`Research article`

Effects of Swimming with Added Respiratory Dead Space on Cardiorespiratory Fitness and Lipid Metabolism

Stefan Szczepan [1]✉, **Kamil Michalik** [2], **Jacek Borkowski** [2] and **Krystyna Zatoń**[1]
[1] Department of Swimming, and [2] Department of Physiology and Biochemistry, University School of Physical Education in Wroclaw, Poland

Abstract

The aim of this study was to investigate the circulatory, respiratory, and metabolic effects of induced hypercapnia via added respiratory dead space (ARDS) during moderate-intensity swimming in recreational swimmers. A mixed-sex sample of 22 individuals was divided into homogeneous experimental (E) and control (C) groups controlled for maximal oxygen uptake (VO_2max). The intervention involved 50 min of front crawl swimming performed at 60% VO_2max twice weekly for 6 consecutive weeks. ARDS was induced via tube breathing (1000 ml) in group E. An incremental exercise test was administered pre- and post-intervention to assess cardiorespiratory fitness (CRF) by measuring VO_2max, carbon dioxide volume, respiratory minute ventilation, respiratory exchange ratio (RER), and heart rate at 50, 100, 150, 200 W and at maximal workload. Body mass index (BMI), fat mass (FM), and fat-free mass (FFM) were also measured. The mean difference in glycerol concentration (ΔGLY) was assessed after the first and last swimming session. No significant between-group differences were observed at post-intervention. No within-group differences were observed at post-intervention except for RER which increased in group E at maximal workload. A 6-week swimming intervention with ARDS did not enhance CRF. The RER increase in group E is not indicative of a substrate shift towards increased lipid utilization. No change in ΔGLY is evident of a lack of enhanced triglyceride hydrolyzation that was also confirmed by similar pre- and post-intervention BMI, FM, and FMM.

Key words: Cardiorespiratory fitness, lipid metabolism, added respiratory dead space, swimming.

Introduction

Improving cardiorespiratory fitness (CRF) concomitant with metabolic function is an important component of swimming training (Jorgić et al., 2011) and serves as a key predictor of swimming performance and fitness (Lahart and Metsios, 2018). Several effective and time-efficient exercise strategies have been identified that improve CRF and induce positive metabolic changes (Latt et al., 2009). The search for more effective training interventions for both competitive swimmers and recreational swimmers has expanded to include several external aids that can enhance CRF variables including oxygen uptake (VO_2), respiratory minute ventilation (VE), tidal volume (VT), respiratory frequency (RF), heart rate (HR), and associated metabolic measures such as lipid oxidation (Heinicke et al., 2005; Koppers et al., 2006; Millet et al., 2010).

One method that has shown promise is inducing a condition known as hypercapnia or the abnormal elevation of the partial pressure of carbon dioxide in arterial blood (pCO_2) above 45 mmHg (Kato et al., 2005; Schneider et al., 2013). A low-cost strategy that can safely induce hypercapnia is by increasing the volume of external respiratory dead space such as by breathing through a tube (Hebisz et al., 2013; Khayat et al., 2003; Zatoń et al., 2010). According to numerous studies (Cathcart et al., 2005; Toklu et al., 2003), the added respiratory dead space (ARDS) supplemented by tube breathing increases the amount of CO_2 that is inspired in subsequent breaths proportional to the added volume. The retention of CO_2 induces a condition known as acute respiratory acidosis (Jones, 2008; Kraut and Madias, 2014). In response to increased CO_2 levels, chemoreceptors provide respiratory feedback (Kumar and Bin-Jaliach, 2007) increasing VE, VT, and RF (Mercier et al., 1992) to levels observed at higher training intensities (Zatoń and Smołka, 2011). Several studies suggested that regular training with induced hypercapnia can condition muscle cells to utilize lipids at a greater rate than other substrates and enhance the reduction of body fat content (Hollidge-Horvat et al., 1999; McLellan, 1991; Østergaard et al., 2012).

Original research on exercise with ARDS dates back to the 1970s in which participants breathed through a 1200 ml tube during 6 min of ergometer cycling corresponding to 30%, 50%, and 70% of predicted maximal oxygen uptake (VO_2max) (Kelman and Watson, 1973). This protocol resulted in an increase in RF and VE with the latter attributed to increased VT. In the previous study Zatoń et al. (2010) have found that a 5-week endurance cycling intervention with 1000-ml ARDS improved performance (total work) in the incremental exercise test with simultaneous decreases in hematocrit, hemoglobin, and red blood cell concentrations. In another study by the same research group, the respiratory response to cycle ergometry at 100 W with increasing ARDS volume (up to 1600 ml) was investigated (Zatoń and Smołka, 2011). In this study, VE increased proportional to the volume of ARDS and both pCO_2 and blood pH decreased. A study on incorporating 1000 ml ARDS in a 10-week training macrocycle in trained cyclists showed increases in VO_2max, VCO_2max, and VE-max during exercise testing although a decrease was observed in mechanical efficiency (Hebisz et al., 2013). Another investigation on cycling training with 1200 ml ARDS at submaximal intensities found no reduction in RER and VCO_2 but an increase in endurance capacity (Smołka et al., 2014). A recent study involving triathlon athletes included ARDS in an 8-week interval training intervention and found greater increases in VCO_2max, VE, maximal power,

and post-exercise lactate concentrations when compared with a non-ARDS cohort (Michalik et al., 2018).

