EFFECTS OF HEAT ON OXIDATIVE STRESS MARKER (REDUCED TO OXIDIZED GLUTATHIONE RATIO), SELECTED PHYSIOLOGICAL RESPONSES AND RUNNING TIME-TRIAL PERFORMANCE AMONG RECREATIONAL ATHLETES

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ABSTRACT

Background: The increased production of free radicals during exercise has been shown to be associated with oxidative stress that impaired exercise performance. However, information on the exercise-induced oxidative stress among individuals living in the hot and humid environment is limited. Thus, the present study was carried out to investigate the effects of heat on oxidative stress, selected physiological responses and time-trial performance among recreational athletes.

Materials and Methods: Twelve male recreational athletes (Age: 23.1 ± 2.1 years old; Body weight: 64.0 ± 8.7 kg; VO₂max: 54.3 ± 4.6 mL.kg⁻¹.min⁻¹) were recruited and assigned to perform prolonged running at 70% of their respective VO₂max for 60 min and immediately followed by a 20 min time-trial performance on a motorized treadmill in a randomized cross-over trial. The two trial conditions were in the heat (H trial; 32˚C room temperature, 70% relative humidity) and thermoneutral (N trial; 25˚C room temperature, 70% relative humidity) environments. During the trials, oxygen uptake (VO₂), heart rate, core body temperature and rate of perceived exertion (RPE) were recorded. Blood samples were drawn before warm-up, at beginning of exercise, and every 20 min of exercise. The samples were analyzed for reduced glutathione (GSH) and oxidized glutathione (GSSG).

Result: There was no significant difference in the GSH: GSSG ratio between trials (p=0.963), where the mean GSH:GSSG ratio at the end of exercise in the N and H trials were 25.4 ± 6.9
and 27.7 ±13.6, respectively. Heart rate was significantly higher in the H trial from the 30th min until the end of the run but was not different from the N trial during the time trial performance. There were also no differences between trials in core body temperature and RPE between trials. The oxygen uptake was significantly higher in the N trial compared to the H trial. The running distance in the H trial was 0.31 km shorter (p< 0.001) compared to the N trial.

**Conclusion:** These results indicated that prolonged running either in the heat or thermo-neutral environment did not seem to enhance oxidative stress. Additionally, running in the heat did not have an effect on core body temperature and RPE compared to a thermoneutral environment among the recreational athletes. However, the time-trial performance of these athletes was impaired in the hot and humid environment.

**Keywords:** Heat, free radicals, oxidative stress, GSH:GSSG ratio, exercise performance

### 1.0 Introduction

Athletic performance is influenced by several factors including physiological (Sleivert and Rowlands, 1996; Tyler et al., 2016), psychological (Bali, 2015; Brown & Fletcher, 2017), nutritional (Baker et al., 2015; Pochmuller et al., 2016) and types of training (Blagrove et al. 2017). Among the physiological factors, heat has been speculated as one of the factors that affects exercise performance (No and Kwak, 2016). A critically high core temperature was proposed as the main factor limiting endurance performance in hot environments (Walters et al., 2000). Heat has also impaired endurance performance in individuals well adapted to living and training in the heat (Chen et al., 2004). Similarly, the running performance of marathon was progressively reduced as the temperature increased from 5°C to 25°C. (Ely et al., 2007). Tatterson et al. (2000) also showed that there was a 6.5% reduction in power output of elite cyclists in a 30 min time-trial performance in high ambient temperature.

An increment in oxidative stress has been reported on various exercise intensity and modes including cycling (Morilla-Ruiz et al., 2005), running (Lawler et al., 1994; Quindry et al., 2008), resistance exercise (Hudson et al., 2008) and Ironman triathlons (Knez et al., 2007). A high intensity and prolonged exercise resulting in accumulation of reactive oxygen species (ROS) and the antioxidant defense system may not be able to buffer the excessive exercise-induced ROS. This will lead to the redox imbalance that may impair skeletal muscles contributing to peripheral fatigue (Reid, 2016) and ultimately reduced exercise performance (Lamina et al., 2013). In addition, there is a 10- to 20-fold increase in whole body oxygen consumption, and 100- to 200-fold increase of oxygen uptake in the active skeletal muscle during endurance exercise (Halliwell and Gutteridge, 1999). The elevated oxygen consumption is thought to increase the production of ROS at rates that exceed the body’s capacity to detoxify them. In return, accumulation of excess ROS results in lipid peroxidation and destruction of nucleic acid and protein (Sjodin et al., 1990; Packer, 1997).

