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# The effects of a respiratory training mask on steady-state oxygen consumption at rest and during exercise

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## ABSTRACT

No studies have directly measured ventilatory and metabolic responses while wearing a respiratory training mask (RTM) at rest and during exercise. Eleven aerobically fit adults (age: 21 ± 1 years) completed a randomized cross-over study while wearing an RTM or control mask during cycling at 50% Wmax. An RTM was retrofitted with a gas collection tube and set to the manufacturer's "altitude resistance" setting of 6,000 ft (1,800 m). Metabolic gas analysis, ratings of perceived exertion, and oxygen saturation ($SpO_2$) were measured during rest and cycling exercise. The RTM did not affect metabolic, ventilation, and $SpO_2$ at rest compared to the control mask (all, effect of condition: $P > 0.05$). During exercise, the RTM blunted respiratory rate and minute ventilation (effect of condition: $P < 0.05$) compared to control. Similar increases in $VO_2$ and $VCO_2$ were observed in both conditions (both, effect of condition: $P > 0.05$). However, the RTM led to decreased fractional expired $O_2$ and increased fractional expired $CO_2$ (effect of condition: $P < 0.05$) compared to the control mask. In addition, the RTM decreased $SpO_2$ and increased RPE (both, effect of condition: $P < 0.05$) during exercise. Despite limited influence on ventilation and metabolism at rest, the RTM reduces ventilation and disrupts gas concentrations during exercise leading to modest hypoxemia.

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KEYWORDS

respiratory muscle training, hypoxemia, altitude, respiratory resistance training

## INTRODUCTION

Athletes, coaches, and general exercisers are continuously trying novel training methods to enhance performance and improve health. Sport scientists and exercise physiologists assess the validity and effectiveness of novel training methods to distinguish whether these methods provide a true ergogenic or pseudoergogenic benefit [1]. High altitude training methods have been studied extensively over the past four decades due to the potential benefits for maximizing endurance performance at sea-level [2–4]. Living at high altitude and training at low altitude has been shown to increase red blood cell mass and hemoglobin content in endurance-trained athletes [2, 5], although the effectiveness of high altitude exposure on endurance performance is debated [6, 7]. Nonetheless, these are two important physiological adaptations to hypoxia that could, in theory, increase oxygen ($O_2$) delivery capacity and performance at sea-level. Due to the proposed benefits, other methods have been developed such as artificial hypoxia (e.g., nitrogen houses, altitude tents, hypobaric chambers) to achieve performance benefits in sub-elite level athletes [8].

More recently, respiratory training masks (RTM) or "elevation" training masks have been developed and marketed as an affordable solution with the goal of training respiratory muscles or simulating [9] "altitude resistance" to improve performance. RTMs utilize an adjustable valve system to create varied inspiratory resistances with a set expiratory resistance load. Recent studies have tested whether an RTM can provide a similar physiological stress (hypoxemia, increased ventilation, sympathoexcitation) as high altitude with regard to corresponding "altitude resistances" during acute and chronic exercise [10–13], however, only two studies have measured metabolic responses during acute exercise. Granados and colleagues (2016) developed a modified resistance valve configuration, similar to an RTM, to measure expired gases during exercise at "altitude resistance" settings stated to be equivalent to 9,000 ft (2,700 m) and 15,000 ft (4,600 m) according to manufacturer documents. The authors found that increasing inspiratory resistance led to modest hypoxemia (reduced oxygen saturation; $SpO_2$) during steady-state treadmill exercise at 60% peak oxygen consumption ($VO_{2peak}$), and postulated the mechanism to be augmented carbon dioxide ($CO_2$) rebreathing and reduced ventilation [10]. However, a similar study using a different modified resistance valve configuration found no change in $SpO_2$ during cycling at 60% maximal power output [14]. Jung and colleagues (2019) provided additional data to support that the RTM significantly reduced $SpO_2$ at a lower "altitude resistance" setting (6,000 ft; 1,800 m) during cycling at 70% $VO_{2peak}$, but not at 50% $VO_{2peak}$ [12]. However, they did not directly measure metabolic or ventilatory responses in their study, and these responses remain unknown at the lower "altitude resistance" setting. To date, all studies examining the impact of inspiratory resistance training during exercise have either failed to measure metabolic gases or have used custom mask configurations that do not sample gases directly from a commercially available RTM. As such, this inhibits the ability to identify the true mechanistic role of metabolic gases on potential benefits and specific adaptations.

The purpose of this study was to assess metabolic and ventilatory responses of participants wearing an RTM set to a moderate "altitude resistance" setting (6,000 ft; 1,800 m) at rest, during submaximal exercise at 50% of maximum workload, and during recovery. We did so by directly

sampling gases within the RTM. We hypothesized that the RTM mask would reduce respiratory rates and minute ventilation, and increase ratings of perceived exertion (RPE), but the lower resistance setting would not alter peripheral $SpO_2$ in healthy fit young adults at rest or during exercise.

