



The Effect of Different Training Loads on the Lung Health of Competitive Youth Swimmers

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ABSTRACT

International Journal of Exercise Science 11(6): 999-1018, 2018. Airway hyperresponsiveness (AHR), airway inflammation, and respiratory symptoms are common in competitive swimmers, however it is unclear how volume and intensity of training exacerbate these problems. Thus, our purpose was to measure AHR, inflammation, and respiratory symptoms after low, moderate, and high training loads in swimmers. Competitive youth swimmers (n=8) completed nine weeks of training split into three blocks (Low, Moderate, and High intensity). Spirometry at rest and post-bronchial provocation [Eucapnic Voluntary Hyperpnea (EVH)] and Fractional Exhaled Nitric Oxide (FeNO) were completed at the end of each training block. A weekly self-report questionnaire determined respiratory symptoms. Session Rating of Perceived Exertion (sRPE) quantified internal training loads. Internal load was significantly lower after Moderate training (4840 ± 971 AU) than after High training (5852 ± 737 AU) ($p = 0.02$, $d = 1.17$). Pre-EVH FEV₁ was significantly decreased after Moderate (4.52 ± 0.69 L) compared to Low (4.74 ± 0.63 L) ($p = 0.025$, $d = 0.326$), but not different from High load. Post-EVH FeNO after Moderate training was significantly decreased (9.4 ± 4.9 ppb) compared to Low training (15.4 ± 3.6 ppb) ($p = 0.012$, $r = 0.884$). Respiratory symptom frequency was significantly correlated with percent decrease in FEV₁ 20 minutes post-EVH after Low and Moderate loads (both $\rho = -0.71$, sig = 0.05), and after High load was significantly correlated with percent decrease in FEV₁ at 10 ($\rho = -0.74$, sig = 0.03), 15 ($\rho = -0.91$, sig = 0.00), and 20 minutes post ($\rho = -0.75$, sig = 0.03). In conclusion, Moderate load training resulted in the worst lung health results, suggesting there may be factors other than the total amount of stress within training blocks that influence lung health. Further research is needed to determine the effect of manipulating specific acute training load variables on the lung health of swimmers.

KEY WORDS: Athletes, elite sport, exercise-induced bronchoconstriction, lung health, swimming

INTRODUCTION

Exercise-induced bronchoconstriction (EIB), known as the “acute airway narrowing that occurs as a result of exercise”(43) and airway hyperresponsiveness (AHR), defined as “an abnormal susceptibility to airway narrowing following exposure to a wide range of bronchoconstricting stimuli”(11) are the most common chronic medical conditions experienced

by Olympic athletes (8%) (23). In particular, elite endurance athletes have greater airway dysfunction, respiratory tract illness and symptoms compared with non-endurance elite athletes (14,23,36,38,40). A distinguishing feature is the high ventilation requirement, a phenomenon ascribed as “high ventilation sport dysfunction” (16,44).

Competitive swimmers undergo high volume, high intensity training while sustaining elevated ventilation rates in chlorinated pool environments (20,41). Training several hours per week under these conditions has been associated with increased exposure to chlorine by-products and unfavourable changes to lung health compared to recreational swimming (11,19,29,48). High ventilation sustained in swimming may negatively affect the airway epithelium by promoting mucus dehydration and increased shear stress on the epithelial wall of the lungs (11,12). The prevalence of EIB is 11-29% in competitive swimmers (28,43), however the pool environment may influence the mechanism in which it occurs. AHR is a key characteristic associated with EIB and is prevalent in up to 79% of elite swimmers (46,57). Airway inflammation, known as the protective response to airway injury (usually associated with asthma), may be increased in swimmers and is likely due to chloramine-induced changes to the lung. Airway inflammation can be determined through induced sputum analysis (12), however an innovative and potentially less invasive technique is measuring Fractional Exhaled Nitric Oxide (FeNO), which may also be used as a surrogate for AHR (2). Further investigation is needed to establish how AHR, airway inflammation, and EIB are interrelated.

There is also a high incidence of respiratory tract infections in competitive swimmers, likely due to acute alterations in immune function and tissue damage repair after intense swimming exercise (6,8). Higher frequency of reported illness also occurs when training surpasses an athlete’s individual optimal training stress threshold (25), or other training program variables including sudden increases in volume, lack of periodization and recovery, and highly monotonous training (38). Thus, the *intensity* of training loads likely influences the development of respiratory problems in competitive swimmers beyond exposure to chlorine by-products (36), and is of key interest for our study. We do know that AHR status might change with training, where transition from “intense” to “light/no training” reduced AHR 67% in previously hyperresponsive younger adult swimmers (19±2 years) (10). However, intensity of training was not objectively quantified.

To our knowledge, no study has quantified both the external training loads – the physical work completed by the athlete (27) – and the internal training loads – the relative amounts of physiological and psychological stress (27) – to determine what influence training has on AHR, inflammation, and respiratory symptoms in competitive swimmers. Further, most studies on elite-level athletes have been performed in adults with very high training ages and often do not reveal detailed information about the training program that would allow us to understand how AHR might increase or decrease from week to week. Although youth swimmers have fewer of years of competition and strenuous exercise compared to adult athletes, they still train and compete several hours per day and undergo intense training. With this identified gap in the literature, we were interested in studying weekly training loads in younger, high performance swimmers. Ideally, we hoped quantifying training loads and measuring the lung

health of youth-aged swimmers would add detail and specificity to improving our understanding of the development of AHR.

Thus, the primary purpose of our study was to examine the effect of training for three weeks at each low, moderate, and high loads, and the resulting internal training stress on the lung health of competitive youth swimmers. Lung health was determined by airway inflammation, AHR, EIB, and respiratory symptoms. We hypothesized that swimmers' lung health would worsen after every three weeks of training with the increase in training load from low, to moderate, to high. Specifically, we predicted swimmers would have greater % decreases in FEV₁ post-EVH challenge (i.e. more hyperresponsive to provocation), increased FeNO levels (more airway inflammation), and increased frequency of respiratory symptoms determined by a self-reported questionnaire.