In the swimming domain, Adam et al. (2015) introduced ARDS in an 8-week training intervention targeting glycolytic power in a sample of competitive swimmers. Two sessions were held per week in which repeated 50-m swims were performed at maximal intensity until volitional exhaustion. This protocol enhanced 100-m freestyle swim speed as well as VO_2max and VE ascertained in an incremental exercise test. Another study examined the effects of a 5-week swimming intervention (two session per week) with ARDS in competitive swimmers and observed reduced HR and a lowered physiological cost of swimming (Zatoń and Ziebura, 2012). Additionally, Zoretić et al. (2014) investigated an 8-week hypercapnic-hypoxic training program in elite swimmers. At the end of the program they observed a 5.35% increase in hemoglobin concentration and a 10.79% increase in VO_2max. Karaula et al. (2016) examined the effects of 8 weeks of hypercapnic–hypoxic training in elite swimmers and reported improved swimming efficacy and increased maximal inspiratory and expiratory muscle strength.

The aforementioned studies (Zatoń and Ziebura, 2012, Adam et al., 2015) involving elite swimmers suggests that swimming with ARDS may enhance swimming performance and CRF and maintain CRF among athletes during the off-season mesocycle when exercise intensity is reduced. However, less is known about the effects of ARDS in recreational swimmers as previous studies recruited elite or trained cohorts. Furthermore, as a reduction in fat tissue is a common goal of recreational swimmers, there is a lack of data on changes in substrate metabolism after several weeks of swimming with ARDS. Hence, investigating the effects of swimming with ARDS on CRF and lipid metabolism in recreational swimmers can provide new insights on the use of this exercise enhancement. Therefore, the aim of this study was to assess the circulatory, respiratory, and metabolic effects of a moderate-intensity swimming intervention with ARDS in recreational swimmers. Moderate intensity exercise was selected as the literature recommends prescribing this intensity level to promote lipid utilization and cardiovascular adaptations (Alkahtani, 2014). It was hypothesized that ARDS would show large improvements in CRF with beneficial metabolic changes towards increased exercise-induced lipid oxidation.

Methods

Subjects
Twenty-two healthy male and female physical education students were recruited. All swam regularly for recreational purposes averaging 2 km twice a week. Subjects were assigned to a control (C) and experimental (E) group based on VO_2max to ascertain similar CRF between the two groups. VO_2max was obtained for each participant in an incremental exercise test (detailed below). The VO_2max scores were ranked from highest to lowest and participants were then alternately assigned to group E (n = 11, age 24.27 ± 2.69 years, body height 1.73 ± 0.09 m, body mass 70.03 ± 13.14 kg, VO_2max 45.55 ± 7.47 mL·kg⁻¹·min⁻¹) and

group C (n = 11; age 24.00 ± 3.35 years, body height 1.68 ± 0.03 m, body mass 72.32 ± 10.13 kg, VO_2max 47.09 ± 8.85 mL·kg⁻¹·min⁻¹). The groups were compared at this stage with the non-parametric Wilcoxon test ($\alpha = 0.05$) and no between-group differences were observed for age (p = 0.72), body height (p = 0.50), body mass (p = 0.65), and VO_2max (p = 0.65). This generated comparable intervention groups with an objective baseline measurement of CRF.

All participants provided their written consent to participate in the study and were informed they could withdraw at any time. They were instructed to maintain their normal lifestyle and diet and refrain from any additional exercise external to the training prescribed in the study. The design was approved by the local ethics committee (No. 14/2017) and all procedures adhered to the prerogatives set out in the Declaration of Helsinki.

