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Original Research Article

Ventilation can exclusively be used to predict ventilatory thresholds: a retrospective analysis

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ABSTRACT

Aim: We retrospectively analyzed a cohort of graded exercise tests (GXT) and compared ventilatory thresholds (VT1 and VT2) predicted from ventilation (PRED) to ventilatory thresholds derived from the gold-standard method using indirect calorimetry (TRUE). **Methods:** A cohort of 202 participants (132 women, 70 men; age range 18–69 years) completed GXT in the High Altitude Performance Laboratory at Western Colorado University between September 2014 and February 2020. Bland–Altman 95% limits-of-agreement were used to quantify the agreement between TRUE and PRED VT1 and VT2. **Results:** For VT1 time point detection, the mean differences between TRUE and PRED were -0.05 ± 1.28 min (95% CI, -2.56 to 2.46 min). For VT2 time point detection, the mean differences between TRUE and PRED were 0.10 ± 1.55 min (95% CI, -2.93 to 3.12 min). **Conclusion:** In this retrospective study, it was shown that modeling ventilation data elicited acceptably accurate estimates for VT1 and VT2 time point detection, workloads, and heart rates during both treadmill and cycle ergometer GXT. These novel findings are encouraging and provide critical preliminary data for the successful translation of the threshold-based training paradigm to a larger demographic of the population.

KEYWORDS: Aerobic Threshold, Anaerobic Threshold, Exercise Prescription, Fitness Assessment, Minute Ventilation, Threshold-Based Training, Threshold Detection.

Introduction

Heterogeneity in the response to exercise training first received attention in the 1980s[1] with a series of standardized studies investigating trainability of sedentary adults. Among these studies was an investigation into responses of maximal aerobic power (VO_2max) in which it was reported that

interindividual differences ranged from 5% to 88%[2]. Even though these original findings were reported over 30 years ago, substantial individual variability in response to prescribed exercise regimes remains a poorly understood phenomena. Nonetheless, it has been purported that a more individualized approach to the exercise prescription may enhance training efficacy and limit training unresponsiveness. For instance, it has been acknowledged as far back as the late 1970s that utilizing a relative percent method (i.e., % heart rate reserve [HRR]) to establish exercise intensity fails to account for individual metabolic responses to exercise[3]. Despite such findings, the relative percent concept remains the gold standard recommendation for exercise intensity[4].

It is believed that a more individualized approach to exercise prescription may better optimize training[5]. Indeed, it is both plausible and practical to think that an intensity set based on an individual's threshold measurement (i.e. ventilatory thresholds) will not only encourage more positive physiological adaptations, but may account for some of the variability in training responsiveness by taking into consideration individual metabolic differences. In recent years, our laboratory has consistently reported that when exercise intensity is titrated according to a threshold-based model (i.e., ventilatory thresholds), the prevalence of a favorable VO_2max training response (i.e., responders) is 100%[6-9]. In comparison, incidence of responders has ranged from 41.7% to 68.8% when the exercise intensity was 'standardized' or prescribed according to a relative percent method (i.e., % HRR)[6-9]. Collectively our findings suggest that design of individualized exercise prescriptions based on ventilatory thresholds will enhance training efficacy and limit training unresponsiveness.

A key factor underpinning the success of threshold-based training at eliciting positive training responsiveness in these above-mentioned studies is the accurate detection of individual ventilatory thresholds (i.e., VT1 and VT2) from graded, maximal exercise testing with concomitant collection of gas exchange data. However, in real world settings it may be more challenging and cost prohibitive for individuals to have regular access to metabolic testing in an academic or performance laboratory. As such, alternative options for valid detection of VT1 and VT2 are necessary to ensure translation of the threshold-based training paradigm to a larger demographic of the population. In the past decade, there has been an explosion in wearable technologies that can be used for guiding exercise training, including apps, smart watches, heart rate monitors, and other devices. A more recent addition to this wearable technology menu is the Tyme Wear smart shirt (https://www.tymewear.com/pages/home). This wearable technology integrates sensors that communicate wirelessly to smart phones. The sensors embedded in the shirt measure ventilation along with body movement and have the potential to detect

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VT1 and VT2 and guide threshold-based training. However, the validity of using ventilation to accurately detect VT1 and VT2 when compared to the gold-standard method of indirect calorimetry remains unknown. Therefore, the purpose of this study was to retrospectively analyze a cohort of maximal exercise tests from our laboratory and compare ventilatory thresholds predicted from ventilation exclusively to ventilatory thresholds derived from the gold-standard method of gas exchange data collected via indirect calorimetry.

Methods

Participants

A cohort of 202 participants (132 women, 70 men; age range 18–69 years) ranging from inactive to recreationally active to endurance trained were included in this retrospective study and completed a valid graded exercise test (GXT) in the High Altitude Performance Laboratory at Western Colorado University between September 2014 and February 2020. This study was approved by the Human Research Committee at Western Colorado University.

Experimental design

Prior to participation, each participant signed an informed consent form. All participants performed a GXT on either a cycle ergometer or treadmill to determine maximal heart rate and maximal oxygen uptake (VO_2max). Resting heart rate, height, and weight were assessed prior to maximal exercise testing. Ventilatory thresholds were determined from gas exchange data, including ventilation (VE), oxygen consumption (VO_2), and carbon dioxide production (VCO_2), obtained during GXT and considered the gold-standard (TRUE) VT1 and VT2 values. Ventilatory thresholds were also predicted from VE data obtained during GXT and considered the predicted (PRED) VT1 and VT2 values.

Gold-standard (TRUE) ventilatory thresholds derived from gas exchange data

Determination of both VT1 and VT2) were made by visual inspection of graphs of time plotted against each relevant respiratory variable (according to 15 s time-averaging). The criteria for VT1 was an increase in VE/VO_2 with no concurrent increase in VE/VCO_2 and departure from the linearity of VE. The criteria for VT2 was a simultaneous increase in both VE/VO_2 and VE/VCO_2[10]. All assessments were done by two experienced exercise physiologists. In the event of conflicting results, the original assessments were reevaluated and collectively a consensus was agreed upon. This method of ventilatory threshold determination is consistent with past work from our group[8, 9].

