DOES COMPETITIVE SWIMMING DURING PUBERTY AFFECT LUNG DEVELOPMENT IN PUBERTAL FEMALES?

by

Joshua Maschio Bovard B.Sc., McGill University, 2012

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ABSTRACT

Whether the large lungs of competitive swimmers result from intensive swim training or genetic endowment has been widely debated. Given that peak growth velocities for the lungs occur during puberty, this longitudinal study aimed to determine if competitive swimming during puberty affected lung development. Female swimmers (n=11) and healthy controls (n=10) aged 11-14 years old were assessed before and after one competitive swimming season. Pulmonary function testing included lung volumes, spirometry, diffusion capacity (D_{L,CO}), and maximal inspiratory (PI_{MAX}) and expiratory (PE_{MAX}) pressures. Ventilatory constraints, including endexpiratory lung volume (EELV), expiratory flow limitation (EFL), and utilization of ventilatory capacity (\dot{V}_E/\dot{V}_{ECAP}), were assessed during an incremental cycling test. Despite being of similar age (p=0.10), maturational development (p=0.27), and height (p=0.38) as controls, swimmers had a larger total lung capacity (p < 0.01), forced vital capacity (p < 0.01), and peak expiratory flow (p=0.03). Although D_{L,CO} was greater in swimmers (p=0.01), there was no difference when expressed relative to alveolar volume (p=0.20). Both PI_{MAX} (p=0.06) and PE_{MAX} (p<0.001) were greater in swimmers. Swimmers and controls achieved a similar relative maximal oxygen consumption (p=0.32) and experienced similar ventilatory constraints as characterized by EELV (p=0.18), severity (p=0.95) and prevalence (p=0.71) of EFL, and \dot{V}_E/\dot{V}_{ECAP} (p=0.95). Changes over time were similar between groups (p>0.05). Pubertal female swimmers already had larger lung capacities, higher flows, and greater indices of respiratory muscle strength, but similar ventilatory constraints while cycling. One competitive swimming season did not further accentuate this enhanced function or alter exercise ventilatory mechanics, suggesting that competitive swimming during puberty did not affect lung development.

LAY SUMMARY

Whether the large lungs of competitive swimmers result from intensive swim training or genetic endowment has been widely debated. Because lung growth is greatest during puberty, this thesis compared lung function before and after one swimming season in 11-14-year-old, similarlysized female swimmers and healthy controls. At the initial measurement, the swimmers already had larger lung capacities, higher flows, and greater indices of respiratory muscle strength that occurred irrespective of training experience. One swimming season did not further accentuate this enhanced lung function, and no associations between changes in lung function and swim training volume were found. Moreover, detailed analyses of physiological development of the lungs and the respiratory challenges imposed by swimming provided no unequivocal evidence that swimming can alter lung development. Despite having greater lung function, swimmers had similar ventilatory responses as controls while cycling. Thus, this thesis concluded that competitive swimming during puberty did not affect lung development.

PREFACE

This thesis contains original data collected and analyzed for partial fulfilment of Joshua Bovard's Master of Science degree. All protocols were approved by the University of British Columbia's Children's and Women's Clinical Research Ethics Board (approval certificate number: H15-00977). The research question and experimental protocol were developed by Joshua Bovard and Drs William Sheel, Kristin Houghton, Donald McKenzie, and James Potts. Data was collected and analyzed by Joshua Bovard.

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LIST OF ABBREVIATIONS

A-aDO ₂	Alveolar-arterial oxygen difference
AHR	Airway hyperresponsiveness
ANOVA	Analysis of variance
β°	β-angle
BL	Resting baseline
BMI	Body mass index
bpm	Beats per minute
BSA	Body surface area
BTPS	Body temperature and pressure, saturated
Cdyn	Dynamic compliance
CFB	Controlled frequency breathing
CO	Carbon monoxide
CO ₂	Carbon dioxide
CON	Controls
Cst	Static compliance
Crs	Respiratory system compliance
CV	Closing volume
CVE	Central vascular engorgement
Cw	Chest wall compliance
D _{L,CO}	Diffusion capacity for the lungs measured using carbon monoxide
DL,COC	D _{L,CO} corrected for hemoglobin
DR	Dysanapsis ratio
EELV	End-expiratory lung volume
EFL	Expiratory flow limitation
EIAH	Exercise-induced arterial hypoxemia
EILV	End-inspiratory lung volume
EPL	Exercise Physiology Laboratory in the Children's Heart Centre
ERV	Expiratory reserve volume
EVH	Eucapneic voluntary hyperpnea
fв	Breathing frequency

FECO ₂	Fraction of expired carbon dioxide
FEF	Forced expiratory flow
FEF25-75%	Maximum mid-expiratory flow (between 25% and 75% of the FVC)
FEF25%	Maximum expiratory flow when 25% of the FVC has been expired
FEF50%	Maximum expiratory flow when 50% of the FVC has been expired
FEF75%	Maximum expiratory flow when 75% of the FVC has been expired
F _E O ₂	Fraction of expired oxygen
FEV ₁	Forced expiratory volume in one second
FR	Flow ratio
FRC	Functional residual capacity
FSB	Freestyle breathing pattern ("hypoxic training")
FVC	Forced vital capacity
FVL	Tidal flow-volume loop
Gaw	Airway conductance
gFVC	Graded forced vital capacity
GH	Growth hormone
Hb	Hemoglobin
**He	Helium
HR	Heart rate
IC	Inspiratory capacity
IRV	Inspiratory reserve volume
Ксо	Transfer coefficient of the lung (i.e., $D_{L,CO}/V_A$)
MCh	Methacholine
MEFV	Maximum expiratory flow-volume curve
MVV	Maximum voluntary ventilation
O ₂	Oxygen
PaCO ₂	Arterial carbon dioxide tension
PaO ₂	Arterial oxygen tension
PAQ	Physical activity questionnaire
PEF	Peak expiratory flow
PE _{MAX}	Maximum expiratory pressure

PFT	Pulmonary function test
PGV	Peak growth velocity
PIMAX	Maximum inspiratory pressure
POST	Follow-up visit
PRE	Initial visit
Pst(L)	Static recoil pressure of the lungs
Pst(L) ₅₀	Static recoil pressure of the lungs at 50% of VC
Q	Cardiac output
Raw	Airway resistance
RC	Respiratory Clinic
RER	Respiratory exchange ratio
RPE	Rating of perceived exertion
rpm	Revolutions per minute
RT	Respiratory therapist
RV	Residual volume
SaO2	Arterial oxygen saturation
SD	Standard deviation
SE	Standard error of the mean
sGaw	Specific airway conductance
SMR	Sexual maturity rating
SR	Slope ratio
SS	Snorkel set
STPD	Standard temperature and pressure, desaturated
SV	Stroke volume
SWIM	Swimmers
TLC	Total lung capacity
UK	Underwater (dolphin or breast) kick
VA	Alveolar volume
Ė₄/ġ	Ventilation-perfusion ratio
Vc	Pulmonary capillary blood volume
VC	Vital capacity

VCO 2	Carbon dioxide production
\dot{V}_{E}	Expired minute ventilation
V ECAP	Ventilatory capacity
V̈ _E /V̈CO ₂	Ventilatory equivalent for carbon dioxide
V E/ V ECAP	Utilization of ventilatory capacity
$\dot{V}_E/\dot{V}O_2$	Ventilatory equivalent for oxygen
Ϋ́Ι	Inspired minute ventilation
ΫO ₂	Oxygen consumption
VO 2MAX	Maximal oxygen consumption
VT	Tidal volume
W _{MAX}	Peak work rate
WOB	Work of breathing

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INTRODUCTION

The respiratory system is generally thought to be ideally designed to meet the demands of exercise in healthy young adults (1). However, unlike the cardiovascular and musculoskeletal systems, the respiratory system does not exhibit significant beneficial adaptations to endurance training (2, 3). There are no known measurable beneficial structural adaptations, and, of the limited number of functional adaptations, the majority are related to changes in respiratory musculature (e.g., increased respiratory muscle endurance or strength) (2). As a result, the respiratory system can become a limiting factor of performance in highly trained athletes (2-4) and can negatively adapt to exercise in cold (5) or chlorinated (6) environments. While chronic exercise training appears to confer no change to the respiratory system, an exception may be in young, competitive swimmers.

1.1 Competitive swimming and lung development

The pulmonary function of competitive swimmers is characterized by large lung capacities (7-31), greater expiratory flows (11, 14, 18, 29-31), and increased diffusion capacities (19, 22, 24, 29, 31-35). This enhanced function has been suggested to be beneficial for swimming (14). For example, increased functional residual capacity (FRC) may act as a reservoir for gas exchange, thereby attenuating oscillations in arterial blood gases between breaths (14). Additional benefits may include greater buoyancy in the water to decrease drag and improved ventilatory capacity (14). In fact, it has been suggested that having large lungs is a pre-requisite for becoming a top swimmer (12). However, it has been widely debated whether this enhanced pulmonary profile is an adaptation to swim training (8, 13, 16, 18, 21, 33), the result of young athletes self-selecting into swimming based on favourable genetic endowments (17), or both (9-11, 14, 15, 23, 27).

The argument for adaptive growth is based on four unique challenges that competitive swimming places upon the developing respiratory system. First, swimming is performed in the prone or supine position with the body partially or fully submerged (36). Second, swimmers use an "obligatory, controlled frequency" breathing pattern that is dependent on both physiological need and timing of arm strokes (36). Third, swim training often involves breath control drills, including "hypoxic training", and sprint swimming where breathing frequency is reduced. Lastly, intense and structured swim training begins as early as 5 y old (17). Hypothetically, the first three stressors may cause changes in ventilatory mechanics, greater inspiratory pressures (14), and excessive upper body work (14) and/or require transient breath-holding maneuvers (11). These have been suggested to augment growth of the thoracic cavity and musculature (14) and/or create an intermittent hypoxic stimulus for lung growth (29). However, mechanistic evidence is lacking. More importantly, competitive swimmers are exposed to these stressors during periods of maximal lung growth between 1 month and 7 years of age (37) and during puberty (38). Thus, if the growing respiratory system is sensitive to induced growth, then competitive swimming during these critical periods is likely to elicit the greatest effects.

Table 1 and Table 2 list 18 cross-sectional (20-34, 39-41) and 16 longitudinal (6 <1 year (7, 14, 19, 35, 42, 43) and $10 \ge 1$ year (8-13, 15-18)) studies that have reported pulmonary function data in young swimmers throughout development. Overall, increased lung volumes of competitive swimmers have been observed compared to predicted values (13, 24, 25, 29, 31), population norms (8-10, 12, 21), and measurements in controls (7, 11, 14-18, 20, 22, 26-30, 40, 41). Greater expiratory flows have been measured in competitive swimmers compared to controls (11, 14, 16, 18, 20, 22, 28-30, 40) and predicted values (24, 25, 29, 31), as have diffusion capacities compared to controls (11, 22, 29, 34, 39) and predicted values (24, 29, 31-33, 35). Conversely, maximal static

mouth pressures, which have been seldom reported, have often been found to be similar in swimmers relative to controls (15, 27-29). However, within the published literature there is considerable between-study variation in the competitive level of the swimmers (e.g., experience and training volume), study length, and experimental design and analysis. Moreover, some have not observed any differences in lung volumes between swimmers and a control group (33, 39, 42, 43). Therefore, a more detailed review of this literature is necessary to provide a comprehensive understanding of the relationship between competitive swimming and lung development. This includes addressing the following questions:

- 1. At what age are swimmers first reported to have larger lung capacities compared to a normal control group or predicted values?
- 2. Given that lung volume increases 13-fold between 1 month and 7 y (37), does swim training prior to age 7 y have any effect on lung capacities?
- 3. When children begin intensive swim training, do they already have greater lung capacities?
- 4. Do longitudinal analyses show greater-than-expected growth of lung capacities?
- 5. If there is accentuated growth, is it most evident during puberty when the lungs reach their peak growth velocities (PGV)?
- 6. At what age are swimmers first reported to have a higher diffusion capacity, and does swim training increase it further?
- 7. What are the main differences between and weaknesses of these studies on competitive swimming and lung development?

Study	Sex	N	Age (y)	Swimming history	Training volume	Volume	Flow	Pressure	Diffusion capacity	Endowment or swim training?	Other conclusions
Newman et al., 1961 (20)	M F	15 S 10 C 15 S 9 C	16.1 (13- 17)	"Leading British swimmers"	-	↑ FVC	$\uparrow FEV_1$	↑ PE _{max}	-	-	Swimmers had greater standing and seated height
Astrand et al., 1963 (21)	F	30 S	12-16	Started training at 10-15 y	6-28 h and 6- 65 km per week	TLC, FRC, FVC > predicted by body size	-	-	-	Swim training	↑ height due to early menarche
		5 S 6 R	19.0	University team	>9 h/week	-	-	-	Similar DLCO during exercise		D _{L,CO} of champion swimmers >
Mostyn et al., 1963 (34)	M&F	8 S 24 O	19.3	Canadian National or Olympic swim team	-	-	-	-	\uparrow absolute and relative $D_{L,CO}$ during exercise	Either	university swimmers Greater D _{L,CO} due to ↑ V _c
Magel and Andersen, 1969 (22)	М	10 S 9 C	$\begin{array}{c} 17.3 \pm 1.4 \\ 17.1 \pm 1.3 \end{array}$	Well-trained	-	↑ TLC, FRC, VC	$\uparrow \mathrm{FEV}_1$	-	↑ D _{L,CO} at rest & exercise ↑ K _{CO} at max exercise	-	-
Ness et al., 1974 (39)	F	20 S 13 C	$\begin{array}{c} 10.2\pm0.7\\ 9.8\pm0.8 \end{array}$	No prior swimming experience	-	TLC, VC, FRC not significantly larger	No difference in FEV ₁	-	$\uparrow D_{L,CO}$ at submaximal exercise	-	Minimal differences in lung function in parents
Vaccaro et al., 1977 (32)	M&F	16 S	10-18	-	Daily, 3000-13,000 yards per day	-	-	-	$D_{i,,\rm CO} > predicted$	Either	-
Eriksson et al., 1978 (23)	М	18 S	10.1 (7.6- 11.8)	Just started swimming	-	TLC, FRC, RV, FVC > predicted by body size	-	-	-	Endowment more likely	-
Vaccaro et al., 1980 (24)	М	12 S	15.1 ± 1.7	≥6 y swim training	4x/week 3600-6400 m per session	TLC, FRC, RV > predicted	FEV ₁ > predicted	-	$D_{L,CO}$ > predicted	Potentially swim training	-
Yost et al., 1981 (33)	M&F	12 S 12 C	$\begin{array}{c} 13.9\pm2.2\\ 14.0\pm2.4 \end{array}$	2-12 y intense swim training	Daily 3-12 km per session	No difference in FVC	-	-	$\uparrow D_{L,CO} \text{ at rest & exercise} \\ and > predicted \\$	Swim training	D _{L,CO} at rest and exercise, FVC ↑ after 10 months in 10 S
McKay et al., 1983 (25)	M&F	25 S	Youngest: 14.1 ± 0.9 Oldest: 18.6 ± 1.3	Scottish National or Youth swim team members	-	FVC 9-25% above predicted	FEV ₁ 19- 25% above predicted	-	-	Either	-
Bloomfield et al., 1984 (26)	M F	53 S 106 C 62 S 123 C	7-12	State Championship finalists	-	↑ FVC	No difference in FEV1	-	-	-	-
Bradley et al.,	М	18 S	20.3 (17- 25)	US Olympic		TLC, FRC,	PEF,		D > predicted	Fither	Greater than other
1985 (31)	F	20 S	18.4 (15- 23)	team	-	predicted	predicted	-	D _{L,C0} > predicted	Liulei	groups

Table 1 – Cross-sectional studies of competitive swim training on lung development.

Study	Sex	Ν	Age (v)	Swimming	Training	Volume	Flow	Pressure	Diffusion	Endowment or	Other
Study	Sen	11	1190 ())	history	volume	volume	110.0	Tressure	capacity	swim training?	conclusions
		7 S 15 C	7.0-8.9	14.7 ± 7.7 mo	5 h per week	↑ TLC, FRC,		No difference in PE_{MAX} or	_		↑ chest wall dimensions, surface area
Zinman and Gaultier, 1986 (27)	F	15 S 17 C	9.0-10.9	$27.2\pm15.0\ mo$	5-12.5 h per week	VC		PI _{MAX} at TLC, FRC, or RV		Both	↑ chest wall dimensions, surface area; ↑ F1max at FRC
		16 S 27 C	11.0-13.3	$44.7\pm19.5\ mo$	12.5 h per week	↑ TLC, FRC, RV, VC	-	↓ PE _{MAX} at FRC, PI _{MAX} at FRC, RV	-		↑ chest wall dimensions, surface area
Pherwani et al., 1989 (40)	M&F	45 S 45 C		6 months to >5 years	6x/week, 2000-5000 m per session	↑ FVC	↑ FEV1, FEF25%	-	-	-	
Cordain et al., 1990 (28)	F	11 S 11 R 10 C	19.0 ± 0.6	Collegiate Division 1 swimmers (9.4 ± 2.8 y start swim)	3000-7000 m per day	↑ TLC, FRC, RV, FVC	$\uparrow {\rm FEV}_1$	No difference in PI _{MAX} , PE _{MAX}	-	Either	-
Armour et al., 1993 (29)	М	8 S 8 R 8 C	$\begin{array}{c} 18 \pm 2.4 \\ 24 \pm 3.2 \\ 22 \pm 4.8 \end{array}$	Start: 11.0 ± 2 y Experience: 6.5 ± 1.9 y	$69.4 \pm 22.1 \text{ km}$ per week	↑ TLC, FRC, VC, RV, FVC	↑ FEV1, FEF50%, PEF	No difference in PI _{MAX} , PE _{MAX}	$\uparrow D_{L,CO},$ no difference in K_{CO}	More likely to be swim training	↑ chest wall dimensions, no difference in F _E max, Fmax, or lung recoil
Doherty and	М	82 S 90 O 66 C	$\begin{array}{c} 15.1 \pm 3.0 \\ 14.1 \pm 2.6 \\ 13.8 \pm 2.7 \end{array}$	Some were	>3v/week	↑ FVC	↑ FEV1,			Fither	Swimmers were
1997 (30)	F	78 S 72 O 70 C	$\begin{array}{c} 14.5 \pm 2.4 \\ 14.4 \pm 2.6 \\ 14.0 \pm 2.5 \end{array}$	swimmers	≥33/ WEEK	FVC	PEF	-	-	Either	older
Lazovic- Popopvic et al., 2016 (41)	М	38 S 271 O 100 C	$\begin{array}{c} 20.9 \pm 2.4 \\ 20.2 \pm 3.6 \\ 21.2 \pm 3.9 \end{array}$	Start: 9.4 ± 2.6 y Experience: 12.8 ± 3.0 y	$\begin{array}{l} 22.0 \pm 7.9 \\ \text{h/week} \end{array}$	↑ FVC and > predicted	↑ FEV ₁ and > predicted, PEF same	-	-	Genetic endowment likely	No relationship between % predicted and starting age, experience, or training volume

Table 1 – Cross-sectional studies of competitive swim training on lung development, continued

Values are expressed as means \pm SD. M, male; F, female; swim, competitive swimmers; con, controls; land, land-based athletes; TLC, total lung capacity; FRC, functional residual capacity; VC, vital capacity; RV, residual volume; FVC, forced vital capacity; FEV_{1.0}, forced expiratory volume in one second; FEF_{50%}, forced expiratory flow at 50% of FVC; PEF, peak expiratory flow; P_Emax, maximal static expiratory pressure at the mouth; P_Imax, maximal static inspiratory pressure at the mouth; D_{LCO}, diffusion capacity of the lungs for carbon monoxide; K_{CO}, transfer factor; F_Imax, maximal inspiratory respiratory muscle force; F_Emax, maximal expiratory respiratory muscle force; V_c, pulmonary capillary blood volume.

Study	Length	Sex	Ν	Starting Age (y)	Swimming history	Training volume	Volume	Flow	Pressure	Diffusion capacity	Endowment or swimming?	Other conclusions
Bachman and Horvath, 1966 (7)	4 mo	М	12 S 9 C	18.8 ± 1.1	Collegiate	-	Swim had ↑ FVC, ↓ FRC, RV, RV/TLC	-	-	-	-	-
Eriksson et al., 1967 (8)	4 y	F	30 S	12-16	See Astrand et al., 1963	Only 4 were still actively training	Pre: see Astrand 1963 Post: still had ↑ FVC	-	-	-	Swimming	-
Gibbins, 1971 (42)	6 mo	F	8 S 6 C	9-10	-	3-4x/week 1000 yards per session	Pre/post: same TLC, FRC, FVC	Pre/post: same FEV1	-	Same D _{L,CO}	Neither	-
Engstrom et al., 1971 (9)	3.6 (1-5) y	F	29 S	9-13	2 (0-5) y training experience	-	Pre/post: ↑ TLC, FRC Swim had ↑ VC during 1-5 v	-	-	-	Both	-
Eriksson et al., 1971 (10)	7-8	F	30 S	12-16	Started training at 10-13 y	Had stopped training for 5 (0-7) y	Pre: see Astrand 1963 Post: still had ↑ FVC	-	-	-	Both	-
Andrew et al., 1972 (11)	3 у	М	71 S 40 C	8-18	-	-	↑ TLC, VC, no difference in FRC; with ↑ height the	↑ maximal mid- expiratory flow	-	↑ DL,co at exercise only in males No	Both	By age 12 the swimmers were taller than
		F	32 S 73 C				greater volumes were more evident			difference in D _{L,CO} /TLC		average
Eriksson et al., 1978 (12)	10 y	F	30 S	12-16	See Astrand et al., 1963	All had stopped training (see Eriksson et al., 1971)	Pre/post: ↑ TLC, VC, FRC	-	-	-	Both	-
Vaccaro and Clarke, 1978 (43)	7 mo	M&F	15 S 15 C	9-11	Just started (in 1 st year of swim training)	4x/week 3000-10,000 yards per session	Pre/post: same FVC No difference in ↑ in FVC	No difference in in FEV1	-	-	Neither	-
Zauner and Benson, 1981	3 v	М	8 S	13.7 (9-19)	Each had competitively	≥6x/week ≥5000 m per	Pre: FVC = predicted	-	-	_	Swimming	-
(13)		F	7 S		swam ≥50% of their life	session	Post: FVC > predicted				6	
Clanton et al., 1987 (14)	12 w	F	8 S 4 C	$\begin{array}{c} 18.9\pm1.2\\ 20.8\pm1.0 \end{array}$	Varsity swimmer for collegiate team	≥5 days per week, 2300- 9000 m per day	Pre/post: ↑ TLC, VC, FRC, RV Swim had ↑ VC, FRC during 12 w	Pre/post: ↑ FEV1, PEF, FIV1, PIF	Pre: no difference Post: ↑ PI _{MAX}	-	Both	↑ Inspiratory muscle endurance

Table 2 – Longitudinal studies of competitive swim training on lung development.

Study	Length	Sex	N	Starting Age (y)	Swimming history	Training volume	Volume	Flow	Pressure	Diffusion capacity	Endowment or swimming?	Other conclusions
Zinman and			7 S	7-10	See Zinman	See Zinman	Pre/post: ↑ TLC, VC		$\uparrow PE_{MAX}$			
Gaultier, 1987 (15)	1 y	F	10 S 10-1	10-12	and Gaultier, 1986	and Gaultier, 1986	Swim had ↑ TLC, VC during 1 y	-	No change in PI _{MAX} or PE _{MAX}	-	Both	-
Miller et al., 1989 (35)	5 mo	М	22 S	18-22 y	Collegiate swim team	-	Pre/post: similar VC	-	-	Pre/post: similar D _{L,CO} , > predicted	Either	Performance and lung function were independent
Bloomfield et al	_	М	38 S 57 C		Group selected		↑ FVC from stage 2	↑ FEV ₁ from stage 3				↑ chest depth and
1990 (16)	5 y	F	57 S 64 C	8-12	finalists	5x/week	↑ FVC from stage 4	↑ FEV1 from stage 4	-	-	Swimming	girth
Baxter-Jones and Helms, 1996 (17)	3 у	M&F	114 S 339 O	8-16	-	9-13 h per week	↑ FVC at start compared to others, no further ↑ after	-	-	-	Endowment	Swimmers were taller after adjusting for age and pubertal status
Courteix et al., 1997 (18)	1 y	F	5 S 11 C	$\begin{array}{c} 9.3\pm0.5\\ 9.4\pm0.5\end{array}$	-	8-12 h per week 10-20 km per week	Pre: no difference Post: ↑ TLC, VC, FRC	Pre: no difference Post: ↑ FEV ₁ , PEF, FEF _{25%} , FEF _{50%} , FEF _{75%}	-	-	Swimming	No difference in R_{aw} ; not- significantly taller
Mickleborough et al., 2008 (19)	12 w	M&F	10 S	18.2 ± 1.6	National and international swimmers	10-12x/week 40-60 km per week	↑ TLC, FVC, no difference RV	\uparrow FEV ₁ , FIV ₁	↑ PI _{MAX} , PE _{MAX}	↑ D _{L,CO} , no change K,co		
This thesis	7 mo	F	11 S 10 C	$\begin{array}{c} 12.4\pm0.8\\ 13.2\pm1.3\end{array}$	3.2 ± 1.8 y	9.1 \pm 3.6 h per week 19 \pm 8 km per week	Pre/Post: ↑ TLC, FVC, VC	Pre/Post: ↑ PEF, FEV1, FEF	Pre/Post: ↑ PI _{MAX} (p=0.06), PE _{MAX}	Pre/Post: ↑ D _{L,CO} , similar K _{,CO}	Endowment	Similar ventilatory constraints during cycling

Table 2 – Longitudinal studies of competitive swim training on lung development, continued

Values are expressed as means \pm SD. M, male; F, female; swim, competitive swimmers; con, controls; TLC, total lung capacity; FRC, functional residual capacity; VC, vital capacity; RV, residual volume; FVC, forced vital capacity; FEV_{1.0}, forced expiratory volume in one second; PEF, peak expiratory flow; FIV, forced inspiratory volume in one second; PIF, peak inspiratory flow; FEF_{25%} FEF_{50%}, and FEF_{75%}, forced expiratory flow at 25%, 50%, and 75% of FVC, respectively; PE_{MAX}, maximal static expiratory pressure at the mouth; D_{LCO}, diffusion capacity of the lungs for carbon monoxide; K_{CO}, transfer factor; R_{aw}, airway resistance; IMT, inspiratory muscle training.

1.1.1 Swedish "girl swimmers" – the foundational studies on swimming and lung development

The foundational research into physiological adaptations to competitive swimming during development came from analyses of three different cohorts of Swedish swimmers (summarized elsewhere (44)). The original was a longitudinal analysis of 30 "girl swimmers", examined first at 12-16 y old by Astrand et al. (21) and again after 2, 4 (8), 7 (10), and 10 y (12). Initially, the girls had significantly larger lung volumes (i.e., total lung capacity (TLC), forced vital capacity (FVC), and FRC) that were 11-13% greater than the average value for their height (21). These larger lung volumes for a given height were maintained throughout the follow-up period, even upon cessation of swimming (12). At the first measurement, significant correlations between lung volume (expressed as the %-deviation in TLC or vital capacity (VC) from the average value for height) and training volume (expressed either as training experience or training volume in metres or hours per week) suggested that the intensity of swim training influenced functional development of the respiratory system (21). Therefore, different training regimens were compared by separating the swimmers into two groups such that 9 top swimmers from one club who trained the most (up to 65,000 m and 28 h per week) were compared to the other 21 swimmers (6,000-30,000 m and 6-20 hours per week). While the top swimmers initially had a larger FVC but statistically similar TLC, FRC, and residual volume (RV), differences between the groups did not change throughout the follow-up period (12). This suggested that intense training did not further increase lung volumes in the top swimmers; however, the question of whether the larger lungs of all swimmers at the initial measurement were due to intense training or genetic endowment was unresolved.

A subsequent longitudinal analysis conducted with 29 9-13 y old Swedish girl swimmers confirmed some of these findings (9). At the initial measurement, TLC and FRC in relation to

height were already significantly larger than normal whereas FVC was not. By the final measurement 3.6 y (range 1-5 y) later, FVC in relation to height had increased significantly while TLC and FRC remained constant. Interestingly, when the girls were stratified into two groups based on training experience before the first measurement, the 18 girls who had been training at least 3 times per week for \geq 1 year had significantly increased lung volumes (TLC, FRC, and FVC in relation to height) whereas those who had trained for \leq 1 year did not. Moreover, when lung volumes were analyzed from 12 to 14 y old, the girls that trained during this period had a significant increase in FVC in relation to both height and TLC, while TLC grew as expected. From these observations, the authors concluded that lung volumes in swimmers are larger at the start of training, and further increases in FVC but not TLC with continued training point to functional rather than anatomical growth. However, no explanation was provided for why the swimmers with more experience had greater lung volumes.

The third study by Eriksson *et al.* assessed 18 boys aged 10.1 y (range 7.6-11.8 y) who had minimal training experience (less than a few months) but had just been selected to competitively swim with the top clubs in Sweden (23). Despite not yet having started intensive swim training, their lung volumes (TLC, FRC, RV, and FVC) already exceeded normal values in relation to height, strongly suggesting that the initially larger lungs of competitive swimmers were due to genetic endowment. A similar result was found in a study comparing untrained girls trying out for a competitive swimming team (39). The 11 girls who qualified for the team had an average TLC of 3.08 ± 0.73 l compared to 2.52 ± 0.39 l in the 9 girls of similar age, maturity, and body size who did not. Furthermore, the fathers of the girls who qualified had a greater TLC and FRC than the fathers of those who did not, strongly pointing to a genetic endowment whereby swimmers with constitutionally larger lungs may select into swimming.

1.1.2 Lung volumes in young swimmers

Other studies have also reported differences in young swimmers. In a 3-year longitudinal analysis of over 70 8-18 y old competitive swimmers (compared to 83 controls analyzed cross-sectionally), Andrew *et al.* found greater lung volumes (TLC and VC) which were apparent even in the youngest swimmers (11). Zinman and Gaultier cross-sectionally assessed 38 7-13 y old trained female swimmers and 59 age- and size-matched controls, and found significantly greater lung volumes (TLC, VC, and FRC) in all ages of swimmers (27). In a cross-sectional analysis of 112 7-12 y old trained swimmers compared to tennis players and non-athletes, Bloomfield *et al.* found a significantly greater FVC that was apparent across all ages (26). Lastly, a large, longitudinal study of 453 8-16 y old young athletes (swimmers, gymnasts, tennis players, and soccer players) found that FVC (adjusted for height, weight, and maturation) was \geq 20% larger in swimmers compared to the other athletes at the initial measurement (17). Cumulatively, these results suggest that swimmers as young as 7-8 y old already have greater lung volumes

However, this has not been the case for all studies. As mentioned, Engstrom *et al.* did not find any initial differences in TLC or FVC in the 9-13 y old swimmers who had less than 1 year of training (9). Similarly, Vaccaro and Clarke compared 15 9-11 y old children in their first year of swim training (3,000-10,000 yards per training session) with 15 controls, but reported no differences in FVC (43). Gibbins and Courteix *et al.* did not find any differences in TLC, FRC, FVC, or VC between 8 9-10 y old female swimmers and 6 controls (42) and 5 9-10 y old prepubertal competitive swimmers and 11 age-, sex-, and size-matched controls (18), respectively. Unfortunately, swimming history was not reported in either of these studies. Zauner and Benson

measured FVC in extensively trained 9-19 y old swimmers and found no initial difference compared to predicted values (13). There is no clear reason for these opposing results.

1.1.3 Longitudinal assessments of competitive swimming and lung volume development

Longitudinal assessments of lung function in growing swimmers are also contradicting, as four different conclusions have been reported. First, there was no difference in lung function before or after a 6-7 month period of training in 8 9-10 y (42) and 15 9-11 y (43) old swimmers compared to control groups. Second, despite similar lung capacities initially, greater lung capacities were measured in 5 9-10 y old swimmers compared to 11 matched controls after 1 y of training (18), 95 8-12 y old trained swimmers compared to 102 maturation-matched controls over 5 y of assessments (16), and 15 9-19 y old very competitive swimmers compared to predicted values after 3 y of training (13). Third, swimmers initially had larger lungs and these did not increase further over 3 y of training in 114 8-16 y old swimmers compared to other athletes (17), 3 y of training in >70 8-18 y old swimmers compared to non-athletes (11), and 1-5 y of training in 29 female swimmers aged 9-13 y compared to population norms (9). Finally, trained swimmers aged 7-12 y initially had larger lungs and these further increased after 1 y of training as compared to a control group analyzed cross-sectionally (15). Unfortunately, no study has assessed lung function in competitive swimmers before 7 y (this may be related to some measurements requiring maximal maneuvers that are not reliable until 8 y (37, 45-47)).

Moreover, only 4 of the aforementioned studies have provided analysis during puberty and the conclusions are conflicting. Engstrom *et al.* found that VC, but not TLC, grew more than expected between 12 and 14 y old (9), whereas Zinman and Gaultier reported accentuated growth of both TLC and VC in 10-12 y old trained swimmers (15). While Bloomfield *et al.* found that

FVC was only statistically significantly larger from stage 2 (puberty) onwards in males and stage 4 (puberty) onwards in females (16), Baxter-Jones and Helms reported that FVC was initially larger and did not increase further during puberty (17). Clearly, more work is needed to determine if competitive swimming affects the development of lung volumes during growth.

1.1.4 Competitive swimming and diffusion capacity during growth

Differences in diffusion capacity may highlight structural or functional changes in the gas exchanging ability of the lungs. Like lung volumes, greater diffusion capacities have been observed in swimmers. One study suggested the greater diffusion capacity for carbon monoxide $(D_{L,CO})$ was partly due to an adaptation to swim training (33). Yost *et al.* observed a greater absolute D_{LCO} at rest and during submaximal exercise at 170 bpm in 12 9-17 y old competitive swimmers (2-12 y experience, 3-12 km per session) compared to 12 matched controls (33). They re-tested 10 of the swimmers after 10 months of training and found that exercise D_{LCO} increased more than expected by growth, leading them to suggest that swim training increased D_{L,CO} to a greater extent than expected by growth. However, resting D_{L,CO} increased only slightly (and as expected given their somatic growth), and the greater exercise D_{LCO} could be explained by the swimmers exercising at a greater metabolic rate (oxygen consumption (VO₂) at 170 bpm was significantly increased) at the second examination. Moreover, when resting $D_{L,CO}$ was correlated with height, the slope between $D_{L,CO}$ and height was identical for swimmers and controls, suggesting that D_{L,CO} was equally greater across all heights studied. This could have been related to greater lung volumes in the swimmers (FVC 4.12±0.93 vs. 3.61±0.86 l), although differences in FVC did not reach statistical significance. Such a difference was found in the longitudinal analysis by Andrew et al., where the greater exercise D_{L,CO} across all heights in 8-18 y old male

swimmers compared to non-athletic male controls (no differences were found between the female cohorts) was no longer apparent when expressed relative to TLC (11). Greater absolute $D_{L,CO}$ but similar relative $D_{L,CO}$ have also been reported in older adolescent (22) and young adult (29) elite swimmers. Thus, the greater diffusion capacity of swimmers is apparent across all heights and ages and is related to their larger lung volumes, yet no conclusive evidence has shown that competitive swim training accentuates the development of $D_{L,CO}$ during development.

1.1.5 Differences between and weaknesses of previous studies

While there is a myriad of literature on competitive swimming and lung development, heterogeneities between studies have made it difficult to systemically analyze and resolve the question of "genetic endowment, training adaptation, or both?". These include differences in participant age; swimming history and level of competition; training status; design and length of study; lung function measurements; comparisons to controls, predictive values, or population norms; and statistical analysis. Moreover, as outlined in Table 3, previous studies have been weakened by short study periods (\leq 7 months (43)), not differentiating boys and girls, not assessing or matching for maturational stage, small sample size (<10 subjects in group), no control group, not statistically comparing swimmers with the control group or reference values, or not performing a comprehensive assessment of lung function (e.g., only FVC and forced expiratory volume in one second (FEV₁), but no lung volumes).
	Short study period (≤7 months) *	Did not separate sexes	Did not match for maturational stage	Small sample size (<10 subjects in group)	No control group	No statistical comparison to controls or reference values	Few measures of lung function (e.g., only FVC, FEV ₁)	Other notes
Gibbins, 1971 (42)	Х			Х				Swim history not stated, training stimulus low
Andrew <i>et al.</i> , 1972 (11)						Х		Controls assessed cross- sectionally but swimmers longitudinally
Ness et al., 1974 (39)				Х				<i>.</i> ,
Vaccaro et al., 1977 (32)		Х			Х		Х	
Vaccaro and Clarke, 1978 (43)	х	Х	Х				Х	Did not state if groups were sex-matched
Vaccaro <i>et al.</i> , 1980 (24)					Х			
Yost et al., 1981 (33)		Х	Х				Х	
Zauner and Benson, 1981 (13)		Х			Х		Х	
Bloomfield et al., 1984 (26)		**	Х				Х	
Zinman and Gaultier, 1986 (27)			Х					
Zinman and Gaultier, 1987 (15)			Х			Х		Controls assessed cross- sectionally but swimmers longitudinally
Bloomfield et al., 1990 (16)							Х	Mixed-longitudinal analysis
Baxter-Jones and							Х	
Helms, 1996 (17) Courteix <i>et al.</i> , 1997				Х				Unusually small lung
(18) Doherty and								growth in control group
Dimitriou, 1997 (30)			Х				Х	

Table 3 – Weaknesses of selected previous studies on competitive swimming during development

*Only for longitudinal studies. **Did not find significant effect of sex, therefore dropped from further statistical analysis. FVC, forced vital capacity; FEV₁, forced expiratory volume in one second.

Vaccaro and Clarke reported similar lung function in 15 9-11 y old swimmers (training 3,000-10,000 yards per session, 3-4 times per week) and 15 age- and size-matched controls before and after a 7-month season of competitive swimming, leading them to suggest that study durations less than 7 months are too short to measure significant differences in lung development (43). The short time period may also underlie the lack of differences in the 6-month swim training study by Gibbins (42). To note, the very low training stimulus (1000 yards per session, 3-4 sessions per week) may have contributed to the negative finding.

Vaccaro and Clarke did not specify if the groups were matched for sex. A variety of sexbased differences in lung growth (48, 49), which lead to differential timing and rates of growth of alveoli and small and large airways between boys and girls, may have caused no effect of competitive swimming on lung development to be observed in this study (43) and affected the results of others (13, 33).