Oxidative damage to adenosine triphosphatase (ATPase) pumps can reduce calcium uptake by the sarcoplasmic reticulum, interfering with muscle excitation-contraction coupling and reducing muscle contractility (Xu et al., 1997). Similarly, ROS may affect the ability to develop action potentials required for muscle contraction through damaging ATPase pumps required for potassium influx back into skeletal muscle cells (Lawler et al., 1998).
Furthermore, oxidative stress which results in muscle-increased ROS concentration is associated with muscular fatigue during contraction and in post-exercise muscular damage (McArdle et al., 2001a; Cooper et al., 2002). The increment of free radicals, such as superoxides has been reported in the mitochondria (Mills et al., 1996; McAnulty et al., 2005). ROS also causes the molecular changes in DNA, proteins, lipids, and other biological molecules that contribute to oxidative stress (Grasso et al., 2003; Zhao et al., 2006; Vadim et al., 2012).

Previous studies have shown that strenuous exercise especially in the hot environment may increase free radicals production, which can lead to oxidative stress (Laitano et al., 2010). Oxidative stress may cause muscle fatigue and subsequently reduce the endurance performance of the athletes. Studies have shown that reduced GSH/GSSG ratio is an indicative of oxidative stress in response to exercise of sufficient duration and intensity (Sen et al., 1994; Laaksonen et al., 1999). Thus, the present study was carried out to investigate the effects of heat on oxidative stress marker (GSH/GSSG), physiological responses and time trial performance in recreational athletes. We hypothesized that exercising in the heat may lead to the occurrence of oxidative stress and subsequently reduced the endurance performance of an athlete.

2.0 Materials and Methods

2.1 Participants

Twelve recreational athletes with the age ranging between 20 to 35 years old were recruited for this study. They must not be taking medication and exercising regularly at least 2 times per week prior to the study. The participants must also be able to run for at least 60 min in the heat at 70% of their VO$_{2\text{max}}$. They must not have any chronic diseases such as hypertension, diabetes mellitus, renal failures, and heart diseases. The participants were given explanation to the nature and risks of experiment procedures and a written informed consent were obtained from them. This study has been approved by the Human Research Ethics Committee of Universiti Sains Malaysia.

2.2 Experimental design

A randomized, cross-over trial was employed for the present study. The actual experiment involved two trials for each participant. They were asked to perform the experimental trials in two different types of environment which were in the heat (H) and thermoneutral (N) environments. They were asked to run at 70% of their respective VO$_{2\text{max}}$ for 60 min and immediately followed by a 20 min time-trial performance. In this study, 3 ml.kg body weight$^{-1}$ of plain water was given to the participants before and every 20 min of exercise during the trials to avoid the adverse effects of dehydration, especially in the heat trial. This amount was similar to that administered to the participants in other studies (Kiew et al., 2001; Chen et al., 2004; Coyle, 2004; Ayu et al., 2010; Wong et al., 2010).
2.3 Preliminary testing

The preliminary testing has been done according to previous research (Chen et al., 2006). The weight and percent body fat of the participants were measured by using an electronic body composition analyzer (Tanita® TBF-410, Japan). A telescoping measuring rod (Seca 220, Germany) was used to measure the height of the participants.

After familiarization to treadmill running, the participants were asked to perform the sub-maximal and maximal oxygen uptake (\(VO_2\text{max}\)) test to establish maximal oxygen uptake (\(VO_2\text{max}\)) and relationship between running speed and oxygen uptake. The participants performed the following tests:

i. A 16-min incremental sub-maximal running test to determine the relationship between speed and oxygen uptake.

ii. An uphill incremental treadmill-running test to determine maximum oxygen uptake (\(VO_2\text{max}\)).

2.4 Familiarization trials

The data from the submaximal and \(VO_2\text{max}\) tests were used to determine the running speed for warm-up (50% \(VO_2\text{max}\)) and endurance running performance (70% of \(VO_2\text{max}\)). The running speed was determined by plotting the graph between speed and oxygen uptake of the participants. The participants then performed a 60 min run at 70% of \(VO_2\text{max}\) on a motorized treadmill at least one week before actual experimental trials. The participants who were unable to cope with this test were excluded from the study.

