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Original Article

Effects of environmental temperature on physiological responses during submaximal and maximal exercises in soccer players

*MiHyun No, Hyo-Bum Kwak**

Department of Kinesiology, Inha University, Incheon, Republic of Korea

ARTICLE INFO

Article history:
Received 12 May 2016
Received in revised form
29 May 2016
Accepted 9 June 2016
Available online 16 June 2016

Keywords:
environmental temperature
heart rate
oxygen uptake
time to exhaustion
ventilation

ABSTRACT

Background: Although thermoregulation is effective in regulating body temperature under normal conditions, exercise or physical activity in extreme cold or heat exerts heavy stress on the mechanisms that regulate body temperature. The purpose of this study was to investigate the effects of environmental temperature on physiological responses and endurance exercise capacity during submaximal and maximal exercises in healthy adults.

Methods: Nine male soccer players participated in this study. In this study, three environmental temperatures were set at 10 ± 1°C, 22 ± 1°C, and 35 ± 1°C with the same humidity (60 ± 10%). The participants cycled for 20 minutes at 60% maximum oxygen uptake (60% VO_2max), and then exercise intensity was increased at a rate of 0.5 kp/2 min until exhaustion at three different environmental conditions.

Results: Oxygen uptake and heart rate were lower in a moderate environment (22 ± 1°C) than in a cool (10 ± 1°C) or hot (35 ± 1°C) environment at rest and during submaximal exercise, and were higher during maximal exercise ($p < 0.05$). Minute ventilation was lower at 22 ± 1°C than at 10 ± 1°C or 35 ± 1°C at rest and during submaximal exercise, and no significant differences were observed in minute ventilation during maximal exercise ($p < 0.05$). Blood lactate concentrations were lower at 22 ± 1°C than at 10 ± 1°C or 35 ± 1°C at rest and during submaximal exercise, and were higher during maximal exercise ($p < 0.05$). Time to exhaustion during exercise was longer at 22 ± 1°C than at 10 ± 1°C or 35 ± 1°C ($p < 0.05$).

Conclusion: It is concluded that physiological responses and endurance exercise capacity are impaired under cool or hot conditions compared with moderate conditions, suggesting that environmental temperature conditions play an important role for exercise performance.

* *Corresponding author*. Department of Kinesiology, Inha University, 100 Inha-ro, Nam-gu, Incheon 22212, South Korea.
E-mail address: kwakhb@inha.ac.kr (H.-B. Kwak).

http://dx.doi.org/10.1016/j.imr.2016.06.002

1. Introduction

The human body is physiologically regulated to keep it homeostatic when environmental conditions change. Humans produce or lose heat through thermoregulation to maintain the homeostasis of body temperature and protect themselves against excessive heat or cold.[1] In the same way, environmental temperature may affect physiological responses to exercise through thermoregulation. For example, our body minimizes heat dissipation by reducing body surface under cold conditions, promoting heat generation by skeletal muscle contraction (i.e., shivering), and preventing heat loss by contracting skin blood vessels (i.e., vasoconstriction).[2] By contrast, our body promotes heat dissipation by sweat evaporation through increased skin blood vessels (i.e., vasodilation) when exposed to heat.[3]

Exposure to cold causes various physiological responses in the human body. It has been reported that cold exposure results in increased heart rate and systolic blood pressure.[4,5] Cold-induced increase in heart rate may be associated with reduced vagal activation compared with sympathetic response to cold.[6] In addition, exposure to cold causes peripheral vasoconstriction leading to increase in systemic vascular resistance and diastolic blood pressure.[2,7] Various physiological responses are also caused by heat stress. Heat increases the heart rate by activating the sympathetic nervous system, but stroke volume is limited because of less return to the heart.[8] Moreover, dehydration caused by heat was associated with decrease in stroke volume, cardiac output, and blood pressure as well as decline in blood flow to the working skeletal muscles.[9]

Physical exercise is involved in skeletal muscle contraction and cardiovascular changes, which include increases in heart rate and systolic blood pressure by both the activation of sympathetic nervous system and the withdrawal of vagal control.[10] Also, dynamic exercise increases stroke volume and oxygen uptake[11,12] and increases blood flow to the skeletal muscle and skin, thereby promoting body heat loss.[3] However, exercise with prolonged and/or repeated cold or heat exposure may impair thermoregulatory control such as shivering and vasoconstriction responses to cold, and evaporation and vasodilation responses to heat.

