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Effects of Deep Slow Breath Training on Performance and Recovery During High Intensity Interval Cycling

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EFFECTS OF DEEP SLOW BREATH TRAINING ON PERFORMANCE AND RECOVERY DURING HIGH INTENSITY INTERVAL CYCLING

By

Andrew David Brown

Accepted in Partial Completion
of the Requirements for the Degree
Master of Science

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Master's Thesis

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Effects of Deep Slow Breath Training on Performance and Recovery During High Intensity Interval Cycling

A Thesis
Presented to
The Faculty of
Western Washington University

In Partial Fulfilment
Of the Requirements for the Degree
Master of Science

by
Andrew David Brown
May 27, 2019

Abstract:

The present investigation sought to delineate the effects of a six-week deep slow breathing (DSB) program on measures of cycling performance (mean power: MP), recovery (heart rate recovery: HRR, and expired carbon dioxide: VCO_2), and pulmonary capacities (vital capacity: VC, forced expiratory volume: FEV_1, and maximum voluntary ventilation: MVV). Twenty male cyclists were divided into training (n=10) and control (n=10) groups, where the training group completed a six-week DSB program in addition to their own training while the control group completed no breathe training. Participants completed two testing sessions, one before and one after the six-week period. Testing sessions involved three repeated Wingate Anaerobic Tests (WAnT) with three minutes of passive recovery between each interval. MP was recorded for each WAnT while measures of VCO_2 and HRR were taken immediately following each WAnT. No significant ($p < 0.05$) differences were found between groups for any of the variables measured, while both groups exhibited increase MP in the second WAnT (T2) following the six-week training period (Treatment: pre: 516.30 ± 20.82 W versus post: 536.38 ± 20.62 W; p = 0.010; Control: pre: 549.93 ± 18.66 W versus post: 567.83 ± 18.44 W; p = 0.010). The results presented here suggest DSB provides no performance benefit relevant to recovery or pulmonary capabilities during high intensity interval cycling, beyond those which are incurred via endurance training.

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INTRODUCTION

Voluntary alterations in breath frequency, tidal volume, and lengths of the inhalation and exhalation components of the respiratory cycle have been utilized for centuries in yogic, qigong, and other meditative practices (Danucalov, Simões, Kozasa, & Leite, 2008; Goyal, Lata, Walia, & Narula, 2014; Pal, Velkumary, & Madanmohan, 2004b; Vinay, Venkatesh, & Ambarish, 2016). While an abundance of data supports their use in cardiovascular, mental, and autonomic nervous system health contexts, the potential benefit of these practices for the goal of enhanced sport performance is unclear. The reported benefits of apnea and deep slow breathing (DSB) include anti-nociceptive effects (Reyes del Paso, Muñoz Ladrón de Guevara, & Montoro, 2015; Ryan & Kovacic, 1966), increased vital capacity (VC) (Zelenkova & Chomahidze, 2016), hypercapnic and hypoxic tolerance (Bernardi, Gabutti, Porta, & Spicuzza, 2001; Lavin, Guenette, Smoliga, & Zavorsky, 2015; Roecker et al., 2014; Smith et al., 2014), buffering capacity (Joulia et al., 2003; Woorons et al., 2008), cardiac vagal tone (Bhargava, Gogate, & Mascarenhas, n.d.; Eckberg, 2003; Jerath, Edry, Barnes, & Jerath, 2006; Pal, Velkumary, & Madanmohan, 2004a; Telles, Nagarathna, & Nagendra, 1996; Vinay et al., 2016), work capacity (Hepburn, Fletcher, Rosengarten, & Coote, 2005; Porcari et al., 2016), enhanced blood pressure response (Anderson, McNeely, & Windham, 2009; Goyal et al., 2014; Monnazzi, Leri, Guizzardi, Mattioli, & Patacchioli, 2002), endogenous antioxidant defense (Joulia et al., 2003), reduced oxidative stress (Joulia et al., 2003), and altered endocrine profiles (Djarova, Ilkov, Varbanova, Nikiforova, & Mateev, 1986; Kox et al., 2014; Monnazzi et al., 2002).

Support for the use of breath training programs to enhance recovery and performance is provided by Woorons et al. (2008). These researchers observed attenuated exercise induced acidosis and increased HCO_3^- concentrations during running at 90% of predicted heart rate max following a four–week dynamic hypoventilation program. Maintenance of acid-base balance was paralleled by an

increase of maximum velocity of 0.5 km/hr in their breath-training group. Given the greater relative contribution of anaerobic energy systems in attaining maximum velocity, their results reflect that glycolytic metabolism may have been altered by their breath program and that anaerobic tests (i.e. 400m sprint or Wingate tests) would better elucidate augmented buffering capacities following breath training compared to exercise during which oxidative systems are dominant.

Another mechanism by which long DSB may enhance recovery during high intensity exercise (HIE) is modified glycolytic metabolism subsequent to repeated hypoxic and hypercapnic stress (Joulia et al., 2003; Lemaître, Joulia, & Chollet, 2010). Joulia et al. (2003) described attenuated rises in blood lactate, oxidative stress, decreased heart rate during static and dynamic apneas, and increased apnea durations following breath training in eight triathletes. These researchers suggest that increased mobilization and use of free fatty acids following breath hold training may reduce glycolytic energy production and subsequently decrease lactate concentrations during exercise. However, these researchers did not address adaptations to hypercapnic tolerance or buffering systems as potential mechanisms for improved apneic durations or attenuated increases in lactate production.

Tests of anaerobic capacity have been utilized as measures of athletic performance (Bogdanis, Nevill, Lakmoy, & Boobis, 1998; Hawley, Williams, Hamling, & Walsh, 1989; Rindom et al., 2016). Of these tests, the Wingate anaerobic test (WAnT) has been routinely used, given its ability to produce reliable and valid results and their correlative strength to sporting performance (Hachana, Attia, Nassib, Shephard, & Chelly, 2012; Langfort, Zarzeczny, Pilis, Nazar, & Kaciuba-Uscitko, 1997; MacDougall et al., 1998; Masterson, 1999; Richard Davison, Swan, Coleman, & Bird, 2000). Given the levels of metabolic acidosis that occur during WAnTs, they are also a useful method to test endogenous buffering capacities. As such, the WAnT is a suitable test that may elucidate the

effects of breath training on performance and recovery during interval exercise with a large anaerobic contribution.

While there is certainly heterogeneity within the literature, much of the data supports the use of hypercapnic or controlled frequency breathing conditions to enhance performance and potentially recovery, though few studies have specifically investigated its potential efficacy during exercise with primarily glycolytic energy contribution (Busch et al., 2012; Jerath et al., 2006; Joulia et al., 2003; Kox et al., 2014; Lavin et al., 2015; Lemaître et al., 2010; Woorons et al., 2008). Further, the specific effects of deep slow breath training on athletic performance have yet to be clearly elucidated. The aim of the present study was to address the research hypothesis that a DSB program would positively influence measures of cycling performance (mean power; MP), recovery (heart rate recovery: HRR, and expired carbon dioxide: VCO_2), and lung function (vital capacity: VC, forced expiratory volume: FEV_1, and maximum voluntary ventilation: MVV). A secondary purpose to the present work was to explore the relationship between VCO_2 and MP, with the hypothesis that VCO_2 would show a positive relationship with MP and partly explain the ability to maintain performance across repeated cycling sprints.

METHODS

Experimental approach to the problem

The study was a randomized control intervention where differences in interval sprint cycling performance were examined pre- and post-six-week DSB or no training intervention. Pre-training (PRE) testing was followed by a six-week intervention period. Subjects then completed post-training (POST) testing during which the pre-training tests were repeated.

Participants

The participants included 20 young, trained cyclists from Western Washington University (Bellingham, WA, USA) and training facilities in the surrounding area. Participants were selected randomly from those who respond to flyers posted in training facilities in Bellingham and social media posts (Facebook). Those selected for participation were divided evenly into to either training (n=10) or control (n=10) groups by age and cycling experience matching.

Exclusion criteria included previous lower-extremity injury within six months, the use of anti-inflammatory medication within the last six weeks, or cardiac or respiratory abnormalities. Participants were excluded from the study if they had consumed creatine, beta alanine, or citrulline malate within six months prior to the intervention (Crisafulli et al., 2018; Pérez-Guisado & Jakeman, 2010; Trexler et al., 2015). Participants were prohibited from consuming caffeine before testing sessions. Prior to participation in the current study, all subjects received and signed an informed consent form, which had been previously approved by the Western Washington University's Institutional Review Board, in accordance with the Department of Health and Human Services guidelines.

Procedure

Baseline measurements of height (cm), mass (kg) and pulmonary measures of VC, FEV_1, and MVV were taken upon arrival to the laboratory with a Pneumoscan S-301 (Vacumed, Ventura, CA). Participants changed into cycling clothing and were fit to the cycle ergometer. Seat height was individually adjusted to achieve a knee flexion angle of 25-30° for all participants and recorded for consistency between measures (Bini, Hume, & Croft, 2011). Handlebar height was adjusted to

achieve a trunk flexion angle of 30° (Ericson, Bratt, Nisell, Arborelius, & Ekholm, 1986). Measurements of bike fit were recorded for future testing. Participants were fit with an electric heart rate monitor and gas sampling mask for gas analysis throughout the test duration.

High-intensity Interval Exercise Procedure (HIE)

A Monark 894E cycle ergometer outfitted with accompanying software (Monark, Vansbro, Sweden), toe-clips, and heel straps was used for all WAnT tests. The participants completed a five-minute warm-up on the Monark cycle ergometer at self-selected cadence and resistance. Following warm up, peak cadence (rev/min) was established during a five second effort during which the participants pedaled as fast as possible. Following a 5-minute rest, subjects were then instructed to begin cycling at maximal intent to attain 80% of peak cadence. When 80% of peak cadence was achieved, a predetermined load equal to 0.075 kp per kg body mass was applied to the flywheel and the participant cycled at maximal effort for 30-seconds. The participants completed a total of three, 30-second Wingate (WAnT) sprints, each interspersed with 3 minutes of passive recovery (Francois Billaut, Giacomoni, & Falgairette, 2003; Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995; Bogdanis et al., 1998; Bogdanis, Nevill, Lakomy, Graham, & Louis, 1996).Verbal encouragement was provided to all participants during each sprint. The Monark Wingate software was used to measure and record MP performed during each sprint.

Carbon Dioxide Output (VCO_2)

Gas exchange was measured for two minutes immediately following each WAnT and recovery interval with a Parvo Medics TrueOne 2400 metabolic cart (Parvo Medics, Sandy, UT, USA). Given the relationship between bicarbonate buffering and endogenous CO_2 production , VCO_2 was taken as

a proxy for buffering capacity (Böning, Klarholz, Himmelsbach, Hütler, & Maassen, 2007; McGinley & Bishop, 2016; Röcker, Striegel, Freund, & Dickhuth, 1994).

Heart Rate Recovery

Heart Rate Recovery (HRR) was taken as the change in heart rate within the first minute immediately following each WAnT, and is associated with vagal tone and autonomic function in athletic and general populations (Goulopoulou et al., 2006; Halson, 2014; Hepburn et al., 2005; Seiler, Haugen, & Kuffel, 2007; Wyatt, Donaldson, & Brown, 2013). Heart rate monitors (Polar T31 Heart Rate Monitor, Polar Electro, Kempele, FI) were secured around the ribcage at the level of the xiphoid process and conducting surfaces were moistened. Heart rate recovery was tracked following the bout of exercise with a Polar FT4 training watch (Polar Electro, Kempele, FI).