Incremental exercise test
An incremental exercise test (IET) on a cycle ergometer was administered 3 days before and after the intervention to assess changes in CRF pre- and post-intervention. The IET was performed in controlled conditions in a climate-controlled (24ºC and 50% relative humidity) exercise laboratory (PN-EN ISO 9001:2009 certified). Testing was executed at the same time of day and participants were instructed to refrain from caffeine 24 hours prior to testing (Atkinson and Reilly, 1996). The cycle ergometer (Excalibur Sport, Lode BV, Netherlands) was adjusted to each participant and calibrated before each trial. Starting workload was 0 W and linearly increased by ~0.27 W s⁻¹ (50 W·3 min⁻¹) until volitional exhaustion. Gas exchange was measured breath-by-breath using a metabolic cart (Quark b2, Cosmed, Italy). The device was calibrated with a reference gas mixture of CO_2 (5%), O_2 (16%), and N_2 (79%). Respiratory function was measured 2 min prior to test start and continued 5 min after test conclusion with data averaged over 30-s intervals. HR was also continually measured with a non-invasive heart rate monitor (S810, Polar Electro, Finland). Primary outcome measures of VO_2 [mL·kg⁻¹·min⁻¹], VCO_2 [ml·min⁻¹], VE [l·min⁻¹], RER [$VCO_2·VO_2^{-1}$], and HR [beat·min⁻¹] were determined at four workloads (50, 100, 150, 200 W) and at maximal power (Max). VO_2max was defined as the highest 30-s average at in which relative VO_2 values plateaued (<1.35 mL·kg⁻¹·min⁻¹) despite an increase in workload or two of the following criteria: (a) RER >1.10, (b) attainment of HRmax (within 10 bpm of age-predicted maximum [220−age]), (c) voluntary exhaustion.

Intervention
Twelve swimming sessions were administered over a period of 6 weeks with at least 72 h of rest provided between each session. All sessions were held in a short course 25-m swimming pool in homogenous conditions (water temperature 27°C, air temperature 28°C at a relative humidity of 60%, pool illuminated at 600 lumens). The participants swam the front crawl for 50 min at a moderate intensity corresponding to 60% IET-obtained VO_2max. This intensity was selected as it was below the lactate threshold and suitable for prolonged exercise in untrained subjects and

most optimal for enhancing lipid metabolism (Aellen et al. 1993). Intensity was translated into a target HR for each individual and monitored by the subject with a waterproof heart rate monitor (RS400, Polar Electro, Finland) during each open turn (lap completion).

Group C breathed using normal front crawl breathing technique. Group E swam with a custom face mask integrated with a polypropylene frontal snorkel and 2.5-cm diameter ribbed tubing to provide 1000 ml of physiologic dead space (Figure 1). Dead space volume was identical for each participant and measured by filling the snorkel with water and then transferring the volume to a graduated cylinder as per Smolka et al. (2014). This volume was added with the volume of the custom face mask to ascertain a total volume of 1000 ml. The snorkel was sufficiently rigid to maintain a constant volume when swimming.

Figure 1. The ARDS apparatus (mouthpiece and snorkel).

Previous reliability and validity testing for this apparatus showed that a 1000 ml volume can be sufficient to provoke hypercapnia (partial pressure of pCO_2 in arterial blood >45 mmHg) and induce respiratory acidosis in which pCO_2 increased with a concomitant decrease in blood pH without causing hypoxia (Zatoń et al., 2010; Zatoń and Smołka, 2011; Hebisz et al., 2013; Smołka et al., 2014). This apparatus had also been previously verified for use during swimming (Zatoń and Ziebura, 2012; Adam et al., 2015). When swimming with the snorkel, the subjects wore a nose clip to eliminate nose breathing. Breathing frequency and the ratio of inhalations to exhalations were not controlled.

Assessment of glycerol concentration
Blood was drawn from the fingertip before and 3 min after completing the first (pre-intervention) and last (post-intervention) swim session to quantify the change in plasma glycerol concentration (GLY, micromol·l⁻¹). GLY was collected at this time point as the swimming sessions were sufficient in duration to fully promote lipolysis (Stallknecht et al. 1995). Quantitative enzymatic determination of GLY was performed using free glycerol reagent (Sigma-Aldrich, USA). The difference (Δ) before and after the first and last swim session was compared to serve as an indirect measure of change in pre- and post-intervention lipid metabolism.

Anthropometric measurements
Body mass index (BMI; $kg·m^{-2}$), fat mass (FM; kg), and fat-free mass (FFM; kg) were determined by near-infrared interactance (6100/XL, Futrex, Great Britain) at the middle of the biceps brachii muscle of the dominant extremity. Measures were collected pre- and post-intervention as an indirect measure of changes in lipid metabolism. The measurements were taken by a laboratory member with the device calibrated before each trial (Fukuda et al., 2017).

Statistical analysis
Data are given as means ± standard deviations (SD) and the difference (Δ) between pre- and post-intervention values. The distribution of the data set was screened for normality using the Shapiro–Wilk test and the homogeneity of variances checked with Levene's test. Cardiorespiratory outcomes (VO_2, VCO_2, VE, RER, HR) for each workload (50, 100, 150, 200 W and Max) were compared using repeated measures multivariate analysis of variance (MANOVA) for dependent samples and Tukey's Honest Significant Difference (HSD) test for pairwise post-hoc comparisons.

