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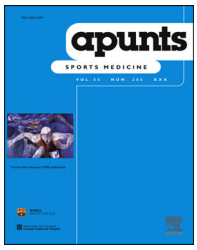

ORIGINAL ARTICLE

# Lung capacity and alveolar gas diffusion in aquatic athletes: Implications for performance and health

Iker García[a,b,*], Franchek Drobnic[c], Beatriz Arrillaga[d], Victoria Pons[a], Ginés Viscor[b]

[a] *Departament de Fisiologia i Nutrició, Centre d'Alt Rendiment (CAR), Av. Alcalde Barnils s/n, E-08173 Sant Cugat del Vallés, Barcelona, Spain*
[b] *Secció de Fisiologia, Departament de Biologia Cel·lular, Fisiologia i Immunologia, Facultat de Biologia, Universitat de Barcelona, Av. Diagonal, 643 E-08028 Barcelona, Spain*
[c] *Shenhua Greenland FC Medical Services, Shanghai, China*
[d] *Real Federación Española de Natación, Madrid, Spain*

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**Abstract**
*Background:* The diffusion capacity of carbon monoxide ($DL_{CO}$) provides a measure of gas transfer in the lungs. Endurance training does not increase lung volumes or diffusion in land-based athletes. However swimmers have larger lungs and better diffusion capacity than other matched athletes and controls.
*Purpose:* The aim of this study was to evaluate pulmonary alveoli-capillary diffusion and lung volumes in elite aquatic athletes, specifically swimmers, artistic swimmers and water polo players.
*Methods:* The participants were 64 international level aquatic athletes including 31 swimmers (11 female and 20 male), 12 artistic swimmers (only female), and 21 water polo players (10 female and 11 male). The single-breath method was used to measure $DL_{CO}$ and pulmonary parameters.
*Results:* The main finding of this study is that $DL_{CO}$ is high in aquatic athletes, clearly above their reference values, both in females ($33.4 \pm 9.4$ mL min$^{-1}$·mmHg$^{-1}$; 135%) and males ($48.0 \pm 5.83$ mL min$^{-1}$·mmHg$^{-1}$; 148%). There was no difference in $DL_{CO}$ between female swimmers, artistic swimmers and water polo players ($34.7 \pm 8.3$ to $33.4 \pm 4.0$ to $32.1 \pm 5.6$ mL min$^{-1}$·mmHg$^{-1}$), but male swimmers had a higher $DL_{CO}$ compared to water polo players ($50.4 \pm 5.3$ to $43.4 \pm 7.0$, $p = 0.014$).
*Conclusions:* Aquatic athletes have larger lungs and better diffusion capacity than the percentage predicted by age and height. Therefore, swimming-based sports could help to improve pulmonary function in many different segments of the population.

* Corresponding author.
*E-mail address:* ikergarciaalday@gmail.com (I. García).

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## Introduction

Endurance training produces a physiological adjustment response in the cardiovascular, musculoskeletal and haematological systems. However, the structural and functional properties of the lungs do not change significantly in response to training.[1,2]

Exercise in the terrestrial environment does not provide sufficient stimulus for the remodelling and growth of the lungs.[3] The biological adaptation of the human body to exercise has not found a way within the laws of physics to increase lung functionality,[3] and the evidence is clear that the lungs do not well adapt to increased fitness.[4] Consequently, the lungs could be a limiting factor for performance within the extraordinary physiology developed by highly trained athletes.

The diffusing capacity of the lungs for carbon monoxide ($DL_{CO}$) provides an integrated representation of the mechanisms involved in the transfer of $O_2$ from atmospheric air to the pulmonary capillaries,[5] and provides a universally accepted measure of gas diffusion in the lungs.[6] During high-intensity exercise, pulmonary diffusion can be limited, leading to an increase in the alveolo-capillary oxygen difference and exercise-induced arterial hypoxaemia,[7] justifying the study of $DL_{CO}$ in high-performance sport.

The scientific literature has not extensively described the changes in $DL_{CO}$ in elite water-based athletes. While land-based sports do not alter $DL_{CO}$,[8] water-based sports have been associated with increased lung capacity and diffusion, including swimming,[9] artistic swimming[10] and free diving.[11]

Swimming has been described as one of the healthiest forms of physical activity, providing complete development of the body.[12] Unlike other sports, and despite the lack of mechanical impact, swimming involves both locomotor and respiratory muscles in a demanding way. Moreover, certain conditions associated with swimming have an impact on the lungs and airways that could favour a physiological or pathophysiological adjustment in the pulmonary system (Fig. 1). These conditions may be present with greater or lesser intensity, depending on the particular exposure to swimming and the structural conditions of each individual.

