



Rating of perceived exertion increases synergistically during prolonged exercise in a combined heat and hypoxic environment

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ABSTRACT

The purpose of this study was to determine the cardiovascular, thermoregulatory, and perceived exertion responses during 2 h of moderate intensity exercise in a combined high heat (38 °C, 40% relative humidity) and hypoxic (15% O₂) environment. Ten healthy volunteers completed 2 h of treadmill walking at 40% of maximal oxygen uptake in four different conditions, each separated by approximately 1 week: (1) control, 23 °C/20.9% O₂, (2) heat, 38 °C/20.9% O₂, (3) hypoxia, 23 °C/15% O₂, and (4) combined heat/hypoxia, 38 °C/15% O₂. Compared to the responses seen in each condition alone, heart rate (HR) and core temperature (T_{core}) showed an additive increase in the combined heat and hypoxic environment after 2 h of moderate intensity exercise. The most important new finding was that the mean rating of perceived exertion (RPE) increased synergistically 3.3 units when exercising in the combined high heat and hypoxic environment, compared to 1.9 units in the heat condition alone. The results suggest that RPE is a conscious perception of effort that plays a regulatory function to ensure that the work rate remains at an intensity that can be safely sustained, rather than simply a marker of exercise intensity. Such results also support previous anecdotal reports that exercise on hot days at altitude seem unusually difficult.

1. Introduction

Human physiological responses (e.g. heart rate, hemoglobin oxygen saturation, core temperature, etc.) during exercise are known to be affected by environmental stressors such as temperature (Hunter et al., 2002) and altitude (Noakes et al., 2001). However, short of anecdotal reports, there are limited data available concerning the physiological responses associated with exercise in a combined hot and hypoxic environment (Girard and Racinais, 2014; Lloyd et al., 2016).

During exercise in high heat environments, there is increased stress placed on both the cardiovascular and thermoregulatory systems. These include, but are not limited to, a continuous and significant rise in both heart rate (HR) (Girard and Racinais, 2014; Gonzalez-Alonso et al. 1998; Parkin et al., 1999) and core temperature (T_{core}) (Girard and Racinais, 2014; Nybo and Nielson, 2001). The increase in T_{core} may inhibit exercise performance by attenuating cardiac function (Lim et al., 2008), decreasing brain function and central motor drive (Nybo and Nielson, 2001), and/or via a self-imposed, feed-forward limitation to prevent severe hyperthermia and catastrophic collapse (Hampson et al., 2001; Crewe et al., 2008). During exercise in the heat, blood flow is increased to both the working skeletal muscles and to the skin to meet

the demands for muscular activity and temperature regulation, respectively. This creates a competition for the available cardiac output (Gonzalez-Alonso et al., 2008) which results in cardiovascular drift (i.e., a reciprocal increase in heart rate and decrease in stroke volume) during prolonged exercise.

Exposure to a hypoxic environment, such as altitude, produces an increase in resting (Halliwill and Minson, 2002) and exercise HR (Girard and Racinais, 2014; Robinson and Haymes, 1990). The increase appears to be the result of an increase in minute ventilation and/or a resetting of the chronotropic baroreflex (Halliwill and Minson, 2002; Robinson and Haymes, 1990).

Most studies examining the effects of hypoxia on thermoregulation have been done in hypothermic or normothermic environments due to the colder air temperature typically seen at high altitude (Cipriano and Goldman, 1975; Robinson and Haymes, 1990; Savourey et al., 1997). With that said, it should be remembered that there are many locations at moderate altitude that experience very warm summer climates. For example, in 2018 Denver, CO, at an altitude of 5500 feet, had 25 days in which the air temperature reached 95 °F (~35 °C) or greater.

When comparing ratings of perceived exertion (RPE) during exercise in different environmental conditions, participants generally

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report higher values in hot conditions compared to those during cold exposure (Raw et al., 1993); thus similar absolute work rates were perceived to be more difficult. During exercise, RPE has also been found to be significantly correlated with both heart rate and T_{core} - demonstrating linear relationships that can be used to predict duration to fatigue (Crewe et al., 2008; Nybo et al., 2003). In addition, there have been certain physiological cues found to alter RPE. For example, local peripheral factors relating to the work of the exercising muscles as well as central factors relating to feelings from the cardiopulmonary exertion are believed to increase RPE (Borg, 1990; Lloyd et al., 2016; Pandolf, 1982).

