

Gradual reduction in ambient temperature of about 5°C does not influence physiological and hormonal response to prolonged exercise

Ogaki, Tetsuro
Institute of Health Science, Kyushu University

Fujishima, Kazutaka
Institute of Health Science, Kyushu University

Hotta, Noboru
Institute of Health Science, Kyushu University

Saito, Atsushi
Institute of Health Science, Kyushu University

他

<https://doi.org/10.15017/10767>

出版情報：健康科学. 28, pp.1-6, 2006-03-25. 九州大学健康科学センター
バージョン：
権利関係：



ORIGINAL

Gradual reduction in ambient temperature of about 5 °C does not influence physiological and hormonal response to prolonged exercise

Tetsuro OGAKI ^{1)*}, Kazutaka FUJISHIMA ¹⁾, Noboru HOTTA ¹⁾,
Atsushi SAITO ¹⁾ and Takashi MIGITA ²⁾

Abstract

The purpose of this study was to determine whether or not a gradual reduction in ambient temperature affected core temperature or physiological and hormonal responses during prolonged exercise in a neutral thermal condition. On two occasions, in random order and separated by 1 week, six male distance runners [mean age: 20 ± 1 years; maximal oxygen uptake ($\dot{V}O_{2max}$): 63.8 ± 5.0 ml/kg/min; mean ± SD] exercised for 60 min on a cycle ergometer at 67% $\dot{V}O_{2max}$ in an environmental chamber, on one occasion at a gradually lowered ambient temperature (DEC-T: ambient temperature lowered during exercise by 0.3°C every 3 min from 23°C at rest) and another occasion at a constant ambient temperature of 23 ± 0.2°C (CONT). Subjects were given 100 ml of water every 15 min during exercise. Rectal temperature and heart rate were recorded continuously throughout the entire test period. Expired gas samples were collected for 10 min before exercise, for the last 5 min of every 10 min during exercise, and at 30 min and 60 min after exercise. Blood samples were collected before exercise, at 10 min intervals during exercise, and 30 min and 60 min after exercise. Exercise intensity was similar for both trials (DEC-T: 67.9 ± 2.9% $\dot{V}O_{2max}$, CONT: 67.3 ± 3.0% $\dot{V}O_{2max}$). Ambient temperature on the DEC-T trial was significantly lower than that of the CONT trial after 17 min of exercise ($p < 0.05$), and reached 17.9 ± 1.6 °C at the end of exercise (CONT trial: 23.0 ± 1.1°C, $p < 0.01$). Rectal temperature was not significantly different for the trials at pre-exercise, during exercise and recovery (at end of exercise; DEC-T: 38.98 ± 0.48°C, CONT: 39.20 ± 0.28°C). Body mass losses showed no significant differences between trials (DEC-T: -1.2 ± 0.4 kg, CONT: -1.2 ± 0.3 kg). Whole-period minute ventilation, heart rate and respiratory exchange ratio also showed no significant differences between the two test periods. Concentrations of plasma-free and sulpho-conjugated catecholamines, serum cortisol, growth hormone, glycerol and free fatty acid, and blood lactate were similar for both trials at all sampling points. These results show that a gradual reduction of ambient temperature of about 5°C does not influence core temperature or physiological and hormonal responses during prolonged exercise. Additionally, these data suggest that physiological and hormonal responses during prolonged exercise might be strongly influenced by core temperature.

Key Words: rectal temperature, cardiorespiration, catecholamine, growth hormone, cortisol, metabolites.