For anthropometric and metabolic outcomes (BMI, FM, FFM, ΔGLY), a normal distribution was not confirmed and the non-parametric Wilcoxon signed-rank test was used. Effect sizes were calculated using Cohen's d and interpreted as small (d=0.20 to 0.49), moderate (d=0.50 to 0.79), and large (d>0.80). All calculations were performed with the Statistica 13.1 software package (StatSoft, USA) and followed accepted methodology (Thoma et al., 2015). Significance was set at an alpha level of 0.05 for all statistical procedures with p values provided for all results. The sample size was estimated using a stand-alone power analysis program for statistical tests (G*Power 3.1.9.2, Kiel University, Germany) (Faul et al., 2007) with a small effect size of $f^2 = 0.29$. Assuming an alpha error of 0.05 and power of 0.80, the required total sample size was estimated to be 26 subjects. However, due to the length and commitment of the intervention, we were able to include only 22 individuals in the final analysis.

Results

Pre- and post-intervention cardiorespiratory and metabolic outcomes are presented in Table 1. No differences between groups were observed at post-intervention for VO_2 (p = 0.75), VCO_2 (p = 0.85), VE (p = 0.93), RER (p = 0.98), HR (p = 0.44), BMI (p = 0.69), FM (p = 0.71), FFM (p = 0.43), and ΔGLY (p = 0.21) (Table 1). Post-hoc analysis revealed a within-group difference only in group E for RER at maximum workload (p = 0.04, d = 1.30).

Discussion

Several studies have investigated the longitudinal effects of incorporating ARDS as an external aid to further enhance exercise-induced cardiovascular adaptations and lipid utilization in a variety of exercise modalities and intensities (Hebisz et al., 2013; Smołka et al., 2014). However, no studies to date have investigated the effects of an extended duration intervention in recreationally trained

Table 1. Pre- and post-intervention VO_2, VCO_2, VE, RER, HR with HSD post-hoc analysis for pre- and post-intervention differences for each group.

Variables	Power [W]	Group C					Group E				
		Pre-intervention	Post-intervention	Δ (post – pre)	P value	Cohen's d	Pre-intervention	Post-intervention	Δ (post –pre)	P value	Cohen's d
VO_2 [$mL·kg^{-1}·min^{-1}$]	50	17.7±3.8	17.2±2.8	-0.52	0.96	0.16	17.9±4.3	15.4 ±2.4	-2.58	0.13	0.77
	100	23.5±3.6	22.5±3.2	-1.08	0.75	0.32	24.5±4.5	22.5±3.2	-2.05	0.26	0.53
	150	29.6±4.4	28.3±3.6	-1.22	0.54	0.31	30.5±4.3	29.1±3.9	-1.45	0.40	0.38
	200	36.0±5.0	34.9±4.7	-1.07	0.70	0.22	35.8±4.5	35.4±5.4	-0.38	0.98	0.08
	Max	47.1±8.9	47.6±10.2	0.50	0.97	0.05	45.6±7.5	46.7±8.3	1.15	0.80	0.15
VCO_2 [$mL·min^{-1}$]	50	977±243	867±143	-109.74	0.97	0.16	988±179	962±153	-26.23	0.36	0.57
	100	1413±211	1316±99	-96.75	0.96	0.22	1390±146	1366±77	-24.21	0.31	0.62
	150	1864±189	1844±92	-19.92	0.74	0.33	1897±150	1950±172	52.68	0.98	0.13
	200	2404±177	2422±88	17.75	0.76	0.27	2505±171	2551±176	46.16	0.98	0.13
	Max	3534±861	3648±780	114.55	0.99	0.04	3811±845	3773±947	-37.51	0.80	0.30
VE [$l·min^{-1}$]	50	28.8±4.8	28.2±4.2	-0.56	0.99	0.12	26.9±6.0	24.6±3.3	-2.30	0.63	0.09
	100	39.0±4.1	37.4±2.6	-1.53	0.79	0.45	36.9±5.0	35.6±2.1	-1.38	0.83	0.13
	150	51.9±5.3	53.4±4.3	1.50	0.88	0.31	47.6±6.2	48.7±3.8	1.09	0.95	0.09
	200	71.5±12.3	72.4±7.0	0.89	0.98	0.09	66.4±12.0	66.8±7.4	0.38	0.99	0.04
	Max	132.3±35.1	135.8±39.8	3.51	0.92	0.09	121.5±39.5	124.8±37.1	3.33	0.93	0.09
RER	50	0.79±0.08	0.81±0.08	0.02	0.90	0.20	0.78±0.05	0.80±0.08	0.02	0.84	0.30
	100	0.84±0.04	0.84±0.05	0.00	0.99	0.04	0.83±0.04	0.84±0.07	0.01	0.98	0.10
	150	0.92±0.05	0.94±0.06	0.02	0.36	0.45	0.88±0.04	0.91±0.06	0.03	0.45	0.49
	200	0.99±0.05	1.00±0.05	0.01	0.60	0.25	0.97±0.04	0.98±0.05	0.01	0.83	0.18
	Max	1.38±0.08	1.45±0.09	0.07	0.26	0.78	1.31±0.12	1.42±0.12	0.11	**0.04***	1.30
HR [$beat·min^{-1}$]	50	113±20	104±11	-10.55	0.17	0.69	109±16	100.0±11.86	-8.91	0.30	0.65
	100	125±20	120±15	-4.55	0.74	0.27	126±15	117±13	-9.27	0.20	0.65
	150	146±18	142±16	-4.09	0.68	0.24	142±16	137±16	-5.00	0.53	0.31
	200	166±19	162±17	-4.01	0.53	0.23	161±17	156±17	-4.73	0.41	0.28
	Max	190±10	188±8	-2.46	0.83	0.28	182±10	185±14	2.18	0.87	0.18
BMI [$kg·m^{-2}$]		22.97±2.17	22.93±2.27	-0.05	0.28	0.02	22.99±2.24	23.05±2.17	0.06	0.64	0.03
FM [kg]		14.51± 6.02	15.12±5.89	0.16	0.858	0.10	16.90±5.17	17.45±5.34	0.55	0.44	0.10
FFM [kg]		57.64±11.46	56.55±8.83	-1.09	0.533	0.11	53.83±12.86	52.70±9.87	-1.13	0.59	0.10
GLY[$micromol·l^{-1}$]		Δ 39.10	Δ 74.40	35.30	0.476	0.55	Δ 41.60	Δ 68.50	26.90	0.28	0.57