Predicted (PRED) ventilatory thresholds from ventilation

Determination of both VT1 and VT2 were made by visual inspection of graphs of time plotted against ventilation (according to 15 s time-averaging) using third order polynomial (cubic) modeling (R^2 = 0.9767). As assessed by visual detection, the apex of the first curvature was defined as VT1 and

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the apex of the second curvature was defined as VT2 (Figure 1). All assessments were done by two experienced exercise physiologists. In the event of conflicting results, the original assessments were reevaluated and collectively a consensus was agreed upon. The exercise physiologists were blinded to TRUE VT1 and VT2 when determining PRED VT1 and VT2.

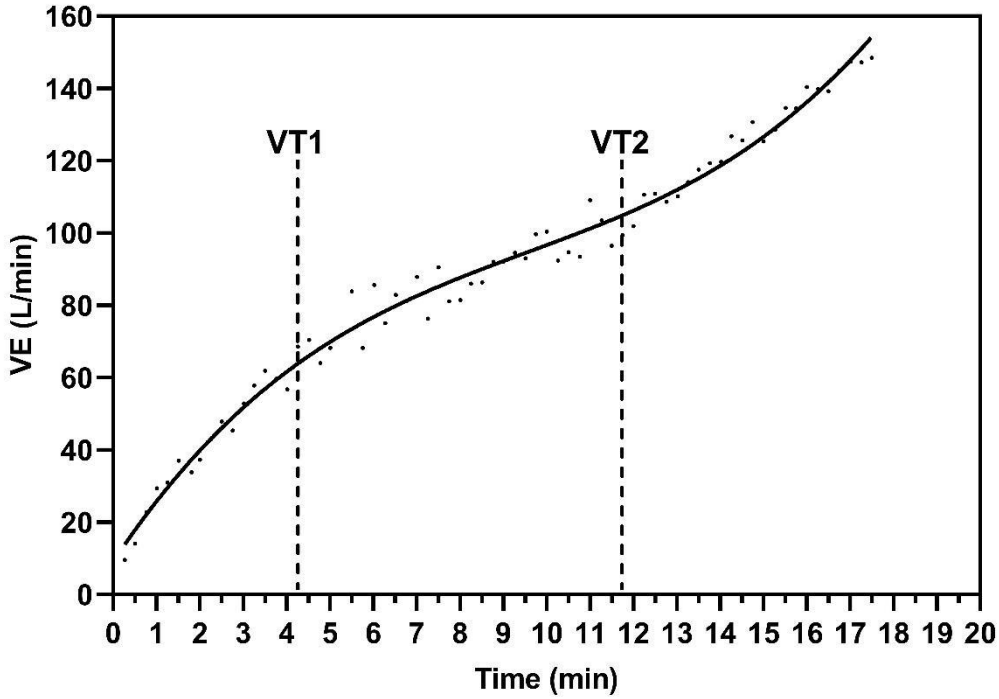

Figure 1. Example of third order polynomial (cubic) modeling on minute ventilation (VE) signal recorded during graded exercise test to determine predicted (PRED) first ventilatory threshold (VT1) and second ventilatory threshold (VT2), respectively.

Procedures

Anthropometric and resting heart rate measurements

Participants were weighed to the nearest 0.1 kg on a medical grade scale and measured for height to the nearest 0.5 cm using a stadiometer. The procedures for assessment of resting heart rate outlined elsewhere were followed[4]. Briefly, participants were seated quietly for 5 min in a chair with a back support with feet on the floor and arm supported at heart level. Resting heart rate was obtained via manual palpation of radial artery in the left wrist and recording the number of beats for 60 s.

Graded exercise testing

Participants completed GXT on either treadmill or cycle ergometer modalities and followed the respective protocols:

Treadmill protocol

Participants completed a 2 min warm up by walking for 2 min between 2.0 and 3.0 mph. Participants then walked or jogged at a self-selected and constant speed for the

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remainder of the GXT. Treadmill incline was increased by 0.5–1% each 30–60sec until the participant reached volitional fatigue. Treadmill workload (speed and incline) was subsequently converted to an equivalent ground zero speed for all further statistical analyses using an online lookup table: (https://www.hillrunner.com/calculators/treadmill-pace-conversions/).

Cycle ergometer protocol

Participants completed 2 min of pedaling at 50 W as a warm-up. During exercise, power output was increased in a step like manner equal to 5 W/30 s for women and 5 W/20 s for men to elicit volitional fatigue in approximately 7–11 min. Pedal cadence was maintained at 70–90 rev/min, with volitional fatigue representing a failure to sustain a pedal cadence greater than 40 rev/min.

Heart rate and gas exchange data collection

Participant heart rate was continuously recorded during the GXT via a chest strap and radio-telemetric receiver (Polar Electro, Woodbury, NY, USA). Expired air and gas exchange data were recorded continuously during the GXT using a metabolic analyzer (Parvo Medics TrueOne 2.0, Salt Lake City, UT, USA). Before each exercise test, the metabolic analyzer was calibrated with gases of known concentrations (14.01 ± 0.07 % O_2, 6.00 ± 0.03 % CO_2) and with room air (20.93%O_2 and 0.03 % CO_2) as per the instruction manual. Volume calibration of the pneumotachometer was done via a 3-Litre calibration syringe system (Hans-Rudolph, Kansas City, MO, USA).

Data processing and confirmation of maximal oxygen uptake

The last 15 s of the GXT were averaged – this was considered the final data point. The closest neighboring data point was calculated by averaging the data collected 15 s immediately before the last 15 s of the test. The mean of the two processed data points represented VO_2max. Maximal heart rate was considered to be the highest recorded heart rate in beats per minute (bpm) during the GXT. The criteria for the attainment of VO_2max were two out of three of the following: (1) A plateau (ΔVO_2 < 150 mL/min) in VO_2 with increases in workload, (2) maximal respiratory exchange ratio (RER) > 1.1, and (3) maximal heart rate within 15 beats/min of the age-predicted maximum (220—age).

