

**MINI REVIEW**

# Determinants of lung function changes in athletic swimmers.

## A review

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**Funding information**

I.R was supported by a professional development fund from the Lausanne University Hospital and the Department of pediatrics, Lausanne, Switzerland.

**Abstract**

**Aim:** To summarise lung function characteristics of athletic swimmers and discuss mechanisms explaining these changes while putting forward the lack of a clear understanding of the precise physiological factors implicated.

**Methods:** Literature search until 07.2021 on Medline and EMBASE using keywords swimming, athletes, respiratory physiology, lung development, lung function tests. Relevant articles in French and English were reviewed.

**Results:** We found insufficient data to perform a meta-analysis. However, there is evidence that swimmers have better expiratory flows and increased baseline lung volumes than non-athletes or non-swimmers. Although these features can result from changes in lung development following intense training over the years, the contribution of a genetic predisposition and positive selection cannot be totally excluded.

**Conclusion:** Disentangling the participation of constitutional factors and years of hard training to explain the larger lung volumes of athletic swimmers is in favour of an adaptative response of the lungs to early swim training through modification of the pathway of lung development. There seems to be an optimal window of opportunity before the end of growth for these adaptational changes to occur. Precise mechanisms, and contribution of adaptative change on lung physiology, remain to be further studied.

**KEYWORDS**

athletes, lung function tests, lung growth and development, swimming

**1 | INTRODUCTION**

There has been a long-standing interest in the lung physiology of swimmers with the earliest studies dating as early as the 1960s.<sup>1</sup> Intense swimming workouts have been shown to increase cardio-respiratory capacity in athletes. In particular, it has a positive impact

on lung, blood and heart volumes, as well as maximal oxygen uptake and aerobic capacity.<sup>1-4</sup> However, aquatic sports, competitive swimming in particular, may impact respiratory health in causing an increase in airway responsiveness and in prevalence of exercise-induced asthma over the years.<sup>5,6</sup> These changes have been associated with airway remodelling as shown in bronchial biopsies studies,

**Abbreviations:** DLCO, lung diffusion capacity for CO; EGF, epithelial growth factor; FEV<sub>1</sub>, forced expiratory volume in one second; FImax, inspiratory force; FRC, functional residual capacity; FVC, forced vital capacity; HGF, hepatocyte growth factor; HIF, hypoxia-inducible factors; MEM<sub>50</sub>, mid-expiratory flows; MVV, maximal voluntary ventilation; pCO<sub>2</sub>, partial pressure of CO<sub>2</sub>; P<sub>E</sub>max, maximal expiratory pressure; PFTs, pulmonary function tests; P<sub>I</sub>max, maximal inspiratory pressure; pO<sub>2</sub>, partial pressure of O<sub>2</sub>; Raw, airway resistance; TLC, total lung capacity; VC, vital capacity; VEGF, vascular endothelial growth factor; VO<sub>2</sub> max, maximal oxygen uptake.

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possibly associated with airways epithelial damage.<sup>7</sup> This phenomenon may be enhanced by the regular exposure to chlorine and its derivatives.<sup>5,6</sup> Nevertheless, there is still a debate about the causes of such differences between swimmers and other athletes mainly because airway structural changes observed in swimmers should theoretically lead to reduced expiratory flows, whereas swimmers usually have higher values of lung function parameters such as expiratory flows and lung volumes, compared to athletes performing in other disciplines.<sup>8-11</sup>

Indeed, it remains uncertain whether the ability to perform in swimming and the associated lung function characteristics of swimmers come from a genetic predisposition, are the result of intense training or a combination of both. In most athletes, many years of training take place during a growth period, when the lungs are also developing rapidly. Training during that particular time may be of great importance in determining future lung structure and function, including performance of the oxygen transport system. New insights on pulmonary development show that continued alveolarisation and microvascular maturation of the capillary bed take place until early adulthood.<sup>12,13</sup> As such, specific consequences of exercising in a water environment and possible adaptative mechanisms to intense swim training might have an influence on the development of airways and alveolar spaces, leading to enhanced lung growth.