While Zinman and Gaultier found significantly greater (27) and accentuated growth (15) of lung volumes in 7-13 y old swimmers compared to similarly-aged controls, it is not clear if they were matched for maturational stage. Male swimmers tend to be early maturers (50, 51); conversely, female swimmers tend to have a slightly intrinsically later (not delayed) menarchal age (13.3-13.4 y (52, 53) compared to the reference 13.0 y (52)) with the best performers having the latest menarchal ages (54). Considering that, first, females who are late maturers might also have a prolonged pubertal growth spurt (55), second, the growth velocity of the lungs differs depending on pubertal stage (56), and, third, a mixed-longitudinal study of swimmers and non-athletes showed different amounts of growth in FVC depending on the maturational stage (16), there is clear need for maturational matching when comparing swimmers with their healthy counterparts. Therefore, differences in maturational stage may have contributed to the larger lungs

of the swimmers in the studies by Zinman and Gaultier and affected the results of others (26, 30, 33, 43).

In the longitudinal analysis by Courteix *et al.*, they found similar lung volumes initially but significantly greater volumes after 1 y of intense swim training in 5 9-10 prepubescent swimmers and maturity- and age-matched controls (18). However, the control group grew an average of 5 cm in height but only 90 ml in TLC, which appears abnormally small compared to reference values for their age and somatic growth (57). Therefore, the difference may have been due to the control group's minimal increase in lung volumes. Moreover, the authors cited the need for a larger group of swimmers.

While comparisons to predicted values provide an idea of lung function relative to population standards, they require predictive equations. These depend on the design (cross-sectional versus longitudinal) of the reference study, size of the reference population, and quality of the statistical modelling. Moreover, selecting the appropriate predictive equations requires demographic similarities between the study sample and reference population as well as methodological similarities between the study and reference measurements. Thus, conclusions from studies lacking a control group (13, 24, 32) must be interpreted with caution.

Longitudinal assessments of swimmers by Andrew *et al.* (11) and Zinman and Gaultier (15) were compared to control groups who were cross-sectionally analyzed using regression lines (with a 95% confidence interval). Although plots of longitudinal changes against these regression lines provided graphical illustration of changes in lung function, neither study statistically analyzed if the changes in swimmers' lung function reached statistical significance.

Lastly, many studies have used only FVC as an indicator of lung size and FEV₁ of airway function (Table 3). More comprehensive analysis of lung volumes (i.e., measuring TLC, FRC, and

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RV), flows (i.e., measuring expiratory flow rates and analyzing maximum expiratory flow-volume curves (MEFV)), diffusion capacity, and static pressures are necessary to draw thorough conclusions about the effect of competitive swimming on lung development. Specifically, assessing changes in TLC and $D_{L,CO}$ may elucidate irreversible anatomical adaptations, whereas FVC may only be indicative of functional changes (9).

1.1.6 Summary

Thus, the following key points can be concluded from the current literature:

- While two small studies have observed no differences in TLC in 9-10 y old (18) or FVC in 9-11 y old (43) swimmers, greater lung volumes have been observed in large cohorts of swimmers as young as 7-8 y (11, 17, 23, 26, 27). This suggests even the youngest swimmers already have enhanced lung function.
- Whether this difference is due to genetic endowment or an adaptation to swim training at an early age is not clear, as lung function has not been measured in swimmers prior to age 7 y.
- Reports of lung volumes in swimmers at the beginning of training are conflicting, as greater lung capacities have been reported in 7-12 y (23) but not in 9-11 y (43) or 9-13 y (9) old children in their first year of swim training.
- Longitudinal analyses has provided four different conclusions regarding changes in lung capacities with swim training: first, no differences before or after (42, 43); second, no differences before but greater capacities after (13, 16, 18); third, greater capacities

before that did not increase further (9, 11, 17); and, fourth, greater capacities before that further increased (15).

- Studies longitudinally assessing lung volumes in swimmers during puberty have found conflicting results. Some have found FVC (9, 16) and TLC (15) increased more than can be expected due to maturational growth alone, while others have not (FVC (17), TLC (9)).
- A greater diffusion capacity has been observed across all ages and heights of swimmers (11, 33), which is likely related to their larger lung volumes. There is no conclusive evidence that competitive swimming accentuates increases in D_{L,CO}.
- Differences between and weaknesses of previous studies underlie the difficulty in determining whether differences are due to genetic endowment, a training adaptation, or both.

There is <u>need for a longitudinal study that comprehensively assesses lung function (i.e.,</u> <u>lung volumes, spirometry, diffusion capacities, and pressures) in pubertal competitive swimmers</u> compared to healthy controls of similar age, size, and sexual maturity to further our understanding of pulmonary adaptations to competitive swimming. Moreover, <u>whether competitive swimming</u> <u>affects ventilatory mechanics during exercise has not been studied</u> and warrants investigation.

1.2 Ventilatory mechanics during growth

Smaller lungs and airways, such as those found in adult women in comparison to adult men, can lead to more constrained ventilatory mechanics during exercise and subsequently an augmented oxygen cost of breathing (58), increased likelihood of experiencing exercise-induced arterial hypoxemia (EIAH) (59), and, ultimately, impaired exercise performance (4). Moreover, higher levels of aerobic fitness and therefore an increased ventilatory demand may also lead to the development of expiratory flow limitation (EFL) at maximal exercise (60). Because children have similar lung structures and hyperventilatory responses to exercise as do adult women, it is possible that they are predisposed to the same ventilatory constraints during exercise (61). However, while much work has been done in adults, only a handful of reports have studied ventilatory mechanics in the healthy pediatric population (61-65).

Ventilatory mechanics can be assessed quantitatively using (1) the degree of EFL, (2) breathing strategy (i.e., regulation of operational lung volumes), and (3) the utilization of ventilatory capacity (\dot{V}_E/\dot{V}_{ECAP}) (66) and qualitatively by superimposing tidal flow-volume loops (FVL) on a graph of the maximum expiratory flow-volume curve (MEFV). Expiratory flow limitation occurs when expiratory flow does not change despite increases in transpulmonary pressure (67). In other words, for a given volume no greater expiratory flow can be generated, thus a shift towards higher operating lung volumes is necessitated to increase flow further. This, however, comes at a cost because operating volumes dictate muscle length (i.e., length-tension relationship) and the work required to overcome the elastic properties of the lung (i.e., pressure-volume relationship). Estimating the ventilatory capacity (\dot{V}_E/\dot{V}_{ECAP}) provides another useful tool, as one can determine the ventilatory demand imposed by the given breathing strategy (66).

The prevalence of EFL in prepubescent children is high, ranging from 56% (63) to 93% (61) at maximal exercise, and both boys and girls are equally susceptible. However, the reason for this high prevalence is unclear. Nourry *et al.* compared 10 flow-limited and 8 non-flow-limited prepubescent children, finding that the two groups had different breathing strategies as exercise progressed despite similar resting pulmonary function (63). In the non-flow-limited group, the

decrease in operational lung volumes upon starting exercise was followed by a leftward shift towards greater operating lung volumes as intensity increased, enabling them to breathe at higher expiratory flows that prevented the onset of EFL. Conversely, the flow-limited group initially decreased and then maintained their end-expiratory lung volume (EELV), operating at lower lung volumes with smaller expiratory flows that made them susceptible to EFL. The leftward shift meant that the non-flow limited group was able to reach higher peak values for minute ventilation (\dot{V}_E) and $\dot{V}O_2$ and utilize a greater percentage (>90%) of their estimated maximum voluntary ventilation (MVV). The study by Swain et al. observed the opposite operating lung volume response (61). They studied 20 prepubescent boys and 20 prepubescent girls, finding a significant correlation between the increase in expiratory reserve volume (ERV) relative to FVC (ERV/FVC) from rest (i.e., the amount of dynamic hyperinflation) and the extent of EFL. In other words, they observed that EFL was associated higher operating lung volumes, a finding consistent with healthy adult populations (61). Given that no relationships were found between the severity of EFL and TLC, peak \dot{V}_E , or peak $\dot{V}O_2$, the authors suggested that prepubescent girls and boys experience similar rates of EFL but for different reasons. Prepubescent girls were limited by their capacity, as their smaller lung volumes and expiratory flows led to a smaller MEFV that then provided the main ventilatory constraint to exercise. On the other hand, prepubescent boys were constrained due to their increased demand. They had a bigger MEFV because of their larger lung volumes and expiratory flows, but it was still encroached upon because they had a higher metabolic demand (as evidenced by a higher peak $\dot{V}O_2$ than girls) (61). The boys (65.5 ± 6.1%) and girls (64.4 ± 5.7%) utilized a similar percentage of estimated MVV at peak exercise, values that may be lower than the previous study because they had a lower fitness level.

A follow-up study done five years later in 21 (11 boys, 10 girls) of the 40 prepubescent children provides the only observation of ventilatory mechanics in postpubescent children (62). The prevalence of EFL was 45% and 20% in boys and girls, respectively, much lower than prepubescent rates despite both the postpubescent boys and girls operating at higher lung volumes at maximal exercise. The authors suggested this was possible because greater growth of lung volumes and expiratory flows compared to improvements in exercise capacity increased the ventilatory capacity well beyond the metabolic demand. Thus, the postpubescent children could operate at higher lung volumes as a strategy to avoid EFL (62). Moreover, maturation may have lowered the sensitivity to CO_2 during exercise (the authors noted a decreased ventilatory equivalent for carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) at maximal exercise in postpubescence compared to prepubescence) which relatively decreased the ventilatory demand, further decreasing the likelihood of EFL. Unfortunately, no estimate of ventilatory capacity was provided postpubescence.

1.2.1 The effect of training on ventilatory constraints during growth

How training affects ventilatory mechanics in children has only been assessed in one crosssectional study. Comparing trained and untrained prepubescent children, larger lungs and therefore a greater MEFV in the trained group was associated with the leftward shift in operational lung volumes observed at maximal exercise (65), similar to the aforementioned difference from pre- to post-puberty. However, the prevalence of EFL was similar between the two groups (69 and 73% in trained and untrained, respectively) and the trained subjects utilized a higher proportion of their MVV. The authors suggested that the equal prevalence of EFL was due to trained subjects having a substantially greater ventilatory drive occupying more of the larger MEFV, while the untrained subjects simply had a smaller MEFV. There was a cost associated with this greater ventilatory drive, as the trained subjects operated at a higher EELV which may have negatively impacted dyspnea and arterial saturation at maximal exercise. More research is needed on this topic, specifically comparing trained and untrained children with similar resting pulmonary structure and function.

1.2.2 The effect of ventilatory constraints on EIAH during growth

Whether these ventilatory constraints ultimately lead to EIAH is unknown. Arterial desaturation during exercise in adults is the result of relative alveolar hypoventilation, increased ventilation-perfusion mismatching, and an alveolar-to-capillary diffusion limitation (64). The small and more mechanically constrained lungs of adult women can directly or indirectly lead to an increased susceptibility to EIAH by preventing an adequate alveolar hyperventilatory response (59). In prepubescent children, EIAH measured noninvasively with pulse oximetry at the ear was reported in approximately 30% of active children at maximal exercise and associated with smaller lungs (as measured by FVC) and greater ventilatory constraints (as assessed using breathing reserve) (64). Conversely, two other studies of prepubescent girls (68) and both boys and girls (61) found that EIAH did not occur in any subjects during maximal exercise, suggesting that the ventilatory constraints experienced were not of sufficient magnitude to cause arterial desaturation. Moreover, in a follow-up of the latter study, no postpubescent boys and girls desaturated during maximal exercise (62). More work is needed to clarify the prevalence of EIAH and its significance during growth.

Since ventilatory capacity is primarily determined by anatomical features (i.e., lung size, airway size and geometry) (69), the larger lung volumes and expiratory flows of swimmers may be advantageous during exercise if it leads to a larger ventilatory capacity that makes them less

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susceptible to ventilatory constraints during exercise. They may be able to generate higher flows at similar lung volumes, decreasing their susceptibility to EFL and allowing them to operate at lower relative lung volumes that may lower their work of breathing (WOB). Alternatively, the increased capacity could facilitate an increased metabolic and ventilatory demand within similar ventilatory constraints. Exploratory work is needed in swimmers to determine if they are afforded any benefits from their larger lungs.

1.3 Significance

As mentioned, increased lung volumes and diffusion capacity may facilitate improved arterial oxygen saturation, increase ventilatory capacity, and provide greater buoyancy in the water. Cumulatively, these may lead to greater performance in meets and success in swimming. There is also benefit for the general population from studying the effect of swim training on lung development. Adolescence is a critical period during which physiological changes can significantly influence health throughout the lifespan (70). Years of competitive swim training during this period may lead to improvements in lung function that can persist into adulthood (12). However, increased exposure to chlorine derivatives while swimming may increase the likelihood of developing reactive airway disease later into a swimmer's career (71). Therefore, studying changes in lung development will give us a greater understanding of the potential benefits and harms of competitive swimming during youth.

1.4 Conclusion

Typically, the lungs do not beneficially adapt to physical activity. However, competitive swimmers consistently display exceptional pulmonary function. Whether this is an inherited or

acquired trait has been widely debated. Previous cross-sectional findings suggest that swimmers as young as 7 y old already have enhanced function, but swimmers before this age have not been examined. Longitudinal analyses of competitive swimmers during growth present contradicting results, and a limited number of studies have focused on changes during puberty. Moreover, a myriad of differences between and weaknesses of previous studies make it difficult to draw definitive conclusions regarding the relationship between competitive swimming and lung development. Additionally, whether this enhanced function improves ventilatory capacity and/or alters ventilatory mechanics during exercise has not been assessed. Given that the PGV for FVC (57, 72) and lung and chest wall dimensions (73) occur between 11-14 y old or Tanner stages 2-4 (16) in girls, a comprehensive and longitudinal assessment of lung function during this period of rapid growth is needed to provide further answers to the question: "genetic endowment, training adaptation, or both?".

1.5 Purposes

This thesis was designed to address the following questions:

- 1. Does competitive swim training during puberty affect lung development in healthy, pubescent girls?
- 2. Does competitive swim training during puberty affect ventilatory mechanics during exercise in healthy, pubescent girls?

Thus, the primary purpose of this longitudinal study was <u>to determine if one season of</u> <u>competitive swimming during puberty affects lung development in female competitive swimmers</u> as compared to healthy controls of similar age, sex, size, and maturity. The secondary purpose was <u>to characterize their ventilatory mechanics during cycling exercise.</u>

1.6 Hypotheses

This thesis hypothesized that:

- 1. Swimmers will have greater increases in pulmonary function measurements.
- 2. Swimmers will have less ventilatory constraints due to their larger lungs.

Specifically, it was hypothesized that swimmers would have a greater increase in TLC as compared to a healthy control group. Moreover, because ventilatory capacity is primarily determined by anatomical features (i.e., lung size) (69), it was further hypothesized that the larger lungs of swimmers would be associated with an increased \dot{V}_{ECAP} and make them less susceptible to ventilatory constraints (i.e., higher operational lung volumes and decreased prevalence and/or severity of EFL) during exercise.

METHODS

2.1 Subjects

Twenty-four healthy females (12 SWIM and 12 CON) aged 11-14 y old were recruited to participate in the study. All of the subjects had no history of smoking, no previous exposure to hypoxia (i.e., altitude) for a period greater than 6 weeks, no pre-existing reactive airway disease (e.g., asthma), and no previous use of an inhaler. The swimming group (SWIM) consisted of 12 competitive swimmers from 7 Greater Vancouver swim clubs, where each swimmer was required to maintain a vigorous training volume as instructed by her coach. Ten swimmers competed in regional or provincial meets, including one who competed at the national level. An eleventh swimmer trained predominantly for water polo during the study period, but was still included because her training involved weekly speed swimming sessions and she competitively swam during the summer season. A twelfth subject in the SWIM group was excluded from analysis due to a non-cardiorespiratory illness that interrupted her swim training. Twelve controls (CON) were recruited from family and friends of the hospital and university staff and the University of British Columbia's recreational activities programs. They primarily participated in gymnastics, dance, and team sports, but none of them competed in any sports or activities that required sport-specific endurance training. Two controls declined to return for the follow-up visit. Thus, 11 swimmers and 10 controls completed the entire experimental protocol and were included in the analysis.

2.2 Experimental overview

Testing was performed in the Respiratory Clinic (RC) and Exercise Physiology Laboratory in the Children's Heart Centre (EPL) at BC Children's Hospital. Each subject completed two identical visits; the first at the beginning of the swim season between September and November (PRE), and the second at the end of the swim season in May or June (POST). Subjects were accompanied by a parent or guardian at all visits. All measurements and procedures were approved by the University of British Columbia's Children's and Women's Clinical Research Ethics Board (approval certificate number: H15-00977), which conforms to the *Declaration of Helsinki*.

After being greeted outside the RC, subjects completed an informed assent (11-13 y old) or adolescent assent (14 y old) form and the parent or guardian provided medical history and informed consent. Sexual maturity rating (SMR) was assessed at all visits using a validated form (74) containing sex-specific illustrations and written descriptions of pubic hair and breast development (Appendix C) corresponding to Tanner's five pubertal stages (75). Each subject had the option of self-reporting her SMR or being evaluated by her parent or guardian. Subjects were then taken into the RC where a respiratory therapist (RT), experienced in working with pediatric patients, measured their height (seca 217, seca GmbH & Co. KG., Hamburg, Germany), weight (Scale-Tronix, White Plains, NY, USA), and hemoglobin (Hb) (Pronto-7, Masimo Corp., Irvine, CA, USA). A pulmonary function test (PFT), consisting of spirometry, lung volumes, and diffusion capacity (MasterScreen[™] PFT system, Jaeger, CareFusion Corp., San Diego, CA, USA), was completed with the subject sitting and wearing nose clips. Subjects were then taken into the EPL where maximal static pressure maneuvers (Mouth Pressure Meter, Micro Direct, Inc., Lewiston, ME, USA) and a graded maximal exercise test on a cycle ergometer (Excalibur Sport, Lode BV, Groningen, Netherlands) were performed. Before the exercise test, five minutes of seated resting metabolic data were obtained, followed by multiple FVC and graded forced vital capacity (gFVC) maneuvers. Forced vital capacity and gFVC maneuvers were also performed after the exercise test. After the second visit, subjects filled out a modified version of a validated

physical activity questionnaire (PAQ) (76-78) and coaches reported the training load during the study period for the SWIM group (Appendix C).

2.3 Measurements and procedures

2.3.1 Spirometry

With the RT, subjects performed FVC maneuvers according to ATS/ERS guidelines (79) to determine FVC, FEV₁, FEV₁/FVC, peak expiratory flow (PEF), maximum mid-expiratory flow (FEF_{25-75%}), and forced expiratory flows (FEF) when 25%, 50%, and 75% of the FVC had been expired (FEF_{25%}, FEF_{50%}, and FEF_{75%}, respectively).

2.3.2 Single-breath carbon monoxide diffusion and helium dilution technique

Using the single-breath technique, FRC, inspiratory capacity (IC), and ERV were measured by helium dilution and $D_{L,CO}$ and alveolar volume (V_A) by carbon monoxide (CO) diffusion according to ATS/ERS guidelines (80, 81). Total lung capacity, RV, and VC were calculated as follows: TLC = FRC + IC, RV = FRC – ERV, and VC = TLC – RV. The transfer coefficient for carbon monoxide ($D_{L,CO}/V_A$), a measurement of diffusion capacity standardized to alveolar volume, was determined by dividing $D_{L,CO}$ by V_A. Because $D_{L,CO}$ depends on the concentration of Hb in the blood, measurements were corrected for Hb ($D_{L,COC}$) using the equation: $D_{L,CO}c = \frac{D_{L,CO} \times 1.7}{9.38+Hb}$ (81).

2.3.3 Maximal static pressures

Maximum inspiratory pressure (PI_{MAX}) from RV and maximum expiratory pressure (PE_{MAX}) from TLC were measured at the mouth using a handheld device according to ATS/ERS

guidelines (82). Maneuvers were performed in the sitting position a minimum of 3 and a maximum of 9 times, with at least one minute of rest in between.

2.3.4 Resting baseline

Subjects then sat on the cycle ergometer for five minutes and breathed quietly through a low-resistance, two-way non-rebreathing valve (model 2700B, Hans Rudolph, Kansas City, MO, USA). The valve was connected by large bore tubing to independently calibrated pneumotachographs (model 3813, Hans Rudolph, Kansas City, MO, USA) to separately measure inspiratory and expiratory flow. Expired gas was collected in a mixing chamber from which independent sampling lines were drawn through *Nafion* tubing and a de-humidification chamber containing *Drierite* and into calibrated O₂ and CO₂ analyzers (#17625 Fast Response O₂ Analyzer and #17630 Silver Edition CO₂ Analyzer, respectively, VacuMed, Ventura, CA, USA). Heart rate (HR) was measured using a HR monitor (T34, Polar Electro, Kempele, Finland) worn around the chest. At the end of the five-minute period, subjects were instructed to perform several IC maneuvers using the instructions, "at the end of a normal breath out, take a maximal breath all the way in until you fill up your lungs, then return to normal breathing."

2.3.5 Forced vital capacity and graded forced vital capacity maneuvers

While still seated on the cycle ergometer, subjects performed multiple FVC and gFVC maneuvers to construct the MEFV. The FVC maneuvers were performed according to ATS/ERS guidelines (79), while the gFVC maneuvers were performed with extensive coaching to ensure the subjects inspired maximally but expired maximal volumes at sub-maximal efforts (83). Both maneuvers were repeated after the maximal exercise test, as gFVC maneuvers minimize thoracic

gas compression and post-exercise maneuvers may reflect exercise-induced bronchodilation, together leading to better representation of the MEFV (83).

2.3.6 Graded maximal exercise test

After warming-up on the cycle ergometer for three minutes at 20 W, intensity was increased stepwise by 20 W every two minutes until exhaustion to ensure a test duration of less than 20 minutes. Subjects were instructed to maintain a pedalling frequency of 60 revolutions per minute (rpm) throughout the test, which was terminated when the subject could no longer maintain 50 rpm for at least five seconds despite strong verbal encouragement from the researchers. During each stage, subjects were asked to perform two IC maneuvers; the first around one minute and 10 seconds (1:10) into the stage and the second approximately 10 seconds before the end of each stage (1:50). Following the first IC maneuver, subjects were asked to provide their rating of perceived exertion (RPE) using the validated Children's OMNI Scale of Perceived Exertion (84).

2.3.7 Data collection and processing

Summary data for spirometry, lung volumes, and diffusion capacity were listed on a standard lab report printed and provided by the RT. Raw ventilatory and metabolic data were recorded continuously during the resting baseline period and exercise test using a 16-channel analog-to-digital data acquisition system (PowerLab/16SP model ML 795, ADInstruments, Colorado Springs, CO, USA) and stored on a laboratory computer for subsequent analysis (LabChart v6.1.3, ADInstrument, Colorado Springs, CO, USA).

2.4 Data analysis

2.4.1 Predictive values

Predictive regression equations used to generate reference values for each subject are listed in Appendix D. Spirometric measurements and lung volumes were compared to predicted values determined using age-, height-, and sex-based regression equations from a large, longitudinal study comprising of subjects with similar age and ethnicity (85). As recommended, predictive values for FRC/TLC, RV/TLC, and VC were derived from those for TLC, FRC, and RV (86). Predictive values for diffusion capacity, V_A, and D_{L,CO}/V_A were determined from age-, height-, and sex-based regression equations from a large, multi-centre cross-sectional study that also comprised of subjects with similar age and ethnicity (87). Regression equations based on age, height, weight, and sex and generated from a large cross-sectional study were used to calculate reference values for PI_{MAX} and PE_{MAX} (45). A sex- and weight-based regression equation for maximal oxygen consumption ($\dot{V}O_{2MAX}$) measured using a similar cycling protocol and 6-17 year old children was used as the reference value for $\dot{V}O_{2MAX}$ (88). When the limits of abnormality based upon the predictive equation's 95% confidence intervals were provided, each subject's %-predicted value was identified as normal or abnormal (85) (Appendix A).

2.4.2 Dysanapsis ratio

The dysanapsis ratio (DR) was calculated according to the equation: $DR = \frac{FEF_{50\%}}{VC \times Pst(L)_{50}}$, where Pst(L)₅₀ was the static recoil pressure at 50% of VC (89). Because static recoil pressures were not measured directly, Pst(L)₅₀ was estimated using a height-based regression equation derived from elastic recoil measurements in 130 children and adolescents aged 6-17 years old: $Pst(L)_{50} = 0.0770 \times Height - 3.3871$ (90). Because there is no evidence of a difference in lung elasticity in swimmers (29), the same regression equation was used for both swimmers and controls.

2.4.3 Maximum expiratory flow-volume curve

Maximum expiratory flow-volume curves were created by superimposing all FVC and gFVC maneuvers and determining the highest flow for each 10 ml volume increment of the FVC. Constructed curves were used to calculate numerical descriptions of the MEFV: the slope ratio (SR), β -angle (β°), and flow ratio (FR). Slope ratio, a quantity describing the emptying of the lungs, was calculated instantaneously at each 10 ml volume increment by comparing the ratio of the tangent to the chord slopes (91). To determine the tangent slope, flows and volumes 200 mL above and below the point of interest were used (92). While tangent slopes were also calculated with increments of 50, 100, and 150 ml, no differences in SR were found; therefore, 200 ml was selected to minimize small oscillatory noise. The instantaneous SR were filtered down to 31 discrete points in 2% increments between 80 and 20% of the expired FVC to ensure all subjects could be compared, regardless of FVC size (92). These filtered values were also averaged to provide an overall SR. The β° , which describes the curvature of the MEFV around 50% of the FVC, was calculated as the angle above and to the right of the MEFV, created from three points: (1) the PEF, (2) the FEF_{50%}, and (3) the point of zero flow and volume (93). Lastly, the FR characterized the curvature at low lung volumes by comparing the FEF75% with an "ideal" FEF75% (94). The latter was calculated by multiplying the $FEF_{50\%}$ by 0.5, thus quantifying the flow had it decreased linearly from 50% of the expired FVC.

2.4.4 Metabolic data

The customized metabolic cart used tidal volume (V_T) and breathing frequency (f_B) measured from expiratory flow to calculate expired \dot{V}_E ; \dot{V}_E and the Haldane conversion to calculate inspired minute ventilation (\dot{V}_1); and \dot{V}_E , \dot{V}_I , and the mixed expiratory fractions of O₂ (F_EO₂) and CO₂ (F_ECO₂) to determine $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), and the respiratory exchange ratio (RER). All ventilatory parameters (e.g., V_T , \dot{V}_E) were reported in BTPS and metabolic parameters (i.e., $\dot{V}O_2$, $\dot{V}CO_2$) in STPD. Baseline and exercise data were presented as the 20-30 second average before the IC maneuver used in the calculation of operational lung volumes (see below). Maximal exercise data were presented as the 20-30 second average before the final successful IC maneuver. Subjects were pooled together according to their group (SWIM vs. CON), time point (PRE vs. POST), and work rate. Because the subject with the shortest test finished during the fifth stage (and other subjects completed up to 10 stages), pooled exercise data was compared at six work rates expressed relative to peak work rate (W_{MAX}) (resting baseline (BL), 30%, 50%, 70%, 90%, and 100% of W_{MAX}).

2.4.5 Operational lung volumes

End-expiratory lung volume was determined by subtracting the IC volume (corrected for pneumotachograph drift using the 5-12 breaths preceding the maneuver) from the subject's FVC, while end-inspiratory lung volume (EILV) was calculated by summing V_T and EELV. The difference between the IC and EILV was the inspiratory reserve volume (IRV). By default, the first IC maneuver of the stage and the preceding 20-30 seconds were used to determine the operational lung volumes and metabolic data, respectively. However, in the case where the first IC maneuver was not performed properly (as assessed using the inspiratory flow), the second IC

maneuver during the stage and its preceding 20-30 second period were used. The last successful IC maneuver in the test was used for maximal exercise data. This meant that both maneuvers were analyzed for subjects who performed two IC maneuvers during their last stage.

2.4.6 Tidal flow-volume loops

Average tidal flow-volume loops (FVL) were generated for each stage by averaging the flows over the 10 ml volume increments of the V_T from the tidal breaths in the same 20-30 second period preceding the IC maneuver as was used for the metabolic data. Thus, FVL were composed of a minimum of 5 tidal breaths during resting baseline to a maximum of 30 tidal breaths during maximal exercise. Apneic and double breaths were excluded. To provide a qualitative assessment of ventilatory constraints, the FVL were then superimposed onto the MEFV by aligning the V_T with the EELV.

2.4.7 Expiratory flow limitation

Expiratory flow limitation EFL was determined by calculating the amount of overlap between the tidal FVL and MEFV. For each 10 m increment of the V_T , the FVL's expiratory flow was compared with the corresponding MEFV's flow. Increments where the FVL's expiratory flow exceeded that of the MEFV were considered flow limited. The number of flow-limited increments could be expressed as a percentage of the total number of increments (i.e., as a percentage of the V_T), and subjects whose V_T was greater than 5% flow limited were deemed to exhibit EFL (95).

2.4.8 Ventilatory capacity

Ventilatory capacity, which is the theoretical maximum minute ventilation based on the subject breathing at the maximum expiratory flow across the entire range of the tidal breath, was calculated for each stage from the MEFV, V_T , EELV, and ratio of inspiratory-to-expiratory time (96, 97). This was accomplished by dividing the V_T into 20-30 ml volume segments and determining the maximum expiratory flow for each. The minimum expiratory time was found by dividing the maximum expiratory flow by the volume segment and summing the resulting times for each volume segment. Using the ratio of inspiratory-to-expiratory time, the minimum inspiratory time was calculated and added to the minimum expiratory time, from which the reciprocal was the theoretical maximum f_B for the given V_T and EELV. The maximum f_B was multiplied by the V_T to produce the \dot{V}_{ECAP} , which was then compared with the \dot{V}_E to provide a quantitative assessment of the extent to which each subject was encroaching on their breathing reserve (expressed as \dot{V}_E/\dot{V}_{ECAP}).

2.4.9 Composite maximum expiratory flow-volume curves and tidal flow-volume loops

Composite MEFV and FVL were created for each group (SWIM and CON) at both time points (PRE and POST) to qualitatively characterize the ventilatory constraints during the maximal exercise test. To construct the composite MEFV, each subject's MEFV had to be reduced to the same number of volume data points by filtering the % FVC expired according to the subject with the smallest FVC. Then, for each % FVC expired increment, the expiratory flow and absolute FVC expired were averaged. A similar process was used to construct composite FVL, except the % V_T expired and subject with the smallest V_T for a given relative work rate were used.

2.4.10 Statistical analysis

Two-way mixed-factorial analysis of variance (ANOVA) tests were used to determine statistically significant interactions and main effects between the groups and time points for descriptive characteristics, spirometry, lung volumes, diffusion capacity, maximal static pressures, DR, MEFV quantities (SR, β° , and FR), and $\dot{V}O_{2MAX}$. For these tests, group served as the betweensubjects independent variable (two levels: SWIM and CON) and time point was the within-subjects independent variable (two levels: PRE and POST). Three-way mixed-factorial ANOVA compared the exercise response between the groups, time points, and work rates for all metabolic and ventilatory parameters except RPE. In addition to the independent variables (group and time point), relative work rate was the second within-subjects independent variable (six levels: BL, 30%, 50%, 70%, 90%, and 100% of W_{MAX}). A generalized estimating equation was used to assess SMR and RPE, as data for these variables were ordinal. A generalized estimating equation was also used to determine if the prevalence of EFL was different between SWIM and CON at PRE and POST. A three-way mixed-model ANOVA compared instantaneous SR for the groups and time points across the expired FVC.

Normality for each combination of levels (e.g., SWIMxPRExBL) was assessed qualitatively by visually inspecting descriptive statistics (including kurtosis), histograms, and Q-Q plots and quantitatively using a suitable Shapiro-Wilk test for small samples, as recommended by Tabachnick and Fidell (98). Outliers were identified through visual inspection of box and whisker plots and were kept in the analysis to maintain sample size, for theoretical reasons (i.e., to avoid subjectively removing data points), and because ANOVA are quite robust to violations of normality. Homogeneity of variances between the groups (i.e., between-subjects factor) for each combination of time point and work rate (i.e., within-subject factors) was tested using Levene's Test of Equality of Error Variances. Given similar group sizes, data that moderately departed from the assumption of equal variances were still interpreted unless qualitative assessments of positive correlations between group and level (i.e., the group with the larger variance also had the greater mean) were found. Mauchly's Test of Sphericity was used to test if the variances of the differences between levels of the within-subject factors for both groups of the between-subjects factor were equal. When a significant difference rejected the assumption of sphericity, the Greenhouse-Geisser adjusted test was interpreted. Statistically significant *F*-ratios, as well as those approaching statistical significance (i.e., $p \le 0.10$), were further analyzed for magnitude and direction using independent and paired *t*-tests for between- and within-subject data, respectively (Bonferonnicorrected levels of significance were not used (99)). Main effects were not interpreted if statistically significant interactions were found.

Independent *t*-tests were used to compare the time between visits and self-reported physical activity levels. Associations between swimming history and pulmonary function as well as swim training volume and changes in pulmonary function were quantified using Pearson's correlation coefficient. A level of significance of p<0.05 was used for all statistical comparisons, and a suitable Shapiro-Wilk test for small samples assessed the assumption of a normal distribution. Statistical tests were performed using SPSS statistics (Version 20, IBM Corporation, Armonk, NY, USA).

RESULTS

3.1 Descriptive data

Anthropometric data is presented for both groups in Table 4. The two groups had similar age (p=0.10) and height (p=0.38) during both PRE and POST; SWIM tended to be heavier (p=0.10) with a larger body mass index (BMI) (p=0.10) and body surface area (BSA) (p=0.12), but these differences were not statistically significant. Most subjects listed their SMR as pubertal (between Tanner stage 2 and 4), and there were no differences between groups (p=0.27). Hemoglobin was similar for all groups (p=0.89) and time points (p=0.15). There were no statistically significant interactions between group and time point and no main effect of group for any anthropometric variable. The average follow-up time was similar between the two groups (7.3 ± 0.5 and 7.6 ± 0.4 months for SWIM and CON, respectively; p=0.16).

		Swimmers (n=11)	Controls $(n=10)$	Interaction p-value	Group p-value	Time point <i>p</i> -value
Age (y)	PRE	12.4 ± 0.8	13.2 ± 1.3	0.53	0.10	< 0.001
	POST	13.0 ± 0.8	13.8 ± 1.3			POST > PRE
Height (cm)	PRE	161.3 ± 7.9	158.3 ± 7.4	0.59	0.38	< 0.001
0	POST	163.4 ± 6.9	160.7 ± 7.0			POST > PRE
Weight (kg)	PRE	52.4 ± 10.8	46.3 ± 5.4	0.71	0.10	< 0.001
	POST	55.8 ± 9.8	49.4 ± 5.6			POST > PRE
BMI (kg·m ⁻²)	PRE	19.9 ± 2.5	18.5 ± 1.7	0.54	0.10	< 0.001
	POST	20.8 ± 2.3	19.1 ± 1.6			POST > PRE
BSA (m ²)	PRE	1.53 ± 0.19	1.43 ± 0.11	0.91	0.12	< 0.001
	POST	1.59 ± 0.17	1.48 ± 0.11			POST > PRE
Hb (g·dl ⁻¹)	PRE	13.3 ± 1.5	13.5 ± 0.6	0.64	0.89	0.15
	POST	13.8 ± 1.0	13.7 ± 1.3			
SMR pubic hair [#]				0.81	0.27	< 0.001
Ι	PRE	1 (9%)	1 (10%)			POST > PRE
	POST	0 (0%)	0 (0%)			
Π	PRE	1 (9%)	1 (10%)			
	POST	1 (9%)	1 (10%)			
III	PRE	2 (18%)	5 (50%)			
	POST	1 (9%)	4 (40%)			
IV	PRE	6 (55%)	2 (20%)			
	POST	6 (55%)	3 (30%)			
V	PRE	1 (9%)	1 (10%)			
	POST	3 (27%)	2 (20%)			
SMR breasts#				0.54	0.23	< 0.01
Ι	PRE	0 (0%)	1 (10%)			POST > PRE
	POST	0 (0%)	0 (0%)			
Π	PRE	2 (18%)	1 (10%)			
	POST	0 (0%)	0 (0%)			
III	PRE	1 (9%)	4 (40%)			
	POST	1 (9%)	4 (40%)			
IV	PRE	8 (73%)	3 (30%)			
	POST	8 (73%)	5 (50%)			
V	PRE	0 (0%)	1 (10%)			
	POST	2 (18%)	1 (10%)			

Table 4 – Anthropometric data

Values are expressed as mean \pm SD except [#] presented as count (relative frequency). BMI, body mass index; BSA, body surface area; Hb, hemoglobin; POST, follow-up visit; PRE, initial visit; SMR, sexual maturity rating.

Self-reported physical activity levels for both groups and swim training history for SWIM are found in Table 5. Both groups self-reported similar levels of physical activity, with 8 out of 10

CON subjects and 10 of the SWIM subjects (1 swimmer did not complete the questionnaire) meeting the Canadian Society for Exercise Physiology's guideline of 60 minutes of moderate-tovigorous intensity physical activity per day (100). The swimmers had a diversity of training backgrounds, with the age of onset of swim training ranging from 6.0 to 10.1 y old and experience ranging from 1.1 to 6.3 y. The 10 year-round swimmers trained 5-7 times per week, ranging from 2.5-4.8 km per session and 7.5-15.5 h and 10-30 km per week. All of them completed weekly breath control drills, with underwater dolphin or breast kick off of the wall being the most common drill. Only 3 swimmers performed freestyle breathing pattern drills ("hypoxic training"). The 11th swimmer's weekly training regimen consisted of seven water polo (totalling 12 h in the pool) and one 3 km speed swimming sessions, but these did not include any breath control drills.

	0 1		
	Swimmers (n=11)	Controls (n=10)	p-value
PAQ ^σ	3.1 ± 0.4	3.1 ± 0.5	0.58
Daily activity (min) ^o	121 ± 25	110 ± 55	0.76
Starting swimming age (y)	9.2 ± 1.4	-	-
Swimming experience (y)	3.2 ± 1.8	-	-
Swim distance per week (km)	19 ± 8	-	-
Swim time per week (h)	9.1 ± 3.6	-	-
Non-swim training time per week (h)	1.0 ± 0.6	-	-
Breath control drills time per week (h)	1.3 ± 1.1	-	-
Breath control drills [#]			
Underwater kick	8 (73%)	-	-
Freestyle breathing pattern ("hypoxic training")	3 (27%)	-	-
Snorkel sets	1 (9%)	-	-
Other	1 (9%)	-	-
None	1 (9%)	-	-

Table 5 – Activity levels and swim training history

All values are expressed as means \pm SD. $^{\sigma}$ swimmers n=10. [#] data presented as count (relative frequency). NS, not statistically significant. PAQ, self-reported physical activity questionnaire score.