METHODS

Participants

Eight competitive swimmers (four males, four females) aged 14-17 years from a high-performance swim program participated. Based on a previous study (46) of elite female swimmers and their responses to airway provocation tests, 5 of 16 swimmers (31%) had positive EVH tests, with a mean fall of 0.70L (SD = 0.35L) in FEV₁ post-EVH, equivalent to 18% (SD = 8.4%). Another study (15) revealed 18 of 33 elite swimmers (55%) had a positive EVH challenge, with a mean fall in FEV₁ of 20.4 (SD = 11.7%) from baseline. Using this information, 11-21 participants would be required to detect a similar change with a 95% confidence level and 5% allowable error, determined using the SD of the % fall in FEV₁ reported in previous studies that was associated with positive EVH tests. Given the intensive nature of this study we aimed for at least 11 swimmers based on the apriori sample size determination. However, the high-performance group that we were able to recruit to this study was smaller than we had hoped and only 8 participants provided assent to participate. Thus, 8 swimmers successfully completed the study.

All participants had the same training and meet competition schedule. The head coach allowed the swim training loads to be determined in length and intensity for the study and all participants were assigned the same workouts. The study received Institutional Research Ethics Board approval, and all participants provided informed consent (Pro00066445). All procedures were in accordance with the ethical standards of the Helsinki Declaration. Participants were included if they were currently training and free of any diagnosed illness, such as flu or fever, respiratory infection, or musculoskeletal injury prohibiting participation in this training group; however, no invited swimmer was excluded based on these criteria. Swimmers with a history of asthma or respiratory symptoms associated with exercise were not excluded, as these individuals could help better understand the profile of swimmers with lung health problems, and we were interested in lung health changes that occur with training.

Protocol

This study utilized a single group, within-subjects, quasi-experimental design. Participants completed the same tests three times, on the last day of Low, Moderate, and High training load blocks in the first nine weeks of their program from September-November. Measurements completed during laboratory visits (Figure 1) included FeNO, spirometry, and EVH challenge testing. A swim fatigue and health questionnaire was completed once per week to determine respiratory symptoms. Session Rating of Perceived Exertion (sRPE) was reported once daily, or twice if there were two training sessions. As per previous recommendations (3), participants did not complete any training or strenuous exercise 24 hours prior to the start of the test for each testing day. The Allergy Questionnaire for Athletes (AQUA©) (7) was completed at the first testing session to screen for previous respiratory symptoms and atopy. The Physical Activity Readiness Questionnaire (PAR-Q+) (50) ensured participants had no contraindications to exercise, as the EVH test involves high ventilation.

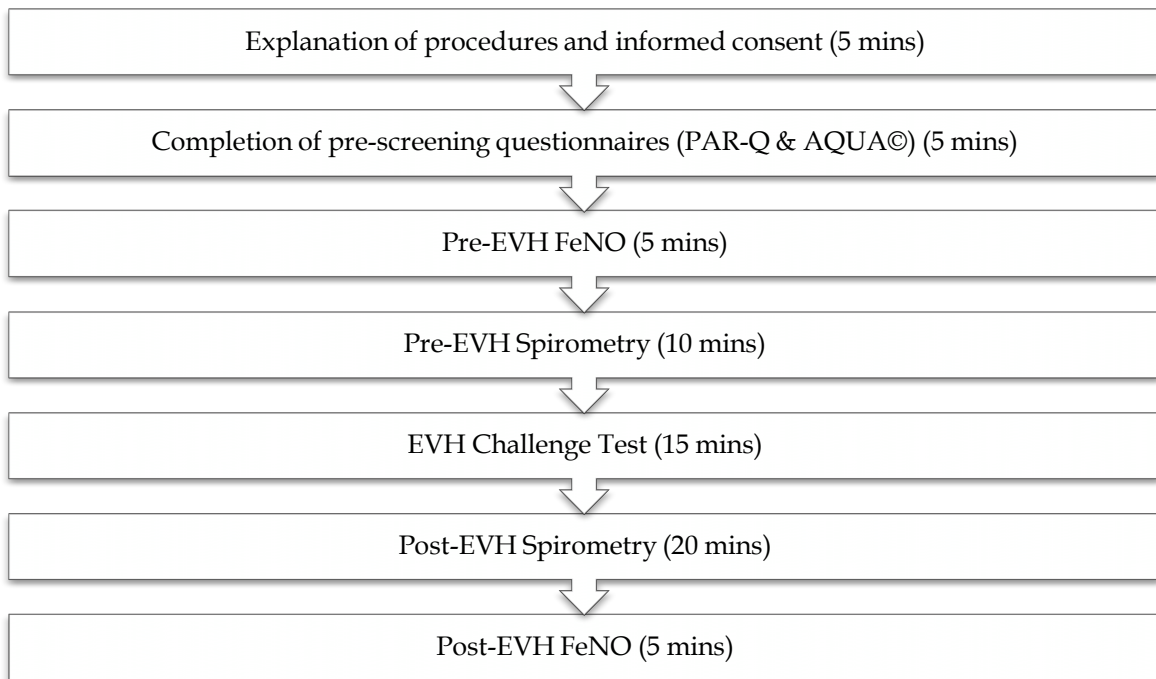


Figure 1. Flow-chart timeline for testing sessions. The process remained the same on all three sessions. The first two steps were completed in the first session. AQUA© = Allergy Questionnaire for Athletes; EVH = Eucapnic Voluntary Hyperpnea; FeNO = Fractional Exhaled Nitric Oxide; PAR-Q = Physical Activity Readiness Questionnaire.

FeNO was measured pre- and post-EVH challenge to respectively determine the level of chronic inflammation and whether the response to EVH reveals a change in FeNO values, so as to improve our understanding of the relationship between inflammation and AHR in swimmers. We used a NIOX MINO® monitor (Aerocrine AB, Solna, Sweden) with a single-breath online technique, following current guidelines and recommendations for measuring exhaled nitric oxide (2,21). Participants inhaled to total lung capacity and exhaled at constant pressure (10-20 cm H₂O) for 10 seconds, guided by visual and auditory aids to stabilize flow

rate (50 ± 5 ml/s) (1). The test requires low flow and maximum inhalation one time, and thus does not aggravate the airway prior to the EVH test. No participant reported the FeNO measurement as difficult or undue symptoms related to the test.

Spirometry was completed pre- and post-EVH challenge to determine the magnitude of AHR and whether a swimmer was EIB positive or negative based on % decreases in FEV₁ from baseline. Measurements included forced vital capacity (FVC) and forced expiratory volume in one second (FEV₁) with a portable handheld spirometer (Spirodoc Touchscreen Spirometer, Medical International Research, Rome, Italy) according to ATS/ERS guidelines for the standardization of spirometry (39). Forced expiratory flow at 25-75% of FVC (FEF 25-75%); forced expiratory flow at 50% (FEF 50%); and peak expiratory flow rate (PEF) were also included in the measurements to better understand small airway obstruction and EIB in competitive athletes (18,51). A minimum of three trials were performed to obtain three FEV₁ values within 150 ml. Spirometry was completed post-EVH test in duplicate within 30 seconds ("immediately post"), 5, 10, 15, and 20 minutes. If FEV₁ differed by ≥ 150 ml at a given time point, an additional FEV₁ trial was performed. A 10% fall in FEV₁ observed at two consecutive time points within 20 minutes post-test was considered a positive indication of EIB (22,34).

