

The elevation training mask induces modest hypoxaemia but does not affect heart rate variability during cycling in healthy adults

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ABSTRACT: This study examined the acute effects of the elevation training mask (ETM) on haemodynamics and heart rate variability (HRV) at rest, during cycling, and during recovery in healthy adults. Fifteen healthy male (N=9) and female (N=6) adults (27.0 ± 1.14 years) completed two trials with the mask (MASK) and without the mask (CON). The 40-minute cycling exercise protocol included 10-minute phases of (1) rest, (2) 50% of VO_{2peak} cycling, (3) 70% of VO_{2peak} cycling, and (4) recovery. Blood pressure and pulse oximetry saturation (S_pO_2) were measured at each phase. An Actiwave-Cardio ECG monitor (CamNtech, UK) was used to measure HRV variables including time and frequency domains. A greater response in systolic blood pressure ($p=.035$) was observed at rest while S_pO_2 ($p=.033$) was lower during high-intensity cycling (70% of VO_{2peak}) in the MASK trial. The HRV indices were not different between trials during cycling. However, heart rate ($p=.047$) was greater while inter-beat interval and sympathovagal balance (the ratio between low-frequency and high-frequency components; $\ln LF/HF$, $p=.01$) were lower in the MASK than the CON trials during recovery. Wearing an ETM during high-intensity cycling (70% of VO_{2peak}) induces modest hypoxaemia. Although this device did not affect HRV changes during cycling, it seems to delay the cardiac-autonomic recovery from exercise. Healthy adults may be required to perform high-intensity exercise with an ETM to simulate a hypoxic environment, but future studies are needed to determine whether repeated exposure to this condition provides similar benefits as altitude training.

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INTRODUCTION

Inspiratory muscle weakness is associated with slow oxygen uptake kinetic, lower limb muscle weakness, and exercise intolerance [1]. It has also been identified as a limiting factor for exercise performance in patients and sedentary individuals [2,3]. Respiratory muscle training (RMT) has been introduced as a clinical approach for those with chronic heart failure or cardiopulmonary disease, as well as the elderly, to improve their inspiratory muscle strength, exercise tolerance, and quality of life [4,5]. RMT is designed to reduce perceived breath effort and breath work, which results in the delay of respiratory muscle fatigue and attenuation of the respiratory muscle metaboreflex [6]. RMT is known to provide health benefits for haemodynamic and autonomic regulation, which may result in decreasing resting sympathetic activity in addition to baroreceptor sensitivity and sympathovagal balance improvements [7].

Traditionally, RMT is performed at rest and mainly applied to patients for improvements in disease outcomes or quality of life [4,5], but in recent years exercise scientists have begun to consider the role of RMT during exercise and its value in providing a synergetic benefit with improvements in health and performance. The Elevation training mask (ETM 2.0, Training Mask LLC, Cadillac, Michigan, USA) was introduced in the commercial fitness market and is purportedly designed for the simulation of altitude training. The adjustable resistance cap provides different inspiratory resistance loads that are claimed as “altitude resistance” which ranges from 914 m to 5,486 m. A previous study reported that additional RMT training (2 times/week) combined with conventional training improved rowing performance in female competitive rowers, both in a 6 min all-out and a 5,000 m trial [8]. Even though RTM combined with training

showed performance improvement, the different equipment (i.e., manufacture or model) may have caused different outcomes in performance improvement. Recently, two studies that utilized the ETM 2.0 device during high-intensity cycling exercise or physical training did not show aerobic performance improvement in healthy adults and Reserve Officers' Training Corps (ROTC) cadets, respectively [9,10]. Although many athletes in American football, various types of martial arts, and CrossFit use this equipment during high-intensity aerobic or resistance training, it is still unclear whether this equipment truly provides synergetic benefits for health and performance.

In this context, we believe that understanding about the physiological responses to wearing an ETM during exercise is important not only to assess its potential as a prospective ergogenic aid, but also to design ETM-utilizing exercise protocols. Heart rate variability (HRV) assessments would provide information on how cardiac auto-regulation is altered by wearing the ETM at rest, during exercise, and recovery because HRV indices reflect the extrinsic regulation of heart rate that involves sympathetic, para-sympathetic activities, and sympathovagal balance [11]. Therefore, the purpose of this study was to examine the acute effects of the ETM on haemodynamics and HRV at rest, during cycling, and recovery. It was hypothesized that the ETM would show significant differences in blood pressure, oxygen saturation level (S_{pO_2}), and HRV responses compared to a control trial without the ETM.