2.5 Experimental trials

The experimental trials were performed in an improvised heat chamber where the temperature of the room was controlled by halogen lamps (Philips – 500W, France) and air conditioner (York®, Malaysia). A water-bath (Memmert W350t, Germany) was used to maintain the relative humidity (RH) of 70% in the chamber for both trials and a standing fan was used to direct air to the participants to mimic the airflow in an open air environment. The relative humidity and temperature of the chamber were measured by using Digital Psychrometer (Extech Instrument RH300, USA).

Food intake of all participants was recorded in a food diary 3 days prior to the first experimental trial. The participants were then instructed to follow the same diet before the subsequent trial to minimize the differences in resting muscle glycogen concentrations. They were also asked to refrain from training the day before each trial and fast for 10 h before their arrival to the laboratory. On the morning of test day, the participants were given a standardized breakfast which was a piece of bread (Gardenia®, Malaysia) and 3 ml.kg body weight\(^{-1}\) of mineral water 30 min before the warm up. The athletes were then asked to empty their urinary bladder and nude body weight was recorded before warm up and after completion of the trial by using an electrical body composition analyzer (Tanita® TBF-410, Japan). An indwelling cannula (Vasocan® - 22 G, 1", B. Braun, Malaysia) was inserted into a forearm vein and an extension tube was connected to it to facilitate repeated blood withdrawals. Approximately 0.8 ml of heparinized saline (10 IU heparin sodium in 1 ml 0.9% NaCl, B. Braun, Malaysia) was injected into the extension tube for each blood withdrawal to maintain the patency of blood. A rectal thermistor (Yellow Springs Instrument, USA) was
inserted to a depth of 10cm beyond the anal sphincter for measurement of core body temperature. The heart rates of participants were measured by a heart rate sensor (Sport Tester PE3000, Polar, Finland) throughout the trials which was fitted onto the chest wall.

Once the participants were ready, they entered the chamber to perform the running performance either in heat (32°C) or thermoneutral (25°C) environment. Blood sample (6 ml) was collected and all physiological parameters (VO₂, heart rate, and RPE values) were measured. The participants then had a warm-up run for 5 min at 50% VO₂max on the treadmill. Then, the participants ran for 60 min at 70% of their VO₂max and immediately followed by a 20 min time-trial performance. The participants were encouraged to run the longest distance possible in 20 min by controlling the speed of the treadmill.

2.6 Analysis of blood parameters

Blood sample was collected from the participants before the warm-up, at the end of warm-up and every 20 min interval during the trials. The participants ingested 3 ml.kg body weight⁻¹ of plain water at the start of trial and at every 20 min during the trials and after completion of the trials. All physiological parameters such as oxygen uptake, heart rate, core body temperature and rate of perceived exertion (RPE) were measured at the end of warm-up period, at 10 min interval throughout the trials. The GSH/GSSG assay procedure has been done as followed:

2.6.1 Sample Collection and Storage

GSSG Sample: 30 µL of Scavenger was added to the microcentrifuge tube. Then 100 µL of whole blood was added to the bottom of the centrifuge tube and mixed gently. Then, the sample was frozen at -70°C for 30 days.

GSH Sample: 50 µL of whole blood was added to the bottom of a microcentrifuge tube. Then, the sample was frozen at -70°C for 30 days.

2.6.2 Sample Preparation

GSSG Sample: The GSSG sample (130 µL) was thawed and mixed immediately. The sample was then incubated in the room temperature for 5 to 10 min. After that, 270 µL of ice-cold 5% metaphosphoric acid (MPA) was added to the tube and vortexed briefly. The sample was then centrifuged at 1000 x g and 4°C for 10 min.

GSH Sample: The GSH sample (50 µL) was thawed and mixed immediately. Then 350 µL ice-cold 5% MPA was added to the microcentrifuge tube and vortexed briefly.