3.2 Spirometry

Swimmers had statistically significantly larger values for all spirometric measurements except FEV₁/FVC (p=0.49) and FEF_{25%} (p=0.08) (Table 6). This is illustrated in Figure 1, where the average MEFV for SWIM at both time points has a wider FVC and higher peak flow. However, the figure also suggests that SWIM and CON produced a similar flow for a given FVC. There was no change in FEV₁/FVC in either SWIM or CON. Comparing the groups to their reference values, swimmers consistently exceeded their predicted function (means ranging from 97 to 125%) and had, on average, an abnormally high FVC. The controls were more similar to their predicted function, on average ranging from 78 to 102% of their reference values. Assessing the pulmonary function data individually, seven swimmers had a %-predicted FVC during at least one visit that was above the limits of abnormality (Appendix A). None had a spirometric value below the limits of abnormality. Conversely, four controls had at least one abnormally low spirometric value and none had a measurement that was abnormally high. While a main effect of group was noted for almost all spirometric measurements (except FEV₁/FVC (p=0.49) and FEF_{25%} (p=0.08)), no statistically significant interactions were found.

		Swimmers (n=11)	Controls (n=10)	Interaction p-value	Group p-value	Time point p-value
FVC (l)	PRE	3.92 ± 0.71	3.13 ± 0.50	0.27	< 0.01	< 0.001
	POST	4.15 ± 0.61	3.28 ± 0.54		SWIM > CON	POST > PRE
FVC (% pred)	PRE	123 ± 11	102 ± 11	0.24	< 0.001	0.28
	POST	125 ± 10	101 ± 11		SWIM > CON	
FEV_1 (1)	PRE	3.34 ± 0.61	2.61 ± 0.43	0.23	< 0.01	< 0.001
	POST	3.55 ± 0.57	2.74 ± 0.43		SWIM > CON	POST > PRE
FEV ₁ (% pred)	PRE	117 ± 11	95 ± 13	0.21	< 0.001	0.35
	POST	119 ± 10	94 ± 12		SWIM > CON	
FEV ₁ /FVC (%)	PRE	85 ± 2	84 ± 7	0.98	0.49	0.63
	POST	85 ± 3	84 ± 7			
PEF $(l \cdot s^{-1})$	PRE	6.48 ± 0.92	5.70 ± 0.86	0.21	0.03	< 0.001
	POST	6.97 ± 0.84	6.00 ± 0.77		SWIM > CON	POST > PRE
PEF (% pred)	PRE	97 ± 9	86 ± 10	0.24	< 0.01	0.02
	POST	101 ± 8	87 ± 10		SWIM > CON	POST > PRE
FEF25-75% (l·s ⁻¹)	PRE	3.56 ± 0.73	2.74 ± 0.81	0.47	0.02	0.04
	POST	3.76 ± 0.84	2.85 ± 0.83		SWIM > CON	POST > PRE
FEF25-75% (% pred)	PRE	100 ± 15	79 ± 22	0.64	0.01	0.80
	POST	101 ± 15	78 ± 22		SWIM > CON	
FEF _{25%} (1·s ⁻¹)	PRE	5.66 ± 0.87	4.82 ± 1.22	0.57	0.08	< 0.001
	POST	6.10 ± 0.99	5.16 ± 1.31			POST > PRE
FEF25% (% pred)	PRE	115 ± 10	98 ± 23	0.65	0.04	0.09
	POST	119 ± 14	101 ± 24		SWIM > CON	
FEF50% (1·s ⁻¹)	PRE	4.03 ± 0.85	3.08 ± 0.90	0.74	0.02	0.14
	POST	4.20 ± 0.95	3.19 ± 0.87		SWIM > CON	
FEF50% (% pred)	PRE	118 ± 20	91 ± 25	0.96	< 0.01	0.75
	POST	117 ± 18	90 ± 23		SWIM > CON	
FEF75% (l·s ⁻¹)	PRE	1.90 ± 0.44	1.39 ± 0.43	0.16	< 0.01	< 0.01
	POST	2.19 ± 0.59	1.48 ± 0.42		SWIM > CON	POST > PRE
FEF75% (% pred)	PRE	107 ± 17	80 ± 24	0.22	< 0.01	0.16
	POST	116 ± 22	81 ± 23		SWIM > CON	

Table 6 – Spirometry

All values are expressed as mean \pm SD. FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 1 – Composite maximum expiratory flow-volume curve from the pulmonary function test. From left to right, data points represent total lung capacity, peak expiratory flow (PEF), forced expiratory flow when 25% (FEF_{25%}), 50% (FEF_{50%}), and 75% (FEF_{75%}) of the forced vital capacity (FVC) has been expired, and residual volume (RV).

3.3 Lung volumes

Individually, swimmers had a greater TLC than controls for nearly all heights (Figure 2), meaning that the swimmers as a group had a larger TLC at both PRE and POST (Table 7). However, there was no statistically significant interaction between group and time point for TLC (p=0.29). Because RV was the same between groups, the greater TLC in swimmers was due to a larger VC whilst RV/TLC tended to be smaller in swimmers compared to controls (p=0.07). Similar to the spirometry results, swimmers exceeded their predictions for TLC (ranging 100-

123%) whereas controls were around their expected values (81-106%; Figure 3). Two swimmers, but no controls, had a TLC considered to be abnormally large. There was no correlation between starting age of swimming and TLC (r=-0.39, p=0.23) or %-predicted TLC (r=0.20, p=0.56), or years of training history and %-predicted TLC (r=-0.12, p=0.72) (Figure 4). Moreover, there were no significant correlations between swim training per week (km) and absolute (r=0.09, p=0.78) or relative (r=-0.02, p=0.95) change in TLC (Figure 5). In fact, there were no trends or significant correlations between any pulmonary function parameter and starting age or training history (for %-predicted values) whereby an earlier age of swimming onset or more years of swim training experience correlated with a greater absolute or %-predicted value (Table 8). Similarly, no significant correlations were found between the absolute or relative change in a pulmonary function parameter and training er week (expressed either in hours or kilometres) (Table 9). Lastly, when the swimmers and controls were combined into one group, no association was found between daily moderate-vigorous physical activity levels and relative change in TLC (r=0.08, p=0.75) (Figure 6).

Statistically significant interactions between group and time point were observed for FRC (p=0.04), %-predicted FRC (p=0.01), and FRC/TLC (p=0.03). Post-hoc comparisons showed that swimmers increased FRC from PRE to POST (p<0.01), but there were no differences between PRE and POST for controls (p=0.80) or between the two groups (PRE: p=0.94, POST: p=0.28). There was no difference in %-predicted FRC between swimmers and controls at PRE (p=0.88), whereas at POST the swimmers were larger (p=0.05). There was no change from PRE to POST in swimmers (p=0.16), while in controls %-predicted FRC decreased (p=0.04). Conversely, FRC/TLC tended to decrease from PRE to POST in the controls (p=0.06) and was larger than the swimmers at PRE (p=0.001) and POST (p=0.07).



Figure 2 - Total lung capacity for individual subjects in relation to their height. Individual data are presented with an open symbol connected by a solid line, while group averages have a closed symbol connected by a hashed line.

		Swimmers (n=11)	Controls (n=10)	Interaction p-value	Group p-value	Time point p-value
TLC (l)	PRE	4.73 ± 0.73	3.93 ± 0.46	0.29	< 0.01	< 0.001
	POST	5.08 ± 0.68	4.19 ± 0.64		SWIM > CON	POST > PRE
TLC (% pred)	PRE	110 ± 7	94 ± 7	0.18	< 0.001	0.15
	POST	112 ± 8	94 ± 7		SWIM > CON	
FRC (1)	PRE	2.18 ± 0.43	2.19 ± 0.28	0.04	-	-
	POST	$2.40 \pm 0.39^{**}$	2.21 ± 0.40			
FRC (% pred)	PRE	102 ± 14	103 ± 4	0.01	-	-
	POST	106 ± 14	$96\pm7*$			
RV (l)	PRE	0.99 ± 0.16	0.96 ± 0.20	0.97	0.70	0.01
	POST	1.04 ± 0.19	1.01 ± 0.25			POST > PRE
RV (% pred)	PRE	96 ± 13	91 ± 14	0.85	0.39	0.46
	POST	95 ± 16	90 ± 16			
VC (l)	PRE	3.74 ± 0.65	2.98 ± 0.45	0.24	< 0.01	< 0.001
	POST	4.03 ± 0.61	3.18 ± 0.55		SWIM > CON	POST > PRE
VC (% pred)	PRE	114 ± 9	94 ± 12	0.17	< 0.001	0.02
	POST	118 ± 11	95 ± 12		SWIM > CON	POST > PRE
FRC/TLC	PRE	$0.46 \pm 0.05^{\text{\#\#\#}}$	0.56 ± 0.04	0.03	-	-
	POST	0.47 ± 0.06	0.53 ± 0.05			
FRC/TLC (% pred)	PRE	$93\pm10^{\#\#}$	110 ± 8	0.02	-	-
	POST	95 ± 13	$103\pm10^{\ast}$			
RV/TLC	PRE	0.21 ± 0.03	0.24 ± 0.05	0.92	0.07	0.72
	POST	0.21 ± 0.03	0.24 ± 0.05			
RV/TLC (% pred)	PRE	88 ± 13	99 ± 20	0.83	0.15	0.10
	POST	85 ± 15	96 ± 21			

Table 7 – Lung volumes

All values are expressed as mean \pm SD. *p<0.05, **p<0.01 statistically significant difference within group between PRE and POST. #p<0.05, ###p<0.001 statistically significant difference within time point between SWIM and CON. TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; VC, vital capacity; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 3 – Percent-predicted total lung capacity (TLC), vital capacity (VC), functional residual capacity (FRC), and residual volume (RV) for each group during the PRE and POST visits. Bars are presented as mean \pm SE. [#]p<0.05, significant difference between PRE to POST. *p<0.05, significant difference within group between PRE and POST.



Figure 4 – Percent-predicted total lung capacity at the initial measurement compared to the number of years of swimming experience for each swimmer.



Figure 5 – Relative change in total lung capacity from PRE to POST compared to the average weekly swim training volume for each swimmer.


Figure 6 – Relative change in total lung capacity from PRE to POST compared to the average daily moderate-vigorous physical activity in all subjects.

	Startin	g age (y)	Years experience (y)		
	r	P-value	r	P-value	
TLC (l)	-0.39	0.23	0.65	0.03	
TLC (% pred)	0.20	0.56	-0.12	0.72	
FRC (l)	-0.49	0.13	0.72	0.01	
FRC (% pred)	-0.18	0.59	0.22	0.51	
RV (l)	-0.06	0.87	0.35	0.29	
RV (% pred)	0.45	0.17	-0.40	0.22	
VC (l)	-0.42	0.19	0.64	0.03	
FRC/TLC	-0.28	0.41	0.32	0.34	
RV/TLC	0.34	0.31	-0.29	0.39	
FVC (l)	-0.42	0.20	0.64	0.03	
FVC (% pred)	-0.08	0.81	0.18	0.59	
FEV ₁ (l)	-0.38	0.25	0.62	0.04	
FEV ₁ (% pred)	-0.03	0.92	0.16	0.64	
FEV ₁ /FVC	0.25	0.46	-0.10	0.77	
PEF $(l \cdot s^{-1})$	-0.36	0.28	0.68	0.02	
PEF (% pred)	-0.14	0.67	0.43	0.18	
FEF _{25-75%} (l·s ⁻¹)	-0.26	0.44	0.50	0.11	
FEF _{25-75%} (% pred)	-0.01	0.98	0.17	0.62	
FEF _{25%} (l·s ⁻¹)	-0.38	0.25	0.66	0.03	
FEF _{25%} (% pred)	-0.09	0.79	0.26	0.43	
FEF _{50%} (l·s ⁻¹)	-0.31	0.35	0.50	0.12	
FEF50% (% pred)	-0.08	0.81	0.16	0.64	
FEF _{75%} (l·s ⁻¹)	-0.20	0.56	0.44	0.17	
FEF75% (% pred)	0.15	0.66	-0.01	0.97	
DL,CO (ml·min ⁻¹ ·mmHg ⁻¹)	0.04	0.91	0.26	0.43	
DL,CO (% pred)	0.61	0.04	-0.49	0.13	
DL,COC (ml·min ⁻¹ ·mmHg ⁻¹)	0.15	0.66	0.08	0.81	
D _{L,CO} c (% pred)	0.67	0.02	-0.67	0.02	
V _A (l)	-0.39	0.24	0.65	0.03	
V _A (% pred)	0.21	0.54	-0.13	0.71	
$D_{L,COC}/V_A (ml \cdot min^{-1} \cdot mmHg^{-1} \cdot l^{-1})$	0.62	0.04	-0.71	0.01	
D _{L,COC} /V _A (% pred)	0.55	0.08	-0.59	0.05	
PI _{MAX} (cm H ₂ O)	0.12	0.71	0.00	1.00	
PI _{MAX} (% pred)	0.34	0.30	-0.31	0.36	
PE _{MAX} (cm H ₂ O)	-0.46	0.15	0.67	0.02	
PE _{MAX} (% pred)	-0.43	0.19	0.54	0.09	

Table 8 – Correlations between swimming history and pulmonary function

TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; VC, vital capacity; FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow; D_{L,CO}, diffusion capacity of the lung for carbon monoxide; D_{L,CO}, D_{L,CO} corrected for hemoglobin; V_A; alveolar volume; PI_{MAX}, maximal inspiratory pressure; PE_{MAX}, maximal expiratory pressure; SWIM, swimmers; CON, controls.

	Training po	Training per week (h) vs. Λ in variable		Training per week (h) vs. $\% \Lambda$ in variable		Training per week (km)		Training per week (km)	
	r	P-value	r r	P-value	R R	P-value	r r	P-value	
TLC (l)	0.35	0.29	0.14	0.67	0.09	0.78	-0.02	0.95	
TLC (% pred)	0.46	0.15	0.47	0.14	0.17	0.62	0.20	0.56	
FRC (1)	-0.30	0.38	-0.21	0.54	0.05	0.87	0.16	0.63	
FRC (% pred)	-0.22	0.51	-0.17	0.62	0.14	0.68	0.21	0.54	
RV (l)	-0.07	0.84	-0.06	0.86	-0.24	0.47	-0.22	0.51	
RV (% pred)	0.00	1.00	-0.01	0.97	-0.17	0.61	-0.19	0.58	
VC (l)	0.34	0.30	0.16	0.64	0.25	0.46	0.12	0.72	
FRC/TLC	-0.35	0.30	-0.29	0.38	0.11	0.76	0.16	0.64	
RV/TLC	-0.10	0.76	-0.10	0.76	-0.23	0.50	-0.21	0.53	
FVC (l)	-0.01	0.98	-0.06	0.86	0.10	0.78	0.06	0.86	
FVC (% pred)	0.19	0.57	0.19	0.57	0.25	0.45	0.29	0.38	
FEV ₁ (l)	-0.12	0.72	-0.25	0.45	-0.22	0.51	-0.26	0.43	
FEV ₁ (% pred)	0.04	0.90	0.01	0.97	-0.11	0.74	-0.09	0.78	
FEV ₁ /FVC	-0.15	0.66	-0.14	0.67	-0.33	0.32	-0.33	0.33	
PEF $(l \cdot s^{-1})$	-0.40	0.22	-0.41	0.21	-0.39	0.23	-0.41	0.21	
PEF (% pred)	-0.30	0.36	-0.31	0.36	-0.32	0.33	-0.32	0.34	
FEF _{25-75%} (l·s ⁻¹)	0.29	0.38	0.22	0.51	0.00	1.00	-0.02	0.95	
FEF _{25-75%} (% pred)	0.30	0.37	0.30	0.37	0.04	0.91	0.04	0.91	
FEF _{25%} (l·s ⁻¹)	0.16	0.63	0.10	0.77	0.13	0.71	0.13	0.70	
FEF _{25%} (% pred)	0.22	0.53	0.19	0.58	0.20	0.56	0.20	0.56	
FEF _{50%} (l·s ⁻¹)	0.42	0.20	0.37	0.26	0.23	0.49	0.23	0.51	
FEF _{50%} (% pred)	0.39	0.24	0.42	0.20	0.26	0.44	0.26	0.43	
FEF _{75%} (l·s ⁻¹)	-0.20	0.56	-0.33	0.33	-0.30	0.37	-0.34	0.31	
FEF _{75%} (% pred)	-0.23	0.50	-0.26	0.43	-0.30	0.37	-0.30	0.38	
$D_{L,CO}(ml\!\cdot\!min^{\text{-}1}\!\cdot\!mmHg^{\text{-}1})$	0.10	0.78	0.10	0.78	0.07	0.83	0.04	0.91	
D _{L,CO} (% pred)	0.25	0.45	0.20	0.56	0.16	0.64	0.12	0.73	
$D_{L,CO}c \ (ml \cdot min^{-1} \cdot mmHg^{-1})$	0.13	0.70	0.22	0.52	0.10	0.78	0.09	0.79	
D _{L,CO} c (% pred)	0.30	0.37	0.24	0.48	0.19	0.57	0.16	0.64	
V _A (l)	0.36	0.28	0.16	0.65	0.09	0.78	-0.02	0.96	
V _A (% pred)	0.59	0.05	0.60	0.05	0.26	0.45	0.29	0.39	
$D_{L,CO}c/V_A (ml \cdot min^{-1} \cdot mmHg^{-1} \cdot l^{-1})$	0.19	0.58	0.08	0.81	0.17	0.61	0.10	0.76	
$D_{L,CO}c/V_A$ (% pred)	0.11	0.75	0.04	0.90	0.12	0.72	0.07	0.83	
PI _{MAX} (cm H ₂ O)	-0.14	0.69	-0.42	0.20	-0.33	0.32	-0.51	0.11	
PI _{MAX} (% pred)	-0.17	0.62	-0.33	0.32	-0.40	0.23	-0.47	0.15	
PE _{MAX} (cm H ₂ O)	0.13	0.70	0.05	0.89	0.16	0.63	0.09	0.79	
PE _{MAX} (% pred)	0.07	0.83	0.04	0.90	0.11	0.75	0.09	0.78	

Table 9 – Correlations between weekly training volume and changes in pulmonary function

TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; VC, vital capacity; FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow; D_{L,CO}, diffusion capacity of the lung for carbon monoxide; D_{L,CO}, D_{L,CO} corrected for hemoglobin; V_A; alveolar volume; PI_{MAX}, maximal inspiratory pressure; PE_{MAX}, maximal expiratory pressure; SWIM, swimmers; CON, controls.

3.4 Diffusion capacity

Swimmers had a statistically significantly greater $D_{L,COC}$ (p=0.01) and V_A (p<0.01) than controls (Table 10). However, when $D_{L,COC}$ was expressed relative to V_A , there were no differences between the groups (p=0.20). Group averages for diffusion capacity did not change from PRE to POST in SWIM or CON, as illustrated in panel A of Figure 7. Conversely, V_A increased (Figure 7B) and $D_{L,COC}/V_A$ decreased (Figure 7C) from PRE to POST. While the swimmers had high %predicted values for $D_{L,COC}$ and V_A , controls were within normal limits. No statistically significant interactions were found.

		Swimmers (n=11)	Controls (n=10)	Interaction p-value	Group p-value	Time point p- value
D _{L,CO} (ml·min ⁻¹ ·mmHg ⁻¹)	PRE	23.29 ± 2.87	20.76 ± 1.93	0.39	0.01	0.09
_	POST	24.33 ± 2.16	21.13 ± 3.10		SWIM > CON	
DL,CO (% pred)	PRE	121 ± 11	110 ± 8	0.28	< 0.01	0.61
	POST	122 ± 12	107 ± 8		SWIM > CON	
D _{L,CO} c (ml·min ⁻¹ ·mmHg ⁻¹)	PRE	23.43 ± 2.58	20.73 ± 1.88	0.63	< 0.01	0.26
	POST	24.09 ± 1.83	21.00 ± 3.18		SWIM > CON	
DL,COC (% pred)	PRE	122 ± 12	110 ± 8	0.55	< 0.01	0.31
	POST	121 ± 13	107 ± 9		SWIM > CON	
V _A (l)	PRE	4.61 ± 0.71	3.83 ± 0.45	0.28	< 0.01	< 0.001
	POST	4.96 ± 0.66	4.08 ± 0.64		SWIM > CON	POST > PRE
V _A (% pred)	PRE	114 ± 7	98 ± 7	0.17	< 0.001	0.05
	POST	117 ± 8	99 ± 8		SWIM > CON	
$D_{L,COC}/V_A (ml \cdot min^{-1} \cdot mmHg^{-1} \cdot l^{-1})$	PRE	5.14 ± 0.60	5.44 ± 0.44	0.65	0.20	< 0.001
	POST	4.91 ± 0.56	5.16 ± 0.38			PRE > POST
$D_{L,COC}/V_A$ (% pred)	PRE	101 ± 10	106 ± 8	0.68	0.25	< 0.01
	POST	97 ± 9	101 ± 8			PRE > POST

Table	10 -	Diffusion	capacity
	-		

All values are expressed as mean \pm SD. D_{L,CO}, diffusion capacity of the lung for carbon monoxide; D_{L,CO}c, D_{L,CO} corrected for hemoglobin; V_A; alveolar volume; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 7 – A) Diffusion capacity, B) alveolar volume, and C) $D_{L,COC}/V_A$ for swimmers (\circ) and controls (Δ) during PRE and POST time points. Individual data are presented with an open symbol, while group averages have a closed symbol.

3.5 Maximal static pressures

Maximal inspiratory mouth pressure tended to be greater in swimmers (p=0.06), and while PE_{MAX} was significantly greater in swimmers (p=0.001) compared to controls (Table 11). Both groups increased PI_{MAX} and PE_{MAX} from PRE to POST (Figure 8), but no statistically significant interactions were noted.

	^	Swimmers (n=11)	Controls (n=10)	Interaction p-value	Group p-value	Time point p- value
PI _{MAX} (cm H ₂ O)	PRE	87 ± 26	71 ± 24	0.19	0.06	< 0.001
	POST	103 ± 22	79 ± 26			POST > PRE
PI _{MAX} (% pred)	PRE	96 ± 26	82 ± 30	0.23	0.13	< 0.01
	POST	109 ± 19	87 ± 30			POST > PRE
PE _{MAX} (cm H ₂ O)	PRE	112 ± 17	85 ± 16	0.77	< 0.001	< 0.01
	POST	127 ± 17	98 ± 18		SWIM > CON	POST > PRE
PE _{MAX} (% pred)	PRE	104 ± 13	77 ± 19	0.83	< 0.001	0.02
	POST	114 ± 13	84 ± 19		SWIM > CON	POST > PRE

Table 11 – Maximal static pressures

All values are expressed as mean \pm SD. PI_{MAX}, maximal inspiratory pressure; PE_{MAX}, maximal expiratory pressure; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 8 – A) Maximal inspiratory pressure B) maximal expiratory pressure for swimmers (\circ) and controls (Δ) during PRE and POST time points. Individual data are presented with an open symbol, while group averages have a closed symbol.

3.6 Dysanapsis ratio

As shown in Figure 9, the DR was similar between swimmers and controls for PRE (0.12 ± 0.02 vs. 0.12 ± 0.03 for SWIM and CON, respectively) and POST (0.11 ± 0.01 vs. 0.11 ± 0.03) (p=0.95). No significant interaction was found (p=0.61) and DR tended to decrease from PRE to POST (p=0.09).



Figure 9 – Dysanapsis ratio for swimmers (\circ) and controls (Δ) during PRE and POST time points. Individual data are presented with an open symbol, while group averages have a closed symbol.

3.7 Maximum expiratory flow-volume curve

No differences between groups or time points were found for any characteristic of the MEFV curve, including FR, β° , and SR (

Table 12). When instantaneous SR was plotted versus FVC (Figure 10), there was a statistically significant main effect for instantaneous SR (p<0.001) but not for group (p=0.28) or time point (p=0.44). There were no significant interactions (Figure 10).

		Swimmers (n=11)	Controls (n=10)	Interaction p-value	Group p-value	Time point p-value
FR (%)	PRE	-3 ± 19	-5 ± 8	0.41	0.71	0.73
	POST	-5 ± 17	0 ± 10			
$\beta^{\circ}(^{\circ})$	PRE	195 ± 9	194 ± 15	0.60	0.64	0.52
	POST	194 ± 14	191 ± 12			
SR (au) PRE	0.83 ± 0.23	0.89 ± 0.23	0.50	0.28	0.43
	POST	0.83 ± 0.21	0.96 ± 0.23			

Table 12 – Maximal expiratory flow-volume curve characteristics

All values are expressed as mean \pm SD. FR, flow ratio; β° , β -angle; SR, slope ratio.



Figure 10 – Instantaneous slope ratio. The box represents the range of values (0.5-2.5) for homogenous emptying of the lung.

3.8 Maximal exercise test

3.8.1 Metabolic and ventilatory responses

The metabolic response to exercise is displayed for selected variables in Figure 11, and maximal exercise data is listed in Table 13. Generally, subjects exercised longer and reached a higher work rate during the follow-up visit. The average RER at peak exercise was greater than 1.1 at both time points for SWIM and CON, suggesting that the tests were maximal.

There were no statistically significant three-way interactions between group, time point, and relative work rate (Table 14), although V_T approached statistical significance (p=0.08). Moreover, no statistically significant two-way interactions were found between group and time point. The only statistically significant two-way interactions involving group were with relative work rate for $\dot{V}O_2$ (p=0.02) and RER (p=0.01), with $\dot{V}CO_2$ approaching significance (p=0.08). Focusing on the main effects of competitive swimming, SWIM tended to have a higher work rate than CON throughout the exercise test (p=0.10); however, when work rate was expressed relative to body mass, there was no difference between groups (p=0.83). Thus, the larger size and therefore absolute work rate of the swimmers may have lead to the greater VCO_2 (p=0.02), stimulating an increased \dot{V}_E (p=0.02) which was achieved by a similar f_B (p=0.99) but greater V_T (p=0.02). Although $\dot{V}O_2$ was greater in SWIM at each stage (p<0.05), these differences were abolished when expressed relative to body mass (p=0.26). Therefore, absolute VO_{2MAX} was greater in swimmers initially (p<0.01) and at the follow-up measurement (p<0.001), but there were no differences in relative \dot{VO}_{2MAX} between groups (p=0.32) or time points (p=0.11). Swimmers had a smaller RER only at 50% W_{MAX} (p=0.04) and 70% W_{MAX} (p=0.04). Lastly, the HR response (p=0.39) and RPE (p=0.85) were similar between the groups. Results were similar when compared at absolute work rates.

	3-way	Group x work rate	Time x work rate	Group x time	Work rate	Group	Time
Work rate (W)	0.63	0.13	< 0.001	0.31	-	0.10	-
Work rate (W·kg ⁻¹)	0.68	0.83	0.08	0.39	< 0.001	0.99	< 0.01
							POST > PRE
HR (bpm)	0.94	0.70	0.56	0.36	< 0.001	0.39	0.04
							POST > PRE
RPE	0.67	0.52	0.57	0.05	< 0.001	0.61	0.97
V _T (l)	0.08	0.62	< 0.01	0.16	-	0.02	-
						SWIM > CON	
V _T /FVC (%)	0.17	0.39	0.10	0.69	< 0.001	0.32	0.69
$f_{\rm B}$ (breaths per minute)	0.78	0.46	0.03	0.19	-	0.99	-
\dot{V}_E (l·min ⁻¹)	0.53	0.11	< 0.001	0.37	-	0.02	-
						SWIM > CON	
[.] VO ₂ (l⋅min ⁻¹)	0.84	0.02	< 0.01	0.85	-	-	-
$\dot{V}O_2$ (ml·kg ⁻¹ ·min ⁻¹)	0.89	0.43	0.30	0.98	< 0.001	0.26	0.17
^{VCO} ₂ (l⋅min ⁻¹)	0.66	0.08	< 0.001	0.50	-	0.02	-
						SWIM > CON	
RER	0.08	0.01	< 0.01	0.61	-	-	-
$\dot{V}_E/\dot{V}O_2$	0.46	0.48	< 0.01	0.69	-	0.76	-
$\dot{V}_E/\dot{V}CO_2$	0.69	0.65	< 0.01	0.87	-	0.95	-
EFL (% V _T)	0.58	0.80	0.09	0.99	< 0.001	0.95	0.07
Ż _{ECAP} (l∙min ⁻¹)	0.17	0.15	0.29	0.70	< 0.001	0.23	0.25
$\dot{V}_{E}/\dot{V}_{ECAP}$ (%)	0.44	0.75	< 0.001	0.90	-	0.96	-

Table 13 - Interactions and main effects p-values for metabolic variables during the exercise test

HR, heart rate; RPE, rating of perceived exertion; V_T , tidal volume; FVC, forced vital capacity; f_B , breathing frequency; \dot{V}_E , expired minute ventilation; $\dot{V}O_2$, oxygen consumption; $\dot{V}CO_2$, carbon dioxide production; RER, respiratory exchange ratio; EFL, expiratory flow limitation; \dot{V}_{ECAP} , ventilatory capacity; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.

Table 14 – Maximal exercise data

di exercise data			
		Swimmers (n=11)	Controls (n=10)
Duration (min)	PRE	14.3 ± 3.0	12.8 ± 2.7
	POST	16.5 ± 1.5	14.6 ± 2.7
Work rate (W)	PRE	167 ± 29	154 ± 25
	POST	191 ± 16	170 ± 27
Work rate (W·kg ⁻¹)	PRE	3.3 ± 0.5	3.3 ± 0.4
	POST	3.5 ± 0.6	3.5 ± 0.5
HR (bpm)	PRE	192 ± 10	196 ± 7
	POST	195 ± 8	198 ± 8
RPE	PRE	9.3 ± 1.8	9.3 ± 1.0
	POST	9.5 ± 1.0	9.4 ± 0.9
V _T (l)	PRE	1.59 ± 0.33	1.44 ± 0.25
	POST	1.77 ± 0.26	1.55 ± 0.26
V _T /FVC (%)	PRE	48 ± 8	52 ± 5
	POST	47 ± 6	52 ± 5
$f_{\rm B}$ (breaths per minute)	PRE	56 ± 16	50 ± 8
	POST	58 ± 13	56 ± 7
\dot{V}_{E} (l·min ⁻¹)	PRE	85.5 ± 20.8	72.0 ± 15.6
	POST	100.4 ± 17.6	85.3 ± 12.5
[.] VO ₂ (l⋅min ⁻¹)	PRE	2.20 ± 0.35	1.85 ± 0.25
	POST	2.42 ± 0.23	2.07 ± 0.27
^{VO} ₂ (ml⋅kg ⁻¹ ⋅min ⁻¹)	PRE	42.9 ± 6.8	40.1 ± 4.2
	POST	44.4 ± 8.1	42.1 ± 5.2
VO _{2MAX} (% pred)	PRE	125 ± 18	115 ± 12
	POST	131 ± 21	122 ± 14
VCO₂ (l·min ⁻¹)	PRE	2.45 ± 0.42	2.13 ± 0.38
	POST	2.77 ± 0.25	2.43 ± 0.33
RER	PRE	1.11 ± 0.05	1.15 ± 0.09
	POST	1.15 ± 0.05	1.18 ± 0.06
$\dot{V}_E/\dot{V}O_2$	PRE	39 ± 5	39 ± 6
	POST	42 ± 6	42 ± 5
Ϋ́E/Ϋ́CO ₂	PRE	35 ± 4	34 ± 4
	POST	36 ± 4	35 ± 4
EFL (% V _T)	PRE	19 ± 24	13 ± 25
	POST	28 ± 28	28 ± 21
Vecap (l∙min ⁻¹)	PRE	122.8 ± 39.2	110.3 ± 32.8
	POST	132.9 ± 37.8	105.3 ± 21.2
V̈́ε/V̈́εcap (%)	PRE	73 ± 19	69 ± 23
	POST	80 ± 21	82 ± 13

All values are expressed as mean \pm SD. HR, heart rate; bpm, beats per minute; RPE, rating of perceived exertion; V_T, tidal volume; FVC, forced vital capacity; *f*_B, breathing frequency; \dot{V}_{E} , expired minute ventilation; $\dot{V}O_2$, oxygen consumption; $\dot{V}CO_2$, carbon dioxide production; RER,

respiratory exchange ratio; EFL, expiratory flow limitation; \dot{V}_{ECAP} , ventilatory capacity; SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 11 – Mean A) heart rate, B) oxygen consumption, C) carbon dioxide production D) breathing frequency, E) tidal volume, and F) V_T /FVC responses during the maximal exercise test. All exercise stages were significantly increased from baseline (BL). [#] significant difference between PRE and POST. Statistical significance was set at the level of p<0.05.

3.8.2 Ventilatory mechanics

As shown in Figure 12, the prevalence of EFL was similar in both groups (p=0.72) but increased from PRE to POST (p=0.03). The interaction was not statistically significant (p=0.12). Moreover, no difference between groups was found for the severity of EFL (p=0.95), \dot{V}_{ECAP} (Figure 13A) (p=0.23), or \dot{V}_E/\dot{V}_{ECAP} (Figure 13B) (p=0.96).



Figure 12 – EFL prevalence for each group and time point.



Figure 13 – Mean A) \dot{V}_E (line and symbols) and \dot{V}_{ECAP} (line only) and B) \dot{V}_E/\dot{V}_{ECAP} during the maximal exercise test. * significant difference between resting baseline and exercise stage for all groups (combined). [#] significant difference between PRE and POST. For \dot{V}_E and \dot{V}_E/\dot{V}_{ECAP} , all exercise stages were significantly increased from baseline (BL). Statistical significance was set at the level of p<0.05.

There were no statistically significant three-way interactions involving group for operational lung volumes (Table 15); however, two-way interactions between group and relative work rate approached significance for EILV (p=0.10), EELV (p=0.11), EELV/FVC (p=0.07), and IRV (p=0.10). A main effect of swimming was found for IRV (p<0.01) such that swimmers had a larger IRV, while differences in EILV (p=0.05), EILV/FVC (p=0.08) and IRV/FVC (p=0.08) approached statistical significance. Swimmers utilized a similar absolute EELV (p=0.18). Although there was a significant interaction between group and time point for IC (p=0.02), IC was greater in SWIM compared to CON at PRE and POST (both p<0.001) and increased from PRE to POST for both SWIM (p<0.01) and CON (p<0.001). The interaction between time and work rate was or approached significance for all volumes. The absolute and relative operational lung

volumes are displayed in Figure 14 and Figure 15, respectively, and IRV, IRV/FVC, IC, and IRV/IC are displayed in Figure 16. Values are maximal exercise are presented in Table 16.

	3-way	Group x work rate	Time x work rate	Group x time	Work rate	Group	Time
EILV (l)	0.17	0.10	0.06	0.70	< 0.001	0.05	< 0.001
							POST > PRE
EILV/FVC (%)	0.37	0.64	0.04	0.27	-	0.08	-
EELV (1)	0.32	0.11	0.09	0.64	< 0.001	0.18	< 0.001
							POST > PRE
EELV/FVC (%)	0.31	0.07	0.03	0.15	-	0.46	-
IRV (l)	0.17	0.10	0.06	0.11	< 0.001	< 0.01	0.13
						SWIM > CON	
IRV/FVC (%)	0.37	0.64	0.04	0.27	-	0.08	-
IC (l)	0.32	0.11	0.09	0.02	< 0.001	-	-
IRV/IC (%)	0.81	0.81	0.10	0.58	< 0.001	0.10	0.37

Table 15 – Interactions and main effects p-values for operational lung volumes during the exercise test

EILV, end-inspiratory lung volume; FVC, forced vital capacity; EELV, end-expiratory lung volume; IRV, inspiratory reserve volume; IC, inspiratory capacity; V_T, tidal volume: SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.

		Swimmers (n=11)	Controls (n=10)
EILV (l)	PRE	2.71 ± 0.73	2.34 ± 0.46
	POST	3.02 ± 0.70	2.48 ± 0.55
EILV/FVC (%)	PRE	80 ± 7	84 ± 3
	POST	80 ± 7	83 ± 6
EELV (l)	PRE	1.12 ± 0.38	0.90 ± 0.27
	POST	1.25 ± 0.43	0.93 ± 0.22
EELV/FVC (%)	PRE	33 ± 5	32 ± 7
	POST	33 ± 7	31 ± 5
IRV (l)	PRE	0.67 ± 0.27	0.42 ± 0.06
	POST	0.74 ± 0.26	0.52 ± 0.20
IRV/FVC (%)	PRE	20 ± 7	16 ± 3
	POST	20 ± 7	17 ± 6
IC (l)	PRE	2.26 ± 0.41	1.87 ± 0.29
	POST	2.51 ± 0.32	2.07 ± 0.37
IRV/IC (%)	PRE	29 ± 10	23 ± 4
	POST	29 ± 8	25 ± 7

Table 16 – Operational lung volumes during maximal exercise

All values are expressed as mean \pm SD. EILV, end-inspiratory lung volume; FVC, forced vital capacity; EELV, end-expiratory lung volume; IRV, inspiratory reserve volume; IC, inspiratory capacity; V_T, tidal volume: SWIM, swimmers; CON, controls; POST, follow-up visit; PRE, initial visit.



Figure 14 – Absolute operational lung volumes for A) swimmers PRE vs. POST, B) controls PRE vs. POST, C) PRE swimmers vs. controls, and D) POST swimmers vs. controls. As shown in (A), the top lines represent end-inspiratory lung volume (EILV) and the bottom lines end-expiratory lung volume (EELV). Data points are presented as mean \pm SE. For all exercise stages, EILV and EELV were significantly increased and decreased from baseline (BL), respectively. Statistical significance was set at the level of p<0.05.



Figure 15 – Relative operational lung volumes for A) swimmers PRE vs. POST, B) controls PRE vs. POST, C) PRE swimmers vs. controls, and D) POST swimmers vs. controls. Data points are presented as mean \pm SE. EILV/FVC and EELV/FVC were significantly increased and decreased from baseline (BL), respectively, for all stages except PREx100% for EELV. There were no differences between PRE and POST except for 50% for EILV/FVC and 30% for EELV/FVC. Statistical significance was set at the level of p<0.05.



Figure 16 – Mean A) inspiratory reserve volume, B) IRV/FVC, C) inspiratory capacity, and D) IRV/IC during the maximal exercise test. All exercise stages were significantly decreased from baseline (BL) for IRV, IRV/FVC, and IRV/IC and increased for IC (except PREx100%). [#] significant difference between PRE and POST. Statistical significance was set at the level of p<0.05.