The present review discusses how swimming-induced changes on lung growth and development during childhood and adolescence leads to enhanced lung function, as the majority of swimmer athletes begin their intense training during this period of life.

## 2 | METHODS

Literature search was conducted until 07.2021 on Medline and EMBASE using the keywords swimming, athletes, respiratory physiology, lung development, lung function tests. Relevant articles in French and English were reviewed. Most retrieved articles were small sample sizes studies, and we found insufficient data to perform a meta-analysis on this topic.

### 2.1 | Lung function in athletic swimmers

It has been repeatedly reported that male and female competitive swimmers have greater lung volumes,<sup>1-4,9,14-18</sup> expiratory flows<sup>2,3,9,19</sup> and diffusing capacities<sup>2,17,20</sup> compared with other categories of athletes or untrained individuals. Furthermore, it has been shown that the level of training correlates with lung function indices, being even more pronounced among elite swimmers than sub-elite athletes. For example, when looking at 150 swimmer athletes, Doherty et al.<sup>19</sup> reported that male national swimmers had a higher forced expiratory volume in one second (FEV<sub>1</sub>), a marker of airway calibre, compared to non-nationals, showing a relationship with the number of years of training and age at which it began. Yet, while some consider increased lung volumes in elite swimmers as the result of

### Key Notes

1. Swimmer athletes have greater airway hyperresponsiveness and lung function values than other athletes or sedentaries; longitudinal studies explaining these precise mechanisms are few.
2. Anthropometry and greater muscle strength do not account for increased lung function values.
3. Years of hard training beginning at an early age and genetic predisposition may contribute; more likely, there is an adaptative response of lung development and growth to early intense swim training.

an adaptative response to intense swim training, others consider that it is rather the manifestation of an innate advantage. It is even speculated that large lungs strongly contribute to becoming an elite swimmer.

### 2.2 | The role of anthropometry on lung function of athletic swimmers

Lung volumes depend on multiple anthropometric parameters, including height. Swimmers tend to be tall and lean: taller boy and girl swimmers after the age of 12 years have been compared to non-athletic peers,<sup>2</sup> as well as taller adult swimmers compared to footballers and sedentary counterparts,<sup>9</sup> or land-based athletes.<sup>19</sup> Overall, however, the average height of swimmers does not significantly differ from age-matched population values<sup>21</sup> or non-athletic children.<sup>3,15,19,22</sup> Favourable anthropometry does not seem therefore to be the explanation for increased lung volumes in swimmers.

### 2.3 | The role of genetic predisposition on lung function of athletic swimmers

Arguments for genetically determined superior lung functions are few. In a longitudinal study looking at swimmers 8–18 years engaged in strenuous year-round training programmes, vital capacity (VC) values in most of the swimmers were above those of non-athletic children. The fact that this difference was present even in the youngest who had just initiated training pointed to an hereditary characteristic, even if the authors could not exclude the effect of a high level of physical activity in those children prior to beginning swimming.<sup>2</sup> Likewise, in a group of 11 to 14-year-old girls, pulmonary function values were higher at the onset of a training season in competitive swimmers compared to physically active but not competitive controls.<sup>4</sup> Finally, among a group of adult sportsmen, swimmers had larger lung volumes than footballers or sedentary controls, which were not influenced by anthropometric nor training factors.<sup>9</sup> Therefore, the contribution of a genetic predisposition and positive

selection cannot definitely be excluded and should still be considered as plausible to explain the greater lung function indices in some swimmers.