2.6.3 Assay procedure

50 µL of standards, samples and blank was added to the corresponding wells on the microplate. Then 50 µL DTNB solution was added to the each well. After that, 50 µL of the blood sample was added to each well. The plate was then mixed on an orbital shaker and incubated at room temperature for 5 min. Lastly, 50 µL NADPH solution was added to each well. The plate was then placed in a kinetic plate reader. The change of absorbance at 412 nm was recorded by taking readings every minute for 10 min.
After completion of the trials, the participants were allowed to cool down for 5 to 10 min and removed the heart rate sensor and thermistors. The participants then towel dried themselves and their post-exercise nude body weight was measured to determine the percentage of body weight loss. The participants performed the same trial protocol in the different environment (either heat or thermoneutral environment) 1 week after the first trial.

2.7 Statistical Analysis

Data were statistically analyzed using the statistical software SPSS version 22.0. ANOVA with repeated measures was performed to determine the significance of the differences between two trials over time. The significant difference between trials was measured by using Student’s paired t-test. The statistical significance was accepted at $p < 0.05$. All the data were expressed as means ± standard deviations (SD).

3.0 Results

3.1 Participants’ Anthropometric Data

The anthropometric data of the participants are shown in Table 1.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>23.1 ± 2.1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171.2 ± 5.4</td>
</tr>
<tr>
<td>Body Weight (kg)</td>
<td>64.0 ± 8.7</td>
</tr>
<tr>
<td>Body Mass Index (m.kg$^{-2}$)</td>
<td>21.7 ± 2.1</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>18.1 ± 3.7</td>
</tr>
<tr>
<td>VO$_{2\text{max}}$ (mL.kg$^{-1}$.min$^{-1}$)</td>
<td>54.3 ± 4.6</td>
</tr>
</tbody>
</table>

3.2 Room temperature and relative humidity

The average room temperature and relative humidity in the heat (H) and thermoneutral (N) environments are shown in Table 2. The room temperature and relative humidity were stable throughout the trials. There was a significant difference in room temperature ($p < 0.01$)
between heat (H) and thermoneutral (N) environments, but there was no significant difference in relative humidity \( (p = 0.717) \) for both environments.

**Table 2.** Room temperature (\(^\circ\)C) and relative humidity (%) in the heat (H) and thermoneutral (N) environments.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Thermoneutral (N)</th>
<th>Heat (H)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Room temperature ((^\circ)C)</td>
<td>25.0 ± 0.1</td>
<td>31.9 ± 0.1**</td>
</tr>
<tr>
<td>Relative humidity (%)</td>
<td>70.5 ± 0.1</td>
<td>70.3 ± 0.1</td>
</tr>
</tbody>
</table>

Note: Values are mean ± SD; ** significantly different from the N trial \((p<0.01)\)

### 3.3 Endurance running performance

Our results showed that the endurance running performance of the participants in the heat (H) was significantly shorter compared to the thermoneutral (N) environment \((p < 0.001; \text{Fig. 1})\). The mean distance covered in the both H and N trials were 3.13 ± 0.50 and 3.44 ± 0.50 km, respectively.

**Figure 1:** The endurance running performance (km) of the participants in the heat (H) and thermoneutral (N) environments. *** significantly different from N trial \((p<0.001)\).
3.4 Oxygen uptake (VO₂)

Oxygen uptake (VO₂) of the participants was measured throughout the trials. Our results showed that the oxygen uptake in the heat (H) and thermoneutral (N) environments were significantly influenced by the period of exercise ($p<0.001$; Fig 2) and the heat trial ($p<0.05$). Oxygen uptake was significantly higher in the N trial. A repeated measures for each trial revealed that VO₂ significantly increased over time ($p<0.001$). The mean VO₂ at the end of warm up in the N and H trials were $23.5 \pm 7.5$ and $21.4 \pm 5.8$ mL.kg$^{-1}$.min$^{-1}$ respectively. The mean oxygen uptake at the end of the time trial for both N and H trials were $46.8 \pm 5.5$ and $39.8 \pm 4.8$ mL.kg$^{-1}$.min$^{-1}$, respectively.

![Graph showing oxygen uptake](image)

**Figure 2:** The oxygen uptake (mL.kg$^{-1}$.min$^{-1}$) of the participants in the heat (H) and thermoneutral (N) environments.

+, ++, +++ , significantly different from respective resting values ($p<0.05$, $p<0.01$ and $p<0.001$, respectively).