Prolonged exercise with elevated body temperatures in the heat markedly influences exercise capacity. For example, there was a progressive impairment of marathon performance as the temperature increased from 5 °C to 25 °C.[13] Similarly, high-intensity running distance during a football game in the heat (approx. 43 °C) declined compared with moderate temperature condition (approx. 21 °C).[14] Thus, both environmental and exercise-induced heat stress may cause hyperthermia and increase core and brain temperature, resulting in impaired performance. However, it has been reported that solar loads were not associated with fast marathon performance.[15] Interestingly, impaired maximal oxygen uptake by hyperthermia-induced cardiovascular dysfunction seems to be associated with during high-intensity exercise,[16] whereas oxygen uptake capacity may be not affected during submaximal exercise.[17]

Similarly, exercise capacity and physiological responses are differentially affected by exercise in the cold. For example, Galloway and Maughan[18] reported that exercise time to fatigue during cycling was higher at 11 °C compared with that at 4 °C, 21 °C, or 31 °C. These results were not consistent with those of Parkin et al,[19] who showed that maximal time to exhaustion at the same intensity was produced after 3 °C, suggesting that environmental temperatures between 3 °C and 11 °C may be beneficial to maximal exercise performance. By contrast, it has been reported that exercise performance was negatively affected by cold ambient temperatures.[2] Moreover, Ball et al[20] showed that cold environmental temperatures leading to lowered skeletal muscle temperatures might be detrimental to exercise. Furthermore, it has been shown that maximum oxygen uptake remained unaffected by the cold environment, and there was no significant difference in aerobic capacity.[21,22]

Previous research regarding the effects of environmental temperature on physiological responses to exercise and endurance exercise capacity has produced inconsistent and conflicting results. Therefore, the purpose of this study was to verify the effects of environmental temperature on physiological responses and endurance exercise capacity during submaximal and maximal exercises in healthy adults.

2. Methods

2.1. Participants

Nine male varsity soccer players voluntarily participated in this study, none of whom had any history of diseases including cardiopulmonary disease. Informed consent was obtained from each participant prior to the experiment. They completed the Physical Activity Readiness Questionnaire and attended an orientation session during which the experimental protocol was explained. The principles outlined in the Declaration of Helsinki were followed in this study. The characteristics of participants are detailed in Table 1.

2.2. Experimental design

All participants were randomly assigned to three groups, and each group ($n = 3$) was set according to a randomized crossover design, as shown in Table 2. There were three environmental conditions: cool temperature (10 ± 1 °C), moderate temperature (22 ± 1 °C), and hot temperature (35 ± 1 °C); the same humidity (60 ± 10%) was used in each chamber. Each

Table 1 – Characteristics of participants

	Participants
n	9
Age (y)	18.56 ± 1.13
Height (cm)	176.89 ± 5.62
Weight (kg)	67.67 ± 5.48
BMI (kg/m^2)	21.38 ± 3.10
VO_2max (L/min)	3.23 ± 0.33
VO_2max (mL/kg/min)	48.20 ± 6.84

BMI, body mass index; VO_2max, maximum oxygen uptake.

Table 2 – Experimental design

Group ($n=9$)	Experimental order (A: 10 ± 1°C, B: 22 ± 1°C, C: 35 ± 1°C)		
Group I ($n=3$)	A	B	C
Group II ($n=3$)	B	C	A
Group III ($n=3$)	C	A	B

participant performed cycling trials at different temperature chambers. The interval of each trial was at least 1 week to avoid previous exercise and adaptation effects. On the day of the exercise testing trials, the participants arrived at the laboratory at the same time (11:00 AM) after at least a 3-hour fast and having refrained from any physical activity or intake of alcohol, tobacco, or caffeine 24 hours prior to exercise testing.

Prior to the experimental trials, all participants completed the pretest of maximal oxygen uptake (VO_2max) under normal laboratory ambient conditions to determine the exercise intensity (60% VO_2max) for submaximal exercise using Astrand protocol, which has long been considered the best measure of the capacity of the cardiovascular system and of calculating aerobic capacity in the cycle ergometer test, at which exercise started at 50 rpm and increased at a rate of 1 kp/2 min. The maximal exercise testing was terminated when one of the following conditions had occurred: (1) the participant could no longer keep pedaling at 50 rpm; (2) the respiratory exchange ratio had exceeded 1.20; (3) the oxygen consumption had plateaued even at an increased intensity; and (4) the heart rate was higher than the maximal target heart rate by age (HRmax = 220 – age).[23]

