 mL kg $^{-1}$ min^{-1} to closely resemble the average of the firefighter population. Subjects were all familiarized with the firefighting gear before the GEAR session. Also, to prevent diurnal effects, GEAR and NOGEAR were scheduled on separate days but at the same time of day.

The participants all followed a specific pre-testing exercise protocol for NOGEAR and GEAR testing sessions. This pre-testing protocol included the following protocols:

Participants refrained from consuming caffeine, alcohol, and nicotine 12 h before testing.
Participants drank at least 3.7 L of water 24 h before testing and at least 500 mL of water two hours before the testing protocol (Convertino et al., 1996). Subjects did not consume water after arriving in the lab. This allowed the core temperature capsule readings not to be influenced by ingested water (Wilkinson et al., 2008).
Participants were instructed to consume their last meal two hours before the experimental sessions.
Participants were instructed to void their bladders completely before NOGEAR and GEAR testing sessions.

The participant's anthropometric values were determined upon arrival at the laboratory for NOGEAR and GEAR testing. Height was measured using a stadiometer (Tanita, USA), and body mass was measured pre- and post-exercise with a scale (Health-o-meter, Continental Scale Corporation, USA). Following these anthropometric measures, participants were equipped with an Equivital Integrated physiological monitoring system for heart rate (HR) measurement. They were then asked to swallow a Jonah core temperature capsule (VitalSense, Health Canada License # 70240) to measure real-time core temperature. The capsule was swallowed 45 min before GEAR and NOGEAR (Domitrovich et al., 2010). The Vitalsense core temperature capsules are pre-calibrated to measure ± 0.1 °C (32 °C to 42 °C) [equivital.com].

To increase thermal stress, the ambient temperature in the laboratory was kept between 25 and 26 °C during the NOGEAR and GEAR sessions. The temperature was maintained by portable heaters positioned around the testing area. Relative humidity was kept between 30 and 50 % for GEAR and NOGEAR. Temperature and relative humidity were continuously monitored via an indoor compact weather monitoring system (Davis Perception II, Davis Instruments Corporation, USA).

The experimental sessions involved a treadmill graded-exercise protocol which included an initial 5-minute stage at 3.5 mph and a 0 % grade, the second stage was 5-minutes at 3.5 mph at 4 % grade, the third stage was 50-minutes at 3.5 mph, and an 8 % grade and the final stage was 1-hour at 3.5 mph and a 12 % grade. HR was recorded at each 0.5 °C increase in core temperature during the treadmill graded exercise protocol (Coehoorn et al., 2020). The treadmill graded exercise protocol was terminated at the onset of any of the following three termination criteria: (1) core temperature reached 39.5 °C, (2) the subject reached a volitional maximum, (3) the participant reached the 2-hour time limit. The subjects wore shorts, a *t*-shirt, socks, and running shoes in the NOGEAR session, and firefighter personal protective equipment (coat, pants, balaclava, gloves, and running shoes) in the GEAR session. Firefighter helmets were not included in the firefighter protective equipment ensemble due to the need to monitor metabolic data, including its own headgear. To match the mass of the firefighter's PPE, during NOGEAR, the ensemble included a backpack with weights equal to the mass of the firefighter's personal protective equipment.

2.3. Cerebral oxygenation and hemodynamics

Throughout the experimental sessions, a continuous wave Near-Infrared Spectroscopy (NIRS) monitor (Portalite, Artinis Medical Systems BV, The Netherlands; Oxysoft version 2.1.6) was used to measure changes in chromophore concentrations of oxyhemoglobin (HbO_2) and deoxy-hemoglobin (HHb), in μM cm^{-1}. Total hemoglobin (tHb) was also reported in μM cm^{-1}, representing the total HbO_2 and HHb at any given point. Total Saturation Index (TSI %) was expressed as a ratio between HbO_2 and THb. The data was collected at 10 Hz with a brain measurement differential pathlength factor (DPF) determined by a scattering medium formula based on age and wavelengths set at 761 nm and 848 nm.

Two NIRS sensors were used to account for oxygenation differences between the prefrontal cortex's left and right sides. The sensors were placed at the height of 15 % of the nasion-inion distance from the nasion and at 7 % of the head circumference to the left and right from the midline to avoid measuring the midline sinus. These locations are used to measure left and right Brodmann's area (BA) 10 and the dorsolateral and anterior PFC (Maidan et al., 2018). The NIRS sensors were also wrapped in thin plastic to prevent sweat from altering the measurement. Lastly, the NIRS sensors were covered with dark fabric to keep out ambient light.