It has been reported that a horizontal body position and water immersion stimulate an increase in lung capacity and diffusion. During exercise in the aquatic environment, swimmers are also exposed to the hydrostatic forces produced by the water, which necessitate stronger inspirations that improve the strength of the inspiratory muscles[13] and longer breathing cycles, mimicking intermittent hypoxic training in which hypercapnia and hypoxia occur.[14]

This divergence between land and water sports may also have implications for the treatment of respiratory diseases and has been studied in the treatment of lung diseases such as asthma[15] and chronic obstructive pulmonary disease (COPD),[16] with positive lung function outcomes demonstrated in patients who practiced aquatic exercise.[17] These results are important considering that $DL_{CO}$ is an important predictor of mortality in the general population.[18]

The clinical management of lung diseases can also be improved based on physiological assessments of elite athletes, who demonstrate physiological optimization at the highest level.[19] As a result, the aim of this study was to evaluate the alveolo-capillary diffusion and lung volume of different elite aquatic athletes: swimmers, artistic swimmers and water polo players.

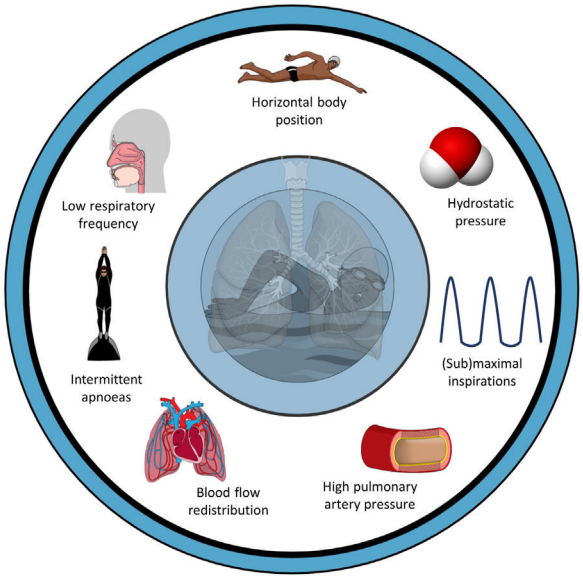


**Figure 1** Factors inherent to the practice of swimming related to the stress on respiratory function.

## Material and methods

### Participants

The participants were 64 elite junior-absolute athletes who train regularly at the Centre d'Alt Rendiment (CAR) in Sant Cugat: 31 swimmers (11 female and 20 male), 21 water polo players (11 female and 10 male) and 12 artistic swimmers (female) from the Spanish national team. Their weekly training volume consists of 8–10 pool sessions and 4–5 fitness sessions, accumulating 25–35 h of training per week.

### Experimental design

The participants performed two $DL_{CO}$ manoeuvres before the start of the study to familiarize themselves with the method. Then, the single breath method was performed under baseline conditions, in the morning and before training, to measure the diffusion and lung volume parameters. The measurements were made in an examination room, 20 m from the pool, in the same facility.

### Lung function measurement

The laboratory test used to measure $DL_{CO}$ was the *single-breath method*. The procedure requires a computerized spirometer (Ganshorn, PowerCube Diffusion+, Niederlauer, Germany) attached to a cylinder with a gas mixture of known concentration. The gas mixture used in our case was: 0.3% CO, 11% He, as reference inert gas, and 20.9% $O_2$ supplemented with $N_2$. The method measures the absorption of CO by the lungs during a short period of time in apnoea. We followed the recommendations given in a recent joint