2.3. *Exercise protocol*

Bicycle exercise protocol and physiological variables to be measured are shown in Fig. 1. All participants performed the bicycle exercise for 20 minutes at 60% maximum oxygen uptake (60% VO_2max) after resting for 10 minutes. Then, the exercise intensity was increased at a rate of 0.5 kp/2 min until exhaustion with three different environmental conditions (10 ± 1 °C, 22 ± 1 °C, 35 ± 1 °C). During the bouts of bicycle ergometer exercise (Monark, Vansbro, Sweden), all participants were instructed to refrain from drinking water and eating food to avoid the effects of drinking and food. The physiological responses such as oxygen uptake (auto gas analyzer, Respina 1H 26; NEC San-Ei, Tokyo, Japan), heart rate (RS3000X, Kempele, Finland), minute ventilation (auto gas analyzer, Respina 1H 26, NEC San-Ei, Tokyo, Japan), blood lactate (lactate analyzer; Y.S.I., Yellow Springs, OH, USA), and time to exhaustion were measured during submaximal and maximal exercises. In particular, we measured oxygen uptake (VO_2) as a predictor of endurance performance including both absolute oxygen uptake (L/min) and relative oxygen uptake (mL/kg/min) because absolute oxygen uptake measures only the whole-body oxygen uptake whereas relative oxygen uptake normalizes whole body oxygen uptake by body weight considering the effect of body weight.

2.4. *Statistical analysis*

Oxygen uptake, heat rate, minute ventilation, blood lactate concentration, and time to exhaustion in three different conditions were analyzed with one-way repeated analysis of variance and Tukey *post hoc* test. Statistical significance was set at $p < 0.05$.

3. Results

3.1. *Effect of environmental temperature on oxygen uptake*

Both absolute oxygen uptake (L/min) and relative oxygen uptake (mL/kg/min) at 35 ± 1 °C during rest were significantly higher compared with those at 22 ± 1 °C ($p < 0.05$; Fig. 2). However, there was no significant difference at rest between 10 ± 1 °C and 22 ± 1 °C in terms of oxygen uptake, even though a high trend was shown at 10 ± 1 °C compared with 22 ± 1 °C. Similarly, oxygen uptake was higher at 35 ± 1 °C compared with 22 ± 1 °C at 5, 10, and 15 minutes during submaximal exercise. However, both absolute and relative maximal oxygen uptake (VO_2max) were significantly higher at 22 ± 1 °C compared with 10 ± 1 °C or 35 ± 1 °C ($p < 0.01$).

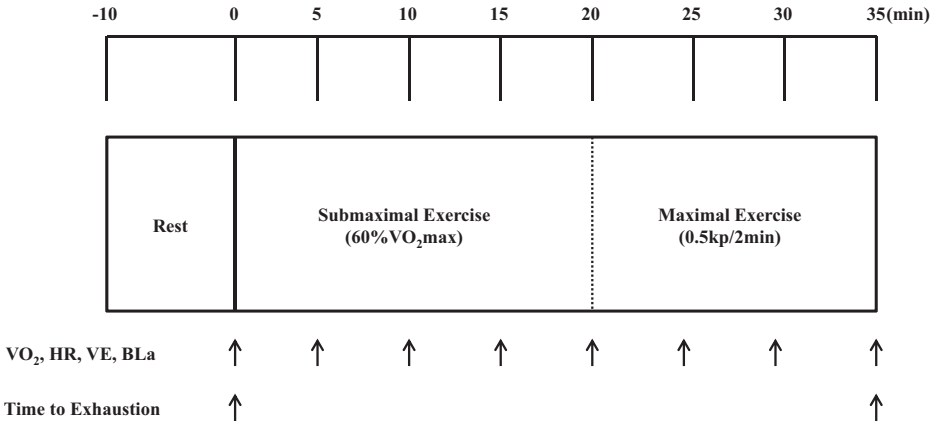

Fig. 1 – Exercise protocol and time schedule to measure physiological variables.
BLa, blood lactate concentration; Duration, time to exhaustion; HR, heart rate; VE, minute ventilation; VO_2, oxygen uptake; VO_2/kg, oxygen uptake/kg in body weight.