Deep Slow Breathing Procedure

The DSB utilized was validated in a pilot study conducted by the researchers. The DSB program aimed to increase parasympathetic nervous system activity and buffering capacity via prolonged exhalation and post expiration apnea durations (Hepburn et al., 2005; Reyes del Paso et al., 2015; Vinay et al., 2016). The breath exhalation and hold durations were constructed progressively to allow familiarization to extended exhalation and breath hold while potentially maintaining parasympathetic stimulation. DSB progressions are presented in Table 1. During the first week, participants inhaled for a count of four, held breath for a count of four, exhaled for a count of four, and held breath for another count of four before repeating the breath cycle. One second was added to the inspiratory pause and expiration components of the breath cycle every week while 2 seconds were added to the expiratory pause every week to provide progressive overload. Following each 10-

minute training session, the participants completed three maximal post-expiratory breath holds with recovery durations indicated by return to normal breathing frequency. Participants were asked to use diaphragmatic breaths for all inhalations and exhalations. Instructional videos were included with written instructions for diaphragmatic breathing and breath holding techniques (Bruton et al., 2018). The Breath + Relaxation and Breath Training application (Dynamic App Design LLC) was utilized as a visual tool for tracking intended for weekly breathing progressions. Post-expiration breath holds were timed by the participants on self-selected timing devices (i.e. smartphone timers) and recorded. Participants were required to complete 10 minutes of DSB every day for six weeks. A maximum of 4 missed sessions was allowed across the six-week training period, after which the participant was dropped from the study.

Table 1. Deep Slow Breathing Progressions

Week	1	2	3	4	5	6
Inhalation (sec)	4	4	4	4	4	4
Hold (sec)	4	5	6	7	8	9
Exhalation (sec)	4	5	6	7	8	9
Hold (sec)	4	6	8	10	12	14

Three maximal post expiratory breath holds after each session

STATISTICAL ANALYSIS

All data analyses were completed with SPSS (SPSS; V. 25.0; SPSS, Inc., Chicago, IL, USA). Descriptive data were generated for each outcome measure and reported as mean ± SD. Separate (time X experimental group X test) mixed model ANOVAs with *a priori* significance set to $p \leq 0.05$ were utilized to determine pre-to-post differences across and within DSB and control groups for measures of MP and VCO_2. Paired t-tests were conducted to determine pre-to-post differences in measures of HRR and pulmonary measures (VC, FEV_1, and MVV). Tukey post-hoc tests were

performed if significant group X time interactions were observed. Calculations of partial eta squared (η_p^2) were included for effect size analysis. Pearson correlations were conducted between the MP of each WAnT (interval 1= T1, Interval 2 = T2, and interval 3 = T3) and the VCO_2 of the subsequent recovery interval, and for the MP recorded in WAnTs T2 and T3 and the VCO_2 of the previous recovery interval.

RESULTS

Participant demographics are presented in Table 2. No significant differences were found between groups for age, mass, or height. Specifically, the sample consisted of recreational mountain bike (n=9), recreationally aerobically trained (n=4), road bike (n=3), professional mountain bike (n=2), cyclocross (n=1), and track cycling (n=1) athletes. All participants in the control group were able to complete the prescribed testing before and after the tests while one participant in the training group was unable to complete the third interval in pre-testing, while another was unable to complete the third interval in post-testing.

Table 2. Participant Demographics (Mean ± SD).

	Control	Treatment
Age (yrs)	24.50 ± 4.50	26.1 ± 2.81
Height (cm)	181.73 ± 5.81	178.96 ± 7.15
Mass (kg)	80.47 ± 8.84	79.25 ± 10.09

Descriptive statistics for MP and VCO_2 are presented in Table 3-4. MP decreased significantly across each WAnT ($p<0.001$; $\eta_p^2= 0.857$) for both pre and post testing conditions. No 3-way interaction (group X time X test) was found for either MP or VCO_2. A significant time X test interaction was found ($p =0.010$) in which post testing MP was greater than pre in test 2 for both groups. There was a systematic decline in VCO_2 across each recovery interval for both groups (p

<0.001; η_p^2=0.797). No significant differences were found between groups for either MP (p =0.336; η_p^2= 0.058) or VCO_2 (p =0.10; η_p^2= 0.170) during any WAnT or recovery interval.

Table 3. Mean Power for each Wingate Test (T1-3) before and after the training period

	Control			Treatment		
	Test 1	Test 2	Test 3	Test 1	Test 2	Test 3
Pre-training Mean Power (W)	622.34± 54.68	549.93 ± 61.46	507.75 ± 83.87	622.44 ± 80.10	516.3 ± 88.50	449.81 ± 82.38
Post-training Mean Power (W)	624.96 ± 52.49	567.83 ± 61.83*	511.30± 72.60	618.24 ± 83.41	536.37 ± 53.44*	475.18 ± 61.86

* Significant differences between pre- and post-training mean power for test 2

Table 4. VCO_2 (Mean ± SD) during and after each Wingate Test (T1-3) before and after training period.

	Control			Treatment		
	Recovery Interval 1	Recovery Interval 2	Recovery Interval 3	Recovery Interval 1	Recovery Interval 2	Recovery Interval 3
Pre-training VCO_2 (L/min)	2.66 ± 0.24	2.27 ± 0.26	2.23 ± 0.33	2.51 ± 0.34	2.09 ± 0.26	1.94 ± 0.32
Post-training VCO_2 (L/min)	2.73 ± 0.26	2.33 ± 0.24	2.08 ± 0.27	2.45 ± 0.24	2.19 ± 0.32	2.05 ± 0.33

Descriptive statistics for HRR, VC, FEV_1, and MVV are presented in Table 5. No significant differences were found between groups for values of HRR (p =0.331; η_p^2= 0.059), VC (p =0.336; η_p^2= 0.058), FEV_1 (p =0.414; η_p^2= 0.037), and MVV (p =0.211; η_p^2= 0.086).

Table 5. Heart Rate Recovery, Vital Capacity, Forced Expiratory Volume, and Maximum Voluntary Ventilation.

	Control		Treatment	
	Pre	Post	Pre	Post
HRR (bpm)	31.56 ± 11.10	31.56 ± 10.79	42.88 ± 18.89	34.13 ± 17.96
VC (L)	5.30 ± 0.74	5.49 ± 0.90	5.45 ± 0.90	5.53 ± 0.88
FEV1 (L)	4.39 ± 0.43	4.36 ± 0.36	4.18 ± 0.45	4.25 ± 0.56
MVV (L/min)	182.70 ± 19.38	183.80 ± 23.03	168 ± 32.69	168.30 ± 34.88

Pearson correlations and p-values are presented in tables 6 and 7. Significant correlations were observed between the MP of each WAnT and the VCO_2 of the subsequent recovery interval before (T1: p<0.001, T2: *p* =0.001, T3: *p* =0.001) and after (T1: *p* =0.005, T2: *p* < 0.001, T3: *p* =0.002) the six-week training period for both groups. Significant correlations were also observed for the MP recorded in WanTs T2-3 and the VCO_2 of the previous recovery interval before (T2 / Recovery 1: *p* =0.014, T3 / Recovery 2; *p* =0.003) and after (T2 / Recovery 1: *p* =0.010, T3 / Recovery 2: *p* =0.017) the six-week training period for both groups.

Table 6. Pearson correlations between MP and VCO_2 before six-week intervention r-value.

		VCO_2		
		R1	R2	R3
T1	r-value	0.725 *	-	-
	p-value	<0.001	-	-
T2	r-value	0.54*	0.704*	-
	p-value	0.014	0.001	-
T3	r-value	-	0.642*	0.69*
	p-value	-	0.003	0.001

R1= Recovery interval 1, R2= Recovery Interval 2, R3= Recovery interval 3;
*Significantly different from pre ($p< 0.05$)

DISCUSSION

The aim of the present investigation was to examine the effects of a DSB program on measures of cycling performance, recovery, and pulmonary capacities. No significant differences were found between the training and control groups for any of the variables measured. Systematic decreases in both MP and VCO_2 were found across successive WAnT and recovery period, respectively, for both groups before and after the 6-week experimental period. These systematic changes in MP and VCO_2 were accompanied by large effect sizes. To the researchers' knowledge, this is the first investigation to address the effects of breath training on recovery

from high intensity anaerobic intervals, though numerous studies have sought to elucidate the relevance of breath training to sporting performance (Joulia et al., 2003; Lavin et al., 2015; Lemaître et al., 2010; Woorons et al., 2008).

Mean Power and VCO_2

The primary finding of the present study was the hypothesis that a DSB protocol would result in maintenance of MP across repeated WAnTs was not supported (Table 2). Moreover, the results demonstrated systematic reductions in MP and VCO_2 across the testing conditions, as would be expected due to increasing metabolic acidosis, reduction in endogenous buffering capacity, and subsequent reductions in glycolytic energy transfer (Baker, McCormick, & Robergs, 2010; Péronnet & Aguilaniu, 2006). The large effect sizes that accompany these data further illustrate the degree of change between intervals and indicate the cycling protocol elicited substantial reductions in the ability of the participants to maintain power production. A secondary finding of this investigation was that VCO_2 was unchanged in both groups but mirrored the systematic decrement in MP across each subsequent WAnT in both pre and post-testing conditions. Lemaitre, Joulia, and Chollet (2010) hypothesized that a primary potential benefit of apneic training may be improvements in muscle buffering capacity and subsequent reduction in acidosis. Given the relationship between HCO_3^- buffering systems and endogenous CO_2 production, it was expected that an increase in buffering capacity via HCO_3^- could be estimated utilizing measurements of VCO_2. However, the results did not demonstrate an increase in VCO_2 and thus it is likely that the DSB protocol did not augment HCO_3^- buffering systems. However, biochemical analyses (pH, HCO_3^-, and blood lactate) were not included in the design, so to state potential changes in any of these variables would be erroneous.

A second finding was that the hypothesis that MP and VCO_2 would display a significant positive relationship was supported. Pearson correlational analysis revealed significant positive relationships between the MP across the WAnTs and the VCO_2 in the subsequent recovery interval, as well as the MP recorded in WAnTs T2-3 and the VCO_2 of the previous recovery interval. These findings are in line with what is currently understood regarding the production of non-metabolic CO_2, with respect to HCO_3^- buffering, and suggest that MP is at least in part explained by the ability to produce greater CO_2 (Böning et al., 2007; Chicharro et al., 2000; McGinley & Bishop, 2016). These relationships suggest VCO_2 may be useful as a performance indicator to consider for athletes whose sport requires a significant proportion of energy transfer from glycolysis. Further research should investigate the efficacy of VCO_2 production as a predictive measure of exercise performance during endurance sport. Within the context of breath training, these relationships support the goals of breath training to increase levels of endogenous CO_2 production. As such, this measure should be utilized to determine the efficacy of future breath training programs, in addition to measures of exercise performance.

A factor that would largely influence CO_2 kinetics in a breath training protocol is manipulating respiratory dead space. Koppers, Vos, and Folgering (2006) elucidated the efficacy of tube breathing in eliciting hypercapnia in healthy subjects. In a training context, Smolka, Borkowski, and Zaton (2014) described increased arterial CO_2 and decreased blood pH during exercise at 60% VO_{2max} with an additional 1200 mL of respiratory dead space. Their results reflect those presented in the current study, in that their six-week training with additional dead space had no effect on VCO_2 or respiratory exchange ratio (RER) values in a cohort of healthy males, though the exercise conditions were not similar to the present study. Arterial CO_2 concentrations were not measured, so whether the degree of hypercapnia elicited in the protocol

used was similar to that of Smolka et al. (2014) is unclear. Additionally, the results presented here, in tandem with those described by Smolka et al. (2014), suggest that hypercapnic breath training does not alter carbohydrate metabolism, as proposed by Woorons (2008) and Joulia (2003).

While the protocol did not result in improvements in VCO_2 or measures of interval cycling performance, previous literature has revealed alternative findings for various physiological and performance measures. Lavin et al. (2015) reported decreases in 150m swim times and increases in running economy in their group of 18 recreational swimmers following a controlled breath-frequency program, while Porcari et al. (2016) demonstrated improvements in power at ventilatory threshold and power at respiratory compensation point following a program utilizing device guided breathing. However, the design of the training program presented by these authors differs significantly from that of the present investigation, so drawing definitive comparisons is difficult.