**Table 1** Physiological and anthropometric parameters of the aquatic athletes studied.

| | Swimming | | Artistic swimmers | Water polo | |
|---|---|---|---|---|---|
| | Female | Male | Female | Female | Male |
| Age (y) | 18.3 ± 3.0 | 18.1 ± 1.9 | 21.5 ± 3.6 | 17.4 ± 0.7 | 17.0 ± 1.7 |
| Height (cm) | 169.5 ± 6.0 | 182.0 ± 6.3 | 170.3 ± 4.5 | 171.4 ± 7.9 | 179.2 ± 9.3 |
| Body weight (Kg) | 58.8 ± 6.2 | 72.6 ± 6.8 | 57.0 ± 6.1 | 68.1 ± 6.5 | 67.0 ± 10.3 |
| BMI | 20.4 ± 1.3 | 21.9 ± 1.4 | 19.7 ± 1.7 | 23.1 ± 1.2 | 20.9 ± 1.7 |
| 6 skinfold (mm) | 77.6 ± 13.0 | 51.5 ± 12.8 | 74.5 ± 8.4 | 109.9 ± 18.6 | 69.5 ± 8.4 |
| $VO_2$max (mL·Kg$^{-1}$ min$^{-1}$) | 54.5 ± 2.5 | 60.1 ± 4.2 | 45.9 ± 4.8 | 43.9 ± 4.5 | 50.6 ± 6.5 |
| $V_E$max (L·min$^{-1}$) | 109.0 ± 12.7 | 150.5 ± 20.9 | 96.5 ± 20.9 | 97.7 ± 10.1 | 125.7 ± 16.5 |

statement of the American Thoracic Society (ATS) and the European Respiratory Society (ERS) when performing the evaluations.[20] Each participant began the test sitting down with a mouthpiece and nose clip correctly positioned so that the gas mixture could not escape from the airway. The manoeuvre started with several basal breaths and, when the subject felt comfortable, he or she was asked to exhale fully into the residual volume (RV). Then, the technician connected the gas mixture to the spirometer and the subject rapidly inhaled up to the total lung capacity (TLC). After this, the technician instructed the participant to hold their breath for 10 s and then exhale completely without interruption in less than 4 s and to finish the test with a normal breath. The haemoglobin concentration was determined using a small capillary blood sample, used to adjust the $DL_{CO}$ to the individual parameters before starting the study. Intervals of at least 4 min were left between tests to ensure complete elimination of the gases. In this test, the CO is used to measure the diffusion properties of the alveolo-capillary membrane while the He is used as an inert reference gas to assess the alveolar volume (VA) and the rest of the lung parameters described. The following were also evaluated: the transfer coefficient of the lungs for CO ($K_{CO}$); the TLC, the inspired vital capacity ($VC_{IN}$) and the RV. The percentage, with respect to their reference for age and height (%-reference), of the pulmonary parameters was calculated according to the supplementary material in Stanojevic et al.[21]

### Incremental maximal test

An incremental maximal test was performed to characterize the aerobic capacity of each participant in the study. Maximum oxygen consumption ($VO_2$max) and maximum ventilation ($V_E$max) were determined. The test was performed on an ergometric treadmill, starting at a speed of 6 km/h and increasing by 1 km/h every minute until exhaustion. This laboratory test does not mimic the biomechanical characteristics of swimming, so it should be noted that the real physiological capacity of the participants could have been underestimated with respect to the physiological involvement during swimming in the aquatic environment.

### Ethical considerations

All study procedures followed the principles of the Declaration of Helsinki for human experimentation and were developed in accordance with the ethical standards of the Clinical Research Ethics Committee of the Direcció General de l'Esport del Consell Català de l'Esport (05-2020-CEICEGC). Informed consent was obtained from participants or their legal guardian before the study began.

### Statistical analysis

Lung parameters are described as mean value ± standard deviation (SD). The differences between groups in respiratory parameters were measured by a one-way analysis of variance (ANOVA). The level of statistical significance was set at $p < 0.05$. The software used for the statistical analysis was the StatGraphics 18 package.

## Results

### Physiological and anthropometric description

The anthropometric and physiological parameters collected from elite aquatic athletes are shown in Table 1 as mean ± standard deviation (SD). Male swimmers had significantly higher values of $VO_2$max (60.1 ± 4.2 vs. 50.6 ± 6.5 mL-Kg$^{-1}$ min$^{-1}$), $V_E$max (150.5 ± 20.9 vs. 125.7 ± 16.5 L-min$^{-1}$) and FVC (6.11 ± 1.0 vs. 4.87 ± 0.54 L) than male water polo players. Among the female athletes, female swimmers had higher values of $VO_2$max (54.5 ± 2.5 vs. 45.9 ± 4.8 vs. 43.9 ± 4.5 mL-Kg$^{-1}$min$^{-1}$) than artistic swimmers and water polo players. However, there were no significant differences in the $V_E$max (109.0 ± 12.7 vs. 96.5 ± 20.9 vs. 97.7 ± 10.1 L).