## 2.4 | The influence of training on lung function of athletic swimmers

Recent reviews in nonelite or noncompetitive swimmers or in children and adolescents with asthma have shown no effect of recreational swimming on lung function measures except on peak expiratory flow,<sup>23</sup> or only a small increase in lung function parameters.<sup>24</sup> However, in athletic swimmers, arguments for an effect of training on lung physiology are many, even if there are few longitudinal data available describing the effect of a swimming career or other high endurance careers on lung physiology from its initiation through its cessation. What is known about the impact of intensive swim training on pulmonary function tests (PFTs) comes mostly from studies in children and young adolescents and displays conflicting results. For instance, Vaccaro et al.<sup>22</sup> showed that boys and girls 9 to 11 years of age undertaking their first year of competitive training (four times weekly) had comparable increases in forced vital capacity (FVC), FEV<sub>1</sub> and maximal voluntary ventilation (MVV) after 7 months compared to controls, whereas their maximal oxygen uptake (VO<sub>2</sub> max) increased significantly more. In that study, training did not lead to an increase in selected lung function parameters, but it could be argued that the training period was too short to have allowed a significant effect of swim training on PFTs. Indeed, when looking at longer training periods (up to 5 years) in young female swimmers aged 9–13 years, mean lung volumes such as total lung capacity (TLC) and functional residual capacity (FRC) were greater than predicted values in the girls who had been training for a year or more, with a greater increase over time in VC in relation to height compared to TLC and FRC.<sup>14</sup> Thus, regular swim training could promote functional over anatomical lung growth, most likely attributable to the changes in breathing pattern and increase in muscular strength induced by swimming. This may also explain why adolescent male swimmers 13–16 years old with at least 6 years of training had TLC, FVC and FEV<sub>1</sub> values 10–16% above predicted values after 6 years of competitive swimming, though comparative values prior to training were not available.<sup>15</sup> Similarly, young swimmers followed longitudinally for 3 years demonstrated higher VC and TLC values in relation to height compared to non-athletic peers, as well as increased MVV and FEV<sub>1</sub>, highlighting the importance of physical training during growth.<sup>2</sup> Finally, a small group of 5 prepubertal girls belonging to a youth swimming team were compared to controls before and after one year of intensive endurance training. Lung volumes, FEV<sub>1</sub> and airway resistance (Raw) did not initially differ. A year later, they were significantly greater in the group of ongoing swimmers. In particular, FEV<sub>1</sub> and mid-expiratory flows (MEM<sub>50</sub>) values had increased by 18% and 15%, respectively, and higher MEF<sub>50</sub>/TLC was seen along with a shift to the right of the flow-volume curves.<sup>3</sup> These arguments provide favourable evidences that swim training

may cause enlargement not only of static but also of dynamic lung volumes.

## 2.5 | Pathophysiology of lung function changes in athletic swimmers

The mechanisms explaining the increased lung dimensions as a result of prolonged training in swimmers are not completely elucidated. Several hypotheses have been proposed and are outlined in the present section.

In regard to increased static lung volumes, one hypothesis suggests that increased ventilatory muscle strength helps the lungs to inflate and deflate more efficiently, as some lung volumes depend on muscle power. Swimming induces conditioning of the accessory muscles of the neck and chest wall, resulting in stronger respiratory musculature which may help expand the chest wall to achieve large lung volumes. In healthy adults<sup>25</sup> and quadriplegic patients,<sup>26</sup> specific inspiratory strength training at TLC was shown to increase the strength and endurance of ventilatory muscles, including lung volumes and maximal static pressures. In varsity female swimmers undergoing a 10-week conditioning programme, a positive correlation was described between changes in FRC and maximal inspiratory pressure (P<sub>I</sub>max). However, those changes were not different in swimmers having additional muscle conditioning compared to those following their regular training programmes.<sup>27</sup> Although this hypothesis seemed interesting, measurements of maximal inspiratory and expiratory pressures (P<sub>I</sub>max and P<sub>E</sub>max) in swimmers go against an increased muscle strength hypothesis. Indeed, comparison of P<sub>I</sub>max and P<sub>E</sub>max in swimmers, runners and controls did not identify any difference in favour of swimmers, but rather a higher P<sub>E</sub>max in runners.<sup>28</sup> Likewise, in a longitudinal study looking at 7–12 years old girls involved in intensive swim training, lung volumes were larger in swimmers but maximal static pressures were surprisingly less in the athletes in comparison to controls.<sup>29</sup> A one-year follow-up later confirmed that swimmers still had normal maximal pressures.<sup>16</sup> Lastly, a study comparing competitive swimmers to long distance runners and non-athletic controls found no difference in P<sub>I</sub>max and P<sub>E</sub>max among groups despite the observation that all lung volumes were greater in swimmers.<sup>11</sup> Again, these data go against the assumption that increased lung volumes in swimmers are due to an increased ability to inflate and deflate the lungs in relation to changes in inspiratory/expiratory pressures.