MATERIALS AND METHODS

Subjects

Initially, 20 healthy male and female adults enrolled in this study. All participants were recruited by advertisements such as flyers and oral presentations in the university. Inclusion criteria of this study are

as follows: 1) participants who were aged 20-40 years, 2) able to participate in cycling exercise, and 3) body fat percentage under 30%. If participants fell outside these parameters and/or had cardiopulmonary-related diseases, they were excluded. This study was approved by the university's Institutional Review Board (IRB) and all participants received information of the study procedure, benefits, and risks of the experiment before providing written consent. During the study period, five participants dropped out for personal reasons. Therefore, 15 participants (male, $n=9$, female, $n=6$) completed this study. Participants' basic characteristics are presented in Table 1.

Study Design

This study was conducted in a randomized and crossover design. Participants reported to the human performance laboratory three times during the study period. In the first visit, participants' height, body weight, body fat percentage, respiratory function, and VO_{2peak} were measured to assess basic characteristics. In the second and third visits, participants randomly completed two trials of cycling exercise protocols with or without an ETM. Forty minutes of the cycling exercise protocol was divided into 10 minutes of 1) resting, 2) 50% of VO_{2peak} cycling, 3) 70% of VO_{2peak} cycling, and 4) recovery, with a seven-day interval between trials. During this seven-day interval, participants were asked to carry on their normal activities and not to change any exercise habits. Heart rate, blood pressure, S_{pO_2} , heart rate variability, self-perceived fatigue and self-perceived breath efforts were measured at each phase. The study procedure is shown in Figure 1.

Methodology

Height, body weight, and body fat percentage. Height and body weight were measured to the nearest 0.1 cm and 0.1 kg, respectively,

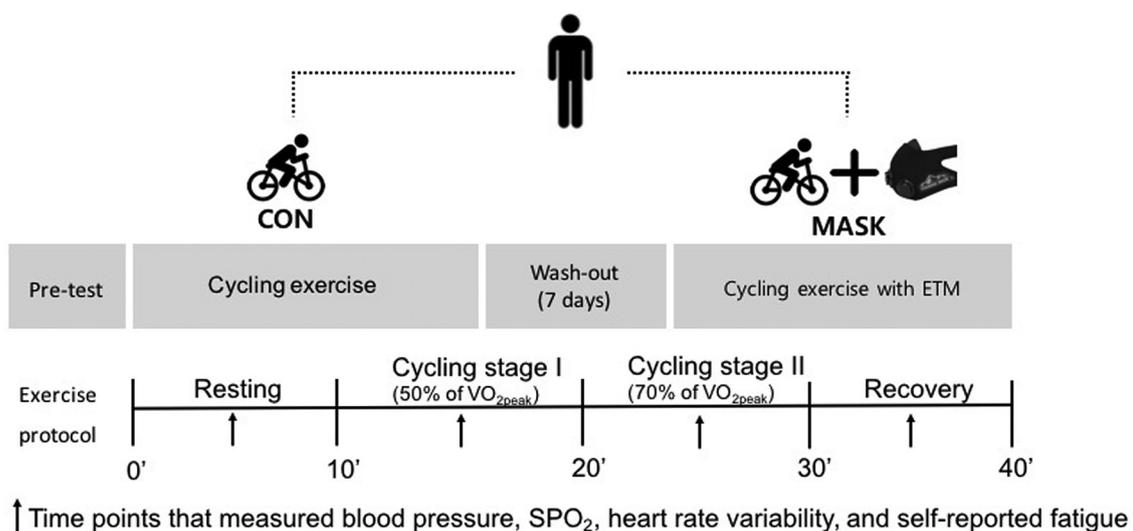


Fig. 1. The study procedure.

TABLE 1. Basic characteristics of participants (mean ± s).

Variables	Total (N=15)	Male (N=9)	Female (N=6)
Age (years)	27.0 ± 1.14	28.1 ± 1.52	25.3 ± 1.63
Height (cm)	171.3 ± 2.60	176.4 ± 3.02	163.6 ± 2.46
Weight (kg)	72.7 ± 4.04	80.1 ± 4.90	61.6 ± 3.96
Body fat percentage (%)	16.4 ± 2.40	11.7 ± 2.00	24.7 ± 1.71
Systolic BP (mmHg)	118.1 ± 3.28	125.4 ± 2.69	107.0 ± 4.16
Diastolic BP (mmHg)	70.3 ± 1.86	72.9 ± 2.13	66.3 ± 2.86
FVC (%)	91.0 ± 2.26	91.6 ± 3.07	90.2 ± 2.26
FEV1/FVC ratio	0.9 ± 0.02	0.9 ± 0.02	0.9 ± 0.04
VO _{2peak} (ml/kg/min)	34.1 ± 1.29	36.1 ± 1.64	31.1 ± 1.50