Fabrice et al. (2003) noted improvements in lactate kinetics, reduced oxidative stress, and changes in arterial concentrations of oxygen and CO_2 following their 12-week dynamic apnea program. These researchers reported increased arterial CO_2 and decreased lactate concentrations at the break point of maximal apneas following their program. These results may suggest that increased carbon dioxide production is an adaptation incurred via breath training. It is possible the increases in arterial CO_2 are explained by longer maximal apnea durations and greater hypercapnic tolerance, though they did not address these questions in their discussion. Additionally, the apneic conditions utilized by these researchers included dynamic apnea, in which the participants were actively exercising during the breath holds while the protocol in the present investigation utilized apneic durations at rest.

Woorons et al. (2008) demonstrated the efficacy of a 4-week hypoventilation protocol in eliciting maintenance of pH and HCO_3^- in a group of 15 male runners at 90% of maximum heart rate, though they did not report any significant changes in exercise performance during VO_{2max} and time to exhaustion tests. In their study, these researchers note while enhanced buffering subsequent to breath training may delay the onset of metabolic acidosis, it may not reduce it at maximal intensities, as their results showed no changes in pH or HCO_3^- in the recovery following their measurements of VO_{2max}. Given these findings, it is possible that the benefit of DSB training may be most apparent at sub-maximal intensities near lactate threshold. As such, future research on this topic should investigate the use of sub-maximal exercise intensities slightly above lactate threshold to further discriminate the potential for performance enhancement following DSB training (Röcker et al., 1994).

Previous researchers have utilized measurements of relative buffering capacity as a function of workload at ventilatory threshold and workload at respiratory compensation point during incremental ramp exercise (Chicharro, Hoyos, & Lucía, 2000; Röcker et al., 1994). Specifically, Chicharro, Hoyos, and Lucía (2000) calculated functional relative buffering capacity as the difference in workload at lactate threshold and respiratory compensation point in of group professional cyclists to elucidate the effects of endurance training on isocapnic buffering and hypocapnic hyperventilation. Their data demonstrated that endurance training had no effect on isocapnic buffering while the phase of hypocapnic hyperventilation decreased significantly. These findings suggest a viable methodology to quantify buffering at sub-maximal intensities that may be useful in later investigations on the effects of breath training

Another potential explanation for a lack of significant findings with respect to values of VCO_2, is that DSB training may augment intramuscular buffering via mechanisms distinct from

HCO_3^-. As noted by Péronnet and Aguilaniu, HCO_3^- buffering represents only a portion of intramuscular pH balance, and may only buffer ~25% of H^+ load during anaerobic exercise (Péronnet & Aguilaniu, 2006). If what these researchers argue is correct, total buffering capacity, as represented by HCO_3^-, histidine, carnosine, phosphocreatine, and ammonia, would need to be measured to capture a holistic representation of the effects of a training program that aims to maintain acid-base homeostasis.

An unexpected finding was an increase in MP in T2 for both groups. A learning effect for WAnTs has been described previously (Barfield, Sells, Rowe, & Hannigan-Downs, 2002). However, if a learning effect had occurred it would be expected to improve the MP values in T1 during the post testing as well. A likely scenario is that both groups experienced improvements in T2 due to improved pacing strategies (François Billaut, Bishop, Schaerz, & Noakes, 2011). Despite the prescribed maximal intensity, it is possible that the participants were able to attain more optimal pacing strategies in the second round of testing. However, one would expect a decrement in MP during T1 in combination with an increase in MP during T2, which was not observed. Another potential explanation is that the physiological readiness of all participants improved enough to result in an improvement in T2 but was not substantial enough to affect the values for MP achieved in T3.

Another interpretation of the results may suggest that training status across the annual training calendar may have influenced the values, given some data were collected during times of the year that may correspond to varying training volumes and intensities and subsequent physiological readiness (Manunzio, Mester, Kaiser, & Wahl, 2016). However, pre and post testing were completed within an 8-week period for each subject, so decrements or improvements in performance would have been relative to their current state of training,

regardless of season. Another potential factor that likely influenced the data were the heterogeneity in the discipline of cycling or training across the sample population (Craig & Norton, 2001; Hays, Devys, Bertin, Marquet, & Brisswalter, 2018; Richard Davison et al., 2000). However, it would appear efforts to homogenize the treatment and control groups were effective, as no differences were found in any of the measured variables before the 6-week training period.

Heart Rate Recovery

The results showed that six weeks of DSB training resulted in no change in HRR. Jerath et al. (2006) explain deep slow breathing results in parasympathetic dominance through activation of mechanosensitive tissues and subsequent hyperpolarization of lung tissue, which induces synchronicity between the brainstem and hypothalamus. Eckberg (2003) provides further elucidation for the mechanisms responsible for an increase in parasympathetic dominance through elongation of the exhalation component of the respiratory cycle. Given the ability of slow breathing to elicit acute and chronic changes in vagal tone, HRR were utilized as a measure of parasympathetic activity and reactivation (Bhargava et al., 1988.; Eckberg, 2003; Jerath et al., 2006; Pal et al., 2004; Telles et al., 1996; Vinay et al., 2016). Unfortunately, technical failures resulted in the reduction of the sample in the training group (n=8) so only HRR data for the first interval was analyzed. No significant differences were found between or within groups for HRR following this DSB protocol.

A potential reason for a lack of significant findings for changes in HRR may be attributable to the trained state of the participants, as chronic endurance training results in increases in parasympathetic activity and decreases in sympathetic activity at rest (Carter, Banister, & Blaber, 2003). It is possible that the stimulus incurred from the DSB program was

not sufficient to drive adaptations that would further increase vagal tone. Had the participants been of a lesser trained state, it is possible that larger changes in HRR would have been observed, though contradictory data have been published elsewhere (Michaelson et al., 2019) . It is important to note that while the statistical analyses revealed no significant differences between groups with respect to HRR, a substantial decrease in HRR (-8.75 bpm) was recorded in the experimental group while minimal change was recorded in the control group. It is possible the variability in HRR response to training was large (shown by large standard deviations) in this group may have masked any potential effect of training.

Pulmonary Measures

No differences were noted between groups for any of the measurements of lung volumes. Previous research has demonstrated the efficacy of breath-hold and diaphragmatic breath training in eliciting increases in VC (Ferretti, 2001; Walterspacher et al., 2011; Yong, Lee, & Lee, 2017). The pretraining values of VC provided by Yong et al. (pre=5.32 ± 1.4 L, post = 6.05 ± 1.3 L) are similar to those reported in the current work, while those reported by Walterspacher et al. in their sample of breath-hold divers are larger (6.2 ± 0.6 L) (Walterspacher et al., 2011; Yong et al., 2017). Greater ventilatory function has been reported in endurance athletes compared to power trained and sedentary individuals (Durmic et al., 2017). The values of VC (5.37 ± 0.80 L), FEV_1 (4.59 ± 0.69 L), and MVV (160.72 ± 35.41) provided by Durmic et al. (2017) are similar to those reported here. Given their findings that endurance athletes exhibited significantly larger values for VC and MVV compared to sedentary individuals, it is possible the breath training stimulus was not sufficient to elicit further adaptations that would improve ventilatory function beyond those incurred by endurance training.

LIMITATIONS

While attempts to homogenize the sample appeared to be effective, greater specificity could be utilized in recruiting a cycling cohort. Recruiting from a single discipline (mountain, road, cyclo-cross, etc.) would further reduce the potential for the confounding factors of the adaptations specific to each of these sub-categories of cycling. Another factor that was not controlled was the effect of season. Data collections were completed over the course of a nine-month period, during which the training volumes of those who completed the training in the late summer and early fall may have been larger than those completed in the winter. By requiring a minimum volume of cycling training, an attempt was made to mitigate this effect. The chosen testing methodology was based on the work of previous researchers who suggested investigation on the effects of breath training at intensities and time domains during which the proportion of energy transfer from glycolysis is relatively larger than those of PCr or oxidative systems (Woorons et al., 2008). Future investigations should utilize sub-maximal protocols that may more accurately quantify the potential effects of breath training.

Conclusions

The results presented here suggest DSB training provides no performance benefit during high intensity interval cycling beyond those which are already stimulated by endurance training, considering the measures of exercise performance utilized. Similarly, the results demonstrate no benefit during the recovery from sprint efforts, with respect to parasympathetic reactivation. While these results are most clearly relevant to repeated sprint efforts with a significant contribution from glycolytic energy systems, their relation to sustained efforts near lactate threshold remain unclear. Given these results, use of DSB training as an effective mode of

preparation for athletes who compete at intensities which correspond to single or repeated glycolytic efforts (400m run, 50m swim, etc) is not warranted.

It would appear DSB training appears to provide no performance or recovery enhancement in trained male cyclists during repeated WAnTs, while further research utilizing ramp exercise testing procedures may more accurately represent the potential effects of DSB training at varying levels of exercise intensity. These findings add to the existing body of knowledge regarding the relationship between anaerobic performance and CO_2 production during high-intensity interval exercise and provide a basis for further research on the efficacy of breath training in the context of sporting performance.

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Review of literature: Effects of breath training on exercise recovery

Introduction

Voluntary alterations in breath frequency, tidal volume, and lengths of the inhalation and exhalation components of the respiratory cycle have been utilized for centuries in yogic, qigong, and other meditative practices (35,62,79). While an abundance of data supports their use in cardiovascular, mental, and autonomic nervous system health contexts, the potential benefit of these practices for the goal of enhanced recovery and subsequent athletic performance is unclear (1,27,42,45,56,62,82,90).

The reported benefits of apnea and deep slow breathing (DSB) include anti-nociceptive effects (Reyes del Paso et al., 2015; Ryan & Kovacic, 1966), increased vital capacity (VC) (Ferretti et al., 2012), hypercapnic and hypoxic tolerance (Bernardi et al., 2001; Lavin et al., 2015; Roecker et al., 2014; Smith et al., 2014), buffering capacity (Joulia et al., 2003; Woorons et al., 2008), cardiac vagal tone (Bhargava et al., n.d.; Eckberg, 2003; Jerath et al., 2006; Pal et al., 2004a; Telles et al., 1996; Vinay et al., 2016), work capacity (Hepburn et al., 2005; Porcari et al., 2016), enhanced blood pressure response (Anderson et al., 2009; Goyal et al., 2014; Monnazzi et al., 2002), endogenous antioxidant defense (Joulia et al., 2003), reduced oxidative stress (Joulia et al., 2003), and altered endocrine profiles (Djarova et al., 1986; Kox et al., 2014; Monnazzi et al., 2002). While there is certainly heterogeneity within the literature, much of the research supports the use of hypercapnic or controlled frequency conditions to enhance performance and potentially recovery during or between training or competition efforts (Engan, Richardson, Lodin-Sundström, van Beekvelt, & Schagatay, 2013; Joulia et al., 2003; Lemaître et al., 2010; Schagatay, van Kampen, Emanuelsson, & Holm, 2000).

The aim of the present review is to explore the primary physiological mechanisms relevant to potential adaptations following DSB and apnea-training including increased vagal tone, decreased chemosensitivity, anti-nociception, and endocrine responses. Applied training studies utilizing breathing within the context of sport performance are evaluated. Methodical considerations for future studies regarding the efficacy of breath training on sport performance are presented given the specificity of adaptations purported across the literature.

Physiological mechanisms to explain increased performance

Vagal Tone. The relationships between vagal tone, heart rate, and exercise capacity are well documented (Anderson, 1998; Anderson et al., 2009; Delapille et al., 2001; Hepburn et al., 2005; Jerath et al., 2006; Kiviniemi et al., 2014; Machhada et al., 2017; Monnazzi et al., 2002; Pal et al., 2004b; Vinay et al., 2016; Walterspacher et al., 2011; Wang et al., 2016; Zelenkova & Chomahidze, 2016). One early study that illustrates the chronic effects of breath training on cardiac activity is presented by Bhargahva et al. (Bhargava et al., n.d.). These researchers investigated the effects of a four-week pranayama breath-training program on indices of heart rate and blood pressure during rest and extended breath holding in ten young male volunteers. Following their program, they reported decreases in inspiratory ($p<0.05$) and expiratory ($p<0.001$) heart rates, systolic and diastolic blood pressure ($p<0.01$), and differential changes in galvanic skin resistance, a measure of sympathetic activity. These researchers explain the attenuated tachycardia during extended breath holding was likely subsequent to enhanced parasympathetic vagal tone given the findings of previous research, though their disparate results for galvanic skin resistance make it difficult to make this conclusion.