Statistical Analyses

All analyses were performed using SPSS Version 25.0 (Chicago, IL) and GraphPad Prism 8.0. (San Diego, CA). The assumption of normality was confirmed by an examination of normal plots of the residuals in ANOVA models and Shapiro–Wilk tests[11]. Measures of centrality and spread are presented as mean ± SD. Paired t-tests were used to compare TRUE and PRED values for VT1 and VT2 time, treadmill workload (equivalent 0%-incline mph) or cycle ergometer workload (Watts), and heart rate, respectively. Bland–Altman 95% limits-of-agreement (LoA) were used to quantify the agreement (bias ± random error (1.96 × SD)) between the time, workload, and heart rate at TRUE and PRED VT1 and VT2 [Bland and

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Altman, 1986]. The probability of making a Type I error was set at $p<0.05$ for all statistical analyses.

Results

The physical and physiological characteristics of participants are presented in Table 1.

Table 1. Physical and physiological characteristics of the cohort.

Parameter	Women (n=132)	Men (n=70)	Overall (n=202)
Age (yr)	30.8 ± 14.5 [a]	29.8 ± 11.6	30.4 ± 13.5
Height (cm)	166.5 ± 6.8	179.2 ± 8.4	170.9 ± 9.6
Weight (kg)	66.6 ± 12.6	81.6 ± 19.0	71.8 ± 16.7
Body Mass Index (kg/m^2)	24.0 ± 3.8	25.4 ± 5.4	24.5 ± 4.5
Resting heart rate (bpm)	65.1 ± 7.0	62.3 ± 9.2	64.1 ± 7.9
Maximal heart rate (bpm	176.9 ± 4.2	182.4 ± 11.7	178.8 ± 14.6
VO_2max ($mL \cdot kg^{-1} \cdot min^{-1}$)	28.0 ± 7.1	46.4 ± 12.6	40.5 ± 10.8

[a] Values are mean ± SD.

Bland-Altman plots indicating the mean differences in VT1 and VT2 time point detection between TRUE and PRED and levels of agreement with 95% confidence intervals (CI) are illustrated in Figure 2. For VT1, the mean differences between TRUE and PRED were -0.05 ± 1.28 min (95% CI, -2.56 to 2.46 min). For VT2, the mean differences between TRUE and PRED were 0.10 ± 1.55 min (95% CI, -2.93 to 3.12 min).

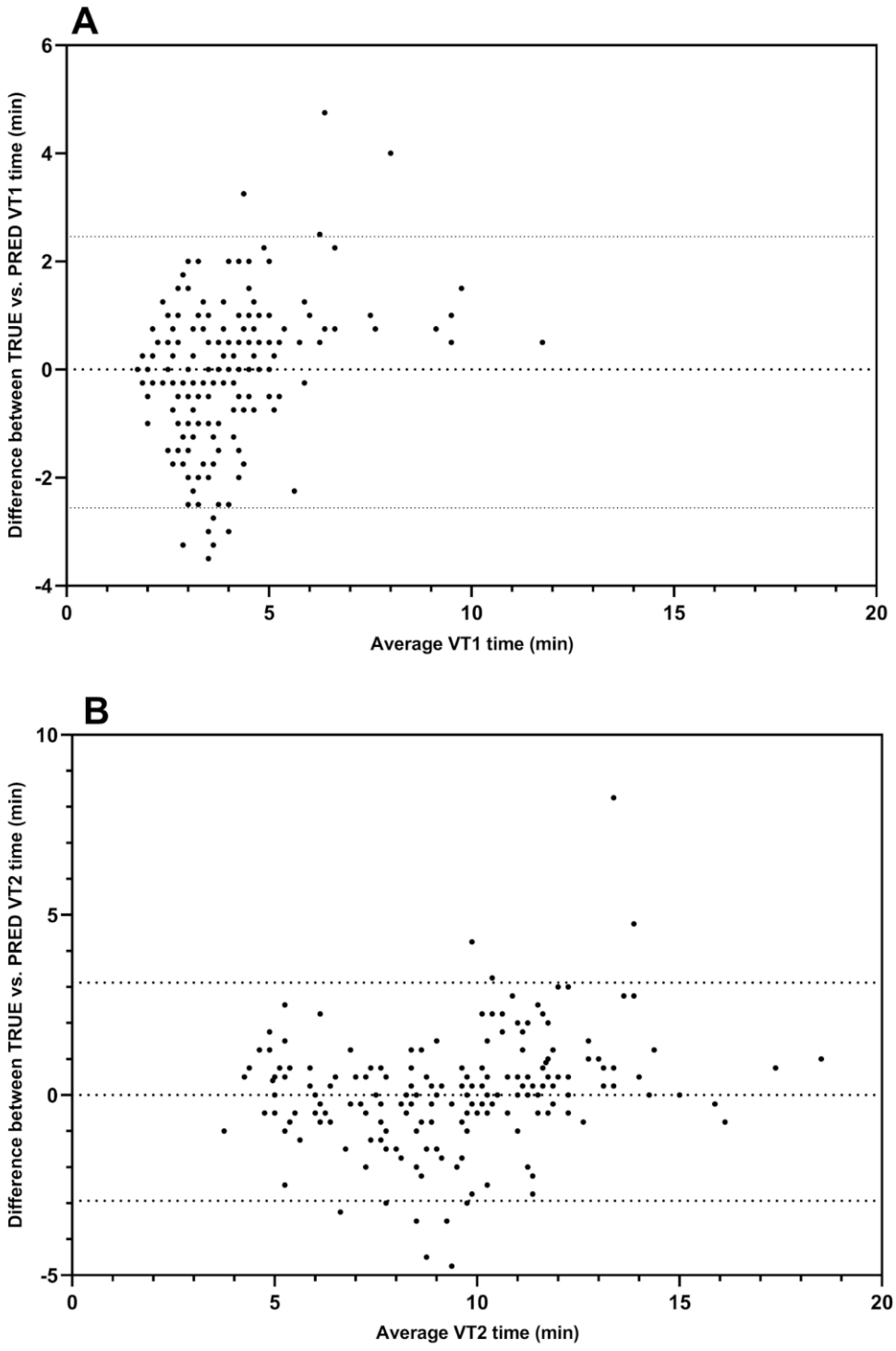

Figure 2. Narrowest 95% limits of agreement (bias ± (1.96 × SDdiff), min) between TRUE and PRED VT1 time (A) and VT2 time (B).