### Pulmonary spirometric parameters among different aquatic athletes

The pulmonary spirometric parameters collected from elite water athletes are shown in Table 2 as mean ± standard deviation (SD). Male swimmers had significantly higher values of FVC (6.11 ± 1.0 vs. 4.87 ± 0.54 L) and FEV1 (4.96 ± 0.78 vs. 4.10 ± 0.49 L) than male water polo players. On the other hand, there were no significant differences between female swimmers, artistic swimmers, and water polo

**Table 2** Comparison of forced spirometry pulmonary parameters in aquatic athletes from different disciplines: swimming, artistic swimming and water polo.

| | Swimmers | | Artistic swimmers | Water polo | |
|---|---|---|---|---|---|
| | Female | Male | Female | Female | Male |
| FVC (L) | 4.31 ± 0.40 | 6.11 ± 1.0* | 4.46 ± 0.45 | 4.20 ± 0.40 | 4.87 ± 0.54* [a] |
| FVC (%-reference) | 107 ± 8 | 116 ± 15 | 106 ± 10 | 97 ± 10 | 96 ± 6 |
| FEV1 (L) | 3.63 ± 0.40 | 4.96 ± 0.78* | 3.68 ± 0.50 | 3.35 ± 0.59[a],[b] | 4.10 ± 0.49* [a] |
| FEV1 (%-reference) | 104 ± 7 | 114 ± 15 | 104 ± 14 | 90 ± 15 | 98 ± 5 |
| FEV1/FVC | 84.24 ± 2.6 | 81.25 ± 5.2 | 82.57 ± 8.9 | 79.63 ± 10.6 | 84.39 ± 7.6 |
| PEF ($L{\cdot}s^{-1}$) | 6.78 ± 0.9 | 9.09 ± 2.0* | 6.84 ± 1.29 | 6.31 ± 1.01 | 8.16 ± 1.27* |
| MEF25-75 ($L\,s^{-1}$) | 3.73 ± 0.64 | 4.71 ± 0.91* | 3.65 ± 1.03 | 3.18 ± 1.14 | 4.20 ± 1.17* |

The values are expressed as mean ± standard deviation (SD).
* Significant differences by sex ($p < 0.05$).
[a] Significant differences compared to swimmers ($p < 0.05$).
[b] Significant differences compared to artistic swimmers ($p < 0.05$).

**Table 3** Comparison of the lung diffusion and volume capacity of aquatic athletes from different disciplines: swimmers, artistic swimmers, and water polo.

| | Swimmers | | Artistic swimmers | Water polo | |
|---|---|---|---|---|---|
| | Female | Male | Female | Female | Male |
| $DL_{CO}$ ($mL{\cdot}min^{-1}{\cdot}mmHg^{-1}$) | 34.7 ± 5.6 | 50.4 ± 5.3* | 33.4 ± 4.0 | 32.1 ± 5.6 | 43.4 ± 7.0* [a] |
| $DL_{CO}$ (%-reference) | 145 ± 18 | 151 ± 15 | 137 ± 15 | 123 ± 20 | 144 ± 17 |
| $K_{CO}$ ($mL{\cdot}min^{-1}{\cdot}mmHg^{-1}{\cdot}L^{-1}$) | 5.93 ± 0.73 | 5.98 ± 0.57 | 5.14 ± 0.65 | 5.83 ± 0.46 | 6.24 ± 0.67 |
| $K_{CO}$ (%-reference) | 125 ± 16 | 116 ± 11 | 109 ± 13 | 112 ± 9 | 126 ± 16 |
| VA (L) | 5.95 ± 0.78 | 8.36 ± 1.06* | 6.53 ± 0.53 | 5.48 ± 0.63[b] | 6.94 ± 0.61* [a] |
| VA (%-reference) | 117 ± 11 | 129 ± 14 | 124 ± 7 | 110 ± 13 | 116 ± 12 |
| TLC (L) | 6.09 ± 0.78 | 8.51 ± 1.06* | 6.66 ± 0.53 | 5.63 ± 0.63[b] | 7.09 ± 0.61* [a] |
| TLC (%-reference) | 116 ± 10 | 133 ± 18 | 124 ± 11 | 110 ± 13 | 115 ± 11 |
| $VC_{IN}$ (L) | 4.29 ± 0.89 | 6.44 ± 1.4* | 4.99 ± 0.32 | 4.17 ± 0.51[b] | 5.26 ± 0.52* [a] |
| RV (L) | 1.80 ± 0.69 | 2.07 ± 1.22 | 1.67 ± 0.39 | 1.46 ± 0.39 | 1.83 ± 0.36* |
| RV (%-reference) | 140 ± 63 | 135 ± 32 | 122 ± 26 | 127 ± 32 | 136 ± 25 |