# MATERIALS AND METHODS

## Participants

All experimental protocols were approved by the Institutional Review Board and were conducted in accordance with the Declaration of Helsinki. Participants were recruited from the local area and population surrounding Plymouth State University. We used convenience sampling methods to recruit participants using flyers, word-of-mouth, and in-person class announcements. All study participants provided written consent before being enrolled in the study. Research personnel completed University-approved Environmental, Health, and Safety training before collecting data on research participants. Prior to participants completing a $VO_{2peak}$ test, participants completed a health history questionnaire that screened for tobacco product consumption, cardiovascular disease, diabetes, cancer, respiratory diseases, or renal disease. Participants were excluded if they had any of the diseases mentioned above, if they did not meet the $VO_{2peak}$ requirement, or if they had excess facial hair, resulting in poor face mask fitting.

## Familiarization and peak oxygen consumption

Participants were tested in the Exercise and Sport Physiology Research Laboratory at Plymouth State University for all testing procedures. Data was collected between January and March in 2018. Each participant completed a familiarization visit, which included a $VO_{2peak}$ test. Prior to the start of the $VO_{2peak}$ test, height, weight, and body composition (Tanita BC-418 Segmental Body Composition Analyzer, Arlington Heights, IL, USA) were measured following a 4-h fast. Participants refrained from caffeine and exercise for 24-h prior to $VO_{2peak}$ testing, as well as subsequent experimental study visits. $VO_{2peak}$ testing was completed using a sex-specific maximal exercise protocol for men [15] and women [16] to ensure all participants were aerobically fit. The protocol required both men and women to pedal at 75 rpm with resistance increasing every three minutes until exhaustion. Women began at a lower resistance setting (65 W) compared to men (95 W). Women progressed to 95 W after the initial three minutes. After 95 W for both men and women, the resistance was increased each stage by 35 W until exhaustion. The test was completed on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), and was accompanied with expiratory metabolic gas analysis (ParvoMedics TrueOne 2400, Salt Lake City, Utah, USA). Gas was analyzed in 15-s intervals to determine $VO_{2peak}$. $VO_{2peak}$ was established as the single highest oxygen consumption value obtained during the final stage of work completed during the maximal test protocol. Participants continued in the study if they met the $VO_{2peak}$ requirement of $\geq$ 45 $mL \cdot kg^{-1} \cdot min^{-1}$ for men, or $\geq$ 38 $mL \cdot kg^{-1} \cdot min^{-1}$ for women to classify participants with "Good" aerobic fitness [17]. After the participants had recovered from the $VO_{2peak}$ test, participants were fitted with the RTM to familiarize them to breathing while wearing the mask. All participants were blinded to the randomized testing order prior to their first experimental protocol visit.

## Experimental protocol

A randomized cross-over study design (Fig. 1) was used to examine the metabolic and physiological responses to the RTM compared to a control mask (RTM vs. Control) at rest and during exercise. The RTM has three valve ports on the mask. The two lateral valve ports on the mask can be adjusted to increase airflow resistance and prevent any backflow. These two lateral valves can be removed to adjust "altitude resistance" by replacing the valves with valves that have a smaller surface area opening to allow airflow into the mask. Airflow resistance was set to an "altitude resistance" setting of 6,000 ft (1,800 m). The RTM was retrofitted with a gas collection tube to directly collect and measure expiratory gases from the center expiratory valve. Proper fitting to ensure a complete seal around the face was performed prior to testing. Each participant completed the experimental protocol at the same time of day and under the same environmental conditions (temperature: control = 22.5 ± 0.7 °C vs. RTM = 21.8 ± 0.3 °C, $P > 0.05$; barometric pressure: control = 745.8 ± 2.6 mmHg vs. RTM = 748.5 ± 3.5, $P > 0.05$; humidity: control = 20 ± 2 mmHg vs. RTM = 20 ± 1, $P > 0.05$).

Participants completed two study visits wearing a control mask (7450 Series Silicone V2™ Oro-Nasal Mask, Hans Rudolph, Inc, Kansas, USA) and an RTM (Elevation Training Mask 2.0, Michigan, USA). The following expiratory gas measurements were assessed: volume of oxygen consumed ($VO_2$), volume of carbon dioxide produced ($VCO_2$), respiratory exchange ratio (RER), fractional expired oxygen ($F_EO_2$), fractional expired carbon dioxide ($F_ECO_2$), minute ventilation ($V_E$), and respiratory rate. Ventilatory equivalents were used as a measure of ventilatory efficiency by dividing $V_E$ by the volume of oxygen consumed, and the volume of carbon dioxide produced ($V_E/VO_2$ & $V_E/VCO_2$, respectively). Control masks used a standard fitting used for oxygen consumption testing and did not restrict breathing. According to manufacturer product details, the control mask and standard T-valve assembly (Series 2,600 medium) increase dead space by 219-172 mL based on mask size (medium to petite). The RTM increases dead space by approximately 250 mL.