*,**,***, significantly different between trials ($p<0.05$, $p<0.01$ and $p<0.001$, respectively).
3.5 Core body temperature

The core body temperature of the participants was measured throughout the trials. Duration of exercise has a significant effect ($p<0.001$) on core body temperature of the participants in the N and H trials. There was no significant effect of environment ($p = 0.508$) on core body temperature for both trials. The mean core body temperature of the participants at the baseline in the N and H trials were 36.3 ± 0.6 and 36.5 ± 0.9 °C, respectively. The core temperature significantly increased over time ($p<0.001$; Fig. 3) in both trials. Mean core temperature of the participants at the end of time trial in N and H trials were 38.4 ± 1.1 and 39.0 ± 0.9 °C, respectively.

Figure 3: The core body temperature (°C) of the participants in the heat (H) and thermoneutral (N) environments. ++, +++ significantly different from respective resting values ($p < 0.01$ and $p < 0.001$ respectively).
3.6 Heart rate

The heart rate of participants in the N and H trials was significantly affected by the duration of exercise ($p < 0.001$; Fig 4). There were significant differences ($p < 0.001$) in heart rate compared to respective resting values in both trials. However, heart rate was significantly higher in the H trial compared to the N trial from the 30th min to the end of the 1 h run. The mean heart rate of the participants at the end of time trial in the heat (H) and thermoneutral (N) environments were 182.7 ± 6.9 and 185.4 ± 10.0 beats.min$^{-1}$, respectively. However, no significant interaction was observed on heart rate between the duration and environment of exercise ($p = 0.253$) for both trials.

Figure 4: The heart rate (beats.min$^{-1}$) of the participants in the heat (H) and thermoneutral (N) environments.

+, ++, +++ significantly different from respective resting values ($p < 0.05$, $p < 0.01$ and $p < 0.001$ respectively).

*, **, *** significantly different between trials ($p < 0.05$, and $p < 0.01$, respectively)
3.7 Rate of perceived Exertion (RPE)

The rate of perceived exertion (RPE) of the participants was significantly affected by duration of exercise ($p < 0.001$) in the N and H trials. Results showed that RPE significantly increased in N and H trials, over time ($p < 0.001$) (Fig. 5). RPE was significantly higher from min 20 to min 50 during the 1 h run. The mean RPE of the participants at the end of time trial in N and H trials were $19.8 \pm 0.5$ and $19.9 \pm 0.3$ Borg’s unit, respectively.

**Figure 5:** The ratings of perceived exertion (Borg’s unit) of the participants in the heat (H) and thermoneutral (N) environments.

+, +++ significantly different from respective resting values ($p < 0.05$ and $p < 0.001$ respectively).

*, significantly different between trials ($p < 0.05$)

3.8 Reduced glutathione:oxidised glutathione (GSH:GSSG) ratio

The GSH: GSSG ratio was not significantly different between trials. The mean GSH: GSSG ratio at the end of time trial in the N and H trials was $25.4 \pm 6.9$ and $27.7 \pm 13.6$, respectively (Fig. 6). The GSH: GSSG ratio at the end of time trial was also not significantly different compared to the corresponding resting levels in both trials.
Figure 6: Reduced glutathione (GSH) and oxidized glutathione (GSSG) ratio of the participants in the heat (H) and thermoneutral (N) trial environments

4.0 Discussion

The mean BMI of the participants was within normal classification of BMI which range from 18.5 to 24.9 kg.m\(^{-2}\) (Shiwa et al., 2004) and the mean VO\(_{2}\)\(_\text{max}\) of the participants was 54.3 ± 4.6 mL.kg\(^{-1}\).min\(^{-1}\), which are considered as ‘excellent’ in term of cardiorespiratory fitness for this age group (McArdle et al., 2001b). Mean room temperature and relative humidity in N trial and H trial were 25.0 ± 0.1 °C; 70.5 ± 0.1 % and 31.9 ± 0.1 °C; 70.3 ± 0.1 % respectively (Table 2). This indicated that the room temperature and relative humidity were stable and well monitored throughout the trials. Numerous studies have shown that the high ambient temperature and relative humidity significantly affects the exercise performance (Walters et al., 2000b; McAnulty et al., 2005)

Our finding showed that the endurance performance of participants was affected by the hot and humid environment. The participants were able to run a longer distance in the thermoneutral environment compared to a hot environment (Fig. 1). The running distance of the participants in the N trial was 310 m further compared to the H trial. This result showed that there was a significant effect of heat on time trial performance in recreational athletes. The present observation was similar to other studies (Febbraio et al., 1994a; Galloway and Maughan, 1997; Casa, 1999; Tatterson et al., 2000; Walters et al., 2000b; McAnulty et al.,
Thus, endurance performance was significantly decreased in a hotter environment.