(Journal of Health Science, Kyushu University, 28: 1- 6, 2006)

1) Institute of Health Science, Kyushu University

2) Institute of Health and Sports Science, Kurume University

*Correspondence to: Institute of Health Science, Kyushu University, 6-1 Kasuga-koen, Kasuga, 816-8580, Japan

Tel/Fax: +81-92-583-7851 e-mail: ogaki@ihs.kyushu-u.ac.jp

Introduction

It is well known that prolonged exercise in hot conditions elevates core temperature, which results in a decrease in physiological responses and performance, compared with prolonged exercise in a neutral thermal condition. Nielsen¹⁾ found that the adjustment of the body temperature was dependent on the work load, but independent of the environmental temperature (5- 30°C) and duration of exercise. Davis²⁾ reported that even at exercise intensities up to 65% of maximal oxygen uptake ($\dot{V}O_{2max}$), the core temperature was largely independent of dry-bulb temperature within the range 5 to 20°C. Berggren and Christensen³⁾ suggested that the body temperature was directly correlated to the metabolic rate of the body.

During prolonged exercise in a neutral thermal environment, a progressive, time-dependent, tachypneic hyperventilation and hypocapnia and a continuous rise in heart rate and splanchnic vasoconstriction take place^{1,2,3,4)}. This ventilatory and cardiovascular "drift" is accompanied by and is probably – at least in part – mediated by gradually rising body temperatures^{4,5)}. Also the hormonal response to exercise – i.e. an increase in the concentrations in plasma of catecholamines, glucagon, growth hormone and cortisol and a decrease in insulin concentrations – is accentuated as the duration of exercise increases^{5) 6)}.

Generally, most endurance races are carried out under natural environmental conditions. This may not necessarily mean that environment temperature is constant during the race; rather, the temperature may decrease or increase. Most of the above stated studies, however, have been conducted in a steady-state condition of ambient temperature. To our knowledge, no study has examined the effects of lowered ambient temperature during exercise on the physiological and/or biological responses to prolonged exercise. Therefore, the purpose of this study was to determine whether or not gradually lowered ambient temperature could affect core temperature or metabolic and hormonal responses during prolonged exercise.

Methods

Subjects

Six male university distance runners participated in this study after giving their informed written consent, which was approved by Ethical Committee of the Institute of Health Science, Kyushu University. The mean age, height, body mass and percentage body fat of the subject were 20 ± 1 (SD) years,

167.4 ± 1.4 cm, 58.1 ± 2.7 kg and $11.0 \pm 1.4\%$, respectively.

The mean $\dot{V}O_{2max}$ was 3.70 ± 2.13 l/min, or 63.8 ± 5.0 ml/kg/min. All the subjects were non-smokers and familiar with the experiment and experimental apparatus used in all the metabolic measurements. None of the subjects were taking any medication for at least 6 weeks or had any history of metabolic or circulatory disease.

Experimental protocol

All the subjects participated in an exhaustion test and two steady-state exercise trials by cycle ergometer. After familiarization with the ergometer and experimental procedures, the subjects undertook an exhaustion test to determine the relationship between workload and oxygen uptake ($\dot{V}O_2$), and to measure $\dot{V}O_{2max}$. The exhaustion test consisted of three stages of steady-state exercise and incremental exercise to exhaustion on a cycle ergometer (Bodyguard-990; Jonas Øglaend A.S., Norway). After a warm-up period, the test was started at an exercise intensity of 75 - 100 W with a constant pedaling rate (50 rpm) for 4 min and then was increased by 50 W every 4 min for 8 min. Thereafter, the exercise intensity was increased by 12.5 - 25 W every minute until the subject reached a state of exhaustion.

On two occasions, in random order and separated by 1 week, the subjects exercised for 60 min on a cycle ergometer at $70\% \dot{V}O_{2max}$ in an environmental chamber. The two trials consisted of exercise at constant room temperature (23 ± 0.2 °C; CONT) and at room temperature gradually lowered by 0.3 °C every 3 min from 23°C at rest (DEC-T). The subjects reported to the laboratory at 9 a.m., after an overnight fast of at least 10 h. They were transported by car or motorbike to minimize the influence of physical activity on the morning of the trial. They were told not to take part in any exercise training for 1 day before the trial and not to make any changes in their lifestyle or dietary habits.