The values are expressed as mean ± standard deviation (SD).
* Significant differences by sex ($p < 0.05$).
[a] Significant differences compared to swimmers ($p < 0.05$).
[b] Significant differences compared to artistic swimmers ($p < 0.05$).

players respectively in FVC (4.31 ± 0.40 vs. 4.46 ± 0.45 vs. 4.20 ± 0.40 L) and FEV1 (3.63 ± 0.40 vs. 3.68 ± 0.50 vs. 3.35 ± 0.59 L).

## Diffusing and lung capacity parameters among different aquatic athletes

The lung parameters of athletes from different aquatic disciplines are shown in Table 3 as mean ± standard deviation (SD). $DL_{CO}$ was higher in all aquatic athletes compared to their population reference for height and age, both in females (33.4 ± 9.4 $mL{\cdot}min^{-1}{\cdot}mmHg^{-1}$; 135%) and males (48.0 ± 5.83 $mL{\cdot}min^{-1}{\cdot}mmHg^{-1}$; 148%), and are presented by sport and sex in Table 3.

Regarding female athletes, there were no significant differences in $DL_{CO}$ between swimmers, artistic swimmers and water polo players (34.7 ± 8.3 vs. 33.4 ± 4.0 vs. 32.1 ± 5.6 $mL{\cdot}min^{-1}{\cdot}mmHg^{-1}$), while artistic swimmers had higher values of VA (6.53 ± 0.53 vs. 5.48 ± 0.63 L), TLC (6.66 ± 0.53 vs. 5.63 ± 0.63 L) and VCIN (4.99 ± 0.32 vs. 4.17 ± 0.51 L) than water polo players.

Regarding male athletes, swimmers had higher $DL_{CO}$ (50.4 ± 5.3 vs. 43.4 ± 7.0 $mL{\cdot}min^{-1}{\cdot}mmHg^{-1}$), VA (8.36 ± 1.06 vs. 6.94 ± 0.61 L), TLC (8.51 ± 1.06 vs. 5.26 ± 0.52 L) and $VC_{IN}$ (6.44 ± 1.40 vs. 5.26 ± 0.52 L) values than water polo players.

## Discussion

This study shows that aquatic athletes have higher lung capacity and gas diffusion than the reference population for their same height and age. By sport, male swimmers have higher lung capacity and diffusion than water polo players, while female artistic swimmers have higher lung capacity than water polo players.

The nature of the three water-based sports presented is different, but they all share, to a greater or lesser extent, movement in the water through swimming. It has previously

been reported that swimmers have larger lungs and better diffusion capacity than land-based athletes or untrained subjects,[9,22,23] but to the best of our knowledge, this is the first study analysing aquatic athletes of different modalities with a considerable number of subjects ($n = 64$). An augmented lung capacity increases the area available for gas exchange and improves flotation in the water, decreasing resistance to progression[24] and promoting performance in this sport.

Immersion of the body in water provokes a well-known physiological response named the ''immersion response''.[25] The immersion response *(diving response)* induces changes mediated by a vagal parasympathetic response in the cardiovascular and respiratory systems. On the one hand, there is a redistribution of peripheral blood flow towards the central circulation, and, on the other hand, peripheral vasoconstriction, increased blood pressure and decreased cardiac output.[26] All this together with the forces caused by the hydrostatic pressure of the water against the chest wall and the high resistance of the airways leads to an increase in the work of breathing during swimming compared to other disciplines such as cycling.[27]

The mechanics of breathing in swimming are complex, combining a forced-inspiration phase with the head out of the water with a prolonged exhalation phase with the head in the water. Therefore, swimmers must establish a controlled breathing pattern, which must be coordinated with the stroke mechanics and the swimming stroke.[9] Swimmers, and to a lesser extent water polo players, alternate short periods of apnoea with almost maximum inspirations while performing a long-duration exercise with high metabolic involvement. Artistic swimmers, in contrast, have long periods of apnoea for a shorter period of time, according to their artistic routine. Both set of conditions cause a low respiratory rate and high pulmonary vascular pressure that can stress the respiratory system through hyper-expansion of the chest wall, hypercapnia, hypoxia and mechanical loading.[28,29]