During the trials, oxygen uptake (Fig. 2) and core body temperature (Fig. 3) gradually increased with increases in intensity and duration of exercise in both trials. These data indicated that the participants complied with the requirements for a time trial in both trials, which was running at a higher intensity in order to run the longest distance as they could in 20 min, particularly towards the end of the trial. This finding was similar with the other studies (Febbraio et al., 1994b; Galloway and Maughan, 1997; Parkin et al., 1999), where the core temperature of the participants who were running in the heat was higher compared to the thermoneutral environment.

Both heart rate and RPE values were increased as the duration of exercise increases and both these values were higher in the heat compared to the thermoneutral environment (Fig. 5 and 6). During exercise, heart rate increases linearly with increasing work rate or VO$_2$. The increased heart rate was due to the increased oxygen demand by the contracting muscle during exercise. Thus, the heart needs to pump the blood rapidly in order to supply more blood to the active muscles. It is well established that heat stress reduces stroke volume (SV) and increases heart rate during moderate intense exercise to the extent to which cardiac output might be compromised (Rowell et al., 1966). In a classic study, Rowell et al. (1966) showed that a significantly lower cardiac output, central blood volume, and SV during exercise in a 43°C than a 26°C environment at 63–73% VO$_{2peak}$. This reduced cardiac output was due to the larger reductions in SV compared with the parallel increases in heart rate. This means that heart rate of athletes were higher in the hot environment compared to thermoneutral environment. However heart rate responses and RPE did not significantly differ in both trials at the end of the 20 min time trial. These data indicated that the participants had tried their best to complete the time trials.

Lactate accumulation, hyperthermia and inadequate oxygen cause the accumulation of radical the superoxide lead to oxidative stress. Studies by Gohil et al. (1988), Sastre et al. (1992) and Aguilo et al. (2005) also reported that oxidative stress occurred after exhaustive exercise. Studies have shown that reduced GSH/GSSG ratio is an indicative of oxidative stress in response to exercise of sufficient duration and intensity (Sen et al., 1994; Laaksonen et al., 1999). The enzyme glutathione reductase replenishes GSH by reducing the GSSG to GSH by using NADPH. Our results showed a slight reduction of this ratio, albeit no statistical significance, at the end of exercise compared to the pre-exercise levels in both trials (Fig. 6). This might be due to changes in glutathione reductase activity. Results of the present study was in contrast with previous studies by Margaritis et al. (2003) and Laitano et al. (2010), where they found that combined heat stress and exercise, independently of dehydration, increased both GSH and GSSG, but did not alter the GSH and GSSG ratio. Theoretically, the decrease in GSH concentration and an increase of GSSG concentration result in a decreased GSH and GSSG ratio, which indicates non-radical induced oxidative stress as the stress is caused by redox imbalance. Physical training can strengthen glutathione and other antioxidant defense systems and may reduce resting and exercise induced oxidative stress (Atalay et al., 1996; Alessio and Goldfarb, 1988). Our findings of no statistical significance in oxidative stress following prolonged running in the heat could be attributed to the enhanced antioxidant status of the participants.
5.0 Conclusion and recommendation

The present study showed that prolonged running in the heat did not induce oxidative stress and some of the selected physiological parameters among the recreational athletes. However, the hot and humid environment impaired the endurance running performance of these athletes. Nevertheless, future studies are warranted to assess the nutritional and antioxidant status of the participants along with more oxidative stress markers when performing prolonged physical activity in the heat.

Acknowledgement

We would like to convey our greatest gratitude to all the participants for their willingness to endure the exercise testing. We would also like to thank Madam Jamaayah, Madam Norlida, and Miss Hafizah for their technical assistance during the data collection for this research. Special thanks to Prof. Dr. Aziz Ahmad for his input on the discussion related to oxidative stress.

Declaration

Author(s) declare that they have no conflict of interest regarding publication of this manuscript.

Authors contribution

Author 1: carried out the experimental trials, performed the statistical analysis and drafted the manuscript
Author 2: participated in the design of the study and edited the manuscript
Author 3: participated in the design of the study and edited the manuscript

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