BP; blood pressure, FVC; forced expiratory volume in one second, FVC/FEV1; forced vital capacity/forced expiratory volume in one-second ratio.

with a stadiometer (PAT #290237, Novel Products, Rockton, USA) and a digital scale (HD-366, Tanita, Tokyo, Japan). Three sites of skinfold thickness were measured by one trained researcher using Lange skinfold calipers (Beta technology, Santa Cruz, CA, USA) to estimate body fat percentage. The three sites of males were chest, abdominal, and thigh, while for females they were triceps, suprailiac, and thigh. Each site was measured three times at the right side of the body, and the average score was recorded. Body density was calculated using the Jackson Pollock method [12] and body fat percentage was estimated by the Siri equation [13].

Respiratory function. The respiratory function test was measured using an electronic spirometer (MicroLab, CareFusion, USA). The test is purportedly performed to examine respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD). If any subjects had symptoms of airflow obstruction (i.e., ratio of forced expiratory volume in one second (FEV₁) / forced vital capacity (FVC) < 0.7, FEV₁ % predicted < 80%), they were excluded from the study [14]. Participants first sat in a chair for 5 minutes before they started the test. After 5 minutes, participants put on a nose clip and a spirometer mouthpiece. The respiratory function test consists of 1) three normal breaths, 2) a maximal deep inhalation followed by a 3) forceful exhalation, and 4) three more normal breaths. All participants were tested three times, and the highest scores of FEV₁ and FEV₁/FVC were recorded.

Blood pressure and S_pO₂. Blood pressure and S_pO₂ were measured once at rest, during cycling (50% and 70% of VO_{2peak}), and during recovery. Blood pressure was measured with a mercurial sphygmomanometer (BMS 12-525, Graham Field Inc., USA) by a trained technician, and S_pO₂ was measured with a pulse oximetry saturation analyzer (Checkmate, Israel). The values were recorded as mmHg and percentages, respectively.

Peak oxygen uptake test. Participants performed a VO_{2peak} test on a cycle ergometer (894E, Monark, Sweden) while interfaced with

a True-One 2400 metabolic cart (ParvoMedics, East Sandy, Utah, USA) to accurately prescribe exercise intensities for the cycling exercise. Participants performed cycling with an initial work rate equal to 60 W (1 kp) and a pedalling rate of 60 revolutions per minute (RPM) for 2 minutes. Thereafter, the work rate increased by 30 W (0.5 kp) every 2 minutes until the participant reached exhaustion. VO_{2peak} was determined when participants met at least 2 of the following conditions; (a) inability to keep up the pedalling rate of 60 rpm for more than 5 seconds with verbal encouragement, (b) respiratory exchange ratio (RER) 1.10, (c) rating of perceived exertion (RPE) ≥19 (Borg 6–20 scale), or (d) volitional fatigue.



Fig. 2. The application of ETM and the HRV measures.

Heart rate variability. Participants were instructed not to drink any caffeine-containing products for at least 24-48 hours before any of the tests. An Acti-Cardio monitor (CamNtech, UK), which traces the electro cardiograph (ECG), measured the HRV variables using two electrodes adhering to the participant's upper chest (5th inter-costal space and 10 cm away on the left side near lead four and five) (Figure 2) [15,16]. Sample rate and resolution were set at 1,024 Hz and 10-bit. Participants' ECGs were recorded for 40 continuous minutes for all phases of the test. For HRV analysis, a 5-minute period of each phase free of noise was selected according to the task force criteria of the European Society of Cardiology [17] and data were analyzed using the Activewave-Cardio Analysis software program (version 3.0.8, CamNtech, UK). The HRV variables reported with time included average heart rate, inter-beat interval (IBI), standard deviation of the IBI interval (IBISD), and root mean squared successive difference (RMSSD). Those reported with frequency domains included low-frequency (LF) and high-frequency (HF) components, and the ratio between LF and HF components (LF/HF ratio).

Self-perceived fatigues and breath effort. To measure perceived muscle fatigue and breath effort, subjects' subjective feelings were evaluated at the end of each phase using Borg's 6-20 rating of perceived exertion scale (RPE) for overall fatigue and a modified scale (range from 0 to 10) for the rate of perceived breath effort (RPBE). The number '0' indicates that the participant perceived no breathing effort at all while '10' indicates the maximal effort of breathing.