*significantly different vs. pre-intervention value; VO_2 – oxygen uptake, VCO_2 – carbon dioxide production, VE – respiratory minute ventilation, RER – respiratory exchange ratio (VCO_2/VO_2^{-1}), HR – heart rate, BMI – body mass index, FM – fat mass, FFM – fat-free mass, GLY – glycogen concentration; data are mean ± SD

swimmers. The present experiment does not corroborate earlier findings, nor does it confirm the positive effects of ARDS-induced hypercapnia on respiratory, cardiovascular, and metabolic function (Toklu et al., 2003; Cathcart et al., 2005; Kato et al., 2005; Kumar and Bin-Jaliach, 2007; Kraut and Madias, 2014; Zoretić et al., 2014; Karaula et al., 2016). Post-intervention VO_2, VCO_2, VE, HR in the IET did not change in any of the selected workloads and only RER increased at maximal workload. No changes were observed in BMI, FM, FFM, and ΔGLY.

Previous research on hypercapnic exercise reported a decrease in RER, which had been interpreted as a substrate shift towards increased lipid utilization (Kato et al., 2005; Østergdard et al., 2012), which was confirmed via tissue analysis (Hollidge-Horvat et al., 1999). Hence, our original assumption that we would observe enhanced lipid oxidation with reduced body fat content when compared with identical albeit non-hypercapnic swim training. Instead, we found an isolated increase in post-intervention RER that was observed only during incremental exercise at maximal workload and not steady-state exercise as in the aforementioned studies. Previous studies have found VCO_2 to increase during incremental exercise as a result of hyperventilation and that the increased buffering of blood lactic acid may cause RER to no longer reflect substrate usage but higher ventilation rates and blood lactate levels

(Kenney et at., 2012).

No change was observed in ΔGLY in either the control or experimental group, confirming a similar level of hydrolyzed triglyceride with no enhanced reduction in adipose tissue. This was also confirmed by similar pre- and post-intervention BMI, FM, and FMM in the experimental group. This lack of change in ΔGLY could be explained by the discrepancy between the exercise modality (swimming with a large upper extremity component) and the testing modality (ergometer cycling with a dominant lower extremity component). Another consideration is the fact that hypercapnia has been found to promote adipogenesis (Kikuchi et al., 2017), possibly negating the fat-reducing effects of moderate-intensity exercise as adopted in the present study. A lack of significant differences can also be explained by the differences in GLY uptake by skeletal muscle during exercise (van Hall, 2002). Considering the above, it is also probable that the lack of difference in post-intervention cardiorespiratory (VO_2, CO_2, VE, RER and HR) and metabolic (BMI, FM, FFM, and GLY) measures can be explained by an inadequate exercise stimulus and not ARDS. The sample population was composed of young recreational swimmers with above average CRF (Kaminsky et al., 2015). The adopted training volume may have been insufficient to induce a quantifiable response in CRF or promote a substrate shift towards increased lipid utiliza-

tion despite the use of an exercise intensity (60% VO_2max) that had been previously used to induce hypercapnia (Graham et al., 1982; Smołka et al, 2014).

