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Scientific Letter

Acute Changes in Lung Diffusing Capacity After Training in Elite Swimmers

Cambios en la capacidad de difusión pulmonar después del entrenamiento en nadadores de élite

Dear Editor:

Swimmers have larger lungs and higher diffusion capacity than other athletes and the general population,[1,2] but there is no clear evidence whether genetic predisposition, swimming training, or a mixture of both factors account for these changes. During incremental exercise, lung diffusion capacity for carbon monoxide (DL_{CO}) increases in a linear fashion with cardiac output following the distension of capillaries that are already perfused and/or the recruitment of additional capillaries that are not perfused under resting conditions[3] but DL_{CO} also has been decreased after exercise in some cases.[4,5] To the best of our knowledge there are no information about possible changes in the lung's alveoli capillary interface after swimming training, although swimming-induced pulmonary oedema (SIPO) has been reported in the literature, as a specific lung injury related to swimming.[6,7]

We evaluate the possible alterations in the pulmonary alveolar-capillary diffusion induced by swimming exercise before and after 10 training sessions over a 4-weeks training phase.

The participants were 21 junior elite swimmers, including 7 females and 14 males [16.1 ± 1.6 years, 21.6 ± 1.3 body mass index, 57.7 ± 3.63 VO_2max, 137.7 ± 18.6 $V_E$max, 110 ± 10 FVC (%-predicted), 108 ± 10 FEV1 (%-predicted)]. Measurements of DL_{CO} were performed pre-training and post-training along 10 swimming training sessions, conducted every 2–3 days, over a period of 4 weeks, with the objective of evaluating the full range of swimming training sessions faced by the swimmers, in terms of intensity, volume, and schedule. The measurements were taken less than 10 min pre- and post-training by a computerized spirometer (Ganshorn, PowerCube Diffusion+, Niederlauer, Germany) by means of the single-breath method and DL_{CO} was corrected to individual haemoglobin (Hb) concentration at the beginning of the study. Differences in DL_{CO} from pre-, to post-training were analyzed using a two-way repeated measures analysis of variance (2w-RM ANOVA) and statistical effects were established at $p < 0.05$. All procedures were in accordance with the ethical standards of the Clinical Research Ethics Committee at the Direcció General de l'Esport of the Catalonian Sports Council.

The changes of the lung diffusing capacity and lung volume along the follow-up period are described in Fig. 1, comparing the changes (%) between averaged pre-training DL_{CO} and the averaged post-training DL_{CO} along the 10 training sessions. There was a significant interaction between changes in DL_{CO} and training ($F = 17.32$, $p = 0.001$, $\eta^2_p = 0.55$), with a slight decrease in lung diffusion capacity for carbon monoxide (DL_{CO}) after swimming exercise during the follow-up (44.4 ± 8.1 to 43.3 ± 8.9 mL min^{-1} mmHg^{-1}, $p = 0.047$; Fig. 1).

Swimmers from this study had large alveolar volumes (125 ± 15%) and extremely developed basal lung diffusing capacity (DL_{CO}) (151 ± 21%). The exact mechanism behind the improved lung volume and diffusing capacity in elite swimmers remains unclear. The respiratory mechanics of swimming comprise a rapid out-of-water phase of forced inhalation and a relaxed underwater phase of prolonged exhalation, which must be coordinated with the stroke mechanics.[1] This condition modifies the respiratory mechanics towards a low respiratory frequency and a high tidal volume pattern which lead to increases in work of breathing[8] and inspiratory muscle strength.[9] Possible metabolic pathways related with hypoxia-activated genes and mechanical stress in the alveoli capillaries are also related with lung growth[10] suggesting that either repeated pulmonary expansion to TLC,[11] or repeated exposure to apnoeic periods[10] during swimming could stimulate lung growth in swimmers. A longitudinal study or a twin study should be performed to fully elucidate the influence of swimming training in lung function.

To the best of our knowledge, this is the first report assessing the changes of lung diffusion capacity in relation to swimming training. We show a consistent slight decrease in DL_{CO} (−2.5%) after swimming exercise, along 207 pre- to post-comparative evaluations. Some studies have already shown that lung diffusion is reduced after exercise in land-based athletes[4,5] leading to the speculation that exercise could trigger interstitial pulmonary oedema, but the novelty of this case report is the follow-up analysis of swimming, an aquatic-based sport specifically associated to pulmonary oedema.[6,7] The most important finding is the large inter-individual variability in the response of DL_{CO} to swim training along the follow-up. Fig. 1 shows individual change in DL_{CO} post-training. We show a decrement in averaged DL_{CO} ($\bar{x}DL_{CO}$)