Cycling exercise. Prior to cycling exercise, participants sat quietly on a chair for 10 min as a rest phase, then performed the cycling exercise (Monark 894E, Sweden) for 20 min at two different intensities: 50% of VO_{2peak} for the first 10 min and 70% of VO_{2peak} for the next 10 min. The average loads of 50% and 70% VO_{2peak} were 1.4 ± 0.37 kp and 2.1 ± 0.55 kp, respectively. Participants maintained a pedalling rate of 60 RPM during cycling. After completion of cycling, participants sat on the chair for 10 min as a recovery phase.

Elevation training mask. The elevation training mask (ETM 2.0, Training Mask LLC, Cadillac, Michigan, USA) is a device on the commercial fitness market that claims to improve athletic performance by simulating training at altitude via restricting airflow. The ETM covers the nose and mouth with a neoprene band and flux valves that adjust the resistance of the respiration. There are four different types of plastic resistance caps (i.e., 8, 4, 2, and 1-hole open caps) with the smaller air hole caps in the flux valves causing increased resistance, making it more difficult to breathe while wearing the mask and thereby simulating higher altitude. In the current study, the ETM mask was adjusted to simulate an altitude resistance of 1,829 m.

Statistical analysis

Statistical analyses were performed with SPSS software version 25.0 (SPSS Inc. Chicago, IL, USA). All data are expressed as means and standard errors. The Kolmogorov-Smirnov test was used to analyze the normal distributions of dependent variables. If any dependent variables were not normally distributed, a natural logarithmic transformation was performed to meet the assumptions of parametric statistical analysis. 2 (trial) x 4 (time) factorial analysis of variances (ANOVAs) for repeated measures were used to assess the effects of wearing an ETM on blood pressure, S_pO_2 , heart rate variability, and self-perceived fatigue and breath effort. If any significant interaction or main effects were detected, Tukey's HSD post-hoc test was applied. Effect sizes were reported as partial eta-squared (η^2_p) indicating a small effect = .01, medium effect = .06, large effect = .14 [18]. The significance level was set at .05 for all tests.

RESULTS

Changes in haemodynamics

The changes of blood pressure and S_pO_2 are presented in Table 2. There were no significant interaction effects for group by time on blood pressure, $p > .05$. However, the systolic BP was greater in the MASK than the CON trial at rest ($p = .035$, $\eta^2_p = .279$). Regarding

TABLE 2. Changes in blood pressure and S_pO_2 at rest, during cycling, and recovery (mean \pm s).

		Rest	50% Cycling	70% Cycling	Recovery	F-value [η^2_p]		
						Group	Time	G x T
Systolic BP (mmHg)	CON	110.1 (2.15)	130.3 (5.87)	147.1 (8.47)	114.5 (3.10)	5.43*	45.92*	0.89
	MASK	114.5 ⁺ (3.42)	136.0 (5.95)	160.4 (7.91)	118.5 (2.43)	[.279]	[.766]	[.060]
Diastolic BP (mmHg)	CON	68.4 (1.94)	72.0 (3.26)	71.0 (3.09)	67.9 (2.68)	0.12	1.32	0.15
	MASK	69.6 (1.98)	73.1 (3.02)	72.5 (3.11)	68.7 (2.40)	[.009]	[.086]	[.011]
S_pO_2 (%)	CON	97.7 (.21)	97.3 (.18)	97.0 (.14)	97.3 (.25)	7.98*	11.96*	3.74*
	MASK	97.9 (.19)	96.9 (.19)	96.1 ⁺ (.17)	97.0 (.17)	[.363]	[.461]	[.211]

BP; blood pressure, G x T; group x time; ⁺ $p < .05$, significant different between the groups; * $p < .05$, significant main or interaction effects.

S_pO₂, there was a significant interaction effect for group by time (p=.033, η²_p=.211). S_pO₂ level was significantly lower in the MASK than the CON trial during high-intensity cycling (70% of VO_{2peak}).

Changes in heart rate variability

Table 3 presents the changes of HRV indices with time and frequency domains. The HRV indices were not significantly different between trials at rest and during cycling. However, heart rate (p=.047, η²_p=.253.) was significantly greater while IBI (p=.05, η²_p=.233) was lower in the MASK than the CON trial during recovery. Regarding frequency domains, the lnLF/HF ratio was significantly lower in the MASK than the CON trial during recovery (p=.01, η²_p=.373). However, no significant interaction or group effects were observed in lnLF and lnHF. The HRV indices including time and frequency domains were significantly changed across time in both trials (p<.001).