It seems reasonable that the increased physiological responses previously reported during exercise in heat or hypoxia alone would be additive in a combined heat and hypoxic environment; however such data is currently limited (Girard and Racinais, 2014). Therefore, the purpose of this study was to determine the cardiovascular (HR), thermoregulatory (core temperature, sweat rate), and RPE responses during 2 h of moderate intensity exercise in a combined high heat (38 °C) and hypoxic (15% O_2) environment. We hypothesized that for healthy adults, the combined stressors of high heat and hypoxia during submaximal exercise would cause an additive increase in HR, T_{core} , and RPE compared to similar exercise in each environment alone.

2. Materials and methods

2.1. Subjects

Approval of this study was obtained prior to the start of data collection by the San Diego State University Institutional Review Board (IRB). The ten participants (5 male, 5 female) were healthy, young adults from the San Diego region. The mean \pm SE age, height, weight, and maximal oxygen uptake for the group was 23.3 ± 0.9 y, 168.1 ± 3.4 cm, 68.7 ± 5.2 kg, and 50.3 ± 3.1 ml/kg/min, respectively. Exclusion criteria for this study included females who were pregnant and any participant with a cardiovascular or orthopedic contraindication to exercise as determined via the Physical Activity Readiness Questionnaire (Par-Q). There were no significant ($p > 0.05$) differences in the responses for the men and women, thus their data were combined.

2.2. Procedures

Participation for this study was made public through flyers posted around the San Diego area and volunteers were asked to contact the researchers. The participants were required to read a detailed description of the study, complete a Physical Activity Readiness Questionnaire (Par-Q), and sign an informed consent form before participation in the study. All of the subjects participated in recreational physical activities, but none was formally acclimated to heat or altitude as determined by self-report. Data were collected during the temperate months of November to May in San Diego, CA to minimize seasonal heat acclimatization in the subjects. Once approved, the participants scheduled the first of five visits to determine their maximal oxygen uptake (VO_2 max). A graded treadmill test to volitional exhaustion was administered during which oxygen uptake was measured using a ParvoMedics TrueOne metabolic cart (Sandy, UT). The remaining four visits were scheduled approximately 1 week apart, thus the total study took most subjects approximately 5–6 weeks to complete. The subjects were informed that prior to each of the next four testing visits, they should be well hydrated by drinking 1 L of fluid both the night before the test and prior to arrival to the laboratory. Furthermore, they were asked not to exercise during the previous 12 h before each lab visit. Lastly, because of the length of the study, no attempt was made to control for the ovarian cycle in the female subjects even though it is well known that core temperature increases approximately 0.3 °C during the luteal phase (Kolka and Stephenson, 1997).

Upon arrival for each of the next four lab visits, the participant's hydration status was checked using urinary specific gravity (USG). If they did not meet the USG hydration criteria of < 1.018 they were asked to drink 1 L of water and then were tested again in 1 h. Next, the subject's dry, semi-nude weight to the nearest 0.05 kg on a balance beam scale was recorded. The subject was asked to apply a Polar Vantage XL heart rate monitor around their chest and insert a rectal probe 10 cm past their anal sphincter.

Each subject completed the following four exercise trials: (1) control, 23 °C/20.9% O_2 , (2) heat, 38 °C/20.9% O_2 , (3) hypoxia, 23 °C/15% O_2 , and (4) combined heat/hypoxia, 38 °C/15% O_2 , in random order approximately 1 week apart. The relative humidity was 40% for all trials. The order of the four exercise tests, which were conducted in an environmental chamber, was blinded to the subjects as they wore the same inspired air delivery mask which covered their nose and mouth for all trials. During the control and heat trials, the subjects breathed ambient air (20.9% oxygen) via the delivery mask from a 100L reservoir bag. During the two hypoxic trials, 15% oxygen was delivered via a Mag-10 Air Generator (Higher Peak, Boston, MA) to the reservoir bag.