The subject emptied his bladder before his body weight was obtained and then a rectal probe was inserted to a depth of about 15 cm. An indwelling catheter was inserted into an antecubital vein and kept patent with a dilute heparin-saline solution (10 U/ml). Thereafter, the subjects sat quietly in an armchair for 60 min. The catheter was kept in place until the trial was finished. The exercise trial began at 10 a.m. The subjects were given 100 ml of water (30 °C) every 15 min during exercise to avoid dehydration. After exercise, the subjects rested quietly in an armchair at a 23°C room temperature for 60 min.

Measurements and analytical procedure

Both the electrocardiograph and heart rate (HR) were monitored continuously by a telemetric monitoring system (DS-882, Fukuda Denshi Co., Japan) throughout the entire test period. The rectal temperature (Rec-T) was also recorded every minute throughout the entire test period using a portable data-recording machine (VMM-67, Vine Co., Japan). Expired gas samples (60 s) were analyzed continuously using a mass-spectra gas analyser (WSMR-1400, Westron Co., Japan) and an automatic respiratory analyser (RM-300i, Minato Ikagaku Co., Japan) during the 30-min rest period before exercise began, and for the last 5 min of every 10 min of exercise. Expired gas samples were also collected at 25-35 and 55-65 min during recovery periods. These analyzers were calibrated before each measurement. Systolic and diastolic blood pressures were measured by auscultatory method at rest, at every 10 min during exercise, and at 30 and 60 min after the cessation of exercise.

A blood sample was collected before exercise after 30 min of rest following the insertion of the catheter, at every 10 min of exercise, and at 30 and 60 min after the cessation of exercise. Each venous blood sample was immediately dispensed into two ice-chilled different tubes. After mixing the blood by gentle inversion and storage on ice, plasma was separated from blood cells by centrifugation (3,000 g) at 4 °C for 10 min. The plasma was then frozen and stored at -80 °C until assayed. The concentrations of plasma-free catecholamine and sulfated catecholamine (HPLC), serum cortisol (radioimmunoassay), growth hormone (immunoradiometric assay), glycerol, blood lactate (enzymatic analysis), free fatty acid (enzymatic analysis), and total protein (STP; Biuret method) were determined by standard techniques at Kitazato Biochemical Laboratory (Sagamihara, Japan). The STP concentration of each subject at any sampling point was used to eliminate the effect of plasma water loss from the determined value of these analyses as previously described⁷.

Data analysis

Any statistical differences were tested using analysis of variance (ANOVA) and post hoc Fisher's PLSD test for multiple comparisons. Significance was set at the 0.05 level of confidence. All results are presented as means \pm SD unless otherwise stated.

Results

The $\dot{V}O_2$ value of each subject was constant throughout exercise for 60 min. The mean values of the $\dot{V}O_2$ were 46.7 ± 2.6 ml/kg/min during the DEC-T trial and 46.3 ± 3.1 ml/kg/min during CONT trial. Therefore, exercise intensity was similar for both trials (DEC-T: $67.9 \pm 2.9\%$ $\dot{V}O_{2max}$, CONT: $67.3 \pm 3.0\%$ $\dot{V}O_{2max}$). Body mass at after the two trials was 56.0 ± 3.3 kg in DEC-T trial and 56.1 ± 3.1 kg in CONT trial, respectively. Body mass losses were not significantly different between the trials (DEC-T: -1.2 ± 0.4 kg, CONT: -1.2 ± 0.3 kg).

Ambient room temperature and Rec-T in the pre-exercise, during-exercise and during-recovery-from-exercise are shown in Fig.1. Ambient temperature for the DEC-T trial was significantly lower than that of the CONT trial after 17 min of exercise ($p < 0.05$), and reached $17.9 \pm 1.6^\circ\text{C}$ at the end of exercise (CONT trial: $23.0 \pm 1.1^\circ\text{C}$, $p < 0.01$). Rec-T was not significantly different for the two trials at pre-exercise, during exercise (at end of exercise; DEC-T: $38.98 \pm 0.48^\circ\text{C}$, CONT: $39.20 \pm 0.28^\circ\text{C}$) or recovery. The relative humidity was $56 \pm 5.5\%$ at pre-exercise in both trials, and $56 \pm 3.5\%$ in CONT trial and $66.0 \pm 9.0\%$ in DEC-T trial at end of exercise.