The exact mechanism by which lung volumes and diffusion capacity improve in swimmers is not known. However, all the aquatic athletes in this study had extremely well-developed lung capacities (female – VA: $6.00 \pm 9.38$ L; 135% and male – VA: $7.88 \pm 1.48$ L; 124%), and an increased diffusion capacity (female – $DL_{CO}$: 135% and male – $DL_{CO}$: 148%). It remains to be elucidated whether these parameters are the result of genetic endorsement or swimming training, although the second explanation seems to be more plausible. It has been reported that intensive swimming training in pre-pubescent children causes isotropic lung growth and better development of the airways and alveolar space.[30] Some metabolic pathways related to genes activated by hypoxia and mechanical strain in the alveolar capillaries seem to be related to lung growth. Both the lung expansion to TLC[24] and the exposure to repeated apnoea in the water[3] that occur during swimming could be the reason why swimmers,[9] free divers[11] and female swimmers[10] have greater lung capacity and diffusion than land-based athletes.

However, Armour et al.[22] demonstrated that the diffusion capacity is higher in swimmers due to their larger ribcage and a larger number of alveoli compared to runners and control subjects. However, the aquatic athletes in the current study had a higher $K_{CO}$ than reference values in both females ($115 \pm 12\%$) and males ($119 \pm 13\%$), suggesting that elite aquatic athletes also have a higher gas transfer coefficient in the lungs for a given alveolar surface.

During swimming, the requirements of rapid submaximum inspirations from the functional residual capacity (FRC) to TLC, for short periods of time, as well as the ventilatory restriction in each respiratory cycle during swimming, constitute a conditional stimulus of intermittent hypoxia and mechanical loading, already described as the main stimuli for the improvement of breathing.[3,31] In this study, the pulmonary parameters of swimmers and artistic swimmers exceeded those of water polo players. This coincides with the fact that both swimmers and artistic swimmers are exposed to a greater extent to the factors that promote improved lung function, such as the abovementioned mechanical strain and intermittent apnoea, suggesting that, in addition to the aquatic environment itself, the type of activity that takes place in the water is also important for improving lung function.

In this context, it has been reported that there is no additional benefit from respiratory muscle training as long as the swimmers train on an elite level basis.[9] Our interpretation of this phenomenon is that swimming training in itself is already a sufficient stimulus for the development of the respiratory muscles' strength, thus improving the pulmonary function of the practitioners. Therefore, it remains possible that other athletes or even patients with respiratory insufficiency could benefit from this characteristic of swimming by improving their functional lung capacity as well as their overall fitness. This is a factor to consider when performing rehabilitation of respiratory diseases in the aquatic environment.

In addition to the aquatic environment, the stroke mechanics combined with a low respiratory rate and intermittent apnoea could improve the prognosis of some lung diseases such as asthma[15] and COPD,[16] although those studies did not use swimming as an exercise method but instead used a combination of strength and mobility exercises in the aquatic environment *(aqua-gym)*. It has also been shown that the distance walked in the shuttle endurance test improved more after an exercise programme in the aquatic environment than on land.[17]

Evidence is currently limited regarding whether training in the aquatic environment offers more benefits than training in the terrestrial environment for improving aerobic capacity. Future studies should include swimming in these aquatic exercise protocols, due to the beneficial effects of mechanical stress and intermittent hypoxia, in addition to strength and mobility exercises, to increase the beneficial effects of aquatic therapy on lung function.

One of the main challenges of pulmonary physiology is to understand the mechanisms underlying lung plasticity, and to find ways to improve structural and functional capabilities.[32] Accordingly, the study of swimming exercise as a conditional model could be useful.

## Conclusions

The elite aquatic athletes studied had larger lungs and better lung diffusion capacity than their reference

population for the same age and height. Therefore, the practice of sports in the aquatic environment could help improve lung function. Specifically, male swimmers had better lung capacity and diffusion than water polo players, and female artistic swimmers had better lung capacity than female water polo players. Future research should include a longitudinal analysis to understand the impact of the practice of different water-based and land-based sports on lung function. The results of such studies would be relevant to the therapeutic exercises applied in the general population and to the rehabilitation of lung function in people with pathologies.

## Conflict of interests

The authors declare that they don't have any conflict of interests.

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