The findings of the previous study are reflected in a more recent investigation presented by Pal et al. (Pal et al., 2004b). The researchers describe enhanced parasympathetic activity as measured by significantly lower resting heart rates (pre 73.61±10.69 versus post 65.02±8.02; $p<0.05$) in 30 male graduate students. These researchers also report lower heart rates immediately following standing (pre 108.28±9.36 vs. post 101.18±8.45; $p<0.05$), and conclude their results demonstrate enhanced vagal tone, given previous findings that the rise in heart rate immediately following standing is an indication of autonomic nervous system activity. However, this work only addresses acute modulations in vagal dominance and thus its relevance to chronic adaptations may be limited.

Further support for the relationship between long slow breathing and enhanced sympathovagal balance is provided by Santaella et al. (Santaella et al., 2011) in their investigation of a four-week yogic breathing program in 19 (14 control, 15 training) elderly patients. The primary findings of their study revealed the yoga-breathing group had significant decreases in low frequency component of heart rate variability (control: pre 40 ± 13 v vs. post 41 ± 13 v; yoga breathing: pre 40 ± 11 v vs. 27 ± 8 v; $p=0.001$). These results demonstrate a shift towards parasympathetic dominance in autonomic modulation of cardiac rhythmicity. Given the population sampled in this study, replication of the efficacy of their breath-program may be limited to elderly populations.

One comprehensive theory that aims to describe the autonomic alterations is provided by Ravinder et al. (Jerath et al., 2006) who describe that deep breathing induces a shift of the autonomic nervous system towards parasympathetic dominance via stretch induced activation of the slow adapting mechanoreceptors and generation of hyperpolarization currents from stretch of lung tissue, which result in synchronization of the brainstem and hypothalamus. These alterations

result in parasympathetic dominance causing decreased heart rate, blood pressure, and oxygen consumption. Further elucidation of this phenomena is provided by Eckberg (Eckberg, 2003) who describes that respiratory activity modifies preganglionic vagal and sympathetic motoneuron membrane potentials. As such, elongation of the expiratory portion of the breath cycle results in vagal dominance and sympathetic withdrawal while the opposite occurs during inspiration. A simple example of such phenomena is respiratory sinus arrhythmia, during which heart rate accelerates during inspiration and decelerates during expiration (Yasuma & Hayano, 2004). The role of this interaction between the cardiovascular and respiratory systems may be explained by potential energy conservation during expiration when gas diffusion across alveolar membranes decreases, thus negating the necessity for increased perfusion to the pulmonary capillary bed (Yasuma & Hayano, 2004).

Sympathetic Tone. While decreasing breath frequency and elongation of the expiratory component of the respiratory cycle may increase vagal dominance, increasing breath frequency through hyperventilation elicits an acute increase in sympathetic nervous system activity. Läderach and Straub (Läderach & Straub, 2001) investigated the effects of voluntary hyperventilation on glucostatic hormones in eight healthy males. During the 20 minutes of hyperventilation, these researchers observed significant ($p<0.05$) increases in plasma free fatty acids (0.35 ± 0.18 mmol/ L vs. 0.345 ± 0.18 mmol/ L; $p=0.01$), insulin (5.5 ± 2.4 mU/ L vs. 8.7 ± 2.7 mU/ L; $p=0.03$), cortisol (350.6 ± 65.4 nmol/ l vs. 429.5 ± 109.7 nmol/ l; $p=0.01$), glucagon (91.0 ± 33.3 pg/ml vs. 100.9 ± 28.8 pg/ml; $p=0.02$), adrenaline (123.0 ± 63.6 pmol/l vs. 194.0 ± 88.9 pmol/l; $p=0.01$), noradrenaline (557.2 ± 256.9 pmol/l vs. 480.6 ± 201.1 pmol/l; $p=0.01$), and heart rate (58 ± 6 bpm vs. 60 ± 6; $p=0.01$). While these authors do not address sympathetic activation specifically, elevations of catecholamine concentrations would be indicative of

increased sympathetic activation. However, these results reflect only acute modulation of these hormones, and consequently their role in chronic adaption is unclear.

Similarly, Telles et al. (Telles et al., 1996) noted increased sympathetic activation in 12 participants (four males, eight females) following 45 minutes of rapid yogic breathing. These researchers observed significant ($p<0.01$) increases in sympathetic tone as measured by decreased skin resistance (treatment: 461.3 ± 312.0 kohms to 183.0 ± 159.2 kohms; control: 440. ± 328.0 kohms to 306.0 ± 205.7 kohms), increased systolic blood pressure (treatment: 100.6 ± 9.8 mmHg to 110 ± 9.6 mmHg; control: 104.5 ± 10.1 mmHg to 101.7 ± 10.9 mmHg), oxygen consumption (treatment: 257.4 ± 54.0 mL/min to 301.1 ± 51.1 mL/min; control: 251.65 ± 40.0 mL/min to 205.3 ± 59.2 mL/min), and digital pulse volume (treatment: 7.0 ± 2.0 mm to 3.8 ± 1.8 mm; control: 6.6 ± 4.6 mm to 6.7 ± 5.0 mm). However, these authors do not mention potential chronic adaptations to this style of breathing, nor do they give implications for populations outside obese and hypertensive populations. Consequently, the relevance of these data to athletic performance is unclear.

Further demonstration of voluntary sympathetic activation through hyperventilation is provided by Kox et al. (Kox et al., 2014). These researchers subjected participants to mild endotoxemia following 5-9 days of voluntary hyperventilation, cold baths, and meditation to address the effects of these modalities on sympathetic activation and attenuation of the innate immune response. Their results demonstrate significantly larger basal epinephrine in their training group (1.02 ± 0.22 vs. 0.35 ± 0.06 nmol/L, $p = 0.007$) and a rise in epinephrine during hyperventilation (up to 5.3 nmol/L). These observations were paralleled with a significant enhancement of immune function and attenuation of the immune response of endotoxin, as measured my increased leukocytes, neutrophils, and monocytes, decreased TNF-α (53% lower),

IL-6 (57% lower) and IL-8 (51% lower) levels, and increased IL-10 (194% higher). While the immune response described by these researchers is beyond the scope of the present review, their findings regarding the trainable activation of the sympathetic nervous system are intriguing and potentially relevant to athletic performance given the inflammatory and immune processes in muscle remodeling (Aoi et al., 2004; Peake, Neubauer, Gatta, & Nosaka, 2016; Weisleder et al., 2014). There are, however, methodical issues to consider in reviewing this article given their training protocol was not comprised only of hyperventilation training, but also included cold bathing and meditation. Consequently, the relative roles of these other factors on their findings are potentially limiting to their application.

While the bulk of these studies do not address the effect of enhanced vagal or sympathetic tone on exercise *per se*, the roles of each during sporting performance have been clearly elucidated (Coote, 2010; Machhada et al., 2017). However, further research is warranted to determine whether the vagal or sympathetic adaptations subsequent to breath training may be beneficial to performance in athletic and performance contexts.

Chemosensitivity and energy systems

Another mechanism by which breath training may influence sport performance is via altered chemo-sensitivity to hypoxia, hypercapnia, and maintenance of acid-base balance, given repeated reductions in arterial oxygen and increases in arterial CO_2 and acidosis. The effects of breath holding, hyperventilation, and a combination of the two on endocrine response and acid-base balance have been described (Djarova et al., 1986). Djarova et al. (Djarova et al., 1986) found three maximal duration breath holds significantly ($p < 0.05$) decreased PO_2 (81.1± 6.6 mmHg to 58.00±5.1 mmHg) and raised PCO_2 (39.04 ± 1.76 mmHg to 45.7 ± 3.70 mmHg) while hyperventilation raised PO_2 (66.48 ± 14.4 mmHg to 89.40 ± 16.2 mmHg) and decreased PCO_2

(36.27± 2.24 mmHg to 19.60 ± 1.6 mmHg). These researchers also describe elevated cortisol (12.77 ± 5.87 ng / mL to 28.0 ±7.59 ng/ mL; $p < 0.05$) and human growth hormone (2.13 ± 2.10 ng / mL to 10.35 ± 8.28 ng / dL; $p < 0.05$) in the breath holding condition. While these data are relevant to potentially enhanced recovery and sport performance, few studies have directly investigated the effects of breath holding or breath patterns on similar endocrine parameters. However, these data clearly demonstrate the effects of breathing or apnea on arterial gas concentrations.

Bernardi et al. (Bernardi et al., 2001) investigated the effects of slow-breathing on the chemoreflex response to both hypercapnia and hypoxia in 15 healthy participants (seven men, eight women). Participants underwent either spontaneous or fixed frequency breathing (6 or 15 breaths per minute) in hypoxic and hypercapnic conditions. Their results demonstrated a blunted chemoreflex to both hypoxia (-0.14 ± 0.03 l/min per % SaO_2 vs. -0.46 ± 0.09 l/min per % SaO_2; $p < 0.01$) and hypercapnia (0.32 ± 0.07 l/min per % SaO_2 vs. 1.38 ± 0.29 l/min per % SaO_2; $p <0.001$) during breathing at six breaths per minute compared to spontaneous breathing. These researchers also describe preserved heart rate variability as measured by electrocardiogram in their six breaths per minute condition compared to spontaneous breathing (713 ± 35 ms versus 680 ± 18ms; $p<0.001$). These researchers indicate that altered baroreflex activity may have been the primary mechanism for their findings, but that altered vagal activity may have also played a role in altered chemo-sensitivity to both hypoxia and hypercapnia. A primary limitation of this study is these data represent only acute modulations in chemo-sensitivity and do not reflect chronic adaptations that may arise following breath training.

Chronic adaptations to hypercapnia and hypoxia are reported consistently in breath-hold divers (BHDs). In their investigation on the chronic respiratory adaptations in BHDs,

Walterspacher et al. (Walterspacher et al., 2011) performed lung function assessments in 12 elite BHDs. Their results demonstrated increased mean pulmonary volumes (FEV_1: 4.5 ± 0.02 L vs. 4.7 0.03 L; total lung capacity: 8.1 ± 0.8 L vs. 8.5 0.7 L; VC: 6.2 ± 0.6 L vs. 6.5 ± 0.5 L) attenuated respiratory drive in response to 6 and 9% CO_2 concentrations in ambient air. However, these authors present only graphical representation of the ventilatory drive measures. These researchers conclude that CO_2 tolerance is likely a training adaptation as opposed to a genetically inherited trait, given comparison of their results to their previous work. These results are representative of the literature on BHDs, as similar chronic adaptations in breath hold divers have been reported consistently (Andersson, Linér, Rünow, & Schagatay, 2002; Delapille et al., 2001; Ferretti, 2001; Smith et al., 2014; Zelenkova & Chomahidze, 2016).

Another area in which breath training may significantly affect sporting performance is through adaptations of respiratory musculature, namely, through attenuation of the respiratory metaboreflex. This phenomenon is best illustrated in the work presented by Witt et al. (Witt, Guenette, Rupert, McKenzie, & Sheel, 2007), who investigated the effects of a six-week respiratory musculature training program on the sympathetically mediated increases in heart rate (HR) and mean arterial pressure (MAP) during fatiguing respiratory work in sixteen healthy men. Following training, participants in the training group demonstrated significantly ($p < 0.05$) increased maximum inspiratory pressure (training: −125 ± 10 to −146 ± 12 cm H_2O; sham: −141 ± 11 to −148 ± 11 cm H_2O) and attenuated rises in MAP and HR during their respiratory work task (HR: 59 ± 3 to 74 ± 2 beats min^{-1}; MAP: 84 ± 1 to 89 ± 2 mm Hg). These researchers hypothesize that improved fiber type composition and enzyme profile likely improved the aerobic capacity of the respiratory musculature, which aided in a decrease in metabolite accumulation and subsequent attenuation of sympathetic mediated metaboreflex. Moreover,

these researchers conclude that their results have direct implications on athletic performance given high work demands of the respiratory musculature during endurance and high-intensity training.