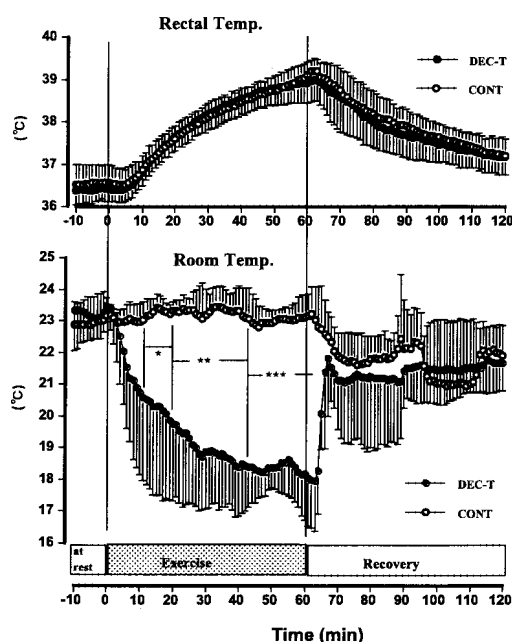


Figure 1. Ambient room temperature and rectal temperature in DEC-T trial and in CONT trial. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$: Significantly different from CONT trial. Values are means \pm SD for 6 subjects.

As is clear in Fig.2, the mean HR and blood pressure in the two trials were similar. The whole-period mean minute ventilation and respiratory exchange ratio also showed no significant differences between the two test periods (data not shown).

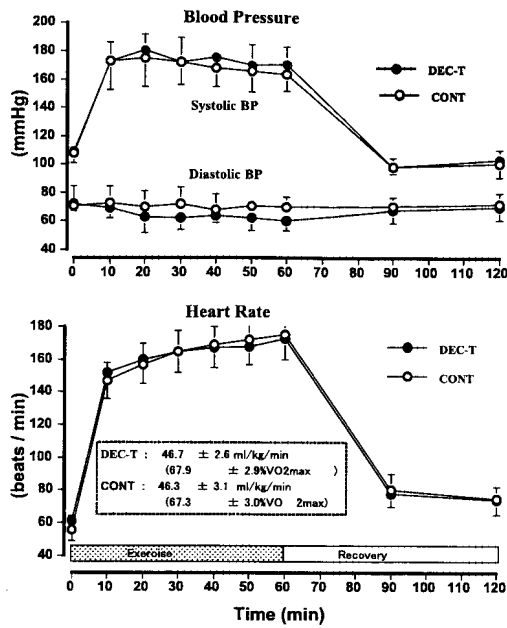


Figure 2. Heart rate, and systolic and diastolic blood pressure in DEC-T trial and CONT trial. Values are means \pm SD for 6 subjects.

The plasma-free CA and CA-S concentrations at the pre-exercise, during-exercise and recovery-from-exercise stages are shown in Fig.3. No significant differences were

observed between the two trials at any sampling points. The concentrations of serum cortisol, growth hormone, glycerol, blood lactate, free fatty acid and STP in the two trials are shown in Table 1. There were no significant differences between the two trials in these parameters at any sampling point.