3.8.3 Individual and composite maximum expiratory flow-volume curves and tidal flow-volume loops

Figure 17 and Figure 18 present the individual MEFV and FVL for swimmers and controls, respectively. Composite MEFV and FVL are displayed in Figure 19.



Figure 17 – Individual MEFV and FVL for swimmers.



Figure 18 – Individual MEFV and FVL for controls.



Figure 19 – Composite MEFV and FVL for A) swimmers PRE, B) swimmers POST, C) controls PRE, and D) controls POST.

DISCUSSION

4.1 Major findings

The primary purpose of this thesis was to determine if one season of competitive swimming during puberty affected lung development in female competitive swimmers in comparison to healthy controls matched for age, sex, size, and maturity. A secondary purpose was to characterize and compare their ventilatory mechanics during cycling exercise. Thus, the two major findings of this thesis were: first, the swimmers initially had an enhanced pulmonary profile as characterized by larger lung capacities, increased expiratory flows, greater diffusion capacity, and greater indices of respiratory muscle strength. These occurred regardless of the starting age of swimming or years of experience, and did not increase further after one competitive swimming season as compared to healthy controls. Second, the competitive swimmers experienced similar ventilatory constraints while cycling, as evidenced by similar EELV, prevalence and severity of EFL, and utilization of \dot{V}_{ECAP} .

4.1.1 Changes in pulmonary function due to competitive swim training during puberty

The primary finding of this thesis was that one season (seven months) of competitive swimming during puberty did not accentuate growth of the lungs in 11-14 y old female swimmers. The swimmers had bigger lungs than controls of similar age, size, and maturational development at the initial visit, but TLC did not become significantly larger over time.

4.1.1.1 Comparisons of training volume, swimming history, and pulmonary function to previous studies

In this thesis, the swimmers averaged 3.6 ± 0.8 km per swim training session and 5.3 ± 1.6 sessions per week for totals of 9.1 \pm 3.6 h and 19 \pm 8 km per week of swim training. They had a mean 3.2 ± 1.8 y of competitive swimming experience, with a range of starting ages from 6.0 to 10.1 y old. These are congruent with previously reported training volumes and swimming histories of adolescent competitive swimmers, including: Courteix et al.'s 9-10 y old female swimmers (8-12 h and 10-20 km per week) (18), Zinman and Gaultier's 7-13 y old female swimmers (5 h per week and 1.2 ± 0.6 y experience in the youngest 7-8 y old cohort, 12.5 h per week and 3.3 ± 1.7 y experience in the oldest 11-13 y old cohort) (27), Bloomfield et al.'s Australian state finalist 8-12 y old swimmers (average 5 swimming sessions per week) (16), and Baxter-Jones and Helm's 114 8-16 y old swimmers (9-13 h per week) (17). The training volume of the 30 female swimmers (1-4 y experience with starting age ranging from 10-15 y old) from Astrand et al.'s study varied depending on swim club, with 21 females from 3 clubs training 6-20 h and 6-30 km per week and 9 females from the most competitive club swimming upwards of 28 h and 65 km per week (21). Thus, the swimmers in this thesis had comparable swimming histories and training volumes to previous reports of competitive, adolescent female swimmers.

The pulmonary function of the swimmers in this thesis is also in accordance with previous reports of adolescent female swimmers (Table 17). In particular, TLC, RV, RV/TLC, and FVC were nearly identical to the 30 girls studied by Astrand *et al.* who had a comparable age and height (21). The 9 most competitive swimmers in the study by Astrand *et al.* (selected from >600 applicants and included 5 Swedish champions, 3 European record holders, and 2 world record holders) had an average age of ~15.0 y, height of 167.6 ± 6.1 cm, and TLC of 5.37 ± 0.811 (21).

While these champion swimmers are slightly older and bigger, their pulmonary function falls in line with potential future values for the swimmers in this thesis when they reach an equivalent age. Wells *et al.* mixed-longitudinally assessed the Canadian National and Youth National Teams, including sprint, middle-distance, and long-distance swimmers (101). The 12 and 13 y old cohorts of female swimmers had a height (163.3 \pm 7.5 (n=12) and 164.6 \pm 6.1 cm (n=44), respectively), mass (53.2 \pm 6.8 (n=12) and 54.9 \pm 6.3 kg (n=44)), FVC (4.3 \pm 0.58 (n=8) and 4.2 \pm 0.5 1 (n=32)), and hemoglobin (13.6 \pm 0.6 (n=8) and (13.5 \pm 0.9 g·dl⁻¹ (n=34)) that were nearly identical to the swimmers in this thesis. Moreover, at the initial measurement the swimmers in this thesis had a similar TLC, FRC, FRC/TLC, PI_{MAX}, and PE_{MAX} to Zinman and Gaultier's eldest cohort of 15 similarly-aged, but slightly shorter, swimmers with the same amount of competitive swimming experience (27). Lastly, initial and follow-up D_{L,CO} were similar to 12 extensively-trained, comparably-sized adolescent swimmers studied by Yost *et al.* (33). Thus, the swimmers in this thesis had similar pulmonary function to previously reports of competitive, pubescent female swimmers, including the Canadian Youth National and National Teams.

The swimmers in this thesis grew an average of 2.1 cm in height and 350 ml in TLC over the 7-8-month duration, equivalent to 3.4 cm and 560 ml per year, respectively. Changes in TLC of other longitudinal assessments have varied based on age. In 8 9-10 y old female swimmers, Gibbins *et al.* observed an average increase of 2.6 cm in height but only 80 ml in TLC after 6 months of swimming 1000 yards 3-4 times per week (42). Courteix *et al.* examined 5 prepubescent 9-10 y old swimmers before and after one year of intensive training, measuring average increases of 7 cm and 420 ml in height and TLC, respectively (18). Zinman and Gaultier assessed two cohorts before and after one year of training (15). In the 7-9 y old group, seven swimmers had a mean increase in height of 7 cm and TLC of 445 ml; the older cohort (10-12 y old), grew 6.9 cm and 738 ml. The aforementioned 9 most competitive swimmers from Astrand *et al.*'s study grew, on average, 2.4 cm in height and 610 ml in TLC over 2 years (equivalent 1.2 cm and 305 ml over 1 year) of intensive swimming training from ~15 to 17 y old (to note, one swimmer had stopped training by the time of the follow-up measurement) (12). This may reflect that, in females, lung volumes continue to increase even when standing height is approaching or has reached adult values (57, 102-104). Thus, in comparison to other studies which included pre- or peri-pubescent (<15 y old) competitive swimmers, the swimmers in this thesis experienced the greatest change in TLC for the given somatic growth. This validates the thesis design, which focused on the pubertal growth spurt when lung growth is greatest.

In terms of training volume, swimming history, and pulmonary function, the swimmers in this thesis were much like previous reports of competitive adolescent female swimmers. The controls also appeared to have "normal" function, as evidenced by mean %-predicted values ranging between 90-105% for most measures. For example, the %-predicted values for TLC were identical before and after $(94 \pm 7\%)$ and within the limits of normality (85), suggesting normal growth of the lungs. Therefore, both groups appear representative of their respective populations. Additionally, %-predicted values were generally similar at PRE and POST within each group. Thus, discrepancies between the groups in pulmonary function are unlikely to be the result of abnormal results (e.g., the swimmers initially having extraordinarily large lungs compared to other groups of swimmers or the control group having too small of a change in TLC for a given the change in height) and instead signify real physiological differences typical of swimmers vs. non-swimmers.

4.1.1.2 Differences in initial pulmonary function

At the initial visit, the swimmers already had enhanced lung function (i.e., TLC, FVC, FEV₁, PEF, mid-expiratory flows, $D_{L,CO}$, PE_{MAX} , and PI_{MAX}) compared to both reference values and the healthy controls, including individually greater TLC than controls for nearly all heights (Figure 2). Individually, swimmers consistently exceeded their predicted values for initial measurements of TLC (range 100-122%), FVC (106-143%), $D_{L,COC}$ (100-142%), and PE_{MAX} (84-125%). This occurred regardless of the starting age of swimming or training history (i.e., number of years of experience), as shown in Table 8. Moreover, as a group the swimmers had an average %-predicted value \geq 110% for initial TLC, FVC, FEV₁, FEF_{25%}, FEF_{50%}, $D_{L,CO}$, $D_{L,COC}$, and V_A . In comparison to the control group, the swimmers initially had a 20% greater TLC and 25% greater FVC.

Table 18 shows that these %-predicted means are akin to those reported in previous studies of competitive swimmers, including the observations by Astrand *et al.* on 30 adolescent female swimmers (21). Interestingly, %-predicted pulmonary function does not show an increase over time, suggesting that it is relatively constant in competitive female swimmers throughout adolescence and into adulthood. For example, the swimmers in this study and the female members of the 1984 U.S. Olympic swim team (aged 15-23 y) (31) had nearly identical mean %-predicted function for TLC (110 vs. 115%), RV (both 96%), FVC (both 123%), FEV₁ (117 vs. 116%), and $D_{L,CO}$ (121 vs. 127%). The 20-25% larger values compared to the control group are similar to the 20% greater TLC reported by Cordain *et al.* (28) in 11 Division I collegiate swimmers (aged 19.0 \pm 0.6 y) and 20% greater FVC throughout development by Baxter-Jones and Helms (17) in 114 swimmers aged 8-16 y.

Taken altogether, two conclusions can be made. First, the lung function of competitive swimmers is already enhanced by the time they reach puberty, regardless of swimming history. Second, the extent of this enhanced function, relative to either a control group or reference values, is fairly constant throughout development, suggesting that the differences in pulmonary function may be based upon genetic endowments in swimmers that are not accentuated by competitive swim training. Moreover, these findings support the conclusion by Baxter-Jones and Helms that children with endowments favourable to swimming success may select themselves into the sport (17). It is possible that competitive swimming leads to early attainment of peak pulmonary function, but reports of children at the start of their competitive swimming career already having larger lungs may refute this claim (23, 39).

C	Bloomfield et al., 1990* (n=34) [#]	Zinman and Gaultier, 1986 (n=15)	This study PRE (n=11)	Astrand et al., 1963 (n=30)	This study POST (n=11)	Bloomfield et al., 1990* (n=51) ^{##}	Yost et al., 1981* (n=12)
Age (y)	12.2 ± 1.0	12.4 (11.0-13.3)	12.4 ± 0.8	12.9	13.0 ± 0.8	13.1 ± 0.9	14.0 ± 2.4
Height (cm)	152.5 ± 6.0	150 ± 8	161.3 ± 7.9	164.8 ± 6.5	163.4 ± 6.9	158.8 ± 6.7	159.3 ± 11.8
Experience (y)	-	3.7 ± 1.6	$\textbf{3.2} \pm \textbf{1.8}$	1-4 y	-	-	2-12 y
TLC (l)	-	4.58 ± 0.70	$\textbf{4.73} \pm \textbf{0.73}$	4.91 ± 0.81	5.08 ± 0.68	-	-
VC (l)	-	3.40 ± 0.51	$\textbf{3.74} \pm \textbf{0.65}$	-	$\textbf{4.03} \pm \textbf{0.61}$	-	-
FRC (l)	-	2.25 ± 0.41	$\textbf{2.18} \pm \textbf{0.43}$	2.04 ± 0.40	$\textbf{2.40} \pm \textbf{0.39}$	-	-
RV (l)	-	1.19 ± 0.33	0.99 ± 0.16	0.95 ± 0.28	$\textbf{1.04} \pm \textbf{0.19}$	-	-
FRC/TLC	-	0.49 ± 0.03	0.46 ± 0.05	0.42 ± 0.04	$\textbf{0.47} \pm \textbf{0.06}$	-	-
RV/TLC	-	0.25 ± 0.06	0.21 ± 0.03	0.19 ± 0.04	0.21 ± 0.03	-	-
FVC (l)	2.78 ± 0.49	-	$\textbf{3.92} \pm \textbf{0.71}$	4.00 ± 0.67	$\textbf{4.15} \pm \textbf{0.61}$	3.31 ± 0.60	4.12 ± 0.93
$FEV_1 (l \cdot s^{-1})$	2.45 ± 0.46	-	3.34 ± 0.61	-	3.55 ± 0.57	2.91 ± 0.49	-
$D_{L,CO} (ml \cdot min^{-1} \cdot mmHg^{-1})$	-	-	23.29 ± 2.87	-	$\textbf{24.33} \pm \textbf{2.16}$	-	22.40 ± 6.10
PIMAX (cm H2O)	-	85 ± 23	87 ± 26	-	103 ± 22	-	-
PE _{MAX} (cm H ₂ O)	-	110 ± 25	112 ± 17	-	127 ± 17	-	-

Table 17 – Lung function of adolescent swimmers

*males and females. [#]Tanner stage 3. ^{##}Tanner stage 4. TLC, total lung capacity; VC, vital capacity; FRC, functional residual capacity; RV, residual volume; FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; D_{L,CO}, diffusion capacity for carbon monoxide; PI_{MAX}, maximal inspiratory pressure; PE_{MAX}, maximal expiratory pressure.

	Age (y)	N, Sex	TLC	FRC	RV	FVC	FEV_1	PEF	$D_{L,CO}$	$D_{L,CO}\!/V_A$
This study	12.4 ± 0.8	11 F	110 ± 7	102 ± 14	96 ± 13	123 ± 11	117 ± 11	97 ± 9	121 ± 12	101 ± 10
Astrand et al., 1963	12.9	30 F	112 ± 11	110 ± 14	100 ± 22	113 ± 12	115 ± 14	-	-	
This study	13.0 ± 0.8	11 F	112 ± 8	106 ± 14	95 ± 16	125 ± 10	119 ± 10	101 ± 8	122 ± 13	97 ± 9
Zauner and Benson, 1981	13.7 (9-19)	8 M, 7 F	-	-	-	104-109*	-	-	-	-
McKay et al., 1983	14.1 ± 0.9	10 F	-	-	-	118	-	-	-	-
McKay et al., 1983	14.7 ± 0.5	6 M	-	-	-	132	-	-	-	-
Vaccaro et al., 1980	15.1 ± 1.7	12 M	116	109	105	115	110	-	113	-
McKay et al., 1983	17.5 ± 0.6	4 M	-	-	-	130	-	-	-	-
Armour et al., 1993	18 ± 2.4	8 M	145 ± 22	137 ± 23	151 ± 37	149 ± 23	131 ± 16	116 ± 11	117 ± 18	93 ± 15
Bradley et al., 1985	18.4 (15-23)	20 F	115	-	96	123	116	113	127	-
McKay et al., 1983	18.6 ± 1.3	5 F	-	-	-	135		-	-	-
Miller et al., 1989	18-22	22 M	-	-	-	-	-		114 ± 15	-
Bradley et al., 1985	20.3 (17-25)	18 M	119	-	91	127	120	110	139	-
Lazovic-Popovic et al., 2016	20.9 ± 2.4	38 M	-	-	-	115 ± 12	112 ± 10	104 ± 13	-	-

Table 18 – Percent-predicted lung function in competitive swimmers throughout development listed according to average age.

*Range of average %-predicted FVC over 3 consecutive years. TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; D_{L,CO}, diffusion capacity for carbon monoxide; V_A, alveolar volume.

4.1.1.3 Changes in pulmonary function after one season of competitive swimming

Over the study duration, nearly all anthropometric and absolute pulmonary function measures significantly increased from PRE to POST for both swimmers and controls (the exceptions were hemoglobin, FEV₁/FVC, FEF_{50%}, RV/TLC, and diffusion capacity). Both groups had similar amounts of somatic growth in height (mean 2.1 vs. 2.3 cm for swimmers and controls, respectively) and mass (mean 3.4 vs. 3.1 kg), and changes in SMR were similar (p=0.23). Although swimmers had, on average, 800 ml greater TLC at the initial measurement, growth in TLC was comparable between the swimmers (350 ml) and controls (260 ml). This was also the case for almost all spirometry values, including FVC and PEF. While diffusion capacity (D_{L,COC}/V_A), decreases were observed from PRE to POST but no group differences were found at either time point. Thus, the lack of significant interactions between group and time for TLC, spirometry, and diffusion capacity suggests that, contrary to the first hypothesis, competitive swimming during puberty does not accentuate lung development.

Whereas changes in absolute pulmonary function can be ascribed to normal somatic and lung growth, changes in relative (%-predicted) lung function may be indicative of lung growth beyond expected for a given increase in age and height. Since measures of lung volumes and spirometry 'track' (i.e., follow percentile curves) growth charts in healthy children (105), deviations in swimmers may indicate an effect of competitive swimming on lung growth. However, almost all subjects in this thesis had similar %-predicted TLC at PRE and POST as evidenced by the change in %-predicted TLC being $\leq 5\%$ for 9/11 swimmers and 8/10 controls. Moreover, there were almost no significant two-way interactions for any %-predicted measure of lung function and the values were generally similar at PRE and POST. Therefore, the changes in

relative lung function do not suggest an effect of competitive swimming on lung development during puberty.

The individual changes in lung function in swimmers occurred irrespective of the training volume, as no significant correlations were found between training volume (expressed as time (h) or distance (km) per week) and change in any measure of pulmonary function (Table 9). This thesis also quantified time spent performing breath control drills, which included underwater kick, freestyle breathing pattern ("hypoxic training"), snorkel sets, or any other as defined by the swimming coach (listed for each swimmer in Table 25 in Appendix A). On average, only 1.3 ± 1.1 h per week were spent performing these drills. Furthermore, no consistent qualitative association was observed between the amount of time spent performing breath control drills and change in pulmonary function. In fact, some of the swimmers (e.g., S04, S09, S10) who spent minimal training time performing breath control drills (0.25-0.5 h per week) had some of the greatest changes in TLC. Thus, the lack of association between training time and change in pulmonary function suggest that any effect of competitive swimming during puberty is not a dose-response relationship. Moreover, the lack of association between relative change in TLC and daily moderate-vigorous physical activity levels in all of the subjects suggest that growth of the lungs occurs irrespective of one's activity level.

The primary finding that changes in pulmonary function are not accentuated by competitive swimming is supported by three large longitudinal studies of adolescent swimmers. First, it is similar to that of Engstrom *et al.*, who reported that TLC in 29 female swimmers age 9-13 y was initially larger compared to population norms and did not increase further up to age 16 (9). The finding also agrees with the observations of a large, longitudinal study by Baxter-Jones and Helms who found that, after adjusting for height, weight, and pubertal stage, the FVC of 114 8-16 y old
swimmers was $\geq 20\%$ larger than 339 other young athletes at the initial measurement and also did not increase further (17). Lastly, it is in accordance with that of Andrew *et al.*, who observed a greater TLC, VC, maximal mid-expiratory flow, and D_{L,CO} (males only), but not FRC or D_{L,CO}/TLC, across all ages in >70 8-18 y old swimmers compared to non-athletes (11).

However, other longitudinal reports of young swimmers compared to healthy controls have reported contrasting results. First, Gibbins (42) and Vaccaro and Clarke (43) found no difference in lung function before or after 6-7 months of training in 8 9-10 y and 15 9-11 y old swimmers compared to control groups, respectively. Compared to this thesis, the training volume in Gibbins's thesis was relatively low as participants only swam 1000 yards 3-4 times per week. This suggests that the lack of differences in pulmonary function may be explained by participants not qualifying as "competitive swimmers." Moreover, despite mean height increases of 2.7 and 3.2 cm in the swimmers and controls, average TLC increased by only 80 ml in swimmers and did not change in the controls. In fact, one swimmer's TLC is listed as decreasing from 3.769 to 2.956 l. These are unusual results when considering their initial age and change in height (57), raising concerns regarding the validity of their measurements. On the other hand, the 15 swimmers in the study by Vaccaro and Clarke had a similar training volume (3,000-10,000 yards 3-4 times per week) and were tested before and after a similar duration (7 months) as the present study. Yet, they observed a similar FVC between swimmers and controls. This lack of difference may have been related to the controls being, on average, 5 cm taller than the swimmers, although this difference was not statistically significant. Alternatively, the groups may not have been matched for sex (sex distribution of the control group was not stated) or maturity (not measured), both of which can lead differential timing and rates of lung growth during development (48, 49, 56).

Second, significantly increased lung function after training despite no initial differences were found in longitudinal studies of 5 9-10 y old prepubescent female (18) and 95 8-12 y old (26) swimmers. The swimmers observed by Courteix *et al.* were comparable to this thesis with regards to training volume and competitive status, and had a similar amount of somatic and lung growth as the 7-9 y old competitive swimmers in Zinman and Gaultier's one-year longitudinal assessment (15). However, over one year their age- and maturity-matched control group grew 6 cm in height but only 90 ml in TLC; again, this appears abnormally small compared to reference values for their age and somatic growth (57) and may underlie the contrasting results. Bloomfield et al. performed a five-year mixed-longitudinal analysis of 95 8-12 y old competitive swimmers and 102 matched non-competitors, separating the groups based on sex and Tanner maturational stage (26). A qualitatively larger FVC was found in swimmers throughout all maturational stages, and was particularly evident in the male cohort. In fact, they found a statistically significant main effect for swimming whereby FVC was larger in swimmers for both sexes, regardless of maturity, and no statistically significant interactions between swimming and maturational stage - this is in agreement with the results of the present thesis. However, only upon interpreting post-hoc tests did Bloomfield *et al.* state that the qualitative differences in FVC became statistically significant at stage 2 in males and stage 4 in females.

Lastly, Zinman and Gaultier studied 17 7-12 y competitive swimmers and observed a greater TLC before and a larger increase in TLC after one year of training (15). While the greater TLC measured initially is in accordance with this study, the observation of a larger increase in TLC due to swim training is not. Their older cohort (the 10-12 y old swimmers) grew, on average, 6.9 cm in height and 738 ml in TLC. As mentioned previously, when these changes in TLC are expressed relative to height, Zinman and Gaultier's swimmers had lower rates of lung growth

compared to the present thesis. Thus, the differing results may be related to the experimental design. Their control group was analyzed cross-sectionally, and no inferential statistics comparing the swimming and control groups were provided. Although the onset of puberty was assessed, there was no further quantification of maturational age and therefore the extent of maturational development of the swimmers was unknown.

4.1.1.4 Summary of changes in pulmonary function

Overall, the swimmers in this thesis were much like previous reports of competitive adolescent female swimmers in terms of training volume, swimming history, and pulmonary function. Their lung function was already enhanced by the time they reached puberty, regardless of swimming history. The lack of significant interactions between group and time for TLC, spirometric values, diffusion capacity, and maximal static mouth pressures suggests that contrary to the first hypothesis, competitive swimming during puberty does not accentuate lung development. This is supported by three large longitudinal studies of adolescent swimmers (9, 11, 17); contrasting results may be explained by discrepancies in the control group used (18, 42, 43) or experimental design and analysis (15, 16), but not by effects of competitive swimming. Collectively, these findings suggest that the differences in pulmonary function may be based upon genetic endowments in swimmers, but are not accentuated by competitive swim training.

4.1.2 Mechanisms underlying differences in pulmonary function

A thorough review of the development of the respiratory system and its mechanical properties is necessary to speculate why the pulmonary function of the swimmers in this thesis was not affected by intensive swim training. However, anatomical, morphological, and physiological changes of the lungs and chest wall during growth are complex, not completely understood, and difficult to measure in healthy humans. Therefore, it is difficult to make inferences of their numerous interactions into causative mechanisms that determine the static and dynamic properties of the respiratory system and its measurable pulmonary function. It is also important to note that this thesis was not designed to address why differences in pulmonary function existed. Thus, this section will attempt to provide a brief review of developmental processes and speculate how they may be affected by the unique challenges imposed by competitive swimming. Then, the extent to which each measure of pulmonary function may be influenced by competitive swimming will be discussed within the context of the results of this thesis. More comprehensive reviews of respiratory mechanics in children (46), biochemical and structural changes during growth (106), and functional development from gestation to adulthood (107) can be found elsewhere.

4.1.2.1 Development of the respiratory system

Development of the alveoli and the elastic properties of the lung. Alveoli appear around 30 weeks of gestational age (48). Extensive alveolar multiplication from alveolar ducts in the first 2-3 years of life form the bulk of the alveoli (108), then alveolar multiplication and hypertrophy continue throughout childhood and adolescence and into early adulthood (38). This normal development of the lung after birth happens through a feedback loop whereby outward recoil of the growing rib cage mechanically stimulates alveolar growth and then alveolar growth relieves the mechanical tension to allow further expansion of the rib cage (109). There is also an increase in the amount and change in the distribution of elastin, collagen, and smooth muscle during growth (46, 110). Combined with alveolar multiplication, these lead to changes in surface and tissue forces and therefore the elastic properties of the lungs (46, 111); more specifically a progressive increase

in lung elastic recoil pressure ($P_{st}(L)$) (37, 90, 112-114), a greater $P_{st}(L)$ for a given fractional lung volume (i.e., % TLC) (37, 90, 113, 115), and a rightward shift and flattening of the pressurevolume (PV) curve (i.e., changes were more dramatic at larger (~90% TLC) than mid-range (~60% TLC) lung volumes) (37, 90, 113, 115). Thus, although static (C_{st}) (90, 113, 116) and dynamic (C_{dyn}) (90, 117-119) lung compliances increase with growth (because of the increase in anatomical size), C_{st} relative to TLC (90) and FRC (90, 113) ("specific" C_{st}) decrease as a result of stiffer, more elastic lungs (90).

Development of the airways and the flow-resistive properties of the lung. Unlike the alveoli, the airways do not increase in number after birth (120). The airways are formed by ~16 weeks of gestational age (120), and by birth, the conducting regions of the lungs are a miniature version of the adult whereby the conducting airways can only increase in length and diameter during growth (121). There is an increase in anteroposterior and transverse diameters of the trachea (122) and a decrease in specific compliance of the trachea and main-stem bronchi (123). It is likely that the airway walls also undergo morphological changes, as the airway walls are much more compliant in children compared to adults (46). These changes lead to an increase in airway resistance (R_{aw}) from infancy to adulthood (46). The susceptibility of the peripheral airways to dynamic compression during expiration does not change during childhood and adolescence and into adulthood, therefore the trachea or main-stem bronchi remain the location of the flow-limiting segment (112).

Dysanapsis. The differing patterns of and disproportionate growth between airways and alveoli is termed *dysanapsis* (dys = unequal, anapitxy = growth) (69). One measure that quantifies dysanapsis is specific airway conductance (sG_{aw}), calculated as the ratio of airway conductance

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(sensitive to airway size) to the lung volume at which it is measured (sensitive to lung size) (46). Decreases in sG_{aw} reflect periods when alveolar growth predominates, whereas increases suggest relatively more airway growth. The ratio is highest during infancy, then from birth until 5 years old, it decreases when alveolar growth is greatest (125-127). During childhood and early adolescence, sG_{aw} remains fairly constant or slightly decreases (46); after puberty, increases in sG_{aw} may be due to late airway growth that stabilizes sG_{aw} into adulthood (125). Thus, dysanapsis is likely to be most prominent when lung growth is greatest in infancy and early childhood (37), and continues into puberty when PGV occur.

Puberty, also known as the adolescent growth spurt, occurs as early as age 7 y in girls and 9 y in boys (75). Lung growth during this stage can be characterized by four phases: (1) prepubescent steady-state lung growth continuing from childhood; (2) maximal acceleration of lung growth at the onset of puberty; (3) postpubescent growth at progressively slower rates; and (4) cessation of lung growth during post-adolescence and early adulthood (56). Mathematical modelling of longitudinal changes in FEV₁ (sensitive to airway size) and FVC (sensitive to lung volume) showed that growth velocities for FVC were consistently greater than FEV₁ throughout puberty until 13-14 years of age (72). Moreover, PGV for lung volume preceded those for flow by one year (48). The relationship between volume and flow can be quantified by the dysanapsis ratio, which, as mentioned, is the ratio of mid-expiratory flow corrected for P_{st}(L)₅₀ (index of airway size) to VC (index of lung size) (89). From late childhood and through adolescence the ratios steadily decrease, suggesting disproportionate growth before, during, and after the adolescent growth spurt (57). However, caution is warranted when assessing changes in DR over time, as interpretation of decreases in DR may reflect isotropic (i.e., proportional) or dysanaptic growth depending on the assumption of turbulent flow during the expiratory maneuver (46, 89). A recent

longitudinal study accounted for this assumption and showed that an estimated DR (using $Pst(L)_{50}$ calculated from a regression equation) decreased from prepubescence to postpubescence in a manner that suggested dysanaptic growth indeed occurs during puberty (128).

Development of the chest wall. The main static change of the respiratory system during development is an increased outward recoil of the chest wall (129). Infants have a chest wall compliance (C_w) that is nearly three times greater than lung compliance (130). However, within the first few years of life, the shape of the thorax changes as the ribs, which extend horizontally at birth, tend downwards and the diaphragm becomes less horizontal (131). Moreover, the ribs mineralize, the gravitational effect of the abdominal contents increases, and the respiratory musculature develops. These changes, especially within the first 2 y of life, cause a substantial reduction in specific C_w (130) such that, throughout the rest of development (132) and into adulthood (133), the contributions of C_{st} and C_w to total respiratory system compliance (C_{RS}) are nearly equal. As with C_{st}, C_w and C_{RS} increase with growth (116, 132); but when expressed relative to lung volume, specific C_w (46, 129) and specific C_{RS} (132, 134) decrease. Further changes in size and shape of the chest wall occur during puberty, as the thorax elongates due to relatively greater thoracic height growth (135). Measurements of sternal length, chest width, and chest depth and estimations of chest wall surface area and thoracic volume index have been shown to increase before and during puberty (102, 136, 137). Furthermore, chest and lung widths reach their PGV simultaneously during puberty, while lung length and chest depth reach PGV six months later (73). Thus, growth of the thorax and, due to the feedback loop, lungs occur until their dimensions stabilize when somatic maturity is reached and epiphyses close (109).

4.1.2.2 Total lung capacity and chest wall size.

The tight coupling between chest wall and lung growth has important implications on the development of pulmonary function. Static lung volumes are determined by lung size, the ability of the inspiratory and expiratory muscles to generate pressure, antagonistic muscle activity, and the elastic recoil pressures of the lungs and chest wall (138). Of these, lung size, inspiratory muscle force generation, and the inwards elastic recoils of the lungs and chest wall interact to determine TLC (139). During childhood, increases in TLC are due primarily to somatic growth of the chest wall (140) and, consequentially, lung size.

Thus, because TLC is primarily determined by lung size and lung size is directly related to chest wall size, it is likely that the greater TLC of the swimmers in this study were related to larger chests. Although not measured here, chest wall dimensions were observed to be larger in competitive swimmers (16, 27, 29). Zinman and Gaultier measured the anteroposterior diameter (i.e., chest depth) at the sternal angle and transthoracic diameter (i.e., chest width) at the xyphoid process at TLC, FRC, and RV, as well as sternal length (27). From these dimensions, they also estimated chest wall surface area at FRC and TLC. They found the dimensions and surface area to be larger in all three cohorts of female competitive swimmers (7-8, 9-10, and 11-13 y old), with all differences becoming statistically significant in the 9-10 and 11-13 y old swimmers. Armour et al. performed the same measurements in 24 collegiate male swimmers, runners, and controls (8) each), finding a greater chest width at TLC (but not chest depth) and chest wall surface area at TLC, FRC, and RV in 8 swimmers compared to 8 runners (differences between swimmers and 8 controls did not reach statistical significance) (29). They also found a significant correlation between increased chest wall surface area and TLC, whereby the swimmers with the largest surface area had the greatest TLC. Lastly, Bloomfield *et al.* measured chest depth and width in 95 8-12 y

old competitive swimmers and 102 matched non-competitors (16). They found a statistically significant main effect for both depth and girth in males and females. Post-hoc testing suggested that males and females had significantly greater chest depth from maturational stage 4, while chest width was greater at each stage in males. Interestingly, significant differences in FVC between swimmers and non-competitors nearly mirrored those of chest dimensions, as FVC was greater in male swimmers from stage 2 onwards and in female swimmers from stage 4 onwards. The observation that greater chest dimensions in swimmers as young as 7-8 y old persisted into young adulthood and were correlated with larger TLC provide further evidence that children with larger chests, and thus lungs, are likely selected into swimming.

4.1.2.3 Total lung capacity, hypoxia, and induced postnatal lung growth

Armour *et al.* suggested that a periodic hypoxic stimulus and enhanced growth hormone response while swimming, specifically during the adolescent growth spurt, may elicit accentuated alveolar hyperplasia in response to the enlarging chest cavity (29). In other words, intermittent hypoxia may be a mediator of greater lung growth in developing swimmers. However, it is not clear if there is a connection between the mechanisms of induced postnatal lung growth (3, 109), including hypoxia, and the unique stressors of intense swim training.

Hypoxia. Exposure to chronic hypoxia is one of the main stimulants of induced postnatal lung growth (109). As explained earlier, growth of the lung occurs via a feedback loop until epiphyseal union of the ribs occurs, at which point the size of the lungs is matched to and limited by the size of the thorax (109). This is critical to speculating the response to chronic hypoxia, because its effect may be dose-dependent whereby moderate levels stimulate lung growth but severe levels retard rib cage growth (109). Comparisons of animal models at different altitudes

effectively describe this interplay (109). At moderate altitudes, hypoxia primarily stimulates lung growth, with passive rib cage expansion and lowering of the diaphragm providing the space for more alveoli. The balance is more delicate at higher altitudes, where the stimulation of lung growth becomes diminished over time by the retardation of rib cage growth. Extreme altitudes illustrate the other end of the spectrum, as severe hypoxia stunts rib cage growth, which prevents absolute increases in lung volume. The effect of hypoxia may also be dependent upon time. Exposure too early blunts lung development, whereas exposure during early and later stages of maturation cause alveolar hyperplasia and hypertrophy, respectively (109). Thus, for hypoxia-induced accentuated lung growth to occur in swimmers, there must be a chronic hypoxic stimulus of appropriate dose during development.

Whether this occurs in swimmers likely depends on stressors unique to swimming. First, swimming is performed in the supine or prone position while partially or fully submerged in water. Second, a swimmer's breathing pattern is not only a result of physiological need, but also of stroke rate and biomechanics (i.e., entrainment). Third, swim training often involves breath control drills, including "hypoxic training", and sprint swimming where breathing frequency is reduced. Lastly, intense swim training often begins at a very young age, as early as 5 years old.

Immersion and the supine and prone positions. Increased hydrostatic pressure and decreased gravitational pull during upright immersion causes the redistribution of ~700 mL of blood into the intrathoracic vascular bed (141) and elevates the diaphragm (142). These alterations lead to major changes in ventilatory mechanics and hemodynamics. Central vascular engorgement (CVE) contributes to increased lung recoil at high lung volumes and decreased lung recoil at low lung volumes (143), shifting the transpulmonary pressure-volume curve to the right by ~16 cm H_2O (144). Moreover, hydrostatic forces oppose the action of the inspiratory muscles, thereby

decreasing the maximum static transpulmonary pressure that can be generated (143). The increased lung recoil and decreased static pressure generated cause a decreased TLC while immersed (143). At RV, the decreased lung recoil but increased hydrostatic pressure assisting expiration may oppose each other and cause no change (145, 146), although some have suggested that CVE leading to increased lung stiffness may increase RV (147). Since VC is the difference between TLC and RV, a decreased TLC and relatively unchanged RV would cause VC to decrease (146) by approximately 10% (148, 149). Furthermore, elevation of the diaphragm to near-resting EELV decreases ERV and therefore FRC (142). Since tidal volume is unchanged (144), lower operating lung volumes decrease airway diameter, increase airway resistance (142, 150), and thereby increases the elastic and overall WOB by 60% (144). Moreover, ERV does not change with increases in V_T, so increasing V_T occurs solely by utilizing IRV (148). Hemodynamically, 25% of the blood displaced from the legs to the thorax upon immersion is located in the heart, increasing stroke volume (SV) and thus cardiac output (Q) by 20-40% (151). The mean pulmonary artery pressure also increases (141), leading to a greater pulmonary capillary blood volume (V_c) and diffusion capacity (152). Moreover, the increased Q and pulmonary artery pressure may create a more homogenous distribution of pulmonary perfusion and therefore ventilation-perfusion ratios (V_A/Q) throughout the lung (153). Cumulatively, these effects may reduce the alveolar-arterial difference (A-aDO₂) and increase arterial oxygen tension (P_aO_2) (153).

Many of the studies on immersion were conducted in the seated, upright position, whereas swimming provides the additional stimulus of being performed while either supine (backstroke) or prone (all other strokes). However, evidence of changes in ventilatory mechanics and hemodynamics due to the supine or prone position suggests the changes are similar to those due to immersion. Like immersion, the supine position is also associated with a redistribution of blood to the thorax that increases SV and Q and decreases A-aDO₂ (154). The greater amount of blood in the lungs may encroach upon pulmonary air space to decrease TLC, and therefore VC, while FRC and ERV are reduced due to the effects of gravity and diaphragm elevation (129, 155). Diffusion capacity increases because of greater V_c and improved distribution of pulmonary perfusion (156). These effects are nearly identical in the prone position (155-158), where \dot{V}_A/\dot{Q} matching is even better (159). Thus, both immersion and the transition to the supine or prone position are associated with changes in blood volume and diaphragm level; therefore, combining the two conditions may not be additive (151). Deroanne et al. measured compliance and conductance in three subjects while out of the water, immersed in three body positions (standing, supine, and prone), and swimming breaststroke and backstroke (149). Compared to measurements out of water, they found compliance to be lower while supine, standing, and during backstroke swimming. They also observed an increased airway resistance in all body positions and swimming strokes. They associated the mechanical changes with both immersion and changes in body position, clearly illustrating that the two combine to constrain the respiratory system while swimming.

Breathing pattern and the ventilatory response to swimming. Unlike other forms of endurance exercise, the ventilatory pattern while swimming is not solely dictated by physiological need. The "obligatory, controlled frequency" breathing of swimmers is influenced by the stroke rhythm and its associated biomechanical events (36). As a result, rapid inspiration occurs above water while a slow, controlled expiration occurs below the surface (160). The interpretation of how these breathing patterns affect the ventilatory and metabolic responses while swimming is affected by the conditions in which swim testing occurs. First, the necessary use of a breathing valve connected to a snorkel to measure metabolic and ventilatory variables enables free

respiration and inherently alters the respiratory pattern (160). The impact may be dependent on swimming intensity, as it was reported that swimmers adhere to swimming stroke breathing patterns at low intensities but breathe more freely at high intensities (161). Second, maximal tests may be performed in a flume, using a tethered pulley-weight system, or while free swimming. Differences in $\dot{V}O_{2MAX}$ but not ventilatory parameters have been noted between free and tethered swimming in highly trained swimmers; however, this may have been due to training-induced improvements in $\dot{V}O_{2MAX}$ between measurements (162). Others have found identical values in $\dot{V}O_{2MAX}$ (163) and \dot{Q} (164) between the modalities in elite swimmers and all respiratory variables in well-trained swimmers (165), Lastly, tests can be performed during breaststroke, backstroke, or front crawl swimming. However, all appear to attain a similar $\dot{V}O_{2MAX}$ (166). Thus, these suggest that the different modes of testing (167) and types of swimming strokes (166) produce similar physiological responses.