Nociception

Anti-nociception presents another mechanism by which breath training may enhance physical performance and athletic capacities (Birrer & Morgan, 2010; Ryan & Kovacic, 1966; Walker, 1971). Paso et al. (Reyes del Paso et al., 2015) demonstrated anti-nociceptive effects in their group of 38 healthy men and women in a breath hold condition during varying pain intensities elicited with progressive algometric fingernail loading. Their data demonstrated smaller increases in pain intensity (slow inhale: $p<0.001$, $\eta^2= 0.708$ vs. breath hold: $p= 0.018$, $\eta^2= 0.141$), unpleasantness (slow inhale: $p <0.001$, $\eta^2= 0.141$ vs. breath hold: $p = 0.006$, $\eta^2= 0.708$), and greater heart rate deceleration ($p = 0.047$) in the breath holding condition. These researchers suggest the anti-nociceptive effects may have been elicited by increased activity of baroreceptors in the aortic arch and lungs implicated by a bi-phasic systolic blood pressure response during the breath hold condition ($p < 0.001$) for all pain intensities during which blood pressure initially rose in the first one to three seconds and decreased in seconds seven and eight (pain intensity 1: $p < 0.005$; pain intensity 2-3: $p < 0.007$). These researchers suggest clinical relevance for these results and do not mention implications for athletic performance, nor do they indicate the potential for chronic adaption subsequent to breathing interventions.

Further support for the role of breathing on anti-nociception and cardiac-modulation is provided by Chalaye et al. (Chalaye, Goffaux, Lafrenaye, & Marchand, 2009) in their investigation of varying breathing conditions on thermal pain threshold and tolerance in 20 healthy adults (11 men, nine women). Their results demonstrated significantly ($p<0.005$) higher

pain threshold (1.0 ± 0.3 DS vs. -0.2 ± 0.3 DS; $p = 0.002$) and pain tolerance (0.5 ± 0.2 DS vs. 0.0 ± 0.1 DS; $p = 0.003$) in their slow breathing intervention during which participants breathed at six breaths pre minute while measures of cardiac rhythmicity revealed significantly ($p<0.005$) greater vagal tone as measured by peak-to-valley amplitude (0.296 ± 0.021 vs. 0.079 ± 0.008; $p < 0.001$) and low frequency power heart rate variability (9,194 ±1433 ms^2 vs. 751 ± 229 ms^2; $p < 0.001$). Peak-to-valley units of amplitude were not provided.

Busch et al. (Busch et al., 2012) also reported altered thermal pain perception following six-weeks of attentive or relaxed deep slow breathing in 15 young (13 female, three male) undergraduate students. In the attentive intervention (aDSB), participants were asked to breathe according to biofeedback and external pacing, while in the relaxed intervention (rDSB) attention was directed on each breath and was intended to induce a meditative state. In both interventions, respiratory frequency was held at seven breaths per minute. Their results demonstrated that stimulus detection ($p = < 0.001$; Cohen's d = .88) and pain threshold ($p = < 0.001$; Cohen's d = 1.01) were significantly increased only in their rDSB (aDSB: 37.86° C ± 2.87, 39.97 ± 3.35° C, 40.19 ± 3.22° C vs. rDSB: 39.76 ± 4.0° C, 40.23 ± 3.39° C, 41.36 ± 3.72° C). Similarly, skin conductance (a measure of sympathetic activity) was decreased significantly ($p = 0.002$; Cohen's d = 1.35) by 18% in their rDSB group only. While these data are not presented within the context of athletic performance *per se*, they certainly illustrate the potential for chronic adaptations for nociception following breath training, and potentially enhanced athletic performance. Further research is warranted to investigate these findings in a sport or training specific environments.

Breath-training for athletic performance.

As described by Joulia et al. (Lemaître et al., 2010), apnea and breath training represents a fertile area of research given the potential of these modalities to induce sporting relevant physiological adaptions. While the potential of these modalities have yet to be fully understood, many studies demonstrate the efficacy of breath training in improving athletic performance. Studies utilizing breath frequency, apnea, and device guided breathing are presented.

Breath frequency training, deep slow breathing, and apnea. Lavin et al. (Lavin et al., 2015) explored the effects of controlled-frequency breathing (CFB) on swimming performance and running economy in 18 recreational swimmers. The CFB group demonstrated greater decreases in 150 m swim time (CFB: -13 ± 9 s vs. control: 8 ± 19 s) and increases in running economy (CFB: −15mL/kg/km vs. Control: −8 mL/kg/km). Kapus et al. (Kapus, Ušaj, & Lomax, 2013) explored the effects of reduced breathing frequency on vital capacity and the ventilatory response to hypercapnia. Their training protocol consisted of a progressive six-week high-intensity interval program utilizing a fixed breathing frequency of 10 breathes per minute during training sessions. Their results demonstrated reduced ventilatory sensitivity to hypercapnia (experimental group: 31.46 ± 21.56 L / min/ kPa to 18.16 ± 13.23 L / min/ kPa vs. control: 38.34 ±28.22 L / min/ kPa to 29.00 ± 10.10 L / min/ kPa; $p = 0.03$), and increased vital capacity (CFB: 5.53 ± 1.16 L to 5.88 ± 0.96 L vs. control: 5.95 ± .4 L to 6.0 ± 0.32; $p = .02$) in their controlled frequency breathing group.

Chronic adaptations to repeated exposures to hypoxia and hypercapnia are well documented in elite breath hold divers. Roecker et al. (Roecker et al., 2014) investigated the ventilatory responses to a graded exercise test in 24 male participants (8 breath hold divers, 8 scuba divers, 8 non-divers). The breath hold divers illustrated distinct ventilatory reactions to

maximal exercise in tandem with significantly ($p < 0.01$) lower lactate threshold, higher blood lactate concentrations at respiratory compensation point (RCP), greater power output at RCP, and lower end tidal CO_2 and estimated arterial CO_2 at lactate threshold, RCP, and VO_{2peak}. The region between lactate threshold and RCP took 43% ± 10% of VO_{2peak} in the BHDs while the same region was 23% ± 7% for the scuba divers and 24% ± 11% for their control group.

While the primary indication of this study is decreased chemo-sensitivity to hypercapnia in the breath hold divers as noted by the delayed onset of RCP, their findings indicate a myriad of chronic adaptations to repeated hypoxia and hypercapnia. These researchers explain increased lactate load and delayed RCP would indicate enhanced anaerobic energy production in tandem with increased buffering capacities, while the earlier onset of lactate threshold likely illustrates the relative decrease in aerobic function in their breath hold divers. While the results presented in this study are clearly relevant to the chronic adaptations of breath training and sporting contexts, it must be noted that this is a cross-sectional examination and its relevancy across populations may be limited.

Fabrice et al. (Joulia et al., 2003) investigated the effects of a 12-week apnea training program on measures of blood acidosis and oxidative stress following dynamic handgrip resistance exercise in eight well trained triathletes. The training protocol consisted of three sessions per week of one-hour cycle efforts at predetermined 30% VO_2max interspersed with 20 seconds of breath holding followed by 40 seconds of spontaneous breathing. Following their program, the researchers noted an attenuated maximal rise in blood lactate (pre: +2.8± 0.34 mmol/L vs. post: 1.99 ± 0.22 mmol/L), reduced oxidative stress as measured by decreased thiobarbituric acid reactive substance (pre: + 35± 10 mg/mL vs. post: +12± 4 mg/mL) and erythrocyte reduced glutathione (pre: -2.82 ± 4 mg/100 mL vs. post: -1.16± 0.3 mg/100 mL)

responses, increased partial pressure of alveolar O_2 (pre: 72 ± 3 mmHg vs. post: 79 ± 3 mmHg), and decreased partial pressure of alveolar CO_2 (pre: 46 ± 1 mmHg vs. post: 42 ± 1 mmHg). These researchers purport their intervention attenuated oxidative stress, and sensitivity to hypercapnia and hypoxemia during the handgrip exercise in tandem with enhanced buffering capacity as noted by lower rises in lactate concentrations and decrease alveolar pressures of CO_2 and attenuated falls in alveolar pressure of O_2 (Joulia et al., 2003).

Woorons et al. (Woorons et al., 2008) demonstrated the potential of a four-week reduced frequency breathing training program to delay blood acidosis during exercise in 15 male runners. While their results did not demonstrate any differences in exercise performance between control and hypoventilation groups, higher venous pH ($p < 0.05$) and higher HCO_3^- ($p < 0.05$) at 90% heart rate max were found in the hypoventilation group. However, it is difficult to comment on the physiological relevance of these increases as they did not provide the data and only indicated the values in the hypoventilation group were higher compared to the control. As no differences were observed between groups with regard to exercise performance, it is possible the impact of these effects were marginal. Their occurrence warrants further investigation

Device guided breath training. Hepburn et al. (Hepburn et al., 2005) utilized a re-breather device which provided resistance to the respiratory musculature while simultaneously inducing a hypercapnic breathing environment. Their data showed enhanced work capacity (+ 0.031 ± 0.022 J/heartbeat), cardiac vagal tone (HF: +13.2 ± 5.7 v; LF: -10.2 ± 5.5 v), and heart rate recovery (3.3 ± 1.5 bpm) following a six-week training period. Further support for the use of device guided breath training is provided by Porcari et al. (Porcari et al., 2016), who investigated the effects of the commercially popular elevation training mask on aerobic capacity, lung function, and hematological variables following a progressive six week high intensity interval

program. While their data did not show improvements in vital capacity, forced expiratory volume, hemoglobin or hematocrit concentrations, they did demonstrate increased ventilatory threshold (VT) (mask: +14.0 % vs. control: +2.1 %), power at VT (mask: +19.0 % vs. control: 9.2 %), respiratory compensation point (mask: +10.2 % vs. control: 1.0 %), and power at respiratory compensation point (mask: +16.4% vs. control: 4.0 %).

While device assisted and controlled frequency breath training differ in modality, the research indicates the similarities between the adaptations following their use may be due in part to the hypercapnic environments produced in each context. Porcari et al. (Porcari et al., 2016) hypothesize the mask acted as respiratory resistance and effectively increased the strength and subsequent work capacity of the respiratory musculature. They also note the training mask induced an increased partial pressure of CO_2 (32.9 ± 6.0 mmHg CO_2 vs. 55.6 ± 12.4 mmHg CO_2) and that hypercapnic breathing conditions may have caused additional respiratory adaptation and subsequent fatigue resistance, though no descriptive statistics were provided. Further support for device guided respiratory training is provided by HajGhanbari et al. (HajGhanbari et al., 2013) in their comprehensive review of respiratory muscle training.

Given the balance of the research presented. It is clear that breath training in its various forms has the potential to impact athletic performance. Differential adaptations to varying modalities of breath training may induce adaptations more relevant to sports that require greater reliance on aerobic energy systems than anaerobic. Given the role of respiratory musculature during aerobic sporting events, adaptations that improve the mechanical and metabolic efficiency of inspiratory and expiratory musculature may improve aerobic performance while their relevancy in anaerobic contexts may be limited (HajGhanbari et al., 2013). Conversely, apnea training results in adaptations specific to anaerobic metabolism, and consequently, may be best

suited for sporting events during which lactic anaerobic energy production is dominant (Roecker et al., 2014; Woorons et al., 2008). Adaptations to enhance vagal tone are largely relevant to the broad spectrum of sport, given their function in maintaining autonomic balance and potentially improving recovery and perception (Anderson, 1998; Anderson et al., 2009; Delapille et al., 2001; Hepburn et al., 2005; Jerath et al., 2006; Kiviniemi et al., 2014; Machhada et al., 2017; Monnazzi et al., 2002; Pal et al., 2004b; Vinay et al., 2016; Walterspacher et al., 2011; Wang et al., 2016; Zelenkova & Chomahidze, 2016). Consequently, sport specific adaptations should be addressed in developing a methodology to address the efficacy of breath training on athletic performance.