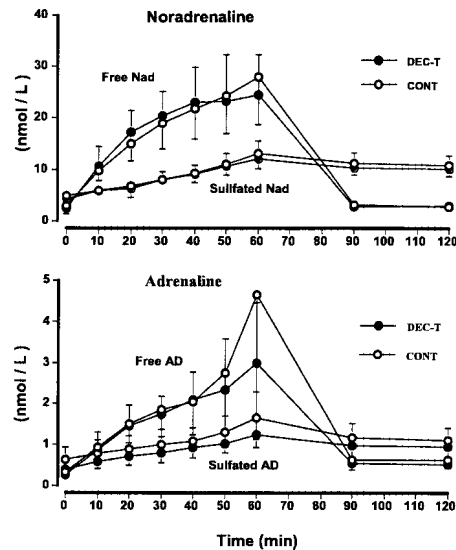


Figure 3. Plasma free and conjugated adrenaline and noradrenaline in DEC-T trial and in CONT trial. Values are means \pm SD for 6 subjects.

Table 1. Concentration of serum growth hormone, cortisol, lactate, glycerol, free fatty acid total protein in DEC-T trial and in CONT trial. Values are means \pm SD for 6 subjects.

	Growth Hormon (ng/ml)	Cortisol (μ g/dl)	Lactate (mg/dl)	Glycerol (mg/dl)	Free Fatty Acid (mEq/l)	Total Pro-tein (g/dl)
At rest						
DEC-T	9.34 \pm 7.67	14.5 \pm 5.6	7 \pm 1	1.2 \pm 0.5	0.45 \pm 0.22	7.2 \pm 0.6
CONT	7.89 \pm 7.37	13.2 \pm 5.5	7 \pm 2	1.2 \pm 0.4	0.62 \pm 0.34	7.2 \pm 0.3
Ex 10 min						
DEC-T			24 \pm 10	1.6 \pm 0.7	0.30 \pm 0.14	7.9 \pm 0.4
CONT			19 \pm 4	1.6 \pm 0.5	0.43 \pm 0.25	7.9 \pm 0.4
Ex 20 min						
DEC-T	28.24 \pm 15.15	14.5 \pm 7.1	31 \pm 14	2.3 \pm 0.7	0.48 \pm 0.20	8.0 \pm 0.4
CONT	20.38 \pm 12.59	15.0 \pm 6.1	23 \pm 8	2.7 \pm 0.7	0.61 \pm 0.20	8.0 \pm 0.4
Ex 30 min						
DEC-T			32 \pm 13	2.7 \pm 0.6	0.55 \pm 0.21	8.0 \pm 0.3
CONT			23 \pm 8	3.7 \pm 1.2	0.81 \pm 0.43	8.0 \pm 0.3
Ex 40 min						
DEC-T	34.56 \pm 8.64	17.6 \pm 7.1	32 \pm 13	3.1 \pm 0.6	0.63 \pm 0.22	8.1 \pm 0.5
CONT	35.56 \pm 17.66	17.4 \pm 6.9	26 \pm 11	4.0 \pm 0.7	0.80 \pm 0.25	8.1 \pm 0.4
Ex 50 min						
DEC-T			28 \pm 12	3.5 \pm 0.8	0.65 \pm 0.17	8.0 \pm 0.4
CONT			25 \pm 13	4.3 \pm 0.9	0.81 \pm 0.31	8.0 \pm 0.4
Ex 60 min						
DEC-T	31.40 \pm 9.45	23.1 \pm 8.5	30 \pm 12	4.1 \pm 0.7	0.79 \pm 0.18	8.2 \pm 0.4
CONT	34.32 \pm 21.53	24.0 \pm 6.7	28 \pm 12	4.6 \pm 1.0	0.81 \pm 0.36	8.1 \pm 0.4
Rec 30 min						
DEC-T	8.43 \pm 2.65	20.7 \pm 8.8		2.1 \pm 1.7	0.78 \pm 0.16	7.1 \pm 0.4
CONT	11.98 \pm 8.52	22.7 \pm 7.5		2.3 \pm 0.9	1.09 \pm 0.71	7.1 \pm 0.3
Rec 60 min						
DEC-T	3.20 \pm 1.13	17.2 \pm 5.5		1.3 \pm 0.4	0.76 \pm 0.19	7.2 \pm 0.4
CONT	4.42 \pm 3.30	17.9 \pm 5.7		1.5 \pm 0.8	0.92 \pm 0.69	7.2 \pm 0.4

Ex: exercise, Rec: recovery from exercise

Discussion

The main finding of this study was that no significant differences exist for core temperature or any physiological parameters, hormones and metabolites between a constant ambient temperature condition and gradually lowered condition of room temperature. Since body mass losses and concentration of serum STP were similar in the two trials, it is likely the hydration status of subjects also showed no differences in the two trials.