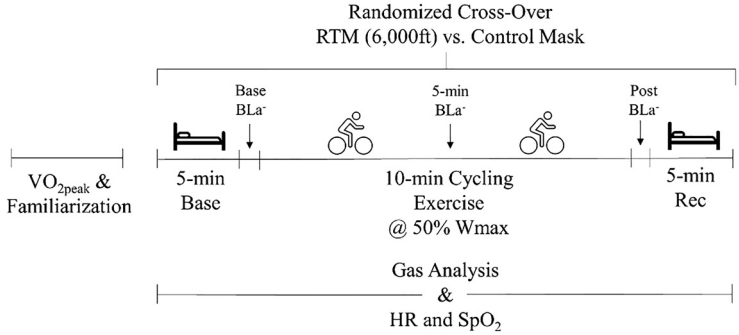


*Fig. 1.* A randomized cross-over study design was used to assess metabolic and ventilatory variables while wearing a respiratory training mask (RTM; 6,000 feet) and control mask
These responses were measured at rest, during cycling at 50% maximum workload (Wmax), and recovery. Study visits were separated by 4–10 days. Blood lactate ($BLa^-$) was collected after a 5 min baseline, 5 minutes into cycling exercise, and immediately post exercise. Gas analysis was measured throughout, heart rate (HR) was measured every minute, and oxygen saturation ($SpO_2$ and rating of perceived exertion (RPE) were measured every 2 minutes.

Upon arrival at the laboratory, all participants were asked to use the restroom to void their bladder. Before the start of the test, participants rested in the supine position on a padded table for 5-min. After the 5-min period, masks were fitted to the face of each participant. Immediately after fitting, a leak test was performed to ensure no air was leaking around the periphery of the masks. A chest strap heart rate monitor (Polar Electro, New York, USA) was placed at the level of the xiphoid process. A pulse oximeter (Pulse Oximeter PC-66H, Georgia, USA) was placed onto the right index finger according to manufacture specification to measure $SpO_2$ during rest, exercise, and recovery. Participants then completed another 5-min of quiet rest while baseline heart rate, $SpO_2$, blood lactate (Nova Biomedical Lactate Plus Analyzer, Massachusetts, USA), and expiratory gases were measured. Participants then completed 10-min of steady-state cycling exercise at 50% of the maximum workload (watts) achieved during their $VO_{2peak}$ test. The same workload was used for each experimental study visit. Participants were instructed to breathe spontaneously while pedaling at 75 rpm. Expiratory gases and heart rate were measured at the end of each minute of exercise. $SpO_2$ and rating of perceived exertion (1–10 Modified Borg Scale) were measured at the end of every 2-min during exercise. Blood lactate was measured at the 5-min time point during steady-state exercise and immediately after exercise.

### Data & statistical analysis

An *a priori* power analysis was performed to determine the sample size necessary to detect a significant reduction in $SpO_2$ based on previously published findings [10]. While we did not hypothesize that $SpO_2$ would reach statistical significance, we wanted to ensure that the study was adequately powered to potentially detect a significant difference in $SpO_2$. A significant difference greater than 2% would ensure that a significant finding in $SpO_2$ would be greater than the measurement error of the pulse oximeter. Therefore, an *a priori* power analysis with significance set at an alpha level of $P < 0.05$, and $\beta = 80\%$ to detect an estimated mean difference $2 \pm 1\%$ $SpO_2$ with a large effect size ($f = 1.0$) yielded a required sample size of 10 participants. All expiratory gas collected for baseline, steady-state exercise, and recovery gas analysis used 60-s averaging. Baseline and post-exercise expiratory gas analysis data were averaged across the 5-min steady-state period. All statistical analyses were performed using GraphPad Prism 8 (GraphPad Prism version 8.4.1 for Windows, GraphPad Software, San Diego, California, USA). Two-way repeated-measures ANOVAs were used to compare expiratory gases, ventilation, blood lactate, RPE, $SpO_2$, and heart rate during exercise between conditions. Post-hoc analysis was performed using Sidak's multiple comparisons test if significance was detected between conditions (Control Mask vs. RTM). When an interaction effect was detected, we analyzed the effect between conditions at each time point. Statistical significance was set to an alpha level of $P < 0.05$. All data are presented as mean ± SEM. Effect sizes are recorded as partial eta-squared ($\eta^2_p$) and are presented in the corresponding table and figure legends.

## RESULTS

### Participant characteristics

A total of 16 participants were recruited into the study. Four participants did not meet the $VO_{2peak}$ requirement, and another participant did not complete the study due to a non-study

*Table 1.* Participant characteristics

| | Mean | | S.D. |
|---|---|---|---|
| Participants (*n*) | | 9M/2W | |
| Age (years) | 21 | ± | 2 |
| Height (cm) | 175 | ± | 7 |
| Weight (kg) | 79 | ± | 11 |
| Body Mass Index ($kg \cdot m^{-2}$) | 25.8 | ± | 3 |
| Body Fat Percentage (%) | 17.3 | ± | 5 |
| Peak Power (watts) | 287 | ± | 47.6 |
| $VO_2$ Peak ($mL \cdot kg^{-1} \cdot min^{-1}$) | 49.9 | ± | 5.2 |

$VO_2$, Oxygen consumption; M, Men; W, Women.

related musculoskeletal injury. Therefore, eleven participants are presented herein (men, $n = 9$; women, $n = 2$). Baseline participant characteristics are provided in Table 1. All participants were healthy, fit men and women (age range = 18–24 years).