Highly trained swimmers performing maximal running and swimming tests have been shown to utilize similar (161, 162) or even increased f_B (168) while swimming. Conversely, V_T is lower, potentially due to the aforementioned effects of hydrostatic pressure and increased flow resistance as well as movement of the chest and involvement of the respiratory muscles in the arm stroke (162, 168). Therefore, at maximal swimming, elite swimmers have a similar (168) or lower \dot{V}_E (162, 163). During both submaximal and maximal swimming and running, elite swimmers have been shown to relatively hypoventilate, as evidenced by high estimated arterial and venous CO₂ contents and very low ventilatory equivalents (168). In other words, swimmers may ventilate normally, instead of the hyperventilatory response often seen with other athletes, by having a higher alveolar ventilation per breath (169). This does not appear to have a major effect on the $\dot{V}O_{2MAX}$ of elite swimmers, as similar (162, 168) or only slightly lower (163) values have been reported. Moreover, despite a lower maximum HR (162, 163, 168), a greater SV maintains the same \dot{Q} and A-aDO₂ during swimming and running, likely a result of improved venous return and more effective muscle pump action in the arms, shoulders, and chest (168). Arterial oxygen saturation (S_aO₂) is also maintained above 95% (170). In non-elite or recreational swimmers the cardiorespiratory responses may be attenuated due to a lower training status (171), as studies have shown a decreased V_T(171), *f*_B (171), \dot{V}_E (168), $\dot{V}O_{2MAX}$ (162, 168, 171, 172), \dot{Q} (168, 171), and SV (168) during maximal swimming compared to running. However, P_aO₂, arterial carbon dioxide tension (P_aCO₂), and S_aO₂ were similar between maximal swimming and running (171), potentially due to the improved extraction of alveolar O₂ as demonstrated by a decreased A-aDO₂ (168, 171).

Thus, if immersion, body position, and the obligatory breathing pattern of swimming impose restrictions on the respiratory response to maximal swimming, the cardiovascular system appears to be able to compensate by maintaining Q and, more importantly, S_aO_2 (167, 169). Slight decrements in $\dot{V}O_{2MAX}$ can be explained by less muscle mass being involved, primarily a result of minimal muscular work being necessary to support the body in the water (171). Since hypoxemia is avoided, it appears unlikely that the stressors of normal swimming create a hypoxic stimulus for postnatal lung growth.

Controlled frequency breathing drills and sprint swimming. However, controlled frequency breathing (CFB) drills, also know as "hypoxic training," performed as part of an intense swim training program were proposed to create an intermittent hypoxic stimulus (173). The theory was that swimmers could become hypoxemic, increase the oxygen deficit, and enhance the anaerobic response by increasing the number of arm strokes between breaths (e.g., breathing every 5th, 6th, etc., arm stroke instead of every 2nd or 3rd), (173). Traditionally, this voluntary

hypoventilation is performed using the "inhale-hold" technique whereby a rapid, large inhalation (to near-TLC) is followed by a short breath-hold (5 (174) to ~8 (175) seconds) and rapid expiration (174). However, there is no evidence to suggest this form of CFB improves maximal aerobic or anaerobic power output (169), as investigations of CFB during submaximal (175-179) and maximal (180) swimming have observed blood lactate concentrations to be similar (175, 177-180) or reduced (178). Moreover, the decreased \dot{V}_E (175-178, 180) and lower alveolar PO₂ (176-178) caused by the lower breathing frequency is compensated for by a significantly increased V_T (175-177, 180) and lower F_EO₂ (175, 176, 178, 180), the latter elucidating that a higher O₂ extraction at the alveolar-arterial membrane compensates for the hypoventilation (175, 176, 178). Thus, this traditional "inhale-hold" method of CFB was shown to have minimal effects on arterial oxygen saturation (174, 176) and is again unlikely to cause hypoxemia.

A recently proposed "exhale-hold" form of CFB while swimming also appears possible for stimulating intermittent hypoxia (174). Whereas the traditional "inhale-hold" breath is held at a high lung volume near TLC, the proposed "exhale-hold" breath is held at a low lung volume at or below FRC (174). This is accomplished by a quick inspiration to near-TLC and expiration back down to near-FRC, a breath-hold for 4-5 s, followed by a complete exhalation to near-RV before the next rapid inspiration (174). Woorons *et al.* compared the two techniques with normal breathing by continuously measuring S_aO_2 via pulse oximetry at the forehead and breath-by-breath metabolics during 10 consecutive 50 m intervals at near-maximal swimming speeds (174). While S_aO_2 during normal breathing averaged 98% and was \geq 94% for most of the "inhale-hold" intervals, S_aO_2 averaged 89% during the "exhale-hold" intervals and 87% at the end. The authors suggested that the severe hypoxemia observed during "exhale-hold" but not "inhale-hold" was partly due to worsened pulmonary gas exchange, previously demonstrated by an increased A-aDO₂ during

"exhale-hold" exercise (181, 182). This is likely related to lung volume, which has been stated to be the most important determinant of hypoxemia during apnea (183). During apnea at high lung volumes, \dot{V}_A/\dot{Q} homogeneity may minimize arterial desaturation; conversely, apnea at low lung volumes is associated with airway closure and a heterogeneous distribution of \dot{V}_A/\dot{Q} , increasing A-aDO₂ and arterial desaturation (183). Toubekis *et al.* also observed a decreased S_aO₂ compared to normal breathing during "exhale-hold" CFB intervals of 75-400 m at submaximal intensities (184). However, even though S_aO₂ decreased to as low as 78%, on average, at the end of some of the intervals, S_aO₂ recovered to baseline values within 60 s for all distances.

Regardless of whether the "inhale-hold" or "exhale-hold" form of CFB is performed, alveolar CO₂ tension is also increased (176-178, 184). This creates such a strong stimulus to breathe that numerous authors mentioned that some subjects were unable to complete trials when breathing rate was most restricted (e.g., breathing every 8-10th arm stroke) (177, 180). Therefore, CFB may provide a form of "hypercapnic training" (185) that can improve tolerance to hypercapnia (176). This is important because swimming during both "inhale-hold" and "exhalehold" CFB was also shown to decrease VO2 and VCO2 at a given intensity compared to normal breathing (174, 175, 177, 178, 180). The reduced level of metabolism may be related to the decreased \dot{V}_E , as the WOB and therefore respiratory muscle $\dot{V}O_2$ may be lower (174). Alternatively, it may indicate a beneficial biomechanical effect with reduced breathing whereby drag is decreased due to less head movement or better body position in the water (167). Therefore, increased hypercapnic tolerance could be advantageous during sprint swimming, as the ability to increase the apneic period would allow some swimmers to breathe minimally and improve movement economy. For example, some swimmers may breathe only 1-2 times during a 50-m race (180). These longer apneic periods may cause intermittent hypoxemia similar to that observed by Matheson and McKenzie (186). Using arterial lines, they directly measured a significant drop in P_aO_2 and S_aO_2 with repeated 15-s breath-holds during very intense cycling exercise. Miyasaka *et al.* observed a 6-14% drop in S_aO_2 in three male swimmers performing 100 m sprints (187). In both reports, however, S_aO_2 recovered quickly upon exercise cessation when normal breathing was resumed.

Taken altogether, the traditional "inhale-hold" form of CFB has not been shown to decrease S_aO_2 , as the apneic period may not be long enough to cause hypoxemia. While the "exhale-hold" technique was demonstrated to alter S_aO_2 , it is not clear if this is a commonly performed drill. Both techniques may improve hypercapnic tolerance, which may be beneficial during sprint swimming when a drastically reduced breathing frequency causing hypoxemia may also have biomechanical advantages. However, any resultant hypoxemia from "exhale-hold" CFB or sprint swimming is only short-lasting, as S_aO_2 quickly recovers once the drill or sprint is finished. Therefore, this hypoxemic exposure is so short that it appears unlikely to provide the chronic hypoxic stimulus necessary for lung growth.

Age of onset of training. Another unique aspect of competitive swimming is the very young age at which some athletes start training. Coaches of elite swimmers have suggested that intensive swim training programs should begin as early as 5 years old (17). This means that young, competitive swimmers are exposed to the stresses of immersion, the prone and supine positions, and the obligatory breathing pattern at an age when the lung is rapidly developing. Between 1 month and 7 y there is a 13-fold increase in lung volume (37) and an additional 3-fold increase in lung volumes from age 7 to the cessation of lung growth (37, 38). If the growing respiratory system is sensitive to swimming, then swimming during these critical periods of maximal growth are likely to elicit the greatest effects. Such an age-dependent adaptation of the growing lung has been

reported in rats (188). At the end of a one-month training period when 2-month-old rats were exposed to swimming five days per week, they had significantly greater alveolar densities and surface area-to-lung volume ratios than the non-exercised rats, suggesting alveolar proliferation. Conversely, the author reported that these differences were not found when 3-month-old rats underwent the same swimming protocol.

However, as discussed earlier, immersion, the prone and supine positions, and the obligatory breathing pattern have not been associated with hypoxemia during swimming. Moreover, it is unlikely that young swimmers spend extensive amounts of time performing CFB drills or sprinting with reduced breathing frequencies during the primary years of lung growth. Since extended breath-holds during "hypoxic training" are documented to nearly cause drowning (189), concerns of improper technique leading to shallow water blackout in youth swimmers may limit the amount of time coaches choose to spend on CFB drills. In this thesis, the time spent performing breath-control drills was quantified. These consisted of underwater (dolphin or breast) kick, CFB or freestyle breathing pattern, snorkel sets, or sprints performed with minimal breathing. Of the total weekly swimming time $(9.1 \pm 3.6 \text{ h})$, breath control drills were only a small portion of this $(1.3 \pm 1.1 \text{ h})$. Underwater kick was the most common, whereas only three out of 11 swimmers performed FSB and one reported sprints with minimal breathing. Comparisons of the %-predicted TLC and time spent per week performing breath-control drills elucidated no qualitative relationships between them. The four swimmers with the largest %-predicted TLC performed 2 h (117%-predicted TLC), 1 h (122%), 2 h (117%), and 0.25 h (113%), whereas the swimmer who spent the most time doing breath-control drills had the lowest %-predicted TLC (100%). This raises further doubts about the possible connection between intermittent hypoxia and enhanced lung growth in young swimmers.

Other stimulants inducing postnatal lung growth. Other stimulants of postnatal lung growth include increased parenchymal or vascular mechanical strain and hormonal mediators (109), but these are poorly understood and therefore interactions with the stressors of swimming are purely speculative. One can speculate that the ventilatory demand of thousands of hours spent intensely swim training during growth may provide additional mechanical stress on the feedback loop, accelerating or increasing thoracic and lung growths, or affect the remodelling and expansion of elastin during lung growth, altering the elastic recoil of the lungs. However, there is no convincing evidence that intense exercise affects lung structure (3). If intense exercise influences lung growth, elite athletes from land-based endurance sports would have increased pulmonary function as well. Moreover, without measurements of operating lung volumes or estimations of WOB during swimming, it is impossible to determine if the ventilatory demand differs in young swimmers versus other endurance athletes. In terms of vascular mechanical strain, since immersion and the prone position increase blood volume in the thorax and pulmonary artery perfusion pressure while swimming, and Q is maintained, one can speculate that these may increase chronic capillary distension and shear forces that could stimulate lung growth or affect protein distribution. Lastly, it has been suggested that increased release of exercise-induced growth hormone (GH), due to transient hypoxia and greater arm exercise while swimming, may stimulate lung growth (29). Two arguments counter this possibility. First, GH may not directly induce lung growth, but instead modulate the response to mechanical strain or hypoxia (109). As thoroughly discussed, it has not been shown if either of these responses occurs. Second, greater circulating GH increases whole body growth homogenously until epiphyseal closure (190); therefore, one would expect swimmers to have enhanced growth of height and weight as well, which does not appear to be the case (8).

Summary. In conclusion, there is a dose- and time-dependency of chronic hypoxia as a stimulus for induced postnatal lung growth. While swimming provides a unique environment where immersion, the prone and supine positions, and an obligatory breathing pattern all stress the respiratory system, there is no unequivocal evidence that they cause hypoxemia. Certain forms of controlled frequency breathing drills and sprint swimming likely cause hypoxemia, but exposure is acute and recovery to normal values occurs rapidly. Moreover, the prevalence of these drills in a young swimmer's training program varies depending on the coach, and some swimmers who spent minimal time performing breath-control drills still have large lungs for their age and size. Therefore, young swimmers do not appear to be exposed to either the dose of or time spent in hypoxemia necessary for hypoxia-induced lung growth. Lastly, no other form of induced postnatal lung growth appears likely to induce substantial lung growth in young swimmers. Thus, given the present evidence, intermittent hypoxia or any other form of induced postnatal lung growth stimulus are unlikely to explain the larger TLC of swimmers in this study.

4.1.2.4 Total lung capacity and respiratory musculature.

Stronger inspiratory muscles may have also contributed to the larger TLC. Maximal static pressures provide an indirect index of respiratory muscle strength (191), and both PI_{MAX} and PE_{MAX} were greater in swimmers in this study. However, Leith and Bradley measured a 55% increase in maximal static pressures but only 4-5% increase in TLC and VC after 5 weeks of respiratory muscle training in 4 subjects (192). While the increases in TLC and VC were statistically significant, they concluded that increasing respiratory muscle strength only had a limited, ceiling effect on TLC because respiratory system compliance is low at TLC and inspiratory muscle force-length relationships become even more strained with modest increases in

volume above TLC. Fanta *et al.* also observed a slight increase in TLC and VC in 8 subjects performing 6 weeks of inspiratory muscle training by inspiring to TLC, breath-holding at TLC for 10 seconds, then exhaling back to FRC (193). However, PI_{MAX} and PE_{MAX} were unchanged in their subjects. Instead, subjects generated an additional 27 ± 8 cm H₂O of inspiratory pressure at their initial TLC (compared to zero before training). Because there were no changes in FRC or RV, and therefore lung elasticity, the authors suggested that greater maximal shortening of the inspiratory muscles may have occurred. This could have resulted from either the force-length relationship of the respiratory muscles shifting to the left due to a decrease in the number of sarcomeres in series, or the inspiratory muscles becoming stronger and producing greater forces at shorter sarcomere lengths near TLC.

These findings may translate to swimmers because, as mentioned earlier, immersion and the prone position constrain lung volumes and increase airway resistance, providing a potential inspiratory muscle conditioning stimulus. The hypothesized respiratory muscle adaptations may explain the small (<10%) but statistically significant increases in TLC and FVC despite large increases in PI_{MAX} (20-50%) after 12 weeks of either swim training or swim and inspiratory muscle training in collegiate swimmers (14, 19). However, swimmers reportedly have a similar maximal static inspiratory mouth pressure compared to controls (14, 27-29), and Zinman and Gaultier observed no change in PI_{MAX} despite a significantly increased TLC after one year of training in 17 7-12 y old swimmers (15). Estimates of respiratory muscle force, which reflect the larger chest surface area over which swimmers must dissipate pressures, were shown to be greater in 7-10 y old female swimmers (27) but not collegiate male swimmers (29). Measurements of operating lung volumes and WOB while swimming are needed to confirm an inspiratory muscle conditioning stimulus, and at present there is no evidence that swimmers routinely inspire to TLC while

immersed in water. Improvements in quantifying respiratory muscle strength may also provide a better understanding of changes in respiratory musculature with swim training. Ultimately, while respiratory muscle changes may have contributed to the larger lungs of the swimmers in the present study, the contribution is likely to be small and could not account for the entire 20% difference in TLC between the swimmers and controls.

4.1.2.5 Summary on total lung capacity

All published studies assessing TLC in competitive young swimmers have measured greater capacities compared to controls (11, 12, 14, 15, 18, 19, 22, 27-29) or predicted values (9, 21, 23, 24, 31). Only the unpublished thesis by Gibbins found a similar TLC between swimmers and controls, and it is important to note the low training stimulus (1000 yards per session, 3-4 sessions per week) and short time period (6 months) of his investigation (42). As stated earlier, lung size, inspiratory muscle force generation, and the inwards elastic recoils of the lungs and chest wall interact to determine TLC. Any contributions from changes in respiratory musculature are likely to be small and could not account for the entire 20% difference in TLC between the swimmers and controls. Given the evidence, an intermittent hypoxic stimulus or alternative form of induced postnatal lung growth is unlikely to contribute to a larger lung size that explains the greater TLC. Moreover, at present, there is no clear indication that the lungs of swimmers have different mechanical properties compared to healthy controls. Therefore, given the tight coupling between chest wall and lung sizes, and that increases in TLC during childhood are due primarily to somatic growth of the chest wall, it is likely that the greater TLC of the swimmers in this thesis were related to larger chests. The facts that greater chest dimensions were observed in swimmers as young as 7-8 y old, persist into young adulthood, and are correlated with larger TLC also provide further evidence that children with larger chests and lungs likely self-select into swimming. Lastly, the tight coupling between chest wall and lung growths could explain the conclusion by Armour *et al.* that an increased alveolar number, not alveolar distensibility, may have been associated with the physically larger chests of swimmers and could explain the greater TLC (29). It is possible that the larger chests of swimmers stimulated an increased rate of alveolar hyperplasia, as per the aforementioned feedback loop, leading to a greater number of alveoli.

4.1.2.6 Functional residual capacity.

Functional residual capacity is the volume remaining in the lungs at the end of a normal, resting expiration. In infants it is determined dynamically; during development, FRC becomes the static passive balance of lung recoil forces inward and chest wall recoil forces outward, potentially a result of the stiffening chest wall (130). Since all lung volumes increase during development due to the growth in lung size, the ratios of lung volumes relative to TLC are useful for assessing the relative contributions of the other determinants. The ratio of FRC to TLC (FRC/TLC) is commonly reported to be independent of height and age, with most reference values ranging from 0.405 to 0.50 (194). However, it was suggested that FRC/TLC may increase slightly during development due to a greater increase in chest wall recoil outwards than lung elastic recoil inwards at FRC (37). This is indicated by some studies reporting a slight but significant increase in FRC/TLC with increasing age (124, 195). Thus, FRC increases during development primarily due to the growing lungs; any additional increases are marginal, due to a slightly greater change in chest wall recoil outwards.

In this study, FRC was similar between the two groups, although only the swimmers increased significantly from PRE to POST (the difference in mean FRC between the groups was

only 190 ml at the follow-up). This result is somewhat surprising, as the swimmers had a significantly larger TLC and therefore one would expect a larger FRC as well. Moreover, previous studies of growing and young adult swimmers reported a larger FRC compared to controls (14, 22, 23, 27-29) and predicted values (9, 10, 12, 24, 31). Conversely, Andrew *et al.* did not observe a distinct difference in FRC compared to controls, but they noted that there was considerable variability in FRC amongst swimmers (11). While Courteix *et al.* observed no initial differences between 5 9-10 y old female swimmers and 11 matched controls, FRC was larger in swimmers after 1 year (18). As mentioned earlier, the control group had an abnormally small increase in TLC despite growing, on average, 6 cm in height. This may also explain the difference in FRC at their follow-up measurement, as FRC did not change in the control group. A similar explanation may underlie the surprising result in this study.

The mean FRC/TLC for the swimmers was 0.46 ± 0.05 (PRE) and 0.47 ± 0.06 (POST). These are comparable to the 0.48 ± 0.07 , 0.46 ± 0.05 , and 0.49 ± 0.03 reported by Zinman and Gaultier in 7-8, 9-10, and 11-13 y old female swimmers (27), and 0.47 ± 0.02 and 0.49 ± 0.04 observed by Courteix *et al.* in 9-10 y old female swimmers before and after 1 year of intense training (18). Therefore, the mean FRC/TLC for the swimmers is comparable to previous reports and within the range of previously stated reference values. Whereas others found no differences between swimmers and controls for FRC/TLC (18, 27), in this study the controls had a larger FRC/TLC at both time points. Their initial mean FRC/TLC of 0.56 ± 0.04 appears remarkably high, and it is unusual that their FRC did not change from PRE to POST despite both somatic and lung growth. Thus, the significant interactions for FRC and FRC/TLC were likely due to abnormalities in the control group and not an effect of swimming.

If the difference in FRC/TLC was due to swimmers having a smaller FRC relative to TLC, and assuming the greater TLC was primarily due to larger lungs (i.e., a greater number of alveoli) and not a greater ability to distend the existing alveoli, then this would necessitate that the swimmers had either an increased lung recoil inwards or decreased chest wall recoil outwards at FRC. However, Armour et al. found no difference in pressure-volume curves between male collegiate swimmers, runners, and controls when lung elastic static recoil pressures were expressed relative to % TLC (29). Moreover, at present there is nothing to indicate that swimming alters the elastin and collagen distribution in the lung. In terms of chest wall mechanics, as discussed earlier the key contributors to increasing outward recoil during development are changes in chest wall shape, increased respiratory muscle strength, and rib mineralization. Zinman and Gaultier measured larger chest wall dimensions in 7-13 y old competitive female swimmers compared to healthy controls; however, growth in each dimension was proportional and the overall configuration was similar between the groups (27). One can speculate that a greater respiratory muscle mass may decrease chest wall recoil outwards at FRC; however, increased respiratory muscle strength and resting tone would have the opposite effect on chest wall recoil. Hypothetically, these opposing effects would cancel each other out. Lastly, bone mass and size increase with weight-bearing exercise due to increasing muscle size and other loading factors (196, 197). This is particularly important during puberty, when augmented bone mineralization is favourable and physical activity, specifically weight-bearing exercise, can have a critical effect (198). Even though swimming is non-weight-bearing, a recent systematic analysis suggested that children and adolescent swimmers have a similar bone mineral density as sedentary controls (199). Thus, the conclusion that differences in FRC were not the result of swimming is further supported by the lack of evidence supporting the idea that long-term, intense swim training affects the static mechanical properties of the respiratory system.

4.1.2.7 Residual volume.

Residual volume is the volume of air remaining in the lungs after a maximal expiration. In young subjects, RV is determined by the balance between expiratory muscle strength and opposing chest and lung elastic recoil forces (200), antagonist muscle contraction (129), and the occurrence of airway closure (102). In children, although the chest wall is more compliant, lower lung elastic recoil and weak expiratory musculature (relative to adulthood) limit their ability to maximally exhale. During adolescence the chest wall stiffens, and despite greater lung elastic recoil (37) and more developed expiratory musculature, expiration becomes limited by the ability to compress the chest wall (91). Closing volume (CV), the volume of air remaining in the lungs once the small dependent airways have closed and remaining expiration is from the upper open airways (201), is high in children (approaching supine FRC) due to earlier airway closure resulting from less elastic recoil of the lungs (201). However, as lung elastic recoil and airway elasticity increase during growth, airway closure decreases. Therefore, CV decreases and converges with RV, reaching a minimum around 16 years where it can be undetectable (37).

Thus, while RV increases primarily due to the growing lungs, these mechanical changes may underline secondary increases as evidenced by a marginal increase in the ratio of RV to TLC (RV/TLC) during development (57, 124, 135, 202). The large, longitudinal Australian study by Hibbert *et al.* reported average values ranging from 0.23-0.26 in early pubescent females (age 8-13 y old) to 0.30-0.31 in older adolescent females (age 17-20 y old) (57). The ratio was slightly lower in males, ranging from 0.22-0.26 in early pubescent males (age 8-13 y old) to 0.25-0.29 in

older adolescent males (age 16-20 y old). They suggested that this might have been related to differences in expiratory muscle strength, chest wall shape, or airway compliance. Conversely, others have reported RV/TLC to be constant throughout development (86) and similar between males and females (86, 195), with most reference values ranging from 0.171 to 0.240 (194).

In this thesis, RV was similar between the groups (p=0.70) and therefore RV/TLC was smaller in swimmers (p=0.07). The former agrees with previous reports that found no difference in RV despite a larger TLC in developing and young adult competitive swimmers compared to controls (18, 22, 27) and predicted values (21). However, others observed a greater RV in swimmers compared to controls (14, 27-29) and predicted values (23, 24, 31). Interestingly, swimmers and controls were reported to have a similar RV/TLC (18, 21, 27-29) regardless of whether or not there was a difference in RV. Only Zinman and Gaultier's oldest cohort had a significantly different RV/TLC, for which swimmers were larger (27). Therefore, the finding that RV/TLC was smaller for the swimmers in this thesis is notable. The average of 0.21 \pm 0.03 is comparable to calculations of RV/TLC from previous studies, including 0.186 ± 0.035 in 12-16 y old female swimmers (21); 0.22 ± 0.05 in 9-10 y old female swimmers (18); 0.24 ± 0.05 , $0.20 \pm$ 0.07, and 0.25 ± 0.06 in 7-8, 9-10, and 11-13 y old female swimmers, respectively (27); and 0.232 \pm 0.06 (28) and 0.213 \pm 0.026 (29) in female and male collegiate swimmers, respectively. Moreover, the controls had an RV/TLC (0.24 ± 0.05) within the range of earlier stated reference values.

Since RV is determined by lung size, lung elastic recoil, chest wall recoil, and the expiratory muscle's ability to compress the chest wall, it seems likely that the opposing effects of larger lungs and stronger expiratory musculature interact to determine whether RV is greater than expected. In cases where RV was similar despite swimmers possessing a greater TLC, it is possible

that the muscularity effect underlying slight differences in RV/TLC between males and females may also apply to swimmers versus controls. Stronger expiratory musculature may not be captured by PE_{MAX} , as PE_{MAX} is measured at TLC and was observed to be larger in swimmers in some (20), including here, but not all (27-29) studies. On the other hand, in cases where RV was larger than expected, it is possible that the lungs of swimmers are of such size, that even with the muscularity effect, RV is still often observed to be greater than controls. Thus, the similar RV but lower RV/TLC of swimmers in this thesis was likely due to their relatively greater ability to compress their chest wall during maximal expiration.

4.1.2.8 Vital capacity

Vital capacity is the difference between TLC and RV; therefore, VC is dictated by the determinants of both TLC and RV. These include lung size, lung and chest wall recoils at RV and TLC, the respiratory musculature's ability to compress and expand the chest wall, and antagonistic muscle activity. Regardless of whether it is determined using spirometry (i.e., FVC) or as the difference between TLC and RV, vital capacity was consistently observed to be greater in swimmers compared to controls (7, 11, 12, 14, 16-18, 22, 26-30, 40, 41) or predicted values (9, 13, 21, 23-25, 31, 41). Only a couple of studies found no differences with controls (33, 42, 43). Thus, it is likely that a greater lung size, increased TLC, the muscularity effect, and decreasing RV, all contribute to the increased vital capacity of the swimmers in this study.

4.1.2.9 Spirometry

Peak expiratory flow (PEF) and peak inspiratory flow near TLC are effort-dependent because they are limited by the rate of muscle shortening and therefore the muscle's ability to generate force (67, 203). At lower lung volumes, expiratory flow becomes an effort-independent function of airway conductance (which is a function of the interaction between the forces across and intrinsic properties of the airway wall (204)) and lung and chest wall recoil, not of muscular effort (67). Maximum expiratory flow-volume curves provide a great tool for analysis of flows and how they change over time. During development, the MEFV geometrically enlarges primarily due to an increasing TLC, leading to a proportionally larger PEF (205). However, changes in lung mechanics lead to changes in curvature of the MEFV as well. Children have higher airway resistance, lower elastic lung recoil, and nonhomogenous emptying of the alveoli; therefore, peak expiratory flows are lower and they are unable to sustain high flows throughout expiration, causing the curve to be convex to the volume axis (46). During adolescence, larger lungs and an increased $P_{st}(L)$ lead to a concave curve with greater PEF and forced expiratory flows that can be sustained for more of the expiratory maneuver. However, adolescents can abruptly "fall off" the curve because greater chest wall stiffness limits the expiratory muscles' ability to compress the chest wall at low lung volumes (91). The curve becomes convex once again with increasing age throughout adulthood as airways become flow-limited and lung elastic recoil decreases.

Given that the swimmers in this study had a significantly larger TLC, it is not surprising that they had a greater PEF and other indices of spirometry including FVC, FEV₁, and forced expiratory flows (FEF_{25-75%}, FEF_{25%}, FEF_{50%}, and FEF_{75%}). A greater TLC and PEF were also observed in previous reports compared to controls (14, 18, 29) or predicted values (31). Moreover, most studies measuring FEV₁ as a surrogate of flow found FEV₁ to be greater in swimmers compared to controls (14, 16, 18, 20, 22, 28-30, 40, 41) and predicted values (24, 25, 31, 41). The ratio FEV₁/FVC was nearly identical between the groups and did not change over time, with all swimmers having a ratio greater than or equal to 0.80. While swimmers had larger forced

expiratory flows, this was likely due to the flows occurring at a higher absolute lung volume. This is highlighted by the average MEFV (Figure 1), which also shows that swimmers and controls generated the same flow for a given volume. Furthermore, the quantitative characteristics of the MEFV were the same. The similar instantaneous and average SR implied similar emptying properties of the lung, while curvature of the MEFV was similar as indicated by comparable β° and FR. Thus, despite having larger lungs and higher peak expiratory flows, swimmers were subject to the same mechanical constraints at low lung volumes as were controls. This result is not surprising when considering that the MEFV was geometrically larger, likely due to a greater TLC, and curvature was the same, likely because there is no evidence of differences in mechanical properties between the groups.

Clanton *et al.* compared 16 collegiate female swimmers and 8 sex-matched controls, finding that the swimmers had a leftward shifted MEFV due to a significantly larger RV and especially TLC (14). Therefore, swimmers produced lower expiratory flows for a given absolute lung volume. However, the swimmers and controls had an identical MEFV when plotted against %-TLC, suggesting that the time constant for lung emptying was the same for swimmers and controls. Thus, the difference between the observations of Clanton *et al.* and this thesis could be explained by differences in RV. In younger swimmers, when RV is likely to be closer to that of controls, the MEFV is geometrically larger due to a larger TLC but flows are the same for a given absolute lung volume. With continued development, TLC becomes so substantially increased, that despite the muscularity effect, RV may become higher in swimmers. While the MEFV is still a geometrically similar shape in swimmers and non-swimmers (because mechanical lung properties are similar), it is shifted to the left due to the higher RV.

These findings appear to contrast those of Courteix *et al.*, who reported that their swimmers had an MEFV shifted rightward and increased forced expiratory flows such that swimming "improved" the flow-volume relationship (18). Moreover, they stated that Raw increased in the control group but not the swimmers, and therefore swimming alters dysanaptic development of the respiratory system. However, there are multiple criticisms of how they reached their conclusions. First, they plotted the MEFV with all groups having their TLC start at the same point along the volume axis. However, TLC was significantly larger at the follow-up measurement in swimmers, hence causing their MEFV to look larger and peak and mid-expiratory flows to look higher. If plotted from RV, which was the same at both time points for both groups, then the MEFV would mirror those reported in this thesis. Second, the difference in changes in forced expiratory flows is confounded by the lack of growth in TLC reported in the control group. Courteix et al. argued that the increased forced expiratory flows over 1 year in swimmers, but not controls, implied that swimming improved conductive properties of the small airways. Yet, given that their control group only increased an average of 90 ml in TLC, one would not expect to measure changes in expiratory flows if minimal changes in TLC were observed. Had the control group experienced normal amounts of lung growth, no differences may have been found. Lastly, Courteix et al. stated that R_{aw} increased only in the control group but there were no statistically significant differences between swimmers and controls at either time point for Raw. Instead, their conclusion was based on a statistically significant difference between the groups in %-change from initial to follow-up. No details of statistical analyses were provided for changes from initial to follow-up in absolute R_{aw}. Thus, critical interpretation of their analysis questions the validity of their statements about the effect of swimming on dysanaptic growth.

4.1.2.10 Dysanapsis ratio

As stated earlier, the DR can be calculated to estimate differences in airway size relative to lung volume and lung static elastic recoil pressure (60). Longitudinal analysis in children and adolescents showed that DR, using the regression equation from Zapletal *et al.* based on height to estimate $Pst(L)_{50}$ (90), range from 0.10-0.20 and steadily decrease with increasing age (57). However, two considerations must be made when interpreting DR. First, because DR is a function of lung size, and airway length is dependent on lung size, DR can be confounded by larger lungs which have longer airways and thus increased airway resistance, causing decreased flows. Therefore, DR must be compared at equal lung volumes to control for airway length. Second, when comparing DR across different lung volumes, interpretation of the growth pattern observed (i.e., isotropic vs. dysanaptic) depends on the exponential function best fitting the theoretical relationship between DR and lung volume. A relationship of VC^{-2/7} suggests isotropic growth with turbulent flow during the MEFV; VC⁻¹ suggests dysanaptic growth whereby airway and lung sizes are independent; lastly, VC^{-4/3} suggests dysanaptic growth where airway diameters are independent of lung size but airway length varies according to lung volume (46, 89). Smith et al. used these functions to determine that a relationship of VC^{-4/3} described dysanaptic growth patterns during prepubescence and VC⁻¹ during postpubescence (128). Their DR were slightly higher (0.37 \pm 0.09 to 0.21 ± 0.06 at pre- and post-pubescence, respectively) because they estimated Pst(L)₅₀ using the regression equations calculated by De Troyer et al. based on age (112). Thus, dysanaptic growth occurs throughout development, with different dysanaptic patterns during pre- versus postpubescence.

In this thesis, DR was similar between the two groups. However, DR could not be compared at similar lung volumes because the average TLC for the swimming group was much larger than the control group. Moreover, Figure 9 shows the considerable variability in DR that made individual comparisons at a given lung volume and estimation of the exponential relationship between DR and lung volume difficult. Therefore, interpretation of the effect of swimming on airway diameter from this thesis is limited. Nevertheless, future work should investigate dysanaptic growth patterns in swimmers throughout development for multiple reasons. First, dysanapsis has recently become an increasingly relevant clinical phenomenon (206, 207). Second, as initially suggested by Zinman and Gaultier, assessing dysanaptic growth patterns in swimmers could hold the key to understanding whether competitive swimming affects lung development (27). No single agent can induce lung growth that replicates the coordinated dysanapsis of the normal lung (109). Instead, induced postnatal lung growth exaggerates dysanaptic growth patterns because accelerated alveolar growth occurs without a correspondingly enhanced airway growth (109). The larger airspaces without compensatory larger airways leads to limitations in airway conductance that diminishes the functional benefits of enhanced alveolar growth (109). This is demonstrated by the hypoxia-induced lung growth of high altitude residents (208). Peruvian natives to 3850 m (highlanders) had significantly greater FVC than and similar lung elastic recoil to Peruvian natives to 800 m (lowlanders), but had lower flows and airway conductance at low lung volumes and at all volumes when corrected for lung size. Therefore, if swimming induces postnatal lung growth, then exaggerated dysanaptic growth patterns and increased airway resistance (especially at low lung volumes) would be expected.

4.1.2.11 Maximal static pressures and respiratory muscle force

Maximal static mouth pressures. Maximal pressures measured at the mouth are indicative of the maximum pressures developed by the respiratory muscles, the passive elastic recoil pressure of the respiratory system (lungs and chest wall), and the lung volume at which the pressure is measured (209). They are also influenced by the shape of the chest wall and relaxation of the antagonist muscles (129). Thus, it provides an indirect index of respiratory muscle strength (191). In children and adolescents, maximal static inspiratory and expiratory mouth pressures increase during development (45, 102, 136, 210, 211). This is likely due to respiratory muscle strengthening (i.e., the "muscularity" effect (212)); increasing chest wall outward recoil at RV and increasing elastic lung recoil at TLC may also contribute to improvements in PI_{MAX} and PE_{MAX}, respectively. Moreover, maximal pressures may be relatively higher in children as compared to adults because of mechanical advantages due to the smaller radius of the costal diaphragm and chest wall (213). Pressures are higher in boys than girls (45, 102, 136, 210, 211); while girls reach adult values by puberty (102, 136), boys continue to increase after the growth spurt due to the muscularity effect (102).

As mentioned earlier, maximal static mouth pressures were seldom measured in young swimmers. Maximal expiratory pressure was observed to be larger in swimmers compared to controls in only one study (20); others found no difference (27-29). Moreover, only similar measures of PI_{MAX} between swimmers and controls were reported (14, 27-29). In fact, Zinman and Gaultier reported a significantly smaller PI_{MAX} in their 11-13 y old swimming cohort compared to controls (27). Therefore, the findings of greater PE_{MAX} and PI_{MAX} in the swimmers in this thesis are somewhat surprising. Swimmers increased, on average, 16 cm H₂O in PI_{MAX} and 13% in %-predicted PI_{MAX} whereas the controls only 8 cm H₂O and 5%. Although the interaction effect was not significant, it is possible that inspiratory muscle conditioning due to swimming may explain some of the difference in PI_{MAX} given that swimming was reported to improve inspiratory muscle strength and endurance (14, 19). It seems more likely that differences in both PI_{MAX} and PE_{MAX}

may be explained by relatively lower values in the control group. The controls had an average %predicted PI_{MAX} of 82 ± 30 cm H₂O and 87 ± 30 % and %-predicted PE_{MAX} of 77 ± 19 cm H₂O and 84 ± 19 % at PRE and POST, respectively. Whereas only one swimmer had a slight decrease in PI_{MAX} (-4%) and PE_{MAX} (-4%), three controls decreased PI_{MAX} (by 10, 10, and 22%) and PE_{MAX} (by 6, 11, and 15%). Moreover, all swimmers had a %-predicted PI_{MAX} greater than 80% at followup, while four controls were <70% of predicted values. One control subject struggled with the maneuver and had a %-predicted PI_{MAX} of 26% at the initial measurement. Thus, differences in maximal static mouth pressures reported in thesis may be due to a combination of respiratory muscle conditioning due to swim training and relatively lower values by the control group.