The EVH challenge is a gold standard bronchoprovocation challenge used for diagnosing EIB in athletes, and thus is the provocation method we chose for our study. The EVH challenge is meant to provoke airway obstruction, therefore it is common to experience chest tightness, shortness of breath, and cough – symptoms that typically resolve within 24 hours. The protocol was based on previous laboratory procedures and IOC-MC recommendations for identifying EIB (3,31,42). Participants breathed at a target rate of 30 breaths/min (metronome) and with a tidal volume equal to FEV₁ (visual feedback from a digital chart recorder [LabChart, ADInstruments, Colorado Springs, USA] which approximates 85% Maximal Voluntary Ventilation (MVV) for 6 minutes (3). An inspired dry gas mixture of 21% O₂, 5% CO₂, and balance N₂ was used to avoid hypocapnia (34). Participants were asked to refrain from caffeine, alcohol, and any medications that might influence lung function (24 hours for short-acting β -agonists and 72 hours for inhaled corticosteroids) (34).

Session Rating of Perceived Exertion (sRPE) (scale 1-10) was used to determine internal training loads (the individual response to training), and whether these were reflective of changes in external training loads and lung function between training blocks. We used sRPE developed by Foster (25), as it is a simple and reliable (56) method for determining exercise intensity in swimmers that can eliminate the need to use heart rate monitors or other equipment that are less practical for use in water. Participants were asked to report their sRPE via text message link within 30 minutes of finishing their training session (typically by 7:00am for morning sessions and by 7:00pm for evening sessions). Each athlete's sRPE was multiplied by the approximate training session duration in minutes (and summed if there were multiple sessions in a day) to determine daily internal training load. This was then used in the calculation of total weekly internal training load, training monotony, and training strain (25). Weekly internal load was the sum of the daily internal loads. Monotony was calculated as the

daily mean training load divided by the standard deviation of the daily training loads. Strain represented the product of the weekly training load and monotony (25).

A traditional linear periodization approach was applied over a period of nine weeks and divided into three training blocks consisting of three weeks each, classified as Low, Moderate, and High training load, as shown in Figure 2. Prescribed pool and dryland training volume were calculated individually and then summed to determine the total prescribed external load per week. For consistency, external monotony and external strain were also calculated like the internal calculations above. Methodology used to determine external load followed that of Mujika et al (41), which multiplied kilometres by intensity factors based on a stress index scale established according to theoretical blood lactate levels in different training sets. Mean training load for each block was 47 ± 17 AU (Low), 75 ± 15 AU (Moderate), and 114 ± 18 AU (High). External load was calculated retrospectively in the first two weeks of training (30 ± 3 AU and 55 ± 2 AU), and prospective participant data collection for the study began in the third week of training. Laboratory tests were completed on the last day of each training block.

A weekly online self-report questionnaire was used to identify frequency of respiratory symptoms, and how symptom frequency changes from week to week with increased training intensity. We also were interested in the correlation of symptoms to EVH challenge spirometry measures (% decrease in FEV₁ at each post-EVH time point), as swimmers may be positive for EIB without consistently reporting symptoms. The questionnaire divided symptoms into Upper (Nose/Sinuses), Middle (Throat), and Lower (Chest) respiratory tract symptoms. The sum of all three categories (18 total possible symptoms) provided each athlete with a weekly total respiratory symptom frequency score.

Statistical Analysis

Statistical analyses were performed using the IBM Statistical Package for Social Sciences (SPSS) for Macintosh, Version 22.0.0 (SPSS Inc., Chicago, IL). An alpha level of 0.05 was chosen to indicate significance for all analyses, as this study has a small sample size and is a novel approach to understanding the relationship between different training loads and lung health in swimmers.

A single-group repeated measures ANOVA (1 group x 3 training loads [Low, Moderate, High]) examined differences in raw values for pre- and post-EVH FVC, FEV₁, FEF 25-75%, FEF 50%, and PEF (at immediate, 5 min, 10 min, 15 min, 20 min post-test), as well as maximal % decreases in FEV₁ post-EVH between training blocks. Repeated measures ANOVA (1 group x 3 training loads [Low, Moderate, High]) also evaluated differences between training blocks in mean respiratory symptoms, and means of each internal load, internal monotony, and internal strain. LSD pairwise comparisons were used to evaluate differences in each variable between training blocks.

An outlier was detected for FeNO measurements (Grubb's outlier test), therefore a Friedman's ANOVA (1 group x 3 training loads [Low, Moderate, High]) was used to determine differences in pre- and post-EVH FeNO across training blocks. Wilcoxon Signed Rank Tests were used to

determine pairwise differences between blocks, as well to evaluate differences in FeNO within testing days between pre- and post-EVH.

Pearson correlation coefficients with 95% confidence interval (CI) (two-tailed) were calculated between External and Internal Load estimates, as we expected there would be a linear relationship between these two variables. Spearman rank order correlation coefficients with 95% CI (two-tailed) were calculated between spirometry measurements and corresponding weekly respiratory symptoms (e.g. Week 3 respiratory symptoms during the last week of the Low block were reported in the corresponding week of Test 1). Spearman correlation was chosen in place of Pearson, as we were interested in evaluating whether participants who ranked higher on symptom frequency also ranked higher on % decreases in FEV₁, yet there is paucity of evidence for a linear relationship between these variables.

RESULTS

Participant characteristics are shown in Table 1. Swimmers had a wide range of years participating in competitive swimming (2-10 years). One female swimmer was diagnosed with mild asthma and was prescribed a short-acting beta-agonist, a long-acting corticosteroid, and a metered corticosteroid nasal spray by a physician and had a positive AQUA[®] score. No other swimmers had a history of asthma. Four other swimmers also had positive (≥ 5) AQUA[®] scores and reported they had previously experienced respiratory symptoms (shortness of breath, chest tightness, cough, and/or itching of the throat) during and/or following training sessions, however no swimmer reported severe respiratory distress or was removed from a swim practice or event due to an asthma or EIB event. The training program consisted of 7-9 swim sessions, 3-4 dryland sessions, and 1 day off per week during the study period.