## $SpO_2$, lactate, and rating of perceived exertion during baseline and steady-state exercise

$SpO_2$, blood lactate, and RPE are presented in Table 2. Two-way repeated-measures ANOVAs found that steady-state exercise reduced $SpO_2$ and increased both blood lactate and RPE

*Table 2.* Assessment of oxygen saturation, blood lactate, and rating of perceived exertion during steady state exercise

| Testing variables | Control mask | | | Respiratory training mask | | |
|---|---|---|---|---|---|---|
| | Mean | ± | S.D. | Mean | ± | S.D. |
| Baseline $SpO_2$ (%) | 98.0 | ± | 1.2 | 98.0 | ± | 0.9 |
| Exercise Minute 2 $SpO_2$ (%) | 96.5 | ± | 1.1 | 95.7 | ± | 1.3 |
| Exercise Minute 4 $SpO_2$ (%) | 96.9 | ± | 0.9 | 96.1 | ± | 1.6 |
| Exercise Minute 6 $SpO_2$ (%) | 96.9 | ± | 0.9 | *96.1 | ± | 1.6 |
| Exercise Minute 8 $SpO_2$ (%) | 97.0 | ± | 1.0 | *95.7 | ± | 1.7 |
| Exercise Minute 10 $SpO_2$ (%) | 96.5 | ± | 0.9 | 95.6 | ± | 1.7 |
| Baseline Blood Lactate ($mmol \cdot L^{-1}$) | 1.5 | ± | 0.5 | 1.6 | ± | 0.8 |
| Exercise Minute 5 Blood Lactate ($mmol \cdot L^{-1}$) | 3.1 | ± | 0.9 | 3.2 | ± | 1.1 |
| Recovery Blood Lactate ($mmol \cdot L^{-1}$) | 3.3 | ± | 0.9 | 4.0 | ± | 1.1 |
| Exercise Minute 2 RPE | 2.4 | ± | 0.7 | *3.5 | ± | 1.6 |
| Exercise Minute 4 RPE | 3.1 | ± | 0.7 | *4.2 | ± | 1.5 |
| Exercise Minute 6 RPE | 3.7 | ± | 0.8 | *4.7 | ± | 1.6 |
| Exercise Minute 8 RPE | 4.3 | ± | 0.9 | *5.1 | ± | 1.3 |
| Exercise Minute 10 RPE | 4.6 | ± | 1.0 | *5.4 | ± | 1.3 |

$SpO_2$, Oxygen Saturation, RPE, Rating of Perceived Exertion (1–10 scale). $SpO_2$ ($\eta^2_p$: condition = 0.384; time = 0.623); Blood Lactate ($\eta^2_p$: condition = 0.051; time = 0.674); RPE ($\eta^2_p$: condition = 0.784; time = 0.894). Sidak's Post Hoc Multiple Comparisons test: * $P < 0.05$ vs. Control Mask.