Respiratory muscle force. Respiratory muscle force cannot be directly measured in vivo, and the best estimation is pleural pressure (67). However, multiple studies have estimated respiratory muscle force production at different lung volumes (RV, FRC, and TLC) from the product of maximal static pressures and estimations of chest wall surface area (102, 136). Inspiratory and expiratory forces increase with age in both 7-13 y old (136) and 12-20 y old (102) children and adolescents. This occurs to a greater extent than corresponding increases in static pressures (136), reflecting the increasing chest surface area. Therefore, changes in respiratory muscle forces may capture differences between swimmers and controls that maximal static pressures alone cannot. Zinman and Gaultier observed a greater inspiratory muscle force in 7-10 y old swimmers despite a similar PI_{MAX}, suggesting that pressure alone did not account for the larger chest wall surface area over which swimmers must dissipate pressure (27). Conversely, no differences in inspiratory or expiratory muscle force were found between collegiate male swimmers, runners, and controls (29). Although not measured in this study, this suggests that respiratory muscle force (i.e., pleural pressure) and other surrogate measures of respiratory muscle

strength (e.g., transdiaphragmatic pressure or diaphragm thickness) may elucidate respiratory muscle training effects of swimming not captured by maximal static mouth pressures.

4.1.2.12 Diffusion capacity of the lungs

Diffusion capacity of the lungs for carbon monoxide is anatomically determined by lung volume, alveolar-capillary surface area, alveolar-capillary membrane thickness, and pulmonary capillary blood volume (109). During growth, increases in alveolar surface area, vascular density, and capillary bed size (107) lead to progressive increases in $D_{L,CO}$ with height and lung size (47, 214). The ratio of diffusing capacity of the alveolar membrane to pulmonary capillary blood volume is similar to that in adults, suggesting that the relationship between alveolar capillary diameter and wall thickness is not affected by growth (214). However, $D_{L,CO}$ standardized for alveolar V_A (also known as the transfer factor (K_{CO})) progressively decreases with height (214). This may be due to greater growth in alveolar volume compared to the gas-exchanging portion of the membrane (i.e., basement epithelial membrane of type I cells) (214). While $D_{L,CO}$ is higher in males, K_{CO} is similar between the sexes because the greater $D_{L,CO}$ in males is proportional to their larger lungs (214).

In this thesis, resting $D_{L,CO}$ was significantly greater in swimmers compared to controls, even when corrected for hemoglobin (which was similar between the groups). The swimmers also had an average %-predicted $D_{L,CO}$ of 121% at PRE and 122% at POST. These are in accordance with previous studies that measured a greater $D_{L,CO}$ in swimmers than controls (11, 22, 29, 34, 39) and predicted values (24, 29, 31-33, 35). However, differences in $D_{L,CO}$ between swimmers and controls in this thesis were abolished when expressed relative to V_A , confirming previous findings (11, 22, 29). Moreover, %-predicted $D_{L,CO}/V_A$ averaged 101 and 97% for swimmers at PRE and
POST, respectively. Thus, it is likely that $D_{L,CO}$ was greater due to a greater TLC, but it is unknown if any changes in alveolar-capillary membrane or pulmonary capillary blood volume occurred.

4.1.2.13 Summary

Development of the respiratory system is characterized by dysanaptic growth of the alveoli and airways, changes in chest wall mechanics, and a tight coupling between chest wall and lung growths. Currently, there is no evidence to suggest that these characteristics are altered by any of the unique challenges imposed by competitive swimming on the developing respiratory system. Therefore, it seems likely that the greater TLC of the swimmers in this thesis and other studies were related to larger chests, with an additional, but limited, contribution by stronger respiratory musculature. The larger TLC can explain why other measures of pulmonary function are greater, including FVC, PEF, mid-expiratory flows, the MEFV, and D_{LCO} (Table 19). It can also explain the greater FRC in other studies, whereas in this thesis the lack of difference in FRC may have been related to an initially high mean FRC in the controls. This initially high mean FRC in controls may underlie the smaller FRC/TLC found in the swimmers in this thesis, whilst values for FRC/TLC paralleled other reports of swimmers. Conversely, FRC/TLC has been similar between swimmers and controls in other studies because there are no known differences in lung mechanics to suggest otherwise. Greater PE_{MAX} and PI_{MAX} indicate stronger respiratory musculature, leading to a "muscularity effect" underlying a similar RV and lower RV/TLC. Thus, an effect of competitive swimming is not definitively associated with any of the differences in pulmonary function (beyond differences in respiratory musculature) or structure between swimmers and controls. To confirm this conjecture, future work should focus on quantifying and comparing dysanapsis between swimmers and controls.

Maaguma	Determinente	Changes with	Finding	Findings in previous reports on	Mechanism of	Comments
Measure	Determinants	growth	here	swimmers	change	
TLC	Lung size, inspiratory muscle force generation, inwards elastic recoils of lungs and chest wall (139)	↑ with height primarily due to chest wall growth (140)	↑	↑ compared to controls (11, 12, 14, 15, 18, 19, 22, 27-29) or predicted values (9, 21, 23, 24, 31) \leftrightarrow compared to controls (42)	-Larger chests -Greater ability of respiratory muscles to distend chest wall	
FRC	Lung size, static passive balance of lung recoil inwards and chest wall recoil outwards	↑ primarily due to growing lungs	\leftrightarrow	↑ compared to controls (14, 22, 23, 27-29) or predicted values (9, 10, 12, 24, 31) \leftrightarrow compared to controls (11)	(in other studies) Larger TLC	↔ FRC potentially due to control group having large FRC at PRE
FRC/TLC	Mechanical properties of lungs at FRC and TLC	Either \leftrightarrow or \uparrow slightly	↓	\leftrightarrow compared to controls (18, 27)	(in other studies) Similar mechanical properties of lung	↓ FRC/TLC potentially due to control group having large FRC at PRE
RV	Lung size, expiratory muscle strength, opposing chest and lung elastic recoil forces (200), antagonist muscles (129), occurrence of airway closure (102)	↑ primarily due to growing lungs	\leftrightarrow	 ↑ compared to controls (14, 27-29) or predicted values (23, 24, 31) ↔ compared to controls (18, 22, 27) or predicted values (21) 	Balance between larger TLC (\uparrow RV) and muscularity effect (\leftrightarrow RV)	
RV/TLC	Mechanical properties of the lungs at RV and TLC	Either ↔ or ↑ slightly	↓ (p=0.07)	↑ compared to controls (27) ↔ compared to controls (18, 21, 27-29)	-Balance between larger TLC (causing ↔ RV/TLC) and muscularity effect (causing ↓ RV/TLC)	
PEF	Lung size, effort, expiratory muscle force generation, lung elastic recoil	↑ due to ↑ TLC, ↑ $P_{st}(L)$	↑	↑ compared to controls (14, 18, 29) or predicted values (31) No difference [refs]	Larger TLC	
FVC or VC	Lung size, RV	↑ primarily due to growing lungs	↑	 ↑ compared to controls (7, 11, 12, 14, 16-18, 22, 26-30, 40, 41) or predicted values (9, 13, 21, 23-25, 31, 41) ↔ compared to controls (33, 42, 43) 	Larger TLC, muscularity effect	
FEF	Lung size, airway conductance, lung and chest wall recoil	↑ due to ↑ TLC, ↑ $P_{st}(L)$	Î	-	Larger TLC (FEF occurs at ↑ volume)	
MEFV	Lung size, lung mechanics	Geometrically enlarges due to ↑ TLC, some changes in curvature	Larger	Leftward shifted (14) Rightward shifted, larger (18)	Larger TLC, similar RV	Quantitative characteristics (β° , SR, FR) all similar
D _{L,CO}	Lung size, alveolar-capillary surface area and membrane thickness, pulmonary capillary blood volume (109)	↑ due to ↑ alveolar surface area, vascular density, capillary bed size (107)	Î	↑ compared to controls (11, 22, 29, 34, 39) or predicted values (24, 29, 31-33, 35)	Larger TLC	
$D_{L,CO}\!/V_A$	$D_{\text{L},\text{CO}}$ and alveolar volume	\downarrow with height (214)	\leftrightarrow	\leftrightarrow controls (11, 22, 29)	-	
PE _{MAX}	Respiratory muscle pressure generation, passive elastic recoil pressure of respiratory system, lung volume at measurement (209), chest wall shape and antagonist muscles (129)	↑ likely due to muscularity effect (212), some mechanical and chest wall shape changes	ſ	↑ compared to controls (20) ↔ compared to controls (27-29)	Muscularity effect	Values slightly lower in controls may exaggerate difference
PI _{MAX}	Same as PE_{MAX}	Same as PE _{MAX}	↑ (p=0.06)	↔ compared to controls (14, 27-29) ↓ compared to controls (27)	Same as PE_{MAX}	Same as PE _{MAX}

Table 19 – Summary of changes in pulmonary function

TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; PEF, peak expiratory flow; $P_{st}(L)$, static lung elastic recoil pressure; FVC, forced vital capacity; VC, vital capacity; FEF, forced expiratory flow; MEFV, maximum expiratory flow-volume curve; β° , β -angle; SR, slope ratio; FR, flow ratio; $D_{L,CO}$, diffusion capacity of the lungs for carbon monoxide; V_A , alveolar volume; PE_{MAX} , maximal static expiratory mouth pressure; PI_{MAX} , maximal static inspiratory mouth pressure.

4.1.3 Metabolic and ventilatory responses during cycling exercise

The secondary finding of this study was that the competitive swimmers, despite having larger lungs and enhanced pulmonary function, experienced similar ventilatory constraints as healthy controls of similar age, size, and sexual maturity while cycling. This was demonstrated by similar EELV, prevalence and severity of EFL, and utilization of \dot{V}_{ECAP} . One season (seven months) of competitive swimming during puberty did not affect the occurrence of these constraints.

4.1.3.1 Metabolic and ventilatory responses during cycling

One season of competitive swimming during puberty did not affect the metabolic response to exercise, as there were no two- or three-way statistically significant interactions involving group and time. Differences in absolute work rate and $\dot{V}O_2$ were due to the swimmers being slightly, albeit not significantly, larger in body size. This likely explains the greater $\dot{V}CO_2$ in SWIM, which led to an increased \dot{V}_E achieved by a larger V_T . However, the absence of any significant interactions between relative work rate and group for \dot{V}_E , f_B and V_T suggested that SWIM and CON had similar relative changes in their ventilatory response as exercise intensity increased. Moreover, nearly identical ventilatory equivalents for oxygen consumption ($\dot{V}_E/\dot{V}O_2$) (p=0.76) and carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) (p=0.95) reinforce that the groups had similar ventilatory responses to increasing metabolic demands during exercise. Significant two-way interactions between time and relative work rate reflect similar metabolic data at baseline during PRE and POST, but increased values throughout exercise in accordance with higher absolute work rates at POST.

The swimmers had similar maximal metabolic responses to cycling exercise as other adolescent female swimmers (Table 20), including relative $\dot{V}O_{2MAX}$ (12, 21, 215-218). However,

contrary to previous reports that male (215, 219) and female (215, 217, 219) swimmers had a higher $\dot{V}O_{2MAX}$ compared to untrained controls (215, 217) or other athletes (219), in this thesis there was no difference in relative $\dot{V}O_{2MAX}$ between the groups. This was likely due to the excellent exercise capacity of the control group. Both groups had, on average, a $\dot{V}O_{2MAX}$ that was 115-131% predicted. Moreover, the similar peak aerobic capacity is not surprising when considering that both groups reported similar physical activity levels and nearly doubled the Canadian guideline of at least 60 minutes of daily moderate-vigorous intensity physical activity.

Absolute $\dot{V}O_{2MAX}$ increased from PRE to POST in both groups, which is in agreement with previous reports of female swimmers (219) and with absolute VO2MAX tending to increase from 8 to 13 y old in healthy, untrained girls (220). Conversely, relative VO_{2MAX} did not significantly increase over time. This has been observed in other longitudinal studies of 10-16 y old (217) and 8-16 y old (219) female swimmers, whereas others found it to increase over 3 consecutive years in 13-17 y old female swimmers (13). Robinson et al. had 12 10-16 y old competitive female swimmers perform maximal cycling exercise tests before and after one season of competitive swim training (swimming up to 12-14 km per day as often as 6 days per week) (217). Their relative $\dot{V}O_{2MAX}$ barely changed after one season (47.7 ± 5.2 and 48.9 ± 3.4 ml·kg⁻¹·min⁻¹ before and after, respectively), and they attributed any slight increases to somatic growth. A similar difference between PRE and POST (42.9 ± 6.8 versus 44.4 ± 8.1 ml·kg⁻¹·min⁻¹) was measured here. It is also important to note that VO_{2MAX} expressed relative to body weight was shown to decrease with sexual maturity (221) and chronological age (220) in healthy, untrained growing girls, most likely due to increasing subcutaneous fat accumulation post-pubescence (221). Therefore, the maintenance of an elevated relative VO_{2MAX} throughout pubescence in female swimmers (219) suggests that intense swim training during adolescence prevents age-related declines in relative $\dot{V}O_{2MAX}.$

Study	Age (y)	Ν	Height (cm)	Mass (kg)	HR (bpm)	\dot{V}_E (l·min ⁻¹)	$\dot{V}O_2$ (l·min ⁻¹)	VO ₂ (ml·kg ⁻¹ ·min ⁻¹)
Cunningham and Eynon, 1973 (216)	12.2 ± 0.4	8	154.8 ± 10.0	43.3 ± 6.7	191 ± 2	60.9 ± 14.9	1.97 ± 0.31	46.2 ± 7.8
This thesis (PRE)*	12.4 ± 0.8	11	161.3 ± 7.9	$\textbf{52.4} \pm \textbf{10.8}$	192 ± 10	$\textbf{85.5} \pm \textbf{20.8}$	$\textbf{2.20} \pm \textbf{0.35}$	$\textbf{42.9} \pm \textbf{6.8}$
Wirth et al., 1978# (218)	12.4	8	155	44.8	186	-	1.94	40.3
Robinson et al. 1978 (PRE)* (217)	12.9 ± 1.9	12	152.9 ± 8.7	45.5 ± 9.9	199 ± 6	64.9 ± 17.6	2.16 ± 0.41	47.7 ± 5.2
Astrand et al. 1963 (21)	12.9	30	164.8 ± 6.5	54.2 ± 7.3	199 ± 8	99.9 ± 16.5	2.80 ± 0.44	51.5 ± 4.4
This thesis (POST)**	13.0 ± 0.8	11	163.4 ± 6.9	55.8 ± 9.8	195 ± 8	100.4 ± 17.6	$\textbf{2.42} \pm \textbf{0.23}$	$\textbf{44.4} \pm \textbf{8.1}$
Cunningham and Eynon, 1973	13.2 ± 0.1	6	160.0 ± 4.54	52.1 ± 8.1	189 ± 7	63.6 ± 12.0	2.24 ± 0.34	43.4 ± 6.5
Robinson et al., 1978 (POST)**	-	12	-	-	199 ± 6	63.7 ± 13.4	2.30 ± 0.40	48.9 ± 3.4
Kramer and Lurie, 1964 (215)	14.7 ± 0.8	6	165.9 ± 2.1	61.1 ± 2.3	197 ± 3	84.1 ± 2.1	3.01 ± 0.09	49.5 ± 2.4
Cunningham and Eynon, 1973	14.9 ± 0.6	5	164.8 ± 7.4	53.7 ± 5.4	189 ± 8	70.0 ± 13.5	2.19 ± 0.34	40.5 ± 2.1
Wirth <i>et al.</i> , 1978 [#]	15.9	6	166	53.8	178	-	2.40	44.4
Eriksson et al., 1978 (12)	17.0	9	170.0 ± 3.7	62.5 ± 8.1	201 ± 6	105.6 ± 5.4	3.14 ± 0.22	51.1 ± 5.9

Table 20 – Metabolic responses at maximal cycling exercise in adolescent female swimmers

*before one season of swim training. **after one season of swim training. *Standard deviations not provided. HR, heart rate; \dot{V}_E , minute ventilation; $\dot{V}O_2$, oxygen consumption.

4.1.3.2 Ventilatory mechanics during cycling exercise

As mentioned earlier, ventilatory capacity is primarily determined by anatomical features (i.e., lung size, airway size and geometry) (69). Therefore, this thesis explored whether the larger lung volumes and expiratory flows of swimmers lead to larger ventilatory capacities and alleviate ventilatory mechanical constraints during exercise. It was previously suggested that the increased ventilatory capacity could facilitate increased metabolic and ventilatory demands within similar ventilatory constraints. Alternatively, alleviation of ventilatory constraints could be possible by generating higher flows at similar absolute lung volumes, decreasing the susceptibility to EFL and allowing swimmers to operate at lower relative lung volumes that may lower their WOB.

In terms of group differences in quantitative measures of ventilatory mechanics, there were no statistically significant differences between swimmers and controls for absolute or relative EELV, prevalence or severity of EFL, \dot{V}_{ECAP} , or utilization of \dot{V}_{ECAP} . As shown in Figure 14C and Figure 14D, swimmers utilized a similar absolute EELV (p=0.18) and attained a larger V_T (p=0.02) by tending towards a greater absolute EILV (p=0.05). This was possible because of a larger IC (p<0.001) in swimmers (Figure 16C). In fact, IC was so much larger that swimmers still had a larger IRV (p<0.01), even with a greater EILV (Figure 16A). Given that EILV/FVC was slightly smaller (Figure 15C and Figure 15D, p=0.08), IRV/FVC was slightly larger (Figure 16B, p=0.08), and EELV/FVC was the same (Figure 15C and Figure 15D, p=0.46), these suggest that swimmers utilized a smaller relative amount of their greater inspiratory capacity. This is depicted in Figure 16D, where differences in IRV/IC favoured swimmers but did not reach statistical significance (p=0.10).

Despite the significantly greater IC in swimmers, ventilatory mechanics were still similar between the groups. This was because of the similar absolute EELV combined with a MEFV that was only geometrically larger and had similar quantitative characteristics. Recalling Figure 1, swimmers and controls produced similar flows for a given absolute volume on the MEFV. Therefore, the swimmers and controls were subjected to identical flow constraints when operating at a similar EELV. This may explain why, contrary to our secondary hypothesis, they did not operate at a lower relative EELV, they were equally susceptible to EFL, and there were no significant differences in \dot{V}_{ECAP} and \dot{V}_E/\dot{V}_{ECAP} . The qualitative differences in ventilatory constraints can also be seen in the composite MEFV and superimposed FVL (Figure 19). Hypothetically, in order to alleviate ventilatory constraints by increasing ventilatory capacity or avoiding EFL, the swimmers would have to operate at a higher EELV. However, this would come at the cost of a greater WOB due to breathing along a less compliant segment of the P-V curve. The similar \dot{V}_{ECAP} also negates the possibility of increased metabolic and ventilatory demands within similar ventilatory constraints. Instead, any increase in demand would necessitate greater susceptibility to ventilatory constraints. Thus, the significantly larger TLC and PEF of competitive swimmers did not affect the occurrence of ventilatory constraints while cycling.

There were no statistically significant three-way interactions or two-way interactions involving group. The lack of differences in the two-way interactions between group and time implies that one season of competitive swim training did not affect ventilatory mechanics during exercise. The interaction effects between group and work rate for EELV/FVC (p=0.07), EILV (p=0.10), and IRV (p=0.10) approached statistical significance, but post hoc testing revealed few differences. For EELV/FVC, the only difference between swimmers and controls was at baseline (p=0.02). Moreover, only at baseline was EILV not significantly greater in swimmers (p=0.09), and swimmers also had a greater IRV at all work rates. Therefore, the pattern of the ventilatory responses was similar between swimmers and controls, with swimmers having a response of

greater magnitude for most measures. Lastly, all interactions between time and work rate were either significant (p<0.05) or approached significance (p<0.10). Akin to the metabolic responses, this likely reflects similar ventilatory data at baseline during PRE and POST, but increased differences throughout exercise in accordance with higher absolute work rates and metabolic demands at POST. Taken altogether, these results suggest that one season of competitive swim training did not affect ventilatory mechanics during exercise.

Regardless of training status, the prevalence of EFL was much higher in this study than previously reported for postpubescent girls. Emerson et al. observed EFL in only 2 out of 10 (20%) post-pubescent girls (mean age 14.1 ± 1.0 y and Tanner stage 3.7 ± 0.7) (62) compared to 52% (22) out of 42 total tests) here. There are multiple possible explanations for this difference. First, the subjects in this study were slightly younger and prevalence of EFL was more comparable to the high rates of EFL observed during prepubescence (56% (63) to 93% (61)). It is possible that, with further maturational development and continued lung growth upon cessation of somatic growth in height, the ventilatory capacity increases such that the occurrence of EFL in females becomes less frequent. Alternatively, the difference could be explained by differing fitness levels. The subjects in this thesis had an average relative $\dot{V}O_{2MAX} > 40 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ compared to only $33.0 \pm 6.7 \text{ ml} \cdot \text{kg}^{-1}$ $1 \cdot \min^{-1}$ in the 10 girls tested by Emerson *et al.* (62). The greater metabolic demand could have led to a greater ventilatory response, increasing the susceptibly to EFL. It is interesting to note that the prevalence of EFL was reported to be 45% in healthy young women (60) and 90% in trained young women (58) and occurred in women of all levels of cardiovascular fitness (59, 60). Thus, further investigation of changes in female susceptibility to ventilatory constraints with maturation is clearly needed.

4.1.3.3 Individual responses

Figure 17 and Figure 18 display the individual MEFV and FVL for each subject. It is interesting to note the different ventilatory mechanics utilized throughout exercise in subjects with the largest lungs and higher cardiovascular fitness levels. Three of the swimmers with the largest %-predicted FVC (S01, 131-132%; S04, 129-130%; and S07, 143%) became dynamically hyperinflated at maximal exercise (i.e., EELV at maximal exercise was greater than baseline). This occurred despite a \dot{V}_E/\dot{V}_{ECAP} that was near or well below average (69-77%, 46-44%, and 66-73%, respectively), but none of them experienced EFL at peak exercise. Conversely, S08 (132-136%predicted FVC) did not dynamically hyperinflate and instead became flow-limited, utilizing all of her ventilatory capacity (94-120%). She was also the swimmer with the highest relative and %predicted VO_{2MAX} (values at POST were 56.0 ml.kg⁻¹.min⁻¹, 162%-predicted). The swimmer with the second-highest values (S12 POST, 55.0 ml.kg⁻¹.min⁻¹, 159%-predicted) had the opposite ventilatory response, avoiding EFL and using a lower proportion of her \dot{V}_{ECAP} (65%) by dynamically hyperinflating. Thus, the high ventilatory demand comes at the cost of either dynamic hyperinflation with a concurrent decrease in \dot{V}_E/\dot{V}_{ECAP} , or EFL and an increased \dot{V}_E/\dot{V}_{ECAP} . The different individual responses of subjects with large lungs or high fitness levels display no preference for either breathing strategy.

4.1.3.4 Summary of ventilatory and metabolic responses to cycling exercise

Taken altogether, the swimmers and controls had similar metabolic and ventilatory responses to cycling exercise. Differences in absolute work rate and therefore $\dot{V}O_2$ and $\dot{V}CO_2$ could be attributed to the swimmers being slightly larger in size. The greater absolute metabolic demand likely caused the increased \dot{V}_E , which was achieved in swimmers by increasing V_T. To

facilitate a greater V_T , the swimmers operated at a similar EELV and higher EILV, utilizing a smaller relative portion of their significantly greater IC. However, because swimmers and controls had similar flows for a given absolute FVC on the MEFV, the swimmers were therefore subjected to identical flow constraints when operating at a similar EELV. This likely explains why they were equally susceptible to EFL, and why there were no significant differences in \dot{V}_{ECAP} and \dot{V}_E/\dot{V}_{ECAP} despite the swimmers having larger lungs.

4.2 Methodological considerations

4.2.1 Tanner level of maturation

When investigating growth in youth, standardizing for maturational age is important because gestational age does not account for differences in the age of onset of puberty (75). Methods of standardization include radiography of the carpal bones (to determine skeletal age), and grouping subjects according to their maturational stage measured by Tanner level of maturation (75). Tanner staging uses the assessment of primary and secondary sex characteristics (penis, testes, and pubic hair for boys; pubic hair and breasts for girls) to classify five stages of maturation (stage 1 to stage 5), and can be self-assessed or done through a physician assessment. However, it must be acknowledged this method has limitations because maturation is a continuous process whereas the assessment of Tanner stages places individuals on an integer scale. Therefore, it is possible for individuals to spend different amounts of time in each Tanner stage. For example,, for two individuals who both reported a SMR of 4, the first individual could have been in stage 4 for many months whereas the second just entered it; in such a case, the two individuals would be documented at the same level of maturity, whereas the first individual is more developed. Thus, in this study it is possible that the reported SMR between swimmers and controls was similar, but the

swimmers could have been at a further point of maturation in each given SMR. This could have contributed to the difference in lung volumes. Despite these limitations, self-reported Tanner staging was used because the method has been validated against a physician assessment (74) and allows sexual maturity to be assessed quickly and non-invasively.

Once separated into the five stages, growth velocities for each stage can be determined. In a mixed-longitudinal analysis of competitive swimmers versus controls that used Tanner staging, the largest increases in both female swimmers and controls in FVC and FEV1 occurred between Tanner stages 3 (12.2 ± 1.0 y old) and 4 (13.1 ± 0.9 y old) and between Tanner stages 4 and 5 (14.1 \pm 1.1 y old) (16). Although the changes between Tanner stages 2 (11.9 \pm 0.9 y old) and 3 were smaller in magnitude, they were still greater in swimmers (mean 0.28 l) than controls (mean 0.14 1) (16). A separate longitudinal analysis showed that the PGV for VC in girls occurred between the ages of 11.5-13.5 y (57). Mathematical modelling of longitudinal data determined that the PGV for girls occurred at age 11.25 ± 0.94 y for FVC and 11.75 ± 0.51 y for FEV₁ (72), and radiographic analysis showed lung and chest lengths and widths peaked around age 12 y in girls (73). Therefore, the period of PGV for the lungs in girls is during Tanner stages 2-4, or a gestational age of 11-14 y. This thesis focused on Tanner stages 2-4 with the rationale that these stages of maturation would likely elicit the greatest effects if competitive swimming were to affect lung development during puberty. However, because potential subjects were not screened for a Tanner stage between 2 and 4, this thesis recruited girls between the ages of 11-14 y old.

4.2.2 Technique

Children can reliably perform FVC maneuvers at ~8 y old (46) (with some as young as 3-6 y old (222)), while measurements of maximal static pressures are reproducible above 8 y of age (45), TLC above 6 y of age (37), and diffusion capacity above 6 y (47). Regardless, given the age of the subjects in this study and that some of the maneuvers are maximal, effort-dependent, and require substantial coordination, some subjects may not have the necessary coordination or motivation to properly perform these maximal maneuvers. This would cause underestimation of their true physiological measurements. Although this could affect all subjects in the study, it was more likely to occur in controls and confound data for maximal maneuvers in favour of swimmers. Competitive swimming requires coordinated maximal inspirations alternating with maximal expirations, therefore swimmers have much more training and experience in performing respiratory maneuvers. It is possible that this could have contributed to the differences in pulmonary function observed in this study. However, the controls appeared to have "normal" function, as evidenced by mean %-predicted values ranging between 90-105% for most measures and averaging $94 \pm 7\%$ for TLC. Moreover, Respiratory Therapists experienced with the pediatric population performed the pulmonary function testing to ensure all subjects elicited maximal efforts. Thus, it is unlikely that poor technique explains the difference in lung function between swimmers and controls.

Calculations of operational lung volumes, EFL, and \dot{V}_{ECAP} during exercise all depend on proper technique and adequate effort during the FVC, gFVC, and IC maneuvers. Inadequate effort during the FVC maneuvers could lead to a smaller MEFV, which would translate into a false positive determination of EFL, lower \dot{V}_{ECAP} , and higher $\dot{V}_{E/VECAP}$. Conversely, poor effort during the IC maneuver could have caused an overestimation of EILV and EELV, false negative determination of EFL, higher \dot{V}_{ECAP} , and lower $\dot{V}_{E/VECAP}$. As stated in the previous paragraph, these were more likely to occur in the control group and confound the outcome measures in favour of the swimmers. However, multiple steps were taken to minimize the likelihood of these errors. First, all subjects practiced and performed FVC and IC maneuvers with visual feedback and verbal coaching from the Respiratory Therapists during the lung function testing, prior to entering the Exercise Physiology Laboratory, Upon entering the laboratory, they received further coaching and practiced the FVC and IC maneuvers while becoming familiarized with the equipment (e.g., breathing through the mask, sitting on the cycle ergometer). Second, multiple FVC and gFVC trials were performed before and after the exercise test, again with extensive coaching. Third, two IC maneuvers were performed during each stage, in case the first maneuver was later deemed to be inadequate (e.g., an inspiration was performed with a closed glottis). Lastly, although esophageal pressure was not measured, inspiratory flow was used as a surrogate for assessing effort during the IC maneuver. Visual inspection of the MEFV and FVL in Figure 17 and Figure 18 suggests that most subjects adequately performed the FVC, gFVC, and IC maneuvers. In most cases, the first and second IC maneuvers during each stage produced similar inspiratory flows and volumes. Moreover, all subjects were motivated, focused, and compliant, especially during exercise. Thus, there is no evidence to support a systematic difference in technique between the swimmers and controls that could have affected the results of this thesis.

4.2.3 Predictive equations

When selecting reference equations, it was recommended that the reference subjects have similar anthropometric and ethnic backgrounds and, if possible, all reference equations are taken from the same source (223). The healthy Australian population studied by Hibbert *et al.* had a similar age (8-18 y old), height, and ethnic background (Caucasian) as most subjects in this thesis (85). Moreover, they published a comprehensive set of reference equations that could be used for all measures of spirometry (including mid-expiratory flows) and lung volumes determined here,

unlike those by Cook and Hamann (224) (which were previously recommended for children aged 5-18 y old (86)) which lacked spirometry. Although Hibbert *et al.* used body plethysmography to measure lung volumes, they found similar values compared to previous studies using helium dilution (85). A recent, large study from The Netherlands involving over 500 2-18 y old children comprehensively measured spirometry, lung volumes, and diffusion calculation using similar techniques to this thesis (225). While this appeared to be an ideal set of predictive equations, calculated %-predicted values were very high for some measures (e.g., average %-predicted RV was 126-129% in all groups) and low for others (e.g., average %-predicted FRC was 75-80% in all groups). Therefore, the equations by Hibbert *et al.* were preferred for spirometry and lung volumes and those for diffusion capacity came from a large study of American and Australian 5-19 y old Caucasian children with similar heights and measurement techniques to the present investigation (87).

Reference values for maximal static mouth pressures were derived from measurements in a Spanish population of 8-17 y old children and adolescents (45). Domenech-Clar *et al.* stated that their predictive equation for PE_{MAX} in females was not suitable for calculating reference values (45). However, upon calculating %-predicted values from predictive equations for this study and the large cross-sectional study by Wilson *et al.* (210), the equations of Domenech-Clar *et al.* provided more reasonable values and were used in analysis. To note, their predictive equations only had a predictive power ranging between 0.21-0.51, despite rigorous methodology (45). They attributed this to high variability of maximal static pressures in the population.

Given that height and age are the most important determinants in spirometry reference equations, height was measured on the day of lung function testing to the nearest 1 mm and age was calculated as a decimal (with the date of birth rounded to the 1st or 15th day of the month for

subject confidentiality) to avoid prediction bias (226). Moreover, all the reference equations used in this thesis included height and age in their statistical modelling, with most including both in the published equation (45, 85, 87).

4.2.4 Interpretation of changes in lung function

In healthy subjects, variation between pulmonary function tests may be technical (e.g., equipment, procedure, calibration, technician expertise, subject, and their interaction) or biological (227). Therefore, it is important to interpret whether differences reflect a change in pulmonary function (signal) or test variability (noise) (223, 227). For example, it was suggested that year-toyear changes of $\geq 15\%$ in FVC and FEV₁ and $\geq 10\%$ in D_{LCO} are needed for changes in adults to be considered clinically significant (223). Measurement noise is often assessed using the coefficient of variation, but assessing test reproducibility in children and adolescents is difficult because of their rapidly growing lungs (227). The within-subject coefficient of variation for TLC measured using plethysmography ranged from 3-9% in 61 6-18 y old healthy boys and girls (205). More recently, the large Dutch study of 500 2-18 y old children calculated absolute coefficients of variation for FVC (0.11), FEV₁ (0.11), PEF (0.15), FEF_{25%} (0.18), FEF_{50%} (0.23), FEF_{75%} (0.31), FEF_{25-75%} (0.24), TLC (0.10), FRC (changed with height and age), RV (0.24), RV/TLC (0.0023), $D_{L,CO}$ (changed with age and sex), V_A (0.11), and $D_{L,CO}/V_A$ (0.23) (225). In this thesis, the swimmers had a 20% greater TLC than controls. This far exceeds the 3-9% coefficient of variation and further supports that swimmers had larger lungs at the initial time point. From PRE to POST, the change in TLC averaged $0.35 \pm 0.131(7.8 \pm 3.6\%)$ and $0.26 \pm 0.251(6.3 \pm 5.8\%)$ in swimmers and controls, respectively. Given that these absolute changes in TLC are greater than the

coefficient of variation determined by Koopman *et al.* (225), it suggests that the swimmers and controls both had a measurable amount of growth in TLC.

In order to reduce between-measurement variability, the same experienced Respiratory Therapists performed both PRE and POST measurements using the same equipment and protocols at the same location. Given that the testing was performed in the Respiratory Clinic of a large, tertiary children's hospital, equipment calibration and quality control checks are both performed routinely. These ensured high reproducibility and precision. Lastly, subjects were highly motivated and had extensive coaching and practice before conducting the maneuvers.

4.2.5 Exposure to chlorine and FEV₁/FVC

The relationship between swimming, chlorine, and asthma has been reviewed elsewhere (228) and will only be briefly discussed here. Chronic exposure to chlorine derivatives has been suggested to cause exercise-induced bronchial epithelial damage (6) that may underlie the increase in respiratory symptoms, airway inflammation, and airway hyperresponsiveness (AHR) observed in adult elite swimmers (229). However, evidence in young swimmers is inconclusive. Swimming was not shown to increase the risk of asthma in children longitudinally followed from birth until age 10 years (230). In a cross-sectional analysis of male and female swimmers aged 8-20, no significant relationship was found between duration of exposure to chlorination products and methacholine (MCh) challenge response or asthma-like symptoms score (231). Chlorine exposure was not found to affect airway inflammation in elite adolescent swimmers (232), and no differences were found in the prevalence of AHR to a bronchial provocation test (either eupcanic voluntary hyperpnea (EVH) or MCh challenge) in elite adolescent swimmers, recreational adolescents with asthma, all aged 12-16 y (229). Moreover, there were no

differences in the prevalence of respiratory symptoms between the swimmers and recreational controls, leading the authors to conclude that elite swimmers do not develop respiratory symptoms, airway inflammation, and AHR until later in their swimming careers (229). Analysis of elite swimmers aged 15-25 y may support this notion, as both male and female swimmers had a very high prevalence of lower respiratory symptoms and AHR in response to both MCh and EVH challenges (71) that exceeded the prevalence in winter sport athletes and recreational controls (233). Longitudinal analysis of swimmers from childhood to young adulthood is needed to determine if this later-onset of AHR is indeed the case.

In this thesis, only those with no history of asthma, reactive airway disease, other lung problems, or use of an inhaler were included. Over the study period, no swimmers were diagnosed with asthma or prescribed an inhaler due to exercise-induced bronchoconstriction. Moreover, the ratio FEV₁/FVC was nearly identical between the groups and did not change over time, with all swimmers having a ratio greater than or equal to 0.80. This suggests that any exposure to chlorine during the study period for the swimmers did not affect their spirometry. Because EVH tests were not performed, it is unclear if there were any changes in clinical indicators of asthma or exercise-induced bronchoconstriction. It is also unknown if the findings of this thesis would be different if young swimmers with a history of asthma, reactive airway disease, or use of an inhaler were included.

4.3 Methodological improvements

4.3.1 Sample size and study duration

Prior to recruitment, a sample size of 16 subjects per group was determined to be necessary to detect an effect size of 0.92 using a significance level of α =0.05 and power of β =0.80 (G*Power

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3.1.9.2; http://www.gpower.hhu.de). The effect size was estimated from the average change in TLC after one year in young, competitive female swimmers aged 10.8 years (15) compared to similarly-aged, healthy, normal children from a separate study (57). Moreover, in anticipation of a 25% dropout rate, 20 subjects per group was proposed to be required to appropriately power this semi-longitudinal analysis. Because only 21 subjects total were used in the final analysis, it is possible that this thesis was underpowered to detect the estimated effect size. Furthermore, the effect size used in the sample size estimate was extracted from one-year longitudinal studies; however, this thesis only lasted 7-8 months. Therefore, the magnitude of the effect size between the groups in this thesis was even smaller and a sample size greater than 16 subjects per group would have been necessary. This further highlights the possibility that the study was underpowered.

While increasing the number of subjects and the duration of time between visits (i.e., at least one year) are two definitive methodological improvements, it is interesting to speculate how these would have affected the results of this thesis. The initial differences in pulmonary function between the swimmers and controls were so great (e.g., TLC was, on average, ~800 ml larger in the swimmers) that they were unlikely to be affected by a larger sample size. On the other hand, the changes from PRE to POST were similar between the groups. It is unknown if a larger sample size would have caused the changes to be larger in swimmers or only reduce the variability such that the same difference in means became statistically significant. In terms of duration, a longer period between visits would have led to a disproportionately greater amount of lung growth in swimmers. It is also important to recall that the measured changes in TLC were similar to expected amounts of growth for both swimmers and controls. Lastly, larger and longer studies by Andrew

et al. (11), Baxter-Jones and Helms (17), and Engstrom *et al.* (9), have observed similar results to this thesis. Conversely, smaller (Courteix *et al.* (18)) or similarly-sized (Zinman and Gaultier (15)) studies have observed differences between swimmers and controls over the course of 1 year of intense swim training. Thus, whether these two methodological improvements would have affected the first major finding of this thesis is unknown.

The secondary purpose of this thesis was to characterize and compare their ventilatory mechanics during cycling exercise. Given its exploratory nature, effect and sample sizes were not estimated beforehand and it is not possible to determine if this thesis was appropriately powered to detect differences in the exercise responses. However, once again one can speculate if the results would have been different with a larger sample size or longer duration. Given that most of the p-values for the interaction and main effects were >0.30 for the metabolic variables, these responses are unlikely to be affected by more subjects or time between visits. However, for the operational lung volumes many p-values ranged from 0.03 to 0.18 for the two-way interactions as well as the main effect for group. Thus, it is possible that a larger sample size or time between visits may have affected the statistical significance and therefore the interpretation of the operating lung volumes throughout exercise.