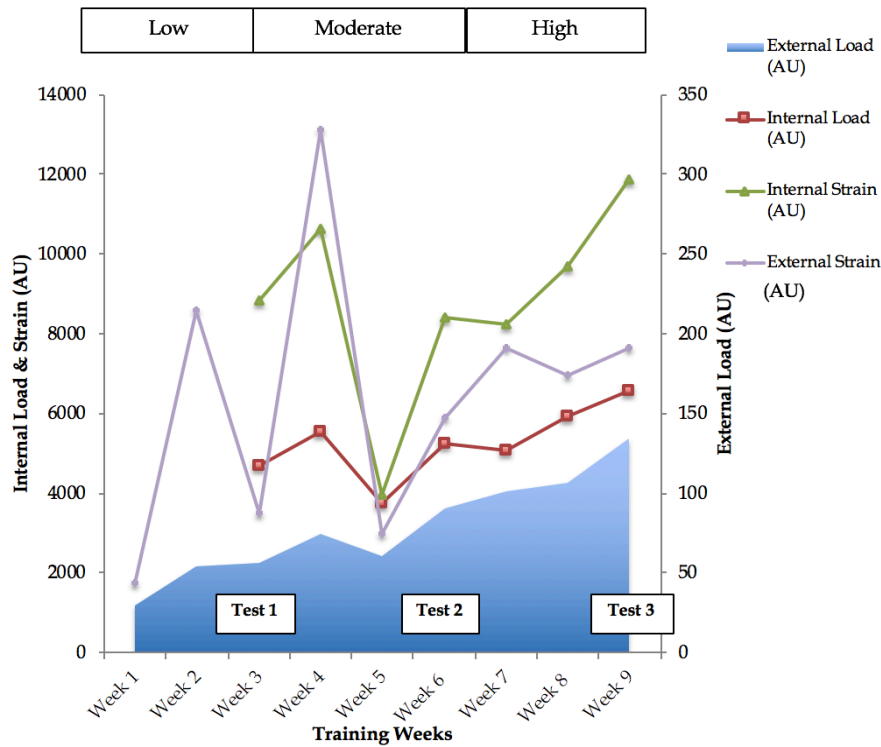


Figure 2. Weekly external load, external strain, internal load, and internal strain over nine weeks of swim training. Training loads are indicated for three-week blocks, with tests occurring at the end of the blocks. Data on internal load and strain were not collected until Week 3. External Load and Strain were calculated retrospectively for the first two weeks of training.

Table 1. Participant characteristics.

ID #	Sex (M/F)	Age (yrs)	Mass (kg)	Height (cm)	Competitive swimming (yrs)	Weekly training (h)	Training Load			AQUA [®]	EIB positive tests
							Low (h) ^b	Moderate (h) ^b	High (h) ^b		
1	F	16	61.4	178	3	15	16	15	16	13 ^a	Low, Moderate
2	F	16	59.1	175	3	13	16	15	11	15 ^a	Low, Moderate, High
3	M	17	79.5	183	10	16	16	14	19	5 ^a	None
4	F	14	58.2	165	8	19	16	19	21	0	Low, High
5	M	17	95.0	175	4	19	16	19	21	2	Moderate
6	F	17	59.1	170	9	19	16	19	20	8 ^a	Moderate, High
7	M	16	70.9	191	5	19	16	19	20	2	Low, Moderate, High
8	M	16	76.2	178	2	19	16	19	21	5 ^a	Low, Moderate, High
Mean		16.1	69.9	176.9	5.5	17.4	16	17.4	18.6	6.3	
SD		1.0	13.1	7.9	3.1	2.4		2.3	3.5	5.4	2 ^c

Note: cm = centimetres; F = female; h = hours; ID # = participant identification number; kg = kilograms; L = low; M = male; yrs = years; ^a = positive score (≥5) on Allergy Questionnaire for Athletes (AQUA[®]); ^b = presented as estimated mean training hours per week; ^c = median number of positive tests.

Friedman's test revealed pre-EVH FeNO did not differ across training loads ($p = 0.42$), however post-EVH FeNO was significantly different ($p = 0.006$) (Table 2, Fig. 4). Wilcoxon Signed Ranks Test showed post-EVH FeNO after Low (15.4 ± 3.6 ppb) was significantly higher than after Moderate training loads (9.4 ± 4.9 ppb) ($Z = -2.5$, $p = 0.012$, $r = 0.884$). Wilcoxon Signed Ranks Test also showed post-EVH FeNO was significantly lower than pre-EVH after Moderate (pre = 16.6 ± 7.4 ppb, post = 9.4 ± 4.9 ppb, $Z = -2.5$, $p = 0.011$, $r = 0.884$) and High training loads (pre = 16.8 ± 3.5 ppb, post = 13.6 ± 4.5 ppb, $Z = -2.1$, $p = 0.034$, $r = 0.742$).

Table 2. Pre- and post-EVH FeNO after Low, Moderate, and High training loads.

	Low	Moderate	High
FeNO Pre-EVH (ppb)	19.0 ± 8.4	16.6 ± 7.4	16.8 ± 3.5
FeNO Post-EVH (ppb)	$15.4 \pm 3.6^*$	$9.4 \pm 4.9^{*\ddagger}$	$13.6 \pm 4.5^\ddagger$

Note: * = Wilcoxon Signed Ranks between training blocks $p < 0.05$; † = Wilcoxon Signed Ranks pre- to post-EVH $p < 0.05$; ppb = parts per billion

Seven swimmers (88%) were positive for EIB (>10% fall in FEV₁ on two consecutive time points post-EVH) for at least one testing session (Table 1). Five (63%) swimmers were positive for EIB after Low load; six (75%) after Moderate; and five (63%) after High. The mean maximum % falls in FEV₁ after low, moderate, and high training loads were $-14.3 \pm 9.2\%$, $-13.9 \pm 4.1\%$, and $-12.0 \pm 7.6\%$, respectively.

Repeated measures ANOVA showed a main effect of training load on pre-EVH FEV₁ values ($F(2,14) = 4.036$, $p = 0.041$, $\eta_p^2 = 0.366$), shown in Table 3 and Figure 3. LSD pairwise comparisons revealed significantly lower mean pre-EVH FEV₁ after Moderate (4.52 ± 0.69 L) compared to after Low (4.74 ± 0.63 L) ($p = 0.025$, $d = 0.326$), while pre-EVH FEV₁ values after High (4.52 ± 0.63 L) were not significantly different from Low ($p = 0.083$) or Moderate ($p = 1.00$). There was also a main effect of training load on 20-min post FEF 25-75% ($F(2,14) = 3.930$, $p = 0.044$, $\eta_p^2 = 0.360$). These values after Moderate (3.73 ± 0.94 L/s) were significantly lower than after High (4.07 ± 1.02 L/s) ($p = 0.02$, $d = 0.372$) (Table 3). Maximum % changes in FEV₁ did not differ significantly across training blocks ($p = 0.763$) (Figure 4). None of the other spirometry measures at pre- or post-EVH sampling points differed between training blocks.