Regarding the lack of differences in the present study, several limitations should be considered. First, the literature strongly supports sports-specific fitness testing (Fernandes and Vilas-Boas, 2012). A swimming ergometer or swimming flume would have better replicated the swimming task and enhanced the validity of CRF testing (Demarie et al., 2001). However, there is evidence that there are no significant differences in peak VO_2 between simulated swimming on an incline bench, tethered swimming, and bicycle ergometry, in which both land- and water-based testing are a reliable and valid method of assessing VO_2max in swimmers (Kimura et al. 1990). Nonetheless, exercise testing similar to the exercise modality would have enhanced our findings.

A second issue was that pulmonary function was not assessed. Ventilation may have been modulated with the incorporation of ARDS and influenced the results. Future studies should consider the inclusion of spirometry testing in order to better quantify and interpret the effects of ARDS. Third, the diet of the participants was not strictly controlled nor was energy expenditure estimated. Fourth, previous studies on ARDS in swimmers applied an 8-week training program (Adam et al., 2015). However, we employed a 6-week intervention to determine if a shorter duration protocol could induce similar effects. It is possible that 8 weeks is the minimum timeframe required to fully promote the beneficial adaptations of ARDS.

Finally, a relatively small, homogeneous sample of health young adults was used. Future research should involve an untrained sedentary or overweight/obese population to further investigate training with ARD including modulated exercise frequency, exercise intensity, and tube volume. However, if future works consider modifying the exercise protocol, it needs to be mentioned that several participants in the experimental group reported headaches during training. This may be due to the dilation of cranial blood vessels and may have limited exercise performance. Furthermore, increasing the volume of the snorkel/tubing may induce hyperventilation, reducing pCO_2 and therefore minimizing the post-training effects of respiratory acidosis on RER and VCO_2 (Hollidge-Horvat et al., 1999). This could negate the promoted enhancement of cardiorespiratory and metabolic adaptations by ARDS. As many questions pertaining to ARDS remain unanswered, further research appears necessary. The present study may serve as a baseline and provide a theoretical and methodological grounding for future research to provide more objective data on the effects of ARDS on cardiorespiratory and metabolic variables in recreational swimmers.

Conclusion

A 6-week swimming intervention with ARDS performed at a moderate intensity corresponding to 60% VO_2max did not improve CRF (VO_2, VCO_2, VE, HR) measured in an IET. While post-intervention RER measured at maximum workload increased, this does not suggest a substrate shift towards increased lipid utilization during exercise. No difference was also observed in ΔGLY, suggesting a similar level of hydrolyzed triglyceride in the last swim session when compared with the first that was confirmed by a lack of change in adipose tissue (FM and BMI). Despite various limitations, the present study can serve as a guide for future research on the implications of ARDS-enhanced exercise.

References

Adam, J., Zatoń, M. and Wierzbicka-Damska, I. (2015) Physiological adaptation to high intensity interval training with added volume of respiratory dead space in club swimmers. *Polish Journal of Sports Medicine* **314**, 223-237.

Aellen, R., Hollmann, W. and Boutellier, U. (1993) Effects of aerobic and anaerobic training on plasma lipoproteins. *International Journal of Sports Medicine* **14(7),** 396-400.

Alkahtani, S. (2014) Comparing fat oxidation in an exercise test with moderate-intensity interval training. *Journal of Sports Science and Medicine* **13(1)**, 51-58.

Atkinson G. and Reilly T. (1996) Circadian variation in sports performance. *Sports Medicine* **21**, 292-312.

Cathcart, A.J., Herrold, N., Turner, A.P., Wilson, J. and Ward, S.A. (2005) Absence of long-term modulation of ventilation by dead-space loading during moderate exercise in humans. *European Journal of Applied Physiology* **93(4)**, 411-420.

Demarie, S., Sardella, F., Billat, V., Magini, W. and Faina, M. (2001) The VO2 slow component in swimming. *European Journal of Applied Physiology* **84**, 95-99.

Faul, F., Erdfelder, E., Lang, A.-G. and Buchner, A. (2007) G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods* **39(2),** 175-191.

Fernandes, R. J. and Vilas-Boas, J. P. (2012) Time to exhaustion at the VO2max velocity in swimming: A review. *Journal of Human Kinetics* **32**, 121-34.

Fukuda, D.H., Wray, M.E., Kendall, K.L., Smith-Ryan, A.E. and Stout, J.R. (2017) Validity of near-infrared interactance (FUTREX 6100/XL) for estimating body fat percentage in elite rowers. *Clinical Physiology and Functional Imaging* **37(4)**, 456-458.