4.3.2 Sex-based differences

Given the extensive literature on female swimmers, as well as time and cost constraints, this thesis only assessed female swimmers during the pubertal growth spurt. It is unknown if the same results would be observed in a male cohort. While a variety of sex-based differences in lung growth underlie normal dysanaptic development of the alveoli and airways (48, 49), it is not clear if mechanisms of induced postnatal lung growth differ between males and females. Thus, the inclusion of male swimmers and controls may have elucidated if the effect of competitive swimming during puberty differed in males. However, considering that no unequivocal evidence connected the respiratory challenges imposed by swimming and induced postnatal lung growth, one can speculate that competitive swimming during puberty would not affect lung development in males.

4.3.3 Control group

Comparing different exercise modalities, such as running and cross-country skiing, may elucidate if lung development is comparable between swimmers and other endurance-trained athletes. Given that this thesis found no effect of competitive swimming during puberty despite the numerous stressors that are unique to intensive swim training (i.e., submersion, the prone and supine positions, an obligatory breathing pattern, breath control drills, and the young starting age of intense training), it is difficult to hypothesize mechanisms by which land-based endurance sports could cause beneficial adaptations within the lungs. Specifically, it is not clear how intensive, land-based endurance training may cause induced postnatal lung growth through a chronic hypoxic stimulus, increased parenchymal or vascular mechanical strain, or altered hormonal mediators. Previous studies assessed lung function in land-based, endurance-trained athletes and found: absolute and %-predicted TLC, VC, and D_{L,CO} were similar between 8 collegiate male runners and controls of similar age and height (29); lung volumes were comparable between 11 Division I cross-country female runners and 10 controls matched for height and age (28), and D_{L,CO} during exercise was not different between collegiate male runners and similarlyaged male students (34). Thus, while adding land-based endurance athletes may control for the effect of chronic endurance training, it is not clear if the results of this thesis would be any different.

4.3.4 Additional measures of pulmonary structure and function

Although differences in lung structure and function were observed, it is only possible to speculate on the underlying physiological mechanisms. Further investigations measuring esophageal and gastric pressures (using an esophageal balloon), airway resistance (using full body plethysmography or the forced oscillation technique), and lung elastic static recoil could provide further insight on the mechanical (e.g., compliance, resistance, WOB) differences between swimmers and controls. This may highlight if the greater lung capacities of swimmers are related to alveolar hyperplasia, alveolar hypertrophy, increased distensibility, or another related mechanism. It could also facilitate quantification of the dysanapsis ratio without estimating Pst(L)50. Measurements of chest wall dimensions would have confirmed if the larger lungs of swimmers correlated with a larger chest wall surface area. As discussed earlier, chest wall dimensions were observed to be larger in multiple cohorts of competitive swimmers (16, 27, 29), and larger chests were correlated with a greater TLC (29). Partitioning diffusion capacity can elucidate whether the increased diffusion capacity in swimmers was solely due to larger alveolar volumes, or if there were other differences in alveolar membrane thickness, pulmonary capillary blood volume, or other structural or functional determinants of gas exchange. Lastly, measuring arterial saturation may provide insight on exercise-induced arterial hypoxemia during exercise and any possible correlation with the occurrence of ventilatory constraints.

4.4 Unresolved questions and future directions

Moving forward, three primary unresolved questions can guide future research questioning whether competitive swimming affects lung development. The first future direction is to longitudinally quantify dysanapsis (using the dysanapsis ratio, imaging, or other) in a large sample of male and female swimmers, land-based endurance athletes, and controls starting in prepubescence (e.g., 7-8 y old) and continuing into postpubescence. This will address the unresolved question: does competitive swimming exaggerate dysanaptic growth patterns? As mentioned earlier, altered dysanaptic growth patterns may highlight an effect of competitive swimming on lung development by accelerating alveolar growth without corresponding enhanced airway growth. The second future direction is to compare mechanical properties of the lungs between swimmers, land-based athletes, and controls during development, including airway resistance and conductance, lung compliance, and elastance. This will address the unresolved question: do competitive swimmers have mechanically different lungs? Lastly, the third future direction is to measure work of breathing and operating lung volumes in competitive swimmers while swimming. This will address the unresolved question: do the unique stressors of swimming affect the work of breathing and create a stimulus for respiratory muscle conditioning?

Beyond these three questions, another direction not addressed in this thesis is *why* competitive swimmers have larger lungs. This posits several interesting questions, including:

Is enhanced pulmonary function beneficial to swimmers? The second paragraph of this thesis stated that larger lungs may be beneficial to swimmers because of increased FRC (which may act as a reservoir for gas exchange, thereby attenuating oscillations in arterial blood gases between breaths), improved ventilatory capacity, and greater buoyancy in the water to decrease drag. First, it was mentioned earlier that the cardiovascular system appears to be able to maintain \dot{Q} and S_aO_2 and compensate for any respiratory limitations while swimming. Therefore, it is not clear to what extent an increased FRC is beneficial to swimmers. Second, in this thesis, \dot{V}_{ECAP} was similar between swimmers and controls because of the same EELV. It is not clear how the MEFV

and operating lung volumes differ when in the water. Lastly, the energetics of swimming are complicated (234). Yet, it has been suggested that arm position may be of more importance to buoyancy characteristics (specifically the distance between the centres of mass and buoyancy) than lung volume (235). Future work is necessary to clarify how larger lungs contribute to improved swimming performance.

If self-selection occurs, do children self-select into swimming because of favourable genetic endowments beyond pulmonary function such as chest size, shoulder girth, or alternative anthropometric characteristics? It is possible that benefits relating to larger chests or alternative anthropometric advantages, specifically with regards to buoyancy or drag, may be the favourable endowments that lead to self-selection into swimming. In this case, since chest wall and lung growths are tightly coupled, by necessity swimmers with larger chests would also have larger lungs. In other words, the direction of causality can be questioned: is it that children with larger lungs become swimmers, or that children who became swimmers also happen to have larger lungs?

If swimmers have larger lungs because of genetic endowment, at what point are these favourable genetic endowments important? Miller et al. measured $D_{L,CO}$ in 22 collegiate swimmers and found that most successful swimmers have an above-average $D_{L,CO}$ (35). However, the authors observed no difference in performance between those with lower (<100%) and higher (\geq 110%) %-predicted $D_{L,CO}$, nor did they find a relationship between performance $D_{L,CO}$ or VC. It is possible that, while large lungs may be a prerequisite for becoming a top swimmer (12), they do not differentiate performance between top swimmers. Thus, it is possible that the favourable genetic endowments may be more important at a young age when entering the sport and relatively less important at an older age when one is already a top swimmer. This highlights the Matthew effect whereby the favourable genetic endowments, which may have originally led to self-selection into

swimming, may also lead to early success and therefore subsequent opportunities and affordances that lead to further success (236). This may be particularly important in swimming where the relative age effect has a limited effect on age-group swimming performance (237, 238), and other factors such as swimming technique and race distance are more predictive of performance (237).

CONCLUSION

By the time they reach puberty, competitive swimmers already have enhanced lung function compared to healthy controls regardless of the age they started swimming or number of years of experience. One season of training did not further accentuate this enhanced function, and no associations between changes in lung function and swim training volume were found. Moreover, detailed discussion of physiological development of the lungs and the respiratory challenges imposed by swimming provided no unequivocal evidence that swimming can alter lung development. Interestingly, swimmers experienced similar ventilatory constraints during cycling exercise despite having larger lungs. This is the first study to longitudinally and comprehensively measure lung function and ventilatory mechanics in pubescent, female swimmers compared to matched healthy controls. It suggests that <u>competitive swimming during puberty does not affect lung development</u> and that <u>competitive swimmers experience similar ventilatory constraints during constraints during cycling exercise</u>. This thesis also furthers our understanding of pulmonary adaptations to exercise and lung development during adolescence.

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APPENDICES

Appendix A: Individual subject data – group tables

Participant		Age (y)	Height (cm)	Mass (kg)	BMI (kg/m ²)	BSA (m ²)	SMR pubic hair	SMR breasts	Hemoglobin (g/dL)
S01	Pre	13.8	163.1	61.3	23.0	1.67	5	4	16.6
	Post	14.4	164.4	59.7	22.1	1.65	5	4	15.6
S02	Pre	12.5	160.9	44.7	17.3	1.41	3	4	12.9
	Post	13.2	164.6	51.1	18.9	1.53	4	4	12.8
S04	Pre	13.7	177.9	71.2	22.5	1.88	4	4	14.4
	Post	14.3	177.9	70.8	22.4	1.87	4	4	14.4
S05	Pre	12.3	166.5	59.3	21.4	1.66	4	4	13.3
	Post	12.9	168.4	65.8	23.2	1.75	5	5	14.2
S06	Pre	12.2	157.9	43.5	17.4	1.38	4	4	13.4
	Post	12.8	161.2	50.2	19.3	1.50	4	4	13.8
S07	Pre	12.7	167.0	67.5	24.2	1.77	4	4	13.4
	Post	13.3	168.0	71.8	25.4	1.83	5	4	14.6
S08	Pre	11.6	153.3	46	19.6	1.40	3	2	13.6
	Post	12.3	156.3	48.1	19.7	1.45	4	3	14.4
S09	Pre	11.3	147.3	39.2	18.1	1.27	2	2	11.5
	Post	11.9	151.2	44.5	19.5	1.37	2	4	12
S10	Pre	12.1	157.0	45.3	18.4	1.41	1	3	11.5
	Post	12.6	160.0	48.9	19.1	1.47	3	4	13.7
S11	Pre	12.3	162.5	52.4	19.8	1.54	4	4	14.1
	Post	12.8	163.1	56.4	21.2	1.60	4	4	13.2
S12	Pre	12.3	161.2	45.7	17.6	1.43	4	4	11.4
	Post	12.9	162.7	46.6	17.6	1.45	4	5	12.7

BMI, body mass index; BSA, body surface area; SMR, sexual maturity rating.

Participant		Age (y)	Height (cm)	Mass (kg)	BMI (kg/m ²)	BSA (m ²)	SMR pubic hair	SMR breasts	Hemoglobin (g/dL)
C01	Pre	11.1	147.0	43.4	20.1	1.33	2	2	13.3
	Post	11.7	149.3	46.3	20.8	1.39	2	3	13.4
C02	Pre	12.2	153.7	40.2	17.0	1.31	3	3	13.6
	Post	12.8	159.1	45.3	17.9	1.41	4	4	17
C03	Pre	12.8	166.0	47.8	17.3	1.48	3	3	13.4
	Post	13.5	168.9	51.4	18.0	1.55	3	4	14.2
C04	Pre	14.2	164.2	49.9	18.5	1.51	3	4	14.6
	Post	14.9	166.7	53.8	19.4	1.58	5	4	14
C05	Pre	11.4	145.6	35.9	16.9	1.20	1	1	13.4
	Post	12.1	149.3	37.5	16.8	1.25	3	3	13.7
C08	Pre	13.6	164.6	49.5	18.3	1.50	4	4	13.6
	Post	14.2	167.6	56.6	20.1	1.62	4	4	13.4
C09	Pre	13.5	158.9	48.9	19.4	1.47	4	4	13.5
	Post	141	161.1	51.9	20.0	1.52	4	4	13.5
C10	Pre	14.3	164.9	47.4	17.4	1.47	3	3	13.4
	Post	14.8	165.4	48.4	17.7	1.49	3	3	13.4
C11	Pre	14.3	160.8	45.5	17.6	1.43	3	3	12.1
	Post	14.8	161.9	47.7	18.2	1.46	3	3	12.2
C12	Pre	14.8	156.9	54.9	22.3	1.55	5	5	13.7
	Post	15.5	157.7	54.7	22.0	1.55	5	5	12.3

Table $2\underline{2}$ – Individual anthropometric data for CON

BMI, body mass index; BSA, body surface area; SMR, sexual maturity rating.

Participant	#1	#2	#3	#4	#5	#6	#7	#8	#9	Average
	All activities	PE class	Recess	Lunch	After school	Evenings	Weekend	Typical week	Typical week	
S01										
S02	1.6	3	3	1	3	4	4	5	4.4	3.22
S 04	1.9	4	1	1	5	4	2	1	3.0	2.55
S05	1.5	4	1	1	4	1	2	4	2.7	2.36
S 06	1.9	5	4	1	5	5	2	5	4.3	3.69
S 07	1.6	4	3	4	3	2	2	4	3.6	3.01
S08	2.0	4	3	1	4	3	3	4	3.4	3.04
S09	1.3	4	3	3	5	1	3	4	4.3	3.18
S 10	2.0	5	2	3	1	4	2	4	4.3	3.04
S11	1.5	5	2	3	1	4	3	5	5.0	3.28
S12	1.4	4	4	4	2	4	2	4	3.6	3.22
C01	2.6	4	4	4	3	3	3	4	4.0	3.52
C02	2.2	5	2	2	3	1	1	3	2.7	2.43
C03	1.9	5	-	2	4	4	3	4	4.0	3.49
C04	2.3	5	2	3	4	4	4	5	4.1	3.72
C05	2.1	5	5	4	3	3	4	3	4.1	3.69
C08	2.0	4	2	1	3	3	3	4	3.9	2.87
C09	1.2	5	2	2	5	4	3	4	3.9	3.34
C10	1.9	1	1	1	5	4	5	4	4.6	3.05
C11	2.1	1	2	1	5	3	5	4	4.4	3.06
C12	1.2	4	1	1	2	3	3	1	2.4	2.07

Table 23 – Individual physical activity questionnaire data (each question is scored out of 5)

Participant	Vi	Vigorous intensity		te intensity per week	Total	time	Guideline met?
	Frequency	Time per session (min)	Frequency	Time per session (min)	Per week (min)	Per day (min)	
S01							
S02	6.5	150	1.5	75	1088	155	YES
S04	5	120	3	100	900	129	YES
S05	3	135	5	105	930	133	YES
S06	6	150	2	60	1020	146	YES
S07	5	90	3	45	585	84	YES
S08	6	90	3	60	720	103	YES
S09	5	120	2	30	660	94	YES
S10	5	120	1	90	690	99	YES
S11	6	90	2	150	840	120	YES
S12	6	120	5	60	1020	146	YES
C01	3	120	2	180	720	103	YES
C02	5	40	3	20	260	37	NO
C03	5	120	1	150	750	107	YES
C04	7	90	7	20	770	110	YES
C05	4	105	3.5	34	540	77	YES
C08	3	180	1.5	90	675	96	YES
C09	3	45	5	180	1035	148	YES
C10	3	20	6	180	1140	163	YES
C11	2	45	6	240	1530	219	YES
C12	5	45	7	10	295	42	NO

Table 24 – Individual moderate and vigorous intensity physical activity data

Participant	Age (y)	Training history (y)	S	wimming se per weel	ssions «	Weekly sw	imming	Nor	n-swimming tr sessions	aining		B	reath control drills
			#	Distance (km)	Time (h)	Distance (km)	Time (h)	# per week	Time per session (h)	Time per week (h)	Time per week (h)	Drills	Details
S01	8.8	5.7	7	4.5	1.75	25	12	3	1	2	2	UK	Go at least 6 m underwater off the wall within sets
S02	10.0	3.2	1	3	1.25	3	1	7	2	12	0	None	-
S04	7.4	6.9	7	3.5	2	24.5	15.5	1	2	2	0.5	Other	Sprints (12.5-25m) are always done with minimum breathing, though this was regularly backstroke.
S05	6.0	6.9	6	4	1.75	24	11	0	0	0	1	UK	Sets of 8x25 working on dolphin kick distance and kicks between breaths
S06	10.1	2.8	6	4.8	1.75	30	10.5	3	1	3	4	UK	Specific metres off walls within sets
S07	10.3	3.0	5	3.5	1.5	18	9	2	1	3	1	UK, FSB, SS	
S08	9.5	2.8	6	3	1.75	17	10.5	2	1	2	2	UK	Normally go by number of kicks; typically, 5-15m off the walls; average being around 6.5-7m
S09	10.2	1.8	5	2.5	1.5	10	8	5	0	1	0.25	UK	off the wall depending on set and if she had fins
S 10	8.6	4.0	5	3.9	1.5	20.8	7.5	5	1	3.5	0.5	UK, FSB	
S11	10.1	2.8	5	4	1.5	20	7.5	5	1	2.5	1	UK, FSB	
S12	10.1	2.8	5	2.5	1.5	12	7.5	2	1	2	1.5	UK	Normally go by number of kicks; go 5-10m off the wall with the average being around 6m

Table 25 – Individual training data for SWIM

FSB, freestyle breathing pattern ("hypoxic training"); UK, underwater (dolphin or breast) kick; SS, snorkel set

Participant		I	FVC	F	EV_1	FEV ₁ /FVC	F	PEF	FEI	25-75%	FE	F _{25%}	FE	F _{50%}	FE	F _{75%}
		(1)	% pred	(1)	% pred	(%)	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred
S01	Pre	4.35	131*	3.85	129	88	8.04	115	4.26	115	6.65	127	4.77	132	2.30	121
	Post	4.54	132*	4.00	129	88	8.06	112	4.36	114	7.17	132	4.93	131	2.33	117
S02	Pre	3.57	114	3.06	109	86	6.65	100	3.96	113	5.97	122	4.52	133	1.77	101
	Post	3.72	111	3.39	113	91	7.09	102	4.13	111	6.22	120	4.27	118	2.57	135
S04	Pre	5.31	129*	4.40	119	83	7.95	104	4.35	99	7.46	126	4.67	111	2.39	100
	Post	5.43	130*	4.65	124	86	8.02	103	5.22	117	7.86	129	5.73	133	2.88	117
S05	Pre	4.13	122*	3.53	117	85	6.30	91	3.82	103	5.45	107	4.62	129	2.03	108
	Post	4.20	119	3.74	118	89	6.36	90	4.12	107	5.56	105	4.71	126	2.67	134
S06	Pre	3.16	106	2.64	99	84	6.32	98	2.47	73	4.82	103	2.67	82	1.36	82
	Post	3.68	116	2.94	103	80	6.69	99	2.63	74	5.64	114	2.93	85	1.32	74
S07	Pre	4.92	143*	4.18	136	85	7.01	100	4.31	114	6.11	118	4.90	135	2.40	124
	Post	5.03	143*	4.31	136	86	7.90	111	4.49	116	6.70	125	5.35	143	3.07	153
S08	Pre	3.63	132*	3.15	128	87	6.25	102	3.55	113	5.81	133	4.35	144	1.78	117
	Post	3.97	136*	3.36	129	85	7.09	111	3.36	101	6.60	143	3.64	114	1.90	117
S09	Pre	3.09	123*	2.55	114	83	4.89	85	2.49	85	4.35	107	2.76	99	1.32	96
	Post	3.52	131*	2.87	119	82	5.23	86	2.71	87	4.32	99	2.97	100	1.41	95
S 10	Pre	3.42	117	2.79	106	81	6.03	94	2.56	77	5.08	110	2.96	92	1.23	76
	Post	3.74	121	3.05	110	82	6.45	97	2.83	81	5.47	113	3.23	96	1.74	100
S11	Pre	4.02	126*	3.49	122	87	5.88	88	3.86	109	5.28	108	4.34	127	2.21	124
	Post	4.13	127*	3.44	118	83	6.76	99	3.33	92	5.17	103	3.96	113	1.84	100
S12	Pre	3.50	112	3.10	110	88	5.95	90	3.50	100	5.25	108	3.80	112	2.16	123
	Post	3.71	114	3.27	112	88	7.05	103	4.22	116	6.38	127	4.52	129	2.36	129

Table 26 – Individual spirometry data for SWIM

*Above or **below the limits of abnormality in percent predicted, based on equations for healthy children and adolescents. FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow.

Participant		F	FVC	F	EV_1	FEV ₁ /FVC	P	ΈF	FEF	25-75%	FE	F _{25%}	FE	F50%	FE	F _{75%}
		(1)	% pred	(1)	% pred	(%)	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred	$(l \cdot s^{-1})$	% pred
C01	Pre	2.53	102	2.09	94	83	4.77	84	1.92	66**	3.94	97	2.11	76	1.09	80
	Post	2.68	103	2.23	96	83	5.00	85	2.03	67	4.15	98	2.28	79	1.18	82
C02	Pre	2.97	106	2.57	102	86	5.47	88	2.97	92	5.28	117	3.44	111	1.36	88
	Post	3.31	108	2.85	103	86	6.43	97	3.24	93	6.11	126	3.82	114	1.53	89
C03	Pre	2.88	85	2.40	79**	83	4.95	71	2.33	62**	3.92	76	2.60	72	1.13	59
	Post	2.88	80**	2.43	75**	84	4.98	69	2.48	63**	4.29	79	2.89	76	1.26	61
C04	Pre	3.62	106	2.47	81**	68**	5.16	72**	1.76	46**	3.14	59**	1.92	52**	0.90	46**
	Post	3.96	110	2.82	87	71**	5.74	78**	2.08	52**	3.65	65**	2.33	60**	1.10	52**
C05	Pre	2.28	93	1.95	89	86	4.49	79	2.08	72	3.59	89	2.38	87	1.03	77
	Post	2.31	88	2.00	85	87	5.08	85	2.06	68	3.60	84	2.35	80	1.31	90
C08	Pre	3.29	97	2.87	94	87	6.34	90	3.17	84	4.95	94	3.45	94	1.73	90
	Post	3.78	106	3.24	101	86	6.51	89	3.5	89	6.14	111	3.79	98	1.81	87
C09	Pre	3.87	124	3.36	120	87	6.9.0	102	3.8	108	6.08	122	4.22	123	2.00	113
	Post	3.86	119	3.33	114	86	6.94	100	3.65	100	6.37	123	3.96	111	1.76	94
C10	Pre	3.46	100	2.62	84	76**	5.93	83	2.14	56**	4.37	81	2.53	67	0.94	47**
	Post	3.58	102	2.57	81**	72**	5.87	80	1.81	46**	4.02	72**	2.12	55**	0.81	39**
C11	Pre	2.96	91	2.68	92	90	6.2.0	89	3.22	88	6.10	117	3.57	99	1.66	89
	Post	3.11	93	2.94	97	94	6.48	91	3.62	96	6.45	120	3.91	106	2.08	107
C12	Pre	3.47	111	3.11	111	90	6.81	99	4.04	113	6.79	132	4.54	130	2.01	112
	Post	3.35	105	3.00	104	90	6.92	98	3.99	109	6.78	128	4.41	122	1.97	106

Table 27 – Individual spirometry data for CON

*Above or **below the limits of abnormality in percent predicted, based on equations for healthy children and adolescents. FVC, forced vital capacity; FEV₁, forced expiratory volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow.

Table 28 – Individual lung volume data for SWIM

Participant]	ГLC	FRC			RV	VC	FRC/TLC	RV/TLC
		(1)	% pred	(1)	% pred	(1)	% pred	(1)	(au)	(au)
S01	Pre	5.28	116	2.84	123	1.17	103	4.11	0.54	0.22
	Post	5.58	117	2.90	119	1.19	99	4.39	0.52	0.21
S02	Pre	4.49	106	2.18	104	1.15	114	3.34	0.49	0.26
	Post	4.66	102	2.46	108	1.15	104	3.51	0.53	0.25
S04	Pre	6.22	108	2.98	103	1.25	88	4.97	0.48	0.20
	Post	6.55	111	2.83	93	1.28	85	5.27	0.43	0.20
S05	Pre	4.75	104	2.15	97	0.80	75	3.95	0.45	0.17
	Post	5.16	107	2.66	112	0.95	83	4.21	0.52	0.18
S06	Pre	4.01	100	1.57	79	0.94	98	3.07	0.39	0.23
	Post	4.44	103	2.08	97	0.83	80	3.61	0.47	0.19
S 07	Pre	5.71	122*	2.34	102	1.04	94	4.67	0.41	0.18
	Post	5.96	123*	2.42	101	1.05	90	4.91	0.41	0.18
S08	Pre	4.34	117	2.07	113	0.87	98	3.47	0.48	0.20
	Post	4.86	123*	2.31	118	0.92	97	3.94	0.48	0.19
S09	Pre	3.83	113	1.83	108	0.90	109	2.93	0.48	0.23
	Post	4.38	121	1.87	103	0.91	103	3.47	0.43	0.21
S10	Pre	4.37	110	2.25	115	1.01	107	3.36	0.51	0.23
	Post	4.72	112	2.63	127	1.13	112	3.59	0.56	0.24
S11	Pre	4.59	107	1.66	79	0.73	72	3.86	0.36	0.16**
	Post	4.75	107	1.68	77	0.75	70	4.00	0.35	0.16**
S12	Pre	4.39	104	2.09	100	1.03	102	3.36	0.48	0.23
	Post	4.81	109	2.53	116	1.33	125	3.48	0.53	0.28

*Above or **below the limits of abnormality in percent predicted, based on equations for healthy children and adolescents. TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; VC, vital capacity.

Table 29 – Individual lung volume data for CON

Participant		TLC FRC		FRC]	RV	VC	FRC/TLC	RV/TLC	
		(1)	% pred	(1)	% pred	(1)	% pred	(1)	(au)	(au)
C01	Pre	3.39	101	1.75	104	0.78	96	2.61	0.52	0.23
	Post	3.40	97	1.53	87	0.74	86	2.66	0.45	0.22
C02	Pre	3.67	97	1.94	103	0.74	81	2.93	0.53	0.20
	Post	3.92	94	1.83	88	0.69	68	3.23	0.47	0.18
C03	Pre	3.88	84	2.15	94	1.11	100	2.77	0.55	0.29
	Post	4.02	81**	2.47	100	1.16	96	2.86	0.61	0.29
C04	Pre	4.46	95	2.56	107	1.17	99	3.29	0.57	0.26
	Post	5.09	101	2.58	99	1.31	101	3.78	0.51	0.26
C05	Pre	3.11	94	1.81	108	0.86	105	2.25	0.58	0.28
	Post	3.13	88	1.61	90	0.87	99	2.26	0.51	0.28
C08	Pre	4.22	91	2.34	100	1.11	97	3.11	0.55	0.26
	Post	4.98	100	2.49	98	1.26	101	3.72	0.50	0.25
C09	Pre	4.48	106	2.25	105	0.71	67	3.77	0.50	0.16**
	Post	4.68	105	2.51	110	0.78	69	3.9	0.54	0.17
C10	Pre	4.32	91	2.43	100	0.99	82	3.33	0.56	0.23
	Post	4.59	94	2.49	98	1.08	86	3.51	0.54	0.24
C11	Pre	3.78	85	2.47	108	1.26	111	2.52	0.65	0.33
	Post	4.04	87	2.43	101	1.37	115	2.67	0.60	0.34
C12	Pre	4.01	94	2.21	99	0.83	75	3.18	0.55	0.21
	Post	4.07	92	2.12	91	0.86	74	3.21	0.52	0.21

*Above or **below the limits of abnormality in percent predicted, based on equations for healthy children and adolescents. TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; VC, vital capacity.

articipant		$D_{L,CO}$		$D_{L,CO}c$			VA	$D_{L,CO}c/V_A$	
		$(mL \cdot min^{-1} \cdot mmHg^{-1})$	% pred	$(mL \cdot min^{-1} \cdot mmHg^{-1})$	% pred	(1)	% pred	$(mL \cdot min^{-1} \cdot mmHg^{-1} \cdot l^{-1})$	% pred
S01	Pre	24.89	124	22.91	114	5.15	121	4.45	88
	Post	28.37	137	26.72	129	5.44	124	4.91	98
S02	Pre	26.72	140	27.14	142	4.39	110	6.18	121
	Post	25.30	125	25.79	127	4.55	106	5.67	113
S04	Pre	27.32	114	26.54	111	6.07	113	4.38	92
	Post	24.73	102	24.02	99	6.40	118	3.75	79
S05	Pre	20.40	100	20.47	100	4.62	106	4.43	89
	Post	23.49	111	22.94	109	5.02	110	4.57	92
S 06	Pre	20.22	110	20.22	110	3.91	103	5.17	100
	Post	20.75	107	20.51	106	4.33	107	4.73	93
S07	Pre	26.20	127	26.20	127	5.56	125	4.71	95
	Post	26.07	123	25.19	119	5.80	127	4.34	88
S 08	Pre	23.43	136	23.29	136	4.24	122	5.49	104
	Post	25.82	143	25.08	139	4.75	128	5.28	102
S09	Pre	18.69	118	19.96	126	3.75	119	5.33	99
	Post	22.00	130	23.06	137	4.29	126	5.38	101
S10	Pre	21.29	117	22.74	125	4.27	114	5.33	103
	Post	22.67	120	22.46	118	4.61	117	4.87	95
S11	Pre	23.52	121	23.04	119	4.48	110	5.14	101
	Post	23.22	117	23.36	118	4.63	111	5.05	100
S12	Pre	23.53	123	25.23	132	4.29	107	5.88	115
	Post	25.26	128	25.83	131	4.71	113	5.48	108

Table 30 – Individual diffusion capacity data for SWIM

D_{L,CO}, diffusion capacity of the lung for carbon monoxide; D_{L,CO}c, D_{L,CO} corrected for hemoglobin; V_A; alveolar volume.

	$D_{L,CO}$		D _{L,CO} c			V _A	$D_{L,CO}c/V_A$	
	$(mL \cdot min^{-1} \cdot mmHg^{-1})$	% pred	$(mL \cdot min^{-1} \cdot mmHg^{-1})$	% pred	(1)	% pred	$(mL \cdot min^{-1} \cdot mmHg^{-1} \cdot l^{-1})$	% pred
Pre	18.31	116	18.37	116	3.29	105	5.58	103
Post	17.09	104	17.09	104	3.29	100	5.19	97
Pre	18.88	108	18.76	108	3.59	101	5.23	100
Post	18.95	101	17.30	92	3.82	98	4.52	88
Pre	22.40	109	22.40	109	3.78	86	5.93	119
Post	22.95	107	22.42	105	3.91	84	5.73	116
Pre	22.16	108	21.41	104	4.35	100	4.92	98
Post	24.45	114	24.02	112	4.97	108	4.84	97
Pre	18.69	120	18.69	120	3.03	99	6.17	114
Post	16.76	101	16.61	100	3.04	92	5.46	102
Pre	22.60	111	22.46	110	4.11	95	5.47	109
Post	25.67	120	25.67	120	4.85	105	5.29	107
Pre	23.88	126	23.80	125	4.37	111	5.44	106
Post	23.90	121	23.83	121	4.57	110	5.22	102
Pre	20.80	100	20.80	100	4.22	96	4.93	98
Post	21.30	101	21.30	101	4.49	100	4.75	95
Pre	20.46	104	21.36	108	3.68	89	5.81	114
Post	20.95	104	21.79	108	3.93	92	5.54	109
Pre	19.43	102	19.25	101	3.89	99	4.95	95
Post	19.25	99	19.95	103	3.95	98	5.05	98
	Pre Post Pre Post Pre Post Pre Post Pre Post Pre Post Pre Post Pre Post Pre	DLCO (mL·min ⁻¹ ·mmHg ⁻¹) Pre 18.31 Post 17.09 Pre 18.88 Post 18.95 Pre 22.40 Post 22.95 Pre 22.16 Post 24.45 Pre 16.76 Pre 23.88 Post 25.67 Pre 23.88 Post 25.67 Pre 23.88 Post 25.67 Pre 23.88 Post 21.30 Pre 20.80 Post 21.30 Pre 20.46 Post 20.95 Pre 19.43 Post 19.25	DLCO (mL·min ⁻¹ ·mmHg ⁻¹) % pred Pre 18.31 116 Post 17.09 104 Pre 18.88 108 Post 18.95 101 Pre 22.40 109 Post 22.95 107 Pre 22.16 108 Post 22.95 107 Pre 22.16 108 Post 22.40 109 Post 22.95 107 Pre 22.16 108 Post 24.45 114 Pre 18.69 120 Post 25.67 101 Pre 23.88 126 Post 23.90 121 Pre 20.80 100 Post 21.30 101 Pre 20.46 104 Post 20.95 104 Pre 19.43 102 <tr tbre<="" td=""></tr>	$D_{L,CO}$ $D_{L,COC}$ $(mL \cdot min^{-1} \cdot mmHg^{-1})$ % pred $(mL \cdot min^{-1} \cdot mmHg^{-1})$ Pre18.3111618.37Post17.0910417.09Pre18.8810818.76Post18.9510117.30Pre22.4010922.40Post22.9510722.42Pre22.1610821.41Post24.4511424.02Pre18.6912018.69Post16.7610116.61Pre22.6011122.46Post25.6712025.67Pre23.8812623.80Post23.9012123.83Pre20.8010020.80Post21.3010121.30Pre20.4610421.36Post20.9510421.79Pre19.4310219.25Post19.259919.95	DL.CO DL.coc(mL·min ⁻¹ ·mmHg ⁻¹) % pred (mL·min ⁻¹ ·mmHg ⁻¹) % pred (mL·min ⁻¹ ·mmHg ⁻¹) % pred Pre 18.31 116 18.37 116 Post 17.09 104 17.09 104 Pre 18.88 108 18.76 108 Post 18.95 101 17.30 92 Pre 22.40 109 22.40 109 Post 22.95 107 22.42 105 Pre 22.16 108 21.41 104 Post 24.45 114 24.02 112 Pre 18.69 120 18.69 120 Post 16.76 101 16.61 100 Pre 22.60 111 22.46 110 Post 25.67 120 25.67 120 Pre 23.88 126 23.80 121 Pre 20.80 100 20.80 100	$D_{L,CO}$ $D_{L,COC}$ $(mL \cdot min^{-1} \cdot mmHg^{-1})$ % pred(nl.Pre18.3111618.371163.29Post17.0910417.091043.29Pre18.8810818.761083.59Post18.9510117.30923.82Pre22.4010922.401093.78Post22.9510722.421053.91Pre22.1610821.411044.35Post24.4511424.021124.97Pre18.6912018.691203.03Post16.7610116.611003.04Pre23.8812623.801254.37Post23.9012123.831214.57Pre20.8010020.801004.22Post21.3010121.301014.49Pre20.4610421.361083.68Post20.9510421.791083.93Pre19.4310219.251013.95	D_{LCO} D_{LCOC} V_A (mL·min ⁻¹ ·mmHg ⁻¹)% pred(n)% predPre18.3111618.371163.29105Post17.0910417.091043.29100Pre18.8810818.761083.59101Post18.9510117.30923.8298Pre22.4010922.401093.7886Post22.9510722.421053.9184Pre22.1610821.411044.35100Post24.4511424.021124.97108Pre18.6912018.691203.0399Post16.7610116.611003.0492Pre23.8812623.801254.37111Post23.9012123.831214.57110Pre20.8010020.801004.2296Post21.3010121.301014.49100Pre20.4610421.361083.6889Post20.9510421.791083.9392Pre19.4310219.251013.8999Post19.259919.951033.9598	D_{LCO} D_{LCOC} V_A D_{LCOC}/V_A (mL·mir ¹ ·mmHg ⁻¹)% pred(n)% pred(n)% pred(mL·min ⁻¹ ·mmHg ⁻¹ ·1 ⁻¹)Pre18.3111618.371163.291055.58Post17.0910417.091043.291005.19Pre18.8810818.761083.591015.23Post18.9510117.30923.82984.52Pre22.4010922.401093.78865.93Post22.9510722.421053.91845.73Pre22.1610821.411044.351004.92Post24.4511424.021124.971084.84Pre18.6912018.691203.03996.17Post16.7610116.611003.04925.46Pre22.6011122.461104.11955.47Post25.6712025.671204.851055.29Pre23.8812623.801254.371115.44Post23.9012123.831214.571105.22Pre20.8010020.801004.22964.93Post21.3010121.301014.491004.75Pre20.461

Table 31 – Individual diffusion capacity for CON

D_{L,CO}, diffusion capacity of the lung for carbon monoxide; D_{L,CO}c, D_{L,CO} corrected for hemoglobin; V_A; alveolar volume.

Participant		PIMA	AX	PE _M	AX
		(cm H ₂ O)	% pred	$(cm H_2O)$	% pred
S01	Pre	71	72	146	125
	Post	80	81	158	130
S02	Pre	55	64	106	99
	Post	83	91	102	91
S04	Pre	124	113	121	105
	Post	150	136	137	114
S05	Pre	68	71	131	124
	Post	97	95	135	122
S06	Pre	121	145	117	112
	Post	126	141	123	112
S07	Pre	122	120	104	96
	Post	118	111	113	100
S08	Pre	75	90	101	100
	Post	91	106	119	113
S09	Pre	87	112	92	94
	Post	99	120	108	105
S10	Pre	66	78	88	84
	Post	82	93	123	114
S11	Pre	101	112	119	113
	Post	113	120	151	138
S12	Pre	64	75	104	98
	Post	95	108	124	112

Table 32 – Individual maximal static pressure data for SWIM

 PI_{MAX} , maximum inspiratory pressure; PE_{MAX} , maximum expiratory pressure.