Mean respiratory symptoms did not significantly differ by training block. Spearman correlations revealed total respiratory symptoms in the last week of the Low training load were significantly negatively correlated with % change in FEV₁ 20 minutes post ($\rho = -0.71$, $p = 0.05$) in the corresponding week. Respiratory symptoms in the last week of the Moderate training load were also significantly negatively correlated with % change in FEV₁ 20 minutes post ($\rho = -0.71$, sig = 0.05) in the corresponding week. Respiratory symptoms in the last week of the high training load were significantly negatively correlated with % change in FEV₁ at 10 minutes post ($\rho = -0.74$, sig = 0.03), 15 minutes post ($\rho = -0.91$, sig = 0.00), and 20 minutes post ($\rho = -0.75$, sig = 0.03) in the corresponding week. All other sampling points and spirometry values tested were not significantly correlated with symptoms.

There was a positive Pearson correlation between weekly external load and weekly internal load ($r = 0.83$, $p = 0.02$), as shown in Table 4 and Figure 5. Repeated measures ANOVA revealed significant differences among training blocks on internal load ($F(2,14) = 5.166$, $p = 0.02$, $\eta_p^2 = 0.425$) and internal monotony ($F(2,14) = 5.472$, $p = 0.02$, $\eta_p^2 = 0.439$), but not internal strain ($p = 0.074$). LSD pairwise comparisons showed internal load was lower in the Moderate block (4840 ± 971 AU) than in the High block (5852 ± 737 AU) ($p = 0.02$, $d = 1.17$). Internal monotony was lower in the Moderate block (1.5 ± 0.4 AU) than the Low block (1.9 ± 0.3 AU) ($p = 0.01$, $d = 1.13$) (Table 4).

Table 3. Spirometry pre-EVH testing and at each post-EVH sampling point after low, moderate, and high training loads.

		Pre-EVH	Immediately				
			Post	5 mins	10 mins	15 mins	20 mins
FVC (L)	Low	5.84 ± 0.74	5.70 ± 0.76	5.54 ± 0.58	5.43 ± 0.75	5.41 ± 0.84	5.49 ± 0.98
	Moderate	5.65 ± 0.86	5.48 ± 0.82	5.44 ± 0.84	5.24 ± 0.77	5.44 ± 1.29	5.41 ± 1.04
	High	5.56 ± 0.94	5.31 ± 0.80	5.36 ± 0.75	5.34 ± 0.70	5.31 ± 0.76	5.31 ± 0.78
FEV ₁ (L)	Low	4.74 ± 0.63*	4.48 ± 0.63	4.26 ± 0.45	4.14 ± 0.46	4.15 ± 0.54	4.22 ± 0.64
	Moderate	4.52 ± 0.69*	4.26 ± 0.62	4.12 ± 0.61	3.99 ± 0.42	4.12 ± 0.72	4.18 ± 0.68
	High	4.52 ± 0.63	4.15 ± 0.69	4.13 ± 0.56	4.14 ± 0.53	4.23 ± 0.50	4.25 ± 0.54
FEF 25-75% (L/s)	Low	4.69 ± 1.12	4.08 ± 0.87	3.72 ± 0.62	3.42 ± 0.66	3.52 ± 0.80	3.81 ± 0.83
	Moderate	4.34 ± 1.04	3.80 ± 0.89	3.52 ± 0.94	3.43 ± 0.58	3.51 ± 0.57	3.73 ± 0.94*
	High	4.47 ± 1.05	3.82 ± 1.04	3.67 ± 0.91	3.69 ± 1.07	3.93 ± 0.77	4.07 ± 1.02*
FEF 50% (L/s)	Low	5.23 ± 1.39	4.35 ± 0.98	4.27 ± 0.89	3.96 ± 0.80	4.09 ± 0.97	4.21 ± 0.85
	Moderate	4.71 ± 1.12	4.30 ± 1.14	4.08 ± 1.10	3.85 ± 0.71	4.02 ± 0.75	4.20 ± 1.11
	High	4.90 ± 1.22	4.25 ± 1.26	3.88 ± 1.16	4.26 ± 0.98	4.39 ± 0.95	4.45 ± 1.03
PEF (L/s)	Low	8.91 ± 1.30	8.32 ± 1.34	7.83 ± 1.36	7.55 ± 1.08	7.65 ± 1.26	7.56 ± 1.25
	Moderate	8.43 ± 1.52	7.92 ± 1.29	7.76 ± 1.18	7.49 ± 1.24	6.92 ± 3.12	8.05 ± 1.70
	High	8.42 ± 1.53	7.98 ± 1.63	7.78 ± 1.58	7.76 ± 1.62	7.93 ± 1.72	7.80 ± 1.61

Note: FEF 25-75% = mean forced expiratory flow at 25-75% of FVC; FEF 50% = forced expiratory flow at 50% of FVC; FEV₁ = forced expiratory volume in 1 second; FVC = forced vital capacity; PEF = peak expiratory flow; mins = minutes; * = LSD pairwise comparison between training blocks for corresponding time points $p < 0.05$.

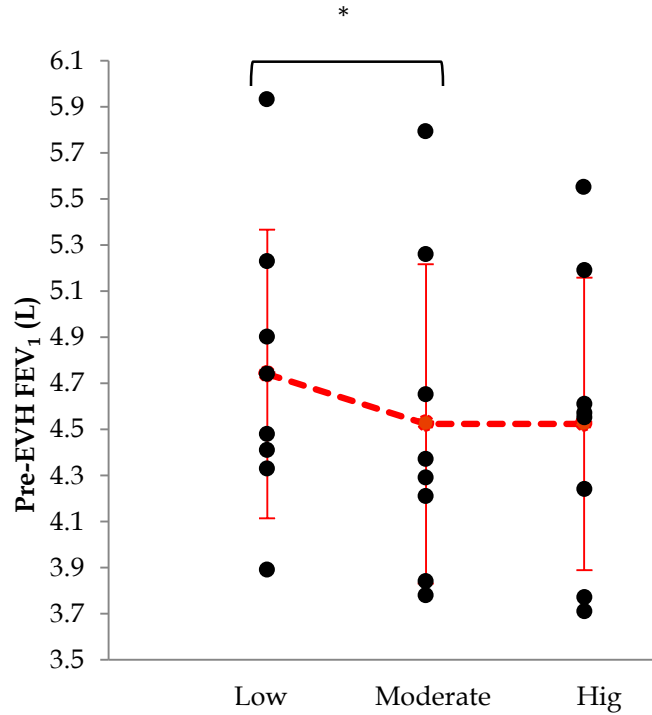


Figure 3. Individual FEV₁ (L) values prior to the EVH test for each testing session. Mean indicated by orange dashed line with standard deviation error bars. * = LSD pairwise comparison between training blocks $p < 0.05$.