Graham, T.E., Wilson, B.A., Sample, M., Van Dijk, J. and Goslin, B. (1982) The effects of hypercapnia on the metabolic response to steady-state exercise. *Medicine & Science in Sports & Exercise* **14**, 286-291.

van Hall G., Sacchetti M., Rådegran G. and Saltin B. (2002) Human skeletal muscle fatty acid and glycerol metabolism during rest, exercise and recovery. *The Journal of Physiology* **15**, 543(Pt 3), 1047-58.

Hebisz, P., Hebisz, R. and Zatoń, M. (2013) Changes in breathing pattern and cycling efficiency as a result of training with added respiratory dead space volume. *Human Movement* **14(3),** 247-253.

Heinicke, K., Heinicke, I., Schmidt, W. and Wolfarth, B. (2005) A threeweek traditional altitude training increases hemoglobin mass and red cell volume in elite biathlon athletes. *International Journal of Sports Medicine* **26**, 350-355.

Hollidge-Horvat, M.G., Parolin, M.L., Wong, D., Jones, N.L. and Heigenhauser, G.J. (1999) Effect of induced metabolic acidosis on human skeletal muscle metabolism during exercise. *American Journal of Physiology* **277**, 647-658.

Jones, N.L. (2008) An obsession with CO2. *Applied Physiology, Nutrition, and Metabolism* **33(4)**, 641-650.

Jorgić, B., Puletić, M., Okičić, T. and Meškovska, N. (2011) Importance

of maximal oxygen consumption during swimming. *Physical Education and Sport* **9(2)**, 183-191.

Kaminsky, L. A., Arena R. and Myers, J. (2015) Reference Standards for Cardiorespiratory Fitness Measured With Cardiopulmonary Exercise Testing: Data From the Fitness Registry and the Importance of Exercise National Database. *Mayo Clinic Proceedings* **90(11),** 1515-1523.

Karaula, D., Homolak, J. and Leko, G. (2016). Effects of hypercapnic-hypoxic training on respiratory muscle strength and front crawl stroke performance among elite swimmers. *Turkish Journal of Sport and Exercise* **18**, 1, 17-24.

Kato, T., Tsukanaka, A., Harada, T., Kosaka, M. and Matsui, N. (2005) Effect of hypercapnia on changes in blood ph, plasma lactate and ammonia due to exercise. *European Journal of Applied Physiology* **95**(5-6), 400-408.

Kelman, G.R. and Watson, A.W.S. (1973) Effect of added dead-space on pulmonary ventilation during sub-maximal, steady-state exercise. *Quarterly Journal of Experimental Physiology* 58, 305-313.

Kenney, W.L., Wilmore, J.H. and Costill, D. (2012) *Physiology of Sport and Exercise*. 5th edition. Human Kinetics, Champaign, Illinois. 247-282.

Khayat, R.N., Xie, A., Patel, A.K., Kaminski, A. and Skatrud, J.B. (2003) Cardiorespiratory effects of added dead space in patients with heart failure and central sleep apnea. *Chest* **123(5)**, 1551-60.

Kikuchi, R., Tsuji, T., Watanabe, O., Yamaguchi, K., Furukawa, K., Nakamura, H. and Aoshiba, K. (2017) Hypercapnia accelerates adipogenesis: a novel role of high CO2 in exacerbating obesity. *American Journal of Respiratory Cell and Molecular Biology* **57(5)**, 570-580.

Kimura, Y., Yeater, R.A. and Martin, R.B. (1990) Simulated swimming: a useful tool for evaluation the VO2 max of swimmers in the laboratory. *British Association of Sport and Medicine* **24(3)**, 201-206.

Koppers, R.J., Vos, P.J. and Folgering, H.T. (2006) Tube breathing as a new potential method to perform respiratory muscle training: safety in healthy volunteers. *Respiratory Medicine* **100**, 714-720

Kraut, J.A. and Madias, N.E. (2014) Lactic acidosis. *New England Journal of Medicine* **371(24)**, 2309-2319.

Kumar, P. and Bin-Jaliach, I. (2007) Adequate stimuli of the carotid body: More than an oxygen sensor? *Respiratory Physiology and Neurobiology* **157(1)**, 12-21.

Lahart, I.M. and Metsios, G.S. (2018) Chronic physiological effects of swim training interventions in non-elite swimmers: A systematic review and meta-analysis. *Sports Medicine* **48(2)**, 337-359.

Latt, E., Jurimae, J., Haljaste, K., Cicchella, A., Purge, P. and Jurimae, T. (2009) Longitudinal development of physical and performance parameters during biological maturation of young male swimmers. *Perceptual and Motor Skills* **108 (1)**, 297-307.

McLellan, T.M. (1991) The influence of a respiratory acidosis on the exercise blood lactate response. *European Journal of Applied Physiology and Occupational Physiology* **63(1)**, 6-11.

Mercier, J., Ramonatxo, M. and Prefaut C. (1992) Breathing pattern and ventilatory response to CO2 during exercise. *International Journal of Sports Medicine* **13**, 1-5.