In 1938 Nielsen¹⁾ put forward the idea that the magnitude of the body core temperature rise in steady-state exercise was independent of the environment. He came to this conclusion after studying one subject performing exercise at a variety of intensities under environmental conditions ranging from 5 to 36°C and low humidity. These findings were extended and substantially supported by the work of Robinson et al.⁸⁾ and Lind⁹⁾. Lind⁹⁾ tried to examine the relationship between environmental conditions and work rates so that safe limits for work environments and work rates (as opposed to recreation) might be construed. He proposed the so-called prescriptive zone¹⁰⁾. Åstrand¹¹⁾ was the first to report the importance of relative exercise intensity rather than absolute metabolic rate on the rise in body core temperature during exercise, and Saltin and Hermansen¹²⁾ extended these findings. Further clarification of the relationship between exercise intensity and ambient conditions was provided in the study by Davis²⁾. Over a wide range of environmental conditions, with dry-bulb temperatures from 5 to 25°C and relatively low humidity, he examined subjects exercising at between 20% and 90% $\dot{V}O_{2max}$ and demonstrated a curvilinear relationship between steady-state core temperature and relative intensity. Even at exercise intensities up to 65% $\dot{V}O_{2max}$, the core temperature was largely independent of dry-bulb temperature within the range of 5 to 20°C²⁾. In this study, the dynamics and values of core temperature, heart rate, blood pressure and other physiological parameters in the DEC-T trial were similar to those of the CONT trial regardless of the sampling point. This is in line with the findings of previous studies¹⁾²⁾³⁾⁸⁾⁹⁾. Therefore, our results support the view that body temperature is correlated to work rate⁸⁾¹²⁾ or the metabolic rate of the body³⁾¹³⁾ and is independent of ambient temperature within 5 to 20°C²⁾ or within 10 to 25°C⁹⁾.

The responses of hormones and metabolites to prolonged exercise have been shown to be influenced not only by the intensity and duration of exercise but also by the ambient temperature⁵⁾⁶⁾¹⁴⁾. It was reported that the concentrations of

plasma or serum hormones and metabolites during exercise vary in extremely hot or cold environments compared with the thermoneutral condition⁵⁾⁶⁾¹⁴⁾. Therefore, the possibility exists that an exercise-induced increase in body temperature enhances the hormonal responses to exercise and metabolites⁵⁾⁶⁾. The present study showed no significant difference in rectal temperatures between the two trials; however, no significant differences were observed in the concentrations of plasma catecholamines, and serum cortisol, growth hormone, glycerol, blood lactate, free fatty acid and STP between the two trials at any sampling points, either. These results show that the gradually lowered ambient temperature, at least about 5°C decrease, does not influence hormonal responses and metabolites during prolonged exercise. Moreover, these data suggest that physiological and hormonal responses during prolonged exercise might be influenced strongly by core temperature or metabolic rate in the thermoneutral condition.

Incidentally, it is well known that marked elevations in body temperature during exercise reduce the exercise performance. Gonzalez-Alonso et al.¹⁵⁾ demonstrated that high internal body temperature per se causes fatigue during exercise and time to exhaustion was inversely related to the initial temperature and directly related to the rate of heat storage. MacDougal et al.¹⁶⁾ previously showed that subjects always became exhausted at a rectal temperature of 39.4°C. Although our study was conducted in the thermoneutral conditions, the rectal temperature reached $38.98 \pm 0.48^\circ\text{C}$ in DEC-T trial and $39.20 \pm 0.28^\circ\text{C}$ in CONT trial at end of exercise. Our result showed that there is no influence in the core temperature if ambient temperature during exercise is reduced by about 5 °C. Therefore, in order to decrease body temperature during exercise, it may be necessary to depend on physical means, such as drinking a cold water, cooling using water, a wet towel or a wet sponge etc.