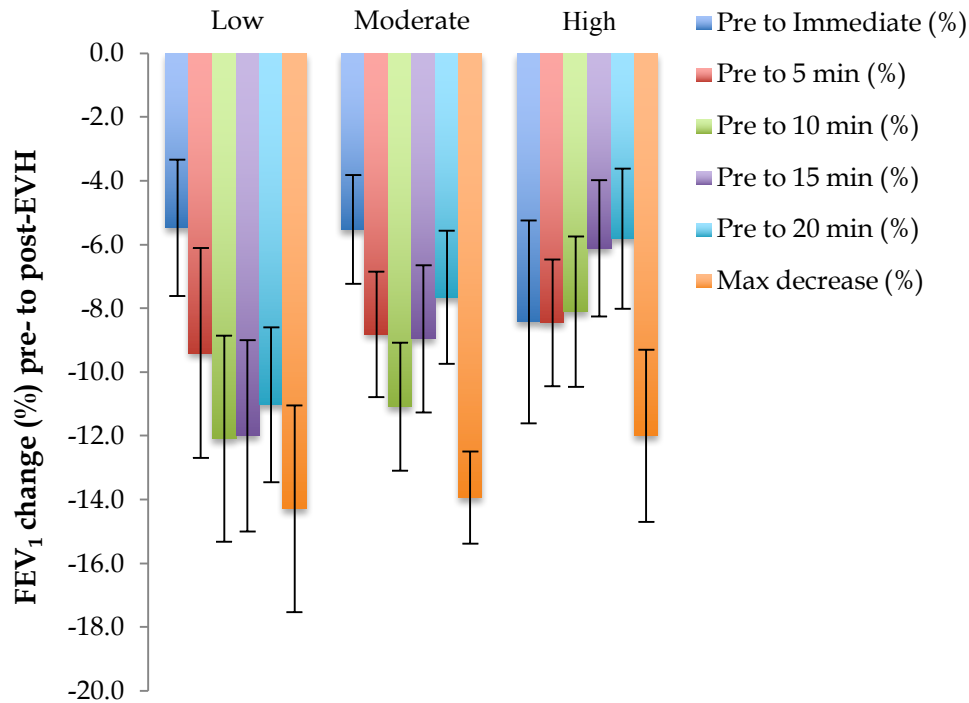


Figure 4. Percent changes in FEV₁ from pre-EVH to each post-EVH sampling point after low, moderate, and high training loads, displayed as means with standard error bars. Maximal FEV₁ decreases did not differ significantly across training loads. Max = maximum; min = minute.

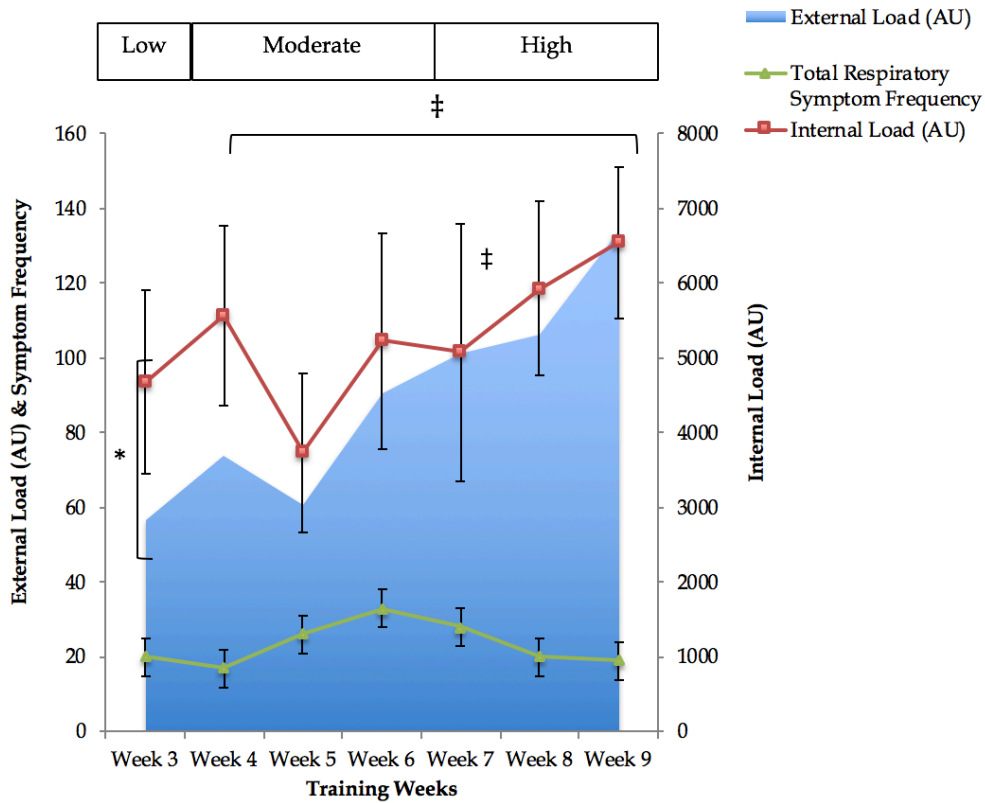


Figure 5. External training load, internal training load, and total respiratory symptom frequency over Weeks 3-9 displayed with standard deviation error bars. Training blocks are separated by dashed line. Symptom frequency is displayed as a group sum of reported upper, middle, and lower respiratory tract symptoms. AU = arbitrary units. * = Pearson correlation between weekly internal and external training loads $r = 0.83$, $p < 0.05$; ‡ = LSD pairwise comparison between Moderate and High internal load blocks $p < 0.05$; $r = 0.83$.

Table 4. External training load and internal training stress summary.