There is, however, a risk that pressures at the mouth may underestimate the strength of the respiratory muscles because the pressures in swimmers are applied over a larger lung surface area. Hence, forces obtained by the product of Pmax and surface area were also compared. In 9–10 years old, only inspiratory force (FImax) was significantly higher compared to controls,<sup>29</sup> whereas in older swimmers and runners, forces were not different between the groups.<sup>11</sup> Even if there might be a possible training effect on inspiratory muscle force, the lack of increased inspiratory flows in swimmers weakens the significance of this finding.<sup>28</sup> With the available evidence, the

hypothesis that supranormal lung volumes in swimmers are directly related to increased respiratory muscle strength and pressures cannot be confirmed.

Other hypotheses propose that swim training induces an adaptational expansion of the lungs and chest surface area different than land-based training programmes due to the specific constraints imposed by swim training upon the pulmonary system. Cordain summarised these unique aquatic stressors in five points: (a) negative pressure breathing and prolonged expiration into the water, (b) restricted breathing pattern and altered alveolar gas tensions, (c) repeated expansion of the lungs to TLC, (d) prone body position, (e) early childhood intensive training programmes.<sup>17</sup> As such, some peculiarities of intense swimming may be viewed as a conditioning stimulus for lung growth.

The fact that the body of swimmers is immersed below the neck during exercise while their lungs remain at atmospheric pressure was hypothesised to be a contributor to lung physiology changes. Swimmers are submitted to a low but constant transthoracic pressure of about 10 cm H<sub>2</sub>O leading to increased airway resistance. This constitutes an additional load, albeit modest, upon the inspiratory muscles. Heightened inspiratory muscle training may also result from repeated rapid forceful inspiration to TLC during swimming, explaining the positive correlation found between VC and P<sub>I</sub>max.<sup>17</sup> However, as discussed previously, studies up to now have not been able to put forward constant superior respiratory muscle strength in swimmers. It may just be that swimmers are more resistant to inspiratory muscle fatigue, as reported in cyclists and rowers. Indeed, prevention or delayed exercise-induced diaphragmatic fatigue is one of the potential mechanisms explaining the ergogenic effect of respiratory muscle training.<sup>30</sup>

Next, important hypotheses rely on the fact that the breathing pattern of a competitive swimmer is more dictated by biomechanical constraints than physiological needs. Inspiration becomes a rapid forced manoeuvre, and expiration, also occurring in the water, is limited and controlled. Consequently, minute ventilation is reduced, and swimmers' lungs must accommodate large pCO<sub>2</sub> and pO<sub>2</sub> gradients. This translates into increased pulmonary diffusion capacity disproportional to changes in growth compared to controls.<sup>2,15,17,20</sup> Increase in DLCO is also facilitated by the horizontal position unique to swimming as lung perfusion becomes more uniform when the body is prone rather than standing.

Moreover, stroke-induced obligatory controlled frequency breathing may also cause a reduction in oxygen saturation and intermittent hypoxia.<sup>17</sup> Hypoxia is recognised as a strong growth factor for the lungs through the upregulation of hypoxia-inducible factors (HIF) acting as important transcriptional activators for the expression of other molecules such as vascular endothelial growth factor (VEGF).<sup>31</sup> For example, in high-altitude residents chronically submitted to a low PaO<sub>2</sub>, larger lungs and increased DLCO are reported, consistent with high-altitude-enhanced lung growth. Likewise, breath hold divers who repeatedly undergo oxygen deprivation during apnoea have higher lung functions compared to non-breathold divers.<sup>32</sup> This environment specific adaptation has also been well documented in animals, including models mimicking preterm

birth.<sup>33,34</sup> In swimmers, even if the exact pathway is not yet defined, stroke-induced recurrent hypoxia could possibly induce a positive adaptation of pulmonary growth responsible for the greater lung capacities observed. Finally, we may wonder whether the particular breathing pattern induced by swimming resulting in repetitive expansion of the lungs at TLC, followed by breath-holding to increase the stroke duration without the need to take another breath, could lead over time to increased lung volumes and flows as an adaptive hyperinflation-induced structural change.