Consideration for study design and potential limitations

Prior to designing a study to address the efficacy of deep slow breathing in augmenting anaerobic exercise performance, dependent variables, exclusion criteria, and inclusion criteria must be established to select sensitive and accurate measures of recovery and to control for population homogeneity with regard to training, sex, training history, injury, and supplement use.

Dependent Variables. A multitude of measures presently exist to objectively and subjectively quantify fatigue and recovery between and within training sessions. Tests of anaerobic capacity (Wingate; WAnT) serve the dual purpose as a test of performance (peak power, mean power, total work) and a method to induce metabolic acidosis. Heart rate recovery (HRR) and respiratory exchange ratio (RER) have been effective objective methods of measuring recovery and buffering capacity, while sessions ratings of perceived exertion (sRPE) has been consistently used as subjective measures of perception and recovery during and following exercise.

Heart Rate Recovery (HRR). HRR is a measure of autonomic recovery (parasympathetic reactivation) and is defined as the rate at which heart rate decreases immediately following exercise and has been projected as a strong indicator of mortality risk and athletic readiness (Borresen & Lambert, 2008; Halson, 2014; Lamberts, Swart, Capostagno, Noakes, & Lambert, 2009; Mayo, Iglesias-Soler, Fariñas-Rodríguez, Fernández-del-Olmo, & Kingsley, 2016; Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000; Seiler et al., 2007). Lamberts et al. (Lamberts et al., 2009) evaluated the efficacy of HRR in predicting athletic performance in a group of 14 trained cyclists that participated in a 4-week high intensity interval training program. These researchers divided the 14 cyclists into two groups based on whether HRR increased or decreased over the 4-week HIIT protocol ($HRR_{increase}$ or $HRR_{decrease}$). The $HRR_{increase}$ group improved PP more than the $HRR_{decrease}$ group during a 40 km time trial ride (p= 0.010) and demonstrated a tendency for faster 40-km time trial completions, while both groups displayed improvements in relative PP (p= 0.001). These researchers conclude that HRR is an effective measure by which to monitor and prescribe training loads, and predict performance in athletic populations. In their comprehensive review, Daanen et al. (Daanen, Lamberts, Kallen, Jin, & Van Meeteren, 2012) confirm the potential efficacy of measurements of HRR in determining the recovery status in athletes.

Buffering: CO_2 and Lactate. Lactate is a product of glycolytic ATP production and historically has been viewed as a potential mechanism for the occurrence of fatigue during high intensity exercise. Contemporary models of fatigue reveal decreased pH subsequent to ion accumulation is more likely the culprit linking lactic acidosis to the development of fatigue during exercise (Brooks, 2001; Goodwin, Harris, Hernández, & Gladden, 2007; Sahlin, 2014). Lactate kinetics during and following exercise have been implicated in improvements in exercise

performance and recovery as illustrated by measurements of anaerobic threshold, onset of blood lactate accumulation (OBLA), and their respiratory equivalents (ventilatory threshold and respiratory compensation point) (Sahlin, 2014; Tanaka et al., 1983). Messonnier et al. (Messonnier, Freund, Denis, Féasson, & Lacour, 2006) investigated the effects lactate of clearance capacity on exhaustive exercise performance in eight active participants following endurance training. Their results demonstrated improved performance during a 5-minute cycling test at 90% PP output by 8% for PP output (p= 0.0687) and improved lactate clearance in the following 90-minute recovery session (p< 0.05).

Intrinsic buffer systems regulate acid-base balance to maintain homeokinesis, which is challenged by rising H^+ concentrations during high intensity exercise (Böning et al., 2007; Sahlin, 2014; Yunoki, Horiuchi, & Yano, 2000). The bicarbonate buffering system (equation 1) offers proton acceptors in the form of bicarbonate (HCO_3^-):

Equation 1. $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$ (Yunoki et al., 2000)

HCO_3^- binds hydrogen protons and produces excess CO_2, which can be measured by gas exchange analysis.

Yunoki et al. (Yunoki et al., 2000) found significant correlations between $CO_{2excess}$ (VO_2 -VCO_2), and peak blood lactate concentrations (La_{peak}) in six male sprinters (r = 0.920, p< 0.01) and seven long distance runners (r = 0.588 , p < 0.05). Similar results are provided by Yano et al. (Yano, Yunoki, Matsuura, & Arimitsu, 2009) who described a significant relationship (r = 0.845) between $CO_{2excess}$ and blood lactate concentrations in their group of eight male participants during high intensity cycling intervals.

While bicarbonate buffering (β_{bi}) represents the primary mechanism by which blood pH is maintained during exercise, hemoglobin, phosphates, and blood proteins offer protection

against rising H^+ concentrations during exercise. Boning et al. (Böning et al., 2007) demonstrated the capacity of non-bicarbonate buffering (β_{nbi}) estimated from measures of La, HCO_3^-, pH, and PCO_2 in trained and untrained males derived from the relationship between their measured variables:

Equation 2: $\beta_{nbi} = -\Delta [La] \times \Delta pH^{-1} - \Delta [HCO_3^-] \times \Delta pH^{-1}$

With regard to total buffer capacity, their data demonstrated larger $\beta_{bi} + \beta_{nbi}$ for the untrained state (78± 2 mmol x L^{-1} vs. 68 ± 2 mmol x L^{-1}; $p < 0.02$). These data demonstrate while buffer capacity may relate to enhanced physical performance, other factors such as increased plasma volume, greater hemoglobin, and an increase in La efflux efficiency may present larger determinants of pH regulation in the trained state. However, their methods do allow for a distinct comparison of extracellular β_{bi} versus β_{nbi} buffering capacities.

Respiratory exchange ratio (RER; VCO_2 / VO_2) demonstrates skeletal muscle's oxidative capacity and differentiates utilization of substrates utilized for energy production. Given the relationship between HCO_3^- buffering and CO_2 production, respiratory exchange ratio presents another measure that effectively quantifies the amount of HCO_3^- buffering occurring as values increase beyond one and excess CO_2 is expired. (Ramos-Jiménez et al., 2008).

Wingate Anaerobic Test (WAnT). The rise in H^+ concentrations during anaerobic exercise presents the ability of anaerobic exercise to act as an acid-base titration model for the human body *in vivo*. The Wingate anaerobic test (WAnT) has been utilized as a standard for testing anaerobic power production and consistently raises blood La (Gratas-Delamarche, Le Cam, Delamarche, Monnier, & Koubi, 1994; Vandewalle, Pérès, & Monod, 1987). The WAnT consists of a maximal effort sprint on cycle ergometer against 0.075 kg x kg $bodyweight^{-1}$ typically ranging from ten to 60 seconds in duration. Given the time course of bioenergetics

supply of ATP from creatine phosphate, shorter WAnTs target the phosphagen system while longer (>20 sec) WAnT involve greater contributions from glycolytic and aerobic energy systems and subsequently greater blood lactate accumulations (Attia et al., 2014; Baker et al., 2010; Bogdanis et al., 1998). However, instrument type, duration, and type of recovery interval have been shown to affect measures of WAnT performance.

Mechanically and electronically braked cycle ergometers have been utilized to perform WAnTs. The Monark Ergometer 894E cycle ergometer is a mechanically braked cycle ergometer designed specifically for the high forces produced during WAnTs (Astorino & Cottrell, 2012; Hachana et al., 2012; Harbili, 2015). The Velotron Racermate (Racermate Inc., Seattle, WA) is an electronically braked cycle ergometer that has been shown to produce test-retest reliability during 30-second WAnTs for PP ($r = 0.70$, $p < 0.05$), MP ($r = 0.90$, $p < 0.01$), and minimum power ($r = 0.79$, $p < 0.05$). PP was significantly ($p < 0.05$) greater on the Racermate Velotron (9.95±1.39 W / kg) compared to the Monark Ergometer 894E utilizing automated weight (9.13±1.26 W / kg), while significantly greater MP was displayed on the Monark (6.95 ± 0.89 W / kg) compared to the Racermate Velotron (6.11 ± 0.52 W / kg) (Astorino & Cottrell, 2012). Further validation for the use of the Velotron Racermate and electronically braked cycle ergometers is provided elsewhere, while some articles suggest specific chain ring to maintain validity during WAnTs (Astorino & Cottrell, 2012; Clark, Wagner, & Heath, 2017; Micklewright, Alkhatib, & Beneke, 2006; Vandewalle et al., 1987). The potential variation between ergometers with respect to measurement of PP and MP must be taken into account when comparing results of these measures between studies.

Aside from duration and mode of WAnT, many researchers have utilized the WAnT in a repeated sprint format with varying recovery interval durations and active versus passive

recovery. Repeated 30-second WAnTs with one and two-minute recoveries resulted in significantly lower PP (one-minute, $p<0.05$; two- minute $p<0.05$) while one, two, and three-minute recoveries resulted in significantly lowered MP across conditions (one minute, $p<0.05$; two minutes, $p<0.05$; three minutes, $p<0.05$). Billaut et al. (Francois Billaut et al., 2003) found recovery of PP following brief cycle sprints was significantly lower after only 15 and 30 seconds of recovery ($p < 0.001$ and $p < 0.05$, respectively) compared to 60, 120, and 240 second recoveries. However, these sprints only lasted 8 seconds in duration and their relevance to lactate recovery are limited. Bogdanis et al. (Bogdanis et al., 1995) investigated the effects of 1.5, three, and six minutes recoveries between two consecutive 30-second WAnTs. Their results demonstrated rapid recovery of PP (88.7%) and pedal speed (93.5%) during the first three minutes of recovery, a 3% increase of the MP in the first six seconds (MP_6: 30) between the three and six minutes recoveries, while MP throughout the second test (MP_{30}; W) followed a slower recovery than PP and MP_6 for each recovery duration. Nearly full recovery of creatine phosphate (resting 39.0 ± 3.2 mmol / kg; after 2^{nd} interval 19.8 ± 3.5 mmol / kg; after 1^{st} recovery 39.9 mmol / kg) was demonstrated in a four minute passive recovery between three consecutive 30-second maximal isokinetic bike sprints, while total work (kJ), MP, and glycogenolysis decreased from 78.5% and 64.5%, 15% and 20%, and 32%, respectively, across the three intervals (Spriet, Lindinger, McKelvie, Heigenhauser, & Jones, 1989). Active recovery allows significantly greater recovery of PP (90 ±3% active versus 87 ± 3% passive) and MP (603 ± 17 W active versus 589 ± 15 W passive) during four-minute recoveries between two consecutive WAnTs. This was due to increased blood flow and subsequent removal of H^+ and metabolites and delivery of O_2 down their respective concentration gradients.

The WAnT presents the dual capacities as a test of anaerobic exercise performance and as a method by which to induce metabolic acidosis. The use of electronically and mechanically braked cycle ergometers has been validated for WAnT use while test duration and recovery interval duration will affect the energy system targeted (phoshpagen versus glycolytic) and the degree to which this energy system is recovered between efforts (shorter for phosphagen compared to glycolytic). Active recovery intervals increase recovery of PP and MP between WAnT intervals over passive recovery.

Sessions Rating of Perceived Exertion (sRPE). The (sRPE) is a tool developed by Foster et al. (Foster et al., 2001) to quantify perceived training stress during non-steady state exercise. These researchers utilized a two-part design in which 12 active participants first completed maximal, steady state, and interval exercise on cycle ergometer during which HR, blood lactate, and Borg RPE were tracked. The second part involved basketball practice during which only HR and practice RPE were recorded. While session RPE derived training impulse (TRIMP) was significantly larger than HR derived TRIMP scores across all 10 exercise durations and intensities (30-minute steady state: 110 ± 24 bpm vs. 130 ± 57 RPE; 60-minute steady state: 216 ± 39 bpm vs. 270 ± 63 RPE; 90-minute steady state: 350 ± 44 bpm vs. 432 ± 57 RPE; 30s/30s intervals: 107 ± 14 bpm vs. 131 ± 45 RPE; 60s / 60 s intervals: 117± 18 bpm vs. 148 ± 54 RPE; 120s / 120s: 114 ± 17 bpm vs. 146 ± 47 RPE; +10% interval: 114 ± 16 bpm vs. 136 ± 60 RPE; +25% interval; 117 ± 18 bpm vs. 148 ± 54 RPE; +50% interval: 114 ± 11 bpm vs. 161 ± 46 RPE; basketball: 652 ± 59 bpm vs. 744 ± 84 RPE; $p < 0.05$) regression analyses revealed the session RPE method was consistent in determining training load. The researchers conclude sRPE is an effective, consistent, and simple method by which to quantify subjective training stress.