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Treadmill GXT

The VT1 and VT2 time point, workload, and heart rate values during treadmill GXT for TRUE vs. PRED are presented in Table 2. Paired t-tests revealed statistically significant ($p<0.05$) mean differences for overall VT1 time point detection between TRUE and PRED. Additionally, there were statistically significant ($p<0.05$) mean differences between TRUE vs. PRED for VT1 time point detection and VT1 workload in women. All other TRUE vs. PRED comparisons were not statistically significant ($p>0.05$).

Bland-Altman plots indicating the mean differences in treadmill VT1 and VT2 workload between TRUE and PRED and levels of agreement with 95% CIs are illustrated in Figure 3. For VT1, the mean differences between TRUE and PRED were -0.05 ± 0.40 mph (95% CI, -0.83 to 0.73 mph). For VT2, the mean differences between TRUE and PRED were 0.02 ± 0.32 mph (95% CI, -0.60 to 0.63 mph). Bland-Altman plots indicating the mean differences in treadmill VT1 and VT2 heart rate between TRUE and PRED and levels of agreement with 95% CIs are illustrated in Figure 4. For VT1, the mean differences between TRUE and PRED were -0.8 ± 7.0 bpm (95% CI, -14.5 to 13.0 bpm). For VT2, the mean differences between TRUE and PRED were 0.1 ± 6.3 bpm (95% CI, -12.2 to 12.4 bpm).

Table 2. The time point, workload, and heart rate values during treadmill GXT for TRUE vs. PRED VT1 and VT2 separated by sex and overall.

Parameter	Women (n=90)		Men (n=58)		Overall (n=148)	
	TRUE	PRED	TRUE	PRED	TRUE	PRED
VT1						
Time point (min)	3.85 ± 1.59 [a]	4.20 ± 1.01*	4.61 ± 2.38	4.75 ± 1.81	4.15 ± 1.97	4.42 ± 1.40*
Workload (mph)	4.31 ± 1.14	4.40 ± 1.03*	4.83 ± 1.36	4.83 ± 1.25	4.52 ± 1.25	4.57 ± 1.14
Heart rate (bpm)	130.3 ± 21.7	131.7 ± 20.2	136.5 ± 18.5	136.3 ± 17.5	132.7 ± 20.7	133.5 ± 19.2
VT2						
Time point (min)	10.00 ± 2.57	9.79 ± 1.86	10.80 ± 3.10	10.92 ± 2.49	10.31 ± 2.81	10.23 ± 2.19
Workload (mph)	5.71 ± 1.24	5.69 ± 1.23	6.56 ± 1.76	6.56 ± 1.76	6.04 ± 1.52	6.03 ± 1.52
Heart rate (bpm)	161.8 ± 20.8	161.5 ± 20.8	166.9 ± 15.7	167.1 ± 15.3	163.8 ± 19.1	163.7 ± 20.0

[a] Values are mean ± SD; * denotes statistically significant within-group difference.

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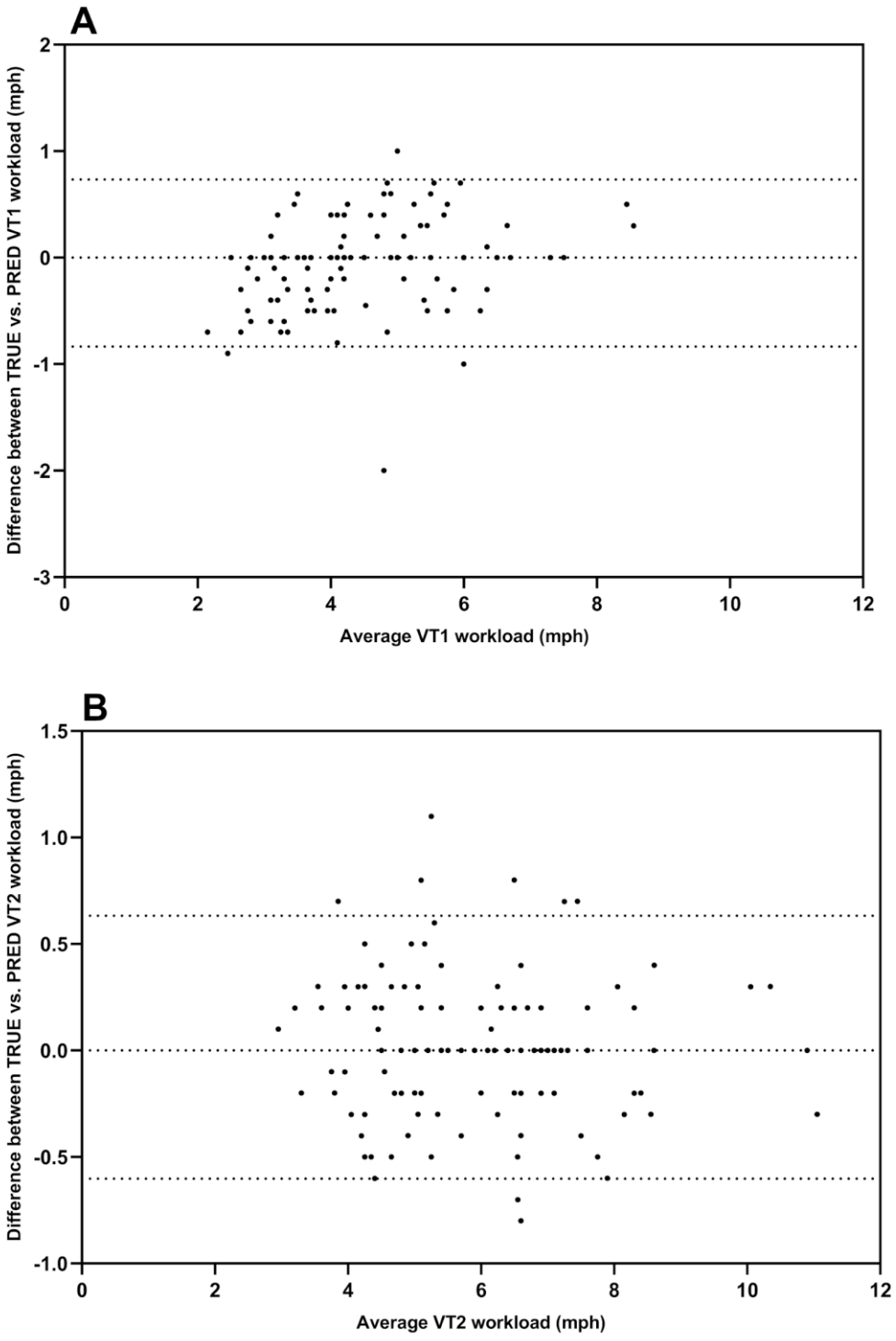