Changes in self-perceived fatigue and breath effort

There were no significant interaction effects for group by time on RPE and RPBE during the study. RPE (F=24.43, p<.001, η²_p=.636) and RPBE (F=70.83, p<.001, η²_p=.835) increased significantly as cycling intensity increased in both trials, but the RPBE was greater in the MASK than the CON trial across time (F=40.18, p<.001, η²_p=.742).

DISCUSSION

Elevation training masks have been widely used in the athletic and recreational communities. Although the company claims that the

increase in respiratory load, known as ‘altitude resistance’, improves respiratory muscle weakness and aerobic performance, it is unknown how cardiovascular function is altered in response to wearing an ETM during exercise and recovery. This study examined the acute effect of wearing the ETM on haemodynamics and HRV at rest, during cycling, and during recovery in healthy adults. The main findings of the study were as follows; 1) a greater response in systolic BP was observed at rest while S_pO₂ was lower during high-intensity cycling (70% of VO_{2peak}) in the MASK trial compared to the CON trial, 2) the HRV indices were not different between trials during cycling, but heart rate was greater and IBI was lower in the MASK than the CON trials during recovery, 3) sympathovagal balance (lnLF/HF ratio) was lower in the MASK than the CON trial during recovery, and 4) perceived breath effort was greater in the MASK than the CON trial across all time points.

Haemodynamic changes

In the present study, a greater response in systolic BP was observed at rest while S_pO₂ was lower during high-intensity cycling (70% of VO_{2peak}) in the MASK than the CON trials. Cardiovascular functions are interactively adjusted in response to respiratory function changes, such as breath frequency. The increase in respiratory load reduces the breath frequency rate, and this change influences the kinetics of oxygen consumption and CO₂ expiration [19]. It is commonly accepted that acute exposure at high altitude (>3,000 m) immediately increases the ventilation and decreases the arterial partial pressure of oxygen due to the low partial pressure of oxygen [20].

TABLE 3. Changes in HRV indices with time and frequency domains at rest, during cycling, and recovery (mean ± s).

				Rest	50% Cycling	70% Cycling	Recovery	F-value [η ² _p]		
								G	T	G x T
Time domain	Heart rate (beat/min)	CON		74.5 (3.34)	129.3 (5.89)	157.4 (4.61)	99.5 (4.25)	4.86*	332.42*	1.04
		MASK		78.4 (2.20)	131.7 (4.49)	164.0 (4.53)	106.7 ⁺ (3.61)	[.253]	[.964]	[.030]
	IBI (sec)	CON		.826 (.035)	.477 (.021)	.384 (.012)	.610 (.023)	3.97*	299.28*	1.01
		MASK		.773 (.021)	.462 (.016)	.370 (.010)	.570 ⁺ (.017)	[.233]	[.958]	[.103]
	IBISD (sec)	CON		.082 (.009)	.022 (.002)	.012 (.002)	.089 (.043)	.56	9.61*	1.20
		MASK		.088 (.006)	.024 (.006)	.009 (.001)	.040 (.005)	[.038]	[.407]	[.079]
RMSSD (sec)	CON		.07 (.011)	.02 (.004)	.01 (.002)	.03 (.007)	.26	35.53*	1.02	
	MASK		.10 (.041)	.01 (.003)	.01 (.002)	.03 (.006)	[.018]	[.717]	[.068]	
Frequency domain	lnLF	CON		6.4 (.33)	3.0 (.39)	0.8 (.33)	4.9 (.27)	.02	120.66*	3.10
		MASK		6.9 (.22)	2.6 (.34)	0.2 (.38)	5.3 (.32)	[.001]	[.896]	[.181]
	lnHF	CON		5.4 (.39)	1.6 (.44)	0.01 (.42)	2.9 (.44)	2.84	83.25*	2.48
		MASK		6.1 (.20)	2.3 (.47)	-0.4 (.47)	4.2 (.36)	[.169]	[.856]	[.150]
	lnLF/HF	CON		1.2 (.07)	1.9 (.41)	0.7 (.78)	2.2 (.29)	8.34*	5.53*	1.55
		MASK		1.1 (.03)	1.1 (.41)	-0.5 (.26)	1.3 ⁺ (.09)	[.373]	[.283]	[.100]

G; group, T; time, G x T; group x time, IBI; inter beat interval, IBISD; standard deviation of the IBI, RMSSD; root mean squared successive difference, lnLF; natural logarithm of low frequency, lnHF; natural logarithm of high frequency; ⁺p<.05, significant different between the groups; *p<.05, significant main or interaction effects.