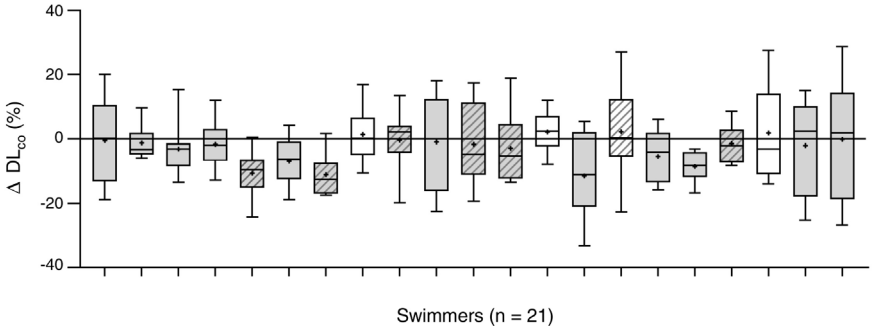

Fig. 1. Intra-individual changes in DL_{CO} after swimming exercise. Comparison of the pre-training DL_{CO} versus the post-training DL_{CO} along the 10 training sessions in the 21 participants. Individual data is showed as a box plot (average decrease, in grey; and average increase, in white). Mean (+), and median (line) values are also represented. Female swimmers are represented by diagonal lines inside their box plot and male swimmers with not-dashed box plots.

in 17 of 21 participants along the 10 training sessions evaluated. Large inter-individual differences in $\bar{x}DL_{CO}$ are also showed after swimming exercise, including 6 subjects showing a large decrease (−5.6–11.2%), 11 subjects with a small decrease (−0.4–3.1%) and 4 subjects showing a slight increase (+1.3–2.2%). Therefore, at least 6 of 21 participants of the study suffer a post-training diffusion limitation consistently more pronounced than the repeatability of DL_{CO} in healthy adults (±3.1%).[12]

Several causes has been proposed to explain this decrease, including the redistribution of central blood volume to peripheral areas[13] and the development of an exercise-induced pulmonary oedema.[4] Beside, the changes in Hb during training were not considered, which could account to some extent for the differences in DL_{CO} after training. The first possible explanation is the redistribution of the blood flow to the peripheral tissues after the training through a significant redistribution of fluid shift from the thorax to the peripheral vascular space.[13] In our study, we measured DL_{CO} less than 10 min after exercise and the decrease in DL_{CO} occurs despite a slight increase in the alveolar volume after training, which conflict with this hypothesis. The second possible explanation is the presence of swimming-induced pulmonary oedema (SIPO) during exercise which has been related to the ultra-structural mechanical stress in the pulmonary capillaries under a condition of high pulmonary artery and capillary pressures[14] such as swimming exercise. Currently there is no evidence as to why certain individuals are susceptible to SIPO, although symptoms normally resolve rapidly within 48 h[7] and do not provoke the development of clinically relevant pulmonary oedema which remains as a rare event.[15]

In summary, this study shows that swimmers experience subclinical decrease in lung diffusing capacity after training, although elite swimmers have larger lungs and higher diffusing capacity than the general population. Therefore, the swimming-induced decrement in DL_{CO} is a transient phenomenon that does not lead to chronic impairment in pulmonary gas exchange. In fact, we suggest that the highly developed pulmonary function of the elite swimmers could be the result of repeated stress to the alveolar-capillary barrier during training. We also found large inter-individual variability, including some swimmers with a large decrease in lung diffusing capacity after exercise. Therefore, doctors and coaches should pay attention to the individual changes in alveolar-capillary diffusing capacity among elite swimmers exposed to highly demanding training regimes.

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Therapeutic adherence of COPD patients according to levels of involvement in health education in their sites☆

Adhesión terapéutica de los pacientes con EPOC según los niveles de implicación en educación sanitaria de sus centros

To the Editor:

According to recent data obtained from the RE-TAI study, 48.5% of patients with chronic obstructive pulmonary disease (COPD) in Spain have poor therapeutic adherence[1] (determined by the Test of Adherence to Inhalers [TAI]).[2] When the TAI data were combined with information from pharmacy refill rate (PRR) electronic records, the percentage of patients with poor adherence increased to almost 55%.[1]

Factors influencing therapeutic adherence are heterogeneous and include, but are not limited to, patient education, inhaler ease-of-use, and the complexity of the therapeutic regimen.[3,4] Lack of adherence is, in turn, associated with poor disease control and reduced quality of life, and an increase in the use of resources and costs.[3] Consequently, acting on factors such as health education that improve the therapeutic adherence of COPD patients can contribute substantially to an improvement in the disease. It is estimated that reductions in morbidity and mortality among respiratory patients who followed an education program were due primarily to their increased therapeutic adherence. No studies have been conducted to evaluate differences in

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