Lastly, and perhaps most importantly, the swim career of a competitive swimmer begins early in life, during a period of intense growth when the responses to growth stimuli are greater. Postnatal development of the human lungs can be divided in 2 or 3 phases. The first one is a period of classical alveolarisation occurring from term birth to about 18–24 months of age. During that stage, the number of alveoli multiplies greatly but they remain fairly immature. In parallel, there is a stage of microvascular maturation, characterised by restructuration of the double capillary network in a more functional single layer capillary system. Then, the stage of late alveolarisation, or continued alveolar formation, happens until young adult age during which additional alveoli are added by peripheral septation.<sup>12,35</sup> These different stages tend to overlap, and lung development progressively blends into normal growth that mostly follows growth in body dimensions, influenced also by the development of the thorax.

The lungs of young swimmers are exposed at an early age to intense exercise with increased mechanical strain, known in some circumstances to directly accelerate lung growth. Lung growth gene expression is activated by mechanical strain during artificial ventilation, or distortion and stretch after pneumonectomy, as evidence by subsequent increased lung mass, collagen, elastin and number of alveolar cell walls.<sup>31,33</sup> Similar mechanical stress may occur following repetitive inspiration to TLC during swim training, ensuing enhanced expression of growth factors such as vascular, epithelial or hepatocyte growth factors (VEGF, EGF and HGF). In this regard, Armour et al.<sup>11</sup> previously suggested that swimmers may develop greater lung volumes than other athletes or non-athletes by developing physically wider chests, containing an increased number of alveoli, rather than an increase in alveolar distensibility or size. Wider chest and higher bi-acromial breadth for age has also been measured by others.<sup>36</sup>

These are only speculative mechanisms by which competitive swimming could be a conditioning stimulus for lung growth, and more research is required on swimmers. Overall, the high capabilities of the healthy lung to adapt, and the greater responses of younger subjects to growth stimuli, are strong indicators of a positive effect of early swim training to explain the greater respiratory capacities of swimmer athletes.

### 3 | CONCLUSION

The advantages of enhanced lung volumes for swimming are well established: improved buoyancy and streamlined body position in the

water, increased gas exchange surface area and reduced respiratory oxygen cost. Disentangling the participation of constitutional factors and years of hard training to explain the larger lung volumes of swimmer athletes seems in favour of an adaptative response of the lungs to early swim training, particularly as recreational swimming does not have the same impact on lung function parameters. Intense swimming leads to a modification of the pathway of lung development, and there even seems to be an optimal window of opportunity before the end of growth for these adaptational changes to occur at best. Nevertheless, genetic factors and inherited pulmonary traits should not be excluded definitively and may certainly be relevant in some swimmers.

To improve our understanding of the precise physiological factors participating in the making of an elite swimmer, in addition to intense prolonged physical training, longitudinal studies from the initiation of training in childhood to the end of a swimmer's career should be undertaken. The interrelation between the rate of growth of lung volumes and chest wall, changes in maximal static pressures and lung recruitment during swimming should be considered cautiously. Functional imaging and activation of molecular pathways underlying lung growth, including hormonal influences, could also bring us further insights to the development of functional dimensions of hard swim training.

Still, it should not be forgotten that aquatic sports, competitive swimming in chlorinated pools in particular, may also have a negative impact on respiratory health in promoting the development of airway hyperresponsiveness and exercise-induced asthma. Precise mechanisms, and perhaps contribution of adaptative change on lung physiology, remain to be further studied.