•	Participant		PI _M	AX	PE _M	AX	
			(cm H ₂ O)	% pred	$(cm H_2O)$	% pred	
	C01	Pre	73	92	100	103	
		Post	66	80	108	107	
	C02	Pre	105	130	100	95	
		Post	122	142	94	86	
	C03	Pre	71	80	87	79	
		Post	64	69	77	67	
	C04	Pre	78	84	101	85	
		Post	101	104	119	95	
	C05	Pre	79	105	98	98	
		Post	88	113	117	112	
	C08	Pre	56	61	51	44	
		Post	95	97	111	93	
	C09	Pre	97	108	80	70	
		Post	98	106	94	79	
	C10	Pre	76	84	86	72	
		Post	59	64	109	88	
	C11	Pre	23	26	68	57	
		Post	41	45	82	66	
	C12	Pre	48	50	78	63	
		Post	53	55	66	51	

Table 33 – Individual maximal static pressure data for CON

 PI_{MAX} , maximum inspiratory pressure; PE_{MAX} , maximum expiratory pressure

Participant		Duration (min)	Work rate (W)	Work rate (W·kg ⁻¹)	HR (bpm)	RPE	V _T (l)	$f_{\rm B}$	[.] V _E (l∙min ⁻¹)	^V O ₂ (l·min ⁻¹)	\dot{VO}_2 (ml·kg ⁻¹ ·min ⁻¹)	V̈O ₂ (% pred)	VCO ₂ (l·min ⁻¹)	RER	EFL (%)	EFL?	V _E ∕V _{ECAP} (%)
S01	Pre	15.8	180	2.9	202	10	1.30	94	120.4	2.42	39.4	119	2.62	1.09	0	NO	77
	Post	18.5	220	3.7	205	10	1.58	88	137.4	2.46	41.2	124	2.97	1.21	0	NO	69
S02	Pre	10.6	140	3.1	194	9	1.39	44	59.7	1.68	37.7	108	1.77	1.05	58	YES	94
	Post	15.0	180	3.5	191	10	1.79	49	87.1	2.33	45.6	133	2.53	1.09	37	YES	85
S04	Pre	18.2	200	2.8	182	10	2.21	43	95.3	2.59	36.4	112	2.89	1.12	0	NO	46
	Post	18.0	200	2.8	182	10	2.17	38	83.3	2.63	37.1	114	2.82	1.08	0	NO	44
S05	Pre	10.0	120	2.0	181	4	1.48	37	54.4	1.71	28.8	86	1.78	1.04	0	NO	40
	Post	14.0	160	2.4	195	10	1.79	45	79.7	2.14	32.6	99	2.39	1.12	0	NO	58
S06	Pre	13.1	160	3.7	182	9.5	1.23	71	86.4	2.16	49.6	141	2.49	1.16	44	YES	91
	Post	16.0	180	3.6	185	7	1.62	61	98.2	2.34	46.7	136	2.77	1.18	60	YES	99
S07	Pre	19.3	220	3.3	208	10	2.24	48	106.9	2.70	40.0	122	3.02	1.12	0	NO	66
	Post	18.0	200	2.8	208	10	2.30	52	118.3	2.69	37.4	115	3.09	1.15	0	NO	73
S08	Pre	13.4	160	3.5	205	10	1.59	65	101.4	2.25	49.0	141	2.60	1.17	57	YES	94
	Post	16.3	200	4.2	201	10	1.69	71	118.3	2.69	56.0	162	3.07	1.14	73	YES	120
S09	Pre	11.5	140	3.6	186	10	1.58	44	70.1	1.79	45.8	128	2.16	1.21	16	YES	71
	Post	16.0	180	4.0	194	10	1.85	52	96.3	2.19	49.1	140	2.68	1.23	47	YES	89
S 10	Pre	13.8	160	3.5	185	10	1.47	46	68.3	2.19	48.2	138	2.28	1.05	0	NO	66
	Post	16.4	200	4.1	191	10	1.55	60	93.2	2.53	51.7	150	2.77	1.11	49	YES	88
S11	Pre	15.8	180	3.4	195	10	1.58	61	96.3	2.54	48.4	142	2.87	1.13	30	YES	90
	Post	14.9	180	3.2	195	8	1.52	60	90.4	2.04	36.2	108	2.38	1.17	39	YES	87
S12	Pre	15.8	180	3.9	197	10	1.42	58	81.8	2.21	48.5	139	2.47	1.12	0	NO	70
	Post	18.0	200	4.3	201	10	1.61	63	102.0	2.56	55.0	159	2.96	1.16	0	NO	65

Table 34 – Individual maximal exercise data for SWIM

HR, heart rate; bpm, beats per minute; RPE, rating of perceived exertion; V_T , tidal volume; f_B , breathing frequency; \dot{V}_E , expired minute ventilation; $\dot{V}O_2$, oxygen consumption; $\dot{V}CO_2$, carbon dioxide production; RER, respiratory exchange ratio; EFL, expiratory flow limitation; \dot{V}_{ECAP} , ventilatory capacity.

Participant		Duration (min)	Work rate (W)	Work rate (W·kg ⁻¹)	HR (bpm)	RPE	V _T (l)	$f_{\rm B}$	[↓] V _E (l·min ⁻¹)	[.] VO ₂ (l∙min ⁻¹)	\dot{VO}_2 (ml·kg ⁻¹ ·min ⁻¹)	V̈O ₂ (% pred)	VCO ₂ (l·min ⁻¹)	RER	EFL (%)	EFL?	V _E ∕V _{ECAP} (%)
C01	Pre	9.8	120	2.8	186	10	1.09	57	61.8	1.64	37.7	107	1.59	0.98	0	NO	63
	Post	12.0	140	3.0	186	10	1.15	58	67.0	1.68	36.4	105	1.88	1.11	50	YES	83
C02	Pre	11.4	140	3.5	204	9.5	1.26	46	57.4	1.73	42.9	120	1.84	1.07	0	NO	47
	Post	14.5	180	4.0	208	9.5	1.65	48	79.1	2.21	48.7	140	2.45	1.12	9	YES	75
C03	Pre	13.7	160	3.3	183	8	1.28	60	76.4	1.98	41.3	120	2.19	1.11	6	YES	84
	Post	14.0	160	3.1	185	8	1.27	70	88.2	2.05	39.9	117	2.27	1.11	0	NO	78
C04	Pre	17.9	200	4.0	195	9	1.53	54	81.8	2.23	44.7	130	2.56	1.15	60	YES	101
	Post	20.0	220	4.1	197	10	1.69	55	92.0	2.54	47.3	140	3.03	1.19	35	YES	79
C05	Pre	8.9	120	3.3	199	10	1.06	40	42.5	1.45	40.3	110	1.59	1.10	0	NO	58
	Post	11.8	140	3.7	198	8	1.15	54	62.3	1.67	44.5	123	2.03	1.22	20	YES	77
C08	Pre	13.2	160	3.2	199	10	1.61	60	95.3	2.03	41.0	120	2.56	1.26	3	NO	82
	Post	14.5	180	3.2	197	10	1.59	63	98.8	2.05	36.2	108	2.57	1.27	28	YES	83
C09	Pre	12.7	160	3.3	201	7	1.69	42	70.6	1.87	38.2	111	2.18	1.17	0	NO	49
	Post	13.5	160	3.1	203	8	1.80	54	97.6	2.01	38.7	114	2.60	1.30	0	NO	69
C10	Pre	16.0	180	3.8	194	10	1.76	47	82.5	2.17	45.8	133	2.59	1.19	62	YES	108
	Post	18.0	200	4.1	202	10	1.90	47	89.7	2.33	48.1	139	2.72	1.17	66	YES	115
C11	Pre	12.5	160	3.5	199	9	1.58	42	65.9	1.70	37.3	107	1.98	1.17	0	NO	55
	Post	15.4	180	3.8	202	10	1.65	51	83.2	2.15	45.1	131	2.48	1.15	42	YES	88
C12	Pre	11.6	140	2.6	200	10	1.59	54	86.0	1.71	31.2	93	2.18	1.27	0	NO	49
	Post	12.0	140	2.6	203	10	1.62	58	94.9	1.98	36.1	107	2.31	1.17	26	YES	77

Table 35 – Individual maximal exercise data for CON

HR, heart rate; bpm, beats per minute; RPE, rating of perceived exertion; V_T , tidal volume; f_B , breathing frequency; \dot{V}_E , expired minute ventilation; $\dot{V}O_2$, oxygen consumption; $\dot{V}CO_2$, carbon dioxide production; RER, respiratory exchange ratio; EFL, expiratory flow limitation; \dot{V}_{ECAP} , ventilatory capacity.

Participant		EILV (1)	EILV/FVC (%)	EELV (1)	EELV/FVC (%)	IRV (l)	IRV/FVC (%)	IC (l)	V _T /FVC (%)
S01	Pre	2.94	75	1.64	42	1.00	25	2.30	33
	Post	3.67	90	2.09	51	0.42	10	2.00	39
S02	Pre	2.14	86	0.75	30	0.36	14	1.75	55
	Post	2.68	82	0.89	27	0.60	18	2.39	55
S04	Pre	4.07	84	1.86	39	0.75	16	2.96	46
	Post	4.07	83	1.91	39	0.86	17	3.02	44
S05	Pre	2.65	74	1.17	33	0.94	26	2.42	41
	Post	2.80	76	1.00	27	0.90	24	2.70	48
S06	Pre	2.16	73	0.93	31	0.81	27	2.04	41
	Post	2.65	78	1.02	30	0.75	22	2.38	48
S07	Pre	3.69	86	1.45	34	0.59	14	2.83	52
	Post	3.97	86	1.67	36	0.64	14	2.94	50
S08	Pre	2.42	81	0.83	28	0.58	19	2.17	53
	Post	2.72	79	1.03	30	0.70	21	2.39	49
S09	Pre	2.38	91	0.81	31	0.25	9	1.82	60
	Post	2.86	89	1.01	31	0.35	11	2.20	58
S10	Pre	2.59	89	1.12	39	0.32	11	1.79	50
	Post	2.53	73	0.98	28	0.93	27	2.48	45
S11	Pre	2.35	69	0.77	23	1.03	31	2.61	47
	Post	2.47	66	0.95	25	1.30	34	2.82	40
S12	Pre	2.39	76	0.96	31	0.75	24	2.18	45
	Post	2.76	81	1.15	34	0.67	19	2.28	47

Table 36 - Individual maximal exercise operational lung volume data for SWIM

EILV, end-inspiratory lung volume; FVC, forced vital capacity; EELV, end-expiratory lung volume; IRV, inspiratory reserve volume; ERV, expiratory reserve volume; V_T, tidal volume.

Participant		EILV (l)	EILV/FVC (%)	EELV (1)	EELV/FVC (%)	IRV (l)	IRV/FVC (%)	IC (l)	V_T/FVC (%)
C01	Pre	1.97	82	0.89	37	0.45	18	1.53	45
	Post	1.92	78	0.77	31	0.55	22	1.70	47
C02	Pre	2.08	85	0.82	33	0.38	15	1.64	51
	Post	2.52	84	0.87	29	0.48	16	2.13	55
C03	Pre	2.26	90	0.98	39	0.27	10	1.55	51
	Post	2.33	90	1.06	41	0.26	10	1.53	49
C04	Pre	2.52	85	0.99	33	0.44	15	1.97	52
	Post	3.13	89	1.44	41	0.38	11	2.07	48
C05	Pre	1.62	79	0.57	28	0.42	21	1.47	52
	Post	1.80	79	0.65	29	0.48	21	1.63	50
C08	Pre	2.58	85	0.97	32	0.46	15	2.07	53
	Post	2.54	76	0.95	29	0.78	24	2.37	48
C09	Pre	3.12	87	1.44	40	0.46	13	2.14	47
	Post	2.84	76	1.04	28	0.88	24	2.68	48
C10	Pre	2.45	83	0.69	23	0.50	17	2.26	60
	Post	2.72	85	0.82	26	0.47	15	2.37	59
C11	Pre	2.10	83	0.52	20	0.44	17	2.02	62
	Post	2.50	89	0.85	30	0.29	11	1.94	59
C12	Pre	2.70	87	1.12	36	0.40	13	1.98	51
	Post	2.46	79	0.84	27	0.64	21	2.26	52

Table 37 - Individual maximal exercise operational lung volume data for CON

EILV, end-inspiratory lung volume; FVC, forced vital capacity; EELV, end-expiratory lung volume; IRV, inspiratory reserve volume; IC, inspiratory capacity; V_T, tidal volume.

Appendix B: Individual subject data – individual tables and figures

This appendix contains the individual subject data for the ventilatory response to exercise. Each subject has two pages. The first page displays selected anthropometric, pulmonary function, and maximal exercise data as well as their MEFV and FVL for all stages of exercise, PRE (top) and POST (bottom). The MEFV, baseline, and maximal exercise stage are bolded, and exercise stages positive for EFL are presented in dashed lines. The second page presents their EFL severity and \dot{V}_E/\dot{V}_{ECAP} in tabular form and their ventilatory mechanics; absolute operational lung volumes (top left), relative operational lung volumes (top right), ventilation and ventilatory capacity (bottom left), and breathing frequency and tidal volume (bottom right). On all graphs, PRE is represented with open symbols, POST with closed.

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	Age	SMR pubic	Height (cm)	Weight	TLC	Work rate	HR (hnm)	\dot{V}_E	$\dot{V}O_{2MAX}$	$\dot{V}CO_2$	RER
	(9)	liali	(CIII)	(kg)	(1)	(\mathbf{w})	(opin)	(1.11111)	(IIII·Kg ·IIIII)	(1.11111)	
PRE	13.8	5	163.1	61.3	5.28	180	202	120.4	39.4	2.62	1.09
POST	14.4	5	164.4	59.7	5.58	220	205	135.7	41.2	2.97	1.21



Figure 20 - S01 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	PRE MAX	200 W	220 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	0	0	-	-
	POST	0	0	0	0	0	0	0	0	0	-	0	0
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	6	15	17	18	25	27	31	44	65	77	-	-
	POST	6	13	16	20	21	26	29	37	46	-	57	69

Table 39 – S01 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$



Figure 21 – S01 ventilatory mechanics.

Table 40 – S02 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.5	3	160.9	44.7	4.49	140	194	59.7	37.7	1.77	1.05
POST	13.2	4	164.6	51.1	4.66	180	191	87.1	45.6	2.53	1.09



Figure 22 – S02 MEFV and FVL for PRE (top) and POST (bottom).

Table 41 – S02 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W
EFL (% V _T)	PRE	0	0	0	4	0	52	58	-	-
	POST	0	0	0	0	0	0	0	0	37
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	14	50	40	58	68	90	94	-	-
	POST	9	32	27	35	42	49	66	66	85



Figure 23 – S02 ventilatory mechanics.

Table 42 – S04 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	13.7	4	177.9	71.2	6.22	200	182	95.3	36.4	2.89	1.12
POST	14.3	4	177.9	70.8	6.55	200	182	83.3	37.1	2.82	1.08



Figure 24 – S04 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	200 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	0	0	0
	POST	0	0	0	0	0	0	0	0	0	0	0
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	5	10	13	16	21	20	26	31	38	43	46
	POST	6	12	17	19	24	24	29	33	38	43	44

Table 43 – S04 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$



Figure 25 – S04 ventilatory mechanics.

Table 44 – S05 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.3	5	166.5	59.3	4.75	120	181	54.4	28.8	1.78	1.04
POST	12.9	5	168.4	65.8	5.16	160	195	79.7	32.6	2.39	1.12



Figure 26 – S05 MEFV and FVL for PRE (top) and POST (bottom).

Table 45 – S05 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}.$

		REST	40 W	60 W	80 W	100 W	120 W	PRE MAX	140 W	160 W	POST MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	0	0
	POST	0	0	0	0	0	0	0	0	0	0
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	8	19	27	28	34	36	40	-	-	-
	POST	7	18	20	26	30	37	-	48	56	58



Figure 27 – S05 ventilatory mechanics.

Table 46 – S06 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.2	4	157.9	43.5	4.01	160	182	86.4	49.6	2.49	1.16
POST	12.8	4	161.2	50.2	4.44	180	185	98.2	46.7	2.77	1.18



Figure 28 – S06 MEFV and FVL for PRE (top) and POST (bottom).

Table 47 – S06 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	44	-	-
	POST	0	0	0	0	0	0	0	51	72	60
$\dot{V}_{E}/\dot{V}_{ECAP}$ (%)	PRE	12	32	38	45	52	60	67	91	-	-
	POST	14	35	41	41	47	56	56	92	115	99



Figure 29 – S06 ventilatory mechanics.

Table 48 – S07 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.7	4	167	67.5	5.71	220	208	106.9	40.0	3.02	1.12
POST	13.3	5	168	71.8	5.96	200	208	118.3	37.4	3.09	1.15



Figure 30 – S07 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	200 W	POST MAX	220 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	0	0	-	0
	POST	0	0	0	0	0	0	0	0	0	20	0	-
$\dot{V}_{E}\!/\!\dot{V}_{ECAP}\left(\%\right)$	PRE	6	18	23	24	26	30	31	35	46	63	-	66
	POST	6	21	23	30	33	41	45	58	70	74	73	-

Table 49 – S07 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$



Figure 31 – S07 ventilatory mechanics.

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	Age	SMR pubic	Height	Weight	TLC	Work rate	HR	Vе	VO _{2MAX}	VCO ₂	RFR
	(y)	hair	(cm)	(kg)	(1)	(W)	(bpm)	$(l \cdot min^{-1})$	$(ml \cdot kg^{-1} \cdot min^{-1})$	$(l \cdot min^{-1})$	KLK
PRE	11.6	3	153.3	46	4.34	160	205	101.4	49.0	2.60	1.17
POST	12.3	4	156.3	48.1	4.86	200	201	118.3	56.0	3.07	1.14



Figure 32 - S08 MEFV and FVL for PRE (top) and POST (bottom).
Table 51 – S08 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	200 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	57	-	-
	POST	0	0	0	0	0	0	0	0	59	73
$\dot{V}_{E}/\dot{V}_{ECAP}$ (%)	PRE	10	32	32	31	38	47	63	94	-	-
	POST	13	27	33	33	41	43	49	62	99	120



Figure 33 – S08 ventilatory mechanics.

Table 52 – S09 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	11.3	2	147.3	39.2	3.83	140	186	70.1	45.8	2.16	1.21
POST	11.9	2	151.2	44.5	4.38	180	194	96.3	49.1	2.68	1.23



Figure 34 – S09 MEFV and FVL for PRE (top) and POST (bottom).

Table 53 – S09 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	16	-	-	-
	POST	0	0	0	0	0	0	0	7	49	47
$\dot{V}_{E}/\dot{V}_{ECAP}$ (%)	PRE	7	26	37	38	47	54	71	-	-	-
	POST	6	25	24	26	35	39	46	68	87	89



Figure 35 – S09 ventilatory mechanics.

Table 54 – S10 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.1	1	157	45.3	4.37	160	185	68.3	48.2	2.28	1.05
POST	12.6	3	160	48.9	4.72	200	191	93.2	51.7	2.77	1.11



Figure 36 – S10 MEFV and FVL for PRE (top) and POST (bottom).

Table 55 – S10 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	-	-
	POST	0	0	0	0	0	0	0	0	29	49
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	8	16	21	33	32	50	58	66	-	-
	POST	11	20	25	29	36	45	51	61	81	88



Figure 37 – S10 ventilatory mechanics.

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13 me 36 = 811 select	ed anthronome	etric nulmon	ary function	and maximal	evercise (iata
	cu anunoponic	cure, pumon	ary runction,	and maxima	CACICISC C	Jaia

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	VO _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.3	4	162.5	52.4	4.59	180	195	96.3	48.4	2.87	1.13
POST	12.8	4	163.1	56.4	4.75	180	195	90.4	36.2	2.38	1.17



Figure 38 - S11 MEFV and FVL for PRE (top) and POST (bottom).

Table 57 – S11 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	30
	POST	0	0	0	0	0	0	0	0	39
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	16	25	20	22	34	38	56	76	90
	POST	8	30	31	34	37	50	63	77	87



Figure 39 – S11 ventilatory mechanics.

Table 58 – S12 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.3	4	161.2	45.6	4.39	180	197	81.8	48.5	2.47	1.12
POST	12.9	4	162.7	46.6	4.81	200	201	102.0	55.0	2.96	1.16



Figure 40 – S12 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	200 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	0	-	-
	POST	0	0	0	0	0	0	0	0	0	0	0
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	8	19	26	30	38	40	48	52	70	-	-
	POST	7	16	22	21	24	26	35	42	57	63	65

Table 59 – S12 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}.$



Figure 41 – S12 ventilatory mechanics.

Table 60 – C0	1 selected	anthropor	metric. 1	pulmonarv	function.	. and	maximal	exercise	data
14010 00 00	1 50100100	ununopoi	mouro,	pullional y	ranction	, unu	mannan	Chercibe	uuuu

	Age	SMR pubic	Height (cm)	Weight (kg)	TLC	Work rate	HR (bpm)	\dot{V}_E	$\dot{V}O_{2MAX}$ (ml. kg ⁻¹ . min ⁻¹)	VCO ₂	RER
	(y)	nan	(CIII)	(kg)	(1)	(\mathbf{w})	(opin)	(1.1111)	(mixg min)	(1.11111)	
PRE	11.1	2	147	43.4	3.39	120	186	61.8	37.7	1.59	0.98
POST	11.7	2	149.3	46.3	3.4	140	186	67.0	36.4	1.88	1.11



Figure 42 - C01 MEFV and FVL for PRE (top) and POST (bottom).



Table 61 – C01 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}\!.$

Figure 43 – C01 ventilatory mechanics.

Table 62 - C02 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	[.] VO _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.2	3	153.7	40.2	3.67	140	204	57.4	42.9	1.84	1.07
POST	12.8	4	159.1	45.3	3.92	180	208	79.1	48.7	2.45	1.12



Figure 44 – C02 MEFV and FVL for PRE (top) and POST (bottom).

Table 63 – C02 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	-	-
	POST	0	0	0	0	0	0	0	0	9
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	7	21	22	24	38	43	47	-	-
	POST	6	20	29	33	35	45	51	66	75



Figure 45 - C02 ventilatory mechanics.

Table 64 – C03 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	[.] VO _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	12.8	3	166	47.8	3.88	160	183	76.4	41.3	2.19	1.11
POST	13.5	3	168.9	51.4	4.02	160	185	88.2	39.9	2.27	1.11



Figure 46 – C03 MEFV and FVL for PRE (top) and POST (bottom).

Table 65 – C03 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	POST MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	6	-
	POST	0	0	0	0	0	0	0	0	1
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	10	24	36	39	49	55	70	84	-
	POST	7	20	24	35	36	49	61	78	78



Figure 47 – C03 ventilatory mechanics.

Table 66 - C04 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	[.] VO _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	14.2	3	164.2	49.9	4.46	200	195	81.8	44.7	2.56	1.15
POST	14.9	5	166.7	53.8	5.09	220	197	92.0	47.3	3.03	1.19



Figure 48 – C04 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	200 W	220 W	POST MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	3	51	60	-	-
	POST	0	0	0	0	0	0	0	0	0	0	0	35
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	14	32	41	49	57	50	68	73	94	101	-	-
	POST	6	22	26	32	34	30	42	44	55	69	79	65

Table 67 – C04 EFL severity and $\dot{V}_{E}/\dot{V}_{ECAP}.$



Figure 49 – C04 ventilatory mechanics.

Table $68 - C$	205 selected	anthro	pometri	ic, pul	monary i	function,	and r	naximal exerc	ise data.
1 33	SMD muhio	Haight	Waight	TIC	Work note	IID	<u> </u>	VO	ŴCO.

	Age	SMR pubic	Height	Weight	TLC	Work rate	HR	ЙE	VO _{2MAX}	VCO ₂	DED
	(y)	hair	(cm)	(kg)	(1)	(W)	(bpm)	$(l \cdot min^{-1})$	$(ml \cdot kg^{-1} \cdot min^{-1})$	$(l \cdot min^{-1})$	KEK
PRE	11.4	1	145.6	35.9	3.11	120	199	42.5	40.3	1.59	1.10
POST	12.1	3	149.3	37.5	3.13	140	198	62.3	44.5	2.03	1.22



Figure 50 - C05 MEFV and FVL for PRE (top) and POST (bottom).

Table 69 – C05 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}\!.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W
EFL (% V _T)	PRE	0	0	0	0	0	0	-
	POST	0	0	0	0	0	0	20
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	10	24	34	42	52	58	-
	POST	10	19	23	34	41	52	77



Figure 51 – C05 ventilatory mechanics.

Table 70 - C08 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	13.6	4	164.6	49.5	4.22	160	199	95.3	41.0	2.56	1.26
POST	14.2	4	167.6	56.6	4.98	180	197	98.8	36.2	2.57	1.27



Figure 52 – C08 MEFV and FVL for PRE (top) and POST (bottom).

Table 71 – C08 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}\!.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	3	-
	POST	0	0	0	0	0	0	0	36	28
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	8	23	28	38	48	54	71	82	-
	POST	8	19	21	25	38	46	60	83	83



Figure 53 – C08 ventilatory mechanics.

|--|

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	VO _{2MAX} (ml⋅kg ⁻¹ ⋅min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	13.5	4	158.9	48.9	4.48	160	201	70.6	38.2	2.18	1.17
POST	14.1	4	161.1	51.9	4.68	160	203	97.6	38.7	2.60	1.30



Figure 54 - C09 MEFV and FVL for PRE (top) and POST (bottom).

Table 73 – C09 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0
	POST	0	0	0	0	0	0	0	0
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	4	15	13	15	25	30	43	49
	POST	6	10	17	18	18	26	55	69



Figure 55 – C09 ventilatory mechanics.

Table 74 – 0	C10 se	elected a	anthrop	pometric, j	oulmonary	function,	and	maximal	exercise	data.
						,				

	Age	SMR pubic	Height (cm)	Weight	TLC	Work rate	HR (hnm)	\dot{V}_E	$\dot{V}O_{2MAX}$	$\dot{V}CO_2$	RER
	(y)	liali	(CIII)	(kg)	(1)	(\mathbf{w})	(opin)	(1.11111)	(IIII·Kg ·IIIII)	(1.11111)	
PRE	14.3	3	164.9	47.4	4.32	180	194	82.5	45.8	2.59	1.19
POST	14.8	3	165.4	48.4	4.59	200	202	89.7	48.1	2.72	1.17



Figure 56 - C10 MEFV and FVL for PRE (top) and POST (bottom).

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W	PRE MAX	200 W	POST MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	17	39	51	62	-	-
	POST	0	0	0	0	0	0	15	27	44	-	66	66
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	11	27	31	37	49	47	66	79	103	108	-	-
	POST	8	23	32	36	41	44	59	75	83	-	116	115

Table 75 – C10 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.



Figure 57 – C10 ventilatory mechanics.

Table 76 - C11 selected anthropometric, pulmonary function, and maximal exercise data.

	Age (y)	SMR pubic hair	Height (cm)	Weight (kg)	TLC (l)	Work rate (W)	HR (bpm)	V _E (l∙min ⁻¹)	^V O _{2MAX} (ml·kg ⁻¹ ·min ⁻¹)	VCO ₂ (l·min ⁻¹)	RER
PRE	14.3	3	160.8	45.5	3.78	160	199	65.9	37.3	1.98	1.17
POST	14.8	3	161.9	47.7	4.04	180	202	83.2	45.1	2.48	1.15



Figure 58 – C11 MEFV and FVL for PRE (top) and POST (bottom).

Table 77 – C11 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}\!.$

		REST	40 W	60 W	80 W	100 W	120 W	140 W	160 W	180 W
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0	-
	POST	0	0	0	0	0	0	1	24	42
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	6	21	25	36	33	46	48	55	-
	POST	10	27	29	37	54	49	56	74	88



Figure 59 – C11 ventilatory mechanics.

Table 78 –	C12 s	selected	anthro	nometric.	pulmonary	z functio	on, and	maximal	exercise	data.
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	Age	SMR pubic	Height (cm)	Weight	TLC	Work rate	HR (bpm)	\dot{V}_E	VO _{2MAX}	VCO ₂	RER
PRE	14.8	5	156.9	54.9	4.01	140	200	86.0	31.2	2.18	1.27
POST	15.5	5	157.7	54.7	4.07	140	203	94.9	36.1	2.31	1.17



Figure 60 - C12 MEFV and FVL for PRE (top) and POST (bottom).

Table 79 – C12 EFL severity and $\dot{V}_{E}\!/\dot{V}_{ECAP}$.

		REST	40 W	60 W	80 W	100 W	120 W	140 W	MAX
EFL (% V _T)	PRE	0	0	0	0	0	0	0	0
	POST	0	0	0	0	0	0	0	26
$\dot{V}_{E}\!/\dot{V}_{ECAP}\left(\%\right)$	PRE	4	16	18	30	34	46	59	49
	POST	7	21	28	33	36	49	68	77



Figure 61 – C12 ventilatory mechanics.

Appendix C: Questionnaires, forms, and documents





Children's Heart Centre B.C. Children's Hospital 4480 Oak Street, 1F Clinic

Vancouver, B.C. V6H 3V4

Does competitive swimming during puberty affect lung development?

Subject identifier:		Date (DD/MM/YYYY):			
DEMOGRAPHICS:	Age (MM/YYYY):	Race (optional):			

HISTORY:

- 1. Are you currently taking any medications?
 - Please list: ______
- 2. Do you currently smoke? YES/NO
- 3. Are you a past smoker? YES/NO
- 4. Have you even been diagnosed with asthma, reactive airway disease, other lung
 - problems, anemia, or significant illness?
 - Please list: ______
- 5. Do you currently use an inhaler? YES/NO
- 6. Have you used an inhaler in the past? YES/NO
- 7. Have you ever travelled to a location above 2000 m for a period of time greater than six weeks? YES/NO

OTHER: Family doctor: _____

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Children's Heart Centre B.C. Children's Hospital 4480 Oak Street, 1F Clinic Vancouver, B.C. V6H 3V4

Does competitive swimming during puberty affect lung development?

t identi	fier: Date (DD/MM/YYYY):
GRAPH	IICS: Age (MM/YYYY):
RY:	
Are yo	u currently taking any medications?
0	Please list:
Do you	u currently smoke? YES/NO
Since	you were seen last, have you:
i.	Smoked? YES/NO
ii.	Been diagnosed with asthma, reactive airway disease, other lung problems,
	anemia, or significant illness?
	o Please list:
iii.	Used an inhaler? YES/NO
iv.	Travelled to a location above 2000 m for a period of time greater than six weeks?
	t identi GRAPH Are yo O Do you Since y i. ii. ii.

YES/NO

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Checked by:

Physical Activity Questionnaire

ID: _____ Today's date (dd/mm/yyyy): _____

Date of last visit: ____

We would like to know about the physical activity you have since your last visit. This includes sports or activities that make you sweat or make your legs feel tired, or games that make you huff and puff, like tag, skipping, running, and climbing.

- There are no right or wrong answers this is not a test.
- Please answer all questions as honestly and accurately as you can this is very important.

1. Physical activity in your **SPARE TIME (this <u>does not</u> include P.E classes)** – have you done any of the following activities **since your last visit**? If yes, how many times and for how long on average?

No	1-2	3-4	5-6	7 or more times	time per session
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	Q	Q	Q	Q	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	Q	Q	Q	Q	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
0	0	0	0	0	
		N_0 1-2 O <td>N_0 1-2 3-4 O O</td> <td>No 1-2 3-4 5-6 0 0 0 0 0 <td< td=""><td>No $1-2$ $3-4$ $5-6$ 7 or more times 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0</td></td<></td>	N_0 1-2 3-4 O	No 1-2 3-4 5-6 0 0 0 0 0 <td< td=""><td>No $1-2$ $3-4$ $5-6$ 7 or more times 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0</td></td<>	No $1-2$ $3-4$ $5-6$ 7 or more times 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0

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Competitive swimming and lung development study 2015 PAQ questionnaire

2. Since your last visit, during your PHYSICAL EDUCATION (PE) CLASSES at school, how often were you very active (playing hard, running, jumping and throwing)? Check only one.

- O I don't do PE
- O Hardly ever
- O Sometimes
- O Quite often
- O Always

3. Since your last visit, what did you most of the time at school during RECESS? Check only one.

- O Sat down (talking, reading, doing school work)
- O Stood around or walked around.
- O Ran or played a little bit.
- O Ran around and played quite a bit.
- O Ran and played hard most of the time.

4. Since your last visit, what did you normally do at school during LUNCH (besides eating lunch)? Check only one.

- O Sat down (talking, reading, doing school work)
- O Stood around or walked around.
- O Ran or played a little bit.
- O Ran around and played quite a bit.
- O Ran and played hard most of the time.

5. Since your last visit, during a typical week (Monday-Friday) on how many days **RIGHT AFTER SCHOOL** did you do sports, dance, or play games in which you were very active? Check only one.

None.
 1 time.
 2 or 3 times.
 4 times.
 5 times.

6. Since your last visit, during a typical week (Monday-Friday) on how many **EVENINGS** did you do sports, dance, or play games in which you were very active? Check only one.

O None. Q. 1 time. Q. 2 - 3 times. Q. 4 - 5 times. Q. 6 - 7 times. Version 1.1, 2016-May-03

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7. Since your last visit, during a typical WEEKEND (Saturday-Sunday) how many times did you do sports, dance, or play games in which you were very active? Check only one.

O None. Q.__1 time. Q.__2 - 3 times. Q.__4 - 5 times. Q.__6 or more times.

8. Which one of the following five statements describes you best for a TYPICAL WEEK (Monday-Sunday) since your last visit? Read all 5 before deciding on the one answer that describes you. Please note "free time" refers to all time outside of school hours.

O All or most of my free time was spent doing things that involved **little physical effort** (e.g. watching TV, homework, playing computer games, Nintendo).

Q. I sometimes (1-2 times last week) did physical things in my free time (e.g. played sports went running, swimming, bike riding, did aerobics).

O I often (3-4 times last week) did physical things in my free time.

O I quite often (5-6 times last week) did physical things in my free time.

O I very often (7 or more times last week) did physical things in my free time.

9. Mark how often you did physical activity (like playing sports, games, doing dance, or any other physical activity) for each day during a **TYPICAL WEEK** (Monday-Sunday) since your last visit.

	None	Little bit	Medium	Often	Very often	
Monday	0	Q	0	Q	0	
Tuesday	0	Q	Q	Q	Q	
Wednesday	0	Q	Ö	0	Q	
Thursday	0	Q	Q	Q	Q	
Friday	0	Q	Ö	Q	Q	
Saturday	0	Q	Q	Q	Q	
Sunday	0	Q	Ö	Q	Ö	

10. Were you sick since your last visit, or did anything prevent you from doing your normal physical activities for a prolonged period of time?

O Yes O No

If yes, what prevented you? _____

For how long did this prevent you from doing activities?

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Competitive swimming and lung development study 2015 PAQ questionnaire

Next, we are going to ask you about the intensity of the various activities that you perform during a typical week.

11a. Since your last visit, during a typical **WEEK** (Monday-Sunday) on how many days did you do **VIGOROUS** physical activities that made you sweat and be "out of breath" (such as running or fast bicycle riding).

Think about only those activities that you did for at least 10 minutes at a time.

____ days per week

11b. How much time in total did you usually spend on one of those days doing VIGOROUS physical activities?

_____ hours _____ minutes

12a. Since your last visit, during a typical WEEK (Monday-Sunday) on how many days did you do MODERATE physical activities that made sweat a little and to breathe harder (such as skating or easy bicycle riding).

Think about only those activities that you did for at least 10 minutes at a time.

____ days per week

12b. How much time in total did you usually spend on one of those days doing MODERATE physical activities?

__ hours _____ minutes

Lastly, we want to know about the time you spent <u>sitting</u> during a typical week while at school, at home, and during leisure time. This includes time spent sitting at a desk, visiting friends, reading, traveling on a bus, or sitting or lying down to watch television.

13. Since your last visit, during a typical WEEK (Monday-Sunday) how much time in total did you usually spend sitting on a day?

____ hours _____ minutes

THANK YOU!

Version 1.1, 2016-May-03

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Tanner Pubertal Staging - Girls

Sexual Maturity Rating (SMR) Stage	Pubic Hair	Breasts
1	Preadolescent	Preadolescent
2	Sparse, lightly pigmented, straight, medial border of labia	Breast and papilla elevated as small mound; areolar diameter increased
3	Darker, beginning to curl, increased amount	Breast and areola enlarged, no contour separation
4	Coarse, curly, abundant but amount less than in adult	Areola and papilla form secondary mound
5	Adult feminine triangle, spread to medial surface of thighs	Mature; nipple projects, areola part of general breast contour









Ref.: Tanner JM: Growth at Adolescence, 2nd ed. Oxford, England, Blackwell Scientific Publications, 1962.
Com	petitive swimming	g and lung	develo	pment study	/ - Swimming	g load d	uestionnaire	for coaches

Type here		
Please enter your unique coaching ID	:	
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Curies testistics local		
Swim training load It is important to quantify the amount of s	swimming each participant has performed. Since the start of the season in S	eptember:
, , ,		
1. How many swim training sessions F	PER WEEK did the participant attend?	
0 sessions		14 session
2. What was the average swim distan	ce (in kilometres) PER SWIM TRAINING SESSION?	
0 km	3	6 k
3. What was the average training time	(in hours) PER SWIM TRAINING SESSION?	
0 hours	(2)	4 hour
1 What was the average swim distan	ca (in kilometres) DED WEEK2	
h km		40 k
	20	40 10
What was the average training time	(in hours) PER WEEK?	
) hours	(15)	30 hou
Non-swim-training load t is also important to quantify the amour sessions. Since the start of the season in 6. How many non-swim training session	nt of training each participant has performed beyond the pool, such as during n September: ons PER WEEK did the participant attend?	g dryland or cross-trainin
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10. What was the average time spent (in hours) performing "breath-control drills" PER WEEK?

0 hours

5 Submit 10 hours

Appendix D: Predictive equations

Measurement	Study	Equation	Limits of abnormality (%)	Notes
TLC	Hibbert et al., 1989 (85)	$e^{0.0075 \times Age \times Ht^3 + 1.1808 \ln(Ht) + 0.4927}$	<83,>121	Age is in y; Ht is in m
FRC		$e^{0.0130 \times Age \times Ht^3 + 0.0606}$		
RV		$e^{0.0153 \times Age \times Ht^3 - 0.4694 \ln(Ht) - 0.5604}$	<63, >159	
FVC		$e^{1.4393 \times Ht + 0.0221 \times Age - 1.4500}$	<83, >121	
FEV_1		$e^{1.4267 \times Ht + 0.0247 \times Age + 1.5717}$	<82	
PEF		$e^{-0.0079 \times Age \times Ht^2 + 1.7246 \ln(Ht) + 0.0477 \times Age + 4.8292}$	<79	
FEF _{25-75%}		$e^{1.1257 \times Ht + 0.0259 \times Age - 0.8787}$	<67	
FEF _{25%}		$e^{1.4917\ln(Ht)+0.0377\times Age+0.4041}$	<73	
FEF _{50%}		$e^{1.7560\ln(Ht)+0.03305 \times Age-0.0269}$	<64	
FEF _{75%}		$e^{0.0143 \times Age \times Ht^2 + 1.4952 \ln(Ht) - 0.6127}$	<69	
RV/TLC			<0.17, >0.38	
$D_{L,CO}$ and $D_{L,CO}c$	Kim et al., 2012 (87)	e ^{0.796+0.012×Ht+0.018×Age}		Age is in months; Ht is in cm
VA		$e^{-1.424+0.016 \times Ht+0.019 \times Age}$		
$D_{L,CO}c/V_A$		$8.458 - 0.021 \times Ht$		
$P_{st}(L)_{50}$	Zapletal et al., 1976 (90)	$Pst(L)_{50} = 0.0770 \times Height - 3.3871$		
PI _{MAX}	Domenech-Clar <i>et al.</i> , 2003 (45)	$-33.854 - (1.814 \times Age) - (0.004 \times Ht \times Wt)$		Age is in y; Height is in cm
PE _{MAX}		$17.066 + (7.22 \times Age)$		
[.] VO _{2MAX}	Cooper et al., 1984 (88)	$28.5 \times Wt + 288.2$		In mL/min

Table 80 – Predictive equations and the limits of abnormality

TLC, total lung capacity; Ht, height; FRC, functional residual capacity; RV, residual volume; FVC, forced vital capacity; FEV₁, forced expired volume in one second; PEF, peak expiratory flow; FEF, forced expiratory flow; $D_{L,CO}$, diffusion capacity of the lungs for carbon monoxide; V_A , alveolar volume; PI_{MAX} , maximal inspiratory pressure; Wt, weight; PE_{MAX} , maximal expiratory pressure; VO_{2MAX} , maximal oxygen consumption.