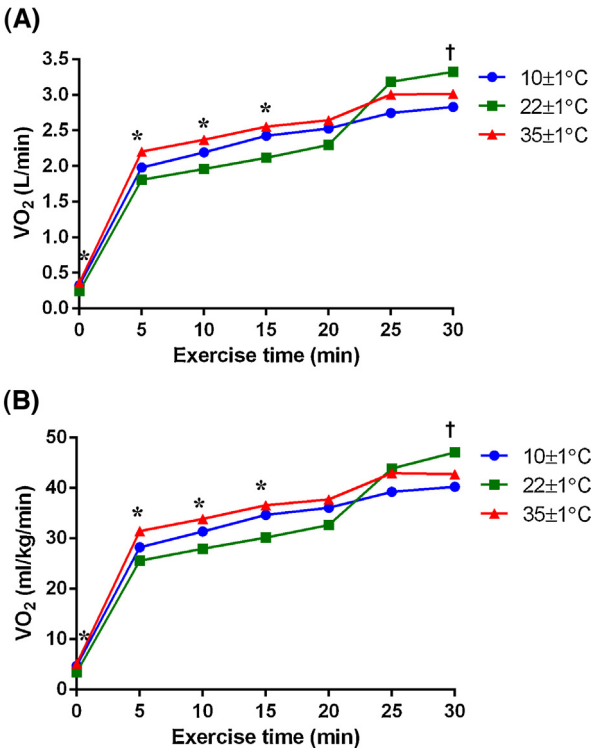

Fig. 2 – Effect of environmental temperature on oxygen uptake. (A) Absolute VO_2 during rest, submaximal, and maximal exercise. (B) Relative VO_2 during rest, submaximal, and maximal exercise.
***$p < 0.05$ versus $10 \pm 1°C$ or $35 \pm 1°C$.**
†$p < 0.01$ versus $10 \pm 1°C$ or $35 \pm 1°C$.
VO_2, oxygen uptake.

3.2. *Effect of environmental temperature on heart rate*

Heart rate at $22 \pm 1°C$ during rest were significantly lower compared with both $10 \pm 1°C$ and $35 \pm 1°C$ ($p < 0.01$; Fig. 3). Also, heart rate was significantly lower at $22 \pm 1°C$ compared with both $10 \pm 1°C$ and $35 \pm 1°C$ at 5 minutes, 10 minutes, and 15 minutes during submaximal exercise ($p < 0.05$). However, maximal heart rate during maximal exercise were

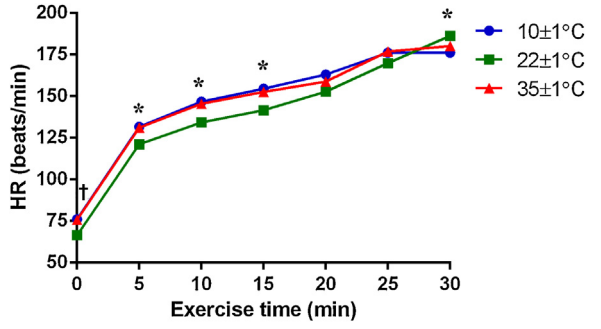

Fig. 3 – Effect of environmental temperature on heart rate during rest, submaximal, and maximal exercise.
***$p < 0.05$ versus $10 \pm 1°C$ or $35 \pm 1°C$.**
†$p < 0.01$ versus $10 \pm 1°C$ or $35 \pm 1°C$.
HR, heart rate.

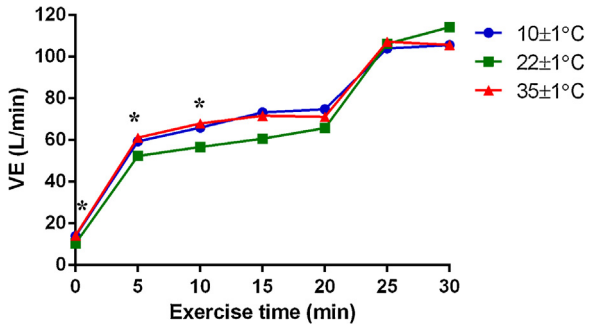

Fig. 4 – Effect of environmental temperature on minute ventilation during rest, submaximal, and maximal exercise.
***$p < 0.05$ versus $10 \pm 1°C$ or $35 \pm 1°C$.**
VE, minute ventilation.

significantly higher at $22 \pm 1°C$ compared with both $10 \pm 1°C$ and $35 \pm 1°C$ ($p < 0.05$).