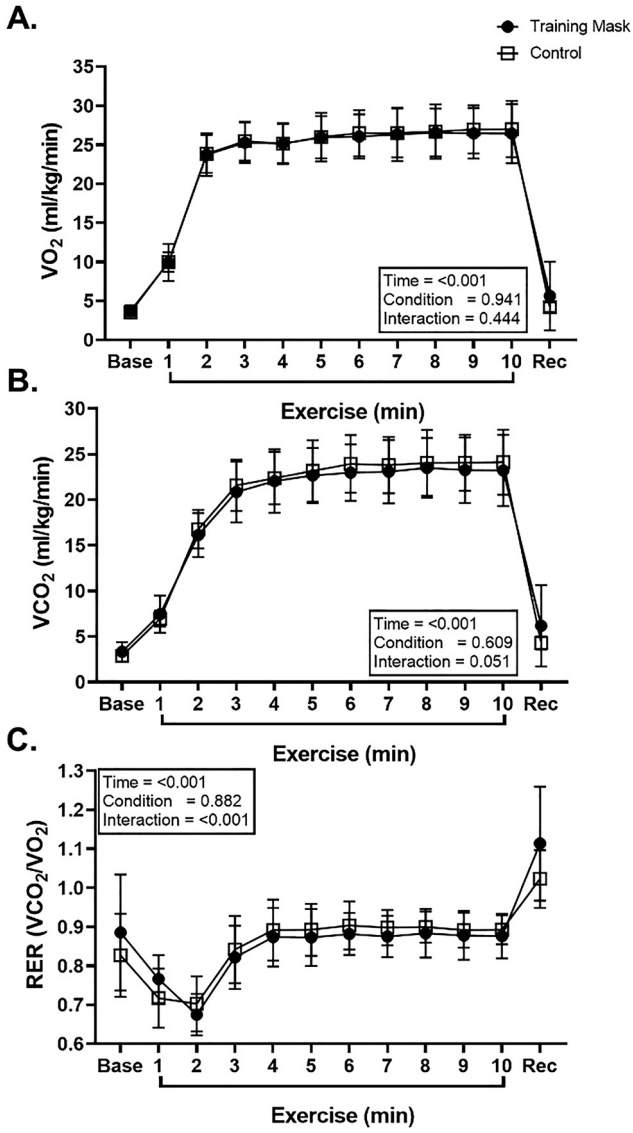


*Fig. 2.* Measured oxygen consumption (**A**; $VO_2$), carbon dioxide production (**B**; $CO_2$), and respiratory exchange ratio (**C**; RER) at baseline, during steady state exercise, and recovery. Oxygen consumption ($\eta^2_p$: condition = 0.001; time = 0.991) and carbon dioxide ($\eta^2_p$: condition = 0.0340; time = 0.991) production were not different between the two exercise conditions. Respiratory exchange ($\eta^2_p$: condition = 0.001; time = 0.915) ratio was not different between the two exercise conditions

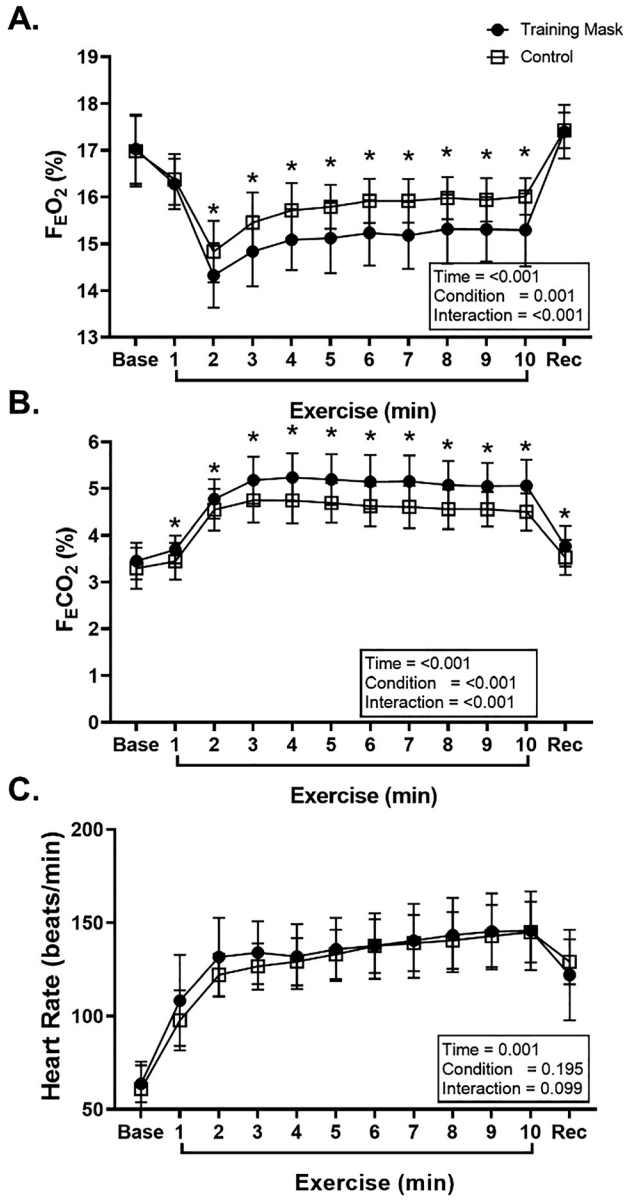


*Fig. 3.* Measured fractional expired oxygen (**A**; $FEO_2$), fractional expired carbon dioxide (**B**; $FECO_2$), and heart rate (**C**) at baseline, during steady state exercise, and recovery. Fractional expired oxygen ($\eta^2_p$: condition = 0.664; time = 0.948) was reduced, while fractional expired carbon dioxide ($\eta^2_p$: condition = 0.784; time = 0.967) was increased during steady state exercise when wearing the respiratory training mask. Heart rate ($\eta^2_p$: condition = 0.077; time = 0.948) was not different between the two exercise conditions. Sidak's Post Hoc Multiple Comparisons test: * $P < 0.05$ vs. Control Mask

($P < 0.05$ for all, main effect of time). The RTM reduced $SpO_2$ ($P < 0.05$, main effect of condition) and augmented RPE ($P < 0.05$, main effect of condition) compared to the control mask. However, there was no effect of condition ($P > 0.05$) on blood lactate. Post-hoc analysis revealed no significant differences between conditions at baseline for $SpO_2$ and blood lactate (RTM vs. Control, $P > 0.05$) compared to the control mask. During exercise, $SpO_2$ was reduced at the sixth and eighth minute of steady-state exercise compared to the control mask. The RTM increased RPE across all time points compared to the control mask ($P < 0.05$).