A NIRS event-related design and time to termination (TTT) % design were used to analyze PFC oxidative metabolism. NIRS events were marked at the start of the treadmill graded exercise protocol, at each 0.5 °C increase in core temperature, and the termination of exercise. TTT % was recorded at each 10 % increase. Data around each event marker was average and recorded for further analysis and to determine trends.

2.4. Statistical analysis

All statistical analyses were conducted in R Studio (Version 1.1.456 – © 2009–2018 RStudio, Inc.). The alpha level for significance was set at $p \leq 0.05$. All mean NIRS values (TSI%, HbO2, and tHb) were compared using a 2 × 3 repeated measures ANOVA design - two conditions (GEAR, NOGEAR) by three times (start, Tc 38 °C, end). Additionally, 95 % confidence levels were reported when necessary. Mauchly's test was used to test for the assumption of sphericity (Mauchly, 1940).

2.5. All NIRS parameter data were averaged and separated into 1 – Minute sections, representative of each NIRS event or TTT %.

The magnitude of change in the NIRS parameters was determined as the difference between a given start and end value. The mean start value was set at 0, and the following values were compared to that start point to show the change over time to 100 % TTT or at increasing Tc points.

3. Results

3.1. Prefrontal cortex oxygenation and hemodynamics

There was a gradual increase from rest (set at 0) to 80 % TTT (6.7 + 4.2 μM cm^{-1}) in NOGEAR and from rest to 60 % TTT (6.2 + 5.9 μM cm^{-1}) in GEAR when evaluating PFC HbO_2. There was also a gradual increase from rest (set at 0) to 80 % TTT (9.1 + 6.9 μM cm^{-1}) in NOGEAR and from rest (set at 0) to 60 % TTT (8.8 + 8.8 μM cm^{-1}) in GEAR when evaluating PFC tHb. These increases in both NOGEAR and GEAR were followed by a plateau and then a decrease until 100 % of TTT. V_E/VO_2 deflection points were compared to HbO_2 and tHb peaks during NOGEAR and GEAR. There were deflection points in the V_E/VO_2

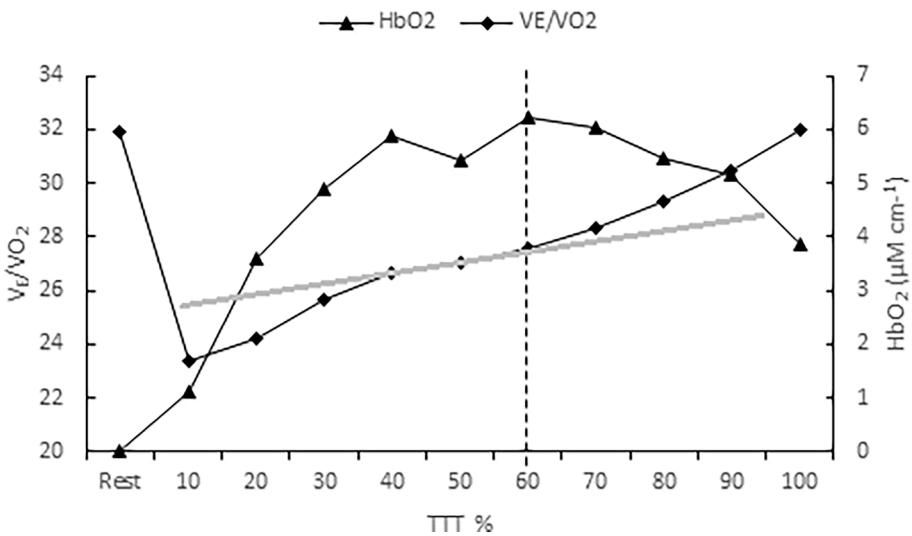

Fig. 2. Evaluation of V_E/VO_2 and HbO_2 during GEAR. Vertical broken line represents the breakpoint for V_E/VO_2 and the point at which HbO_2 begins to decrease. This point is referred to as the respiratory compensation threshold (RCT). The grey line is used to emphasize the deflection point.