The treadmill was set to the appropriate speed and grade to reach the target value of 40% of each subject's normoxic VO_2 max. Participants walked for 2 h and HR, T_{core} and RPE were recorded at rest and every 30 min. Oxygen uptake was measured at min 10 to verify the workload. The treadmill speed and grade remained constant for all 4 trials. Termination of the test occurred when the participant completed the 2 h of exercise. Water was allowed ad lib during the exercise bouts and the volume consumed was recorded. The subject's dry, semi-nude weight to the nearest 0.05 kg on a balance beam scale was recorded following exercise and whole body sweat rate (WBSR) was calculated after correcting for fluid consumption.

2.3. Statistics

Ending HR, T_{core} , RPE, and WBSR for the 4 trials were each analyzed using a 2×2 repeated measures ANOVA. Using the rationale previously outlined by Lloyd et al. (2016), main effects were used to examine the effect of heat and hypoxia alone on each variable, while the combined effect of both heat and hypoxia were categorized as either additive or synergistic via the interaction effect. The level of significance was set at $p < 0.05$.

3. Results

The mean \pm SE ending heart rates (bpm) for the four trials are shown in Fig. 1. There was a significant ($p < 0.05$) temperature main effect, and a non-significant inspired oxygen (i.e., hypoxia) main effect

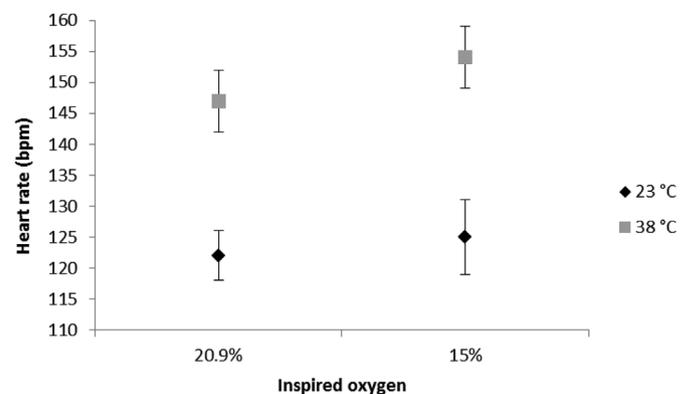


Fig. 1. Mean \pm SE ending heart rate (bpm) for the four trials. There was a significant ($p < 0.05$) temperature main effect, and a non-significant inspired oxygen main effect and non-significant interaction.

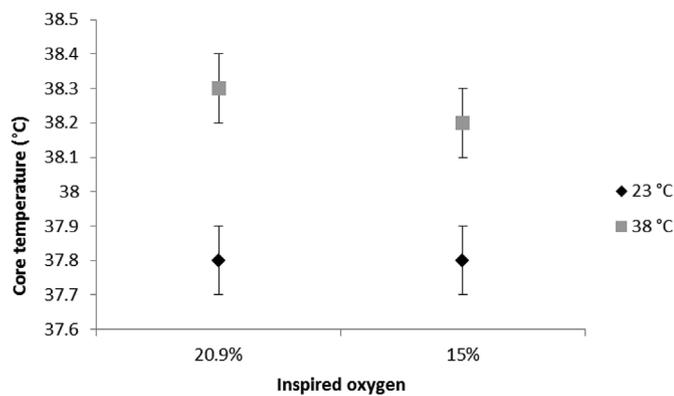


Fig. 2. Mean \pm SE ending core temperature ($^{\circ}$ C) for the four trials. There was a significant ($p < 0.05$) temperature main effect, and a non-significant inspired oxygen main effect and non-significant interaction.

and non-significant interaction. Specifically, during the heat trial, the mean HR increased 25 bpm compared to the control condition which had a mean HR of 122 bpm. Exercise in the hypoxic condition increased mean HR 3 bpm vs. control. HR in the combined heat and hypoxic trial increased 32 bpm compared to the control trial. These results suggest that the HR response in the combined heat and hypoxic environment was additive of the individual responses seen in the separate heat and hypoxic trials.