## Expiratory gas, respiratory, and heart rate responses at baseline, during steady-state exercise, and during recovery

$VO_2$ and $VCO_2$ increased from baseline to steady-state exercise and decreased during recovery ($P < 0.05$, main effect of time), however, there was no difference in $VO_2$ and $VCO_2$ between the RTM and control mask (Fig. 2A and B, $P > 0.05$, main effect of condition). Similarly, exercise increased RER ($P < 0.05$, main effect of time) but did not differ by condition (Fig. 2C). $F_EO_2$ decreased from baseline during exercise and returned to baseline levels during recovery ($P < 0.05$, main effect of time). During exercise, the RTM resulted in a significantly lower $F_EO_2$ compared to the control mask (Fig. 3A, $P < 0.05$, main effect of condition). Oppositely, $F_ECO_2$

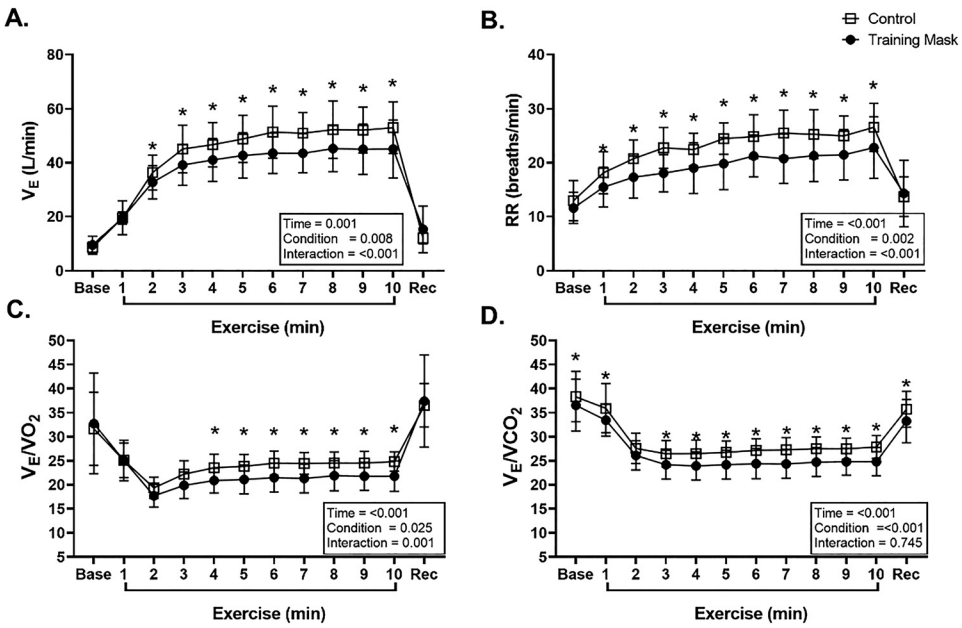


*Fig. 4.* The effect of the respiratory training mask (RTM) and control mask at rest, exercise, and recovery. The RTM resulted in a blunted (**A**) ventilation minute ($\eta^2_p$: condition = 0.611; time = 0.984) and (**B**) respiratory rate ($\eta^2_p$: condition = 0.605; time = 0.893) compared to the control (effect of condition, $P < 0.05$). The RTM reduce ventilatory equivalents (**C, D**) for oxygen ($\eta^2_p$: condition = 0.355; time = 0.939) and carbon dioxide ($\eta^2_p$: condition = 0.685; time = 0.962) compared to the control mask (effect of condition, $P < 0.05$). Sidak's Post Hoc Multiple Comparisons test: * $P < 0.05$ vs. Control Mask

increased from baseline during steady-state exercise and returned to near baseline following steady-state exercise ($P < 0.05$, main effect of time). $F_ECO_2$ was augmented during exercise and recovery while wearing the RTM compared to the control mask (Fig. 3B, $P < 0.05$, effect of condition). Heart rate was not different between the RTM and control mask (Fig. 3C).

Lastly, exercise resulted in a robust increase in $V_E$ and respiratory rate from baseline during steady-state exercise (Fig. 4A and B, $P < 0.05$, main effect of time) under both conditions. $V_E$ and respiratory rates were suppressed during exercise while wearing the RTM compared to the control mask ($P < 0.05$, main effect of condition). Ventilatory equivalents for $VO_2$ and $VCO_2$ decreased in the RTM compared to the control mask (Fig. 4C and D, $P < 0.05$, effect of condition).