In contrast to altitude exposure, Grandos and colleagues reported that an increase in respiratory load during steady state exercise did not increase the ventilation rate, but increased respiratory load may change metabolic CO₂ in the respiratory tract without significant changes in oxygen consumption kinetics [19]. The author assumed that the increased respiratory load might influence the ventilatory equivalents, leading to a decrease of the peripheral oxygen saturation level during exercise. The present study also found a similar result, as a greater decrease in O₂ saturation level was found in the MASK trial than the CON trial during cycling, but the difference was only observed during high-intensity (70% of VO_{2peak}) cycling. The differences between the present and previous studies may be associated with various factors such as different altitude resistance (1860 m vs 2743 m, 4572 m), subjects (both male and female vs male alone), exercise type (cycling vs treadmill), and intensity (50% and 70% vs 60% of VO_{2peak}). Additionally, it was assumed that respiratory function may not be a limiting factor for peripheral oxygen carrying capacity at rest or during moderate-intensity cycling in healthy adults despite the loaded respiration. However, the increased respiratory load during high-intensity cycling may be less sufficient for the kinetics of oxygen consumption to meet the exercise demands in working muscle (i.e., metabolic demands); thus it can be postulated that wearing an ETM during high-intensity cycling induces modest arterial hypoxaemia in healthy adults. Despite the ETM inducing modest arterial hypoxaemia, it is difficult to confirm that this device could provide similar beneficial effects of altitude training on haematological changes such as increased red blood cell and erythropoietin concentrations. A previous study reported that exposure to hypoxic conditions with an ETM for 60 minutes per week for 6 weeks was not sufficient to change the haematological variables [9]. Another study also revealed that 6 weeks of physical training with an ETM did not improve aerobic capacity in ROTC cadets [10].

Heart rate variability

The present study hypothesized that wearing an ETM during cycling would greatly influence HRV compared to not wearing an ETM. Although the HRV indices were not different between the trials at rest and during cycling, the heart rate was greater while the IBI was lower in the MASK than the CON trial during recovery. It has been demonstrated that respiration plays an important role in modulating HRV as well as the baroreflex [21]. The increase in respiratory resistance changes the inspiration process from passive to active, which results in recruiting an additional respiratory muscle group (i.e., sternocleidomastoid) [22]. Along with the additional muscle recruitment, slow oxygen consumption kinetics with loaded respiration may change the signalling in the central cardiovascular centre, which results in increasing heart rate and blood pressure. Another explanation may involve the intensive activation of the diaphragm metaboreflex, which can lead to increased sympathetic activity [23]. In agreement with our results, McConnell and Griffiths found that 30% of

maximal inspiratory pressure increased HR and blood pressure in healthy males [24].

Regarding frequency domains, the lnLF and lnHF powers decreased as cycling intensity increased in both trials, but the variables were not different between the MASK and CON trials in the present study. Traditionally, LF power represents both sympathetic and parasympathetic modulation while HF power indicates parasympathetic activity [11]. It has been demonstrated that autonomic transition occurred toward sympathetic dominance when shifting from rest to exercise, such as cycling [25]. Previous studies also reported that frequency domains including LF and HF powers decreased as exercise intensity increased until the heart rate reached 120 to 180 beat/min [25,26], and this change continued until the first ventilation threshold [27]. Moreno's study also supports our result that frequency domains of HRV (i.e., LF, HF) are reduced while HR increases during moderate-intensity exercise (60% of VO_{2peak}) [28]. However, while the lnLF/HF ratio increased from rest (1.2) to moderate-intensity cycling (1.9) in the CON trial, this score did not change in the MASK trial (1.1 at rest and moderate-intensity cycling) in this study. It was believed that the sympathetic dominance during moderate-intensity exercise caused the increase in LF/HF ratio (sympathovagal balance) due to HF power withdrawal concomitant with increased LF power [29]. As respiratory variation is a primary factor for vagal modulation, it was assumed that an ETM-induced longer respiration phase might not diminish the vagal activity. After cycling exercise, lnLF/HF ratio was lower in the MASK than the CON trial in this study and this particular finding may be due to the restoration of the HF power from the cycling exercise. Although there was no significant difference in lnHF power between the trials during recovery, the mean lnHF power was greater in the MASK than the CON trial (4.2 vs 2.9), whereas the mean lnLF power showed similar values (5.3 vs 4.9). Nevertheless, it is premature to definitively conclude that our result is directly related to parasympathetic predominance, but perhaps it is because other physiological variables, including heart rate and IBI, were greater in the MASK than the CON trial despite the lower sympathovagal balance during recovery. We carefully assumed that even though extrinsic controls of heart rate (i.e., sympathetic and parasympathetic control) are changed by the ETM, the intrinsic controls of heart rate such as SA node might increase to compensate the changed autonomic regulations to remove the metabolic by-products after exercise. The present study confirms that wearing an ETM delays the cardiac-autonomic recovery from cycling exercise.