The mean ending T_{core} ($^{\circ}$ C) for the four trials are shown in Fig. 2. There was a significant ($p < 0.05$) temperature main effect, and a non-significant inspired oxygen (i.e., hypoxia) main effect and non-significant interaction. Specifically, during the heat trial, the mean ending T_{core} was increased 0.5 $^{\circ}$ C compared to the control condition (37.8 $^{\circ}$ C). Exercise in the hypoxic condition had no effect of mean T_{core} (37.8 $^{\circ}$ C). T_{core} in the combined heat and hypoxic trial increased 0.4 $^{\circ}$ C compared to the control trial. These results suggest that the T_{core} response in the combined heat and hypoxic environment was additive of the individual responses seen in the separate heat and hypoxia trials.

The mean ending RPE for the four trials are shown in Fig. 3. There was a significant ($p < 0.05$) temperature main effect, a significant interaction, and a non-significant inspired oxygen (i.e., hypoxia) main effect. Specifically, during the heat trial the mean ending RPE was increased 1.9 units compared to the control condition (10.5 units). Exercise in the hypoxic condition had no effect on mean RPE (10.4 units). However, RPE in the combined heat and hypoxic trial increased 3.3 units compared to the control trial. This would suggest that RPE during the combined heat and hypoxic environment increased in a synergistic manner.

Mean \pm SE WBSR was 0.39 ± 0.07 , 0.82 ± 0.11 , 0.34 ± 0.04 ,

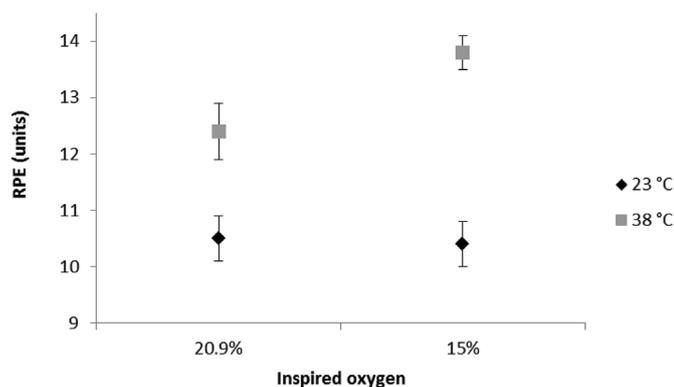


Fig. 3. Mean \pm SE ending RPE for the four trials. There was a significant ($p < 0.05$) temperature main effect and significant interaction, and a non-significant inspired oxygen main effect.

and 0.81 ± 0.09 L/hour for the control, heat, hypoxia, and combined heat and hypoxia condition, respectively. There was a significant temperature main effect and a non-significant hypoxia main effect and a non-significant interaction.

4. Discussion

Previous studies have clearly shown that exercise in the heat at a fixed work rate significantly increases the RPE compared to exercise in cool conditions (Galloway and Maughan, 1997; Jentjens et al., 2002; Pitsiladis et al., 2001; Smith et al., 1997). The results of the current study agree with past findings as the mean increase in RPE between the heat and control trials was 1.9 units (Fig. 3) and is consistent with what others have reported (Gonzalez-Alonso et al. 1998) during exercise in the heat.

The cause of the increase in RPE is believed to be related to elevations in HR and core temperature during heat stress. This is supported by the fact that RPE has been shown to be correlated with increases in body temperature and cardiovascular drift during both exercise hyperthermia (Galloway and Maughan, 1997) and passive heating (Gonzalez-Alonso et al. 1998).

Unlike heat, the effect of altitude/hypoxia on RPE during exercise is still in question and complicated by absolute vs. relative work rates (Horstman et al., 1979; Young et al., 1982). The general consensus is that moderate altitude (5000–8000 ft above sea level) has only minimal effects on RPE during low to moderate intensity exercise (Beidleman et al. 2008). This agrees with the current findings as RPE during moderate intensity exercise (40% of VO_2 max) was not different between the control and hypoxic trials.