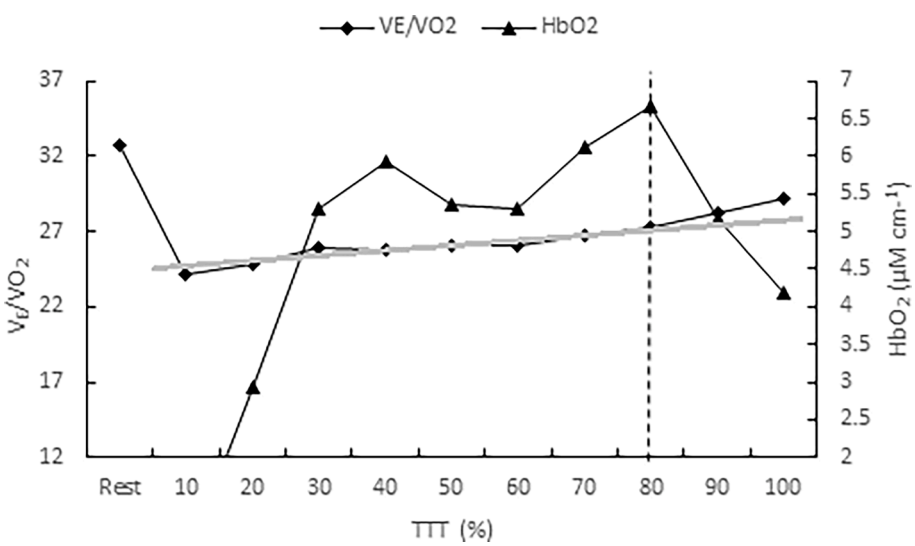

Fig. 3. Evaluation of V_E/VO_2 and HbO_2 during NOGEAR. Vertical broken line represents the breakpoint for V_E/VO_2 and the point at which HbO_2 begins to decrease. This point is referred to as the respiratory compensation threshold (RCT). The grey line is used to emphasize the deflection point.

measurements at 60 % of TTT in GEAR (Figs. 2 & 4) and 80 % of TTT in NOGEAR (Figs. 3 & 5). HbO_2 (Fig. 6) and tHb (Fig. 7) were also evaluated concerning specific NIRS event marker points. There was a plateau in HbO_2 at Tc 38 °C in both the NOGEAR and GEAR sessions. Additionally, there was a plateau in tHb at Tc 38 °C in GEAR.

When evaluating TSI % there was a statistically significant difference ($p \leq 0.05$) in regards to the magnitude of change during GEAR when considering left-side PFC values (Start = 70.9 ± 4.9 %; End = 68.2 ± 5.9 %). It should also be noted that there was a significant difference ($p \leq 0.05$) in the magnitude of change between the start of exercise and Tc 38 °C in left-side TSI % values in GEAR. No TSI % magnitude of change differences were observed when evaluating the right-side PFC during GEAR. Additionally, there was no significant magnitude of change differences on either the right or left sides during the NOGEAR session. Fig. 8.

4. Discussion

This study using NIRS is the first study to demonstrate a Tc point associated with a peak, plateau, and subsequent decrease in cerebral oxygenation. Additionally, it is the first study to demonstrate an increased left-side PFC activation when exercising to a termination point in a rapid heat stress scenario (GEAR). A significant decrease indicated this in TSI % from the start to the end of the exercise. This supports our hypothesis that rapid heat stress directly influences cerebral oxygenation and hemodynamics.

4.1. Cerebral oxygenation and hemodynamics

The interpretation of the NIRS data can be explained as follows: first, during incremental exercise to a termination criterion, there was activation of the PFC in both NOGEAR and GEAR as indicated by increases in HbO_2 and tHb. There was more activation in the left-side PFC during GEAR, as noted in a significant decrease in TSI % (reflecting increased O_2 extraction) from start to Tc 38 °C and from start to termination of exercise. There were no significant decreases in the right-side PFC TSI % values. Additionally, there were no significant differences between the start and any other point during NOGEAR when evaluating TSI %. This