3.3. *Effect of environmental temperature on minute ventilation*

Minute ventilation at $22 \pm 1°C$ during rest were significantly lower compared with both $10 \pm 1°C$ and $35 \pm 1°C$ ($p < 0.05$; Fig. 4). Also, minute ventilation was significantly lower at $22 \pm 1°C$ compared with both $10 \pm 1°C$ and $35 \pm 1°C$ at 5 and 10 minutes during submaximal exercise ($p < 0.05$). However, there were no significant differences in maximal minute ventilation among $10 \pm 1°C$, $22 \pm 1°C$, and $35 \pm 1°C$ conditions.

3.4. *Effect of environmental temperature on blood lactate concentration*

Blood lactate concentration at $22 \pm 1°C$ during rest were significantly lower compared with that at $10 \pm 1°C$ and $35 \pm 1°C$ ($p < 0.01$; Fig. 5). Similarly, blood lactate concentration was significantly lower at $22 \pm 1°C$ compared with that at $10 \pm 1°C$ and $35 \pm 1°C$ at 5 minutes and 10 minutes during submaximal exercise. However, maximal lactate concentrations in blood

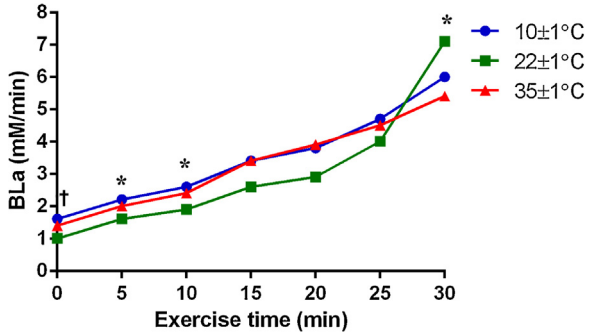

Fig. 5 – Effect of environmental temperature on blood lactate concentration during rest, submaximal, and maximal exercise.
***$p < 0.05$ versus $10 \pm 1°C$ or $35 \pm 1°C$.**
†$p < 0.01$ versus $10 \pm 1°C$ or $35 \pm 1°C$.
BLa, blood lactate concentration.

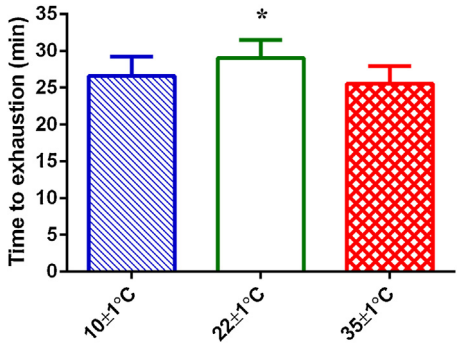

Fig. 6 – Effect of environmental temperature on time to exhaustion during exercise.
***p* < 0.05 versus 10 ± 1 °C or 35 ± 1 °C.**

were significantly higher at 22 ± 1 °C than at 10 ± 1 °C or 35 ± 1 °C ($p < 0.05$).

3.5. *Effect of environmental temperature on time to exhaustion*

In order to measure the effect of environmental condition on endurance exercise capacity, we measured the time to exhaustion during submaximal and maximal bicycle exercises. The time to exhaustion at 22 ± 1°C was significantly higher ($p < 0.05$) compared with that at 10 ± 1°C or 35 ± 1°C (10 ± 1°C, 26 minutes 59 seconds; 22 ± 1°C, 29 minutes 00 seconds; 35 ± 1°C, 25 minutes 50 seconds; Fig. 6).

4. Discussion

Our results demonstrate that aerobic exercise markers (e.g., VO_2, heart rate, ventilation) at 10 ± 1°C or 35 ± 1°C conditions were reduced during rest, submaximal exercise, and maximal exercise compared with those at 22 ± 1°C. In addition, the anaerobic exercise marker (e.g., blood lactate concentration) was decreased during rest, submaximal exercise, and maximal exercise at 10 ± 1°C or 35 ± 1°C compared with that at 22 ± 1°C. We likewise observed that the time to exhaustion during submaximal and maximal exercise was also reduced at 10 ± 1°C or 35 ± 1°C compared with that at 22 ± 1°C. Our results are consistent with the hypothesis that cool (10 ± 1°C) or hot (35 ± 1°C) environmental conditions negatively affect endurance exercise capacity compared with moderate (22 ± 1°C) conditions in humans.