Figure 3. Narrowest 95% limits of agreement (bias ± (1.96 × SDdiff), min) between TRUE and PRED VT1 treadmill workload (A) and VT2 treadmill workload (B).

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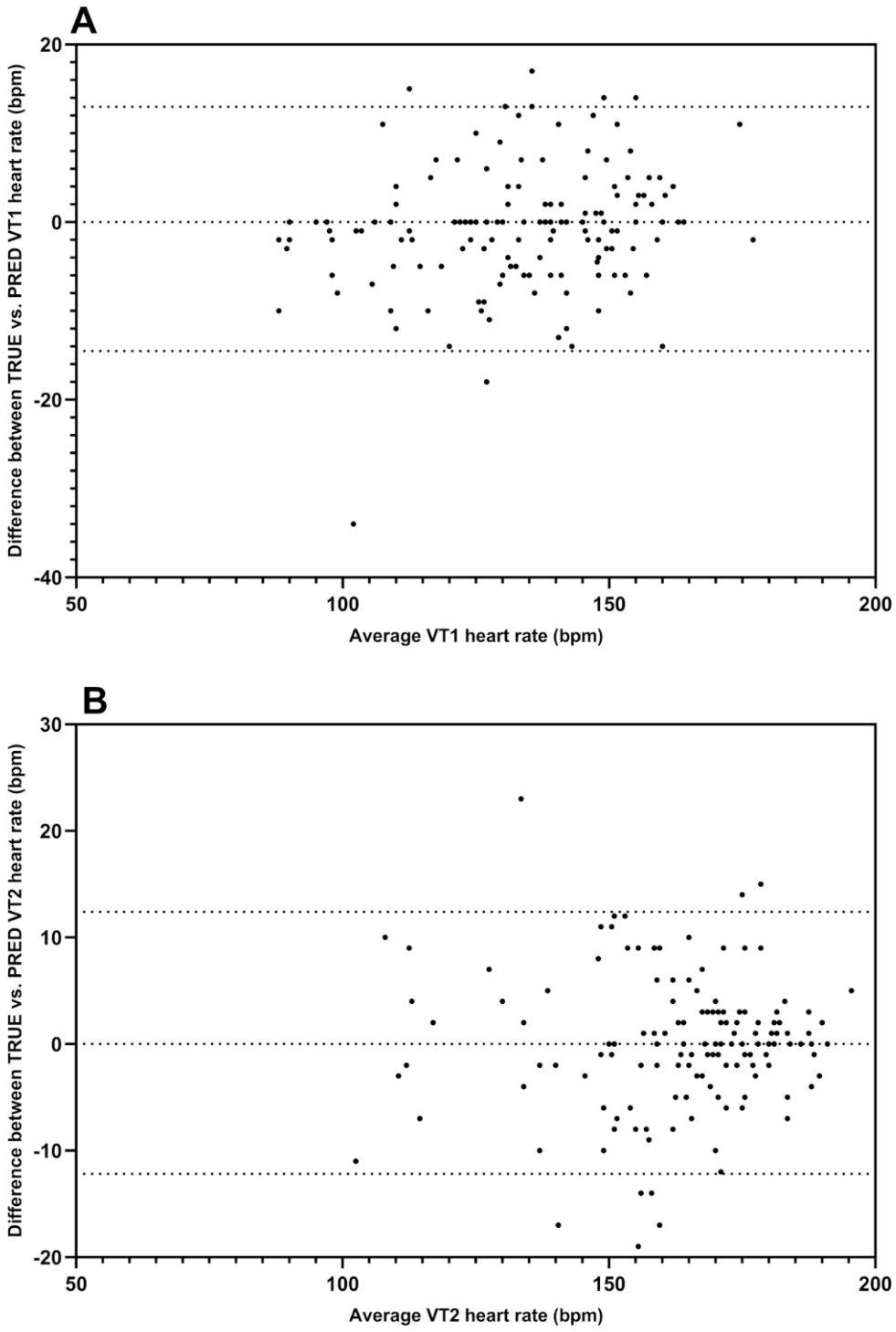

Figure 4. Narrowest 95% limits of agreement (bias ± (1.96 × SDdiff), min) between TRUE and PRED VT1 treadmill heart rate (A) and VT2 treadmill heart rate (B).

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Cycle Ergometer GXT

The time point, workload, and heart rate values during cycle ergometer GXT for TRUE vs. PRED VT1 and VT2 are presented in Table 3. Paired t-tests revealed statistically significant ($p<0.05$) mean differences for overall VT1 time point detection, workload, and heart rate between TRUE and PRED. There were also statistically significant ($p<0.05$) mean differences between TRUE vs. PRED for VT1 time point detection, workload, and heart rate in women. Additionally, there were statistically significant ($p<0.05$) mean differences between TRUE vs. PRED for VT1 workload in men. All other TRUE vs. PRED comparisons were not statistically significant ($p>0.05$).