Michalik K., Zalewski I., Zatoń M., Danek N. and Bugajski A. (2018) High intensity interval training with added dead space and physical performance of amateur triathletes. *The Polish Journal of Sports Medicine* **4(4)**, 247-255.

Millet, P.G., Roels, B., Schmitt, L., Woorons, X. and Richaled, P. (2010) Combining hypoxic methods for peak performance. *Sports Medicine* **40(1)**, 1-25.

Østergaard, L., Kjaer, K., Jensen, K., Gladden, L.B., Martinussen, T. and Pedersen, P.K. (2012) Increased steady-state VO2 and larger O2 deficit with CO2 inhalation during exercise. *Acta Physiologica* **204**, 371-381.

Schneider, A.G., Eastwood, G.M., Bellomo, R., Bailey, M., Lipcsey, M., Pilcher, D. and Suzuki, S. (2013) Arterial carbon dioxide tension and outcome in patients admitted to the intensive care unit after cardiac arrest. *Resuscitation* **84(7)**, 927-934.

Smołka, L., Borkowski, J. and Zatoń M. (2014) The effect of additional dead space on respiratory exchange ratio and carbon dioxide production due to training. *Journal of Sports Science and Medicine* **13(1)**, 36-43.

Stallknecht, B., Simonsen, L., Billpw, J., Vinten, J. and Galbo, H. (1995) Effect of training on epinephrine-stimulatedmlipolysis determined by microdialysisnin human adipose tissue. *American Journal of Physiology* **269(32),** E1059-E1066.

Thoma, J.R., Nelson, J.K. and Silverman, S.J. (2015) *Research Methods in Physical Activity*. 7th edition. Human Kinetics, Champaign, Illinois. 166-167.

Toklu, A.S., Kayserilioğlu, A., Ünal, M., Özer, Ş. and Aktaş, Ş. (2003) Ventilatory and metabolic response to rebreathing the expired air in the snorkel. *International Journal of Sports Medicine* **24(3)**, 162-165.

Zatoń, M, Hebisz, R. and Hebisz, P. (2010) The effect of training with additional respiratory dead space on haematological elements of blood. *Isokinetics and Exercise Science* **18**, 137-143.

Zatoń, M. and Smołka, L. (2011) Circulatory and respiratory response to exercise with added respiratory dead space. *Human Movement* **12(1)**, 88-94.

Zatoń, M. and Ziebura, Z. (2012) The significance of training with additional respiratory dead space in development of physical capacity in swimming. In: *Science in Swimming*. Eds: Zatoń, K., Rejman, M., Klarowicz, A. 4 edition. Wrocław: AWF Wrocław. 125-148.

Zoretić, D., Grčić-Zubčević, N. and Zubčić, K. (2014) The effects of hypercapnic-hypoxic training program on hemoglobin concentration and maximum oxygen uptake of elite swimmers. *Kinesiology: International Journal of fundamental and Applied Kinesiology* **46**, 40-45.

Key points

- The study compared the effects of a 6-week swimming intervention with and without added respiratory dead space (ARDS).
- Swimming with ARDS did not improve cardiorespiratory fitness measures (VO_2, VCO_2, VE, HR) obtained in an incremental exercise test.
- Post-intervention RER increased after the ARDS swimming intervention, but this change does not suggest a substrate shift towards increased lipid utilization during exercise as evidenced by a lack of change in post-exercise glycerol concentration.
- No changes were observed in BMI, FM, and FFM.

AUTHOR BIOGRAPHY

Stefan SZCZEPAN
Employment
Department of Swimming, University School of Physical Education in Wroclaw, Poland
Degree
PhD
Research interests
Motor control and learning, motor skill acquisition processes, swimming performance, swimming science
E-mail: stefan.szczepan@awf.wroc.pl

Kamil MICHALIK
Employment
University School of Physical Education in Wroclaw, Poland, Department of Physiology and Biochemistry
Degree
PhD
Research interests
Exercise physiology, performance testing, added respiratory dead space, interval training
E-mail: kamil.michalik@awf.wroc.pl

Jacek BORKOWSKI
Employment
University School of Physical Education in Wroclaw, Poland, Department of Physiology and Biochemistry
Degree
PhD
Research interests
Exercise biochemistry, immunochemistry
E-mail: jacek.borkowski@awf.wroc.pl

Krystyna ZATON
Employment
Department of Swimming, University School of Physical Education in Wroclaw, Poland
Degree
Full Professor
Research interests
Movement science and swimming science, swimming teaching and learning, didactic communication
E-mail: krystyna.zaton@awf.wroc.pl

✉ **Stefan Szczepan, PhD**
Department of Swimming, University School of Physical Education in Wroclaw, Poland