It has been reported that endurance exercise capacity was affected by various physiological factors including red to white muscle fiber ratio, density of capillary vessel in skeletal muscles, muscle glycogen content, blood flow, oxygen transport ability, and mitochondrial density and activation.[24,25] Furthermore, as the present study showed, environmental temperature conditions may be an important factor to regulate endurance exercise capacity,[26] emphasizing the interaction between the body's mechanisms for heat balance and environmental conditions. During heat stress, vasodilation occurs in skin blood vessels so that the heat is lost from the skin and sweat glands become more active to increase evaporative heat loss.[3] In contrast, during cold stress, vasoconstriction occurs in skin blood vessels so less heat is lost to the environment and skeletal muscle shivering is activated to generate metabolism and heat.[2]

Oxygen uptake (VO_2) is determined by stroke volume, heart rate, and arteriovenous oxygen difference. When it is hot, blood pools in the skin vessels to dissipate more heat across the skin, which results in reduced venous return to the heart, and stroke volume decreases and heart rate increases to compensate for the lower stroke volume. These mechanisms to control cardiovascular responses in the heat are consistent with what we have observed in the present study, wherein VO_2 and heart rate at rest and during submaximal exercise under hot condition (35 ± 1°C) were higher compared with those under a more moderate condition (22 ± 1°C; Fig. 2, 3). However, VO_2max and maximal heart rate during maximal exercise were reduced at 35 ± 1°C compared with those at 22 ± 1°C because of the decrease in stroke volume, arteriovenous oxygen difference, or blood flow to the working muscles.[27,9] Furthermore, blood lactate concentration at rest and during submaximal exercise in the heat was increased compared with that under moderate condition (Fig. 5), indicating that muscle glycogen may be more utilized and depleted in the heat.[28] In addition, increased sweating in the heat during exercise may result in reduced blood volume and increased loss of minerals and electrolytes, leading to impaired endurance exercise capacity and reduced time to exhaustion during exercise (Fig. 6).

There is a growing body of evidence indicating that the effects of both hyperthermia in the heat and exercise stress may be associated with the central nervous system.[3] The central nervous system seems to be critical during exercise in hot environments. The combination of environmental and exercise-induced heat stress may cause severe hyperthermia and elevate brain temperature, leading to impaired exercise capacity. For example, it was reported that the human brain temperature during prolonged exercise was much higher compared with the core temperature.[29] In addition, Nybo and Nielsen[30] showed that cerebral artery blood velocity during prolonged exercise was significantly reduced with hyperthermia, suggesting that the brain may play an important role in the control of fatigue in the heat. Furthermore, Watson et al[31] showed that exercise performance in the heat was improved by the administration of bupropion, a dopamine receptor agonist.

The primary human physiological responses during cold exposure are peripheral vasoconstriction and shivering. Peripheral vasoconstriction improves thermal insulation of the body and inhibits heat loss, whereas shivering increases thermogenesis to replace body heat loss due to the cold.[1,2] However, prolonged cold exposure and exercise in the cold can impair peripheral vasoconstriction and shivering, leading to increased fatigue and reduced exercise capacity. For example, exercising in cold condition increased muscle fatigue and injury markers (e.g., creatine kinase, lactate dehydrogenase) compared with exercising in warm condition.[32] In addition, exercise-induced free fatty acid mobilization may be impaired because of the vasoconstriction of subcutaneous blood vessels. For example, Layden et al[33] showed that cold exposure reduced fat utilization as evidenced by lower blood glycerol

compared with moderate temperature during submaximal exercise, which is consistent with a previous study that noted that cold-induced reduction in blood flow to subcutaneous adipose tissue might be associated with the impairment of lipid mobilization.[34] In addition, Lloyd et al[35] showed that exposure to cold (5 °C) significantly reduced time to exhaustion of knee extension exercise compared with moderate temperature (23 °C), which is consistent with the present findings. Furthermore, physical performance was lower in cool room temperature (15 °C) compared with moderate temperature (25 °C) for leg extensor power, sit-to-stand performance velocity, and gait speed in old adults.[36] However, the mechanisms by which the thermoregulaton is impaired during exercise in the cold are not clear. Future research addressing the mechanisms associated with exercise in hypothermia is therefore warranted.

In conclusion, physiological responses (e.g., VO_2, heart rate, ventilation, blood lactate concentration) and endurance exercise capacity during rest, submaximal exercise, and maximal exercise were impaired under cold or hot temperature conditions compared with those under moderate conditions, suggesting that environmental temperature may play an important role for exercise performance.

Conflicts of interest

All authors have no conflicts of interest to declare.

Acknowledgments

This work was supported by Inha University Research Grant.

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