Nutrient Intake.

Given the potential for macronutrient and total kilocalorie intake to affect indices of mean power, controlling for these variables is crucial in investigations that aim to identify performance increases during exercise bouts which derive a majority of energy production from glycolytic ATP production. Langfort et al. (50) compared the effects of moderate carbohydrate (130 kJ/kg of body mass daily, 50% carbohydrate, 30% fat, 20% protein) and isocaloric low carbohydrate (up to 5% carbohydrate, 50% fat, 45% protein) diets on indices of MP during 30-second WAnTs. Their results demonstrated significantly diminished MP (533 ± 7 W vs. 581 ± 7 W; $p < 0.05$) and lactate concentrations (9.5 ± 0.4 mmol x L^{-1} vs. 10.6 ± 0.5 mmol x L^{-1}; $p < 0.05$) in the low carbohydrate group. Similar data has been presented by Simonsen et al. (Simonsen et al., 1991) in their investigation on the effects of moderate (5 g x kg^{-1} x day^{-1}) versus high (10 g x kg^{-1} x day^{-1}) carbohydrate diets on mean power during rowing efforts. Their results illustrate 65% greater muscle glycogen content ($p <0.05$) and significantly greater mean power output increases (10.6 % versus 1.6%; $p < 0.05$) in the high carbohydrate group. These results clearly demonstrate the necessity of controlling carbohydrate intake during testing procedures that derive a significant source of ATP production form glycolysis

Inclusion and Exclusion Criteria

Previously resistance trained individuals have shown attenuated muscle damage and enhanced recovery following successive bouts of resistance exercise [69]. Newton et al. [69] reported attenuated muscle function reduction following 30 eccentric elbow extensions in resistance-trained individuals compared to the untrained group as noted by decreased rises in CK (Trained: 2-fold increase, Untrained: 20-fold increase; $p =0.007$) and greater decrements in

isometric torque (Trained: -25%, Untrained: -47%). These results are mirrored in the data provided by Zourdos et al. [70] which showed attenuated decrements in isometric torque at 24 hrs post exercise (Session 1: -8%, Session 2: no change from baseline) though *p*-values were not provided. This protective mechanism has been previously referred to as the repeated bouts effect (RBE) and persists for up to 6 months following a bout of resistance training [71].

Prior injury reduces skeletal muscle function. Holder-Powell et al. [72] demonstrate the long term effects of lower extremity injury on decrements on isometric peak torque in injured compared to uninjured limbs (p=0.001). Similar results are presented in a later study, which demonstrated decrements in concentric isometric (Injured: Uninjured: 90.47 ± 13.7%; p=0.003), concentric isokinetic strength ($30°s^{-1}$: 84.07 ± 14.2%; p= .0001; $120°s^{-1}$: 89.37 ± 17.8%; p= .0015), and eccentric isokinetic strength ($30°s^{-1}$: 83.07 ± 15.3%; *p=0* .0001) [73].

Summary

A review of the pertinent literature around breath training identifies autonomic, chemo-sensitive, anti-nociceptive, and endocrine mechanisms that potentially explain how breath training may augment athletic performance and recovery within or between training sessions. Adaptations to the respiratory musculature may be more relevant to aerobic sporting efforts while enhanced buffering and anaerobic metabolism may have greater positive effects on sporting effects in which ATP production from anaerobic energy systems is dominant. Other adaptations, including enhanced vagal tone and augmented endocrine profile, may have relevance across the sporting spectrum given their role in recovery and general health contexts. However, a large majority of this literature has not been conducted within the realm of sport performance, so the ability to generalize the results across populations may be limited. Further

research is warranted to elucidate the potential of breath training on indices of performance and recovery within athletic populations. The aim of the proposed article is to address the null hypothesis that breath-training will have no effect on measures of performance and recovery during anaerobic interval exercise.

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Appendix A: Journal of Sport Science and Medicine Guidelines

Journal of Sport Science and Medicine

Journal guidelines to authors:

https://www.jssm.org/newauthors.php

Appendix B: Raw Data

Subject	Group	Age (Years)	Height (cm)	Weight (kg)
1	1	24	171.45	75
5	1	23	187	92.7
6	1	27	182.88	90.11
9	1	19	187.96	80.4
10	1	19	185.42	71.36
13	1	24	181.61	73.6
17	1	24	186.69	92.3
18	1	24	184	85
19	1	26	175	72.9
20	1	35	175.26	71.36
2	2	28	175.26	72
3	2	28	170.18	70.5
4	2	28	186	96.4
7	2	26	173	67.6
8	2	21	185.42	90.68
11	2	27	190.5	84
12	2	30	181.6	80.45
14	2	26	172	66.4
15	2	22	182.88	79
16	2	25	172.72	85.45

Subject	Group	MEAN PWR 1_Pre (W)	MEAN PWR 2 PRE (W)	MEAN PWR 3 PRE (W)	MEAN PWR 1 Post (W)	MEAN PWR 2 Post (W)	MEAN PWR 3 Post (W)
1	1	550.56	484.23	442.35	532.89	489.62	457.21
5	1	621.04	506.97	433.29	602.26	539.35	468.71
6	1	635.68	580.6	581.45	646.88	637.69	591.11
9	1	620.78	580.8	592.97	646.65	618.08	592.66
10	1	586.99	541.59	488.77	630.86	549.03	466.57
13	1	607.86	519.55	439.69	593.1	532.43	464.32
17	1	742.45	632.8	588.44	731.94	664.51	588.63
18	1	680.21	627.24	607.75	643.25	598.71	577.53
19	1	581.75	458.5	370.96	582.34	477.5	388.48
20	1	596.08	566.98	531.79	639.41	571.36	517.82
2	2	570.12	516.81	470.95	570.12	516.81	470.95
3	2	511.09	481.43	430.47	511.09	511.09	430.47
4	2	681.79	570.06	515.6	669.32	596.12	541.89
7	2	505	450.15	371.99	494.72	418.14	-
8	2	651.2	537.67	450.65	680.43	550.91	464.59
11	2	673.81	620.84	579.37	673.81	620.84	579.37
12	2	635.8	493.7	-	580.32	455.12	420.47
14	2	510.8	451.66	411.23	484.47	450.63	401.87
15	2	713.87	509.4	445.54	667.81	509.94	415.8
16	2	666.86	442.5	294.67	688.89	534.66	496.52

Subject	Group	VCO2 1 Pre (L/min)	VCO2 2_Pre (L/min)	VCO2 3 Pre (L/min)	VCO2 _ Post (L/min)	VCO2 2 Post (L/min)	VCO2 3 Post (L/min)
1	1	2.42	2.21	1.89	2.36	2.11	1.87
5	1	2.76	2.22	2.49	2.73	2.41	1.99
6	1	2.76	2.14	2.25	2.62	2.48	2.21
9	1	2.50	2.62	2.31	2.62	2.49	1.96
10	1	2.54	2.54	2.26	3.21	2.56	2.33
13	1	2.69	2.08	2.08	2.56	2.02	1.83
17	1	2.94	2.38	2.76	3.00	2.61	2.56
18	1	3.03	2.29	2.34	2.72	2.44	2.30
19	1	2.40	1.81	1.74	2.69	2.03	1.88
20	1	2.62	2.62	2.20	2.99	2.60	2.17
2	2	2.42	1.82	1.56	2.41	2.08	1.85
3	2	1.88	1.91	1.71	2.27	1.96	1.74
4	2	2.75	2.43	2.13	2.72	2.46	2.38
7	2	2.37	1.60	1.44	2.13	1.64	-
8	2	2.59	2.08	2.08	2.87	2.43	1.91
11	2	2.62	2.49	2.24	2.49	2.44	2.43
12	2	2.02	1.92	-	2.68	1.96	1.76
14	2	2.17	1.87	1.68	2.14	1.67	1.52
15	2	2.70	1.92	2.43	2.38	1.95	2.33
16	2	2.93	2.19	1.66	2.33	2.53	2.20

Subject	Group	VE 1 Pre (L/min)	VE 2 Pre (L/min)	VE 3 Pre (L/min)	VE 1 Post (L/min)	VE 2 Post (L/min)	VE 3 Post (L/min)
1	1	58.84	74.87	67.99	67.99	64.76	64.76
5	1	91.30	98.64	104.33	78.78	79.08	80.34
6	1	80.87	73.37	88.63	67.70	74.97	72.07
9	1	72.63	88.54	90.88	74.20	77.23	82.69
10	1	72.90	92.83	92.79	82.92	98.80	99.81
13	1	81.12	81.00	76.82	70.26	74.34	61.09
17	1	82.70	88.68	97.52	85.91	103.86	101.64
18	1	82.11	80.64	95.74	84.63	91.68	97.37
19	1	59.22	65.12	66.38	71.11	74.41	80.15
20	1	65.18	96.72	79.95	83.64	96.20	86.72
2	2	76.85	73.67	63.14	68.06	70.79	73.60
3	2	47.66	52.39	53.81	49.06	53.43	54.30
4	2	63.06	67.52	75.12	67.76	67.56	86.40
7	2	67.59	56.82	68.94	50.27	52.17	-
8	2	79.59	83.12	89.00	84.40	86.40	88.73
11	2	74.97	85.83	78.72	74.82	85.50	78.28
12	2	63.37	78.26	-	69.29	67.31	65.81
14	2	62.80	67.26	60.49	58.95	61.64	62.55
15	2	91.46	81.56	90.11	72.05	84.61	86.36
16	2	85.84	92.84	82.25	79.20	91.93	90.30

Subject	Group	HRR 1 Pre (bpm)	HRR 2 Pre (bpm)	HRR 3 Pre (bpm)	HRR 1 Post (bpm)	HRR 2 Post (bpm)	HRR 3 Post (bpm)
1	1	41	20	29	29	27	30
5	1	28	30	25	35	34	33
6	1	34	25	26	47	37	37
9	1	55	61	46	50	43	46
10	1	20	17	16	21	17	10
13	1	24	34	40	32	32	33
17	1	20	15	14	21	12	12
18	1	30	28	27	27	32	27
19	1	32	19	16	22	18	14
20	1	37	23	20	40	34	21
2	2	46	23	30	27	-	31
3	2	73	59	50	76	51	54
4	2	31	29	35	23	23	32
7	2	32	40	48	39	24	-
8	2	23	18	16	21	14	17
11	2	30	36	37	27	31	34
12	2	70	23	-	25	4	12
14	2	38	31	28	35	29	27
15	2	-	16	26	23	19	17
16	2	16	26	49	-	-	-

Subject	Group	VC PRE (L)	VC Post (L)	FEV1 Pre (L)	FEV1 Post (L)	MVV Pre (L/min)	MVV Post (L/min)
1	1	5	4.9	4.3	4	191	152
5	1	6.1	6.2	4.8	4.3	195	187
6	1	6	5.6	3.8	4.4	202	197
9	1	4.2	5	3.8	3.7	152	171
10	1	5.3	5.1	4.3	4.5	157	166
13	1	6.2	6.1	4.8	4.9	160	201
17	1	5.9	5.9	4.5	4.7	205	214
18	1	5.3	6.8	5.1	4.7	195	216
19	1	4.2	4.8	4.1	4.2	187	155
20	1	4.8	4.5	4.4	4.2	183	179
2	2	5.2	5.1	4	3.9	155	164
3	2	4	4.2	3.5	3.4	154	122
4	2	5.2	5.2	4.1	4.3	172	198
7	2	5.1	5.1	4.3	4.5	210	205
8	2	6	6.1	4.9	5.5	190	204
11	2	7.4	7.3	4.8	4.3	160	132
12	2	5.9	6.2	4.5	4.6	191	192
14	2	4.7	4.8	3.9	4	90	114
15	2	5.3	5.3	4.1	4.1	185	189
16	2	5.7	6	3.7	3.9	173	163