Bland-Altman plots indicating the mean differences in cycle VT1 and VT2 workload between TRUE and PRED and levels of agreement with 95% CIs are illustrated in Figure 5. For VT1, the mean differences between TRUE and PRED were 12.3 ± 21.3 Watts (95% CI, -29.5 to 54.0 Watts). For VT2, the mean differences between TRUE and PRED were 4.9 ± 21.5 Watts (95% CI, -37.2 to 47.0 Watts). Bland-Altman plots indicating the mean differences in cycle VT1 and VT2 heart rate between TRUE and PRED and levels of agreement with 95% CIs are illustrated in Figure 6. For VT1, the mean differences between TRUE and PRED were 2.6 ± 5.6 bpm (95% CI, -8.4 to 13.6 bpm). For VT2, the mean differences between TRUE and PRED were 0.4 ± 5.9 bpm (95% CI, -11.3 to 12.0 bpm).

Table 3. The time point, workload, and heart rate values during treadmill GXT for TRUE vs. PRED VT1 and VT2 separated by sex and overall.

Parameter	Women (n=42)		Men (n=12)		Overall (n=54)	
	TRUE	PRED	TRUE	PRED	TRUE	PRED
VT1						
Time point (min)	3.05 ± 0.92 [a]	2.54 ± 0.65*	5.04 ± 1.63	4.38 ± 0.84	3.50 ± 1.38	2.94 ± 1.04*
Workload (Watts)	79.8 ± 18.0	70.2 ± 10.9*	128.3 ± 19.5	106.5 ± 32.9*	78.3 ± 23.4	90.6 ± 27.3*
Heart rate (bpm)	128.7 ± 14.0	126.3 ± 14.0*	130.1 ± 13.7	126.7 ± 13.3	129.0 ± 13.8	126.4 ± 13.7*
VT2						
Time point (min)	6.25 ± 1.54	6.02 ± 1.54	8.60 ± 1.60	8.81 ± 1.10	6.78 ± 1.83	6.64 ± 1.86
Workload (Watts)	145.5 ± 31.4	139.8 ± 26.7	227.5 ± 41.6	225.3 ± 48.4	163.7 ± 48.0	158.8 ± 48.2
Heart rate (bpm)	155.8 ± 12.7	154.8 ± 12.6	157.8 ± 16.4	160.1 ± 13.7	156.3 ± 13.5	156.0 ± 12.9

[a] Values are mean ± SD; * denotes statistically significant within-group difference.

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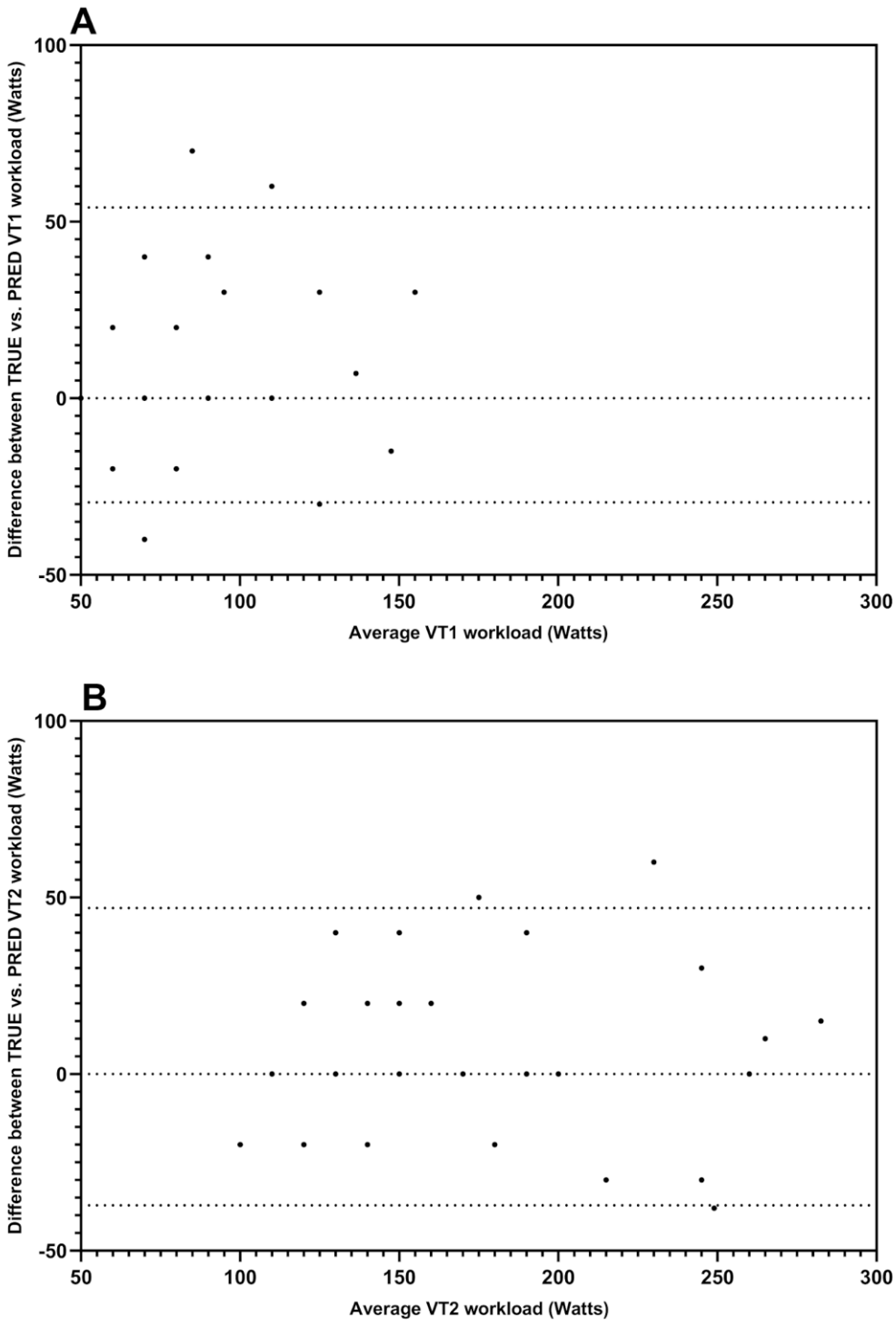

Figure 5. Narrowest 95% limits of agreement (bias ± (1.96 × SDdiff), min) between TRUE and PRED VT1 cycle ergometer workload (A) and VT2 cycle ergometer workload (B).