Perceived fatigue and breath effort

Rate of perceived exertion is a practical method to measure fatigue rate during exercise. In the present study, perceived fatigue did not differ between trials, but perceived breath effort was greater in the MASK than the CON trials across time. It was assumed that increased respiratory load decreased ventilation, and decreased ventilation might be associated with psychological discomfort. Our assumption

is supported by a previous study which found that perceived breath effort is positively associated with ventilation [30].

Limitations

Some limitations should be considered in this study. First, breath frequency rate was not measured in the present study. Even though previous studies demonstrated that the increase in respiratory load reduced the breath frequency rate [19], the absence of breath frequency rate measurement may limit explanation of the association between breath frequency rate and HRV in this study. Secondly, this study only tested in one condition with altitude of resistance of 1,829 m, even though the company introduces various altitude resistance conditions (914 m to 5,486 m). Therefore, applying different altitude resistances during exercise may result in different outcomes.

CONCLUSIONS

Our findings suggest that wearing an ETM (altitude resistance, 1,829 m) during high-intensity cycling (70% of VO_{2peak}) induces modest hypoxaemia. Although this device does not affect HRV changes during cycling, it seems to delay the cardiac-autonomic recovery from exercise. Based on this study, healthy adults may be able to follow an ETM-utilizing exercise programme to simulate a

hypoxic environment, but future studies are needed to investigate whether repeated exposure to this condition provides similar benefits as altitude training in healthy adults.

Practical applications

The elevation training mask has been used in the health and athletic communities to improve performance. Although the company claims the beneficial effect of ETM on aerobic performance, the evidence is still debatable. Based on our results, wearing an ETM during high-intensity exercise ($\geq 70\% VO_{2max}$) simulates modest hypoxaemia, but future studies are needed to determine whether this modest hypoxic condition can provide performance improvement. Additionally, professionals should consider the psychological discomfort (i.e., perceived breath effort) and delayed cardiac-autonomic recovery when designing an ETM-utilizing exercise programme for healthy adults.

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Disclosure statement

No potential conflict of interest was reported by the authors.