## ACKNOWLEDGEMENTS

The authors wish to thank M-E. Boulay for her careful lecture and appreciation of the manuscript, and S. Carette for her secretarial assistance. Open Access Funding provided by Universite de Lausanne.

## CONFLICT OF INTEREST

None to declare.

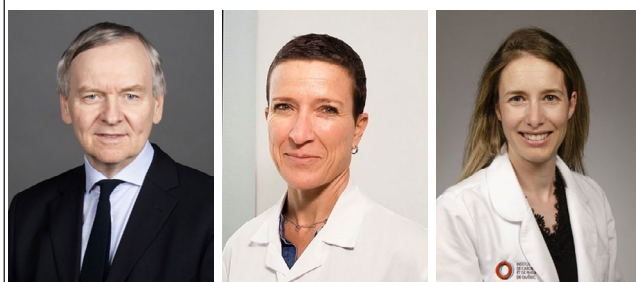
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## REFERENCES

1. Astrand PO, Eriksson BO, Nylander I, et al. Girl swimmers. with special reference to respiratory and circulatory adaptation and gynaecological and psychiatric aspects. *Acta Paediatr Suppl.* 1963;SUPPL147:141-175.
2. Andrew GM, Becklake MR, Guleria JS, Bates DV. Heart and lung functions in swimmers and nonathletes during growth. *J Appl Physiol.* 1972;32:245-251.
3. Courteix D, Obert P, Lecoq AM, Guenon P, Koch G. Effect of intensive swimming training on lung volumes, airway resistance and on the maximal expiratory flow-volume relationship in prepubertal girls. *Eur J Appl Physiol Occup Physiol.* 1997;76:264-269.
4. Bovard JM, Welch JF, Houghton KM, McKenzie DC, Potts JE, Sheel AW. Does competitive swimming affect lung growth? *Physiol Rep.* 2018;6:e13816.
5. Mountjoy M, Fitch K, Boulet LP, Bougault V, van Mechelen W, Verhagen E. Prevalence and characteristics of asthma in the aquatic disciplines. *J Allergy Clin Immunol.* 2015;136:588-594.
6. Bougault V, Turmel J, Levesque B, Boulet LP. The respiratory health of swimmers. *Sports Med.* 2009;39:295-312.
7. Bougault V, Loubaki L, Joubert P, et al. Airway remodeling and inflammation in competitive swimmers training in indoor chlorinated swimming pools. *J Allergy Clin Immunol.* 2012;129(351-358):358.e1.
8. Boulet LP, O'Byrne PM. Asthma and exercise-induced bronchoconstriction in athletes. *N Engl J Med.* 2015;372:641-648.
9. Lazovic-Popovic B, Zlatkovic-Svenda M, Durmic T, Djelic M, Djordjevic Saranovic S, Zucic V. Superior lung capacity in swimmers: some questions, more answers! *Rev Port Pneumol.* 2006;2016(22):151-156.
10. Sable M, Vaidya SM, Sable SS. Comparative study of lung functions in swimmers and runners. *Indian J Physiol Pharmacol.* 2012;56:100-104.
11. Armour J, Donnelly PM, Bye PT. The large lungs of elite swimmers: an increased alveolar number? *Eur Respir J.* 1993;6:237-247.
12. Schittny JC. Development of the lung. *Cell Tissue Res.* 2017;367:427-444.
13. Hsia CC. Signals and mechanisms of compensatory lung growth. *J Appl Physiol.* 1985;2004(97):1992-1998.
14. Engstrom I, Eriksson BO, Karlberg P, Saltin B, Thoren C. Preliminary report on the development of lung volumes in young girl swimmers. *Acta Paediatr Scand Suppl.* 1971;217:73-76. <https://doi.org/10.1111/j.1651-2227.1971.tb05698.x>. PMID: 5289804.
15. Vaccaro P, Clarke DH, Morris AF. Physiological characteristics of young well-trained swimmers. *Eur J Appl Physiol Occup Physiol.* 1980;44:61-66.
16. Zinman R, Gaultier C. Maximal static pressures and lung volumes in young female swimmers: one year follow-up. *Pediatr Pulmonol.* 1987;3:145-148.
17. Cordain L, Stager J. Pulmonary structure and function in swimmers. *Sports Med.* 1988;6:271-278.
18. Rosser-Stanford B, Backx K, Lord R, Williams EM. Static and dynamic lung volumes in swimmers and their ventilatory response to maximal exercise. *Lung.* 2019;197:15-19.
19. Doherty M, Dimitriou L. Comparison of lung volume in Greek swimmers, land based athletes, and sedentary controls using allometric scaling. *Br J Sports Med.* 1997;31:337-341.
20. Yost LJ, Zauner CW, Jaeger MJ. Pulmonary diffusing capacity and physical working capacity in swimmers and non-swimmers during growth. *Respiration.* 1981;42:8-14.
21. Sobolova V, Seliger V, Grussova D, Machovcova J, Zelenka V. The influence of age and sports training in swimming on physical fitness. *Acta Paediatr Scand Suppl.* 1971;217:63-67.
22. Vaccaro P, Clarke DH. Cardiorespiratory alterations in 9 to 11 years old children following a season of competitive swimming. *Med Sci Sports.* 1978;10:204-207.
23. Lahart IM, Metsios GS. Chronic physiological effects of swim training interventions in non-elite swimmers: a systematic review and meta-analysis. *Sports Med.* 2018;48:337-359.
24. Beggs S, Foong YC, Le HC, Noor D, Wood-Baker R, Walters JA. Swimming training for asthma in children and adolescents aged 18 years and under. *Cochrane Database Syst Rev.* 2013;CD009607.
25. Gross D, Ladd HW, Riley EJ, Macklem PT, Grassino A. The effect of training on strength and endurance of the diaphragm in quadriplegia. *Am J Med.* 1980;68:27-35.
26. Leith DE, Bradley M. Ventilatory muscle strength and endurance training. *J Appl Physiol.* 1976;41:508-516.
27. Clanton TL, Dixon GF, Drake J, Gadek JE. Effects of swim training on lung volumes and inspiratory muscle conditioning. *J Appl Physiol.* 1985;1987(62):39-46.