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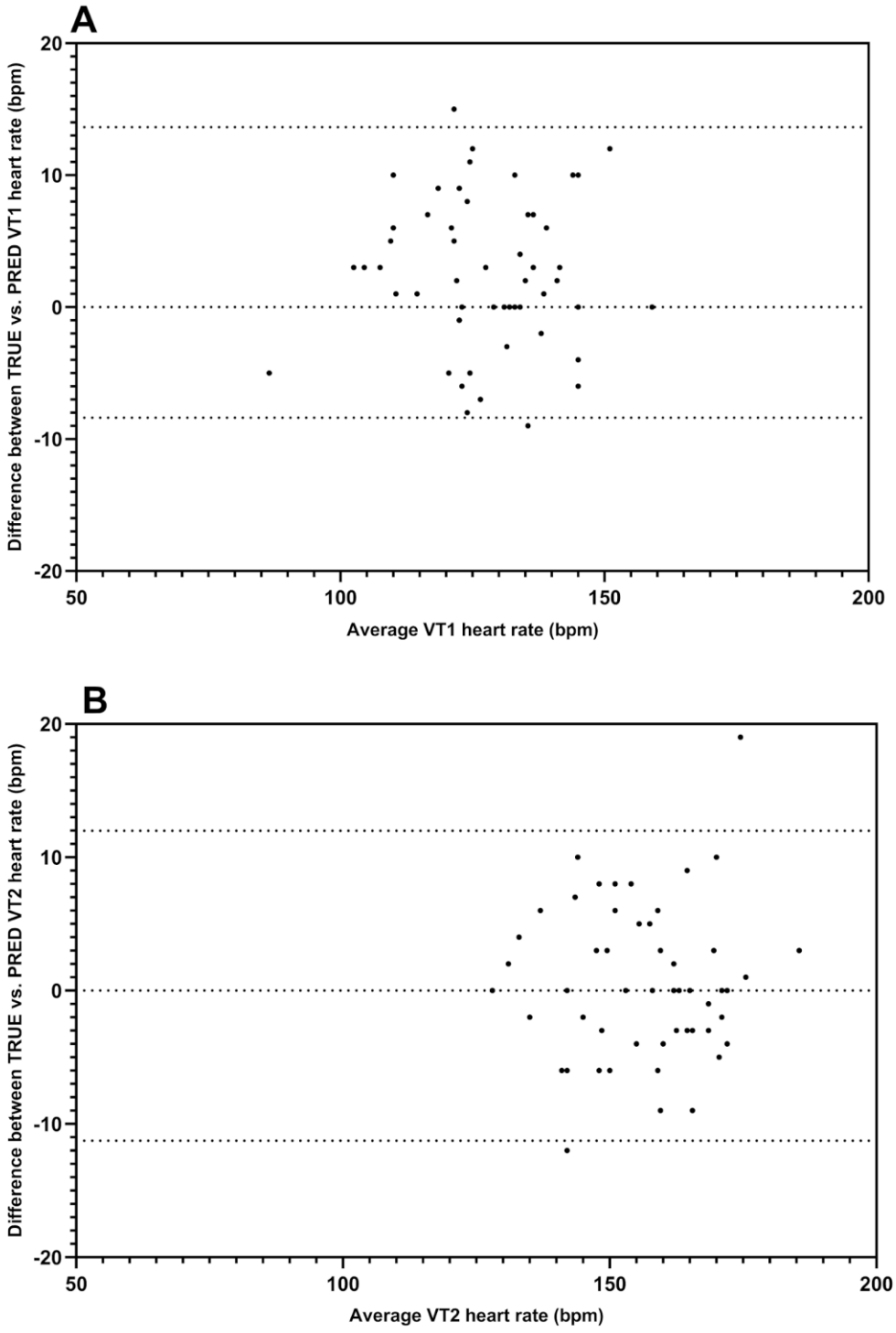

Figure 6. Narrowest 95% limits of agreement (bias ± (1.96 × SDdiff), min) between TRUE and PRED VT1 cycle ergometer heart rate (A) and VT2 cycle ergometer heart rate (B).

Discussion

This is the first study to investigate whether ventilation measurements alone could be used to detect accurately the ventilatory thresholds. In this retrospective study, it was shown that modeling ventilation data elicited acceptably accurate estimates for VT1 and VT2 time point detection, workloads, and heart rates during both treadmill and cycle ergometer GXT. These novel findings are encouraging and provide critical preliminary data for the successful translation of the threshold-based training paradigm to a larger demographic of the population.

Wide variability (-33.2% to +58%) in the individual response to exercise training has been previously described in the literature[12-15]. It has been suggested that the method of exercise intensity prescription may underpin the inter-individual variation to exercise training[16]. Those previous studies[5, 12, 14, 15] that have reported wide variability in the individual response to exercise training have used one of several relative exercise intensity methods, including %HRmax, %HRR, or %VO_2max. However, it has been demonstrated that these exercise intensity prescription methods elicit large inter-individual variation in the metabolic responses to exercise training[16, 17]. On this basis, it has been postulated that the individual variation in metabolic response will subsequently lead to differences in the overall homeostatic stress from each training session which will ultimately result in heterogeneity in the exercise training response (i.e., change in VO_2max). Alternatively, it has been suggested that use of a threshold-based method for establishing exercise intensity, such as ventilatory thresholds, might better normalize the metabolic stimulus for individuals with varying fitness levels[3, 18]. Indeed, the changes in ventilation that accompany exercise are actually precise indicators of the metabolic stress of exercising muscle.