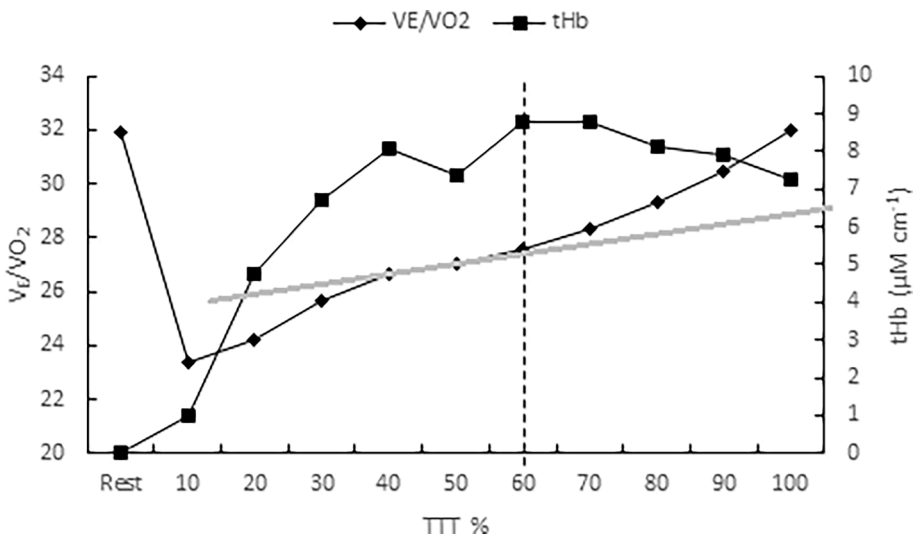

Fig. 4. Evaluation of V_E/VO_2 and tHb during GEAR. Vertical broken line represents the break point for V_E/VO_2 and the point in which tHb begins to decrease. This point is referred to as the respiratory compensation threshold (RCT). The grey line is used to emphasize the deflection point.

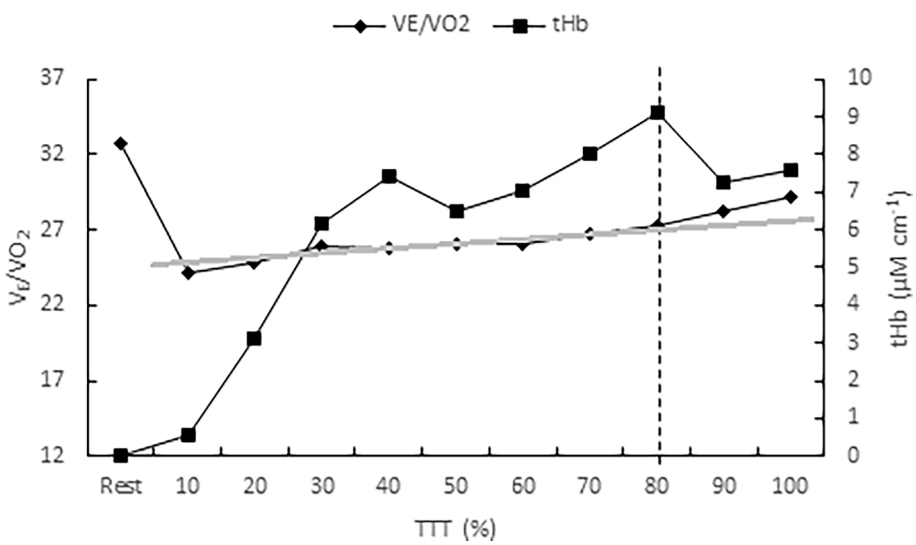

Fig. 5. Evaluation of V_E/VO_2 and tHb during NOGEAR. Vertical broken line represents the break point for V_E/VO_2 and the point in which tHb begins to decrease. This point is referred to as the respiratory compensation threshold (RCT). The grey line is used to emphasize the deflection point.

demonstrates the effect of the thermal acquisition rate on the oxygen uptake rate by the left PFC. Previous research indicates that changes in cerebral oxygenation result from increased neuronal activation (Ferrari et al., 2004). Previous research has found that left-side PFC activation increases are associated with approach-related emotion, whereas increased right-side prefrontal activation is associated with withdrawal (Davidson, 2002; Fox, 1991; Schmidt & Trainor, 2001). Participants in this study were most likely approaching GEAR with a higher approach-related emotional state. The significant difference in HR further supports this before the exercise portion. Mean HR was significantly higher before exercise in GEAR when compared to NOGEAR (NOGEAR: 76.8 ± 8.6 bpm; GEAR: 86.5 ± 9.3 bpm). This potentially indicates that there was an anticipatory effect of wearing the PPE.

Higher left-side PFC activation, as reflected by the changes in oxygenation parameters while undergoing rapid heat stress, could affect cognitive processes. Increased PFC activation is associated with compensatory efforts to maintain a desired level of performance (Liu, 2014). Additionally, there is a relationship between the left PFC and motor areas (Wu et al., 2017). Although speculative, the left PFC could be working harder to prevent fatigue in PPE. This could affect cognitive processes following exercise in the heat while wearing PPE. Hyperthermia causes increased activity in the dorsolateral PFC (Jiang et al., 2013), which plays a significant role in decision-making. Further research is warranted to confirm these results.