28. Cordain L, Tucker A, Moon D, Stager JM. Lung volumes and maximal respiratory pressures in collegiate swimmers and runners. *Res Q Exerc Sport*. 1990;61:70-74.
29. Zinman R, Gaultier C. Maximal static pressures and lung volumes in young female swimmers. *Respir Physiol*. 1986;64:229-239.
30. McConnell AK, Romer LM. Respiratory muscle training in healthy humans: resolving the controversy. *Int J Sports Med*. 2004;25:284-293.
31. Wagner PD. Why doesn't exercise grow the lungs when other factors do? *Exerc Sport Sci Rev*. 2005;33:3-8.
32. Diniz CM, Farias TL, Pereira MC, et al. Chronic adaptations of lung function in breath-hold diving fishermen. *Int J Occup Med Environ Health*. 2014;27:216-223.
33. ad hoc Statement Committee, ATS. Mechanisms and limits of induced postnatal lung growth. *Am J Respir Crit Care Med*. 2004;170:319-343.
34. Asikainen TM, Chang LY, Coalson JJ, et al. Improved lung growth and function through hypoxia-inducible factor in primate chronic lung disease of prematurity. *FASEB J*. 2006;20:1698-1700.
35. Burri PH. Structural aspects of postnatal lung development—alveolar formation and growth. *Biol Neonate*. 2006;89:313-322.
36. Malina RM. Physical growth and maturity characteristics of young athletes (15–25). In: Magill RA (ed). *Children in Sport* (2nd ed). Champaign, IL: Human Kinetics; 1982:309.



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**How to cite this article:** Rochat I, Côté A, Boulet L-P. Determinants of lung function changes in athletic swimmers. A review. *Acta Paediatr*. 2022;111:259–264. <https://doi.org/10.1111/apa.16095>