		External Training Load			Internal Training Stress			
Training Block	Week	External Load* (AU) ^b	External Monotony (AU)	External Strain (AU)	sRPE	Internal Load* (AU) ^b	Internal Monotony (AU) ^b	Internal Strain (AU) ^b
Low	1 ^a	30 ± 3	1.4	43				
	2 ^a	55 ± 2	3.9	214				
	3	56 ± 5	1.5	87	5.2 ± 1.1	4684 ± 1238	1.9 ± 0.3	8839 ± 2954
Block Mean ± SD		47 ± 17	2.3 ± 1.4	115 ± 89				
Moderate	4	74 ± 2	4.4	328	4.9 ± 1.4	5559 ± 1207	1.9 ± 0.2	10608 ± 2368
	5	61 ± 7	1.2	74	4.8 ± 0.8	3735 ± 1062	1.1 ± 0.3	3956 ± 1932
	6	90 ± 7	1.6	147	5.1 ± 1.1	5226 ± 1456	1.5 ± 0.4	8416 ± 3922
Block Mean ± SD		75 ± 15	2.4 ± 1.8	183 ± 131	5.0 ± 1.0	4840 ± 971‡	1.5 ± 0.4‡	7660 ± 3390
High	7	101 ± 6	1.9	191	5.6 ± 1.0	5078 ± 1721	1.6 ± 0.5	8248 ± 4309
	8	106 ± 7	1.6	173	5.8 ± 1.1	5931 ± 1161	1.6 ± 0.4	9679 ± 3686
	9	134 ± 7	2.1	280	6.5 ± 1.0	6547 ± 1017	1.8 ± 0.3	11872 ± 3290
Block Mean ± SD		114 ± 18	1.9 ± 0.2	215 ± 57	6.0 ± 0.5	5852 ± 737‡	1.7 ± 0.1‡	9933 ± 1825

Note: AU = Arbitrary Units; RPE = Rating of Perceived Exertion; ^a = internal stress data not collected, and external load calculated retrospectively in these weeks; ^b = weekly mean ± SD; * = Pearson correlation $p < 0.05$; ‡ = LSD pairwise comparison between blocks $p < 0.05$.

DISCUSSION

The aim of this research was to better understand the influence of training load on lung health. Specifically, we investigated how external training load and internal training stress affect airway responsiveness to an EVH challenge, airway inflammation, and exacerbate respiratory symptoms in competitive youth swimmers. A Eucapnic Voluntary Hyperpnea (EVH) challenge has been recommended as the gold standard to understand AHR status for EIB diagnosis (32), however it is typically done only once in annual medical screens, usually in athletes that have respiratory symptoms associated with AHR. Thus, we measured responses to an EVH challenge after prescribed training blocks that differ in intensity; this has not been done previously in any high ventilation sport population. Additionally, how Fractional Exhaled Nitric Oxide (FeNO) as an index of airway inflammation changes over varied training loads in swimmers is not known.

We did find that an increase in external training load affects resting lung function (FEV₁), although a decrease only occurred from low to moderate (4.74 ± 0.63 L to 4.52 ± 0.69 L; $p = 0.04$ with a 60% load increase) (Figure 3, Table 3). From moderate to high training load, pre-EVH FEV₁ was not significantly different (4.52 ± 0.69 L compared to 4.52 ± 0.63 L with a 52% load increase), thus other factors besides total volume overload might be influencing this resting lung function. There may be a chronic effect of accumulated training load – in other words, swimmers might be more susceptible at the beginning of a training season when they are adjusting to a new training program, while later in the season they are able to tolerate higher training loads without concomitant changes in airway obstruction. It may also be that the

magnitude threshold of increase between blocks that negatively influences lung health lies somewhere between 52-60%.

Regardless of training load, seven swimmers (88%) had positive EVH tests during at least one of the testing sessions (Table 1) and swimmers in our study reached their maximum % decreases in FEV₁ around 10 minutes post-EVH, comparable to other research (4). Such responses have been found in adult elite swimmers (10,46) while much less is known about youth swimmers. A previous study (45) suggested youth involvement in competitive swimming for approximately two years is not sufficient for development of respiratory symptoms or airway inflammation, but could possibly result in minor increases in AHR. Our swimmers were on average two years older (16.1 ± 1.0 compared to 14.3 ± 1.2), had been in competitive swimming for three years longer (5.5 ± 3.2 compared to 2.7 ± 1.3), and presented a more vigorous response to provocation than the swimmers Pedersen et al (45) studied. Furthermore, three swimmers were positive on all three tests, but had only been swimming competitively for 2-5 years. Thus, our results suggest that about three additional years of competitive swimming (about 2,700 total hours based on an estimate of annual training for the three years preceding the present study) influences AHR, and could mark an important transition to severity and prevalence of AHR reflective of adult elite swimmers. Future studies should investigate how years of experience correlate to EIB status and AHR severity.

Eosinophilic airway inflammation is a characteristic feature of asthma (5), thus we predicted that individuals with positive EVH tests would also have elevated FeNO levels at rest. However, contrary to our hypothesis, swimmers who were EIB positive showed normal resting FeNO values (<25 ppb) in each training block (Table 2), except for one asthmatic swimmer who had intermediate values (>25 ppb but <50 ppb) (21); resting FeNO values were also not significantly different between blocks. Others have found that FeNO measured in swimming (45,47) and other high ventilation sports may not be considered high (>50 ppb) (53).

Post-EVH FeNO was significantly lower after Moderate training (9.4 ± 4.9 ppb) (Table 2), which might be explained by the low 20-min FEF 25-75% (3.73 ± 0.94 L/s) characteristic of increased airway obstruction (Table 3). After high training loads, post-EVH FeNO (13.6 ± 4.5 ppb) and 20-min FEF 25-75% (and 4.07 ± 1.02 L/s) increased from moderate, which also supposes post-EVH FeNO might be affected by greater airway obstruction from bronchoprovocation. Deykin et al (17) found that NO measured in the expired gas of asthmatic patients decreased after repeated spirometry efforts, but that bronchoprovocation may counteract this fall in expired NO. However, given that the swimmers in our study completed a bronchoprovocation challenge followed by repeated spirometry, the significant decreases in post-EVH FeNO between training blocks suggests swimmers who are hyperresponsive may express lower FeNO values after provocation rather than high expired NO levels at rest.