## DISCUSSION

New training devices intended to improve athletic performance are continually developed and marketed within the fitness industry. Training masks that restrict airflow with proposed claims of mimicking high altitude environments or alternatively as respiratory training devices have increased in popularity among athletes and recreational exercisers despite limited evidence supporting their efficacy [10, 12, 18]. The primary findings of the present study were that resting and recovery ventilatory ($V_E$, respiratory rates, $V_E/VO_2$, $V_E/VCO_2$), metabolic ($VO_2$, $VCO_2$, RER), and $SpO_2$ variables were not affected by a moderate "altitude resistance" setting while wearing the RTM. Steady-state exercise at 50% of $VO_{2peak}$ while wearing an RTM led to a slight but significant decrease in $SpO_2$ in healthy, fit young adults. The RTM also increased $F_ECO_2$, increased RPE, and reduced $F_EO_2$ levels during steady-state exercise. These occurred at a lower exercise intensity and air resistance setting than those previously studied [10, 12]. The study's findings are further strengthened by the study design of measuring respiratory and metabolic parameters while participants were wearing an actual RTM and not a modified configuration [10, 14].

Previous studies have tested the metabolic and ventilatory responses to modified RTM configurations during exercise, but not at rest [10, 14]. We found that oxygen consumption and ventilatory measures of minute ventilation and respiratory rate were not different while wearing an RTM at rest. Interestingly, we did observe changes in $V_E/VCO_2$, but not $V_E/VO_2$. These findings might be related to increased $CO_2$ accumulation inside the RTM as a byproduct of exercise intensity and skeletal muscle metabolism. We found no difference at rest between the RTM and control mask using moderate inspiratory resistance. The lack of significance during rest could be due to using a lower "altitude resistance" setting. It is possible that higher inspiratory "altitude resistances" are needed to increase $VO_2$ and reduce ventilatory measures at rest, as passive airflow resistance devices are greatly influenced by respiratory rate [19]. During rest, the RTM did not reduce $SpO_2$. A reduction in $SpO_2$ at rest might suggest the possibility for hematological adaptations with the prolonged wearing of an RTM. This finding would be considered noteworthy for those seeking to gain benefits from hypoxemia exposure.

Slight hypoxemia during exercise was found. Interested readers might see this as a plausible ability to simulate a moderate altitude environment. Indeed, a physiological stimulus for hematological adaptations at altitude is caused by hypoxemia [2]. However, the RTM would need to create a similar reduction in blood oxygen saturation at rest to practically provide the user

adequate time at the equivalent physiological stimulus required to achieve the desired "altitude" adaptation. Prior literature examining rest and exercise at an altitude of 1,800 m [20] found resting $SpO_2$ (~96%) and exercising $SpO_2$ (~92%) levels much lower than that of the current investigation (resting $SpO_2$ ~98%; exercise SpO2 ~96%). This suggests the RTM used in the current investigation is unlikely to provide adaptations associated with reduced $SpO_2$ at the labeled "altitude resistance" setting. The observed reduction in $SpO_2$ during exercise in our study is counter to the findings of Jung and colleagues that found no reduction in $SpO_2$ at a similar exercise intensity (50% $VO_{2peak}$) and higher resistance setting (6,000 ft) [12]. This discrepancy in exercising $SpO_2$ may be explained by the limited number of $SpO_2$ measurements (a single measurement at 5 min of the 10 min cycling bout) made by Jung et al. (2019) compared to our study that measured $SpO_2$ every two minutes during exercise. These results can be further explained by the larger mean difference (i.e., ~1% reduction compared to control) detected in our study, which occurred after 5 min, thereby suggesting that exercise beyond 5 min might induce a slight hypoxemia. Our findings confirm that the significant reduction in $SpO_2$ can occur at a lower "altitude resistance" setting in parallel with reduced ventilation and increased $F_ECO_2$ accumulation. Importantly, hypoventilation or lowering alveolar partial pressure of $O_2$ has been demonstrated to reduce $SpO_2$ during exercise [21]. However, the small reductions in $SpO_2$ during exercise in our study might have limited application to promote adaptations similar to high altitude, and previous chronic exercise training studies using the RTM do not support hypoxemia related adaptations [13, 22]. Additionally, our reported reduction in $SpO_2$ can be achieved during higher intensity aerobic exercise without using an RTM [23].

Further, acute and chronic moderate to high altitude exposure leads to increased ventilation due to hypoxemia stimulating chemoreceptors in the absence of an exercise stimulus [24]. Conversely, the RTM reduced respiratory rate, $V_E$, and ventilatory equivalents ($V_E/VO_2$ & $V_E/VCO_2$) during steady-state exercise, but not at rest or recovery while at sea-level. Although the application of altering airflow resistance to improve performance in athletic and recreational settings is new, similar physiological mechanisms have been previously studied during exercise [25, 26], as well as in occupational health settings with the use of respirator masks [27–29]. These studies similarly reported reductions in respiratory rates and $V_E$ with increased inspiratory airflow resistance, contrary to standard responses to acute and chronic altitude exposure.