In conclusion, a gradual reduction of ambient temperature of about 5°C does not influence core temperature or physiological and hormonal responses during prolonged exercise. Moreover, these data suggest that physiological and hormonal responses during prolonged exercise might be influenced not only by exercise intensity but also strongly by core temperature.

Acknowledgements

This work was supported in part by a grant-in-aid for Scientific Research (C) (No. 08680126) from the Ministry of Education, Science, Sports and Culture of Japan. This

study was presented at the 10th annual meeting of the East Asian Sports and Exercise Science Society, Shanghai China, 2005 (abstract 49-50).

References

- 1) Nielsen M (1938): Die regulation der korpe-temperataur bei muskelarbeit. *Skand Arch Physiol*, 79: 193-230.
- 2) Davis CTM (1979): Thermoregulation during exercise in relation to sex and age. *Eur J Appl Physiol*, 42: 71-79.
- 3) Berggren G, Christensen EH (1950): Heart rate and body temperature as indices of metabolic rate during work, *Arbeitsphysiologie*, 14: 255-260.
- 4) Rowell LB (1974): Human cardiovascular adjustments to exercise and thermal stress. *Physiol Rev*, 54: 75-159.
- 5) Galbo H, Houston ME, Christensen NJ, Holst JJ, Nielsen B, Nygaard E, Suzuki J (1979): The effect of water temperature on the hormonal response to prolonged swimming. *Acta Physiol Scand*, 105: 326-337.
- 6) Galbo H (1983): Hormonal and metabolic adaptation to exercise. *George Thieme Verlag, Struttgart*, pp.2-61.
- 7) Ohira Y, Ito A, Ikawa S (1977): Correlation of water content and solute concentration in blood during hemoconcentration. *J Appl Physiol: Respirat Environ Exercise Physiol*, 42: 739-743.
- 8) Robinson S, Dill DB, Wilson JW, Nielsen M (1941): Adaptation of white men and Negroes to prolonged work in humid heat. *Am J Trop Med*, 21: 261-287.
- 9) Lind AR (1963): A physiological criterion for setting thermal environmental limits for everyday work. *J Appl Physiol*, 18: 51-56.
- 10) Sutton JR (1994): Physiological and clinical consequences of exercise in heat and humidity. In Harries M, Williams C, Stanish WD, Micheli LJ (eds), *Oxford textbook of sports medicine*. Oxford University Press, Oxford, pp.231-238.
- 11) Åstrand I (1960): Aerobic work capacity in men and women. *Acta Physiol Scand*, 49 (suppl. B): 64-73.
- 12) Saltin B, Hermansen L (1966): Esophageal, rectal and muscle temperature during exercise. *J Appl Physiol*, 21: 1757-1762.
- 13) Sawka MN, Pimetal NA, Pandolf KB (1984): Thermoregulatory responses to upper body exercise. *Eur J Appl Physiol*, 52: 230-234.
- 14) Ogaki T, Hotta N, Kanaya S, Fujishima K, Shimizu T, Shono T (1995): Hormonal and metanolic responses to low intensity prolonged swimming at three water temperatures. *Japan J Phys Educ*, 40: 80-88. (In Japanese)
- 15) Gonzalez-Alonso J, Teller C, Andersen S, Jensen FB, Hyldig T, Nielsen B (1999): Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol*, 86: 1032-1039.
- 16) MacDougal JD, Reddan WG, Layton CR, Dempsey JA (1974): Effects of metabolic hyperthermia on performance during heavy prolonged exercise. *J Appl Physiol*, 36: 538-544.