Table 3 shows the progression of airway responsiveness before and after the EVH test. Our preliminary data indicates that those who were more hyperresponsive (greater % decreases in FEV₁ post-provocation) and slower to recover from the EVH test (FEV₁ remained decreased for longer post-provocation) also had greater respiratory symptom frequency. Closer examination

of individual symptom data (not shown here) revealed more hyperresponsive swimmers tended to report at least one upper respiratory symptom in the week coinciding with the EVH test, primarily rhinorrhea (runny nose) and/or phlegmy throat. Thus, monitoring symptoms on a regular basis could provide additional insight into the development of AHR, despite previous research indicating AHR may be observed in the absence of symptoms or vice versa (13). A previous study (26) also determined training 1.5-4 hours a day, 3-5 times per week – similar to our study – is associated with high prevalence (74%) of rhinitis symptoms. Thus, participation in competitive swimming at this frequency and volume is perhaps enough of a stimulus to provoke chronic respiratory symptoms, and provides some indirect evidence about risk for AHR (i.e. those with rhinitis and the subsequent length of time rhinitis is reported) likely are more hyperresponsive. What measurable effect symptoms and AHR have on performance requires further investigation, but it can be hypothesized that coaches would observe speed or time-related decrements given the linear relationship that exists between oxygen consumption and swimming velocity (35).

In the recent IOC consensus statement on load in sport and risk of illness (52), only two studies have examined the changes in training load and the risk of illness in swimmers (30,49). Furthermore, training monotony as a risk factor for illness has only been studied recently in elite cross-country skiers (54) and rugby league players (55). Our data suggests that monotony is a key feature of training load that could negatively impact athlete health, including lung health and symptoms. Specifically, the moderate external training loads were the most monotonous (2.43 ± 1.76 AU) which resulted in the greatest total respiratory symptom frequency and a concomitant decrease in pre-EVH FEV₁, (six swimmers were EIB positive in the moderate block). Moreover, in the moderate block the coaching staff increased training frequency to achieve higher volume while maintaining similar training intensity as the low block. Thus, this data supports the premise that the manipulated training variables of frequency and pattern of training (monotony) might be important features of how a high ventilation athlete responds to the overall training load. Yet, the internal stress data in the moderate block does not agree with this explanation because we would expect internal training variables to be higher if lung health is poor. Persistent respiratory symptoms might have caused swimmers to purposefully ease off in training to allow sufficient rest and to prevent overreaching, which could explain the relatively lower RPEs and thus the lower internal load (4840 ± 971 AU) and internal monotony (1.5 ± 0.4 AU) that we observed in the moderate block (38). Low internal load and monotony with moderate external training loads – yet obstructed airways at rest – suggests prescribed increases in external training load could negatively influence lung health more than internal markers of training stress (37).

From an EIB status standpoint, our results do not indicate status stability across time-points. Some swimmers were positive on one test, yet negative on the next (when we hypothesized that lung health gets worse). We cannot discount that a small sample size ($n=8$) increases the possibility of Type 1 Error, and that other factors, such as EIB phenotype, might influence a swimmer's response to a provocation test. We also did not measure markers of the airway remodeling process, such as Goblet cell hyperplasia obtained by bronchial biopsies, or inflammatory cells (neutrophils, T cells, eosinophils, and mast cells) (9). These may be

influenced by the possible reversibility of AHR, which could affect whether a swimmer is positive or negative for EIB at the time of measurement (10). Although the EVH test has been shown to be reproducible in swimmers over a short time frame (24 hours) (34), our findings show swimmers can change from EIB positive to EIB negative in just three weeks, which could have implications for therapeutic use exemption (TUE). Considering an approved TUE for an inhaled beta-2 agonist (IBA) is valid for 4 years (24) testing athletes more frequently to determine the need for an IBA may be more appropriate. Moreover, we question whether the EVH test is an appropriate evaluation of EIB in swimmers given the environmental differences of air in indoor swimming pools (warm and humid) versus air used for the EVH test (room temperature and dry). Although swim specific field tests has shown poor predictability for lung health as well (33), future studies should endeavor to recruit a larger cohort of swimmers and consider a controlled hyperventilation challenge that mimics the unique environmental conditions of indoor swimming.

While we had aimed to recruit at least 11 swimmers, our homogeneous cohort of 8 swimmers provided a significant volume of meaningful data. It was certainly challenging to carry out a training study with a cohort of athletes who were all actively training for the same competitions, and who were willing to participate in research that they would perceive as beneficial (or would not affect) the quality of training. Furthermore, negotiation with head coaches and the swim club was necessary to ensure scientific integrity of the data, including the ability to agree upon the prescribed training intensity and workouts that would accommodate the schedule of important qualifying meets. Many sport scientists do not undertake studies such as ours that require daily monitoring for many reasons, which may include lack of resources, not reaching agreements with clubs and coaches, or simply that swimming coaches (especially of high-profile clubs) or athletes themselves would prefer not to publish training information. Determining external load and internal load in this cohort required extreme diligence and attention to detail, which we believe has strengthened our conclusions.

In summary, it appears that increases in training load affect the development of chronic airway obstruction, inflammation, and respiratory symptoms in swimmers, however how lung health is impacted by training load is more complex than simply higher training loads equal worse lung health. Specifically, the transition from low to moderate challenged lung health in these swimmers, and the pattern of training load in the moderate block led to worse health than in the high training block. These results indicate that a provocation test such as an EVH is not imperative to determining lung function in youth swimmers. In fact, tracking occurrence of respiratory symptoms *and* measuring resting spirometry on a weekly basis – or at least at the beginning of a defined training block – could provide a cost-effective method to monitor overall lung health status in competitive youth swimmers. Waiting to test lung function in athletes only when they present with chronic respiratory symptoms at rest or only one time in an annual medical screen could result in overlooking those who may not have consistent symptoms, but are hyperresponsive after more intense training. We recommend that coaches, athletes, and the integrated support team (e.g. sport scientists, physicians, etc.) establish several lung function testing periods throughout the season to ensure all athletes can receive

appropriate treatment, especially leading up to key swim meets. However, further research in a larger sample is necessary to better understand relationships between weekly respiratory symptoms and spirometry. Based on preliminary data, seemingly minor symptoms such as a runny nose or phlegmy throat that persist over several weeks may be early indicators of AHR, and therefore should not be disregarded. Additionally, having a means of quantifying both external and internal training loads has merit, as it could help coaches manage the overall variation in the training prescription. Load patterns could be adjusted within microcycles to possibly lower the risk of undue fatigue and illness. Implementing a monitoring system could also allow coaches to enhance relationships and communication with athletes and support staff not only in day-to-day swimming practice, but also regarding overall health and wellness that impacts swimming performance.

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