REFERENCES

1. Wolpat A, Lima FV, Silva FM, et al. Association between inspiratory muscle weakness and slowed oxygen uptake kinetics in patients with chronic obstructive pulmonary disease. *Appl Physiol Nutr Metab.* 2017; 42(12):1239-1246.
2. Charusisin N, Dacha S, Gosselink R, et al. Respiratory muscle function and exercise limitation in patients with chronic obstructive pulmonary disease: a review. *Expert Rev Respir Med.* 2018; 12(1):67-79.
3. Langer, D. Inspiratory Muscle Training. In *Textbook of Pulmonary Rehabilitation* (pp. 233-249). Springer, Cham; 2018.
4. Archiza B, Simões RP, Mendes RG, Fregonezi GA, Catai AM, Borghi-Silva A. Acute effects of different inspiratory resistive loading on heart rate variability in healthy elderly patients. *Braz J Phys Ther.* 2013;17(4):401-408.
5. Mello PR, Guerra GM, Borile S, et al. Inspiratory muscle training reduces sympathetic nervous activity and improves inspiratory muscle weakness and quality of life in patients with chronic heart failure: a clinical trial. *J Cardiopulm Rehabil Prev.* 2012; 32(5):255-261.
6. Witt JD, Guenette JA, Rupert JL, McKenzie DC, Sheel AW. Inspiratory muscle training attenuates the human respiratory muscle metaboreflex. *J Physiol.* 2007;584(3):1019-1028.
7. Jaenisch RB, Quagliotto E, Chechi C, et al. Respiratory muscle training improves chemoreflex response, heart rate variability, and respiratory mechanics in rats with heart failure. *Can J Cardiol.* 2017;33(4):508-514.
8. Volianitis S, McConnell AK, Koutedakis Y, McNaughton LR, Backx K, Jones DA. Inspiratory muscle training improves rowing performance. *Med Sci Sports Exerc.* 2001; 33(5):803-809.
9. Porcari JP, Probst L, Forrester K, et al. Effect of wearing the elevation training mask on aerobic capacity, lung function, and hematological variables. *J Sports Sci Med.* 2016;15(2):379.
10. Sellers JH, Monaghan TP, Schnaiter JA, Jacobson BH, Pope ZK. Efficacy of a ventilatory training mask to improve anaerobic and aerobic capacity in reserve officers' training corps cadets. *J Strength Cond Res.* 2016; 30(4):1155-1160.
11. Acharya UR, Joseph KP, Kannathal N, Lim CM, Suri JS. Heart rate variability: a review. *Med Biol Eng Comput.* 2006; 44(12):1031-1051.
12. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. *Br J Nutr.* 2004;91(1):161-168.
13. Siri WE. Body composition from fluid spaces and density: Analysis of methods. 1961. *Nutrition.* 1993; 9(5):480-491.
14. National Institute for Clinical Excellence. Chronic obstructive pulmonary disease in over 16s: diagnosis and management. *Clinical Guideline*, 2010;101:23.
15. Bridget A. Actiwave cardio: the feasibility and validation of an innovative new ambulatory. *Proceedings of Australian Cardiovascular Health and Rehabilitation Association at Brisbane, Australia*; 2014.
16. Thompson AG, Swain DP, Branch JD, Spina RJ, Grieco CR. Autonomic response to tactical pistol performance measured by heart rate variability. *J Strength Cond Res.* 2015; 29(4):926-933.
17. Malik M, Bigger JT, Camm AJ, et al. Heart rate variability-standards of measurements, physiological interpretation, and clinical use; Task Force of the European Society of Cardiology. *Eur Heart J.* 1996; 17(3):354-381.
18. Lakens, D. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for t-tests and ANOVAs. *Front Psychol.* 2013; 4:863.
19. Granados J, Gillum TL, Castillo W, Christmas KM, Kuennen MR. "Functional" respiratory muscle training during endurance exercise causes modest hypoxemia but overall

- is well tolerated. *J Strength Cond Res.* 2016;30(3):755-762.
20. Helfer S, Quackenbush J, Fletcher M, Pendergast DR. Respiratory muscle training and exercise endurance at altitude. *Aerosp Med Hum Perform.* 2016;87(8):704–711.
21. Bernardi L, Porta C, Gabutti A, Spicuzza L, Sleight P. Modulatory effects of respiration. *Auton Neurosci.* 2001;90(1):47-56.
22. Hellyer NJ, Folsom IA, Gaz DV, Kakuk AC, Mack JL, Ver Mulm JA. Respiratory muscle activity during simultaneous stationary cycling and inspiratory muscle training. *J Strength Cond Res.* 2015;29(12):3517-3522.
23. Sheel AW, Derchak PA, Morgan BJ, Pegelow DF, Jacques AJ, Dempsey JA. Fatiguing inspiratory muscle work causes reflex reduction in resting leg blood flow in humans. *J Physiol.* 2001;537(1):277-289.
24. McConnell AK, Griffiths LA. Acute cardiorespiratory responses to inspiratory pressure threshold loading. *Med Sci Sports Exerc.* 2010; 42(9):1696-1703.
25. Casties JF, Mottet D, Le Gallais D. Non-linear analyses of heart rate variability during heavy exercise and recovery in cyclists. *Int J Sports Med.* 2006;27(10):780-785.
26. Povea C, Schmitt L, Brugniaux J, Nicolet G, Richalet JP, Fouillat JP. Effects of intermittent hypoxia on heart rate variability during rest and exercise. *High Alt Med Biol.* 2005; 6(3):215-225.
27. Cottin F, Leprêtre PM, Lopes P, Papelier Y, Médigue C, Billat V. Assessment of ventilatory thresholds from heart rate variability in well-trained subjects during cycling. *Int J Sports Med.* 2006;27(12):959-967.
28. Moreno IL, Pastre CM, Ferreira C, de Abreu LC, Valenti VE, Vanderlei LCM. Effects of an isotonic beverage on autonomic regulation during and after exercise. *J Int Soc Sports Nutr.* 2013; 10(1):2.
29. Perini R, Veicsteinas A. Heart rate variability and autonomic activity at rest and during exercise in various physiological conditions. *Eur J Appl Physiol.* 2003;90(3-4):317-325.
30. Suzuki S, Sato M, Okubo T. Expiratory muscle training and sensation of respiratory effort during exercise in normal subjects. *Thorax.* 1995; 50(4):366-370.