Appendix C: Statistical Analysis

Mean Power:

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
Time	Pillai's Trace	.137	2.549[b]	1.000	16.000	.130	.137	2.549	.323
	Wilks' Lambda	.863	2.549[b]	1.000	16.000	.130	.137	2.549	.323
	Hotelling's Trace	.159	2.549[b]	1.000	16.000	.130	.137	2.549	.323
	Roy's Largest Root	.159	2.549[b]	1.000	16.000	.130	.137	2.549	.323
Time * Group	Pillai's Trace	.011	.176[b]	1.000	16.000	.680	.011	.176	.068
	Wilks' Lambda	.989	.176[b]	1.000	16.000	.680	.011	.176	.068
	Hotelling's Trace	.011	.176[b]	1.000	16.000	.680	.011	.176	.068
	Roy's Largest Root	.011	.176[b]	1.000	16.000	.680	.011	.176	.068
Test	Pillai's Trace	.857	44.891[b]	2.000	15.000	.000	.857	89.783	1.000
	Wilks' Lambda	.143	44.891[b]	2.000	15.000	.000	.857	89.783	1.000
	Hotelling's Trace	5.986	44.891[b]	2.000	15.000	.000	.857	89.783	1.000
	Roy's Largest Root	5.986	44.891[b]	2.000	15.000	.000	.857	89.783	1.000
Test * Group	Pillai's Trace	.108	.904[b]	2.000	15.000	.426	.108	1.808	.177
	Wilks' Lambda	.892	.904[b]	2.000	15.000	.426	.108	1.808	.177
	Hotelling's Trace	.121	.904[b]	2.000	15.000	.426	.108	1.808	.177
	Roy's Largest Root	.121	.904[b]	2.000	15.000	.426	.108	1.808	.177
Time * Test	Pillai's Trace	.382	4.637[b]	2.000	15.000	.027	.382	9.273	.691
	Wilks' Lambda	.618	4.637[b]	2.000	15.000	.027	.382	9.273	.691
	Hotelling's Trace	.618	4.637[b]	2.000	15.000	.027	.382	9.273	.691
	Roy's Largest Root	.618	4.637[b]	2.000	15.000	.027	.382	9.273	.691
Time * Test * Group	Pillai's Trace	.092	.758[b]	2.000	15.000	.486	.092	1.516	.155
	Wilks' Lambda	.908	.758[b]	2.000	15.000	.486	.092	1.516	.155
	Hotelling's Trace	.101	.758[b]	2.000	15.000	.486	.092	1.516	.155
	Roy's Largest Root	.101	.758[b]	2.000	15.000	.486	.092	1.516	.155

a. Design: Intercept + Group
Within Subjects Design: Time + Test + Time * Test

b. Exact statistic

c. Computed using alpha = .05

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	44493.889	1	44493.889	128.985	.000	.890	128.985	1.000
Group	347.222	1	347.222	1.007	.331	.059	1.007	.157
Error	5519.250	16	344.953					

a. Computed using alpha = .05

VCO_2:

Multivariate Tests[a]

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[c]
Time	Pillai's Trace	.028	.425[b]	1.000	15.000	.524	.028	.425	.094
	Wilks' Lambda	.972	.425[b]	1.000	15.000	.524	.028	.425	.094
	Hotelling's Trace	.028	.425[b]	1.000	15.000	.524	.028	.425	.094
	Roy's Largest Root	.028	.425[b]	1.000	15.000	.524	.028	.425	.094
Time * Group	Pillai's Trace	.052	.820[b]	1.000	15.000	.379	.052	.820	.136
	Wilks' Lambda	.948	.820[b]	1.000	15.000	.379	.052	.820	.136
	Hotelling's Trace	.055	.820[b]	1.000	15.000	.379	.052	.820	.136
	Roy's Largest Root	.055	.820[b]	1.000	15.000	.379	.052	.820	.136
Test	Pillai's Trace	.908	69.112[b]	2.000	14.000	.000	.908	138.224	1.000
	Wilks' Lambda	.092	69.112[b]	2.000	14.000	.000	.908	138.224	1.000
	Hotelling's Trace	9.873	69.112[b]	2.000	14.000	.000	.908	138.224	1.000
	Roy's Largest Root	9.873	69.112[b]	2.000	14.000	.000	.908	138.224	1.000
Test * Group	Pillai's Trace	.015	.110[b]	2.000	14.000	.897	.015	.220	.064
	Wilks' Lambda	.985	.110[b]	2.000	14.000	.897	.015	.220	.064
	Hotelling's Trace	.016	.110[b]	2.000	14.000	.897	.015	.220	.064
	Roy's Largest Root	.016	.110[b]	2.000	14.000	.897	.015	.220	.064
Time * Test	Pillai's Trace	.094	.723[b]	2.000	14.000	.503	.094	1.446	.148
	Wilks' Lambda	.906	.723[b]	2.000	14.000	.503	.094	1.446	.148
	Hotelling's Trace	.103	.723[b]	2.000	14.000	.503	.094	1.446	.148
	Roy's Largest Root	.103	.723[b]	2.000	14.000	.503	.094	1.446	.148
Time * Test * Group	Pillai's Trace	.202	1.767[b]	2.000	14.000	.207	.202	3.534	.308
	Wilks' Lambda	.798	1.767[b]	2.000	14.000	.207	.202	3.534	.308
	Hotelling's Trace	.252	1.767[b]	2.000	14.000	.207	.202	3.534	.308
	Roy's Largest Root	.252	1.767[b]	2.000	14.000	.207	.202	3.534	.308

a. Design: Intercept + Group
Within Subjects Design: Time + Test + Time * Test

b. Exact statistic

c. Computed using alpha = .05

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	537.060	1	537.060	1771.487	.000	.992	1771.487	1.000
Group	.930	1	.930	3.067	.100	.170	3.067	.374
Error	4.548	15	.303					

a. Computed using alpha = .05

HRR:

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	44493.889	1	44493.889	128.985	.000	.890	128.985	1.000
Group	347.222	1	347.222	1.007	.331	.059	1.007	.157
Error	5519.250	16	344.953					

a. Computed using alpha = .05

VC:

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	32290634.00	1	32290634.00	1561.235	.000	.990	1561.235	1.000
Group	20351.215	1	20351.215	.984	.336	.058	.984	.154
Error	330924.029	16	20682.752					

a. Computed using alpha = .05

FEV_1:

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	737.881	1	737.881	2014.539	.000	.991	2014.539	1.000
Group	.256	1	.256	.699	.414	.037	.699	.124
Error	6.593	18	.366					

a. Computed using alpha = .05

MVV:

Tests of Between-Subjects Effects

Measure: MEASURE_1

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	1234819.600	1	1234819.600	913.129	.000	.981	913.129	1.000
Group	2280.100	1	2280.100	1.686	.211	.086	1.686	.233
Error	24341.300	18	1352.294					

a. Computed using alpha = .05

Pearson Correlation between MP and VCO_2:

[DataSet1] C:\Users\buddhah\Documents\Mentoring\Commitee member\Andrew Brown\ThesisData_4_2_2019.sav

Correlations

		Avg_Power_Pre_T1	Avg_Power_Pre_T2	Avg_Power_Pre_T3	Avg_Power_Post_T1	Avg_Power_Post_T2	Avg_Power_Post_T3	VCO2_Pre_T1	VCO2_Pre_T2	VCO2_Pre_T3	VCO2_Post_T1	VCO2_Post_T2	VCO2_Post_T3
Avg_Power_Pre_T1	Pearson Correlation	1	.635**	.450	.920**	.676**	.536*	.725**	.452*	.745**	.497*	.651**	.792**
	Sig. (2-tailed)		.003	.053	.000	.001	.018	.000	.045	.000	.030	.002	.000
	N	20	20	19	20	20	19	20	20	19	19	20	19
Avg_Power_Pre_T2	Pearson Correlation	.635**	1	.947**	.671**	.892**	.860**	.540*	.704**	.763**	.576**	.694**	.700**
	Sig. (2-tailed)	.003		.000	.001	.000	.000	.014	.001	.000	.010	.001	.001
	N	20	20	19	20	20	19	20	20	19	19	20	19
Avg_Power_Pre_T3	Pearson Correlation	.450	.947**	1	.457*	.818**	.809**	.340	.642**	.690**	.480*	.541*	.508*
	Sig. (2-tailed)	.053	.000		.049	.000	.000	.154	.003	.001	.044	.017	.031
	N	19	19	19	19	19	18	19	19	19	18	19	18
Avg_Power_Post_T1	Pearson Correlation	.920**	.671**	.457*	1	.791**	.642**	.755**	.628**	.706**	.612**	.832**	.846**
	Sig. (2-tailed)	.000	.001	.049		.000	.003	.000	.003	.001	.005	.000	.000
	N	20	20	19	20	20	19	20	20	19	19	20	19
Avg_Power_Post_T2	Pearson Correlation	.676**	.892**	.818**	.791**	1	.944**	.647**	.766**	.760**	.566*	.861**	.757**
	Sig. (2-tailed)	.001	.000	.000	.000		.000	.002	.000	.000	.012	.000	.000
	N	20	20	19	20	20	19	20	20	19	19	20	19
Avg_Power_Post_T3	Pearson Correlation	.536*	.860**	.809**	.642**	.944**	1	.611**	.727**	.559*	.325	.782**	.656**
	Sig. (2-tailed)	.018	.000	.000	.003	.000		.005	.000	.016	.188	.000	.002
	N	19	19	18	19	19	19	19	19	18	18	19	19
VCO2_Pre_T1	Pearson Correlation	.725**	.540*	.340	.755**	.647**	.611**	1	.481*	.617**	.392	.676**	.741**
	Sig. (2-tailed)	.000	.014	.154	.000	.002	.005		.032	.005	.097	.001	.000
	N	20	20	19	20	20	19	20	20	19	19	20	19
VCO2_Pre_T2	Pearson Correlation	.452*	.704**	.642**	.628**	.766**	.727**	.481*	1	.664**	.577**	.821**	.623**
	Sig. (2-tailed)	.045	.001	.003	.003	.000	.000	.032		.002	.010	.000	.004
	N	20	20	19	20	20	19	20	20	19	19	20	19
VCO2_Pre_T3	Pearson Correlation	.745**	.763**	.690**	.706**	.760**	.559*	.617**	.664**	1	.679**	.658**	.689**
	Sig. (2-tailed)	.000	.000	.001	.001	.000	.016	.005	.002		.002	.002	.002
	N	19	19	19	19	19	18	19	19	19	18	19	18
VCO2_Post_T1	Pearson Correlation	.497*	.576**	.480*	.612**	.566*	.325	.392	.577**	.679**	1	.715**	.500*
	Sig. (2-tailed)	.030	.010	.044	.005	.012	.188	.097	.010	.002		.001	.035
	N	19	19	18	19	19	18	19	19	18	19	19	18
VCO2_Post_T2	Pearson Correlation	.651**	.694**	.541*	.832**	.861**	.782**	.676**	.821**	.658**	.715**	1	.747**
	Sig. (2-tailed)	.002	.001	.017	.000	.000	.000	.001	.000	.002	.001		.000
	N	20	20	19	20	20	19	20	20	19	19	20	19
VCO2_Post_T3	Pearson Correlation	.792**	.700**	.508*	.846**	.757**	.656**	.741**	.623**	.689**	.500*	.747**	1
	Sig. (2-tailed)	.000	.001	.031	.000	.000	.002	.000	.004	.002	.035	.000	
	N	19	19	18	19	19	19	19	19	18	18	19	19

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).