The most important new finding of the current study was that RPE was synergistically increased during the combined heat and hypoxic trial as evidenced by a significant interaction. For this paper, the working definition of a synergistic response is when two individual effects combine to produce a net response greater than the sum of the individual responses. An additive response is when two individual effects produce a net response equal to the sum of the individual responses. As can be seen in Fig. 3, the increase in mean RPE in the combined heat and hypoxic trial (3.3 units) was much greater than the sum of the responses from the individual heat and hypoxia trials (1.9 units). Such results produced a significant interaction suggesting that combined heat and hypoxia synergistically increase RPE during exercise. It should be remembered that the RPE measurement used in the current study is a ratio scaled method which has been shown to increase according to a positively accelerating function with physical work (Borg, 1990), and it seems unlikely that the underlying psychophysical interval widths would get compressed with heat or hypoxia.

To our knowledge, this is the first time such findings have been reported in the scientific literature. RPE involves a variety of integrated sensory cues from peripheral muscles and joints, cardiorespiratory function and the central nervous system, which in the past have been believed to measure relative exercise intensity (Borg, 1990; Lloyd et al., 2016; Horstman et al., 1979). However, the results of the current study question this concept. As can be seen in Figs. 1 and 2, traditional markers of exercise intensity, namely heart rate and core temperature, increase in an additive manner during the combined heat and hypoxic trial, while RPE increases synergistically, as seen in Fig. 3. Such findings support the recent concept that RPE is a conscious perception of effort that plays a regulatory function to ensure that the work rate remains at an intensity that can be safely sustained, rather than simply a marker of exercise intensity (Tucker et al., 2006). It is well known that separately, heat and hypoxia both reduce exercise duration (Gonzalez-Alonso et al. 1998; Beidleman et al. 2008). Thus, the synergistic increase in RPE seen in the current study during the combined heat and hypoxic trial would suggest that exercise duration under such conditions would likewise be reduced. This is supported by the data of Girard and Racinais (2014) who reported that time to exhaustion while exercising at 66% of VO_2

max was reduced significantly more in a combined heat and hypoxic environment, compared to exercise in either condition alone. Specifically, mean time to exhaustion was 61 min in the control trial, 40 min in the heat (35 °C) trial, and 39 min in the hypoxic trial (15% O₂) and 30 min in the combined heat and hypoxic trial.

The physiological mechanisms responsible for the synergistic increase in RPE during the combined heat and hypoxic trial are currently unknown. However, at least two possibilities exist. First, it has previously been shown that increased body temperature increases the peripheral chemoreceptor sensitivity and thus the hypoxic ventilatory response during exercise (Chu et al., 2007). Specifically, Chu et al. reported that during moderate intensity exercise, hyperthermia in a normoxic environment increased minute ventilation by 2 L/min, compare to the normothermic condition. However, in a hypoxic environment (12% oxygen), the same degree of hyperthermia (~38.5 °C) caused exercise ventilation to increase by 10.2 L/min. Thus, hyperthermia and hypoxia caused a synergistic increase in minute ventilation during moderate intensity exercise. Furthermore, it has been shown that minute ventilation and RPE are significantly correlated during exercise (Harms et al., 2000). Thus, it could be hypothesized that increased minute ventilation during the combined heat and hypoxia trial may have caused the synergistic increase in RPE. Second, it has been reported that *in vitro*, both hypoxia and hyperthermia independently decrease intracellular pH. Furthermore when the two conditions were simultaneously applied the decrease in intracellular pH was disproportionately greater (Vexler and Litinskaya, 1986). Muscle and blood acidification has been implicated to stimulate nociceptors and chemoreceptors during exercise thus increasing RPE (Hampson et al., 2001). Certainly further work is warranted to determine the mechanisms for the synergistic increase in RPE during a combined heat and hypoxic environment.

5. Conclusion

In conclusion, the results of this study indicate that although HR and T_{core} showed only an additive increase when exercising in a combined heat and hypoxic environment, RPE increased synergistically. This suggests that RPE is a conscious perception of effort that plays a regulatory function to ensure that the work rate remains at an intensity that can be safely sustained, rather than simply a marker of exercise intensity. Such results also support previous anecdotal reports that exercise on hot days at altitude seem unusually difficult. Such information may aid recreational athletes, wildland firefighters, and military personnel who regularly find they are required to exercise in a combined heat and hypoxic environment.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jtherbio.2019.06.006>.

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