Second, there was a peak followed by a plateau at Tc 38 °C in both NOGEAR and GEAR when evaluating O_2Hb and in GEAR when evaluating tHb. It should be noted that the TTT % at which the peak and subsequent decrease in O_2Hb and tHb was at 60 % of TTT in PPE and 80 % of TTT in NOGEAR. This indicates that the point at which the reduction in oxygenation occurred was at a lesser percentage of meeting a termination criterion in GEAR when compared to NOGEAR. The mean values at these points of TTT (%) were not significantly different ($p > 0.05$) than the mean values at Tc 38 °C during both NOGEAR and GEAR. This supports that at approximately Tc 38 °C, there is a peak, plateau, and subsequent decrease in cerebral oxygenation regardless of exercise condition. The peak, plateau, and subsequent reduction in cerebral oxygenation are associated with declining end-tidal CO_2 ($PETCO_2$) and arterial CO_2 content ($PaCO_2$) that happens beyond the respiratory

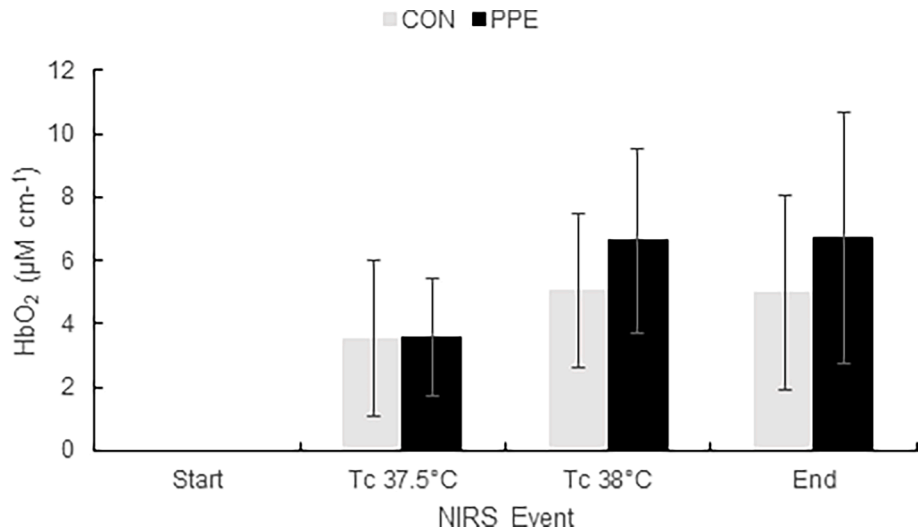

Fig. 6. Mean left PFC changes in HbO_2 from baseline (Start) between NOGEAR and GEAR when evaluating NIRS event points. Error bars = 95 % confidence intervals.

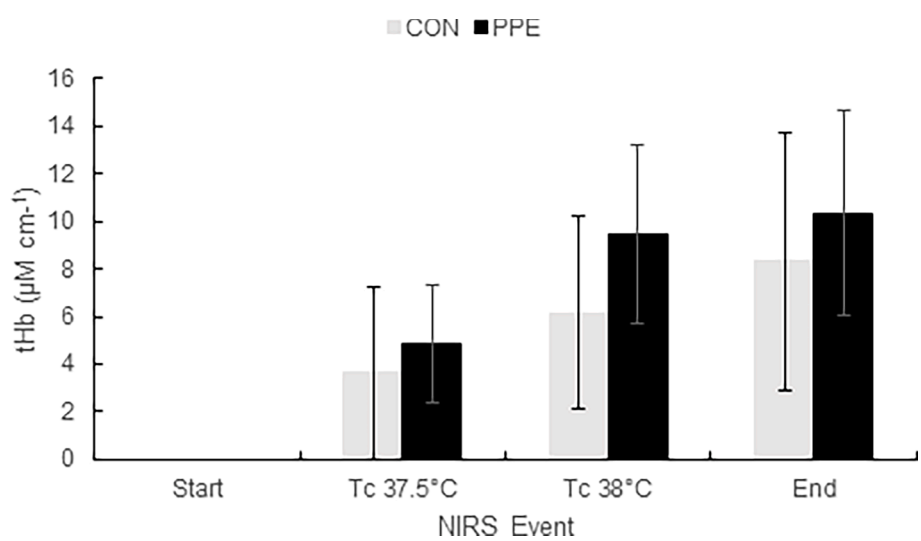

Fig. 7. Mean left PFC changes in tHb from baseline (Start) between NOGEAR and GEAR when evaluating NIRS event points. Error bars = 95 % confidence intervals.