The observed hypoventilation in the current study also manifested as disturbances in expired fractional gases ($F_EO_2$ & $F_ECO_2$), but with slight reductions in $SpO_2$. Our study protocol required participants to maintain spontaneous breathing throughout exercise. Previous studies using voluntary hypoventilation through reduced breathing frequency reported hypoxemia and hypercapnia [21, 30]. Therefore, the mask may produce a compensatory breathing mechanism to prevent excess hypoxemia and hypercapnia during exercise. The compensatory mechanism might be caused by an elongated inhalation period that could allow for increased gas diffusion across the alveolar membrane [31, 32]. A prolonged inspiratory cycle due to increased airflow resistance might allow for increases in pulmonary blood volume and perfusion time, thus increasing time for gases to diffuse across the alveolar membrane. Nonetheless, the reduced pulmonary ventilation found here challenges the notion that an RTM simulates high altitude, as acute high altitude exposure increases respiratory rates and sympathoexcitation (increased heart rate, blood pressure, and regional vasoconstriction). While the RTM may lead to acute perceptual similarities (increased RPE) to breathing at altitude, the mechanisms by which the adaptations are stimulated at altitude are in opposition to those produced by the RTM.

RTMs cause excess $CO_2$ and decreased $O_2$ concentrations within the mask leading to altered inspiratory gas concentrations. These likely influence assessments of $VO_2$ and $VCO_2$ due to the mathematical dependence on standard atmospheric gas concentrations (i.e., normal atmospheric gas concentrations: 20.90% $O_2$; 0.04% $CO_2$) of inspired air. This does limit the ability to adequately determine changes in metabolism while wearing the RTM despite potential increases in respiratory muscle work caused by increased inspiratory resistance. Although our data suggest no difference in $VO_2$ between the RTM and control masks, it is possible that the alteration of inspiratory gas concentrations obscures the increased respiratory muscle oxygen consumption. With this limitation noted, Barbieri and colleagues (2020) found increased $O_2$ consumption during aerobic exercise while wearing a modified resistance valve configuration [14], which may suggest that the mask increases respiratory muscle work. This supports the notion that the RTMs primary training stimulus is increased respiratory muscle work and not decreased $SpO_2$ [13].

Respiratory muscle training and devices for respiratory muscle training have been well-studied in healthy and diseased populations [33]. Additionally, respiratory muscle training may have beneficial effects for improving performance in a hypoxic environment [34]. Respiratory muscle training methods in athletes are specific toward reducing respiratory muscle fatigue by improving respiratory muscle strength, endurance, and potentially blunting the metaboreflex [33, 35, 36]. Respiratory muscle training devices can be classified into three categories: passive flow resistance devices, dynamic adjusted flow resistance devices, and pressure threshold valve devices [19]. The RTM falls into the passive flow resistance device category. The design of the RTM allows for different "altitude resistance" caps to be attached to the mask. The smaller the orifice surface area for air to enter the mask, the larger the inspired resistance or "altitude resistance." Additionally, the resistance load created by the RTM is greatly influenced by inspiratory flow rate [19], which makes this style of respiratory training device difficult to prescribe a set training load and progression for exercisers attempting to reap the benefits of respiratory muscle training. Future studies may include protocols to control for respiration rate (e.g. biofeedback) and fluctuations in $O_2$ and $CO_2$ to normalize training across participants. The use of electromyography may aid in establishing RTMs in improving breathing mechanics, which may be implicated in improving ventilatory efficiency by improving respiratory muscle strength and endurance. This might help establish the mask as an inspiratory training device. Therefore, there could be the possibility for the RTM to be efficacious in improving performance in hypoxic environments, albeit, not as a simulator of hypoxic conditions.

## LIMITATIONS

The findings of the current study do come with limitations. First, both men and women were recruited to participate in the study despite known sex differences with pulmonary physiology [37]. The influence of sex may have introduced increased variability in our study, however, the within-participant design eliminates sex bias in the data. Statistical analyses with women excluded were performed. No statistical findings for the main effects of condition or time for any variable tested were altered. Few studies have included women, thus, the inclusion of women also helps to increase the ecological validity of the study. Second, we did not observe increased oxygen consumption despite the potential increase in respiratory muscle activation to overcome

the RTMs altitude resistance. It is possible that inspiratory gas concentrations within the mask obscured increased respiratory muscle oxygen consumption. Third, we were unable to examine the ratio between inspiratory and expiratory time to determine changes in breathing patterns despite significant reductions in minute ventilation and respiratory rate with the RTM during exercise. The plausible compensatory change in respiratory patterns would help to support $O_2$ and $CO_2$ exchange during exercise.

## CONCLUSION

In summary, the RTM exerts no influence on resting metabolism, pulmonary ventilation, and $SpO_2$. Moderate intensity exercise led to hypoventilation, slight hypoxemia, and increased RPE. The hypoventilatory response contributes significantly to the $CO_2$ accumulation while wearing the RTM, which plausibly alters pulmonary gas diffusion gradients linked to a slight reduction in $SpO_2$. These findings do not support the notion that RTMs produce similar levels of hypoxemia as occurs at moderate altitudes. However, RTMs may provide benefits to users as a respiratory muscle training device.

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