While the regulation of ventilation during exercise is not completely understood, there is broad agreement of integrated regulation by a variety of systemic and central receptors that augment ventilation to maintain control of PCO_2 and blood pH. A key factor that reflects the cellular stress at VT1 and VT2 is increased metabolic production of CO_2 from the working muscle. Increases in PCO_2 as a result of accelerated tissue metabolism during exercise stimulates ventilation. Increased respiratory removal of CO_2 attempts to reduce arterial PCO_2 concentrations to maintain homeostasis. This negative feedback loop maintains a balance in CO_2 production and elimination. Along with elevated CO_2 production at VT1, more vigorous exercise intensities associated with VT2 accelerates hydrogen proton accumulation derived from bioenergetic processes that generate ATP which reduces blood pH. In an attempt to control pH, blood buffers attempt to neutralize

the hydrogen protons, but greater amounts of CO_2 are produced. It is thought that peripheral chemoreceptors located in the aortic and carotid bodies, and central chemoreceptors located in the cerebrospinal fluid in the brain[19, 20] sense increases in PCO_2 and hydrogen proton accumulation. The sensitivity of these chemoreceptors is very precise; they are able to detect very small changes in CO_2 and hydrogen protons on a breath-by-breath basis which are then transmitted to the respiratory control center in the medulla[21].

In addition to the peripheral and central chemoreceptors, other specialized sensors have been identified that communicate other aspects of metabolic stress associated with exercise (i.e., temperature, acid-base status, mechanical alteration of the muscle and lungs) to the respiratory control center. For example, muscle mechanoreceptors are likely to transmit signals related to more vigorous contractions, muscle length and tension, as well as PCO_2 and pH changes to the respiratory control center. Other muscle receptors detect precise changes in ion levels that augment ventilation[22, 23]. Findings from animal models indicate the existence of pulmonary stretch receptors[24] that might also play a role with rises in ventilation during exercise. Finally, it is likely that thermal receptors in the hypothalamus[25, 26] that also contributes to the exponential rise in ventilation at intensities associated with VT2. Collectively, an integration of many types of receptors sense and communicate changes in PCO_2 and pH during exercise to the respiratory control center with the result being ventilation deflection points at VT1 and VT2. A deflection point in ventilation reflects the level of exercise intensity in which the cell reaches a specific threshold of metabolic stress. Prescribing exercise around these metabolic thresholds, therefore, has the advantage of producing greater physiological adaptations to training because it can reveal what is going on at the cellular level.

The most accurate method for determining ventilatory thresholds is accomplished during a GXT by using indirect calorimetry. Although it is the most precise method, direct measurement and analyses of gas exchange data via indirect calorimetry may be unsuitable and unfavorable because the calorimetry equipment is expensive and requires trained laboratory personnel. Furthermore, laboratory-based ventilatory threshold procedures are time consuming, making the assessment less than ideal when testing a large number of individuals. All of the above factors limit accessibility of these tests, often only to professional or elite athletes, which can limit endurance performance assessment and correct training intensity prescription of many individuals. Therefore, for many decades, researchers have looked to alternative and simpler methods for

estimating ventilatory thresholds. One such method that is well-accepted in the scientific literature is the talk test[27, 28].

The talk test derives from the advice given by Professor Grayson, in 1939, to British mountaineers to "climb no faster than you can speak". The talk test is usually performed as a GXT where the subjects' ability to speak comfortably is evaluated. The first stage of a GXT where participants can no longer talk comfortably is labeled as equivocal (EQ) and equates to VT1. The stage of a GXT where participants can definitely not talk is labeled as negative (NEG) and equates to VT2. Considerable research has shown the talk test to be a surrogate of the ventilatory thresholds in a variety of populations[27, 28], and therefore a potentially viable alternative to standard methods of prescribing exercise training intensity[29]. For instance, in a cohort of active and healthy volunteers, Dehart-Beverley and colleagues (2000)[30] demonstrated no statically significant differences between heart rate and rating of perceived exertion (RPE) at VT1 and the EQ stage. Persinger et al. (2004)[28] showed the talk test approximated VT1 and VT2 on both treadmill and cycle ergometer. Rodriquez-Marroyo et al. (2013)[31] studied well-trained cyclists who performed two separate GXT: one with gas exchange data collected to determine ventilatory thresholds, and a second to perform the talk test and identify EQ and NEG stages. No statistically significant differences were found between the workload at VT1 vs. EQ and VT2 vs. NEG. For VT1, the mean bias between gas exchange and talk test heart rates were 3.0 ± 6.0 bpm. The mean bias ± 95% confidence interval of the between-method differences for workload and heart rate were found to be lower for VT2 vs. NEG than VT1 vs. EQ. Collectively, these previous results are consistent with our findings and taken together suggest modelling ventilation is a valid method for estimation of the ventilatory thresholds.

There are a few experimental considerations regarding the present study. First, as with all retrospective studies, there may have been an introduction of selection bias that could have contributed to the findings. Second, determination of both VT1 and VT2 were made by visual inspection of graphs of time plotted against ventilation using third order polynomial (cubic) modeling. It is possible that our findings may have differed with the application of other alternative statistical modeling techniques.

Conclusion

To our knowledge, this is the first study to investigate whether ventilation measurements alone could be used to detect accurately the ventilatory thresholds. Our findings provide important preliminary evidence that ventilation exclusively can be used to detect validly VT1 and VT2. Future research is needed to determine if wearable technologies that can measure

ventilation are reliable and valid. If such wearable technologies were demonstrated to be reliable and valid, this would subsequently permit a larger demographic of the population to test their thresholds without the need of expensive metabolic carts and laboratory time, which in turn would remove barriers and help promote the threshold-based training paradigm to the larger population.

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