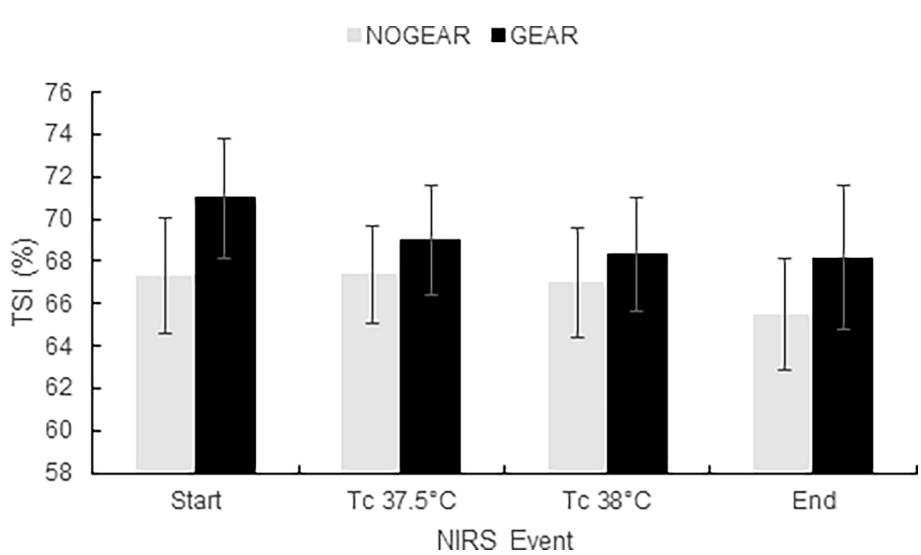

Fig. 8. Mean left PFC TSI % comparison between NOGEAR and GEAR when evaluating NIRS event points. Error bars = 95 % confidence intervals.

compensation threshold (RCT). When exercise continues past the RCT, decreased $PaCO_2$ decreases cerebral blood flow (Bhambhani et al., 2007; Nybo & Rasmussen, 2007). Our study showed a decline in tHb (a representation of blood flow) following 60 % of TTT in GEAR and following 80 % of TTT in NOGEAR. Our study also supported this when evaluating V_E/VO_2 during NOGEAR and GEAR. A deflection point in V_E/VO_2 occurred at roughly 80 % of TTT in NOGEAR and approximately 60 % of TTT in GEAR. These deflection points are representative of RCT (Simon et al., 1983) and further support the statement that there was a decrease in cerebral blood flow at an earlier percentage in GEAR. It further supports the argument that cerebral blood flow peaks, plateaus, and decreases at around Tc 38 °C.

4.2. Summary and conclusion

This study has demonstrated that rapid heat stress causes cerebral oxygenation and hemodynamics changes. This study has shown that Tc 38 °C is associated with a peak, plateau, and subsequent decrease in cerebral oxygenation and hemodynamics. This study also showed a difference in unilateral prefrontal cortex activation when exercising in the heat while wearing PPE. There was a significant ($p \leq 0.05$) change in left side TSI % values from the start until Tc 38 °C and from the beginning to the termination of the exercise. Increased left-side activation could affect cognitive processes during exercise in the heat while wearing PPE. Further research is warranted to confirm the effect of increased left-side PFC activation on cognition.

5. Limitations

A potential limitation of this study is that not all the subjects were career firefighters. Ten of the fourteen subjects were career firefighters, while the remaining four were athletes from the University of Victoria population. The four non-firefighter subjects could have had less long-term acclimation to heat stress. These individuals were most likely not exposed to regular repeated heat stress like the ten career firefighters in the study. That being said, research has demonstrated that heat acclimation has no significant benefit in uncompensable heat stress environments (Cheung & McLellan, 1998). Firefighter PPE creates an uncompensable heat stress environment. Uncompensable heat stress is a scenario where the evaporative capacity of the immediate external environment is less than the evaporative dissipation necessary to maintain heat balance (Cheung et al., 2000). The firefighter PPE creates a microclimate with limited evaporative potential and therefore an uncompensable heat stress environment.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Acknowledgments

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